ISSN:2362-1192(Print)
ISSN:2362-1206 (Online)

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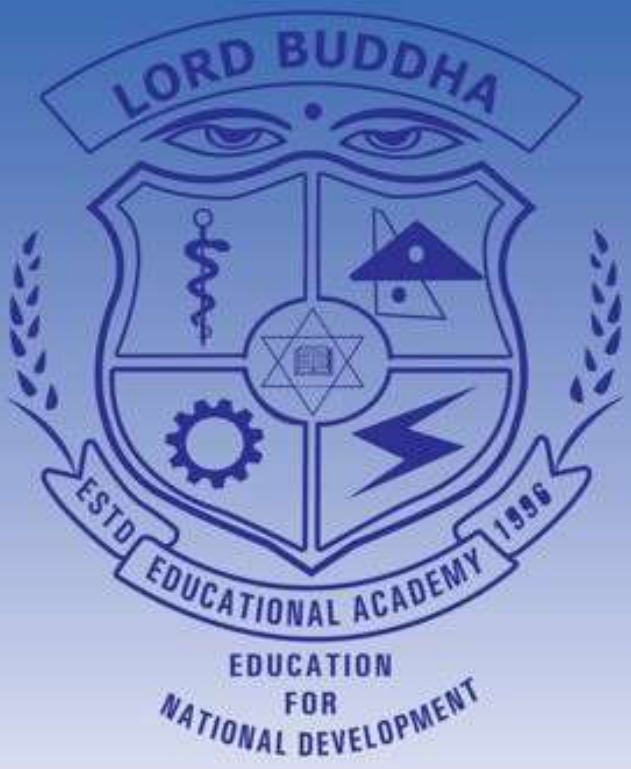
A Journal of Nepalgunj Medical College



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Peer Reviewed Journal

Vol.23 No.2
December 2025

Published by
NEPALGUNJ MEDICAL COLLEGE
Kohalpur, Banke (Nepal)

Vol. 23 No: 2 December 2025

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ORIGINAL ARTICLE

Clinical Patterns, Comorbidities and Stressors in Dissociative Disorders: A Hospital-Based Cross-Sectional Study

Bagban MA, Belbase M, Adhikari P

ABSTRACT

Introduction: Dissociative disorders are characterised by disruptions in memory, consciousness, identity and perception of the self and environment. The high rate of comorbidity complicates diagnosis, emphasising the need for a comprehensive evaluation. Psychosocial stressors play a pivotal role in the onset and exacerbation of the disorder. Despite their high prevalence, there is a lack of systematic research on their clinical patterns, comorbidities, and psychosocial stressors. Most existing literature focuses on case studies or small-scale investigations. A detailed exploration of these aspects of the disorder facilitates early identification and management, ultimately enhancing patient outcomes. **Aims:** To analyse the clinical presentations, comorbid psychiatric conditions, and psychosocial stressors in dissociative disorders. **Methods:** A descriptive cross-sectional study was conducted from April 2025 to June 2025 at the Psychiatry Department of Nepalgunj Medical College among 50 patients aged 18-60 years, diagnosed with dissociative disorders, enrolled by convenience sampling. Unco-operative patients, those with severe psychiatric/medical illnesses, or cognitive disorders, were excluded. Sociodemographic and clinical data were recorded using a semi-structured proforma. **Results:** The majority of patients (88%) were from the age group 16 to 35, female (86%), married (60%), residing in a rural area (62%), belonging to nuclear families (70%) with lower middle-economic status (40%) and middle school education (32%) and anxiety disorder as a comorbidity. The most common clinical presentation was non-epileptic seizures, with the commonest stressor being family conflict. **Conclusion:** Our findings emphasise the need for family-based intervention as well as early psychosocial intervention and mental health support targeting these vulnerable groups.

***Keywords**:* Clinical Patterns, Comorbidity, Dissociative disorders, Stressor

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INTRODUCTION

Dissociative disorders (DDs) are a range of psychiatric conditions characterized by disruptions in memory, consciousness, identity, and perception of the self and environment, often manifested in response to psychological stress or trauma.[1] The prevalence of DDs varies across populations, influenced by cultural, psychological, and biological factors. Despite their clinical significance, DDs remain underdiagnosed and poorly understood, leading to challenges in treatment and management. Individuals with dissociative disorders frequently present with a wide spectrum of symptoms, including amnesia, depersonalization /derealization, identity disturbances, and conversion symptoms, often creating a diagnostic dilemma. The clinical presentation often overlaps with other psychiatric disorders, such as depression, anxiety disorders, post-traumatic stress disorder (PTSD), and somatic symptom disorders. This high rate of comorbidity complicates diagnosis and underscores the need for a comprehensive evaluation of associated mental health conditions. Psychosocial stressors play a pivotal role in the onset and exacerbation of DDs.[2] Childhood trauma, abuse, neglect, interpersonal conflicts, and adverse life events have been identified as major risk factors. Despite the growing recognition of DDs, there remains a lack of systematic research on their clinical patterns, comorbidities, and associated stressors in hospital-based settings. Most existing literature focuses on case studies or small-scale investigations. A detailed exploration of these aspects can contribute to a deeper understanding of these disorders, which could aid in early identification, ultimately improving patient outcomes. This hospital-based cross-sectional study aims to bridge this gap by analyzing the clinical presentations, comorbid psychiatric conditions, and psychosocial stressors in patients diagnosed with DDs.

METHODS

This study followed a hospital-based descriptive cross-sectional study design and was conducted at the Department

of Psychiatry, Nepalgunj Medical College Teaching Hospital, Kohalpur. A convenient sampling method was applied among the patients attending the psychiatry OPD and IPD from April 2025 to June 2025. The study population consisted of patients diagnosed with dissociative disorders. The diagnosis of dissociative disorder and its subtypes was made according to the eleventh revision of the International Classification of Diseases (ICD-11) by a registered psychiatrist. Patients aged 18-60 years diagnosed with a dissociative disorder were included. Uncooperative patients who were not willing to consent, patients with severe psychiatric disorders, severe cognitive impairment, and severe medical illness requiring intensive care were excluded. The calculated sample size was 50, considering the prevalence of dissociative disorder was 10%,[3] with 8.5% margin of error and 95% confidence interval. Ethical approval was obtained from the Institutional Review Committee (Ref. 61/081-082), Nepalgunj Medical College Teaching Hospital, Kohalpur. All the patients were informed about the purpose of the study in detail, and written informed consent was obtained. For patients who were unable to give consent, written consent was taken from their parents or guardians. The identity of the respondents and their responses were kept confidential. Patients were evaluated and diagnosed based on ICD-11 criteria. After taking a detailed history, a semi-structured pro forma was used to collect information about socio-demographic and clinical variables as well as types of stressors. The categorisation of stressors was done based on the most common types of stress mentioned in the presumptive stressful life events scale given by Singh et al (1984).[4]

Statistical Analysis

Descriptive statistics were performed to summarise the demographic characteristics and clinical findings of the study population. Continuous variables (e.g., age) were presented as mean ± standard deviation (SD). Categorical variables (e.g., gender) were summarised as frequencies and percentages. All data analyses were performed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Table I shows the sociodemographic variables of the study population. Among the 50 participants, the majority were female (86.0%), while only 14.0% were male. The age of the participants ranged from 18 to 54 years, with a mean age of 26.48 years and a standard deviation of 7.96. The majority of patients (44.0% each) were within the age group of 18-24 years and 25-34 years, followed by 35-44 years (8.0%). Only a smaller proportion (4.0%) was from the older age group, 45–54 years. This indicates that most participants were young adults, with some variability in age distribution across the sample. Most participants (62.0%) were from rural areas, and the remaining 38.0% were from urban settings. A majority (70.0%) of the respondents were identified as Hindu, while Christianity, Buddhism, and Islam were practised by 14.0%, 12.0%, and 4.0% of the participants, respectively. The majority of respondents were married (60.0%), while 38.0% were unmarried and 2.0% were either divorced or widowed. More than half of the participants were unemployed (54.0%). Unskilled workers made up 18.0%, semi-skilled and skilled workers accounted for 6.0% each, 10.0% were clerks or shop owners, 4.0% were semi-professionals, and only 2.0% were professionals. The largest proportion had a middle school level of education (32.0%), followed by high school (28.0%), and primary school and diploma holders each accounted for 18.0%, 2.0% were illiterate, and 2.0% were post-graduates.

Sociodemographic variables		Frequency (Percentage)
Sex	Female	43 (86.0)
	Male	7 (14.0)
Age	18-24	22 (44.0)
	25-34	22 (44.0)
	35-44	4 (8.0)
	45-54	2 (4.0)
Residence	Rural	31 (62.0)
	Urban	19 (38.0)
Religion	Hindu	35 (70.0)
	Christianity	7 (14.0)
	Buddhism	6 (12.0)
	Islam	2 (4.0)
Marital status	Married	30 (60.0)
	Unmarried	19 (38.0)
	Divorced or widowed	1 (2.0)
Occupation	Unemployed	27 (54.0)
	Unskilled	9 (18.0)
	Clerk, shop owner	5 (10.0)
	Skilled	3 (6.0)
	Semi-skilled	3 (6.0)
	Semi-profession	2 (4.0)
	Profession	1 (2.0)
Education	Illiterate	1 (2.0)
	Primary School	9 (18.0)

Education	Middle school	16 (32.0)
	High school	14 (28.0)
	Diploma	9 (18.0)
	Postgraduate	1 (2.0)
Type of family	Nuclear	35 (70.0)
	Joint	11 (22.0)
	Extended	4 (8.00
Family history of dissociative disorder	Present	10 (20.0)
	Absent	40 (80.0)
Socioeconomic status	Lower-lower	7 (14.00)
	Lower upper	16 (32.0)
	Lower middle	20 (40.0)
	Upper middle	7 (14.0)

Total study population (n)=50. Mean age of the study sample =26.48±7.96 years

Table I: Demographic and clinical profiles of the patients

The majority (70.0%) belonged to nuclear families, followed by joint (22.0%) and extended families (8.0%). Less than 1/3rd (20%) of the patients had a family history of dissociative disorder. The highest proportion of participants (40.0%) belonged to the lower middle socioeconomic class. The lower upper class made up 32.0%, while both the lower-lower and upper middle classes each accounted for 14.0% of the participants.

Clinical patterns	Frequency (Percentage)
Non-epileptic seizure	22 (44.0)
Trans and possession	19 (38.0)
Paresis or weakness	4 (8.0)
Speech disorder	2 (4.0)
Visual disturbance	2 (4.0)
Sensory disturbances	1 (2.00)
Total	50 (100.0)

Table II: Distribution of Clinical Patterns Among Study Participants (N = 50)

The clinical presentation of the participants was varied (Table II). Non-epileptic seizures, reported by 44.0% of the participants, were the most frequent clinical presentation. Trans and possession states were the second most common, observed in 38.0% of cases. Paresis or weakness was reported by 8.0% of participants. Speech disorders and visual disturbances were each reported by 4.0%. The least common symptom was sensory disturbance, seen in 2.0% of cases.

Type of stressor	Frequency (Percentage)
Family conflict	9 (18.0)
Academic	6 (12.00)
Divorce or separation	5 (10.0)
Financial	4 (8.0)
Physical or sexual abuse	3 (6.0)
Death of close family member	2 (4.0)
Work related	2 (4.0)
Personal injury	1 (2.0)
Marriage	1 (2.0)
None	17 (34.0)
Total	50 (100.0)

Table III: Distribution of Stressors Among Study Participants (N = 50)

Out of the total participants, the majority (66%) reported an identifiable stressor; the most frequently reported stressor was family conflict, reported by 18.0% of respondents (Table III). Academic-related stress was noted by 12.0% of participants. Divorce or separation was reported by 10.0%, while financial stress affected 8.0% of individuals. Physical or sexual abuse was reported by 6.0% of participants. Work-related stress and the death of a close family member were each reported by 4.0%. Marriage and personal injury/illness were the least reported stressors, each by 2.0% of respondents. Notably, 34.0% of participants reported experiencing no identifiable stressor.

Comorbidity	Frequency (Percentage)
Anxiety	12 (24.0)
Depression	11 (22.0)
Bipolar affective disorder	9 (18.0)
Personality disorder	4 (8.0)

Substance use disorder	3 (6.0)
Post-traumatic stress disorder	2 (4.0)
Others	1 (2.0)
None	8 (16.0)
Total	50 (100.0)

Table IV: Distribution of Psychiatric Comorbidities Among Study Participants (N = 50)

In terms of psychiatric comorbidities among the participants, anxiety disorders were the most reported comorbidity, present in 24.0% of participants (Table IV), which was followed by depression in 22.0% and bipolar affective disorder (BPAD) in 18.0% of cases. Personality disorders were reported by 8.0% of participants. Substance use disorders were present in 6.0%, and Post-Traumatic Stress Disorder (PTSD) was seen in 4.0%. Other psychiatric conditions accounted for 2.0% of cases. Notably, 16.0% of the participants reported no psychiatric comorbidity.

DISCUSSION

This study aimed to explore the clinical patterns, psychiatric comorbidities and psychosocial stressors in patients diagnosed with dissociative disorders at a tertiary hospital. One of the most striking findings was a significantly higher predominance of female patients, which constituted 86.0% of the study sample. This finding, however, is consistent with multiple prior studies where the majority of patients with dissociative disorders were females, with proportions ranging from 80% to 86%.[5,6] In a study by Mohammad Y et al, in India, dissociative stupor was reported in 77% of females, supporting this gender disparity.

This gender difference may be attributed to socio-cultural as well as emotional factors. In many traditional societies, women often grow up in emotionally restrained environments with limited opportunities for emotional expression. The onset of puberty, societal expectations and gender roles may further compound emotional stress, contributing to the higher incidence of dissociative symptoms among females.

In terms of age distribution, the participants in our study ranged from 18 to 54 years, with a mean age of 26.48 ± 7.96 years. These findings are comparable to other studies that reported similar age distributions, including those by Chowdhury JM et al[7], and Sharma et al.[2] Like the finding by Sharma et al where the majority of patients were from the 21 to 30 age group;[5] our study also showed participants (44.0% each) in the age groups of 18-24 and 25-34 years, indicating that young adulthood remains a vulnerable period for the development of dissociative disorders. A significant portion of our sample (62.0%) came from rural backgrounds, with 70.0% from nuclear families. These findings are in concordance with multiple prior studies, including that by Nizam et al, that observed a similar pattern, with 66.66% of cases from rural areas and 74.50% from nuclear families.[8] Limited mental health resources, stigma, and lack of awareness in rural communities may contribute to the higher reporting or emergence of dissociative disorders in such populations. Higher prevalence in nuclear families may be attributed to epidemiological trends, which show a growing number of nuclear families, as well as the lack of a proper support system in nuclear families. Additionally, 60.0% of our participants were married, reflecting earlier findings that showed dissociative disorders, more common in married individuals, with reported rates up to 74%.[5] Marital responsibilities, interpersonal conflicts, and social pressures could serve as major stressors contributing to symptom manifestation in this group.

Educationally, the largest subgroup had middle school education (32.0%) and 54.0% were unemployed, with 40.0% belonging to the lower middle socioeconomic class. These figures align closely with other studies, where most participants were young adults, females, unemployed, from rural settings, low socioeconomic backgrounds, and nuclear families.[9] These socioeconomic variables likely reflect the limited coping resources and increased vulnerability in individuals from disadvantaged backgrounds. Regarding clinical presentation, the most frequently observed symptom in our study was non-epileptic seizures, reported by 44.0% of patients. This is consistent with clinical patterns described by Bhat et al[10] who noted that dissociative convulsions were the most common form of dissociation. Similarly, Gupta et al[11] reported pseudo-seizures as the predominant presentation (29.7%). These manifestations might be socially acceptable ways of expressing psychological distress, especially in communities where direct expression of emotional turmoil is discouraged. The most frequently reported psychosocial stressor in our sample was family conflict (18.0%). This aligns with findings by Reddy et al[12] who reported family disharmony in the majority (41.82%) of their cases, and Thapa et al[13] who also found family conflict as the most common precipitating factor. Another study observed that 44% of patients experienced stress related to family problems.[14] In general, the literature highlights that stressors such as discord with in-laws, forced marriage, and strained relationships with spouses or parents are highly prevalent in dissociative disorder cases.[15]

From a psychiatric standpoint, anxiety disorders were the most common comorbidity in our study (24.0%), followed by depression (22.0%). This is consistent with other findings that show anxiety frequently coexisting with dissociative symptoms.[16] However, some studies, including that by Thapa et al, have reported a higher prevalence of depression (35%) in comparison to anxiety in these patients.[17] These variations may be influenced by the personal judgement of the clinician due to overlapping symptoms between depression and anxiety, as well as diagnostic criteria used, population differences, and the nature of clinical assessments.

LIMITATIONS

The study was conducted in a single tertiary care hospital. It is cross-sectional nature does not allow for establishing a causal

relationship between psychosocial stressors and dissociative symptoms. A relatively small sample size in our study may limit its generalisability. No standardised tool was used to quantify the degree of psychosocial stressors. Recollection of stressors during the interview can have some recall bias. The diagnoses were made clinically, which may be subject to individual bias.

CONCLUSION

Our study found a higher prevalence of dissociative disorders among young adult females, particularly those from rural backgrounds, nuclear families, and lower socioeconomic strata. The most common clinical presentation was non-epileptic seizures, with family conflict emerging as the leading psychosocial stressor. Anxiety and depressive disorders were the most frequent psychiatric comorbidities. These findings highlight the importance of integrating early psychosocial intervention and mental health education into treatment plans for better outcomes, especially targeting this vulnerable population. Psychoeducation to family members and family-based therapies could be of great help. Also, the higher prevalence of associated psychiatric comorbidity highlights the need for routine screening of other psychiatric disorders in patients presenting with dissociative symptoms.

REFERENCES

1. Diagnostic and Statistical Manual of Mental Disorders | Psychiatry Online [Internet]. DSM Library. [cited 2025 Nov 11]. Available from: https://psychiatryonline.org/doi/book/10.1176/appi.books.9780890425787

2. Frankel AS, Dalenberg C. The Forensic Evaluation of Dissociation and Persons Diagnosed with Dissociative Identity Disorder: Searching for Convergence. Psychiatr Clin North Am [Internet]. 2006 [cited 2025 Nov 11];29:169–84. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0193953X05000857

3. Tutkun H, Sar V, Yargiç LI, Özpulat T, Yanik M, Kiziltan E. Frequency of Dissociative Disorders Among Psychiatric Inpatients in a Turkish University Clinic. Am J Psychiatry [Internet]. 1998 [cited 2025 Jul 17];155:800–5. Available from: https://psychiatryonline.org/doi/10.1176/ajp.155.6.800

4. Singh G, Kaur D, Kaur H. PRESUMPTIVE STRESSFUL LIFE EVENTS SCALE (PSLES) — A NEW STRESSFUL LIFE EVENTS SCALE FOR USE IN INDIA. Indian J Psychiatry [Internet]. 1984 [cited 2025 Oct 29];26:107–14. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3012215/

5. Sharma S, Halder A, Kumar K, Shahani R, Ravindran NP, Kulkarni Y, et al. Psychiatric comorbidities in patients with conversion disorder – A longitudinal study. Telangana J Psychiatry [Internet]. 2023 [cited 2025 Mar 30];9:128–33. Available from: https://journals.lww.com/10.4103/tjp.tjp_35_23

6. Shastri R, Mohanty R, Sahoo S. Dissociative Experiences and Stressful Life Events in Dissociative Disorders - A Cross Sectional Study. Univers J Public Health [Internet]. 2021 [cited 2025 Mar 27];9:477–83. Available from: http://www.hrpub.org/journals/article_info.php?aid=11717

7. Chowdhury JM, Saha S. Socio-demographic and Clinical Profile of Persons with Dissociative Disorder. Natl J Prof Soc Work [Internet]. 2021 [cited 2025 Mar 30];22. Available from: https://pswjournal.org/index.php/njpsw/article/view/285

8. Nizam Ud Din Dar, Abdul Majid Gania, Tajamul Hussain Dhar, Aijaz Mohi Ud Din Bhat. Correlative analysis of dissociative disorder among Kashmiri population. Asian J Med Sci [Internet]. 2024 [cited 2025 Mar 27];15:226–32. Available from: https://www.nepjol.info/index.php/AJMS/article/view/62819

9. Bhusan S, Soni A, Jain S. Clinical Profile of Patients with Conversion Disorder: A Cross-Sectional Study Int J Acad Med Pharm [Internet]. 2023[cited 2025 Mar 27] 5 (4); 313-317 Available from: https://academicmed.org/Uploads/Volume5Issue4/64.%20[1001.%20JAMP_Dheerap%20Singh]%20313-317.pdfhttps://academicmed.org/Uploads/Volume5Issue4/64.%20[1001.%20JAMP_Dheerap%20Singh]%20313-317.pdf

10. Bhat M, Kakunje A, Mithur R. A Study of Clinical Profile and Stressors in Patients Presenting with Dissociative Disorder to a Tertiary Care Teaching Hospital. Indian J Soc Psychiatry [Internet]. 2024 [cited 2025 Mar 30];40:84–9. Available from: https://journals.lww.com/10.4103/ijsp.ijsp_349_21

11. Gupta AK, Saini M, Singh TB, Rai M. Socio-demographic factors and pattern of stressor in patients with conversion disorder. 2023;

12. Reddy LS, Patil NM, Nayak RB, Chate SS, Ansari S. Psychological Dissection of Patients Having Dissociative Disorder: A Cross-sectional Study. Indian J Psychol Med [Internet]. 2018 [cited 2025 Mar 30];40:41–6. Available from: https://journals.sagepub.com/doi/10.4103/IJPSYM.IJPSYM_237_17

13. Thapa R. Dissociative disorders: A study of clinico-demographic profile and associated stressors. J Psychiatr Assoc Nepal [Internet]. 2015 [cited 2025 Mar 30];3:25–30. Available from: https://www.nepjol.info/index.php/JPAN/article/view/12386

14. Mohammad Y, Kumar R, Sinha N, Kumar P. A study of stressors, family environment, coping patterns, and family burden in persons with dissociative disorder. Ind Psychiatry J [Internet]. 2023 [cited 2025 Mar 28];32:317–22. Available from: https://journals.lww.com/10.4103/ipj.ipj_42_23

15. Anuradha, Srivastava M, Srivstava M. A Comparative Study of Psychosocial Factors In Male and Female Patients of Conversion Disorder. Indian J. Prev. Soc. Med Researchgate [Internet]. [Cited 2025 Jul 7]; 219(3) 231-236 Available from: https://www.researchgate.net/publication/344341535_A_COMPARATIVE_STUDY_OF_PSYCHOSOCIAL_FACTORS_IN_MALE_AND_FEMALE_PATIENTS_OF_CONVERSION_DISORDER

16. Samanta S, Nandi S, Saha I, Roy S, Bandyopadhyay G. A study to assess perceived stress, life events and prevalence of dissociative experiences in patients with anxiety disorders. Int J Res Med Sci [Internet]. 2024 [cited 2025 Mar 27];12:2428–35. Available from: https://www.msjonline.org/index.php/ijrms/article/view/13638

17. Thapa R. Dissociative disorders: A study of clinico-demographic profile and associated stressors. J Psychiatr Assoc Nepal [Internet]. 2014 [cited 2025 Mar 27]Diarrhoea may be underestimated: a missing link in 2019 novel coronavirus. Gut. 2020 Jun 1;69(6):1141-3.

ORIGINAL ARTICLE

A Comparative Study of Automated Blood Pressure Device and an Approved Standard Blood Pressure Measuring Device in Young Healthy Population

Jha RK[1], Shrewastwa MK[2], Poudel A[3]

ABSTRACT

Introduction: Blood pressure is a key indicator of cardiovascular health. Automated devices are widely used for convenience, but their accuracy compared with the auscultatory method in young healthy adults remains under investigation. **Aims:** To determine whether automated and auscultatory blood pressure measurement methods show clinically meaningful differences in a young healthy cohort and whether sex or arm differences influence readings. **Methods:** A comparative cross-sectional study using purposive sampling was conducted among 80 medical students aged 20–30 years. Although student samples may not represent the general population, they provide controlled and stable baseline physiological conditions for comparative device evaluation. Blood pressurewas measured three times per arm using an automated device and a standard auscultatory method under stable room temperature, controlled lighting, and a quiet environment. **Results:** Systolic blood pressure measured by the auscultatory method was significantly higher than that obtained by the automated device (right arm: mean difference 2.94 mmHg; left arm: 2.50 mmHg). Diastolic blood pressure differences were not statistically significant. Inter-arm differences were minimal. Males had higher systolic BP than females across both arms, while diastolic differences were negligible. **Conclusion:** Automated devices slightly underestimate systolic blood pressure compared with the auscultatory method in young healthy adults, while diastolic readings are comparable. Automated monitors are suitable for screening and home monitoring, but clinical decisions near systolic thresholds should preferentially rely on validated auscultatory measurements.

Keywords: Auscultatory method, Automated device, Blood pressure, Inter-Arm difference, Sex variation, Young adults

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INTRODUCTION

Blood pressure (BP) is a critical cardiovascular biomarker. The auscultatory method remains the gold standard but requires training and is prone to observer error.[1-3] Automated BP devices are widely used because they are convenient and suitable for home monitoring[4,5] and with wearable monitors, device accuracy has become an important concern.[6] Accurate BP measurement in young adults is important because they undergo limited routine health checks despite risk for early-onset hypertension that may persist into later life. Device variability may cause misclassification and delay intervention.[7,8] Hypertension is a major concern in South Asia, including Nepal. A meta-analysis reported hypertension and prehypertension prevalence of 27.3% and 35.4%.[9] National surveys and gender comparisons show substantial BP burden[10,11,] though these reflect general adults rather than younger cohorts. Studies among Nepalese students show similar trends: in 250 medical students, prehypertension and hypertension were 20.8% and 4%, with basal metabolic index-(BMI) significantly associated.[12] Among 189 nursing students, prehypertension was 11.1%, with modest anthropometric correlations.[13] In 350 medical students in Pokhara, BP correlated strongly with BMI.[14] Adolescents (15-19 years) showed prevalence of 20.8% and 7.1%.[15] Although guidelines do not recommend sex-specific BP thresholds, national data report higher prevalence in men.[16] BMI is strongly associated with BP, whereas body fat percentage is less predictive.[14] Autonomic studies show increased sympathetic activity with higher body mass.[17] Given rising BP in young adults and limited validation of oscillometric devices, accurate assessment is essential. Medical students are an appropriate group to compare automated and auscultatory BP measurements, including possible sex-based variation.

METHODS

A comparative cross-sectional study was conducted at Nepal-

gunj Medical College. Ethical approval was granted by the institutional review board (Approval No. Ref 43/081-082). Eighty medical students (43 females, 37 males; age 20–30 years) voluntarily participated. Participants with a known history of cardiopulmonary disorders were excluded. All provided written informed consent. Sample size was calculated using the formula described by Al-Metha et al[18] with a population of 100 first-year MBBS students. The required sample was 80. Blood pressure was measured three times per arm using a validated automated oscillometric monitor (OMRON Smart Elite+ HEM-7600T) and a standard auscultatory method with a calibrated mercury sphygmomanometer and stethoscope. Measurements were conducted in a quiet, well-lit room at a stable ambient temperature of 22–24 °C. Participants rested for at least ten minutes in a seated position, with the arm supported at heart level, feet flat on the floor, and back supported. Body temperature was recorded to ensure participants were normothermic. Mean values of the three readings per arm were calculated and used for analysis.

Statistical Analysis

Paired-sample t-tests compared automated and auscultatory readings per arm and inter-arm differences. Independent-sample t-tests compared sexes. Statistical significance was set at $p < 0.05$.

RESULTS

Comparison of Systolic and Diastolic BP: Systolic blood pressure measured by the auscultatory method was significantly higher than that obtained by the automated device. The mean difference was 2.94 mmHg for the right arm and 2.50 mmHg for the left arm. Diastolic BP differences were not statistically significant (Table I).

Inter-Arm Differences: Inter-arm differences for both systolic and diastolic BP were minimal and not statistically significant, indicating consistency between the right and left arms in this population (Table II).

Gender-Based Differences: Males had higher systolic BP than females across both arms, while diastolic BP differences were negligible. These differences were observed in both auscultatory and automated measurements (Table II)

Arm	Auscultatory (mmHg)	Automated (mmHg)	P-Value
Right Arm (Systolic)	119.61 ± 6.10	116.67 ± 8.11	0.0013
Left Arm (Systolic)	119.01 ± 6.86	116.51 ± 8.02	0.0066
Right Arm (Diastolic)	73.93 ± 5.29	74.71 ± 8.78	>0.05
Left Arm (Diastolic)	73.93 ± 5.29	74.71 ± 8.78	>0.05

Table I: Comparison of Automated vs Auscultatory Blood Pressure Readings

Category	Parameter (mmHg)	Males (Mean ± SD)	Females (Mean ± SD)	P-Value	Method
Inter-arm Differences	Systolic	119.9 ± 6.5	116.9 ± 6.9	>0.05	Auscultatory
	Diastolic	74.8 ± 5.4	73.1 ± 5.2	>0.05	Auscultatory
	Systolic	117.2 ± 7.8	115.1 ± 7.5	>0.05	Automated
	Diastolic	75.1 ± 8.2	73.6 ± 8.1	>0.05	Automated
Gender Differences	Right-arm Systolic	122.2 ± 6.0	117.4 ± 5.8	0.0013	Auscultatory
	Left-arm Systolic	121.5 ± 6.8	116.2 ± 6.5	0.0066	Auscultatory
	Right-arm Diastolic	74.8 ± 5.3	73.1 ± 5.1	>0.05	Auscultatory
	Left-arm Diastolic	74.7 ± 5.3	73.0 ± 5.0	>0.05	Auscultatory
	Right-arm Systolic	120.0 ± 8.1	115.5 ± 7.9	0.0021	Automated
	Left-arm Systolic	119.2 ± 8.0	114.8 ± 7.8	0.0034	Automated
	Right-arm Diastolic	75.2 ± 6.2	73.5 ± 5.8	>0.05	Automated
	Left-arm Diastolic	75.1 ± 6.1	73.4 ± 5.7	>0.05	Automated

Table II: Comparison of Inter-Arm and Gender Differences in Blood Pressure (Auscultatory vs Automated)

DISCUSSION

In this pilot study of healthy young adults, systolic blood pressure (SBP) measured by the auscultatory method was modestly but significantly higher (~2–3 mmHg) than readings from a validated automated oscillometric device, while diastolic BP (DBP) was similar. Although the difference is small, systematic underestimation by automated devices may be clinically relevant, particularly for individuals near diagnostic or treatment thresholds, as this bias could lead to misclassification or delayed recognition of elevated BP.[19] These findings are consistent with prior research: a study of 337 adults reported that SBP measured by a mercury manometer was on average 1.95 mmHg higher than oscillometric readings[20] and a systematic review indicated that oscillometric devices may be less accurate than auscultatory measurements in certain populations, though generally sufficient for routine clinical use.[21]

Males in our cohort had higher SBP than females, while DBP was similar. Such sex-based differences may reflect vascular compliance, autonomic regulation, and hormonal influences. Women exhibit enhanced vasodilatory responses mediated by nitric oxide (NO), and estrogen can upregulate endothelial β1- and β3-adrenoceptors, promoting vasorelaxation.[22,23] Autonomic regulation also contributes: females often show blunted sympathetic vasoconstrictor responsiveness and enhanced NO-mediated sympatholysis, which may lower SBP relative to males.[24] These physiological differences can influence the oscillometric pulse waveform, potentially causing systematic underestimation of SBP in certain subgroups. Similar patterns have been observed in young adults in Nepal and India, where males generally have higher SBP and BMI is positively correlated with BP.[23,24]

Automated devices require local validation, as accuracy may vary by device model and patient characteristics. For instance, the Microlife BP3T01 1B device demonstrated mean differences of –2.56 ± 7.53 mmHg for SBP and –3.10 ± 5.65 mmHg for DBP in validation studies.[25] Other home BP monitors have shown variable accuracy, with several failing ISO 81060-2 standards.[26] Device performance may also be influenced by arm circumference and body size, with larger arms associated with greater SBP underestimation.[27]

From a public health perspective, underestimation of SBP in young adults may impede early detection and intervention. Young adults are often considered low risk and may not undergo routine BP screening. Systematic underreading, particularly in males or individuals with higher BMI, could delay lifestyle or pharmacological interventions. Accurate measurement is crucial, as BP trajectories established in early adulthood strongly predict long-term cardiovascular risk. Our findings support the need for local validation of automated BP devices in Nepal, and suggest that screening protocols incorporate confirmatory auscultatory measurements or repeated automated readings to minimize misclassification.

Strengths of this study include the within-subject comparison of two measurement methods, repeated measurements to reduce random error, and focus on a low-comorbidity young adult population. Limitations include the small convenience sample, use of a single device model, and absence of direct vascular or body-composition measurements (e.g., arterial stiffness, lean mass, endothelial function), which may help explain the observed bias. Future research should include larger, community-based validation studies across Nepal, compare multiple automated devices to assess device-specific biases, investigate physiological determinants of measurement discrepancies, and develop screening strategies that combine automated and auscultatory measurements to reduce misclassification risk.

CONCLUSION

In this study of healthy young adults, automated oscillometric devices modestly underestimated systolic blood pressure (SBP) compared to the auscultatory method, while diastolic blood pressure (DBP) was similar between techniques. Males exhibited higher SBP than females, reflecting known physiological differences, whereas inter-arm variations were minimal and not clinically significant. These findings underscore the importance of validating automated BP devices in local populations, carefully interpreting readings near diagnostic thresholds, and incorporating improved screening strategies to ensure accurate detection of elevated blood pressure in young adults. Automated devices remain suitable for routine screening and home monitoring, but clinical decisions should rely on validated auscultatory measurements when precision is critical.

REFERENCES

1. O'Brien E, Asmar R, Beilin L, et al. Comparison of automated blood pressure measurement devices and conventional cuff-based devices. Hypertension. 2000;36(5):899–904.
2. Pickering TG, Hall JE, Appel LJ, et al. Recommendations for blood pressure measurement in humans and experimental animals. Hypertension. 2005;45(1):142–61.
3. Stergiou GS, Palatini P, Asmar R, et al. Blood pressure measurement: recommendations of the European Society of Hypertension. J Hypertens. 2004;22(5):1077–88.
4. Liu Y, Li Y, Zhang L, et al. Hypertension in Nepal following application of the 2017 ACC/AHA guideline. JAMA Netw Open. 2020;3(6):e208724. doi:10.1001/jamanetworkopen.2020.8724
5. Karki P, Shrestha S, Koirala S, et al. Trends in the prevalence, awareness, treatment, and control of hypertension in Nepal. Front Public Health. 2020;8:580. doi:10.3389/fpubh.2020.00580
6. Zhang Y, Wang X, Li Y, et al. Wearable cuffless blood pressure monitoring devices: A systematic review. Eur Heart J Digit Health. 2022;3(2):323–32. doi:10.1093/ehjdh/ztac011
7. Allen NB, Siddique J, Wilkins JT, et al. Blood pressure trajectories in early adulthood and long-term cardiovascular outcomes. JAMA. 2014;311(5):490–7. doi:10.1001/jama.2013.284427
8. Hardy ST, Loehr LR, Butler KR, et al. Reducing misclassification of hypertension using proper measurement approaches. Hypertension. 2015;65(2):259–66. doi:10.1161/HYPERTENSIONAHA.114.04318
9. Huang Y, Guo P, Karmacharya BM, et al. Prevalence of hypertension and prehypertension in Nepal: a systematic review and meta-analysis. Glob Health Res Policy. 2019;4:11. doi:10.1186/s41256-019-0102-6
10. Ministry of Health, Nepal; New ERA; ICF. Nepal Demographic and Health Survey 2016. Kathmandu: Ministry of Health; 2017.
11. Gyawali B, Baral KP, Pant B, et al. Prevalence of prehypertension and hypertension in Nepal: analysis of the Nepal Demographic and Health Survey 2016. BMC Public Health. 2020;20:1234. doi:10.1186/s12889-020-08397-z
12. Bhaila A, Shakya B, Nepal GB, et al. Prevalence of prehypertension and its association with body mass index among the medical students. J Chitwan Med Coll. 2021;11(2):84–87. doi:10.54530/jcmc.305
13. Pun DB, Thapa B. Prevalence of prehypertension and its associated risk factors among students of a nursing campus in Nepal. J Physiol Soc Nepal. 2021;2(2):17–23. doi:10.3126/jpsn.v2i2.50175
14. Tiwari I, Bhattarai A, Karmacharya P, Gurung P. Correlation Between Body Fat Composition and Blood Pressure Level Among Medical Students in a Tertiary Care Teaching Hospital in Pokhara, Nepal. JCMS Nepal. 2023;19(2):186–193.
15. Ghimire R, Baral B, Pokhrel S. Prehypertension and hypertension prevalence among adolescents in eastern Nepal: a cross-sectional study. Children (Basel). 2023;10(5):803. doi:10.3390/children10050803.
16. Agho KE, Osuagwu UL, Ezeh OK, Ghimire PR, Chitekwe S, Ogbo FA. Gender differences in factors associated with prehypertension and hypertension in Nepal: a nationwide survey. PLoS One. 2018;13(9):e0203278. doi:10.1371/journal.pone.0203278.

17. Yadav RL, Khadka R, Agrawal K, Thakur D, Sharma D, Shah DK, Yadav PK, Sapkota NK, Paudel BH, Islam MN. Analysis of cardiac autonomic modulation in normotensive obese and eutrophic adults of Nepal. Int J Res Med Sci. 2016;4(1):105–110. doi:10.18203/2320-6012.ijrms20160013

18. Al-Metha AF, Ibrahim MM, Shaltout AA, et al. Sample size estimation in cross-sectional studies: application in hypertension research. Saudi J Med Med Sci. 2019;7(3):161–7. doi:10.4103/sjmms.sjmms_236_18

19. Alpert BS, Quinn D, Rehak NN, et al. Comparison of automated oscillometric versus auscultatory blood pressure measurement. J Clin Hypertens (Greenwich). 2010;12(11):930–936. doi:10.1111/j.1751-7176.2010.00300.x

20. Ogedegbe G, Pickering TG. Principles and techniques of blood pressure measurement. Cardiol Clin. 2010;28(4):571–586. doi:10.1016/j.ccl.2010.07.002

21. Kallioinen N, Hill A, Horswill M, et al. Sources of inaccuracy in adult BP measurement: a systematic review. J Hypertens. 2017;35(3):421–441. doi:10.1097/HJH.0000000000001187

22. Reckelhoff JF. Gender differences in the regulation of blood pressure. Hypertension. 2001;37(5):1199–1208. doi:10.1161/01.HYP.37.5.1199

23. Shrestha P, Mishra SR, Manandhar K, et al. Sex differences in BP among young adults in Nepal: a cross-sectional study. BMC Cardiovasc Disord.2021;21:432. doi:10.1186/s12872-021-02345-6

24. Gupta R, Singh K, Misra A. Differences in BP and cardiovascular risk factors in young adults: evidence from India. Indian Heart J. 2020;72(4):325–332. doi:10.1016/j.ihj.2020.04.009

25. Stergiou GS, Alpert B, Mieke S, et al. Validation of the Microlife BP3T01 1B device according to ISO 81060-2. Blood Press Monit. 2018;23(1):1–7. doi:10.1097/MBP.0000000000000302

26. Omboni S, Palladini G, Gazzola T, et al. Accuracy of home BP monitors: results from a multi-device evaluation. Hypertens Res. 2017;40(11):1036–1044. doi:10.1038/hr.2017.90

27. Myers MG, Godwin M, Dawes M, et al. Conventional versus automated office BP measurement in the office: a systematic review. J Hypertens. 2010;28(12):2316–2324. doi:10.1097/HJH.0b013e32834049a8

ORIGINAL ARTICLE

Sonographic Evaluation of Pancreatic Morphology in Patients with Diabetes Mellitus

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ABSTRACT

Introduction: Diabetes mellitus is a chronic metabolic disorder linked to pancreatic structural changes detectable by imaging. This study evaluates pancreatic size and echogenicity in Type 1 and Type 2 diabetes mellitus compared to healthy controls, emphasizing sonography's utility in resource-limited settings where advanced imaging is scarce. **Aims:** To assess pancreatic morphological changes via ultrasound in Type 1 and Type 2 diabetes patients, correlating findings with clinical parameters such as disease duration, HbA1c, and body mass index. **Methods:** A prospective cross-sectional study included 300 subjects: 100 Type 1, 100 Type 2 diabetes mellitus and 100 age- and sex-matched healthy controls. Standardized fasting transabdominal ultrasound measured anteroposterior diameters of the pancreatic head, body, and tail were taken. Echogenicity was graded (0–3) relative to liver echogenicity. Clinical data disease duration, HbA1c, and body mass index were collected. Statistical analyses compared groups and assessed correlations. **Results:** Type 1 diabetes mellitus patients exhibited marked pancreatic atrophy across all segments, with the most severe reduction in the tail. Type 2 diabetes patients showed mild reduction in pancreatic head diameter but relatively preserved body and tail dimensions. Pancreatic size inversely correlated with disease duration in Type 1 ($r=-0.45$, $p<0.001$) and Type 2 Diabetes ($r=-0.28$, $p<0.001$). Increased echogenicity was observed in 68% of Type 1 and 72% of Type 2 patients, versus 12% of controls. In Type 2 patients, echogenicity positively correlated with body mass index ($r=0.34$, $p<0.001$). **Conclusion:** Sonography is a valuable, non-invasive, and cost-effective tool for assessing pancreatic changes in diabetes. Type 1 diabetes mellitus is associated with marked pancreatic atrophy correlating with disease duration, while Type 2 diabetes mellitus demonstrates primarily increased echogenicity related to fatty infiltration and body mass index, with only mild size reduction. These findings highlight sonography's potential as a primary diagnostic and monitoring tool in resource-limited settings.

Keywords: Body mass index, Diabetes, Magnetic resonance imaging, Pancreas, Ultrasonography

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INTRODUCTION

Diabetes mellitus (DM) is a global pandemic, with prevalence rising sharply, especially in low- and middle-income countries.[1] Characterized by chronic hyperglycemia due to impaired insulin secretion or action, DM's systemic complications are well-documented, but morphological changes in the pancreas remain understudied.[2] Advanced imaging like CT and MRI has revealed reduced pancreatic volume in Type 1 diabetes, while Type 2 diabetes shows increased pancreatic fat content with variable size changes.[3–5] However, these modalities are limited by cost, radiation exposure, and availability, particularly in resource-constrained regions. In South Asia, including Nepal, the diabetes burden is escalating, yet access to advanced diagnostic tools is restricted.[6] Transabdominal ultrasonography offers a practical solution as a non-invasive, radiation-free, and widely accessible tool that can reliably assess pancreatic dimensions and echotexture when standardized protocols are followed. [7,8]

While prior studies suggest reduced pancreatic size in diabetes, comprehensive evaluations differentiating Type 1 (T1DM) and Type 2 diabetes (T2DM) and correlating findings with clinical parameters are lacking. This study evaluates the utility of sonography in assessing pancreatic morphological changes in T1DM and T2DM patients compared to healthy controls. We correlate sonographic findings with disease duration, glycemic control (HbA1c), and body mass index (BMI), hypothesizing distinct morphological patterns between diabetes types.

METHODS

This prospective, cross-sectional study was conducted from September 2023 to August 2024 at Nepalgunj Medical College and Teaching Hospital, Nepal. The study population included 300 subjects, evenly divided into three groups: 100 patients with T1DM, 100 patients with T2DM, and 100 age- and sex-matched healthy controls (HC). Ethical approval for the study was obtained from the Institutional Review Committee (IRC) of Nepalgunj Medical College and Teaching Hospital, with the approval number 05/080-081. Written informed consent was obtained from all participants prior to their enrollment in the study.

Participants with T1DM and T2DM were diagnosed according to the criteria established by the American Diabetes Association (ADA). Healthy controls were selected based on normal fasting glucose levels and the absence of any known chronic diseases. Exclusion criteria for all groups included a history of acute or chronic pancreatitis, pancreatic surgery, pancreatic malignancy, cystic fibrosis, alcoholism, or any condition known to affect pancreatic morphology. A consecutive sampling technique was employed to recruit participants from the outpatient and inpatient departments of Nepalgunj Medical College and Teaching Hospital. Healthy controls were selected from the general population and matched for age and sex to the diabetic groups. Sample size was estimated for the primary outcome, the anteroposterior (AP) diameter of the pancreatic head, using pilot standard deviations (T1DM = 2.9 mm, T2DM = 1.7 mm, controls = 3.8 mm). For a two-sided two-sample t-test to detect a clinically meaningful difference of 3.0 mm with an alpha of 0.05 and 80% power, the minimum required per-group sample sizes were approximately 20 for T1DM versus controls, 16 for T2DM versus controls, and 10 for T1DM versus T2DM. To allow for multiple comparisons, subgroup analyses, and potential missing data, we enrolled 100 participants per group, providing over 99% power to detect the prespecified 3.0 mm difference given the pilot variability.

For all subjects, demographic data (age, sex), anthropometric measurements (height, weight, and body mass index [BMI]), and clinical parameters, including fasting plasma glucose (FPG), glycosylated hemoglobin (HbA1c), and disease duration (for the diabetic groups), were collected.

All ultrasound examinations were performed by a single experienced radiologist using a General Electronics P6 ultrasound machine with a 3.5 MHz curvilinear transducer. To ensure standardization and minimize operator dependence, a consistent protocol was followed. All subjects were required to fast for at least 8 hours to minimize bowel gas interference. Patients were scanned in the supine position and, if necessary, in a right posterior oblique position to optimize visualization of the pancreas. The pancreas was visualized in both transverse and longitudinal planes, and the AP diameters of the pancreatic head, body, and tail were measured at standardized anatomical landmarks. The head was measured at the level of the gastroduodenal artery, the body at the level of the superior mesenteric artery, and the tail just anterior to the splenic vein.

Pancreatic echogenicity was qualitatively assessed and graded on a scale of 0 to 3 relative to liver echogenicity. Grade 0 indicated echogenicity isoechoic to the liver, Grade 1 indicated mildly hyperechoic compared to the liver, Grade 2 indicated moderately hyperechoic compared to the liver, and Grade 3 indicated markedly hyperechoic echogenicity, similar to retroperitoneal fat. The radiologist was blinded to the subjects' clinical status (T1DM, T2DM, or HC) to minimize bias.

STATISTICAL ANALYSIS

Descriptive statistics were used to summarize the data. Group comparisons were performed using one-way analysis of variance (ANOVA) with post-hoc Tukey's Honestly Significant Difference (HSD) test for continuous variables and the Chi-square test for categorical variables. Pearson's correlation coefficient was used to assess the relationship between pancreatic dimensions, echogenicity, and clinical parameters. A p-value of <0.05 was considered statistically significant. All analyses were performed using SPSS version 25.0.

RESULTS

The clinical characteristics of the study participants are summarized in Table I. The mean age was comparable across all three groups. The T2DM group had a significantly higher mean BMI than the T1DM and HC groups. As expected, HbA1c and FPG levels were significantly higher in both diabetic groups compared to the healthy control group.

Variable	T1DM Group (n=100)	T2DM Group (n=100)	Control Group (n=100)	P-value
Age (years, mean ± SD)	45.2 ± 11.5	52.1 ± 10.8	48.8±12.6	-
Male Gender (n, %)	52 (52.0%)	55 (55.0%)	47 (47.0%)	-
Diabetes Duration (years, mean ± SD)	14.2 ± 6.1	9.8 ± 4.5	N/A	<0.001
HbA1c (%, mean ± SD)	8.1 ± 1.5	7.5 ± 1.2	5.4 ± 0.5	<0.001
BMI (kg/m2, mean ± SD)	25.1 ± 3.8	31.4 ± 5.2	24.8 ± 3.5	<0.001

Table I: Study Population Characteristics and Demographics

The sonographic measurements of pancreatic AP diameters are presented in Table II. The T1DM group showed marked atrophy with significantly smaller mean AP diameters across all three pancreatic segments compared to controls ($p < 0.001$ for all). The T2DM group showed significant reduction only in the pancreatic head diameter ($p < 0.001$), while the body and tail dimensions were relatively preserved with only mild, non-significant reductions compared to controls. Significant inverse correlation was found between pancreatic size and disease duration. Specifically, the combined pancreatic diameter (head + body + tail) was negatively correlated with the duration of illness in both T1DM ($r=-0.45$, $p<0.001$) and T2DM ($r=-0.28$, $p<0.001$) groups.

Pancreas part	T1DM (Mean AP Diameter +/- SD in mm)	T2DM (Mean AP Diameter +/- SD in mm)	Controls (Mean AP Diameter +/- SD in mm)	P-value (T1DM vs Controls)	P-value (T2DM vs Controls)	P-value (T1DM vs T2 DM)
Head	17.8 ± 2.9	21.13 ± 1.68	23.31 ± 3.80	<0.001	<0.001	<0.001
Body	10.93 ± 2.0	20.17 ± 1.16	22.05 ± 2.06	<0.001	0.08	<0.001
Tail	11.2 ± 2.5	20.44 ± 1.17	23.31 ± 2.49	<0.001	0.12	<0.001

Table II: Pancreatic Morphometric Dimensions in Diabetes Mellitus vs. Controls

Pancreatic echogenicity was markedly different among the groups. Increased echogenicity (grades 1, 2, or 3) was observed in a large proportion of diabetic patients (68% in T1DM and 72% in T2DM), while only 12% of healthy controls exhibited this finding. In T2DM patients, there was a significant positive correlation between increased pancreatic echogenicity and BMI ($r=0.34$, $p<0.001$). This correlation was not observed in the T1DM group. No statistically significant correlation was observed between echogenicity and HbA1c in either diabetic group.

DISCUSSION

This study provides compelling evidence of significant pancreatic morphological changes in patients with both T1DM and T2DM compared to healthy controls, as detected by sonography. Conducted in a resource-limited setting like Nepal, our findings underscore the clinical utility of ultrasonography in environments where advanced imaging modalities are not widely available.[1] This research reinforces the value of radiology in understanding and monitoring pancreatic pathologies and provides a practical, real-world framework for using ultrasound to assess pancreatic health.

Our study revealed distinct morphological patterns between T1DM and T2DM. T1DM patients exhibited marked pancreatic atrophy across all segments, with the most pronounced reduction in the tail, which is anatomically rich in islet cells.[9] This observation strongly supports the prevailing understanding that T1DM, an autoimmune disease, leads to progressive destruction of insulin-producing β-cells and subsequent pancreatic volume loss.[10] The progressive nature of this atrophy is further supported by our finding of a strong inverse correlation between pancreatic size and disease duration in T1DM patients ($r=-0.45$, $p<0.001$). This is consistent with other studies that have shown a decline in pancreas volume during the first year after T1DM diagnosis.[11]

In contrast, T2DM patients showed only mild reduction in pancreatic head diameter, while body and tail dimensions remained relatively preserved. This finding aligns with the pathophysiology of T2DM, where the primary pancreatic change is fatty infiltration rather than atrophy, particularly in the context of obesity and metabolic syndrome. The weaker correlation between pancreatic size and disease duration in T2DM ($r=-0.28$, $p<0.001$) likely reflects the heterogeneous nature of T2DM and the competing effects of fatty infiltration (which may increase size) and long-term β-cell exhaustion (which may cause mild atrophy). These findings are especially relevant in resource-limited areas where patients often present with advanced disease due to delayed diagnosis and management.

Advanced imaging modalities like MRI have corroborated and quantified these atrophic changes with greater precision.[2] A comprehensive systematic review and meta-analysis of imaging studies, which included over 3,400 subjects, concluded that individuals with T1DM have significantly reduced pancreas size compared to controls, while findings in T2DM are more variable.[12] This meta-analysis highlighted that the volume reduction was substantially more significant in T1DM. These robust findings from large-scale analyses, combined with our sonographic data, confirm that pancreatic atrophy is a fundamental characteristic of T1DM, while T2DM shows heterogeneous size changes. The fact that ultrasound, a far more accessible tool, can detect these changes with statistical significance underscores its clinical utility as a screening and monitoring tool, particularly in settings where a full-body MRI is a distant reality. However, sonographic assessment may be limited in obese individuals due to acoustic attenuation and overlying bowel gas, which can obscure pancreatic visualization.

Beyond simple atrophy, our study demonstrates a significant increase in pancreatic echogenicity in diabetic patients, a finding present in a large majority of both T1DM (68%) and T2DM (72%) patients, compared to only 12% of controls. This sonographic finding, a key radiological indicator, reflects underlying histopathological changes. In T2DM, the positive correlation we found between increased echogenicity and BMI ($r=0.34$, $p<0.001$) strongly supports the concept of pancreatic steatosis, or fatty infiltration, as a primary driver of this appearance. This is a well-documented phenomenon in T2DM and is supported by MRI studies that have quantified pancreatic triglyceride content, finding it to be 23% greater in T2DM subjects than in controls.[3] In contrast, our study found no such correlation between echogenicity and BMI in the T1DM group. This suggests that while increased echogenicity is present in T1DM, its cause is fundamentally different and reflects the distinct pathophysiology of the two diabetes types. The histopathological hallmark of T1DM is insulitis, an inflammatory process that leads to fibrosis of the pancreas. Our sonographic findings, there-

fore, likely represent a combination of pancreatic atrophy and fibrosis in T1DM, distinct from the lipotoxic changes seen in T2DM. This differentiation is a crucial point for radiologists to consider when evaluating pancreatic sonograms in diabetic patients.

The radiological changes we identified are not merely morphological assessments but hold significant clinical implications. A recent MRI study highlighted the potential of using MRI-based pancreatic morphology and clinical characteristics to predict the risk of T2DM.[13] Their logistic regression analysis revealed complex relationships between pancreatic morphology and T2DM risk, achieving a predictive accuracy of 90.20%.[13] While these findings were based on MRI, they underscore the diagnostic and prognostic value of assessing pancreatic morphology. Such predictive models, once validated by simpler modalities, could be invaluable in a low-resource clinical setting.

Furthermore, another study found that serration of the pancreatic limbus, a morphological change seen in MRI, was more often observed in subjects with diabetes mellitus and was associated with older age and higher HbA1c values.[5] This corroborates our finding of correlations between pancreatic morphology and metabolic control, particularly the strong relationship observed in T1DM. The finding that these morphological changes, such as a smaller pancreatic volume and serrated changes, are associated with the progression of vascular complications elevates their importance from a simple observation to a potentially critical component of risk assessment, particularly in T1DM where atrophy is pronounced.[5,13] In a resource-limited setting such as this study site, where access to specialized vascular assessments may be limited, a simple sonographic finding could provide an early warning sign for clinicians to intensify metabolic control and screen for complications.

Our study has several limitations inherent to its design. The cross-sectional nature prevents us from drawing conclusions about causality and the temporal progression of these changes in individual patients. The single-center nature and a sample size of 300, while large for a sonography study, limit the generalizability of our findings. The inherent operator-dependent variability of ultrasound was however mitigated by the use of a single, experienced, and blinded radiologist.

CONCLUSION

In a disease as complex and multifaceted as diabetes, radiology, and specifically the accessible modality of ultrasound, has a vital role to play in enhancing our understanding and improving clinical management, especially in regions where resources are limited. Based on our findings, we recommend that ultrasound be integrated into routine diabetes care in resource-limited settings as a first-line tool for assessing pancreatic morphology. In T1DM, ultrasound can detect progressive atrophy and monitor disease progression, while in T2DM, it can identify fatty infiltration associated with metabolic syndrome. This approach could facilitate early detection of type-specific pancreatic changes, guide therapeutic decisions, and improve monitoring of disease progression. Future research should focus on multicenter, longitudinal studies to validate these sonographic markers over time. Furthermore, combining sonographic evaluation with quantitative imaging biomarkers from MRI (such as pancreatic fat fraction) and clinical markers (such as C-peptide and β-cell function indices) could provide a more comprehensive, integrated assessment of pancreatic health in diabetes. Ultrasound should therefore be considered a cost-effective first-line modality, supplemented by advanced imaging when clinically indicated. Such efforts could significantly enhance diabetes management and reduce the burden of complications in vulnerable populations.

REFERENCES

1. Magliano DJ, Boyko EJ; IDF Diabetes Atlas Committee. IDF Diabetes Atlas. Brussels: International Diabetes Federation; 2021.
2. Macauley M, Percival K, Thelwall PE, Hollingsworth KG, Taylor R. Altered volume, morphology and composition of the pancreas in type 2 diabetes. PLoS One. 2015;10(5):e0126825. DOI: 10.1371/journal.pone.0126825. PMCID: PMC4423920.
3. Chai J, Liu P, Jin E, Su T, Zhang J, Shi K, et al. MRI chemical shift imaging of the fat content of the pancreas and liver of patients with type 2 diabetes mellitus. Exp Ther Med. 2016;11(2):476-80. DOI: 10.3892/etm.2015.2925. PMCID: PMC4734085.
4. Higashi M, Tanabe M, Tanabe K, Okuya S, Takeda K, Nagao Y, et al. Multiparametric magnetic resonance imaging findings of the pancreas: a comparison in patients with type 1 and 2 diabetes. Tomography. 2025;11(2). DOI: 10.3390/tomography11020016. PMCID: PMC11861380.
5. Iwamoto Y, Kimura T, Tatsumi F, Sugisaki T, Kubo M, Nakao E, et al. Association between changes in pancreatic morphology and vascular complications in subjects with type 2 diabetes mellitus: a retrospective study. Sci Rep. 2022;12(1):17166. DOI: 10.1038/s41598-022-21688-1. PMCID: PMC9562404.
6. Smits MM, van Geenen EJ. The clinical significance of pancreatic steatosis. Nat Rev Gastroenterol Hepatol. 2011;8(3):169-77. DOI: 10.1038/nrgastro.2011.4.
7. Li S, Su L, Lv G, Zhao W, Chen J. Transabdominal ultrasonography of the pancreas is superior to that of the liver for detection of ectopic fat deposits resulting from metabolic syndrome. Medicine (Baltimore). 2017;96(37):e8060. DOI: 10.1097/md.0000000000008060. PMCID: PMC5604670.
8. D'Onofrio M, Zamboni G, Faccioli N, Capelli P, Pozzi Mucelli R. Ultrasonography of the pancreas. 4. Contrast-enhanced imaging. Abdom Imaging. 2007;32(2):171-81. DOI: 10.1007/s00261-006-9010-6.
9. Abdulrahman S, Ibrahim AA, Mohamed MA, Gameraddin M, Alelyani M. Sonographic evaluation of the pancreas in type 1 diabetes mellitus: a case-control study. J Med Ultrasound. 2021;29(3):167-70. DOI: 10.4103/jmu.Jmu_89_20. PMCID: PMC8515619.
10. Campbell-Thompson M, Fu A, Kaddis JS, Wasserfall C, Schatz DA, Pugliese A, et al. Insulitis and β-cell mass in the natural history of type 1 diabetes. Diabetes. 2016;65(3):719-31. DOI: 10.2337/db15-0779. PMCID: PMC4764143.

11. Virostko J, Williams J, Hilmes M, Bowman C, Wright JJ, Du L, et al. Pancreas volume declines during the first year after diagnosis of type 1 diabetes and exhibits altered diffusion at disease onset. Diabetes Care. 2019;42(2):248-57. DOI: 10.2337/dc18-1507. PMCID: PMC6341292.

12. Garcia TS, Rech TH, Leitão CB. Pancreatic size and fat content in diabetes: a systematic review and meta-analysis of imaging studies. PLoS One. 2017;12(7):e0180911. DOI: 10.1371/journal.pone.0180911. PMCID: PMC5524390.

13. Yan Y, Wu T, Huang Z, Song X, Huang X, Liu N. Risk Prediction of Type 2 Diabetes Mellitus by MRI-based Pancreatic Morphology and Clinical Characteristics: A Cross-sectional Study. Current Medical Imaging. 2024 Jan;20(1):e15734056304038.

Comparison of Mephentermine, Ephedrine and Phenylephrine for Treating Hypotension after Spinal Anesthesia in Caesarean Section

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ABSTRACT

Introduction: Hypotension is a common complication during spinal anesthesia in the caesarean section, often requiring vasopressors for correction. This study compares the effects of Mephentermine, Ephedrine, and Phenylephrine in maintaining arterial pressure. **Aims:** To evaluate the onset time and efficacy of three vasopressors in reversing hypotension, comparing heart rate responses (bradycardia or tachycardia) and assessing side effects such as nausea, vomiting or shivering. Additionally, neonatal outcomes are assessed using Apgar scores at 1 and 5 minutes to evaluate the impact of each vasopressor. **Methods:** A prospective, randomized interventional study was conducted among 105 parturients undergoing elective or emergency caesarean section under spinal aesthesia at Nepalgunj Medical College Teaching Hospital. Participants were randomly divided into three equal groups (n=35) to receive intravenous boluses of either Mephentermine 6 mg, Ephedrine 6 mg, or Phenylephrine 80 µg when hypotension occurred. Hemodynamic parameters (Systolic blood pressure, diastolic blood pressure and heart rate), neonatal outcomes and complications were recorded. **Results:** The Phenylephrine group demonstrated a significantly greater and quicker rise in systolic blood pressure during the first six minutes post-administration ($p<0.05$). However, Bradycardia was more frequent in this group. Ephedrine and Mephentermine maintained heart rate more effectively with fewer bolus doses. Neonatal APGAR scores at 1 and 5 minutes were comparable among all three groups. **Conclusion:** All three vasopressors effectively correct spinal anesthesia-induced hypotension. Phenylephrine results in higher blood pressure and reduces heart rate, offering an advantage when tachycardia is undesirable. None show significant adverse maternal or neonatal effects.

Keywords: Apgar score, Caesarean section, Hypotension, Spinal anesthesia, Vasopressors

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INTRODUCTION

Spinal anesthesia is the most commonly used regional anesthetic techniques for caesarean sections due to its rapid onset, profound sensory blockade and minimal fetal drug exposure. However, spinal anesthesia often causes maternal hypotension through sympathetic blockade, potentially compromising both maternal and fetal health. During caesarean delivery, this hypotension can lead to maternal nausea, dizziness, and reduced cardiac output, as well as fetal complications including transient hypoxia and acidosis.[1] Effective management of hypotension is therefore crucial for achieving optimal maternal and fetal outcomes. Several vasopressors are used to counteract spinal-induced hypotension, each with different mechanisms and safety profiles. Mephentermine, an indirectly acting sympathomimetic stimulates both alpha- and beta-adrenergic receptors, thereby increasing cardiac output and vascular tone. Ephedrine, a mixed-acting sympathomimetic with alpha- and beta-agonist properties, causes vasoconstriction while also increasing heart rate and cardiac contractility. Phenylephrine, a direct alpha-1 agonist, causes rapid vasoconstriction and is increasingly preferred because it effectively restores blood pressure with minimal effects on fetal acid–base status.[2] The rationale for comparing these vasopressors in treating hypotension after spinal anesthesia is to identify the most effective and safest vasopressor for improving maternal blood pressure and fetal outcomes. This study aims to evaluate the onset time and efficacy of three vasopressors in reversing hypotension, comparing heart rate responses (bradycardia or tachycardia) and assessing side effects such as nausea, vomiting or shivering. Additionally,

neonatal outcomes are assessed using Apgar scores at 1 and 5 minutes to evaluate the impact of each vasopressor.

METHODS

This prospective, comparative clinical study was conducted in the Department of Anaesthesia at Nepalgunj Medical College Teaching Hospital, Kohalpur from November 2024 to April 2025. Ethical approval was obtained from the Institutional Review Committee of Nepalgunj Medical College, and written informed consent was secured from all participants.

The sample size was calculated with an α error of 5% and β error of 20%[4] resulting in 105 patients. Patients (N = 105) who developed hypotension after spinal anesthesia were randomly assigned to one of three groups (n = 35 per group) using a sealed opaque envelope method.

Group A: Mephentermine 6 mg in 1 ml as intravenous (IV) bolus,

Group B: Ephedrine 6 mg in 1 ml as IV bolus, and

Group C: Phenylephrine 80 mcg in 1 ml as IV bolus was used as indicated.

Inclusion Criteria:

• Pregnant women aged 18-40 years undergoing elective caesarean section under spinal anesthesia.

• ASA Physical Status I or II (patients who are healthy or have mild systemic disease).

Exclusion Criteria:

• Patients' refusal for consent, fetal distress and contraindications to spinal anesthesia.

• Patients with known allergies to Mephentermine, Ephedrine, or Phenylephrine.

• Patients with comorbidities such as preeclampsia, eclampsia, pregnancy-induced hypertension (PIH), and heart disease.

On arrival of the patients in operation theater, IV line was initiated with 18-G cannula; all patients were given 20 ml/kg of Ringer's lactate solution intravascular loading before spinal anesthesia. All patients were given premedication consisting of 50 mg of intravenous ranitidine and 10 mg of metoclopramide before surgery, according to institutional protocol, to prevent the risk of regurgitation and aspiration. Under aseptic precautions, spinal anaesthesia was administered in the sitting position using a 25-gauge Quincke needle at the L3-L4 level. A total dose of 2.2 mL of heavy Bupivacaine was given for spinal anaesthesia. The level of sensory blockade was targeted to T4-T6 dermatomes. Sensory block was assessed using the pinprick method, and motor block was evaluated with the Bromage scale to ensure adequate anaesthesia for lower segment cesarean section.The wedge was placed under right buttock for left uterine displacement in all patients. Additionally, all patients were kept in oxygen at 5L/min through facemask and intravenous oxytocin 5U were given after the delivery of baby.

Hypotension is defined as a decrease in systolic blood pressure of ≥20% from the baseline value or an absolute value of <100 mmHg, whichever will be higher.[5] Hemodynamic parameters (SBP, DBP, HR) of patient were recorded at baseline, immediately after the intervention, and at 2, 4, 6, 8, 10, 15 and 20 minutes post-intervention and every 10 minutes until the end of surgery. Intraoperatively, hypotension was recorded and managed with intravenous study drugs to restore blood pressure.

The incidence of adverse effects like nausea, vomiting, shivering and bradycardia were noted and managed according to the institution protocol. Nausea and vomiting was treated with 4mg of intravenous odansetron. The bradycardia i.e. a pulse rate of 60/min or less was treated with atropine 0.6mg intravenous while shivering was managed with pethidine 25 mg IV slowly. Paediatrician blinded to the study drugs assessed Apgar score of every neonate at 1 and 5 minutes after delivery and recorded.

Statistical analysis

Data were collected and recorded as per proforma. Statistical analysis was done by SPSS version 20. Data were presented in tables and figures as appropriate and comparability of groups were analyzed with Analysis of variance (ANOVA) test and Chi-square test. A p-value of <0.05 was considered statistically significant.

RESULTS

Demographic and Baseline Parameters: There were no statistically significant differences between the three groups in terms of age, weight, physical status and duration of surgery.

Variables	Group A	Group B	Group C	p-value
Age(yrs)	26.54± 4.84	27.11 ± 5.58	25.31 ± 3.91	0.28
Weight(Kg)	77.89 ± 7.5	77.37 ±7. 9	77.51 ±7. 8	0.96
Physical Status				
ASA I	28(80)	25(71.4)	24(68.6)	0.5
ASA II	7(20)	10(28.6)	11(31.4)	
Duration of Surgery(min)	66.86 ±8.67	64.00±8.81	62.57±7.41	0.09

Table I: Study population demographic data

Hemodynamic Response:

Intervals	Systolic Blood Pressure			Value		Inter Group Comparison		
	Group A	Group B	Group C	F	p	A-B	A-C	B-C
Baseline	128.03± 9.46	127.40± 10.68	129.34± 9.7	0.346	0.71	-	-	-
B1T0	95.63± 4.4	96.77± 4.83	95.31± 4.4	0.995	0.373	-	-	-
B1T2	113.57± 9.78	117.34± 11.93	127.03± 11.01	14.086	0.001	-	++	++
B1T4	119.86± 13.48	117.11± 13.17	127.66± 10.49	6.76	0.002	-	-	++
B1T6	126.71± 11.78	127.06± 11.81	119.06± 11.92	5.11	0.008	-	-	+
B1T8	128.31± 9.48	129.51± 7.01	128.00± 11.05	0.257	0.77	-	-	-
B1T10	123.57± 14.71	119.63± 16.31	122.06± 15.33	0.579	0.56	-	-	-
B1T15	127.77± 11.61	129.06± 9.32	126.97± 11.43	0.330	0.72	-	-	-
B1T20	127.94± 10.17	126.43± 14.85	126.66± 15.41	0.125	0.88	-	-	-
B1T30	119.23± 13.83	124.03± 13.57	122.24± 12.98	1.134	0.326	-	-	-
B1T40	121.49± 12.85	126.54± 11.89	126.06± 11.02	1.911	0.15	-	-	-
B1T50	127.77± 11.61	129.06± 9.32	126.11± 12.99	0.585	0.56	-	-	-
B1T60	121.64± 16.56	118.27± 16.55	118.45± 16.05	0.429	0.65	-	-	-
B1T70	127.88± 11.57	125.13± 12.21	126.09± 15.93	0.241	0.79	-	-	-
B1T80	125.40± 11.33	134.00± 4.32	135.00± 7.07	1.415	0.298	-	-	-

Table II: Comparison of the Systolic BP among three groups

B1T0: At the time of hypotension B1T2: 2 minutes after study drugs

(Within 2 groups: ++: p value <0.0001 +:p value<0.05 -: p value>0.05)

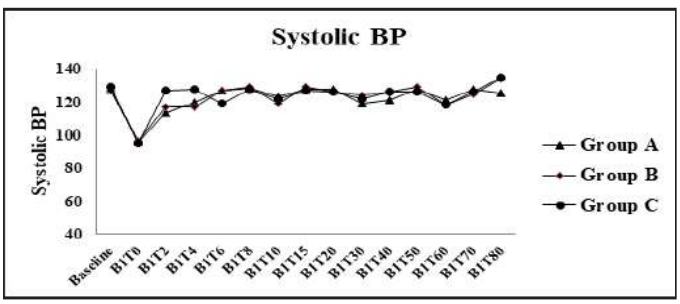

Figure 1: Comparison of the Systolic BP among three groups

The baseline systolic blood pressure among all three groups was statistically similar. Both systolic and diastolic pressures decreased similarly at the onset of hypotension and increased following the bolus dose in all groups. A significant rise in systolic and diastolic blood pressure was observed in the Phenylephrine group compared to the other two groups at 2 and 4 minutes post-intervention (p = 0.001 and 0.002, respectively). On intergroup comparison, the increase in diastolic pressure at 2 and 4 minutes was less pronounced in the Ephedrine and Mephentermine groups than in the Phenylephrine group (p < 0.001). No significant difference was noted between the Ephedrine and Mephentermine groups (p > 0.05).

Intervals	Diastolic Blood Pressure			Value		Inter Group Comparison		
	Group A	Group B	Group C	F	p	A-B	A-C	B-C
Baseline	82.83± 9.46	81.86± 10.91	83.26± 10.19	0.173	0.84	-	-	-
B1T0	57.89± 4.39	56.63± 4.13	55.17± 4.54	3.39	0.052	-	+	-
B1T2	65.00± 3.89	70.91± 10.18	78.09± 10.47	19.75	0.001	-	++	+
B1T4	75.09± 11.22	72.57± 11.05	79.80± 9.93	4.08	0.02	-	-	+
B1T6	80.43± 5.12	81.26± 3.63	81.29± 2.69	0.534	0.59	-	-	-
B1T8	81.66± 10.83	83.37± 6.23	80.74± 10.37	0.708	0.50	-	-	-
B1T10	80.00± 11.46	78.60± 11.47	80.20± 11.67	0.200	0.82	-	-	-
B1T15	81.69± 11.02	80.74± 9.75	82.00± 8.70	0.154	0.86	-	-	-
B1T20	82.29± 3.82	76.83± 11.61	80.40± 11.26	2.92	0.06	-	-	-
B1T30	74.77± 11.38	79.17± 10.72	76.50± 11.42	1.377	0.26	-	-	-
B1T40	82.17± 7.97	83.00± 5.68	82.29± 7.01	0.146	0.86	-	-	-
B1T50	82.66± 4.84	82.63± 4.79	81.37± 5.26	0.764	0.47	-	-	-
B1T60	77.48± 11.53	76.80± 11.83	74.87± 12.38	0.409	0.67	-	-	-
B1T70	81.24± 9.55	82.63± 6.39	78.45± 11.06	0.698	0.50	-	-	-
B1T80	74.00± 8.94	80.00± 8.20	85.00± 7.07	1.359	0.31	-	-	-

Table III: Comparison of the Diastolic BP among three groups

B1T0: At the time of hypotension B1T2: 2 minutes after study drugs

(Within 2 groups: ++: p value <0.0001 +:p value<0.05 -: p value>0.05)

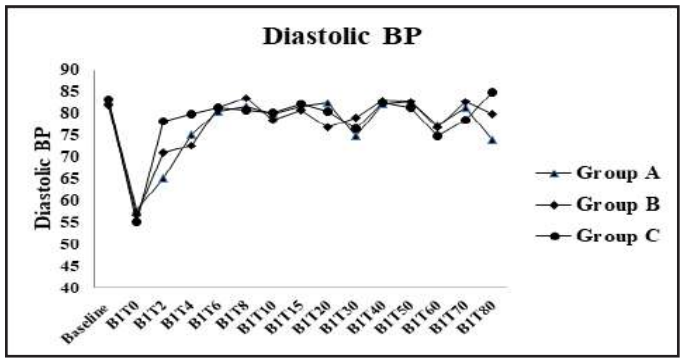

Figure 2: Comparison of the Diastolic BP among three groups

The baseline heart rate among all three groups was statistically similar. A clinically significant rise in heart rate was observed in all groups during hypotension, though this was not statistically significant. However, a significant decrease in heart rate was noted in the Phenylephrine group compared to the Ephedrine and Mephentermine groups at 4, 6, and 8 minutes after administration of the study drug. No significant difference in heart rate changes was observed between the Ephedrine and Mephentermine groups.

Intervals	Heart Rate			Value		Inter Group Comparison		
	Group A	Group B	Group C	F	p	A-B	A-C	B-C
Baseline	83± 11.14	85.03± 12.7	86.20± 11.65	0.654	0.52	-	-	-
B1T0	103.14± 7.69	104.00± 8.22	104.63± 8.01	0.306	0.74	-	-	-
B1T2	78.97± 12.54	80.60± 12.28	83.77± 9.57	1.57	0.2	-	-	-
B1T4	84.00± 13.22	81.26± 14.97	72.34± 8.77	8.2	0.0001	-	++	+
B1T6	81.63± 13.85	87.20± 14.10	65.86± 9.50	28.76	0.0001	-	++	++
B1T8	81.63± 13.86	90.11± 13.68	68.11± 10.66	26.24	0.0001	-	++	++
B1T10	84.20± 12.95	86.26± 14.07	82.51± 13.17	0.68	0.51	-	-	-
B1T15	85.80± 13.49	83.54± 11.06	83.80± 12.79	0.34	0.71	-	-	-
B1T20	85.09± 13.78	81.86± 14.50	85.43± 13.48	0.70	0.50	-	-	-
B1T30	85.34± 13.38	80.20± 11.74	83.77± 14.08	1.42	0.25	-	-	-
B1T40	84.26± 14.12	81.89± 13.42	85.26± 13.44	0.56	0.57	-	-	-
B1T50	84.11± 12.94	81.49± 13.41	85.03± 13.85	0.66	0.52	-	-	-
B1T60	80.00± 10.37	85.03± 13.97	80.65±1 2.77	1.49	0.23	-	-	-
B1T70	80.38± 10.41	87.33± 13.99	84.27± 12.84	1.45	0.25	-	-	-
B1T80	82.67± 15.16	92.50± 9.85	75± 7.07	1.34	0.32	-	-	-

Table IV : Comparison of the Heart Rate among three groups

B1T0: At the time of hypotension B1T2: 2 minutes after study drugs

(Within 2 groups: ++: p value <0.0001 +:p value<0.05 -: p value>0.05)

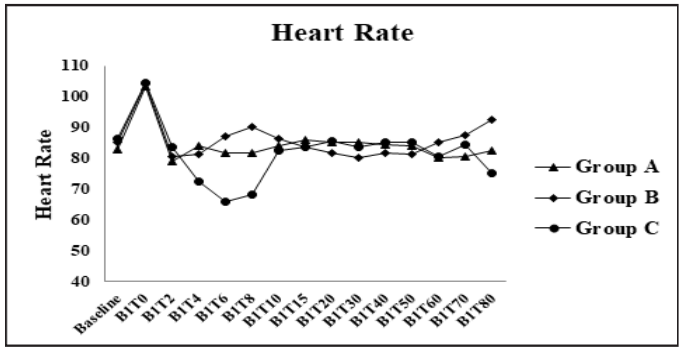

Figure 3: Comparison of the Heart Rate among three groups

Nausea was observed in 5.7% of patients in both the mephentermine and phenylephrine groups, and 2.9% in the ephedrine group. Shivering occurred in 11.4%, 8.6%, and 14.3% of patients in the mephentermine, phenylephrine, and ephedrine groups, respectively. Bradycardia was reported in 2.9%, 5.7%, and 8.6% of patients across the groups.(Figure 4) None of these differences were statistically significant (nausea p = 0.81; shivering p = 0.75; bradycardia p = 0.59).

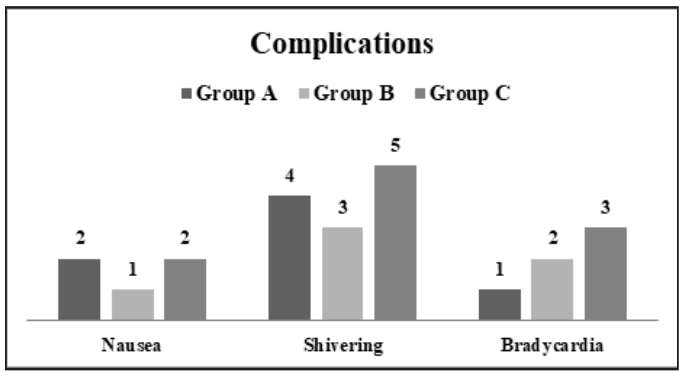

Figure 4: Comparison of the adverse effects among three groups

There was no statistical significant difference among three groups in context of total number of bolus of study drugs (p=0.74). Apgar scores at 1 and 5 minutes were ≥7 in all neonates among the three groups. No significant differences in neonatal outcomes were observed (p<0.05).

	Group A	Group B	Group C	p-value
Number of Bolus	1.26±0.51	1.26±0.50	1.34±0.60	0.74
Apgar Score@				
1min	7.54±0.51	7.57±0.50	7.6±0.55	0.9
5min	9.09±0.51	9.06±0.48	9.11±0.47	0.89

Table V: Comparison of the mean Number of Bolus and Apgar score of Neonates among three groups

DISCUSSION

Hypotension is one of the serious complications seen during caesarean section performed under subarachnoid block. This can be minimized with intravenous fluid, avoidance of aortocaval compression and judicious use of vasopressor agent. Careful positioning and volume preloading with intravenous crystalloid and colloid solution have been standard practice for prevention of hypotension but these are not complete measures. Primary cause of arterial pressure reduction is vasodilation. So, it is better to use vasopressor to correct hypotension.[3]

Our study included a total of 35 patients in each group receiving Mephentermine, Ephedrine and Phenylephrine. Percentage decrease in placental perfusion is related to percentage reduction in maternal arterial pressure and not to absolute reduction in pressure. In this study, Hypotension is defined as a decrease in mean arterial pressure below 20% of baseline or systolic blood pressure of < 100 mm of Hg.[5,6]

Among the vasopressors used Ephedrine have got a mixed action directly as well as indirectly on alpha and beta receptors whereas Mephentermine is an indirectly acting sympathomimetic amine and phenylephrine has pure alpha receptor activity.[2]

Ganeshanavar et al[6] stated that rise of systolic and diastolic blood pressure at 2, 4 and 6 minutes were significantly less in ephedrine and mephentermine group as compared to phenylephrine group. No significant difference (p>0.05) were observed between changes in systolic and diastolic pressure of ephedrine and mephentermine group which shows similar result as our group. In our study arterial blood pressure was maintained within 20% baseline limit by all three vasopressors though phenylephrine maintained better in first 6 minute of bolus dose (p<0.05) as compared to ephedrine and mephentermine. This may be due to the reason that phenylephrine has peak effect within one minute whereas ephedrine has 2-5 minutes and mephentermine has 5 minutes.[3]

Simin et al[7] and Nazir et al[8] concluded ephedrine and phenylephrine are effective vasopressors for treatment of hypotension associated to spinal shock during caesarean section without adverse effect on neonates.

Bhardwaj et al[9] compared phenylephrine, ephedrine and mephentermine and found all these vasopressors were equally effective in maintaining maternal blood pressure as well as umbilical pH without any detrimental effect on foetal and maternal outcome which shows similar result as our study.

According to study done by Sahu D et al[3] cardiovascular stability was better with phenylephrine as it caused significant reduction in heart rate after the bolus dose. In case of ephedrine and mephentermine group heart rate is increased compared to preoperative value which showed similar result as our study.

In this study, the mean number of bolus doses required to maintain systolic blood pressure within 20% of baseline was similar among the three groups indicating no statistically significant difference (p>0.05). This suggests that all study drugs were equally effective in stabilizing intraoperative hemodynamics. These findings align with previous literature. Ngan Kee et al[5] observed comparable rescue bolus requirements between phenylephrine and ephedrine in caesarean sections under spinal anesthesia, with no significant difference in total doses administered. There were no appreciable differences (p>0.05) in the incidences of nausea and shivering between any of the three groups which is in accordance with studies by Singh PM et al[10] and Tiwari JP et al.[11]

Our study showed the APGAR Scores at 1 and 5 minutes after delivery of baby were >7 among all the three study groups (p >0.05). Our findings were similar to the study conducted by Sahu D. et al[3], Thomas D.G. et al[12], Gunda C.P. et al[13] and Kee WD. et al[14] who gave IV boluses of these vasopressors and observed that APGAR scores at 1 and 5 minutes were >7 for all the neonates among the groups. Overall, mephentermine, phenylephrine, and ephedrine appear to have similar safety profiles, supporting their use as effective and well-tolerated vasopressors for intraoperative hemodynamic management.

LIMITATIONS

This study was limited by its single-centre design and short follow-up period restricted to intraoperative and immediate neonatal outcomes. Arterial blood gas analysis was not performed, limiting precise evaluation of fetal acid–base status.

CONCLUSION

In conclusion, we found that all three vasopressors mephentermine ephedrine and phenylephrine are effective as IV boluses form in maintaining maternal arterial pressure within 20% of baseline values. Among them, phenylephrine resulted in higher BP and reduction in heart rate, which may be advantageous when tachycardia is undesirable. All the three vasopressors had no significant adverse effects on maternal and neonatal outcome.

REFERENCES

1. Fakherpour A, Ghaem H, Fattahi Z, Zaree S. Maternal and anaesthesia-related risk factors and incidence of spinal anaesthesia-induced hypotension in elective caesarean section: a multinomial logistic regression. Indian J Anaesth. 2018;62(1):36–46.

2. Chandak AV, Bhuyan D, Singam AP, Patil B. Comparison of bolus phenylephrine, ephedrine and mephentermine for maintenance of arterial pressure during spinal anaesthesia in caesarean section. Res J Pharm Technol. 2021;14(3):1349–52.

3. Sahu D, Kothari D, Mehrotra A. Comparison of bolus phenylephrine, ephedrine and mephentermine for maintenance of arterial pressure during spinal anaesthesia in caesarean section–a clinical study. Indian J Anaesth. 2003;47(2):125–8.

4. Sharma H, Senger VS, Gajbhiye S. A comparative study of bolus phenylephrine, ephedrine and mephentermine for maintenance of arterial pressure during spinal anaesthesia in caesarean section. Eur J Mol Clin Med. 2021;8(4):1744–53.

5. Kee WD, Khaw KS, Lee BB, Lau TK, Gin T. A randomized double-blinded comparison of phenylephrine and ephedrine for the maintenance of arterial pressure during spinal anesthesia for cesarean section. Anesthesiology. 2001;95(2):307–13.

6. Ganeshanavar A, Ambi US, Shettar AE, Koppal R, Ravi R. Comparison of bolus phenylephrine, ephedrine and mephentermine for maintenance of arterial pressure during spinal anesthesia in caesarean section. J Clin Diagn Res. 2011;5(5):948–52.

7. Simin A, Zahra F, Pouya H, Reza T. Comparison of effect of ephedrine and phenylephrine in treatment of hypotension after spinal anesthesia during cesarean section. Open J Obstet Gynecol. 2012;2(3):192–6.

8. Nazir I, Bhat MA, Qazi S, Buchh VN, Gurcoo SA. Comparison between phenylephrine and ephedrine in preventing hypotension in spinal anesthesia for cesarean section. J Obstet Anesth Crit Care. 2012;2(2):92–7.

9. Bhardwaj N, Jain K, Arora S, Bharati N. A comparison of three vasopressors for tight control of maternal blood pressure during cesarean section under spinal anesthesia: effect on maternal and fetal outcome. J Anaesthesiol Clin Pharmacol. 2013;29(1):26–31.

10. Singh PM, Singh NP, Reschke M, Ngan Kee WD, Palanisamy A, Monks DT. Vasopressor drugs for the prevention and treatment of hypotension during neuraxial anaesthesia for caesarean delivery: a Bayesian network meta-analysis of fetal and maternal outcomes. Br J Anaesth. 2020;124(3):e95–e107. doi:10.1016/j.bja.2019.09.045

11. Tiwari JP, Verma SJ, Singh AK. A prospective randomized study comparing the bolus doses of norepinephrine and phenylephrine for the treatment of spinal-induced hypotension in cesarean section. Cureus. 2022;14(7):e27166. doi:10.7759/cureus.27166

12. Thomas DG, Robson SC, Redfern N, Hughes D, Boys RJ. Randomized trial of bolus phenylephrine or ephedrine for maintenance of arterial pressure during spinal anaesthesia for caesarean section. Br J Anaesth. 1996;76:61–5.

13. Gunda CP, Malinowski J, Tegginmath A, Suryanarayana VG, Chandra SBC. Vasopressor choice for hypotension in elective caesarean section: ephedrine or phenylephrine? Arch Med Sci. 2010;6:257–63.

14. Kee WD, Khaw WD, Lau KS, Ng TK, Chui FF, et al. Randomised double-blinded comparison of phenylephrine vs ephedrine for maintaining blood pressure during spinal anaesthesia for non-elective caesarean section. Anaesth. 2008;63:1319–26.

Relationship between Anterior Cruciate Ligament Thickness and Intercondylar Distance in Magnetic Resonance Imaging

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ABSTRACT

Introduction: The anterior cruciate ligament is crucial for maintaining knee biomechanics. It attaches proximally to the posteromedial aspect of the lateral femoral condyle and distally to the anterior intercondylar region. Intrinsic factors such as a narrow femoral intercondylar distance and a small notch width index increase the risk of its injury. A narrow notch often corresponds to a thinner, weaker anterior cruciate ligament, making it more prone to rupture. Identifying individuals with smaller intercondylar distances may help implement preventive strategies during sports and physical activities. **Aims:** To determine the relationship between anterior cruciate ligament thickness and intercondylar distance of the knee on Magnetic Resonance Imaging. **Methods:** This hospital-based, cross-sectional study was conducted with a total of 57 patients undergoing knee Magnetic Resonance Imaging for various indications. Data were analyzed using statistical package for social sciences version 26, applying Pearson's correlation coefficient with a 5% significance level ($p \leq 0.05$). **Results:** Of 57 patients (24 males, 33 females; mean age 40.6 years), 31 were of the right knee and 26 of the left. The mean intercondylar distance was 19.43 mm, while mean mediolateral and anteroposterior anterior cruciate ligament thicknesses were 4.60 mm and 4.76 mm, respectively. Males showed slightly larger values than females, but the differences were not statistically significant. A strong positive correlation was found between intercondylar distance and both mediolateral ($r = 0.79$, $p < 0.001$) and anteroposterior ($r = 0.78$, $p < 0.001$) anterior cruciate ligament thickness. **Conclusion:** The study demonstrated a significant positive correlation between anterior cruciate ligament thickness and intercondylar distance, with no significant gender difference. Wider intercondylar distances are associated with thicker anterior cruciate ligaments, indicating anatomical variation may influence ligament strength and injury risk.

Keywords: Anterior Cruciate Ligament, Intercondylar width, Magnetic Resonance Imaging

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INTRODUCTION

The anterior cruciate ligament (ACL) is crucial for knee stability and is the most commonly injured ligament.[1] The intercondylar notch, located between the femoral condyles, houses ACL, posterior cruciate ligament(PCL), the meniscofemoral ligaments, other central fibrous attachments of the menisci and peri cruciate fat.[2,3] Variations in notch width and shape, which differ among ethnic groups, are significant anatomical risk factors for ACL injury along with hormonal, genetic and biomechanical factors.[4,5,6] A narrow or abnormally shaped notch can cause ACL impingement, thinning, and increased rupture risk, and is also linked to degenerative joint diseases and osteochondritis dissecans in children.[3,4,6] As the incidence of ACL) injury continues to rise in children and adolescents, identifying independent risk factors such as narrow intercondylar distance and evaluating ACL morphology in relation to anthropometric data and other knee structures including intercondylar width can help apply preventive strategies and guide the selection of appropriate graft thickness.[7, 8] Since Nepalese individuals gen-

erally have shorter stature and narrower notches than western populations, they may face a higher risk of ACL injury.[4,6] Magnetic Resonance Imaging (MRI) is the preferred tool for accurate ACL morphology assessment as well as in establishment of its relationship with intercondylar width.[8,9] This study was undertaken as a pioneer to evaluate the relationship between anterior cruciate ligament thickness and intercondylar distance of the knee on Magnetic Resonance Imaging in western region of Nepal.

METHODS

This cross-sectional study was carried out in the department of radiology at Nepalgunj Medical College Teaching Hospital, Kohalpur, Banke from February 2025 to October 2025. Ethical clearance was obtained from the Institutional Review Committee, Nepalgunj Medical College and Teaching Hospital. The study included 57 cases with age above 16 years, who came for MRI of knee joint for various indications. Patients with skeletal immaturity, previous surgery or degenerative alterations in the knee, not willing to give consent and those with ACL injury were not included in the study. Age, history of knee disease, and prior surgery were recorded. MRI scans were performed using a 1.5T GE Signa Creator with an eight-channel coil. T2WI and Proton density–weighted sequences in sagittal, coronal, and axial planes (with and without fat saturation) were obtained using TE 1642, TE 30, matrix 512×256, FOV 16×16, slice thickness 3.5 mm, and interval 0.3 mm. Images were analyzed, and each measurement was taken three times, with the average value used for analysis.

The following measurements were obtained from the MRI

• Intercondylar distance which is the distance from the medial articular cartilage margin of the lateral femoral condyle to the lateral articular margin of the medial femoral condyle was obtained from the PD-weighted or T2WI sequences in the axial plane.

• Antero-posterior thickness of the ACL was obtained from the PD or T2WI weighted sequences in the sagittal plane, by means of linear measurement in its middle third, perpendicular to the long axis of the ligament fibers.

• Mediolateral (transverse) thickness of the ACL was obtained from the PD-weighted or T2WI sequences in the axial plane, by means of transverse linear measurements in its middle third, taking the greatest diameter of the ligament fibers.

Data were tabulated in Microsoft Excel and analyzed using SPSS version 26. Descriptive statistics, including mean, standard deviation, minimum, and maximum values, were calculated. Comparisons were made using paired samples t-tests, with a p-value of <0.05 considered statistically significant.

RESULTS

Patient's ages ranged from 16 to 78 years, with a mean of 40.61 ± 13.78 years. Of the 57 patients, 24 (42.1%) were male and 33 (57.9%) were female. Among the 57 knee MRIs, 31 (54.39%) were of the right side and 26 (45.61%) of the left side. Most patients underwent knee MRI due to knee pain (n=37), followed by trauma (n=13), swelling around the knee joint (n=5), and other complaints such as burning or tingling sensations, knee deformity, or tumor around the knee (n=2). The mean mediolateral ACL thickness on the axial plane was 4.60 ± 0.76 mm (range 2.90–5.90 mm), and the mean anteroposterior thickness on the sagittal plane was 4.76 ± 0.93 mm (range 2.18–6.10 mm). The mean axial intercondylar distance was 19.43 ± 2.89 mm (range 14.20–27.00 mm). Among males, the mean mediolateral and anteroposterior ACL thicknesses were 4.73 ± 0.61 mm and 5.01 ± 0.88 mm, respectively, with an intercondylar distance of 20.35 ±2.58mm.Among females, the mean mediolateral and anteroposterior ACL thicknesses were 4.50 ± 0.84 mm and 4.57 ± 0.94 mm, respectively, with an intercondylar distance of 18.77 ± 2.96 mm. Overall, males demonstrated slightly higher ACL thickness and intercondylar distance compared to females.

N=57(M=24, F=33)	ML ACL thickness in axial plane (mm)			AP ACL thickness in sagittal plane(mm)			Axial intercondylar distance (mm)		
	M	F	Total	M	F	Total	M	F	Total
Mean	4.73	4.50	4.60	5.01	4.57	4.76	20.35	18.7	19.43
Std. Deviation	0.61	0.84	0.76	0.88	0.94	0.93	2.58	2.96	2.89
Std.Error of Mean	0.12	0.14	0.10	0.18	0.16	0.12	0.52	0.51	0.38
Minimum	3.80	2.90	2.90	2.18	2.90	2.18	16.3	14.2	14.20
Maximum	5.90	5.90	5.90	6.10	6.0	6.10	27.0	26.0	27.00

Table I: Measurement of Medio-lateral(ML) ACL Thickness (in axial plane), Antero-posterior(AP) ACL thickness (in sagittal plane) and Intercondylar distance

Correlation Analysis of Intercondylar Distance and ACL Thickness

Pearson's correlation analysis demonstrated a strong, positive, and statistically significant relationship between intercondylar distance and both ACL thickness parameters (Table II and III). The correlation between intercondylar distance and mediolateral ACL thickness was r = 0.79 (n = 57, p < 0.001), and between intercondylar distance and anteroposterior ACL thickness was r = 0.78 (n = 57, p < 0.001). Fisher's r-to-z transformation yielded 95% confidence intervals of 0.67–0.87 and 0.65–0.86, respectively. Linear regression analysis further confirmed these findings, showing a statistically significant positive correlation between intercondylar distance and both mediolateral and anteroposterior ACL thickness (Figures 1 and 2)

	Parameter	ML ACL thickness	AP ACL thickness
Intercondylar Distance	Pearson Correlation	0.79	0.78
	Significance(2-tailed)	<0.001	<0.001
	N	57	57

Table II: Correlation of intercondylar distance with mediolateral(ML) and anteroposterior(AP) ACL thickness

	Parameter	ML ACL thickness	AP ACL thickness
Intercondylar Distance	95% confidence intervals(2-tailed)		
		Lower	Upper
	ML ACL thickness	0.67	0.87
	AP ACL thickness	0.65	0.86

Table III: Confidence interval between intercondylar distance and ACL thickness

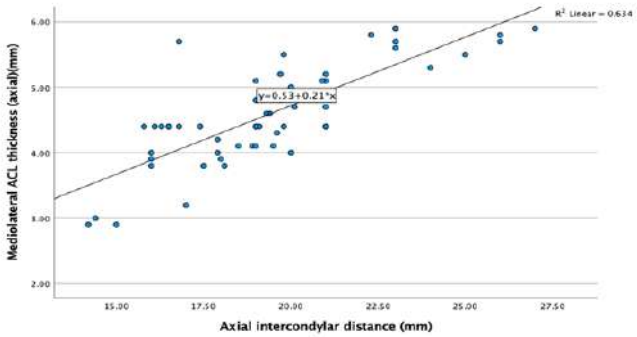

Figure 1: Linear regression analysis of intercondyar distance and mediolateral ACL thickness in axial plane

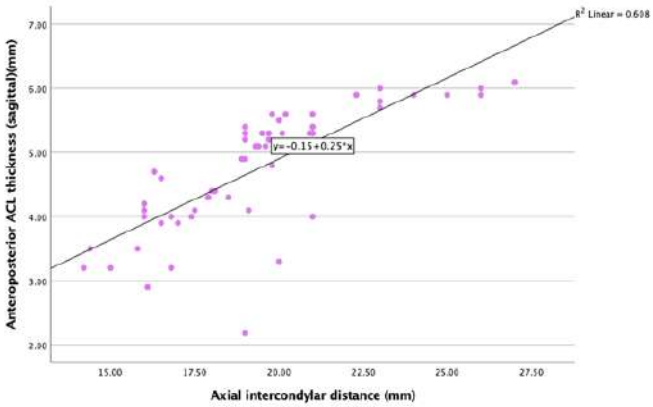

Figure 2: Linear regression analysis of intercondyar distance and anteroposteriro ACL thickness in sagittal plane

CORRELATION BETWEEN TWO GENDERS FOR MEAN INTERCONDYLAR DISTANCE AND ACL THICKNESS

Independent two-sample t-tests (equal variances assumed) showed no significant difference between males and females in mediolateral (p = 0.27, 95% CI: –0.18 to 0.63) and anteroposterior ACL thickness (p=0.80, 95% CI:–0.05 to 0.93). However, a significant difference was observed in intercondylar distance (p = 0.04, 95% CI: 0.67 to 3.09), with males demonstrating higher mean values as shown in table IV.

Parameters	F	Sig	t	df	p (2-tailed)	Mean difference	95 % CI for difference
Mediolateral ACL thickness	1.65	0.20	1.11	55	0.27	0.22	−0.18 to 0.63
Anteroposterior ACL thickness	1.11	0.29	1.78	55	0.80	0.44	−0.05 to 0.93
Intercondylar distance	0.96	0.33	2.09	55	0.04	1.58	0.67 to 3.09

*** Significant at p < 0.05**

Table IV: Independent t-test comparison of ACL thickness and intercondylar distance between genders(equal variance assumed)

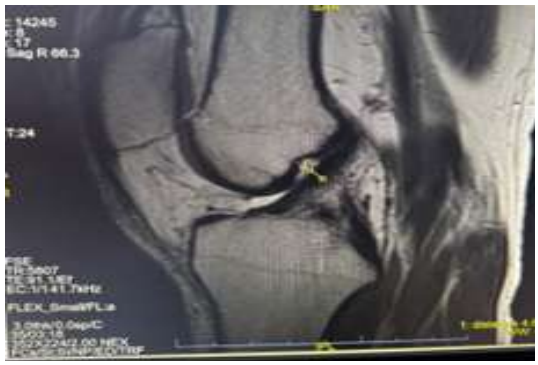

Figure 3: ACL Ligament in Sagittal T2WI

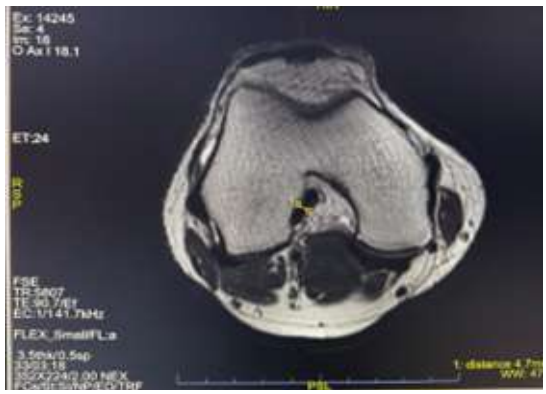

Figure 4: ACL in Axial T2WI

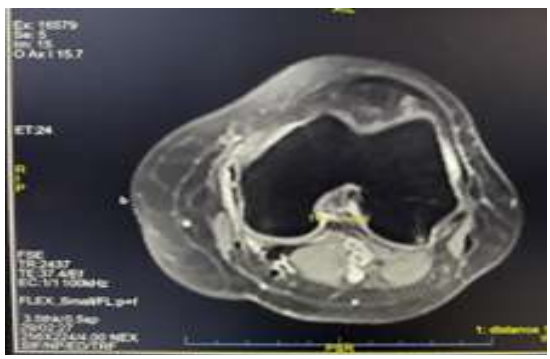

Figure 5: Intercondylar distance in Axial PD

DISCUSSION

MRI is the primary imaging modality for evaluating the knee joint, including ligaments, menisci, and soft tissues. Its superior soft tissue contrast and precise digital measurements allow accurate assessment of bony and ligamentous structures.[10]

Our study analyzed 57 knee MRI scans (mean age 40.6 years; range 16-78), comprising 24 males (42.1%) and 33 females (57.9%). Right knees accounted for 54.4% and left knees for 45.6%. The mean intercondylar distance was 19.43 ± 2.89 mm (range 14.2–27.0 mm), averaging 20.35 ± 2.58 mm in males and 18.77 ± 2.96 mm in females. Study done by De Oliveira VM et al showed mean axial intercondylar distance of 21.7mm (range 15.8 -30.00mm).[8] Hirtler L et al found intercondylar distance ranges from 16.23 ± 2.71 mm before the age of 11 years to 19.38 ± 2.90 mm in middle age and then decreases to 18.6 ± 2.36 mm after the age of 60 years.[3] Shelbourne KD reported significantly wider intercondylar notches in African Americans than in whites, with mean widths of 15.5 ± 2.8 mm vs. 14.1 ± 2.5 mm in women ($p = 0.009$) and 18.0 ± 3.6 mm vs. 16.9 ± 3.1 mm in men ($p = 0.003$).[11]

The mean ACL thickness was 4.60 ± 0.76 mm mediolateral and 4.76 ± 0.93 mm anteroposterior. In males, these measured 4.73 ± 0.61 mm and 5.01 ± 0.88 mm, while in females they were 4.50 ± 0.84 mm and 4.57 ± 0.94 mm, respectively.

Several studies have reported variations in ACL thickness and width measurements. De Oliveira VM et al found a mean mediolateral ACL thickness of 4.3 mm (range 2.9-6.2 mm) and anteroposterior thickness of 4.5 mm (range 3.1-7.2 mm).[8] Kupczik F et al reported a mean ACL width of 4.8 mm (range 3.1-8.3 mm).[12] Anderson AF et al observed mean frontal thicknesses of 4.75 mm in women and 5.6 mm in men, and sagittal thicknesses of 7.6 mm in women and 8.7 mm in men.[13] Marieswaran M et al noted ACL breadth of approximately 10 mm and width ranging from 4–10 mm, comparable to the present study.[14] Ontoh LAP et al found an average ACL width of 9.98 ± 1.5 mm (range 7.59–13.70 mm), with men showing higher values (10.4 ± 1.4 mm) than women (9.1 ± 1.3 mm; $p < 0.0001$).[15] Eleni Triantafyllidi et al reported a midsubstance width of 5 mm in cadaveric ACLs.[16] A Saxena found an average ACL width of 9.38 ± 1.58 mm on MRI, while T.J. Davis et al reported 5.7 ± 1.1 mm in women and 7.1 ± 1.2 mm in men ($p < 0.001$).[17,18]

In our study, a statistically significant linear correlation was found between axial intercondylar distance and mediolateral ACL thickness ($r = 0.796$, $p < 0.001$). De Oliveira VM et al also reported a significant correlation ($r = 0.29$, $p = 0.039$) between these parameters in their study of 48 knee MRIs.[8] Similarly, T.J. Davis et al observed a strong correlation between intercondylar distance and ACL width ($r = 0.87$, $p < 0.001$), consistent with our findings. They also noted smaller intercondylar distances and ACL widths in females (16.2 ± 2.3 mm) compared to males (19.0 ± 2.0 mm).[17] Our study showed a similar trend, with females having a smaller mean intercondylar distance and ACL width than males, though the difference was not statistically significant.

Furthermore, we found a significant correlation between axial intercondylar distance and anteroposterior ACL thickness in the sagittal plane ($r = 0.78$), which contrasts with De Oliveira VM et al, who reported no significant relationship ($r = 0.03$, $p = 0.809$).[8]

Tomas Fernandez et al also found a sex-related difference in notch width (men: 19.3 ± 3.3 mm; women: 17.4 ± 3.1 mm; $p < 0.001$).[20] In contrast, our study also showed a smaller mean intercondylar distance in females than males, but the difference was not statistically significant. Takeshi Muneta et al found results contrary to most studies, showing that both narrow and wide intercondylar notches have ACLs of similar size.[21] Shayan Hosseinzadeh et al reported findings similar to ours, noting smaller ACLs in females.[22] In contrast, M. Rizzo et al, in a cadaveric study of 27 knees, reported significantly larger ACL widths in males (10.59 ± 1.30 mm) than in females (8.09 ± 1.12 mm) ($p < 0.001$). The mean femoral intercondylar notch widths were 20.18 ± 2.20 mm in males and 20.50 ± 1.69 mm in females.[23] Balgovind SR et al reported similar findings, noting that a narrow intercondylar notch often contained a smaller ACL.[4]

LIMITATIONS

The study measured only the axial intercondylar notch distance of the femur, excluding parameters like notch width index and notch angle. MRI was performed in the resting knee position, not in extension or flexion, which might influence ACL width. The study included a heterogeneous population without specific age grouping and did not assess ACL length, focusing instead on the relationship between ACL thickness and intercondylar distance. The small sample size was also a limitation.

CONCLUSION

Our study showed a strong positive correlation between intercondylar distance and ACL thickness indicating that individuals with narrower intercondylar notches tend to have smaller ACLs. Conversely, those with wider intercondylar distances had thicker ACLs. The study also found that ACL size varies significantly among individuals, and axial intercondylar distance can serve as a useful indicator of ACL thickness. Although males generally had larger measurements than females, the differences were not statistically significant.

REFERENCES

1. Ng WHA, Griffith JF, Hung EHY et al.Imaging of the anterior cruciate ligament.World J Orthop.2011;2(8):75-84

2. Sandeep DV, Rao DAS. Magnetic resonance imaging based anthropometric study of knee joint in anterior cruciate ligament insufficient knees. Int J Orthop. 2018;4(2):635–9

3. Hirtler L, Röhrich S, Kainberger F. The Femoral Intercondylar Notch During Life: An Anatomic Redefinition With Patterns Predisposing to Cruciate Ligament Impingement. AJR Am J Roentgenol. 2016;207(4):836–45

4. Balgovind SR, Raunak B, Anusree A. Intercondylar notch morphometrics in Indian population: An anthropometric study with magnetic resonance imaging analysis. J Clin Orthop and Trauma. 2019;10(4):702–5

5. Huang M, Li Y, Guo N, Liao C, Yu B. Relationship between

intercondylar notch angle and anterior cruciate ligament injury: a magnetic resonance imaging analysis.The J of Int Med Res. 2019;47(4):1602–9

6. Basukala B, Joshi A, Pradhan I. The Effect of the Intercondylar Notch Shape and Notch Width Index on Anterior Cruciate Ligament Injuries. J. Nepal Health Res Counc . 2020;17(4):532–6

7. Yellin JL, Parisien RL, Talathi NS, Farooqi AS, Kocher MS, Ganley TJ. Narrow NotchWidth is a Risk Factor for Anterior Cruciate Ligament Injury in the PediatricPopulation: A Multicenter Study. Arthrosc Sports Med Rehabil.2021;3(3):e823–8

8. De Oliveira VM, Latorre GC, Netto A dos S, Jorge RB, Filho GH, de Paula Leite Cury R. Study on the relationship between the thickness of the anterior cruciate ligament, anthropometric data and anatomical measurements on the knee. Rev Bras Ortop Journal. 2016;51(2):194–9

9. Zhao M, Zhou Y, Chang J, Hu J, Liu H, Wang S et al. The accuracy of MRI in the diagnosis of anterior cruciate ligament injury. Ann Transl Med. 2020;8(24):1657

10. Herzog RJ, Silliman JF, Hutton K, Rodkey WG, Steadman JR. Measurements of the intercondylar notch by plain film radiography and magnetic resonance imaging. Am J Sports Med. 1994;22(2):204- 10

11. Shelbourne KD, Gray T, Benner RW. Intercondylar notch width measurement differences between african american and white men and women with intact anterior cruciate ligament knees.Am J Sports Med. 2007;35(8):1304–7

12. Kupczik F, Eduardo M, Schiavon G, Sbrissia B, Caldonazzo R, Valério R. ACL ideal graft : MRI correlation between ACL and hamstrings , PT and QT.Res Bras Ortop. 2013;48(5):441–7

13. Anderson AF, Dome DC, Gautam S, Awh MH, Rennirt GW. Correlation of Anthropometric Measurements, Strength, Anterior Cruciate Ligament Size, and Intercondylar Notch Characteristics to Sex Differences in Anterior Cruciate Ligament Tear Rates.Am J Sports Med.2001;29(1):58–66

14. Marieswaran M, Jain I, Garg B, Sharma V, Kalyanasundaram D. A Review on Biomechanics of Anterior Cruciate Ligament and Materials for Reconstruction. Appl Bionics Biomech.2018;2018:1-14

15. Ontoh LAP, Rahyussalim AJ, Fiolin J. Patient Height may Predict the Length of the Anterior Cruciate Ligament: A Magnetic Resonance Imaging Study. Arthrosc Sports Med Rehabil. 2021;3(3):e733–9

16. Triantafyllidi E, Paschos NK, Goussia A, Barkoula NM, Exarchos DA, Matikas TE, et al. The Shape and the Thickness of the Anterior Cruciate Ligament Along Its Length in Relation to the Posterior Cruciate Ligament: A Cadaveric Study. J Arthrosc Related Surg. 2013;29(12):1963–73

17. Saxena A, Ray B, Rajagopal K V., D'Souza AS, Pyrtuh S. Morphometry and magnetic resonance imaging of anterior cruciate ligament and measurement of secondary signs of anterior cruciate ligament tear. Bratisl Med J. 2012;113(09):539–43

18. Davis TJ, Shelbourne KD, Klootwyk TE. Correlation of the intercondylar notch width of the femur to the width of the anterior and posterior cruciate ligaments. Knee Surg Sports Traumatol Arthrosc. 1999;7(4):209–14

19. Li H, Zeng C, Wang Y, Wei J, Yang T, Cui Y, et al. Association Between Magnetic Resonance Imaging–Measured Intercondylar Notch Dimensions and Anterior Cruciate Ligament Injury: A Meta-analysis. J Arthrosc Surg. 2018;34(3):889–900

20. Fernández-Jaén T, López-Alcorocho JM, Rodriguez-Iñigo E, Castellán F, Hernández JC, Guillén-García P. The Importance of the Intercondylar Notch in Anterior Cruciate Ligament Tears. Orthop J Sports Med. 2015;3(8):232

21. Hosseinzadeh S, Kiapour AM. Age-related changes in ACL morphology during skeletal growth and maturation are different between females and males.J Orthop Res.2021;39(4)841–9

22. Hosseinzadeh S, Kiapour AM. Age-related changes in ACL morphology during skeletal growth and maturation are different between females and males.J Orthop Res.2021;39(4)841–9

23. Rizzo M, Holler SB, Bassett FH. Comparison of males' and females' ratios of anterior-cruciate-ligament width to femoral-intercondylar-notch width: a cadaveric study. Am J Orthop. 2001;30(8):660–4

Prevalence of Hyponatremia in Chronic Liver Disease Patients and its Correlation with Disease Severity

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ABSTRACT

Introduction: Hyponatremia is the most common electrolyte abnormality in patients with chronic liver disease, signaling poor prognosis and contributing to mortality worldwide. In CLD, hyponatremia is often linked to more severe liver disease and is an independent predictor of worse outcomes, including complications like difficult-to-control ascites, hepatic encephalopathy, and increased mortality. **Aims:** To determine the prevalence of hyponatremia in patients with chronic liver disease and its correlation with disease severity. **Methods:** This descriptive cross-sectional study enrolled 104 patients with chronic liver disease admitted to Nobel Medical College from July 2024 to June 2025. Demographic data, medical history, and risk factors were collected via structured questionnaire. Chronic liver disease severity was assessed using the Child-Pugh scoring system. Data were analyzed with SPSS version 20.0; $p < 0.05$ indicated statistical significance. **Results:** Chronic liver disease prevalence was 25.9%, with a mean age of 50.6 years, with male predominance (71.2%). Alcohol was the leading cause (80.8%), followed by metabolic dysfunction-associated steatotic liver disease (13.5%). Common symptoms included ascites (68.2%) and jaundice (58.7%). Major complications were varices (39.4%), portal hypertension (33.7%), and encephalopathy (23.1%). Hyponatremia was observed in 74 (71.2%), and normal sodium in 30 (28.8%). Spearman's rank correlation analysis showed no significant association between Child-Turcotte-Pugh score and hyponatremia ($\rho = -0.068$, $p = 0.494$). **Conclusion:** The prevalence of hyponatremia among the patients with chronic liver disease was found to be higher and found no correlation between Child-Turcotte-Pugh class and hyponatremia.

Keywords: Chronic liver disease, Child-Turcotte-Pugh score, Hepatic encephalopathy, Hyponatremia

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INTRODUCTION

Chronic liver diseases is a progressive destruction of the liver parenchyma over a period greater than 6 months leading to fibrosis and cirrhosis. Cirrhosis is marked by ongoing inflammation, fibrosis, and regenerative nodules that damage the liver architecture and impair the liver function.[1] It has emerged as a major global health issue, ranking as the 13th leading cause of death worldwide and accounting for over one million deaths annually.[2,3] CLD is a growing global health concern, driven by the increasing prevalence of obesity and MASLD,now the most common cause of CLD.[4] Hyponatremia is the most common electrolyte abnormality observed in patients with CLD.[5] Hyponatremia in cirrhosis is defined as a serum sodium level of less than 130 mEq/L.[6] In patients with cirrhosis and ascites, the prevalance of serum sodium level less than 135, 130, and 120 mEq/L was 49.4%, 21.6%, and 1.2%, respectively.[7] It can be due to hypovolemia due to loss of extracellular fluid due to diuretics or expanded extracellular fluid volume due to the inability of the kidneys to excrete solute-free water proportionate.[8] A number of recent studies have shown the association of hyponatremia with greater severity of complications of CLD, namely, difficult-to-control ascites, and greater frequency of complications posttransplant, including neurologic disorders, renal failure, and infectious complications.[9] Hyponatremia is found to be associated with increased morbidity and mortality in patients with cirrhosis.[8] This study was conducted to estimate the prevalence of hyponatremia in chronic liver disease patients and to assess the correlation of hyponatremia with the disease severity.

METHODS

This descriptive cross-sectional study was conducted over one year from July 2024 to June 2025, at the Department of Gastroenterology, Nobel Medical College Teaching Hospital, Biratnagar, Nepal. A non-probability consecutive sampling technique was used to enroll study subjects.

Inclusion criteria: Patients with CLD of any cause of more than 18 years, diagnosed as per clinical, biochemical, and radiological parameters, were included.

Exclusion criteria: Patients less than 18 years old, patients with an existing case of CKD or cardiac failure, patients currently receiving diuretics, and patients who did not give consent were excluded.

The sample size for this study was calculated using the single-population proportion formula:

$n = (Z^2 \times p \times q) / d^2$

A previous study10 reported a hyponatremia prevalence of 41.22% (p = 0.4122) among patients with chronic liver disease, giving q = 1 − p = 0.5878. Using this prevalence, a 95% confidence level (Z = 1.96), and an allowable error of 10% (d = 0.10), the initial calculated sample size was:

n = 93.1

This value was rounded up to 94. To accommodate an anticipated 5% non-response or incomplete data rate, the adjusted sample size was calculated using:

n_final = n_rounded / (1 − 0.05)

n_final = 94 / 0.95 ≈ 98.95

Thus, the final required sample size for the study was 99 participants.

A predetermined proforma was used as the data collection tool. All enrolled patients underwent a detailed clinical history, which included age, sex, and history of alcohol intake, and a clinical examination, which included vitals and a general and systemic examination. Biochemical tests included alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine transaminase (ALT), total and direct bilirubin, prothrombin time/international normalized ratio (PT/INR), serum albumin, urea, creatinine, and sodium.

Upper gastrointestinal (UGI) endoscopy was performed to rule out varices and other pathological conditions, with findings documented accordingly. Abdominal ultrasound assessed liver and spleen size, parenchymal echogenicity, portal vein diameter, and presence of ascites. Portal hypertension was diagnosed using noninvasive tools like USG and CT scans and the presence of splenomegaly, ascitis, low platelet and varices.

Child-Turcotte-Pugh (CTP) score was calculated for all patients. [11]

Vriable	1 point	2 point	3 point
Bilirubin level	<2mg/dl	2-3 mg/dl	>3mg/dl
Albumin level	>3.5g/dl	2.8-3.5g/dl	<2.8g/dl
International normalized ratio	<1.7	1.7-2.2	>2.2
Encephalopathy	None	Controlled	Uncontrolled
Ascitis	None	Controlled	Uncontrolled

Child-Turcotte-Pugh class

Class A =5-6 points

Class B= 7-9 points

Class C=10-15 points

Statistical analysis

Data were entered and analyzed using Statistical Package for the Social Sciences (SPSS) version 20. Point estimates and 95% confidence intervals (CI) were calculated. The categorical variables were analyzed using Chi-square test and Fisher's exact test. The means were compared among the groups of Child-Pugh class using one-way ANOVA. $P < 0.05$ was considered statistically significant.

RESULTS

104 patients (25.9%) admitted to the Department of Gastroenterology during the one-year study period were found to have CLD. The mean age of the patients with CLD was 50.62 ± 11.36 years. A marked male predominance was observed, with 74 (71.2%) male and 30 (28.8%) female patients, resulting in a male-to-female ratio of approximately 2.5:1.

Patients presented with a wide spectrum of clinical features. Ascites was the most common presenting symptom, seen in 71 patients (68.3%), followed by jaundice in 61 patients (58.7%). Fatigue and melena were both observed in 45 patients (43.3%), and altered sensorium was noted in 40 patients (38.5%), reflecting underlying hepatic encephalopathy. Abdominal pain and anorexia were equally prevalent, occurring in 38 patients (36.5%) each. Other presenting complaints included hematemesis (35.6%), peripheral edema (31.7%), vomiting (24%), pallor (20.2%), low urine output (18.3%), fever (16.3%), and spider naevi (9.6%). (Table I)

Clinical Presentation	N	%
Ascites	71	68.3
Jaundice	61	58.7
Fatigue	45	43.3
Hematemesis	37	35.6
Peripheral edema	33	31.7
Melena	45	43.3
Anorexia	38	36.5
Abdominal Pain	38	36.5
Vomiting	25	24

Altered sensorium	40	38.5
Pallor	21	20.2
Low urine output	19	18.3
Fever	17	16.3
Spider naevi	10	9.6

Table I: Clinical Presentation of CLD Patients

Alcoholic liver disease was identified as the leading cause of CLD in this study, accounting for 84 out of 104 patients (80.8%). Non-alcoholic steatohepatitis (NASH) was the second most common etiology, responsible for 14 cases (13.5%). Less common causes included hepatitis C (4 cases), autoimmune hepatitis (1 case), and hepatitis B (1 case). (Table II)

Underlying etiologies		N	%
Alcoholic		84	80.8
NASH		14	13.5
Others	Autoimmune	1	5.8
	Hepatitis-B	1	
	Hepatitis-C	4	
Total		**104**	**100**

Table II: Underlying Etiologies of CLD

Various complications associated with chronic liver disease were noted. Ascites was again the most prevalent, found in 71 patients (68.2%). Esophageal varices were present in 41 patients (39.4%), and portal hypertension in 35 patients (33.7%). Gastrointestinal bleeding was seen in 30 patients (28.8%), and portal hypertensive gastropathy in 29 patients (27.9%). Hepatic encephalopathy occurred in 30 patients (31.2%), while coagulopathy and hepatorenal syndrome were identified in 9 (8.7%) and 6 (5.8%) patients, respectively. (Table III)

Complication	Frequency	%
Portal hypertension	35	33.7
Hepatorenal syndrome	6	5.8
Ascites	71	68.2
Coagulopathy	9	8.7
Hepatic encephalopathy	30	31.2
Gastrointestinal bleeding	30	28.8
Portal hypertensive gastropathy	29	27.9
Esophageal varices	41	39.4

Table III: Complications Observed in CLD Patients

Most patients (62.5%, or nearly two-thirds) had moderate (Class B) liver disease, about a quarter (24%) had advanced (Class C) disease, a smaller group (13.5%) had well-compensated (Class A) disease. (Table IV)

Child-Turcotte-Pugh (CTP)	N	Percentage (%)
A	14	13.5
B	65	62.5
C	25	24
Total	104	100

Table IV: Distribution of Child-Turcotte-Pugh Scores in CLD Patients

The distribution of liver disease severity (Child-Pugh Class) is very similar between patients who have low sodium (Hyponatremia) and those who don't. In both groups, most patients (~62.2%) are in Class B. The proportion of patients in Class A (~13.5%) and Class C (~24.3%) is also nearly identical. (Table V)

Hyponatremia	Child-Pugh Class N%			
	Class A	Class B	Class C	Total
Yes	10 (13.5%)	46 (62.2%)	18 (24.3%)	74 (100%)
No	4 (13.3 %)	19 (63.3%)	7 (23.3%)	30 (100%)

Table V: Prevalence of Hyponatremia

"Spearman's rank correlation analysis showed no significant association between CTP score and hyponatremia ($\rho = -0.068$, $p = 0.494$). This indicates that the severity of liver disease based on CTP score did not correlate with the presence of hyponatremia among the study participants. (Table VI)

Variable	Category	Normal n (%)	Hyponatremia n (%)	Total n (%)	χ^2 (df)	p value
Age	≤45 years	12 (36.4)	21 (63.6)	33 (100)	—	>0.05
	>45 years	18 (25.4)	53 (74.6)	71 (100)		
Gender	Male	22 (29.7)	52 (70.3)	74 (100)	—	>0.05
	Female	8 (26.7)	22 (73.3)	30 (100)		
Child Score Group	A	4 (28.6)	10 (71.4)	14 (100)	0.001 (1)	0.981
	B/C	26 (28.9)	64 (71.1)	90 (100)		
Portal Hypertension	Absent	21 (70.0)	48 (64.9)	69 (66.3)	0.252 (1)	0.616
	Present	9 (30.0)	26 (35.1)	35 (33.7)		

Table VI: Association of hyponatremia with sociodemographic, complications, and severity of chronic liver disease

DISCUSSION

This study highlights the substantial burden of chronic liver disease (CLD) among hospitalized patients, with an overall prevalence of 25.9%. This rate aligns with regional data, including Poudel et al's report of 20.8% among inpatients at a Nepalese tertiary center.[12] Similarly, Suresh et al observed a high admission rate for CLD in Kerala, India, with males accounting for 79.5% of cases,[13] aligning closely with our observed male predominance (71.2%) and mean age of 50.62 years.

The predominant etiology in this study was alcoholic liver disease (ALD), observed in 80.8% of patients. This is consistent with previous studies in Nepal, where ALD ranged from 79% to 85% as the leading cause of CLD.[12, 14, 15] Cultural acceptance of alcohol and lack of community-level awareness interventions remain critical drivers behind this trend.[14] In contrast, MAFLD is now emerging as a dominant global cause of CLD due to the obesity epidemic, especially in Western and urban Asian populations.[16] However, our study found MAFLD/MASH in only 13.5% of patients, reflecting the continued predominance of alcohol-related etiologies in this region.

The clinical presentation in our cohort mirrors established patterns of decompensation. Ascites was the most common presenting symptom (68.3%), followed by jaundice (58.7%) and hepatic encephalopathy (38.5%). These figures align with the findings of JNMA and Kerala-based studies, which similarly reported high rates of ascites, encephalopathy, and upper GI bleeding.[12,13,15] In our study, 39.4% had esophageal varices, consistent with portal hypertension features reported in similar literature.[13,15,17]

Hyponatremia represented the predominant electrolyte derangement in CLD patients, with none exhibiting hypernatremia (>145 mEq/L). In this cohort, it affected 71.2% (74/104), versus 28.8% (30/104) with normal sodium levels; mean serum sodium among hyponatremic cases was 124.6 mEq/L (range, 116–130 mEq/L).

Our findings reinforce the prognostic significance of complications like hepatic encephalopathy (23.1%) and gastrointestinal bleeding (28.8%), which are also strongly linked with mortality in cirrhotic patients.[10,14] Aetiology-wise, Kim et al demonstrated that patients with viral hepatitis plus alcohol-induced CLD had worse CTP scores and a higher rate of acute-on-chronic liver failure (ACLF).[14] While our study predominantly involved alcoholic etiology, the relatively low representation of viral hepatitis may have limited similar observations.

The application of CTP scores in our study enabled disease severity stratification, though individual score-based outcome correlations were not explicitly analyzed. However, other studies have demonstrated that higher CTP scores are associated with increased mortality, particularly in patients with mixed etiologies or active alcoholism.[16,18]

In CLD, hyponatremia severity correlates directly with heightened risks of complications and mortality. Conventional fluid restriction (≤1.5 L/day) is frequently insufficient. Investigational alternatives include albumin infusions and vaptans (selective vasopressin V2 receptor antagonists). By blocking arginine vasopressin's action on renal aquaporin-2 channels, vaptans induce aquaresis- excreting electrolyte-free water- to raise serum sodium. Short-term use enhances urine output and sodium correction; however, hepatotoxicity limits application in advanced liver disease.[19]

In one Nepali study, 47 of the 114 patients under assessment for chronic liver disease exhibited hyponatremia,constituting 41.22% of the cohort, which had an average age of 53.44±7.57 year.[10] In another study, conducted by Amna et al in Pakistan, the prevalence of hyponatremia in the study group (36.9%) and distribution of hyponatremia severity was as follows: 9.2% mild, 21.5% moderate, and 6.2% severe.[20]

The findings underscore an urgent need for preventive strategies targeting alcohol use, early detection programs in high-risk populations, and public health education regarding liver health. As seen globally, transitioning to a broader screening approach that includes metabolic screening (MASLD) and viral markers is necessary for earlier detection and intervention.[16,20]

Hepatic encephalopathy was significantly more common in patients with hyponatremia (86.7%) compared to those with normal sodium (35.1%), p = 0.026. Hyponatremia was linked to a higher occurrence of hepatic encephalopathy, as found by Younas A, Riaz J, Chughtai T, et al[19] in our study, it was also significantly connected. Younus A et al have also shown a similar relationship. Hyponatremia is known to impact brain function and is therefore considered a risk factor for hepatic encephalopathy.[20]

CONCLUSION

In our study the prevalence of hyponatremia among the patient with CLD was found to be higher. However the proportions of those with hyponatremia increased with severity of CLD but for the significance, the levels of sodium did not differ much with severity of liver diseases as per child-Pugh classification. Hyponatremia in patients with CLD is correctable and timely correction could improve their functional status and quality of life.

REFERENCES

1. de la Garza RG, Morales-Garza LA, Martin-Estal I, Castilla-Cortazar I. Insulin-Like Growth Factor-1 Deficiency and Cirrhosis Establishment. J Clin Med Res. 2017;9(4):233–47. doi:10.14740/jocmr2902w

2. Abbas N, Makker J, Abbas H, Balar B. Perioperative Care of Patients With Liver Cirrhosis: A Review. Health Serv Insights. 2017;10:1178632917691270. doi:10.1177/1178632917691270

3. Stasi C, Silvestri C, Voller F, Cipriani F. Epidemiology of Liver Cirrhosis. J Clin Exp Hepatol. 2015;5(3):272–80. doi:10.1016/j.jceh.2015.08.001

4. Li B, Zhang C, Zhan YT. Nonalcoholic Fatty Liver Disease Cirrhosis: A Review of Its Epidemiology, Risk Factors, Clinical Presentation, Diagnosis, Management, and Prognosis. Can J Gastroenterol Hepatol. 2018:2784537. doi:10.1155/2018/2784537

5. Baran D, Hutchinson TA. The outcome of hyponatremia in a general hospital population. Clin Nephrol. 1984;22:72-76. PMID: 6478674.

6. Ginés P, Berl T, Bernardi M, Bichet DG, Hamon G, Jiménez W, Liard JF, Martin PY, Schrier RW. Hyponatremia in cirrhosis: from pathogenesis to treatment. Hepatology. 1998;28:851-864. doi: 10.1002/hep.510280337.

7. Angeli P, Wong F, Watson H, Ginès P. Hyponatremia in cirrhosis: Results of a patient population survey. Hepatology. 2006;44:1535-1542.doi: 10.1002/hep.21412.

8. John S, Thuluvath PJ. Hyponatremia in cirrhosis: Pathophysiology and management. World J Gastroenterol 2015;21:3197-205.doi: 10.3748/wjg.v21.i11.3197.

9. Gaglio P, Marfo K, Chiodo J 3rd. Hyponatremia in cirrhosis and endstage liver disease: Treatment with the vasopressin V_2-receptor antagonist tolvaptan. Dig Dis Sci 2012;57:2774-85.doi: 10.1007/s10620-012-2276-3

10. Bhandari A, Chaudhary A. Hyponatremia in Chronic Liver Disease among Patients Presenting to a Tertiary Care Hospital: A Descriptive Cross-sectional Study. JNMA J Nepal Med Assoc. 2021;59(244):1225-28. doi: 10.31729/jnma.7152.

11. Pugh RN. Pugh's grading in the classification of liver decompensation. Gut. 1992 Nov;33(11):1583. doi: 10.1136/gut.33.11.1583.

12. Poudel SC, Acharya A, Maharjan S, et al. Chronic Liver Disease among Patients Admitted in the Department of Internal Medicine of a Tertiary Care Centre. JNMA J Nepal Med Assoc. 2023;61(259):212–5. doi:10.31729/jnma.8092

13. Suresh A, Praveen M. An Observational Study to Assess the Clinical Profiles of Patients with Chronic Liver Disease. Int J Adv Med. 2023;10(7):511–7. doi:10.18203/2349-3933.ijam2023186.

14. Shrestha AK, Shrestha A, Shah S, Bhandari A. Clinicodemographic Profile of Chronic Liver Disease Patients at a Tertiary Care Hospital: A Retrospective Analysis. Ann Med Surg. 2023;85:399–402. doi:10.1097/MS9.0000000000000248

15. Maharjan S, Poudel SC, Acharya A, et al. Chronic Liver Disease among Patients Admitted in a Tertiary Care Centre. JNMA J Nepal Med Assoc. 2023;61(259):212–5. doi:10.31729/jnma.8092

16. Kim JH, Kim SE, Song DS, et al. Aetiology of Chronic Liver Disease is a Valuable Factor for Stratifying Adverse Outcomes of Acute Decompensation: Prospective Observational Study. Ann Med. 2025;57(1):2428431. doi:10.1080/07853890.2024.2428431

17. Dharel N, Bajaj JS, Zadvornova Y, et al. Clinical implications of portal hypertensive gastropathy in cirrhosis. Am J Gastroenterol. 2010;105(11):2424–31. doi:10.1038/ajg.2010.273

18. D'Amico G, Morabito A, Pagliaro L, Marubini E. Survival and prognostic indicators in compensated and decompensated cirrhosis. Dig Dis Sci. 1986;31(5):468–75. doi:10.1007/BF01296186.

19. Bernardi M, Ricci CS, Santi L. Hyponatremia in Patients with Cirrhosis of the Liver. J Clin Med. 2014;4(1):85-101. doi: 10.3390/jcm4010085. PMID: 26237020

20. Younas A, Riaz J, Chughtai T, et al. (February 06, 2021) Hyponatremia and Its Correlation With Hepatic Encephalopathy and Severity of Liver Disease. Cureus 13(2): e13175. doi:10.7759/cureus.13175

Early Discharge versus 6-hour Observation in Mild Traumatic Brain Injury with a Normal Brain CT Scan in a Tertiary Center

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ABSTRACT

Introduction: Mild traumatic brain injury is a common condition presenting to emergency departments, often managed with a period of observation. **Aims:** To compare the outcomes between early discharge and a 6-hour observation period in mild traumatic brain injury patients with normal brain CT scans. **Methods:** A prospective observational study was conducted on patients with mild Traumatic brain injury and normal CT scans at Nepalgunj Medical College, Kohalpur, Banke from August-October, 2025. A total of 82 patients presenting to the emergency department with clinical features of mild traumatic brain injury were enrolled and divided into two groups: Group A (Patients discharged early within 2 hours) and Group B (Patients observed in the emergency department for six hours before discharge). Parameters such as post discharge complications, re-admission, neurological deterioration and patient satisfaction were recorded and analyzed using SPSS version 25. **Results:** The mean age of the participants was 31.33±18.09 years, with 68.3% male patients. No significant differences were found in the baseline characteristics such as mechanism of injury, or duration of loss of consciousness between the two groups. Revisit rates to the emergency department within 7 days were similar between the groups. No patients in either group required neurological intervention or hospitalization after discharge. **Conclusion:** Our findings suggested that early discharge is equally safe, with no significant differences in post-discharge complications, neurological deterioration, or patient satisfaction compared to a 6-hour observation period.

Keywords: Brain CT, Concussion, Early discharge, Emergency, Mild Traumatic brain injury, Observation

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INTRODUCTION

Traumatic Brain Injury (TBI) is a prevalent neurological condition resulting from external mechanical force, often due to falls, vehicle accidents, or blunt trauma. It represents a major public health concern globally, as it can lead to a range of temporary or permanent cognitive, physical, and psychosocial impairments.[1,2] TBI is typically classified into mild, moderate, and severe categories based on the Glasgow Coma Scale (GCS), duration of loss of consciousness (LOC), and extent of post-traumatic amnesia. Mild Traumatic Brain Injury (mTBI), often referred to as a concussion, accounts for the majority of TBI cases. Patients typically present with transient loss of consciousness or altered mental status. Symptoms may include headache, dizziness, fatigue, irritability, memory problems, and mood disturbances.[3,4] Though most individuals recover fully, a significant subset may experience persistent post-concussion symptoms lasting several weeks to months.[5] The acute management of mTBI often includes diagnostic neuroimaging, observation, symptom monitoring, and patient education. Among patients with mTBI and a normal brain CT scan, there is ongoing debate about the necessity and duration of emergency department (ED) observation. Some guidelines recommend a six-hour observation period to monitor for late-appearing complications, while others suggest that early discharge with adequate instructions and outpatient follow-up may be equally safe and more efficient.[8,9] However, current clinical practices vary significantly, and no consensus has been established regarding the optimal strategy for these patients.

METHODS

This was a prospective observational study conducted in the Emergency Department of Nepalgunj Medical College located in the Western Nepal. The study period was from August to October 2025, following ethical approval from the Institutional Review Board of our institution. This study was aimed to compare and observe the outcomes between two cohorts of pa-

tients with mild traumatic brain injuries and a normal brain CT scan to those who are discharged early (within 2 hours) and those who were observed for six hours. This study consisted of 82 patients divided into two groups, each with 41 patients. The inclusion criteria were patients of age more than 18 years, GCS 14-15 at the time of enrollment, normal non-contrast CT scan of the brain, hemodynamic stability, no prior history of anticoagulant or antiplatelet use, and willing to participate in the study, whereas the exclusion criteria were abnormal CT findings (Intracranial hemorrhage, edema, skull fracture), moderate or severe TBI (GCS 13 or less), polytrauma, pregnancy, known psychiatric illness. For the sample size calculation, the sample size of the study was calculated based on the post-discharge event rate of our centre i.e. 5%, assuming a 5% expected event rate with a 95% confidence interval and a 7% margin of error. The required sample size was calculated by using Cochran's formula. Patients were categorized into two observational groups: Group A, consisting of patients discharged early within two hours of normal CT scan and stable condition, and Group B, patients observed in the Emergency Department for six hours before discharge. Both groups received standard discharge instructions including red flag symptoms, follow-up recommendations, and a contact number for concerns. Demographic and clinical data were recorded. Patients were called for a follow-up visit in the Neurosurgery OPD after one day to assess any new symptoms or deterioration, and after seven days to assess overall neurological status or events on return visit, including neurological deterioration, hospital readmission, need for surgical evaluation, and patient dissatisfaction with the care.

Statistical Analysis

Data management and analysis were conducted using a structured proforma, analyzed by using SPSS version 25 for analysis. Descriptive statistics were used to summarize the demographic data and Chi-square or Fisher's exact test was used to compare categorical outcomes between the two groups. A value of less than 0.05 was considered to be statistically significant.

RESULTS

A total of 82 patients (41 in each group) participated in the study, with a mean age of 31.33 ± 18.09 years (range: 18–70 years). The sample consisted of 56 (68.3%) male and 26 (31.7%) female participants. The severity of traumatic brain injury (TBI) was consistent across both groups, with all patients having a Glasgow Coma Scale (GCS) score of 15, indicating mild TBI. The mechanisms of injury were predominantly Road Traffic Accident 46 (56.09%) followed by fall injury 20 (24.39%) and Physical assault 16 (19.51%). Twenty eight (34.14%) patients experienced loss of consciousness (LOC), duration being less than 5 minutes. Follow-up rates were 100% in both groups at 1 day and 7 days. (Table I) In terms of revisit rates, 9.7% of patients in Group A and 4.8% of Group B revisited the emergency department (ED) within 7 days due to mild symptoms like dizziness and headache, though none required repeat CT scans or further interventions (p = 0.39). Regarding post-concussion syndrome (PCS), at 1 day, group A patients experienced vomiting 8 (19.51%), dizziness 3 (7.3%), headache 6 (14.63%) and anxiety 8 (19.51%). Compared to group B, vomiting 5 (12.19%), dizziness 2 (4.8%), headache 5 (12.19%) and anxiety 2 (4.8%). Follow-up assessments indicated that no patient in either group experienced neurological deterioration requiring further intervention with overall satisfaction rate of 100% (Table II).

Characteristic	Total (n=82)
Age (years), mean	31.33 ± 18.09
Male sex, n (%)	56 (68.3%)
Female sex, n (%)	26 (31.7%)
Mechanism of injury, n (%)	
– Road traffic accident	46 (56.1%)
– Fall injury	20 (24.4%)
– Physical assault	16 (19.5%)
Loss of consciousness (LOC), n (%)	28 (34.1%)
Duration of LOC (all cases)	<5 minutes
Follow-up rate at 1 day	100%
Follow-up rate at 7 days	100%

Table I: Demographic and Clinical Characteristics of Patients

Outcome / Symptom	Group A Early Discharge (n=41)	Group B 6-hour Observation (n=41)	p-value
Revisit to ED within 7 days, n (%)	4 (9.8%)	2 (4.9%)	0.398
Required repeat CT scan	0	0	–
Required admission or neurosurgical intervention	0	0	–
Neurological deterioration	0	0	–
Patient satisfaction	100%	100%	1.000
Post-concussion symptoms at 1-day follow-up			
Any post-concussion symptom	14 (34.1%)	10 (24.4%)	0.489
Headache, n (%)	6 (14.6%)	5 (12.2%)	0.745
Vomiting, n (%)	8 (19.5%)	5 (12.2%)	0.368
Dizziness, n (%)	3 (7.3%)	2 (4.9%)	0.643
Anxiety / Irritability, n (%)	8 (19.5%)	2 (4.9%)	0.041
Persistent symptoms at 7-day follow-up			
Any persistent symptom, n (%)	~5 (12.2%)	~5 (12.2%)	1.000

Table II: Outcomes of Patients with Mild Traumatic Brain Injury (mTBI)

DISCUSSION

A clear definition of mild traumatic brain injury is vital to guide the clinical management. mTBI is characterized by a GCS score of 14–15, brief loss of consciousness (<30 minutes), short duration of post-traumatic amnesia (<24 hours), and absence of structural brain injury on imaging.[15] The management of mTBI with a normal brain CT scan remains one of the most debated topics in emergency medicine and neurosurgery. Although the prognosis of mTBI is generally favorable, studies report that around 18.7% of patients may suffer persistent post-concussion symptoms, including fatigue, forgetfulness, and psychological distress.[9] Several predictors of poor outcomes have been identified, such as pre-existing psychiatric illness, poor communication in the emergency department, and inadequate discharge instructions.

Our study of 82 low-risk adult patients demonstrated that early discharge within 2 hours of a normal CT is as safe as the traditional 6-hour observation period. No patient in either group experienced neurological deterioration, repeat imaging, admission, or neurosurgical intervention during the 7-day follow-up, a figure entirely consistent with the largest published series: 0.09 % in the Scandinavian Neurotrauma Committee cohort of >50 000 patients 16, 0.04 % in a 2021 meta-analysis of >70 000 cases17, and 0.1 % in recent UK National Health Service data incorporating NICE-guided early discharge.[18] These data from high-income settings have now been replicated for the first time in a South Asian tertiary centre, closing a critical evidence gap for low and middle-income countries where prolonged observation remains commonplace despite resource constraints.[6,7] Our findings align with a growing body of international evidence that the risk of clinically significant delayed intracranial hemorrhage in this selected population is extremely low (<0.5–1%) [2,6,7]

Emergency department revisit rates within 7 days were low (9.8 % early discharge vs 4.9 % observation; p=0.398) and exclusively driven by benign post-concussion symptoms, mirroring rates of 7–15 % reported in Canadian, Australian and European cohorts applying similar low-risk criteria (2,6). European surveys reveal that 30–70 % of patients with normal CT are still admitted or observed overnight, largely because of medico-legal anxiety rather than evidence (6).

Technological advancements have introduced biomarkers such as the S100B protein and newer MRI modalities to support diagnosis and reduce unnecessary CT imaging (3,10). These tools may help risk-stratify patients more accurately and potentially guide discharge decisions.[4,10] In a multicenter European study, Foks et al reported that practices for admitting or discharging mTBI patients differed across institutions, often depending on local protocols rather than standardized guidelines.[6] Bazarian et al. found that in the United States, despite normal CT findings, many patients were admitted or observed for extended periods, contributing to resource overuse.[7]

The only statistically significant difference we observed was higher anxiety/irritability at day 1 in the early-discharge group (19.5 % vs 4.9 %; p = 0.041) which was clinically important but entirely expected. Ponsford et al. demonstrated that inadequate discharge education is one of the strongest independent predictors of persistent post-concussion symptoms and psychological distress at 3 months.[9] Silverberg and Iverson similarly showed that early psychoeducation and reassurance reduce symptom reporting by 30-50 %. The rapid resolution of this difference by day 7 in our study, coupled with 100 % patient satisfaction in both arms, underscores that structured verbal and written instructions which we provided uniformly are highly effective at mitigating this transient effect.

Some studies advocate for early discharge in patients with a GCS of 15 and no risk factors (e.g., vomiting, seizures, anticoagulation use, skull fracture signs), as the risk of delayed deterioration in this group is very low.[2,7] However, others suggest a short period of observation (e.g., six hours) may help identify late complications such as intracranial hemorrhage, especially in patients who may not be closely monitored at home.

Special populations, such as the elderly, require particular consideration due to polypharmacy and comorbidities, which may increase the risk of adverse outcomes even in the setting of mild injury.[13] Youth and school-aged populations may also need individualized care plans involving gradual reintegration to cognitive activities.[1] Updated clinical guidelines recommend not only acute symptom management but also addressing mental health, cognitive sequelae, and patient education to reduce long-term morbidity.[14] Rehabilitation strategies should include early patient education and, where indicated, psychological support to reduce persistent post-concussion symptoms.[11]

Overall, while the current evidence supports individualized assessment and safe discharge in selected cases, further research is necessary to validate early discharge protocols compared to observation strategies, particularly in resource-constrained tertiary care settings.

LIMITATIONS

This study was conducted at a single tertiary centre, which may limit the generalizability of the findings to other settings with different management protocols. Also, the follow up time was short which may miss long term complications in the patients.

CONCLUSION

Early discharge in mTBI patients with normal CT scans is as safe as a 6-hour observation period, with no significant differences in return visits, post-concussion symptoms, or neurological deterioration. Early discharge could be implemented as a standard practice to improve ED efficiency and patient satisfaction, particularly in resource-limited settings. Further multicenter trials with larger sample sizes are needed to confirm these findings.

REFERENCES

1. Lee LK. Controversies in the Sequelae of Pediatric Mild Traumatic Brain Injury. Pediatric Emergency Care. 2007;23(8):580–3.

2. Vos PE, Vecsei L, Traubner P, et al. Mild traumatic brain injury. European Journal of Neurology. 2012;19(2):191–8.

3. Zetterberg H, Blennow K, Smith DH. Biomarkers of mild traumatic brain injury in cerebrospinal fluid and blood. Nat Rev Neurol. 2013;9(4):201–10.

4. Parker TD, Griffin C. Mild Traumatic Brain Injury including Concussion. Cambridge University Press; 2023.

5. Nagumo MM, Da Silva DV, De Amorim RLO. Mild Traumatic Brain Injury and Postconcussion Syndrome. In: Springer; 2018. p. 69–73.

6. Foks KA, Cnossen MC, Van Der Naalt J, et al. Management of Mild Traumatic Brain Injury at the Emergency Department and Hospital Admission in Europe: A Survey of 71 Neurotrauma Centers Participating in the CENTER-TBI Study. J Neurotrauma. 2017;34(17):2529–35.

7. Bazarian JJ, Flesher W, Mcclung J, et al. Emergency department management of mild traumatic brain injury in the USA. Emerg Med J. 2005;22(7):473–7.

8. Esselman PC, Uomoto JM. Classification of the spectrum of mild traumatic brain injury. Brain Injury. 1995;9(4):417–24.

9. Ponsford J, Bosch M, Chau M, et al. Factors associated with persistent post-concussion symptoms following mild traumatic brain injury in adults. J Rehabil Med. 2019;51(1):32–9.

10. Oris C, Kahouadji S, Durif J, et al. S100B, Actor and Biomarker of Mild Traumatic Brain Injury. Int J Mol Sci. 2023;24(7):6602.

11. Ponsford J. Rehabilitation interventions after mild head injury. CurrOpin Neurol. 2005;18(6):692–7.

12. Gioia GA. Medical-School Partnership in Guiding Return to School Following Mild Traumatic Brain Injury in Youth. J Child Neurol. 2014;31(1):93–108.

13. Papa L, Braga CF, Mendes ME. Mild Traumatic Brain Injury Among the Geriatric Population. CurrTranslGeriatr Exp Gerontol Rep. 2012;1(3):135–42.

14. Marshall S, Bayley M, McCullagh S, et al. Updated clinical practice guidelines for concussion/mild traumatic brain injury and persistent symptoms. Brain Injury. 2015;29(6):688–700.

15. Stein SC, Narayan RK, Wilberger JE, et al. Classification of Head injury. In: Neurotrauma. New york: Mcgraw- Hall; 1996:31-41

16. Undén J, Ingebrigtsen T, Romner B; Scandinavian Neurotrauma Committee. Scandinavian guidelines for initial management of minimal, mild and moderate head injuries in adults: an evidence and consensus-based update. BMC Med. 2013;11:50.

17. Chen H, et al. Risk of delayed intracranial haemorrhage after an initial negative CT in patients on anticoagulation/ antiplatelet therapy with mild traumatic brain injury: a systematic review and meta-analysis. World J Emerg Surg. 2021;16:59.

18. Marincowitz C, et al. Implementation of the NICE Head Injury Guidelines is associated with reduced rates of CT scanning and admission. Emerg Med J. 2022;39:287-93.

Effect of Short-term Preoperative Dutasteride on Bleeding after Transurethral Resection of the Prostate

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ABSTRACT

Introduction: Transurethral resection of the prostate is the gold-standard surgical treatment for moderate to severe lower urinary tract symptoms due to benign prostatic hyperplasia unresponsive to medical therapy. Bleeding is the most frequent postoperative complication, leading to clot retention, prolonged hospitalization, and increased morbidity. Dutasteride, a 5-alpha-reductase inhibitor, reduces dihydrotestosterone levels, suppresses Vascular Endothelial Growth Factor expression, and decreases prostate vascularity, thereby potentially reducing perioperative bleeding. **Aims:** To evaluate the effect of 2-weeks preoperative dutasteride therapy on bleeding during TURP. **Methods:** This prospective interventional study was conducted at Nepalgunj Medical College from September to November 2025. Fifty patients were divided into two groups: Group A received dutasteride 0.5 mg/day for two weeks before Transurethral resection of the prostate, and Group B underwent surgery without dutasteride. All patients underwent standard preoperative evaluations and surgery was performed using a uniform technique by blinded surgeons. Postoperative outcomes were assessed based on hemoglobin and hematocrit changes, clot retention, and hospital stay. **Results:** Baseline characteristics were similar between groups. Group A showed significantly less postoperative drop in Hemoglobin (0.66 ± 0.55 g/dl vs 1.12 ± 0.72 g/dl; P=0.016) and Hematocrit(4.53 ± 3.09% vs 8.75 ± 2.02%; P=0.02). No patients in Group A developed clot retention, whereas two cases in Group B needed clot evacuation. Hospital stay was significantly shorter in Group A (2.28 ± 0.54 vs 3.08 ± 0.91 days; P=0.000). **Conclusion:** Short-term preoperative dutasteride therapy significantly reduces bleeding during Transurethral resection of the prostate and shortens hospital stay.

***Keywords*:** 5-Alpha-reductase inhibitor, Benign Prostatic hyperplasia, Dutasteride,Transurethral Resection Of Prostate, Vascular endothelial Growth Factor

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INTRODUCTION

Transurethral resection of the prostate (TURP) is the gold standard surgical treatment for patients who have moderate to severe Lower Urinary Tract Symptoms (LUTS) due to benign prostatic hyperplasia (BPH) not responding to pharmacological treatments.[1-3] Post-operative bleeding is one of the prevalent and severe complications of TURP. It increases the risk of post-operative clot retention, longer hospital stay, blood transfusion, clot evacuation and sepsis which increases the morbidity and mortality.[4,5] Finasteride and dutasteride are 5-ARIs administered to inhibit the conversion of testosterone (T) to dihydrotestosterone (DHT). Apart from the daily use as pharmacological treatments for BPH, they are also used to manage hematuria associated with the disease. This effect is achieved by reducing the growth of prostatic tissue and suppressing the androgen-controlled vascular endothelial growth factor (VEGF), which results in reduced angiogenesis and less prostatic bleeding.[6] The available 5-ARIs, namely finasteride and dutasteride, suppress DHT levels, prostate vascularity, and growth. Finasteride inhibits only type II 5-AR, whereas dutasteride blocks both types I and II 5-ARs.[7] Several studies have shown that pretreatment with finasteride has resulted in decreased blood loss during TURP.[8-10] However study on effect of Dutasteride are limited. This study was planned to compare the effect of the short-term pretreatment (2 weeks) with Dutasteride on bleeding after TURP.

METHODS

This prospective interventional study was carried out in the department of urology, Nepalgunj Medical College during the period of September to November 2025. This study was approved by the Ethical Review Committee, NGMC. Written informed consent was taken from every case. Each study subject was evaluated by history, physical examination and investigations. History was taken regarding lower urinary tract syndrome (LUTS), urinary tract infection, bronchial asthma, diabetes mellitus, hypertension, ischemic heart disease, any hematological disorders and any prostatic or urethral surgery. Physical examination included general examination, examination of the renal region and other parts of the genitourinary system. All patients were investigated properly by-Urine for R/M/E and C/S to identify urinary tract infection (UTI); Serum prostate-specific antigen (S. PSA); S. Creatinine; S. Electrolyte; Complete blood count (CBC) was done to see Hemoglobin (Hb) levels, Hematocrit (Hct) levels, Platelet counts, etc; Blood for Bleeding time (BT), Clotting Time (CT), Prothrombin time (PT) and Activated partial thromboplastin time (APTT) ; Ultrasonogram of KUB with post void residue (PVR) was done to evaluate the size, volume and echo-texture of prostate and any hydronephrotic change or hydroureter or any other pathology in the KUB region; Uroflowmetry to see urinary flow rate.

Total Fifty (50) patients were allocated in study. Among them, group A (TURP with dutasteride) consisted of 25 patients who received preoperative dutasteride 0.5 mg/day for 2 weeks before TURP and group B (TURP without dutasteride) consisted of 25 patients who directly underwent TURP.

Monopolar TURP procedure was conducted following a standardized surgical protocol under spinal anesthesia with the patient in the dorsal lithotomy position. After performing urethrocystoscopy to visualize anatomical landmarks and assess prostate enlargement, urethral dilation was carried out using an Otis urethrotome up to 28 Fr. The resection was done using a continuous-flow 26 Fr resectoscope with 1.5% glycine irrigation. Resection began at either the 11 o'clock position for the right lobe or the 1 o'clock position for the left lobe, progressing downward toward the 6 o'clock position. Once one lobe was completed, the other was resected in the same manner. If the median lobe was enlarged, it was addressed first to enhance irrigation flow. Resection depth extended to the prostatic capsule. Systemic hemostasis was then achieved, beginning at the bladder neck, especially around the mucosal margins at the 5 and 7 o'clock positions, followed by re-evaluation of each lobe.

After resection, all prostatic tissue chips were removed using an Ellik evacuator, and the prostate volume was measured and sent for histopathological evaluation. The procedure was performed by urologists, and surgeons remained blinded to the patients' preoperative dutasteride therapy status to eliminate bias. Postoperatively, a 24 Fr tri-channel Foley catheter with a 40 ml balloon was inserted, and continuous bladder irrigation with 0.9% normal saline was maintained until hematuria resolved. During the postoperative period, the two groups were assessed and compared based on clot retention and length of hospital stay. Patients were discharged once they were able to void clear urine. Blood loss was evaluated by measuring reductions in hemoglobin (Hb) and hematocrit (Hct) levels, with comparisons made between preoperative values and those obtained 24 hours after the surgery.

Statistical Analysis

Data collected were entered into and analyzed using SPSS version 24. Mean, standard deviation and Median were calculated. P-values were generated using Mann Whitney U test and Independent T test. For all statistical tests, P-value of less than 0.05 was considered significant.

RESULTS

Patient's age ranged from 55 to 85 (69.76±9.46) years in group A and 56 to 86 years (70.2±6.61 years) in group B. Mean prostate volume (55.68 ± 10.97 grams vs 56.72 ± 11.04 grams, P=0.859), mean preoperative IPSS score (25.88 ± 5.27 vs 24.56 ± 4.06, P=0.213), mean post-void residual urine (49.68 ± 62.05 ml vs 39.6 ± 62.7 ml, P=0.158), mean PSA (2.62 ± 1.18 ng/ml vs 2.43 ± 1.19 ng/ml, P=0.836), mean preoperative Q-max (6.67 ± 2.67 ml/s vs 7.27 ± 2.86 ml/s, P=0.512) were similar in between group A and group B. (Table I)

Parameters	Group A(TURP with Dutasteride)	Group B (TURP without Dutasteride)	P-value
Mean Age (Range) (Years)	69.76±9.46 (50-85)	70.2±6.61 (56-86)	0.859
Mean Prostate Volume (Range) (Grams)	55.68 ± 10.97 (40-80)	56.72 ± 11.04 (42-80)	0.711
Mean Post void residual Urine (PVRU in ml) (Range)	49.68 ± 62.05 (0-200)	39.6 ± 62.7 (0-185)	0.158
Mean PSA (Range) (ng/ml)	2.62 ± 1.18 (0.5-4)	2.43 ± 1.19 (0.58-4)	0.836
Mean Preoperative IPSS score (Range)	25.88 ± 5.27 (15-33)	24.56 ± 4.06 (18-32)	0.213
Mean Preoperative Qmax (Range) (ml/sec)	6.67 ± 2.67 (2.5-11)	7.27 ± 2.86 (2-12)	0.512

Table I: Preoperative Patient's characteristics

The intraoperative duration ranged from 35 to 55 (45.16 ± 27) mins in group A and from 36-57 (46.6 ± 26.2) mins in group B

and amount of irrigation fluid used ranged from 12-25 liters (18.8 ± 5.97) in group A and 12-28 liters (20.48 ± 5.22)

(P=0.988) in group B which showed no statistical significance and were comparable in both groups. The mean preoperative hemoglobin was 13.96±1.83 gm/dl and 13.89±1.22 gm/dl in group A and group B and postoperative hemoglobin was 13.232±1.24 gm/dl and 12.844± 1.67 gm/dl simultaneously. The mean drop in hemoglobin was 0.66 ± 0.55 gm/dl in group A and 1.12 ± 0.72 gm/dl in group B. The drop in hemoglobin level was less in group A and the change was statistically significant (P=0.016).

In the other hand, preoperative Hct was 42.50% ± 3.11% and 42.04% ± 3.24%, respectively in group A and group B. On the other hand, postoperative Hct was 37.97% ± 2.02 % and 33.29% ± 2.12% respectively between the groups. Mean difference of pre-postoperative change of Hct was 4.53 ± 3.09% and 8.75± 2.02% respectively in between group A and group B which was statistically significant(P=0.02). Two patients in group B needed clot evacuation retention whereas no patients in group A developed clot retention which was not statistically significant. No patient in both groups needed blood transfusion.

The mean hospital stay was comparatively shorter in group A (2.28 ± 0.54 days vs 3.08 ± 0.91 days, P=0.000) and was statistically significant as shown in Table II.

Parameter	Group A	Group B	P value
Drop in Hemoglobin (gm/dl)	0.66 ± 0.55	1.12 ± 0.72	0.016
Drop in Hematocrit(Hct)	4.53 ± 3.09%	8.75± 2.02%	0.02
Clot evacuation	0	2	0.651
Hospital stay (in days)	2.28 ± 0.54 (2-4)	3.08 ± 0.91 (2-6)	0.000

Table II: Operative outcomes

DISCUSSION

TURP has long been regarded as the standard surgical approach for managing symptomatic BPH not responding to medical management. Among the complications associated with this procedure, bleeding is the most frequent, occurring both during surgery and in the postoperative phase. In severe cases, excessive bleeding can obstruct urination because of clot retention within the urinary tract.

BPH itself involves an increased growth of stromal and acinar cells surrounding the urethra, a process that is supported by enhanced glandular angiogenesis. This increased blood vessel formation contributes to the substantial bleeding that may occur during and after TURP. Finasteride, a commonly prescribed BPH medication, is a 5-alpha-reductase inhibitor that blocks the conversion of testosterone to DHT, thereby reducing the activation of growth factors that promote angiogenesis.

Studies have shown that administering finasteride before surgery can help minimize bleeding complications.[11,12] It achieves this by lowering VEGF expression and reducing the microvessel density beneath the urethra within the prostate, ultimately improving surgical outcomes. Kim et al in their study reported reduced surgical bleeding and hospitalization days after TURP with preoperative treatment with dutasteride for two weeks before TURP.[13] In our study the mean drop in hemoglobin was 0.66 ± 0.55 gm/dl in group A and 1.12 ± 0.72 gm/dl in group B. The drop in hemoglobin was less in group A and the change was statistically significant (P=0.016).

Similarly, the Mean difference of pre-postoperative change of Hct was 4.53 ± 3.09% and 8.75± 2.02% respectively in between group A and group B which was statistically significant(P=0.02). Two patients in group B needed clot evacuation due to clot retention whereas no patients in group A developed clot retention which was not statistically significant.

The mean hospital stay was comparatively shorter in group A (2.28 ± 0.54 days vs 3.08 ± 0.91 days, P=0.000) and was statistically significant. In a study by Rahman et al they reported Preoperative dutasteride therapy reduces blood loss related to TURP in patients with BPH.[14] Martov et al found a significant reduction in blood loss in patients by using dutasteride for at least one month before TURP compared to the control group.[15] Kravchick demonstrated that 6 weeks of treatment with dutasteride reduced prostatic vascularity, especially in the periurethral area.[16]

In this study, preoperative dutasteride reduced blood loss in terms of pre-postoperative change of hemoglobin and hematocrit levels in the dutasteride group. There was no significant difference in clot retention and no blood transfusion was needed in both groups. Total hospital stay was shorter in dutasteride group.

LIMITATION

It was a single center study with relatively small sample size. All complications of TURP were not included in the study.

CONCLUSION

Short-term preoperative dutasteride therapy significantly reduces bleeding during Transurethral resection of the prostate and shortens hospital stay.

REFERENCES

1. Rocco B, Albo G, Ferreira RC, Spinelli M, Cozzi G, Dell'orto P, Patel V, Rocco F. Recent advances in the surgical treatment of benign prostatic hyperplasia. Ther Adv Urol. 2011 Dec;3(6):263-72.

2. Tanguay S., Awde M., Brock G., Casey R., Kozak J., Lee J., et al. Diagnosis and management of benign prostatic hyperplasia in primary care. J Can Urol Assoc. 2009;3:S92

3. Nickel J.C., Méndez-Probst C.E., Whelan T.F., Paterson R.F., Razvi H. Update: Guidelines for the management of benign prostatic hyperplasia. J Can Urol Assoc. 2010;4: 310–6.

4. Lynch M, Sriprasad S, Subramonian K, Thompson P. Postoperative haemorrhage following transurethral resection of the prostate (TURP) and photoselective vaporisation of the prostate (PVP). Ann R Coll Surg Engl. 2010 Oct;92(7):555-8.

5. Akan S, Ediz C, Özer E, Pehlivanoğlu M, Tavukçu HH, Kıymaz K. Efficacy and Safety of Monopolar Transurethral Resection of the Prostate on Bleeding Control in the Treatment of Benign Prostatic Obstruction: Is It Still a Good Option in Developing Countries?. Bull Urooncol. 2020 Aug 24;19(3):157-61.

6. Bartsch G, Rittmaster RS, Klocker H. Dihydrotestosterone and the concept of 5alpha-reductase inhibition in human benign prostatic hyperplasia. Eur Urol. 2000 Apr;37(4):367-80.

7. Nickel JC. Comparison of clinical trials with finasteride and dutasteride. Rev Urol. 2004;6(Suppl 9):31-9.

8. Uttam Kumar S, Dorairajan LN, Badhe BA, Manikandan R, Singh S. Effect of preoperative finasteride on perioperative blood loss during transurethral resection of the prostate and on microvessel density in patients with benign prostatic hyperplasia: An open label randomized controlled trial. Urol Ann. 2021;13(3):199-204

9. Busetto GM, Del Giudice F, Maggi M, Antonini G, D'Agostino D, Romagnoli D, Del Rosso A, Giampaoli M, Corsi P, Palmer K, Ferro M, Lucarelli G, Terracciano D, et al. Surgical blood loss during holmium laser enucleation of the prostate (HoLEP) is not affected by short-term pretreatment with dutasteride: a double-blind placebo-controlled trial on prostate vascularity. Aging (Albany NY). 2020; 12:4337-47.

10. Joseph IB, Nelson JB, Denmeade SR, Isaacs JT. Androgens regulate vascular endothelial growth factor content in normal and malignant prostatic tissue. Clin Cancer Res. 1997 ;3(12):2507-11.

11. Dutt UK, Kumar S, Dorairajan LN, Badhe BA, Manikandan R, Singh S. Effect of preoperative finasteride on perioperative blood loss during transurethral resection of the prostate and on microvessel density in patients with benign prostatic hyperplasia: An open label randomized controlled trial. Urol Ann. 2021 Jul-Sep;13(3):199-204.

12. Puchner PJ, Miller MI. The effects of finasteride on hematuria associated with benign prostatic hyperplasia: a preliminary report. J Urol 1995;154: 1779–82

13. Kim KS, Jeong WS, Park SY, Kim YT, Moon HS. The effect of two weeks of treatment with dutasteride on bleeding after transurethral resection of the prostate. World J Mens Health. 2015; 33(1):14-9.

14. Rahaman, M. M., Rahman, M. H., Hossain, M., Islam, M. S., & Rahman, M. S. (2020). The Effect of Dutasteride in Peri-Operative Blood Loss During Transurethral Resection of Prostate (Turp). Bangladesh Journal of Urology.2020: 22(2):177–181.

15. Martov AG, Ergakov DV. The experience in dutasteride use before transurethral prostatic resection for large adenoma. Urologiia . 2020; 1(4):46-8.

16. Kravchick S, Cytron S, Mamonov A, Peled R, Linov L. Effect of short-term dutasteride therapy on prostate vascularity in patients with benign prostatic hyperplasia: a pilot study. Urology. 2009;73(6):1274- 8.

ORIGINAL ARTICLE

Prevalence of Urine for Microalbumin in Type 2 Diabetes: A Descriptive Cross-Sectional Study in A Tertiary Care Hospital

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ABSTRACT

Introduction: Microalbuminuria is an early clinical indicator of diabetic nephropathy and a predictor of cardiovascular morbidity and mortality in patients with type 2 diabetes mellitus (T2DM). Early detection is vital for timely management to prevent progression to overt nephropathy and renal failure. **Aims:** To determine the prevalence of microalbuminuria and its association with glycemic control, blood pressure, and body mass index with type 2 diabetes mellitus. **Methods:** A descriptive cross-sectional study was conducted at Nepalgunj Medical College, from October 2024 to April 2025. Two hundred patients with confirmed type 2 diabetes mellitus of at least one-year duration were enrolled using convenience sampling. Patients with urinary tract infections, pregnancy, or non-diabetic renal diseases were excluded. Demographic data, clinical parameters, and laboratory results including fasting blood sugar, HbA1c, lipid profile, and urine microalbumin levels were recorded. Statistical analysis was performed using SPSS version 25. Student's t-test and Chi-square test were applied, and p-value <0.05 was considered statistically significant. **Results:** Among 200 participants, 30 (15%) had microalbuminuria. The mean age of participants with microalbuminuria (65.07 ± 15.05 years) was higher than those without (52.95 ± 13.34 years). Microalbuminuria was more prevalent among females (9.5%) compared to males (5.5%). Participants with microalbuminuria had significantly higher HbA1c (8.30 ± 1.57%) and mean random blood sugar levels was (238.23 ± 25.607) . Individual without microalbuminuria had HbA1c (7.22 ± 1.065%) and mean random blood sugar level was (218.15 ± 46.60). The prevalence was greater among obese individuals and those with stage 2 hypertension. Serum cholesterol and triglyceride differences were not statistically significant. **Conclusion:** The prevalence of microalbuminuria among patients with type 2 diabetes was 15%. Poor glycemic control, hypertension, and obesity were significantly associated with its occurrence.

Keywords: Albuminuria, Diabetes mellitus type 2, Glycemic control, Hypertension

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INTRODUCTION

Diabetes is a significant global metabolic disorder.[1] Type 2 diabetes mellitus have been a significant global public health issue.[2] In 2024, the International Diabetes Federation estimated that approximately 589 million adults (aged 20–79) worldwide are living with diabetes.[3] Among diabetic patients, 20-40% develop diabetic nephropathy, and 10-20% of them succumb to kidney failure.[4-5] The earliest clinical sign of diabetic nephropathy is an increase in urinary protein excretion.[6-9] Microalbuminuria (MA) is the earliest detectable clinical sign of diabetic nephropathy.[10-11] The urine dipstick is a relatively insensitive indicator of proteinuria, as it does not show a positive result until protein excretion surpasses 300-500 mg per day.[12] Elevated urinary albumin levels may indicate broader vascular damage.[13] Microalbuminuria is linked with high blood pressure, dyslipidemia, inflammation, and endothelial dysfunction.[14-17] Microalbuminuria is regarded as an early indicator of diabetic nephropathy.[18] Bruno et al conducted a seven-year study involving 1,253 patients with type 2 diabetes, revealing that 3.7% of them progressed to overt nephropathy annually. Additionally, the presence of microalbuminuria was associat-

ed with a 42% higher risk compared to normoalbuminuria.[19] Microalbuminuria is recognized as a predictor of cardiovascular disease in both diabetic and non-diabetic individuals.[20-21] Recent data from the World Health Organization (WHO) predict a global rise in diabetes prevalence, with developing countries being particularly affected.[22] At present, South Asian countries have the highest number of individuals with diabetes globally.[22-2] In type 2 diabetes mellitus, albuminuria may result from factors unrelated to diabetes.[24-25] Microalbuminuria helps identify patients who require more aggressive management.[26]

METHODS

It is a cross - sectional study. This study was conducted in Nepalgunj Medical College located at Kohalpur, Banke, Nepal. This institution was selected due to their high patient volume and diverse patient demographies, making them representative of the western population of the Nepal. This institution provides a range of services, including outpatient clinics, specialized diabetic care units, and diagnostic laboratories, ensuring a thorough evaluation of patients. The study was conducted over 6 months from 2024 October to 2025 April.

SAMPLE SIZE

From the study done by M. Afkhami-Ardekani, M. Modarresi in 2005 , the overall prevalence of microalbuminuria was 14.2%27. Using the formula given below sample size becomes 187.21, taking 200 final sample size considering 5% of non-responsive errors. Inclusion Criteria includes individual who are diagnosed with T2DM for ≥1 year known through history taking, age >18 years, on treatment or not on treatment for DM and the individual who provided informed consent. Whereas exclusion criteria includes individual who are diagnosed with UTI, Acute illness, Known non-diabetic kidney disease, individual who underwent recent surgery, and individual who are unable to provide consent. Patients with suspected hypertensive nephropathy or diabetic with hypertensive renal disease were excluded based on clinical history, ultrasound and previous medical records

Sources of Data : **Patient Medical Records:** Review of patients' medical records to gather detailed information on diabetes diagnosis, duration, treatment plans, and any other relevant medical history. **Interviews**: Conducting structured interviews with patients to obtain demographic information. Laboratory **Tests**: Collection and analysis of blood and urine samples to measure blood glucose levels, HbAlc, serum creatinine, and urine albumin levels. Microalbuminuria detection was performed using standardized laboratory techniques. Performing physical examinations to assess body mass index (BMI), blood pressure, and other vital health indicators relevant to diabetes and kidney function.

Statistical analysis: Data were analyzed with SPSS independent t-test and chi-square test were used to compare means and proportions.

RESULTS

A total of 200 patients with type 2 diabetes mellitus were included in the study. Among them, 30 (15%) had microalbuminuria, while 170 (85%) did not have microalbuminuria. The mean age of participants with microalbuminuria was 65.07 ± 15.05 years, which was higher compared to those without microalbuminuria (52.95 ± 13.34 years). The prevalence of microalbuminuria was greater among females (19, 9.5%) compared to males (11, 5.5%).

Variable	Without microalbuminurian (%) / Mean ± SD	With microalbuminurian (%) / Mean ± SD
Gender	Male: 106 (53%), Female: 64 (32%)	Male: 11 (5.5%), Female: 19 (9.5%)
Age group (years)	170 (85%) / 52.95 ± 13.34	30 (15%) / 65.07 ± 15.05
Duration of diabetes (years)	8.32 ± 4.31	13.53 ± 6.89
Systolic BP (mmHg)	128.58 ± 20.90	138.67 ± 13.32
Diastolic BP (mmHg)	85.70 ± 12.64	87.67 ± 14.31
Body Mass Index (kg/m²)	21.46 ± 2.45	23.40 ± 2.77
Smoking habits	Yes 74(37%) NO 96(48%)	Yes 11(5.5%) NO 19(9.5%)

Table I: Comparison of study participants with and without microalbuminuria among different study variables

	Without Microalbuminuria N=170, 85% Mean ± SD	With Microalbuminuria N=30,15% Mean ± SD	T value	P value
HbA1C	7.22±1.065	8.30±1.579	-	<0.001
Microalbumin (mg/dl)	12.92±7.128	51.43±16.164	-12.83	<0.001
Fasting Blood Sugar (mmol/l)	182.31±40.54	183.10±30.33	-0.124	0.919
Random Blood Sugar (mmol/l)	218.15±46.600	238.23±25.607	-3.413	0.001
Cholesterol (mmol/l)	195.04±44.347	206.80±43.208	-1.345	0.180
Triglyceride (mmol/l)	240.45±50.718	242.50±56.775	-0.200	0.842

Table II: Comparison of laboratory parameters among patients with and without microalbuminuria (n = 200)

Patients with microalbuminuria had significantly higher mean HbA1c and random blood sugar levels ($p < 0.001$), indicating poor glycemic control. Differences in cholesterol and triglyceride levels were not statistically significant. Among participants

with microalbuminuria, 60.0% had HbA1c > 8%, compared to 15.3% among those without microalbuminuria. The difference was statistically significant ($\chi^2 = 12.83$, $p < 0.001$), suggesting a strong association between poor glycemic control and microalbuminuria.

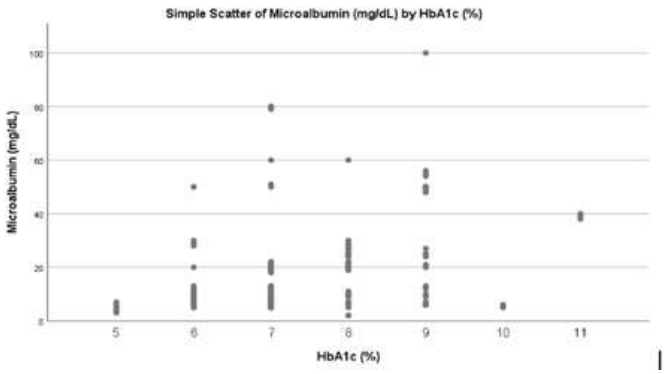

Figure 1: Scatter plot showing correlation between HbA1c and microalbuminuria

HbA1c Category	Without microalbuminuria n (%)	With microalbuminuria n (%)
<7%	57 (33.5%)	5 (16.7%)
7–8%	87 51.2%)	7 (23.3%)
>8%	26 (15.3%)	18 (60.0%)

Table III: Relation between HbA1c category and microalbuminuria

A significant association was observed between HbA1c level and microalbuminuria ($\chi^2 = 12.83$, $p < 0.001$). Patients with HbA1c > 8% had the highest prevalence of microalbuminuria (60%).

Blood Pressure Category	Without microalbuminuria n (%)	With microalbuminuria n (%)
Normal	38 (22.4%)	1 (3.3%)
Elevated	16 (9.4%)	4 (13.3%)
Stage 1 Hypertension	19 (11.2%)	7 (23.3%)
Stage 2 Hypertension	93 (54.7%)	18 (60.0%)

Table IV: Relation between blood pressure category and microalbuminuria

Microalbuminuria was more prevalent among patients with Stage 1 (23.3%) and Stage 2 hypertension (60%) compared to those with normal blood pressure (3.3%), indicating a significant relationship.

BMI Category	Without microalbuminuria n (%)	With microalbuminuria n (%)
Underweight	17 (10.0%)	0 (0.0%)
Normal	106 (62.4%)	17 (56.7%)
Overweight	12 (7.1%)	0 (0.0%)
Obese I	35 (20.6%)	13 (43.3%)

Table V: Relation between BMI category and microalbuminuria

A higher proportion of obese individuals (43.3%) had microalbuminuria compared to normal-weight patients (56.7%), while no microalbuminuria was observed among underweight or overweight participants.

DISCUSSION

The association of clinical and biochemical parameters to determine the prevalence of microalbuminuria was the aim of the present study. About 15% of participants were found to have microalbuminuria, which is similar to findings in studies done in similar settings. Diabetic nephropathy, generalized endothelial dysfunction, and renal and cardiovascular risk among diabetic individuals are early detected with the help of microalbuminuria.[6,7,8]

In a study finding by Lutale et al, the reported prevalence was 43% among African diabetic patients where whereas 15% is observed in our study, which is slightly lower.[11] Prevalence rates ranging from 10% to 30% are reported in studies from developed countries, depending on the duration of diabetes, glycemic control, and ethnicity.[16,17,18] The difference in rate in our study could be due to differences in population characteristics, accessibility to healthcare facilities, duration and other factors.

Our study observed a significant relation between a raised level of HbA1c and the presence of microalbuminuria. The highest prevalence of microalbuminuria (60%) was found in Patients with HbA1c > 8%. This finding is similar to results from previous studies showing that poor glycemic control enhances damage to renal microvessels, which eventually increases urinary albumin excretion.[13,14,15] Nephropathy progression is contributed to by oxidative stress, non-enzymatic glycation of proteins and glomerular hyperfiltration, which are induced by hyperglycemia.[14]

In our study, the prevalence of microalbuminuria was higher among hypertensive patients, mainly among those with stage 2 hypertension, which was about 60%. This is in accordance with previous findings that increased blood pressure is an important predictor for renal damage and microalbuminuria among the population with diabetes.[8,10,18] Association of Body mass index with microalbuminuria was also shown in our study. There are higher rates of obesity (43.3%) who had microalbuminuria compared to normal-weight or underweight individuals. Sys-

temic inflammation, resistance to insulin, and hyperfiltration are contributed to by obesity, which may enhance glomerular injury and albuminuria.[19,20,21] Intraglomerular pressure is increased by persistent elevated blood pressure, which eventually leads to thickening of the glomerular basement membrane and leakage of albumin.[26] The importance of control of BP and in delay in onset and progression of nephropathy is emphasized by these findings.

In accordance with findings reported in the study by Sharma et al in Nepal, our study also has reported obesity, hypertension and diabetes in the Nepalese population as a high prevalence of metabolic risk factors.[5] The growing public health burden and complications of type 2 diabetes in South Asia which strengthen by the findings, uniform with global estimates by the International Diabetes Federation[3] and projections by King et al.[22]

This study coincides with prior research because of significant correlation noted between microalbuminuria and HbA1c. Sustained High blood sugar uneven cholesterol levels can predict early kidney complications as shown by these studies.[12,13,25] Detecting microalbuminuria at an early stage provides a vital opportunity for prompt intervention to prevent the advancement towards severe kidney disease and end-stage renal failure.[24,25]

In summary, our results underscore the necessity of routine microalbuminuria screening for individuals with type 2 diabetes. Keeping track of blood sugar levels, blood pressure, and body weight should be fundamental aspects of diabetes care. Timely identification and management of these factors can significantly reduce the likelihood of kidney and cardiovascular problems.[7,8,26]

LIMITATIONS

As, this study was performed at a single tertiary care center with comparatively small size, which may constraint the accountability of the findings. The capability to establish causal interference is also limited by cross-sectional design. Moreover, confounding influences including specific dietary trends and medication compliance were not adjusted. Hence, larger, multicentric and longitudinal studies are suggested to confirm these outcomes and examine the extended impact of microalbuminuria among patients with type 2 diabetes.

CONCLUSION

In conclusion, our findings emphasize the requirement of routine microalbuminuria screening for subjects with type 2 diabetes. Monitoring blood sugar levels, blood pressure, and body weight should be core elements of diabetes management.

REFERENCES

1. Defronzo RA. Pathogenesis of type 2 diabetes. Diabetes Rev 1997;5:177.
2. King H, Aubert RE, Herman WH. Global burden of diabetes,1995–2025: prevalence, numerical estimates, and projections. Diabetes care. 1998;21(9):1414-31.
3. International Diabetes Federation. IDF Diabetes Atlas 11th Edition — Worldwide, 589 million adults (20 79) living with diabetes in 2024. Brussels: IDF; 2025. Diabetes Atlas+2OUP Academic+2.
4. Gu K, Cowie CC, Harris MI. Mortality in adults with and without diabetes in a national cohort of the US population, 1971–1993. Diabetes care. 1998;21(7):1138-45.
5. Sharma SK, Ghimire A, Radhakrishnan J, Thapa L, Shrestha NR, Paudel N, et al. Prevalence of hypertension, obesity, diabetes, and metabolic syndrome in Nepal. Int J Hypertens. 2011;2011.821971.
6. Mogensen CE. Prediction of clinical diabetic nephr- opathy in IDDM patients. Alternative to microalbuminuria Diabetes 1990;39:761.
7. Ruggeneti P, Remuzzi G. Nephropathy of type 2 dia- betes mellitus. J Am Soc Nephrol 1998;9:2157.
8. Ismail N, Becker B, Strzelczyk P, Ritz E. Renal disease and hypertension in non insulin dependent diabetes mellitus. Kidney Int 1999;55:1.
9. Russo LM, Bakris GL, Comper WD. Renal handling of albumin: A critical review of basic concepts and perspective. Am J Kidney Dis 2002;39:899.
10. Cordonnier D, Bayle F, Benhamou P, Milongo R, Zaoui P, Maynard C, et al. Future trends of management of renal failure in diabetics. Kidney Int Suppl. 1993;41:S8.
11. Lutale JJ, Thordarson H, Abbas ZG, Vetvik K. Microalbuminuria among type 1 and type 2 diabetic patients of African origin in Dar Es Salaam, Tanzania. BMC Nephrol. 2007 Jan 15;8:2.
12. Battisti WP, Palmisano J, Keane WE. Dyslipidemia in patients with type 2 diabetes: Relationships between lipids, kidney disease and cardiovascular disease. Clin Chem Lab Med 2003;41:1174-81.
13. MacIsaac RJ, Cooper ME. Microalbuminuria and diabetic cardiovascular disease. Current Atherosclerosis Reports. 2003;5(5):350-7.
14. Mogensen CE, Steffes MW, Deckert T. Functional and morphological renal manifestation in diabetes mel- litus. Diabetologia 1981;21:89-93.
15. Viberti GC, Keen H. The pattern of proteinuria in diabetes mellitus: Relevance of pathogenesis and pre- vention of diabetes nephropathy. Diabetes 1984;33:686-92.
16. Alzaid A. Microalbuminuria in patients with NIDDM: An overview. Diabetes Care 1996;19:79-89.
17. Parving HH, Gall MA, Skott P. Prevalence and causes of albuminuria non insulin dependent diabetic pa- tients. Kidney Int 1990;41:758-86.
18. Weir MR. CME Microalbuminuria in type 2 diabetics: An important, overlooked cardiovascular risk factor. J Clin Hypertens 2004;6:134-43.
19. Bruno G, Merletti F, Biggeri A, Bargero G, Ferrero S, Pagano G, Perin PC. Progression to overt nephropathy in type 2 diabetes: the Casale Monferrato Study. Diabetes care. 2003 Jul 1;26(7):2150-5.

20. Yudkin J, Forrest R, Jackson C. Microalbuminuria as predictor of vascular disease in non-diabetic subjects: Islington Diabetes Survey. The Lancet. 1988 Sep 3;332(8610):530-3.

21 Damsgaard EM, Frøland A, Jørgensen OD, Mogensen CE. Microalbuminuria as predictor of increased mortality in elderly people. British Medical Journal. 1990 Feb 3;300(6720):297-300.

22 King H, Aubert RE, Herman WH. Global burden of diabetes, 1995–2025: prevalence, numerical estimates, and projections. Diabetes care. 1998 Sep 1;21(9):1414-31.

23 Ramachandran A, Snehalatha C, Latha E, Vijay V, Viswanathan M. Rising prevalence of NIDDM in an urban population in India. Diabetologia. 1997 Jan;40:232-7.

24 Mogensen CE. Preventing end-stage renal disease. Diabetic Medicine. 1998 Dec;15(S4 4):S51-6.

25 Ritz E, Orth SR. Nephropathy in patients with type 2 diabetes mellitus. New England Journal of Medicine. 1999 Oct 7;341(15):1127-33.

26 Weir MR. Microalbuminuria in type 2 diabetics: an important, overlooked cardiovascular risk factor. The Journal of Clinical Hypertension. 2004 Mar;6(3):134-43.

27 Afkhami-Ardekani M, Modarresi M, Amirchaghmaghi E. Prevalence of microalbuminuria and its risk factors in type 2 diabetic patients. Indian J Nephrol. 2008;18(3):112-7. Available from: https://journals.lww.com/ijon/fulltext/2008/18030/prevalence_of_microalbuminuria_and_its_risk.4.aspx

Pattern of Dyslipidemia among Acute Coronary Syndrome (ACS) Patients at Nepalgunj Medical College: A Hospital Based Cross Sectional Study

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ABSTRACT

Introduction: Dyslipidemia is a major risk factor for acute coronary syndrome, with varying patterns across ethnicities and regions, such as higher triglyceride levels in South Asians. **Aims:** To describe the demographic and dyslipidemia patterns among acute coronary syndrome patients. **Methods:** This hospital-based cross-sectional study, conducted from 25 April to 24 October 2024, using convenience sampling to enroll 95 patients diagnosed with acute coronary syndrome (unstable angina), Non-ST-segment elevation myocardial infarction, ST-segment elevation myocardial infarction admitted to the Intensive Care Unit of our center. Data were collected using a structured and semi-structured proforma with demographic details, clinical findings, and laboratory results. Informed written consent was obtained from all participants. **Results:** Among the participants, 54 were male with majority from Nepalgunj (22.1%). Risk factor analysis revealed 64% of patients were smokers, 49% had hypertension, and 9% had type 2 diabetes mellitus. Only 5% of patients demonstrated dyslipidemia with isolated low High Density Lipoprotein Cholesterol. Non-ST-segment elevation myocardial infarction was the most common presentation. **Conclusion:** Isolated low High Density Lipoprotein Cholesterol was the predominant dyslipidemia pattern in statin-naïve acute coronary syndrome patients in western Nepal, despite low overall prevalence suggesting regional variations, possibly due to exclusion of patients on lipid-lowering drugs. These findings highlight the need for targeted lipid screening in early management.

Keywords: Acute Coronary Syndrome, Dyslipidemia, Echocardiography

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INTRODUCTION

Dyslipidemia is defined as the abnormal level of any form of lipid such as total cholesterol, triglyceride (TG), low density lipoprotein-cholesterol (LDL-C), high density lipoprotein-cholesterol (HDL-C), and very low density lipoprotein (VLDL). It remains a major modifiable risk factor for cardiovascular diseases (CVDs) globally.[1] These lipid imbalances can arise from genetic predispositions, poor dietary habits, physical inactivity, or environmental factors and is associated with high risk of cardiovascular diseases (CVDs) that contributes to approximately 4.4 million deaths annually, a significant burden in low and middle-income countries.[1] All forms of lipid can contribute to formation of atherosclerosis, being the potential risk factor for Acute Coronary Syndrome (ACS). ACS includes ST segment elevation Myocardial Infarction (STEMI), Non ST segment elevation Myocardial Infarction (NSTEMI) and Unstable Angina. The pattern of dyslipidemia has shown to be different among different ethnicity and geographical region e.g. South Asians tend to have more triglyceride levels and other forms like LDL-C. In South Asia, including Nepal, the burden of CVDs is rising alarmingly, accounting for nearly 27% of all deaths in the region.[2] Hypertriglyceridemia is mainly treated with drugs like fenofibrates whereas increased LDL-C is treated mainly with statins.[3] Despite the growing burden of CVDs in Nepal, data on dyslipidemia patterns in western Nepal are limited, leaving

a gap in understanding how local factors shape cardiovascular risk. This study seeks to address this gap by understanding the demographic characteristics and dyslipidemia patterns among ACS patients.

METHODS

This cross-sectional observational study was conducted at Nepalgunj Medical College, Nepal, starting from April 25 to October 24, 2024 and enrolled 95 patients using convenient sampling. A self-designed proforma was used to collect demographic (sex, age, address, religion, ethnicity) and clinical data (medical history, ECG, echocardiography, CKMB, Troponin I, and fasting serum lipid profile obtained the morning after ICU admission), with ACS diagnosis based on clinical presentation, ECG changes, and cardiac biomarkers. Inclusion criteria comprised patients with acute coronary syndrome (unstable angina, NSTEMI, STEMI) admitted to the ICU; exclusion criteria included stable ischemic heart disease, chronic coronary syndrome, Q waves or bundle branch block without prior ECG, statin use, and incomplete data. Ethical approval and patient consent were not mentioned. Sample size was calculated using the formula $N = Z^2P(1-P)/d^2$ with 95% confidence and 39% dyslipidemia prevalence.

Statistical Analysis

Categorical variables were reported as frequencies and percentages, numerical variables as means and standard deviations, analyzed using IBM SPSS version 26.0.

RESULTS

The study population consisted of 95 participants, with a gender distribution of 54 male and 41 female. All the demographic findings are listed below (Table I)

Characteristic	Percentage	Number (n)
Age (years), mean ± SD	58.3 ± 12.1	
Male	56.84%	54
Female	43.16%	41
Region		
Nepalgunj	22.10%	21
Banke	11.57%	11
Rukum	9.4%	9
Kailali	8.4%	8
Other (Bajhang, Jajarkot, Jumla, Kalikot)	48.42%	46
Religion		
Hindu	83.15%	79
Muslim	8.4%	8
Buddhist	4.2%	4
Christian	4.2%	4

Table I: Demographic details of Study population

Smoking was prevalent among 64% of participants while Hypertension (HTN) was observed in 49%. Type 2 diabetes mellitus (T2DM) was present in 9% and dyslipidemia in 5%. Only five percentage of participants had a previous diagnosis of acute coronary syndrome (ACS) or chronic coronary syndrome (CCS). (Table II)

Risk Factors	n (%)
Smoking	61 (64%)
Hypertension	47 (49%)
Type 2 DM	9 (9%)
Prior ACS/CCS	5 (5%)
Dyslipidemia (known)	0 (0%)

Table II: Risk factors for dyslipidemia

Overall prevalence of dyslipidemia was 5% (n=5), exclusively due to isolated low HDL-C (<40 mg/dL in males, <50 mg/dL in females). Mean total cholesterol (148.6 ± 28.4 mg/dL) and LDL-C (92.3 ± 22.1 mg/dL) were within normal limits, with only 2.1% and 1.1% exceeding high-risk thresholds, respectively. Triglycerides were mildly elevated on average (118.4 ± 36.7 mg/dL), with 3.2% meeting hypertriglyceridemia criteria (≥200 mg/dL).

Parameter	Mean ± SD (mg/dL)	Abnormal, n (%)
Total Cholesterol	148.6 ± 28.4	2 (2.1%) ≥240
LDL-C	92.3 ± 22.1	1 (1.1%) ≥160
HDL-C	38.4 ± 6.2	5 (5.3%) low
Triglycerides	118.4 ± 36.7	3 (3.2%) ≥200

Table III: Fasting Lipid Profile pattern of dyslipidemia

Electrocardiogram (ECG) findings revealed non-ST-elevation myocardial infarction (NSTEMI) as the most common diagnosis (39%, n=37), followed by inferior wall myocardial infarction (MI) (18%, n=17) and anterior wall MI (15%, n=14). Other ECG findings included unstable angina (4%, n=4), inferoposterior wall MI (3%, n=3), sinus rhythm with intraventricular conduction delay (IVCD) (4%, n=4), extensive anterior wall MI (3%, n=3). Echocardiography (ECHO) results showed a range of findings, with left ventricular diastolic dysfunction (LVDD) with left ventricular ejection fraction (LVEF) of 60% being the most frequent (8%, n=8), followed by normal ECHO with LVEF 65% (10%, n=10) and LVDD with LVEF 55% (5%, n=5). Other ECHO findings included various degrees of akinetic or hypokinetic left anterior descending (LAD) territory, inferior wall, or global hypokinesia, with LVEF ranging from 25% to 65%. (Table IV)

Diagnosis	n (%)
NSTEMI	37 (39%)
Inferior Wall MI	17 (18%)
Anterior Wall MI	14 (15%)
Unstable Angina	4 (4%)
Inferoposterior MI	3 (3%)
Others (IVCD, extensive AWMI, etc.)	20 (21%)

Table IV: ACS diagnostic distribution

DISCUSSION

The findings of this study at Nepalgunj Medical College reveal a significantly low prevalence of dyslipidemia (5%) among patients with acute coronary syndrome (ACS), which contrasts sharply with patterns observed in other South Asian and Nepalese studies. Dyslipidemia, characterized by abnormal levels of lipids such as total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), or triglycerides (TG), is a well-established risk factor for coronary artery disease (CAD) and ACS globally.[1,2] However, the low prevalence in this study prompts a closer examination of regional variations, methodological factors, and the unique characteristics of the study population in comparison to broader South Asian trends. In contrast to our findings, a study by Dhungana et al. at Nobel Medical College Teaching Hospital in Biratnagar, Nepal, reported a significantly higher prevalence of dyslipidemia (62%) among 105 ACS patients.[4] Notably, low HDL-C was observed in 60.8% of their cohort, aligning with the characteristic South Asian lipid profile of elevated triglycerides and low HDL-C, as described in the INTERHEART study.[2] The INTERHEART study, a global case-control study, identified dyslipidemia as the strongest contributor to acute myocardial infarction (AMI) in South Asians, with a population-attributable risk of 49.2%. This is consistent with other South Asian studies, such as one from Lahore, Pakistan, which reported dyslipidemia in 42.6% of 101 ACS patients, with a high prevalence of low HDL-C and elevated triglycerides.[5] These findings mark the typical atherogenic dyslipidemia profile in South Asians, which includes high triglycerides, low HDL-C, and a higher burden of small, dense LDL particles despite normal LDL-C levels.

The discrepancy between the 5% dyslipidemia prevalence in our study and the higher rates reported elsewhere may be attributed to several factors. First, our study excluded patients already on lipid-lowering therapies, such as statins, which likely reduced the number of participants with detectable lipid abnormalities. This exclusion criterion was not consistently applied in other studies, such as Dhungana et al, where only 27% of patients were on statins, potentially inflating the observed dyslipidemia prevalence.[4] Second, the timing of lipid profile measurement was within 24 hours of ACS onset which may have influenced our results. Lipid levels, particularly total cholesterol and LDL-C, can transiently decrease post-ACS due to an acute-phase response, as noted by Gore et al. In contrast, studies like the one from Lahore collected lipid profiles without specifying the exact timing relative to ACS onset, which may have depicted more baseline lipid abnormalities.[5]

Another notable aspect is the regional and demographic context of our study. The majority of participants were from Nepalgunj and surrounding areas, with a high prevalence of smoking (64%) and hypertension (49%), but a relatively low prevalence of type 2 diabetes mellitus (9%). This compared with other South Asian studies, such as one from Noakhali, Bangladesh, which reported dyslipidemia in 73.5% of ACS patients alongside higher rates of diabetes (37.3%) and smoking (38.2%).[7] The low diabetes prevalence in our study may partly explain the lower dyslipidemia rates, as diabetes is strongly associated with atherogenic dyslipidemia, characterized by high triglycerides and low HDL-C.[8] Additionally, the predominantly Hindu population (79%) and rural representation in our study may reflect lifestyle or dietary patterns; such as lower consumption of calorie-rich diets that differ from urban South Asian populations, where dyslipidemia is more prevalent due to sedentary lifestyles and high-carbohydrate diets.

Compared to broader South Asian trends, our study's findings challenge the existing belief that dyslipidemia is a dominant risk factor for ACS in all Nepalese populations. For instance, a study by Pokharel et al on Nepalese patients with type 2 diabetes reported mixed dyslipidemia (high TG, high LDL-C, low HDL-C) in 88.1% of cases, highlighting the synergistic effect of diabetes and dyslipidemia in cardiovascular risk.[8] Similarly, a systematic review of young South Asians with CAD found dyslipidemia prevalence ranging from 2.5% to 97.3%, with Nepal-specific studies reporting rates between 9.6% and 46.8%.[9] The low dyslipidemia prevalence in our study may reflect a unique subset of ACS patients in western Nepal, where smoking and hypertension appear to play a more significant role than lipid abnormalities.

There are several clinical implications of these findings. First, the low dyslipidemia prevalence suggests that routine lipid screening protocols in ACS patients at Nepalgunj may need adjustment to account for patients on lipid-lowering therapies or those with transient lipid changes post-ACS. The 2019 ESC/EAS Guidelines recommend measuring non-fasting LDL-C soon after ACS and initiating high-dose statins with ezetimibe if LDL-C exceeds 55 mg/dL, a threshold that may not be met in many of our patients due to the low dyslipidemia prevalence.[3] Second, the high prevalence of smoking and hypertension in our cohort highlights the need for aggressive risk factor modification beyond lipid management, including smoking cessation programs and blood pressure control, which are critical for secondary prevention in this population.

LIMITATIONS

Included the exclusion of patients on lipid-lowering drugs, which may have skewed the dyslipidemia prevalence, and the lack of apolipoprotein measurements (e.g., ApoB, ApoAI), which are more strongly associated with cardiovascular risk in South Asians. Additionally, the convenience sampling method and relatively small sample size (n=95) may limit generalizability. Future studies should include larger, multicenter cohorts and measure apolipoproteins to better characterize dyslipidemia patterns in western Nepal. Longitudinal studies tracking lipid profiles before and after ACS could also clarify the impact of acute-phase responses on lipid measurements.

CONCLUSION

Isolated low HDL-C is the predominant pattern in ACS patients in our study. Smoking and hypertension are the common modifiable risks. The low rate of dyslipidemia in ACS patients highlights the need for region-specific lipid screening approaches and urgent need for focused efforts to encourage healthier lifestyles and adequate blood pressure management to address ACS in western Nepal.

REFERENCES

1. World Health Organization. Cardiovascular diseases (CVDs) [Fact sheet]. Geneva: World Health Organization; 2021.
2. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937-52.
3. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J. 2020;41(1):111-88.
4. Dhungana SP, Mahato AK, Ghimire R, Shreewastav RK. Prevalence of dyslipidemia in patients with acute coronary syndrome admitted at tertiary care hospital in Nepal: a descriptive cross-sectional study. JNMA J Nepal Med Assoc. 2020;58(224):204-8.
5. Patterns of dyslipidemia among acute coronary syndrome (ACS) patients at a tertiary care hospital in Lahore, Pakistan. Cureus. 2022;14(12):e33045.
6. Gore JM, Goldberg RJ, Matsumoto AS, Castelli WP, McNamara PM, Dalen JE. Validity of serum total cholesterol level obtained within 24 hours of acute myocardial infarction. Am J Cardiol. 1984;54(7):722-5.
7. Islam MT, Saha SK, Shahnaz S, Shill DK, Das A, Rahman MA. Pattern and prevalence of dyslipidemia among patients with acute coronary syndrome admitted in a tertiary level hospital. Bangladesh Heart J. 2019;34(1):31-6.
8. Pokharel DR, Khadka D, Sigdel M, Yadav NK, Acharya S, Kafle RC, et al. Prevalence and pattern of dyslipidemia in Nepalese individuals with type 2 diabetes. BMC Res Notes. 2017;10(1):146.
9. Agrawal A, Lamichhane P, Eghbali M, Xavier R, Cook DE, Elsherbiny RM, et al. Risk factors, lab parameters, angiographic characteristics and outcomes of coronary artery disease in young South Asian patients: a systematic review. J Int Med Res. 2023;51(8):3000605231187806.

ORIGINAL ARTICLE

Obstetric Outcomes: A Comparison of Teenagers and Adults

Adhikari HN, Sinha K, Pandey A

ABSTRACT

Introduction: Teenage pregnancy poses significant risks to maternal and neonatal health, particularly in low-resource settings like Nepal, where adolescent pregnancies are linked to higher morbidity and mortality. **Aims:** To compare obstetric outcome between teenage mothers and adult mothers. **Methods:** A hospital based prospective case-control study was conducted at a tertiary hospital in Nepal over six months, including 94 participants (47 teenagers aged 13–19, 47 adults aged 20–35). Data on antenatal complications, delivery methods, and postpartum outcomes were analyzed using SPSS v 24.0 with Chi-square, independent t-tests, and Fisher's exact tests. **Results:** Teenage mothers were predominantly primigravida (87.2% vs. 29.8%, p<0.001) and had higher rates of intrauterine growth restriction (12.8% vs. 2.1%, OR=6.82, p=0.046). Adults exhibited higher cesarean deliveries (40.4% vs. 19.1%, p=0.002) and gestational diabetes (4.3% vs. 0%). Postpartum complications were more frequent in adults (14.9% vs. 6.4%, p=0.189), while teenagers uniquely experienced postpartum eclampsia (2.1%). The second stage of labor was prolonged in teenagers (45.89 vs. 28.75 minutes, p=0.001). **Conclusion:** Teenage pregnancies are associated with intra uterine growth restriction and prolonged labor, necessitating policies to delay childbearing, improve prenatal care access, and enhance nutritional and educational support for adolescents in community.

Keywords: Adolescent pregnancy, adult pregnancy, maternal health, neonatal outcomes, obstetric complications

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INTRODUCTION

The World Health Organization describes an adolescent or a teenager as those between 13 and 19 years of age and pregnancy between the age are regarded as teenage pregnancy.[1] Pregnancy in a mother's life itself carries a lot of impact to the maternal morbidity.[2] Moreover, teenage pregnancy significantly increases risks of developing anemia, hypertensive disorders, and premature rupture of membrane, ruptured uterus, antepartum/postpartum hemorrhage and chorioamnionitis during/after pregnancy.[3,4,5] These risks could possibly endanger the maternal and fetal outcome.[6] Neonatal complications include birth asphyxia, stillbirth, neonatal deaths, intrauterine death, respiratory distress, low birth weight, neonatal sepsis, IUGR, jaundice, congenital anomalies, meconium aspiration, and low birth weight APGAR score, and so on.[7] Teenage pregnancy stands as a major issue in Nepal since NDHS (2022) reports that motherhood or first pregnancy exists in 14% of females aged 15 to 19. The problem worsens because Nepal has significant rates of child marriage coupled with restricted healthcare access and insufficient education about sexual and reproductive health.[9 8] The risks of teenage pregnancy are heightened in South Asian settings like Nepal because of reduced access to quality prenatal care together with limited skilled medical assistance and limited healthcare systems.[1] This study addresses by examining age-specific obstetric risks to inform targeted interventions.[10] Young mothers generally lose access to both educational and economic opportunities because of these factors which create long-term poverty and negative health results.[11] Research exploring the obstetric results of teenage pregnancies remains scarce throughout Nepal while the situation remains poorly studied within tertiary medical facilities. Thus this research evaluates the delivery results for teenage pregnancies treated at a tertiary hospital.

METHODS

This is a Hospital based prospective case control study conducted at Obstetrics and Gynaecology ward of Nepalgunj Medical College, Kohalpur for 6 months from October 2024 to March 2025. The estimated prevalence of teenage pregnancy was taken as 14% as per Nepal demographic and health Survey 2022 findings. The required sample size calculated according to Cochran's formula and was calculated to be 47 for both the

"cases" and "control" groups. Therefore, the total sample size comprising both the study and control group was 94. This was an unmatched case–control study; cases and controls were not matched on any demographic or obstetric variables. All eligible teenage mothers (13–19 years) presenting for delivery during the study period were consecutively enrolled-no lottery or systematic random sampling methods were used. For each teenage delivery included as a case, the next eligible delivery of an adult mother (20–35 years) was enrolled as the control. Thus, recruitment was consecutive and feasibility-based within the labor ward workflow.

All the study participants were explained about the research in detail in their native language. After obtaining informed written consent, questionnaire was filled with direct interviews on the Performa. The questionnaire was based on the normal obstetric assessment proforma in the institution and already tested survey instruments on maternal health. The expert review of two senior obstetricians served as a means of content validity. Pilot testing was also conducted using 10 participants (not included in analysis), but only slight wording changes were done to enhance clarity. Recall bias was reduced through the use of interviews after delivery and cross validation of self-reported data with the antenatal records in case possible. Research was carried out after obtaining approval from the Institutional Review Committee (IRC) of Nepalgunj Medical College. Any mothers below the 18 years of age, dual written consent from her parents and herself were obtained. The data extracted were strictly confidential.

Inclusion criteria:

1. Cases between 13-19 years of age and controls between 20-35 years who were willing to take part in the study after informed consent were included.

2. Both primigravidae and multigravidae were included in cases and control.

Exclusion criteria:

1. Any pregnancy below 13 years of age and above 35 years of age and those who didn't wish to be included in the study were excluded from the study.

2. Pre-existing conditions like diabetes mellitus, heart diseases, chronic hypertension along with pregnancy were excluded from the study.

Statistical Analysis

Collected data were entered in specially structured proforma and analysed statistically in Microsoft Excel 2019 MSO (Version 2021 Build 16.0.14827.20158) 64- bit and IBM SPSS V 26.0 and Categorical variables were compared using χ^2 or Fisher's exact tests; continuous variables with t-tests/Mann-Whitney U tests.

Variables:

Maternal parameters reviewed were maternal age at delivery, gravida, period of gestation at the time of delivery, antenatal complications along stay at hospital the complications, nature of delivery (spontaneous/induced), mode of delivery (vaginal/instrumental/LSCS), total days of hospital stay during the course of delivery. Antenatal complications included pre-labor rupture of membrane (PROM), Intra-uterine Growth restriction (IUGR) (>2.5 SD below the mean weight for gestational age), oligohydramnios and anemia (Hb <9 gm/dL), infections, Pregnancy Induced Hypertension (PIH), Gestational Diabetes Mellitus (GDM), Antepartum hemorrhage (APH).

RESULTS

A total of 2728 deliveries were accounted at our hospital during the study period. The hospital's teenage (15-19) pregnancy rate was 8.43% (230/2728) compared to 86.8% adult (20-35) pregnancies (2369/2728), with 4.77% belonging to age group >35 years.

1. Demographic Characteristics

Characteristic	Teenage	Adult	Statistical Test	p-value
Primigravida (%)	41 (87.2%)	14 (29.8%)	χ^2	<0.001
Multigravida (%)	6 (12.8%)	33 (70.2%)		

Table I: Baseline Comparison

Among the age group included in the study, teenagers were significantly younger (18.19 ± 0.97 years vs 26.77 ± 4.54 years) (Δ=8.6 years, p<0.001) and were predominantly primigravida (87.2% vs 29.8%, p<0.001). Adults had higher parity (70.2% multigravida vs 12.8%, p<0.001), including 14.9% with ≥ G4.

2. Antenatal Complications

Complication	Teenage (n=47)	Adult (n=47)	p-value (Fisher's Exact)	Odds Ratio (95% CI)
Anemia	5 (10.6%)	4 (8.5%)	0.723	1.28 (0.34–4.83)
Threatened Abortion	1 (2.1%)	1 (2.1%)	1.000	1.00 (0.06–16.30)
PIH	2 (4.3%)	4 (8.5%)	0.403	0.48 (0.08–2.82)
Infection (INF)	2 (4.3%)	6 (12.8%)	0.133	0.31 (0.06–1.63)
GDM	0 (0%)	2 (4.3%)	0.155	-
APH	1 (2.1%)	3 (6.4%)	0.307	0.31 (0.03–3.14)
IUGR	6 (12.8%)	1 (2.1%)	0.046*	6.82 (1.13–41.30)

Oligohydramnios	1 (2.1%)	2 (4.3%)	0.558	0.48 (0.04–5.54)
PROM	1 (2.1%)	0 (0%)	0.316	-
Macrosomia	0 (0%)	1 (2.1%)	0.316	-

Table II: Comparison of Antenatal Complications between study and control group

The most reported complication among the control group was infection 12.8% vs. 4.3% in study group (p=0.133, clinically notable). However, IUGR 12.8% was the most reported complication among the study group vs. 2.1% in adults (p=0.046, OR=6.82). Higher PIH trends were seen (8.5% vs. 4.3%) and GDM (4.3% vs. 0%) in adults along with higher APH (6.4% vs. 2.1%) within the control group. Macrosomia was only reported in the control group (2.1%).

3. Delivery Outcomes

Complication	Teenage (n=47)	Adult (n=47)	p-value (χ^2/ Fisher's Exact)	Risk Ratio (95% CI)
Vaginal Delivery (VD)	38 (80.9%)	28 (59.6%)	0.002*	1.36 (1.11–1.66)
- Spontaneous (SVD)	34 (72.3%)	26 (55.3%)	0.082	1.31 (0.96–1.78)
- Instrumental/ Assisted	0 (0%)	0 (0%)	-	-
Cesarean Section (CS)	9 (19.1%)	19 (40.4%)	0.002*	0.47 (0.24–0.93)
- Elective CS (EL LSCS)	1 (2.1%)	7 (14.9%)	0.025*	0.14 (0.02–1.04)
- Emergency CS (EM LSCS)	8 (17.0%)	12 (25.5%)	0.303	0.67 (0.30–1.48)

Table III: Mode of Delivery

LSCS rate was more than double in adults (40.4% vs 19.1%, p=0.002), primarily due to previous cesareans (8.5% vs 0%). Adults had 7 times higher elective CS rate (14.9% vs. 2.1%, p=0.025). CPD (11.1%), transverse lie (22.2%), PROM (11.1%) were the major indications for LSCS among the cases. However, Severe pre-eclampsia (5.3%), big baby (5.3%) were the major causes of induction in the control group.

4. Hospitalization

Hospitalization	Teenage	Adult	p-value (t-test)
Total Stay (days)	3.34 ± 1.33	3.13 ± 0.64	0.421
ANC Hospitalization	2.1%	17.0%	0.013*

Table IV: Duration of hospital stay among groups

We observed no difference in total stay; (3.34 ± 1.33 days, p=0.421). Adults required more ANC admissions for obstetric related complications; and this was statistically significant (17% vs 2.1%, p=0.013).

5. Duration of Labor

Stage	Teenage (n=38)	Adult (n=28)	p-value (Mann-Whitney)
1st Stage	8.09 ± 1.34 hrs	7.36 ± 1.70 hrs	0.052 (NS)
2nd Stage	45.89 ± 21.56 mins	28.75 ± 12.18 mins	0.001*

Table V: Stage-wise duration of labor

We observed no significant difference in the duration of first stage of labor (8.09 hours vs 7.36 hours, p=0.052) between the study and control groups. Teenagers had a 17-minute prolongation (45.89 minutes vs 28.75 minutes, p=0.001).

6. Postpartum Complications

Complication	Teenage (n=47)	Adult (n=47)	p-value (Fisher's Exact)	Odds Ratio (95% CI)
Any Complication	3 (6.4%)	7 (14.9%)	0.189	0.39 (0.10–1.58)
Postpartum Hemorrhage (PPH)	1 (2.1%)	2 (4.3%)	0.558	0.48 (0.04–5.54)
Infectious Complications	1 (2.1%)	5 (10.6%)	0.092	0.18 (0.02–1.56)
- Puerperal Sepsis	1 (2.1%)	0 (0%)	0.316	-
- Mastitis	0 (0%)	1 (2.1%)	0.316	-
- PP Pyrexia	0 (0%)	4 (8.5%)	0.043*	-
Eclampsia	1 (2.1%)	0 (0%)	0.316	-

Table VI: Complications following delivery, Infections- Mastitis, Puerperal Pyrexia, Puerperal Sepsis

Adults had higher complication rates (14.9% vs. 6.4%), though not statistically significant (p=0.189). Adults had five times higher infections (10.6% vs. 2.1%, p=0.092), driven by pyrexia (8.5% vs. 0%). But Teenagers reported unique post partum eclampsia cases (2.1%).

DISCUSSION

Teenage pregnancies accounted for 8.43% of deliveries, aligning with Nepal's national average (8-10%) but lower than rural cohorts (15-20%).[13] The mean age of teenagers (18.2 ± 0.97 years) and adults (26.8 ± 4.5 years) reflects trends in South Asia, where early marriage persists despite legal restrictions.[14] Similar age gaps were reported in Nepal (18.2 vs. 26.7 years) and Pakistan (18.4 vs. 26.5 years).[15,16] Primigravida dominance

among teenagers (87.2% vs. 29.8%, p<0.001) mirrors findings from India (90%)17 and Bangladesh (84%)18, underscoring societal pressures for early childbearing. Adults' higher parity (70.2% multigravida) aligns with regional norms favoring repeated pregnancies. IUGR was significantly higher in teenagers (12.8% vs. 2.1%, p=0.046), consistent with Pathak et al (12.8% vs. 2.1%)15 and Indian studies (18%).20 Biological immaturity and nutritional competition between mother and fetus likely contribute to such findings.[21]

Anemia rates were comparable (10.6% vs. 8.5%, p=0.723), contrasting sharply with the study by Pathak et al (28.7% vs. 5.3%) 15 and Shah et al (58% vs. 55.9%).[16] PIH and GDM were lower in teenagers (4.3% vs. 8.5%; 0% vs. 4.3%), diverging from studies reporting elevated hypertensive disorders in adolescents[10], teenage mothers had 6.82 times higher odds of IUGR compared to adult mothers, with a 95% confidence interval of 1.13–41.30 (p = 0.046) which was found consistent with the study at Karnali, Nepal.[15]

Lower cesarean rates in teenagers (19.1% vs. 40.4%, p=0.002) align with Pathak et al (13.8% vs. 41.5%)[15] and Kumar et al (18% vs. 40%).[17] This contrasts with Shah et al (Pakistan), where CS rates were comparable.[16] Adults' higher elective CS (14.9% vs. 2.1%, p=0.025) reflects prior obstetric history (e.g., previous scars). Vaginal delivery predominance in teenagers (80.9% vs. 59.6%, p=0.002) is attributed to pelvic flexibility and smaller fetal size, as reported in Nepal and India.[15, 17]

Teenagers had a prolonged second stage (45.9 vs. 28.8 minutes, p=0.001), likely due to inadequate expulsive efforts or pelvic immaturity, corroborating Egyptian studies.[23] However, first-stage duration was comparable (p=0.052), contrasting with reports of prolonged labor in adolescents.[24] Duration for second stage of labor; in teenagers despite statistical significance, this difference is clinically modest (normal 2nd stage: ≤3 hours for primi).[28]

Lower complications in teenagers (6.4% vs. 14.9%, p=0.189) contrast with Shah et al (Pakistan), where infections and anemia were higher in adolescents.[16] Postpartum pyrexia in adults (8.5% vs. 0%, p=0.043) aligns with studies linking advanced age to infectious morbidity.[25] Unique eclampsia in teenagers (2.1%) highlights the need for vigilant postpartum monitoring, targeted nutrition programs for teenage mothers[26] even in low-risk cohorts. Overall reduction is teenage pregnancy and its complication it requires a strict bans on child marriage, as seen in Bangladesh's success.[27]

The teenagers were predominantly primigravida and adults had higher parity which could have had an impact on cesarean section, labor and postpartum complications. These differences in nutritional status, social economic background and access to antenatal care which is not under total control also could be seen to have contributed to the increased IUGR in teenagers. Prior obstetric history of adults, including previous cesarean delivery, also contributes to the confounding of differences in mode of delivery and maternal outcome.

CONCLUSION

Teenage pregnancies showed stronger obstetric risks especially the increased rates of IUGR and longer second stage labor, whereas adult mothers had more cesarean births and postpartum infections. These results make it necessary to put greater emphasis on the antenatal nutrition programs among adolescents and early screening of risks as well as the measures that can minimize unnecessary cesarean section among adults. Specific community-based health initiatives and enhanced access to teenage reproductive health are still needed. The study did not allow evaluating long-term maternal and neonatal outcomes.

REFERENCES

1. Organization WH. World Health Organization fact sheet. Adolescent pregnancy. Retrieved January. 2022;5: 2023.
2. Neiger R. Long-term effects of pregnancy complications on maternal health: a review. Journal of clinical medicine. 2017;6(8):76.
3. Chandra-Mouli V, Camacho AV, Michaud P-A. WHO guidelines on preventing early pregnancy and poor reproductive outcomes among adolescents in developing countries. Journal of adolescent health. 2013;52(5):517-22.
4. Chen X-K, Wen SW, Fleming N, Demissie K, Rhoads GG, Walker M. Teenage pregnancy and adverse birth outcomes: a large population based retrospective cohort study. International Journal of Epidemiology. 2007;36(2):368-73.
5. Edessy M, Gaber M, Maher A. Teenage pregnancy and fetal outcome. American Journal of Research Communication. 2014;2(10):169-75.
6. Todhunter L, Hogan-Roy M, Pressman EK. Complications of pregnancy in adolescents. Semin Reprod Med [Internet]. 2022;40(1–02):98–106. Available from: http://dx.doi.org/10.1055/s-0041-1734020
7. Pun KD, Chauhan M. Outcomes of adolescent pregnancy at Kathmandu University Hospital, Dhulikhel, Kavre. Kathmandu Univ Med J. 2011;9(33):50-3. doi:10.3126/kumj.v9i1.6263
8. UNICEF. The state of the world's children 2021: On my mind – Promoting, protecting, and caring for children's mental health. New York: UNICEF; 2021.
9. Ministry of Health, Nepal. (2022). Nepal Demographic and Health Survey 2022.
10. Raj, A., Saggurti, N., Balaiah, D., & Silverman, J. G. (2010). Prevalence of child marriage and its impact on the reproductive outcomes of young women in India: A crosssectional, observational study. The Lancet, 373(9678), 1883–1889. https://doi.org/10.1016/S0140-6736(09)60246-4
11. Amin, R., Diamond-Smith, N., & Singh, K. (2016). Maternal and child health inequalities in Nepal: Understanding the role of caste. BMC Public Health, 16(1), 1076. https://doi.org/10.1186/s12889-016-3714-6
12. Poudel S, Upadhaya N, Khatri RB, Ghimire PR. Trends and factors associated with pregnancies among adolescent women in Nepal: Pooled analysis of Nepal Demograph-

ic and Health Surveys (2006, 2011 and 2016). PLoS One. 2018;13(8):e0202107. doi:10.1371/journal.pone.0202107.

13. UNICEF. Child marriage in South Asia. 2020. Available from: https://www.unicef.org/rosa/what-we-do/child-protection/child-marriage.UNICEF

14. Pathak P, et al. Perceived stress and its associated factors among pregnant women attending antenatal care in a tertiary hospital in Nepal. JKAHS. 2021;4(2).Karnali Academy Journal

15. Shah N, Rohra DK, Shuja S, Liaqat NF, Solangi NA, Kumar K, et al. Comparison of obstetric outcome among teenage and non-teenage mothers from three tertiary care hospitals of Sindh, Pakistan. J Pak Med Assoc. 2011;61(10):963-7.

16. Kumar A, Singh T, Basu S, Pandey S, Bhargava V. Outcome of teenage pregnancy. Indian J Pediatr. 2007;74(10):927-31. doi:10.1007/s12098-007-0171-2.

17. Rahman M, et al. Maternal health service utilisation of adolescent women in sub-Saharan Africa: a systematic scoping review. BMC Pregnancy Childbirth. 2019;19:366. doi:10.1186/s12884-019-2501-6.BioMed Central

18. Ministry of Health - MOH/Nepal, New ERA/Nepal, and ICF. Nepal Demographic and Health Survey 2016. Kathmandu, Nepal: MOH/Nepal, New ERA/Nepal, and ICF; 2017. Available from: https://www.dhsprogram.com/pubs/pdf/fr336/fr336.pdf.

19. Yasmin G, Kumar A, Parihar B. Teenage pregnancy-its impact on maternal and fetal outcome. Int J Sci Study. 2014;1(6):9-13.

20. Chen XK, Wen SW, Fleming N, Demissie K, Rhoads GG, Walker M. Teenage pregnancy and adverse birth outcomes: a large population-based retrospective cohort study. Int J Epidemiol. 2007;36(2):368-73. doi:10.1093/ije/dyl284.

21. Al-Haddabi R, Al-Farsi Y, Al-Farsi O, Al-Rawahi H, Al-Khabori M. Obstetric and perinatal outcomes of teenage pregnant women attending a tertiary hospital in Oman. Oman Med J. 2014;29(6):399-403.

22. Abbas AM, et al. Pregnancy outcomes among teenagers at a national referral hospital in Uganda. Proc Obstet Gynecol. 2017;7(1):1-10.PMC

23. Gupta N, Kiran U, Bhal K. Teenage pregnancies: obstetric characteristics and outcome. Eur J Obstet Gynecol Reprod Biol 2008;137(2):165-71.doi:10.1016/ j.ejogrb. 2007.06.013.

24. Taffa N, et al. Int J Adolesc Med Health. 2005;15:321-9.

25. Ministry of Health and Family Welfare, Government of India. National Iron+ Initiative: Guidelines for Control of Iron Deficiency Anaemia. 2013. Available from: https://www.nhm.gov.in/images/pdf/programmes/child-health/guidelines/Control-ofIron-Deficiency-Anaemia.pdf.National History Museum

26. Ministry of Health and Family Welfare, Bangladesh. National Strategy for Adolescent Health 2017-2030. 2017. Available from: https://www.unicef.org/bangladesh /sites/ unicef.org.bangladesh/files/2018-10/NationalStrategy-for-Adolescent-Health-2017-2030.pdf.

27. ACOG Committee Opinion No. 766: Approaches to Limit Intervention During Labor and Birth. Obstet Gynecol. 2019;133(2):e164-e173.

ORIGINAL ARTICLE

Trends in Problematic Infection of Lesions: Etiology and Multidrug Resistance

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ABSTRACT

Introduction: Infection of wound and pus lead to delayed healing, prolonged hospital stay, increased healthcare costs and rising mortality and morbidity. Their clinical impact is increased by emerging multidrug resistance, particularly among common pathogens in hospital settings. Understanding local bacteriological trends and antimicrobial susceptibility is crucial for guiding effective treatment. **Aims:** To determine the trends in bacteriological pathogens of infected lesions, assess antimicrobial susceptibility patterns, and evaluate the prevalence and distribution of Multidrug Resistant organisms among patients presenting with wound or pus infections. **Methods:** A retrospective descriptive study was conducted at Dhulikhel Hospital from September 2022 to September 2025. All wound and pus samples submitted for culture and sensitivity testing were included. Standard microbiological methods were used for bacterial isolation, identification, and antimicrobial susceptibility testing following Clinical and Laboratory Standard Institute guidelines. Data were analyzed to determine species distribution, sensitivity patterns, and Multidrug Resistance prevalence across age groups. **Results:** Of 8,199 samples processed, 2,678 (32.7%) showed bacterial growth. Gram-positive organisms were slightly predominant (55.5%) . *Staphylococcus aureus* (33.79%) was the most common pathogen showing high susceptibility to cloxacillin (97.2%) and amoxicillin-clavulanic acid (85.5%), while Methicillin Resistant *Staphylococcus aureus* isolates remained sensitive to linezolid (90.7%) and vancomycin (79.2%). Among gram-negative bacteria, *Escherichia coli* displayed high sensitivity to gentamicin (84.8%) but low susceptibility to β-lactams and carbapenems. *Acinetobacter spp*. demonstrated extensive Multidrug Resistance with poor response to most antibiotic classes. Overall Multidrug resistance prevalence was 51.6%, highest among elderly patients (83.3%), followed by adults (54.2%) and children (36.7%). **Conclusion:** The study highlights *Staphylococcus aureus* and *Enterobacterales* as major pathogens in wound infections and reveals a concerning rise in Multidrug Resistance, especially among gram-negative bacilli. These findings emphasize the need for strengthened antimicrobial stewardship, continuous surveillance, and evidence-based empirical therapy.

Keywords: Drug resistance, *Enterobacterales*, Microbial sensitivity tests, *Staphylococcus aureus*, Wound infection

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INTRODUCTION

Infective lesion develops from disruption of skin's defense barrier which favors growth and colonization of pathogens.[1,2] The infection in lesion delay the healing, leading to long hospital stays, complications, financial burden and higher rate of morbidity and mortality in patients.[3] The incidence of infected lesions may be due to factors like environmental factors, host immune response and virulence factor of the microbes. Etiology of infections also play an important role in treatment outcomes of a patient.[4,5] The cause of infection of the lesions are microorganisms like bacteria, virus, fungus and the bacterial microorganisms may also co-exist as polymicrobial organisms in chronic infections.[6] Bacterial pathogens mostly found causing these infections are *Escherichia coli (E. coli), Staphylococcus aureus (S. aureus)*, different strains of *Klebsiella, Proteus, Pseudomonas* and *Acinatobacter.*[7] The alarming rise in multidrug-resistance (MDR) is a disturbing trend that has emerged in recent years which has sparked concerns in appropriate use of antibiotics to combat with the infection.[5,8,9] Different studies carried out in Nepal have shown increased rate of resistance towards different antibiotics, specially, increasing rate

of Methicillin Resistant *Staphylococcus aureus* (MRSA) and Extended Spectrum Beta-Lactamase (ESBL) producing bacteria has been reported.[6,10] Recognizing the changing patterns of microorganisms and their resistance to antimicrobials is essential for effective treatment of infection. This study intends to explore current trends in bacterial etiology of infections of lesions, prevalence of MDR stains among the causative agents and its implications on treatment of the infected patients.

METHODS

The study is a retrospective descriptive study that was conducted in outpatients and inpatients of Dhulikhel Hospital who had provided wound/pus swab sample for microbiological investigation from the September 2022 to September 2025. The analysis of the report was done at department of Microbiology, Dhulikhel Hospital. All the culture sensitivity reports of wound/pus swab has been included in the study. All age groups of patients were selected. For the microbiological investigation, sterile cotton swabs or sterile syringes were used to collect pus samples from infected wound and were labeled properly with patient's details along with date and time of sample collection. The collection and labeling of samples were done by trained nurses of respective departments. Collected samples were delivered to microbiology laboratory within an hour for microbiological tests. Microscopic examination was done after gram stain for presumptive identification of gram positive and gram-negative bacteria.[11,12]

Culture and identification of isolates: Samples were inoculated into MacConkey agar and Blood agar. They were then incubated at 37^0C for 24 hours. After incubation, grown isolates were identified according to standard microbiological criteria such as colonies morphology, gram stain and biochemical properties.[11,13] Gram positive cocci were identified up to species level by Catalase test, Coagulase test and by using Optochin and Bacitracin disc whereas gram negative bacilli are identified by Catalase test, Oxidase test, Indole test, Motility, Hydrogen sulfide production, Triple sugar iron test, Urease test and Citrate test.[12,14]

Antibiotic Susceptibility test (AST): Antibiotic susceptibility test were performed for all bacterial isolates by a modified Kirby – Bauer disk diffusion method according to the guidelines of Clinical and Laboratory Standard Institute (CLSI) on Mueller Hinton Agar.[15]

Statistical Analysis:

Data were analyzed using SPSS version 16. Categorical variables were summarized as frequencies and percentages. The chi-square test was applied to assess the association between age groups and MDR status, with $p<0.05$ considered statistically significant.

RESULTS

Among 8199 pus samples that were processed for bacterial culture and sensitivity, 2678 (32.7%) showed bacterial growth and 5521(67.3%) showed no growth. Slight male predominance in culture positivity was seen 1523 (56.9%) were from males and 1155 (43.1%) were from females.

Among 2678 bacterial isolates, 1487 (55.5%) were gram positive organisms and 1191 (44.5%) were gram negative. The most pre-dominant organism was found to be *Staphylococcus aureus* (33.8%) followed by *Escherichia coli* (19.6%), *Enterococcus* (9.5%), *Coagulase-negative staphylococci* (9.4%) and *Klebsiella pneumoniae* (9.0%). *Proteus spp., Pseudomonas spp. Streptococcus spp., Enterobacter spp., Acinetobacter spp., Klebsiella oxytoca, Citrobacter spp*. and Methicillin Resistant *Staphylococcus aureus* (MRSA) were among the less frequent isolates.

Antibiotic sensitivity pattern:

Among gram positive organisms, *Staphylococcus aureus* has shown to be highly sensitive to Cloxacillin (97.2%) and Amoxycillin-clavulinic acid (85.5%) and sensitivity was low to Penicillin (17.3%). *Enterococcus spp*. showed high sensitivity to Linezolid (93.7%) and vancomycin (87.8%). For MRSA isolates, Vancomycin (79.2%) and Linezolid (90.7%) proved to be fairly effective.

Among gram negative organisms, *Escherichia coli* was highly sensitive to Gentamicin (84.8%), moderately sensitive to Ciprofloxacin (48.9%) and showed lowed low sensitivity to B-lactam antibiotics (Imipenem and meropenem both 25.6%). Proteus spp. showed high sensitivity to Ceftriaxone (73.8%) and Cefoperazone (66.2%), *Pseudomonas* showed high sensitivity to Cefepime (64.3%) and Ciprofloxacin (67.9%), *Acinetobacter spp.* has shown distinct multidrug-resistance with low sensitivity to most of the antibiotics including carbapenems (Imepenem 39.3%).

MDR

The overall prevalence of MDR was 51.6%, elderly population was highly burdened (83.3%) than adults (54.2%) and pediatric patients (36.7%) ($p<0.05$).

Trends in MDR has revealed significant rise in Gram-Negative Bacilli (GNB). The burden of MDR was heaviest in Acinetobacter, which showed resistance to almost all antibiotic classes like cephalosporins, fluoroquinolones, carbepenems and B-lactam inhibitors. *K. pneumoniae* and *E. coli* showed markedly decreased susceptibility towards cephalosporins, fluroquinolones and carbapenems indicating increasing multidrug resistance and expanding carbapenem-resistant enterobacterales (CRE). MRSA and enterococcus species were the main gram positive organisms where MDR was observed. Last resort drugs like Linezolid and Vancomycin has proven to be still effective.

Organisms	Count	Percentage (%)
Staphylococcus Aureus	905	33.79
Escherichia coli	526	19.64
Enterococcus spp.	254	9.48
Coagulase Negative Staphylococcus (CONS)	251	9.37
Klebsiella Pneumoniae	241	9.00
Proteus spp.	106	3.96
Pseudomonas spp.	100	3.73
Streptococcus spp.	75	2.80
Enterobacter spp.	74	2.76
Acinetobacter spp.	65	2.43
Klebsiella Oxytoca	43	1.61
Citrobacter spp.	36	1.34
Methicillin Resistant Staphylococcus (MRSA)	2	0.07

Table I: Distribution of different organisms isolated from infected lesions

Antibiotic	*Staphylococcus aureus* n (%)	*CoNS* n (%)	*Enterococcus spp.* n (%)	*Streptococcus spp.* n(%)
Penicillin	157 (17)	0 (0)	148 (58.2)	39 (52.6)
Amoxicillin-clavulinic acid	774 (85.5)	6 (2.3)	52 (20.6)	39 (52.6)
Erythromycin	548 (60.6)	6 (2.3)	0 (0.0)	20 (26.3)
Cloxacillin	880 (97.2)	6 (2.3)	0 (0.0)	0 (0.0)
Gentamicin	154 (17.0)	12 (4.7)	227 (89.4)	24 (31.6)
Ciprofloxacin	429 (47.4)	18 (7.0)	129 (50.8)	16 (21.1)
Tetracycline	41 (4.5)	0 (0.0)	110 (43.4)	24 (31.6)
Cotrimoxazole	658 (72.7)	6 (2.3)	0 (0.0)	8 (10.5)
Amikacin	57 (7.3)	12 (4.7)	103 (40.7)	8 (10.5)
Vancomycin	60 (6.6)	0 (0.0)	223 (87.8)	47 (63.2)
Linezolid	72 (8.0)	0 (0.0)	238 (93.7)	43 (57.9)

Table II: Gram positive organisms and its sensitivity to different antibiotics

Antibiotic	*E. coli* n (%)	*Acinetobacter spp.* n (%)	*Proteus spp.* n (%)	*K. pneumoniae* n (%)	*K. oxytoca* n (%)	*Citrobacter spp.* n (%)	*Pseudomonas spp.* n (%)	*Enterobacter spp.* n (%)
Cefuroxime	136 (25.9)	0 (0.0)	41 (38.5)	69 (28.6)	30 (70.8)	6 (15.8)	0 (0.0)	27 (36.2)
Gentamicin	446 (84.8)	37 (57.1)	78 (73.8)	174 (72.2)	43 (100)	32 (89.5)	61 (60.7)	55 (74.5)
Amikacin	292 (55.6)	33 (50.0)	34 (32.3)	78 (32.3)	14 (33.3)	15 (42.1)	75 (75.0)	39 (53.2)
Ciprofloxacin	257 (48.9)	23 (35.7)	65 (61.5)	121 (50.4)	23 (54.2)	21 (57.9)	68 (67.9)	47 (63.8)
Cotrimoxazole	29 (5.6)	0 (0.0)	13 (12.3)	20 (8.3)	5 (12.5)	8 (21.1)	0 (0.0)	8 (10.6)
Cefepime	28 (5.3)	12 (17.9)	15 (13.8)	38 (15.8)	7 (16.7)	0 (0.0)	64 (64.3)	0 (0.0)
Colistin	79 (15.0)	23 (35.7)	11 (10.8)	80 (33.1)	5 (12.5)	0 (0.0)	20 (19.6)	13 (17.0)
Imipenem	253 (48.1)	26 (39.3)	11 (10.8)	62 (25.6)	9 (20.8)	4 (10.5)	23 (23.2)	30 (40.4)
Meropenem	259 (49.2)	21 (32.1)	24 (23.1)	62 (25.6)	16 (37.5)	8 (21.1)	20 (19.6)	33 (44.7)
Cefoperazone	190 (36.1)	7 (10.7)	70 (66.2)	112 (46.6)	22 (50.0)	28 (78.9)	7 (7.1)	38 (51.1)
Ceftriaxone	221 (42.0)	5 (7.1)	78 (73.8)	98 (40.6)	5 (12.5)	21 (57.9)	0 (0.0)	35 (46.8)

Table III: Gram-negative organisms and its sensitivity to different antibiotics

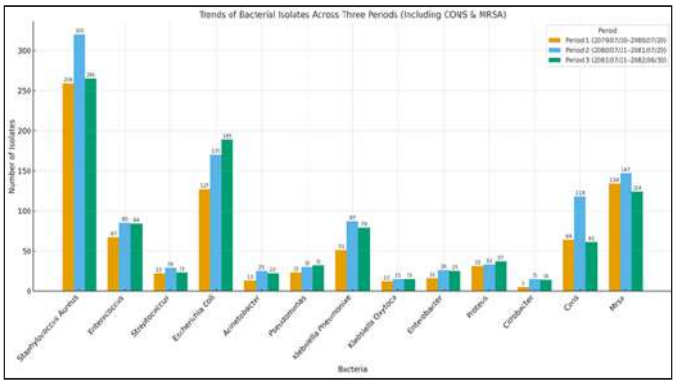

Figure 1: Bacterial Trend analysis across period of three years

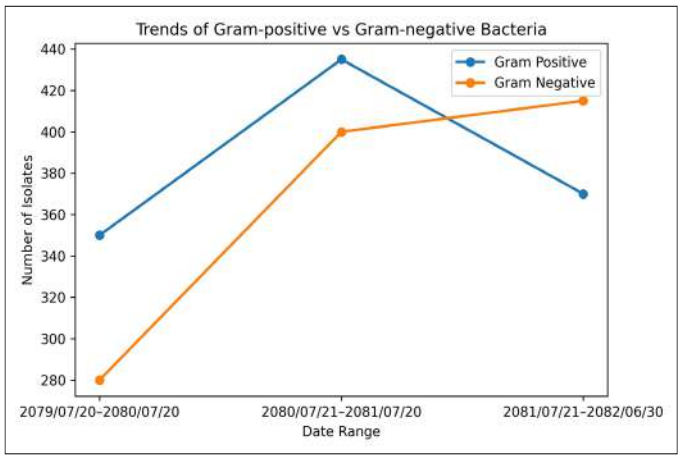

Figure 2: Trends of Gram-negative vs gram positive bacteria

Age Group	Multidrug Resistant n (%)	Non-Multidrug Resistant n (%)	Total	p-value	Remark
Pediatric (<15 yrs)	226 (36.7%)	390 (63.3%)	616	<0.05	Significant
Adult (15–59 yrs)	825 (54.2%)	697 (45.8%)	1522		
Elderly (≥60 yrs)	450 (83.3%)	90 (16.7%)	540	<0.05	Significant
Total	1501 (51.6%)	1177 (48.4%)	2678		

Table IV: MDR according to age-group

DISCUSSION

This study showed 32.7% of bacterial growth in the infected lesions. The positivity rate is comparable to finding from other studies on pus/wound infections of south-Asian countries where the rate ranges from 25-40%.[8] The culture positivity in almost 1/3 of the samples reflects the burden of pyogenic infections and need for appropriate measures for the control. A slight male predominance was observed where 56.87% of male population showed culture-positivity. This predominance is relatable to the studies reporting men to more frequent exposure to outdoor activities and risky occupations leading to trauma and injuries.[16]

Organisms:

The predominance of gram-positive organisms (55.5%), especially *Staphylococcus aureus* as the most common isolate (33.79%) is in accordance to the global data[17] where *S. aureus* has been persistently identified as the main organism responsible for causing wound and soft tissue infections. The presence of gram negative organisms like *Klebsiella pneumoniae* (9%), *Proteus spp., Pseudomonas spp*. and *Acinetobacter spp.* which are usually found in hospital-acquired infections indicates the importance of GNB pathogens in wound infections. Similar observations has been found in other studies from India and Nepal as well.[18] Its detection has highlighted the importance of gram negative organisms in pus/wound infection in hospital settings.

Antibiotic Sensitivity patterns:

Among gram positive organisms, *S. aureus* was highly sensitive to cloxacillin (97.2%) amoxicillin-clavulinic acid (85.5%) and moderately sensitive to erythromycin (60.6%) and ciprofloxacin (47.4%). This trend in sensitivity of gram positive organisms indicates that the dominance of methicillin-susceptible strains is still prevalent in hospital settings. Prior studies from Nepal also shows similar results.[19] The prevalence of MRSA was very low (0.07%) and this demonstration can be considered as a positive sign. The principal drugs used for the MRSA isolates like vancomycin and linezolid[20] demonstrated high susceptibility with Linezolid being 90.7% susceptible and vancomycin being 79.2% susceptible. Global reports demonstrating vancomycin-sensitive Enterococci (VSE)[21] aligns with our findings that show high sensitivity to gentamicin (89.4%), vancomycin (87.8%) and linezolid (93.7%).

Gram negative bacteria, mostly *E. coli, Citrobacter* and *K. oxytoca* showed high sensitivity to gentamicin with sensitivity of 84.8%, 89.5% and 100% respectively. The uprising strains of carbapenem-resistant enterobacterales (CRE)[22] is quite notable and should not be ignored. Cotrimoxazole and cefepime has proven to be not much effective overall, especially against *E. coli* and *acinetobacter* indicating widespread trends in resistance to the antibiotics.[23] Colistin, which is often considered as last-resort antibiotics demonstrated reduced effectiveness in our study which is a matter of concern worldwide.[24]

Multidrug resistance

Multidrug resistance was observed across all age groups. The widespread existence of this multidrug resistant strains is quite alarming. Among pediatric, adult and old age groups, MDR infections were more frequent among old age groups. This finding were also observed in other studies[25] where comorbidities and frequent exposure to antibiotics might have been its cause.

LIMITATIONS

This study has some limitations. Its retrospective design restricts further processing of the clinical samples for more accurate results. Even if our hospital is the main tertiary care center and covers large area and population, it is considered as a single center study-so, the results might not reflect/ cover the situation of our country as a whole.

CONCLUSION

The findings in this study has reflected typical patterns of bacterial microorganisms that are isolated in pus and wound infections where *Staphylococcus aureus* and *Enterobacterales* were the main bacterial pathogens. Antibiotic resistance trends from this study reflects the disturbing rise of the MDR microorganisms and need for urgent address to this situation.

REFERENCES

1. Maillard JY, Kampf G, Cooper R. Antimicrobial stewardship of antiseptics that are pertinent to wounds: The need for a united approach. JAC Antimicrob Resist. 2021;3(1):1-20. DOI
2. Bhatta CP, Lakhey M. The distribution of pathogens causing wound infection and their antibiotic susceptibility pattern. J Nepal Health Res Counc. 2007;5(1): 22–5. LINK
3. Negut I, Grumezescu V, Grumezescu AM. Treatment strategies for infected wounds. Molecules. 2018;23(9):2392. DOI
4. Sahle B, Merid Y. Prevalence and antibiotic resistance of Staphylococcus aureus in wound infections: A hospital study in Hawassa, Ethiopia. J Infect Dev Countries. 2024;18(10):1530-8. DOI
5. Ilyas F, James A, Khan S, Khan S, Haider S, Ullah S, Darwish G,et al. Multidrug resistant pathogens in wound infections: A systematic review. Cureus. 2024;16(4). Full text
6. Parajuli P, Basnyat SR, Shrestha R, Shah PK, Gurung P. Identification and Antibiotic Susceptibility Pattern of Aerobic Bacterial Wound Isolates In Scheer Memorial Hospital. JSM Microbiology. 2014; 2(2):1011. Full text
7. Yeong EK, Sheng WH, Hsueh PR, Hsieh SM, Huang HF, Ko AT, et al. The wound microbiology and the outcomes of the systemic antibiotic prophylaxis in a mass burn casualty incident. J Burn Care Res 2020;41(1):95-103. DOI
8. Divya P, Krishna S, Mariraj J et al. Aerobic Bacteriological Profile of Post-Operative Surgical Wound Infections and Their Antibiogram in A Tertiary Care Hospital. Journal of Medical Science and clinical Research. 2015; 3(6): 6310-6316. LINK
9. Li W, Sadeh O, Chakraborty J, Yang E, Basu P, Kumar P. Multifaceted antibiotic resistance in diabetic foot infections: A systematic review. Microorganisms. 2025;13(10):2311. DOI
10. Maharjan N. Bacteriological pofile of wound infection and antibiotic susceptibility pattern of various isolates in a tertiary care center. J Lumbini. Med Coll. 2020;8(2):218-24. DOI
11. Cheesbrough M. District Laboratory Practice in Tropical Countries. 2nd ed. Cambridge University Press; 2006. LINK
12. Tille PM. Bailey & Scott's Diagnostic Microbiology. 14th ed. St. Louis: Elsevier; 2017. LINK
13. Forbes BA, Sahm DF, Weissfeld AS. Bailey & Scott's Diagnostic Microbiology. 12th ed. St. Louis: Mosby Elsevier; 2007. LINK
14. Perilla MJ. Manual for the laboratory identification and antimicrobial susceptibility testing of bacterial pathogens of public health importance in the developing world: Haemophilus influenzae, Neisseria meningitidis, Streptococcus pneumoniae, Neisseria gonorrhoeae, Salmonella serotype Typhi, Shigella, and Vibrio cholerae. 2003. LINK
15. Clinical and Laboratory Standards Institute (CLSI). Performance Standards for Antimicrobial Susceptibility Testing. 33rd ed. CLSI supplement M100. Wayne, PA: CLSI; 2023. LINK
16. Agbakoba NR, Enweani IB, Udeogu CV, Dilibe EA, Ekelozie IS, Chukwuma LN. Microbial population of wound isolates and sociodemographic characteristics in patients attending clinic in National Orthopaedic Hospital, Enugu, Nigeria. World J Adv Res Rev. 2024;21(2):1652–1659. LINK
17. Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJ, Gorbach SL, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014;59(2):e10-52. DOI
18. Mohanty S, Kapil A, Dhawan B, Das BK. Bacteriological and antimicrobial susceptibility profile of soft-tissue infections from Northern India. Indian J Med Sci. 2004;58(1):10-5. Pubmed
19. Khanal LK, Adhikari RP, Guragain A. Prevalence of Methicillin Resistant Staphylococcus aureus and Antibiotic Susceptibility Pattern in a Tertiary Hospital in Nepal. J Nepal Health Res Counc 2018 Apr-Jun;16(39): 172-4. LINK
20. Boucher HW, Corey GR. Epidemiology of methicillin-resistant Staphylococcus aureus. Clin Infect Dis. 2008;46(S5):S344-9. DOI
21. Arias CA, Murray BE. Enterococcal infections treatment and resistance. N Engl J Med. 2012;366:1995-2005. Full text
22. Logan LK, Weinstein RA. The Epidemiology of Carbapenem-Resistant Enterobacteriaceae: The Impact and Evolution of a Global Menace. J Infect Dis. 2017 Mar 28;215(Suppl 1):S28–S36. LINK
23. Wani FK, Bandy A, Alzeni MJS et al. Resistance Patterns of Gram-Negative Bacteria Recovered from Clinical Specimens of Intensive Care Patients. Microorganisms. 2021 Oct 28;9(11):2246. LINK
24. Binsker U, Kasbohrer A, Hammerl JA. Global colistin use: a review of the emergence of resistant Enterobacterales and the impact on their genetic basis. FEMS Microbiol Rev. 2021 Oct 6;46(1). LINK
25. Magill SS, Edwards JR, Bamberg W et al. Multistate point-prevalence survey of health-care–associated infections. N Engl J Med. 2014;370:1198-1208. Full text

ORIGINAL ARTICLE

Prevalence of Ocular Morbidity Among Road Traffic Accident Patients and its Management at a Tertiary Care Centre

Kafle PA, Chaudhary NP, Goyal N

ABSTRACT

Introduction: Road traffic accidents are a significant cause of preventable ocular morbidity. They can lead to permanent visual impairment and reduction in quality of life. **Aims:** To determine the prevalence, clinical spectrum, and management of ocular injuries among RTA patients presenting to a tertiary care center in Nepal. **Methods:** A hospital-based observational cross-sectional study was conducted at Birat Medical College Teaching Hospital. A total of 278 patients presenting with road traffic accidents were enrolled through consecutive sampling. Comprehensive ophthalmic examination was performed for all patients, and ocular injuries were classified based on standard trauma classifications. Statistical analysis was performed to see the associations between variables. **Results:** Ocular involvement was detected in 19.1%. Male predominance (65.5%) was recorded. Two-wheeler riders constituted 66.5% of road traffic accidents victims and 77.36% of those with ocular injuries. Those with ocular injuries 66.03% were not using helmets, and alcohol intake was reported in 53.8%. Periocular injuries were the most common (65%). A significant association was observed between nature of travel and ocular injury ($p = 0.0002$), and between mode of accident and ocular involvement ($p = 0.0000$). At presentation, 71.7% of patients had normal visual acuity, and most were managed conservatively. **Conclusion:** Ocular injuries represent a significant yet preventable consequence of road traffic accidents. Strengthening road safety regulations, promoting helmet use, and ensuring early ophthalmic evaluation are essential to reduce RTA-related ocular morbidity.

Keywords: Ocular trauma, Periocular injury, Road traffic accident

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INTRODUCTION

Road traffic accidents (RTAs) remain one of the leading causes of preventable injury worldwide and place a substantial burden on healthcare systems. While life-threatening injuries often receive immediate attention, ocular trauma is frequently underestimated. It has potential to cause profound visual disability.[1] Given the anatomical proximity of the eyes to the craniofacial skeleton, ocular structures are vulnerable during high-impact trauma.[2] Injuries may occur as part of polytrauma or as isolated events.[3] The severity of ocular damage depends on the magnitude and direction of force, the nature of the impacting object, and the presence or absence of protective measures. Clinical presentations range from simple eyelid abrasions to devastating injuries such as globe rupture or optic nerve trauma, which may result in irreversible vision loss and long-term visual impairment.[4] We can usually find multiple orbital bone fractures that lead to complicated management protocols requiring multidisciplinary approach.[4] Several contributing factors influence the pattern of ocular involvement in RTAs, including the mode of transportation, helmet or seatbelt use, alcohol intake, and delay in seeking medical care.[5] Studies have shown that 10–15% of all ocular injuries requiring hospitalization are attributed to road traffic accidents, with the majority involving young male adults who are economically productive members of society.[6,7] Although studies from Nepal and neighboring regions have described general ocular trauma patterns, focused data on RTA-related ocular injuries and their management are limited. This study was therefore designed to assess the prevalence, spectrum, clinical characteristics, and management strategies of ocular injuries.

METHODS

This observational cross-sectional study was conducted at Birat Medical College Teaching Hospital, Budhiganga, Nepal, involving patients presenting to the Emergency and Ophthalmology Departments following RTAs. All eligible patients attending during the study period were screened for inclusion. The study duration was from the date of acceptance of IRC(Ref- IRC-41-2081/2082) till data collection of sample size was achieved, which was from June 2025 to October 2025. Patients of all age groups and both genders who sustained injuries due to RTAs were included. Sample size was determined using standard prevalence-based calculation, assuming a prevalence of 23.63%,[8] resulting in a minimum sample size of 278. Consecutive sampling was used, and patients were enrolled until the required number was reached. Individuals were included if they presented directly to the hospital following an RTA and received ophthalmological assessment and initial management. Exclusion criteria included non-RTA-related ocular injuries, prior treatment received elsewhere before presentation, or refusal to participate.

Data collection was carried out using a predefined structured proforma covering socio-demographic factors, mechanism of injury, type of impact, type of vehicle involved, use of protective gear, alcohol intake, systemic injuries, ophthalmic findings, and treatment details. Ophthalmic examination included assessment of visual acuity, slit-lamp evaluation, intraocular pressure measurement (when appropriate), and fundoscopy with pupillary dilation. Imaging modalities such as X-ray or CT scan were used when clinically indicated. Ocular injuries were categorized according to standard trauma classification systems. Visual acuity was graded following WHO criteria, and injuries requiring operative intervention or associated with significant structural damage were considered severe.

Statistical analysis

Data analysis was performed using statistical software. Chi-square tests were used to determine associations between categorical variables, and a p-value of less than 0.05 was considered statistically significant. Ethical clearance was obtained from the Institutional Review Committee of Birat Medical College Teaching Hospital.

RESULTS

Among the 278 RTA patients included in the study, the majority (85.6%) belonged to the 15–59-year age group. Male patients predominated consistent with high-risk working-age population, accounting for 65.5% of cases, with a male-to-female ratio of 1.9:1. Only a small proportion of pediatric age group were involved in RTAs. (figure.1)

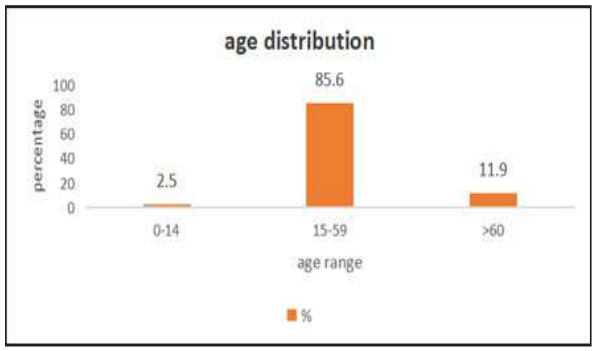

Figure 1: Age distribution among study populations

Two-wheeler users represented the largest group of accident victims (66.5%) followed by 4-wheelers (15.5%) and pedestrians (10.8%). (figure 2). Among those with ocular injuries, 77.36% were two-wheeler riders. Lack of helmet or protective equipment was noted in 66.03% of patients with ocular trauma. Alcohol consumption at the time of accident was documented in 53.8% of cases.

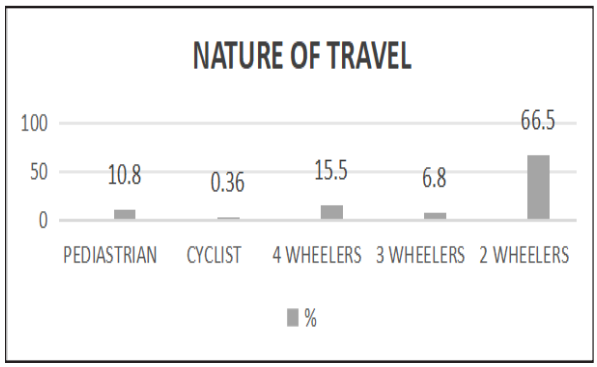

Figure 2: Nature of travel among RTA patients

Collision was the most frequent mechanism of injury (56.8%), followed by sideways (23.7%) and frontal impacts (11.9%). Figure 3

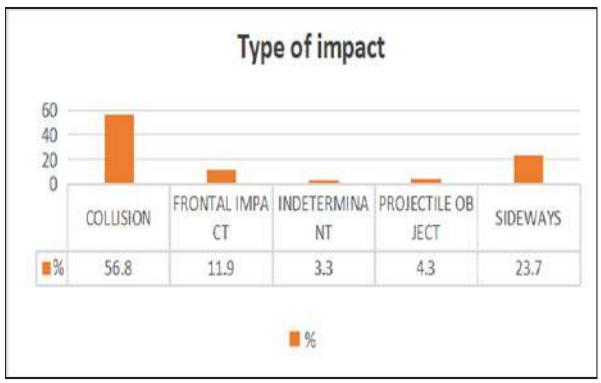

Figure 3 : Showing type of impact among RTA patients

Ocular involvement was detected in 19.1%(n= 53) of all RTA patients. Among them 51% had left eye involved followed by righteye an both eyes (40% and 9%). Periocular injuries were most common (65%), followed by combined globe and periocular injuries (28%) and isolated globe injuries (8%)

(figure 4). Closed globe injuries accounted for the majority, with contusions being the most frequent subtype seen in 17 patients(89.47%) and 2 patients had lamellar laceration (10.52%). Only one case of open globe injury was identified. Among periocular injuries 43 of them had only eyelid injuries and 6 of them had orbit involved along with eyelid in a form of orbital fractures of varying severity.

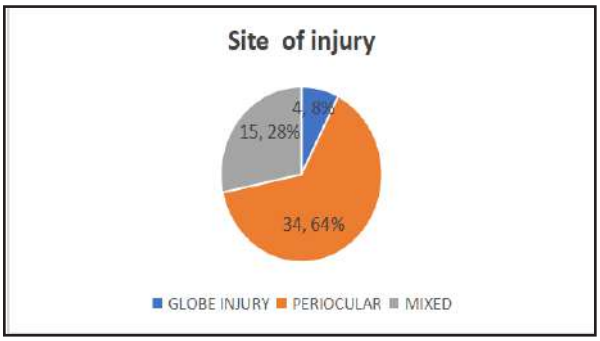

Figure 4: Site of ocular injuries

A statistically significant relationship was found between type of vehicle used and ocular injury (p = 0.0002), as well as between mechanism of accident and ocular involvement (p = 0.0000), particularly in sideways and frontal impacts. Sideways and frontal collisions show notably higher injury rates compared to projectile or in-determinant modes. At presentation, most patients (71.7%) had normal visual acuity. Out of total cases of ocular injuries 2 had severe visual impairment due to severe echymosis with orbital fracture. Three of those who were categorized as blind had closed globe injury leading to rupture, total hyphema and open globe injury. The distribution of visual impairment is shown below:

Visual acuity	Number	Percentage %
Normal	38	71.7
Visual impairment	10	18.9
Severe visual impairment	2	3.8
Blind	3	5.6

Table I: showing distribution of vision according to WHO criteria

Management was primarily conservative or minimally invasive. Medical treatment accounted for 45.3% of cases and surgical management was done in 54.7% of cases. Among surgical management eyelid repair was most common accounting for 45.3% Other interventions included scleral repair, hyphema wash, orbital fracture repair, conjunctival repair, and foreign body removal.

Type of management	Number	Percentage
Medical	24	45.3
Surgical	29	54.7
Total	53	100

Table II: Modalities of management

Types of surgical management	Number	Percentage %
Lid repair	24	45.3
Scleral repair	1	1.9
Lid+ conjunctival repair	1	1.9
Hyphema wash	1	1.9
Orbital # repair	1	1.9
Foreign body repair	1	1.9

Table III: Surgical Management

DISCUSSION

The present study demonstrates that ocular injuries are a notable consequence of RTAs, affecting nearly one-fifth of cases. There were 278 cases of RTAs, a substantial proportion (85.6%) fell within the 15-59 year age range. Young males constituted the most affected population, reflecting their higher exposure to traffic and greater involvement in high-risk driving behaviors. This finding is similar to study conducted by Das S et al at Guhawati Medical college which has reported that 75.2% of ocular injuries from RTA occurred in males, with most patients being in their third and fourth decades of life.[9] Our study had male predominance (male : female = 1.9:1), consistent with studies that report 80 -88.40% of men as more frequent victims of road traffic-related ocular injuries.[8,10] Two-wheeler riders constituted the majority (66.5%) of RTA victims and 77.35% in those who had ocular injuries due to RTAs and poor helmet compliance (66.03%) emerged as a key modifiable risk factor. This is particularly concerning, as the lack of protective gear is a well-established risk factor for facial and ocular trauma. Previous studies similarly report high prevalence of ocular injuries among two-wheeler riders and low usage of helmets - for instance, in a large trauma-database study, lack of protective gear was significantly associated with increased risk of orbital fractures (OR = 2.4; p < 0.0001).[11] A study done in north India shows only 30.53% of 2 wheeler riders sustained eye injuries which is much lesser than our study.[8] where as studies by other authors in India have 87.33% and 73.6% of ocular injuries occurred in two-wheeler riders.[12,13] Not using helmets have greater impact in RTAs and ocular morbidity. Findings of our study (66.03%) regarding this has a similar finding with the study done in Northern India.[8] Thus travelling without wearing protective gears is one of the major modifiable risk factors for RTA related ocular injuries. Alcohol consumption also played a substantial role in accident occurrence and severity(53.8%). Similar results(42.10 %) were seen in other study.[8] Driving under the influence of alcohol is a risk factor for ocular morbidity in RTAs.[5] These findings highlight the urgent need for stronger enforcement of safety regulations and public awareness initiatives.

Regarding the type of impact, collision was the most common mode (56.8%), followed by sideways impact (23.7%) and frontal impact (11.9%). Similarly in another study, frontal collision (37.89%) was the commonest followed by sideways impact (26.32%).[8] There is a significant association between the mode of accident and ocular injury(p = 0.0000),especially with

sideways and frontal collisions. This suggests that certain types of impacts may confer higher risk to ocular structures, maybe due to greater force transmission to the face. This emphasizes the importance of preventive road safety measures. Ocular involvement was present in 19.1% of RTA victims in our study. Approximately 1 in 5 RTA patients presented with ocular morbidity. By putting 95%CI prevalence is 14.4%-23.8%.

Similar prevalence was seen in the study which was done in a tertiary center of northern India.[8] This study shows morbidity was slightly more predominant in the left eye (43.4%). Some of the other studies had right eye involvement (52%, 53.8%).[14,15] Most ocular injuries involved periocular tissues (65%), with closed globe injuries being more prevalent than open globe injury. This pattern aligns with observations from similar regional studies where periorbital edema and eyelid lacerations dominated the spectrum of ocular trauma.[14,16] Within closed globe injuries, contusion was predominant and open globe injuries were rare (only 1 patient had zone 2 open globe injury-scleral perforation). This is similar to findings in several studies where closed globe injuries significantly outnumber open globe trauma in RTA-related eye injuries.[12,17,18] As there were less number of vision threatening injuries 71.7 % of patient's presenting visual acuity was normal(WHO criteria).

The predominance of normal visual acuity at presentation suggests that many injuries were superficial; however, the potential for delayed complications must not be overlooked. In terms of management, nearly half of the patients (45.3%) were treated medically, and an equal proportion underwent lid repair. Surgical interventions such as scleral repair, hyphema wash, orbital fracture repair, or foreign body removal were rare. This again underscores that many RTA-related ocular injuries are superficial or non-penetrating, amenable to conservative or minor surgical management.

LIMITATIONS

This study was limited by its hospital-based design, which may not reflect the true community burden. Self-reported data on helmet use and alcohol intake may be subject to bias. Lack of long-term follow-up prevented assessment of final visual outcomes. Additionally, detailed evaluation of posterior segment involvement was limited, potentially underestimating severe ocular sequelae.

CONCLUSION

Ocular trauma represents a significant yet largely preventable morbidity among RTA victims, particularly among two-wheeler riders. Low usage of protective gear and high prevalence of alcohol involvement underline critical areas for intervention. Despite the fact that most injuries were closed-globe or periocular (with generally good visual prognosis), effective prevention strategies could further reduce the burden. Strengthening emergency eye trauma services and enforcing road safety measures are both essential steps toward mitigating this public health challenge.

REFERENCES

1. World Health Organization. Global status report on road safety 2018. Geneva: World Health Organization; 2018.
2. Maheshwari V, Dhurvey DK, Singh G. Association of ocular injuries in patients with head injury: A tertiary-level armed forces experience. Med J DY Patil Vidyapeeth. 2024;17(1):59-64. doi:10.4103/mjdrdypu.mjdrdypu_83_23
3. Maurya RP, Srivastav T, Singh VP, Mishra CP, Al-Mujaini A. The epidemiology of ocular trauma in Northern India: a teaching hospital study. Oman J Ophthalmol. 2019; 12(2):78-83. doi:10.4103/ojo.OJO_149_2018.
4. Georgouli T, Pountos I, Chang BY, Giannoudis PV. Prevalence of ocular and orbital injuries in polytrauma patients. Eur J Trauma Emerg Surg. 2011;37 (2):135-40. doi:10.1007/s00068-010-0029-6.
5. Das S, Bhuyan D, Addya S. Ocular morbidity following road traffic accidents: a retrospective analysis. Int J Community Med Public Health. 2017;4(4):968.
6. Saxena R, Sinha R, Purohit A, Dada T, Vajpayee RB, Azad RV. Pattern of pediatric ocular trauma in India. Indian J Pediatr. 2002;69(10):863-7. doi:10.1007/BF02723708.
7. Khatry SK, Lewis AE, Schein OD, et al. The epidemiology of ocular trauma in Nepal. Br J Ophthalmol. 2004;88(4):456-60.
8. Maurya RP, Singh VP, Mishra CP, Jain P, Kumar A, Prajapat MK, et al. Eye injuries in motor vehicle accidents: epidemiology, spectrum of injury and analysis of risk factors. IP Int J Ocul Oncol Oculoplasty. 2021;7(1):30-39.
9. Das S, Bhuyan D, Addya S. Ocular morbidity following road traffic accidents: a retrospective analysis. Int J Community Med Public Health. 2017;4(4):968. doi:10.18203/2394-6040.ijcmph20170983.
10. Gagrai J, et al. A study on the pattern of ocular injuries and their visual outcomes following road traffic accidents. Cureus. 2025;17(5):e83985. doi:10.7759/cureus.83985.
11. Lev Ari O, Shaked G, Michael T, Givon A, Bodas M, Tsumi E. Ocular injuries associated with two-wheeled electric transportation devices and motorcycle accidents. Sci Rep. 2022;12(1):20546. doi:10.1038/s41598-022-23860-z.
12. Marudhamuthu E, Sivakumar N, Kumaravel T. Study of ocular injuries in road traffic accident patients. J Evol Med Dent Sci. 2017;6(41):3219-22. doi:10.14260/jemds/2017/697.
13. Kumar J, Singh VP, Chaubey P, Kumar V. Ocular injuries in road traffic accidents. J Dent Med Sci. 2017;16(11):55-60.
14. Reddy DC, et al. Manifestations of ocular injuries in road traffic accidents. Acta Sci Ophthalmol. 2021;4(4):168-71.
15. Farooq F, Quraishy MM, Hassan MU, Hussain M, Mushtaq F. Pattern and magnitude of ocular trauma sustained in road traffic accidents: a trauma centre study. Pak J Ophthalmol. 2022;38(4):266-70. doi:10.36351/pjo.v38i4.1441.
16. Menon L, Mani S, Mathew A. The prevalence of ocular manifestations in road traffic accidents treated at a rural tertiary care hospital in South India: a cross-sectional study. Int J Res Med Sci. 2017;5:4380-4.

17. Shetgar AC, Mallkarjunaswamy DL, Padmaraj JM, Ramanna D, Venkatesh RH. Ocular injuries following road traffic accidents: a hospital based case series study. Indian J Clin Exp Ophthalmol. 2020;6(2):266-9.

18. Kumarasamy R, Velpandian U, Anandan H. Visual outcome in ocular injuries in road traffic accident. Int J Sci Stud. 2016;4(5):151-3.

Histopathological Spectrum of Orofacial Lesions: Insights from a Tertiary Hospital in Western Nepal

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ABSTRACT

Introduction: Oral and maxillofacial region is a composite anatomical area where variety of lesions occurs ranging from congenital, non-neoplastic, precancerous lesions and cancers. Mostly these are asymptomatic and share common characteristic on clinical examination. Histopathology serves as an important aid in making correct diagnosis for proper patient management. **Aims:** To Study the histopathological spectrum of oro- facial lesions and their association with socio- demographic and behavioral factors. **Methods:** This is a hospital based cross sectional study done at Department of Pathology, Nepalgunj Medical College, Kohalpur. It included 75 biopsies from department of Oral and maxillofacial surgery. Samples were processed as per standard protocol, stained with Hematoxylin and Eosin stain and histopathological diagnosis was established for each of them. Data were entered in Excel-sheet and analysis was done using SPSS 25.0 software. **Results:** Age of patients ranged from 10 to 75 years, with a higher proportion of males (62%). Buccal cavity was the commonest site for biopsy, followed by palate and mandible. Among the 29 benign tumors identified, the most frequently encountered lesions were fibroma, Ameloblastoma, and various jaw cysts. A total of 23 malignant cases were recorded, with squamous cell carcinoma accounting for 19 of them. Tobacco use in any form demonstrated a statistically significant association with cancers of the oral cavity. **Conclusion:** Buccal cavity was the most commonly involved site involved in our study with benign tumors being more frequent than malignant ones. Squamous cell carcinoma was the commonest malignancy. Histopathology is vital for accurate diagnosis and to implement appropriate interventions for improved patient outcomes.

Keywords: Biopsy, Oral Cavity, Smoking, Squamous Cell Carcinoma, Tumor

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INTRODUCTION

Oral and maxillofacial region is a composite anatomical area which consists of the oral cavity and its adjoining tissues.[1] This region has complex and diverse relationship of the various structures within the head and neck region and includes the jaws, teeth, salivary glands, temporomandibular joint, facial muscles, and orofacial skin.[1,2] Lesions arising here are relatively common in all age groups and in both sexes. They span a wide pathological spectrum- from benign and premalignant conditions to overt malignancies, along with numerous congenital and acquired disorders.[3] Oral Cavity lesions are mostly asymptomatic and a large number of diseases share a common morphology on clinical examination.[4] Histopathology has been established as a gold standard technique in establishing a diagnosis and thus helps clinicians in deciding the therapeutic modalities.[5] As a tertiary care center, our institution routinely performs biopsies of orofacial lesions. However, no prior study of such type has been conducted in our institution. Therefore, this study aims to evaluate the histological spectrum of biopsy samples submitted to our laboratory by the department of Oral and Maxillofacial Surgery.

METHODS

A hospital Based Cross sectional study was conducted in the department of pathology, Nepalgunj Medical College Teaching Hospital, Kohalpur over a period of One year (September 2024

to August 2025) after obtaining clearance from the IRC, NGMC (IRC ref number-22/081-082)

Inclusion criteria: Patients with biopsy samples from the Department of Oral and Maxillofacial Surgery who provided informed consent.

Exclusion criteria: Non-diagnostic samples and patients who did not provide consent.

A total of 75 cases were taken for the study which was calculated based on the study done by Gaire et al which showed the prevalence of non-neoplastic oral lesions as 45% (Gaire et al).[2] Biopsy specimen were processed and stained with routine H and E stain as per the standard protocols. Slides were screened and reporting done by expert pathologists. A proforma was filled with relevant data. An attempt to establish the association of tobacco consumption with oral cancers was made in this study. For this, patient who have ever consumed tobacoo in any forms (Tobacoo chewing, cigaratte smoking, bidi, tamakhu, gutkha) were taken into a group and the association of these with causation of oral cancer was studied. Data were analyzed using Microsoft excel 2010 and standard statistical software SPSS 25.0.

RESULTS

The age of patients in our study ranged from 10 to 75 years, with mean age of 41.25 ± 15.8 years. Among the 75 cases, 29 (38.7%) were females and 46 (61.3%) were males with ratio M:F 1.58:1. Majority of biopsies were obtained from buccal cavity 25(34%), followed by palate and mandible, each accounting for 9(15%). Less frequently involved sites included gingivobuccal sulcus, maxilla, lip and the tongue (Figure 1).

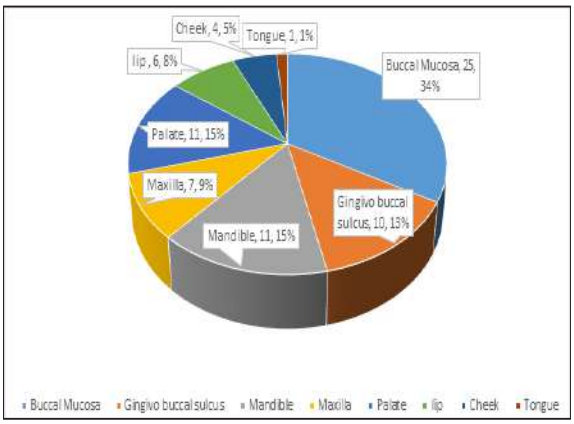


Figure 1: Site of Lesions

Majority of the lesions subjected to biopsy were neoplastic in nature 52(69%) while non-neoplastic ones accounted for 23 (31%) cases. Of the neoplastic ones, 29 (55%) were benign and 23 (45%) were malignant in nature. (Figure 2)

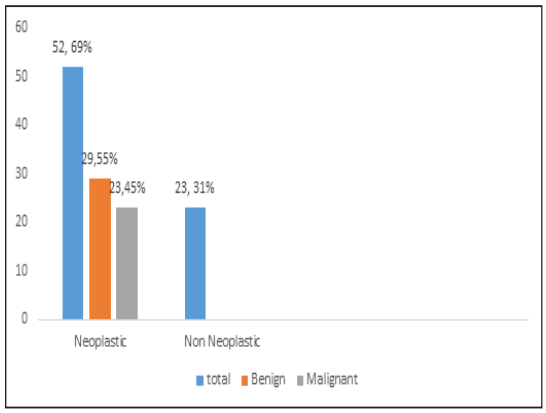


Figure 2: Nature of Lesions

Malignant neoplasms were further sub categorized into different types, of which squamous cell carcinoma was the commonest type occurring in the oral cavity accounting for 19 cases (83%). Two (8%) cases were of mucoepidermoid carcinoma, one case (4%) each were of Adenoid Cystic Carcinoma and Sarcoma. (figure 3)

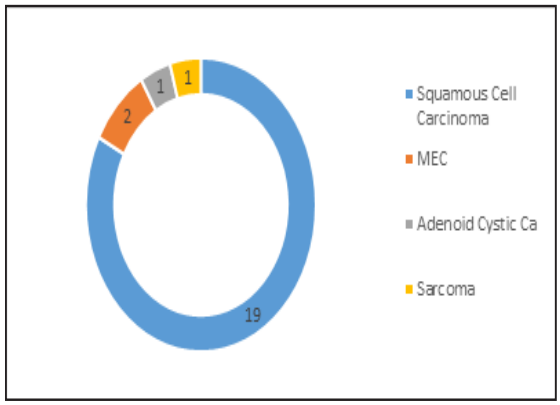
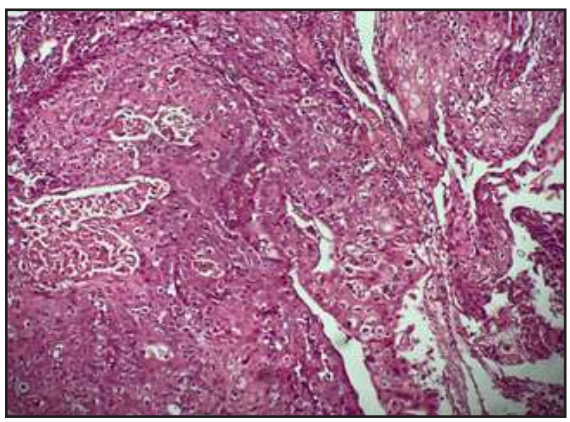


Figure 3: Types of Malignant Lesions



Picture 1: Squamous Cell Carcinoma, H & E, 400x, showing nests of atypical squamous cells

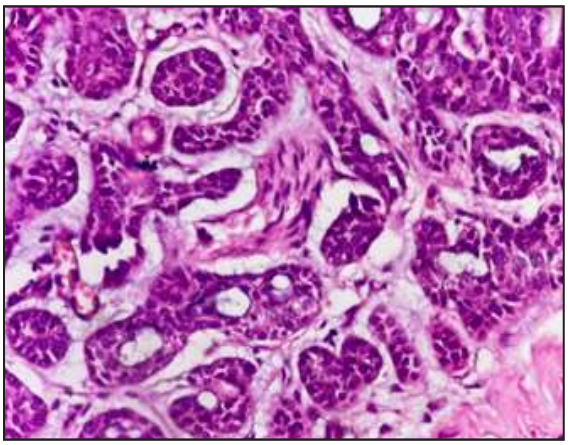


Photo 2: Adenoid Cystic Carcinoma, H &E stain, 400x, showing perineural invasion

Among benign lesions: fibroma, ameloblastoma, dentigerous cyst, radicular cyst and pleomorphic adenomas were common entities. A single case each, of myxoma, neurofibroma and EIC were also diagnosed in our study. (Figure 4)

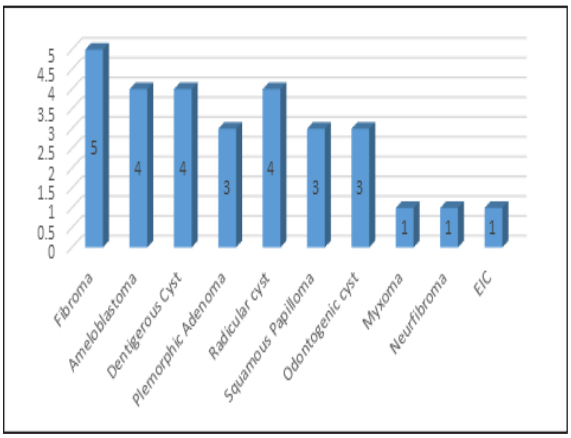


Figure 4: Neoplastic - Benign Lesions

Non Neoplastic lesions were seen in 23 cases (31%). This group comprised of 5 cases (21%) Leucoplakia, 4 cases (17%) of oral submucosal fibrosis, 3 cases (13%) each of pyogenic granuloma and Oral Tuberculosis. Other cases, which comprised of single case, were grouped together into "others" category. This group included cases of lichen planus, traumatic tongue ulcer with eosinophilia, pseudoepitheliomatous hyperplasia etc. (figure 5)

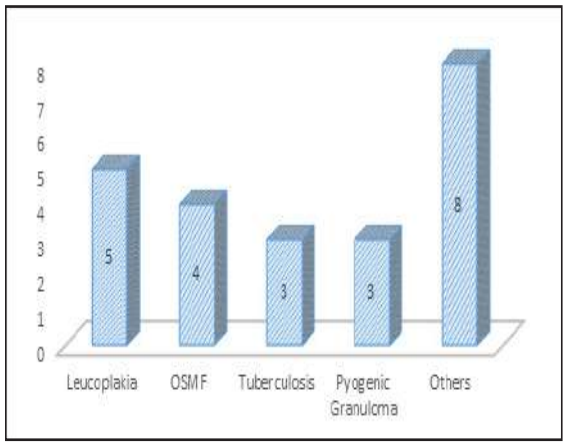
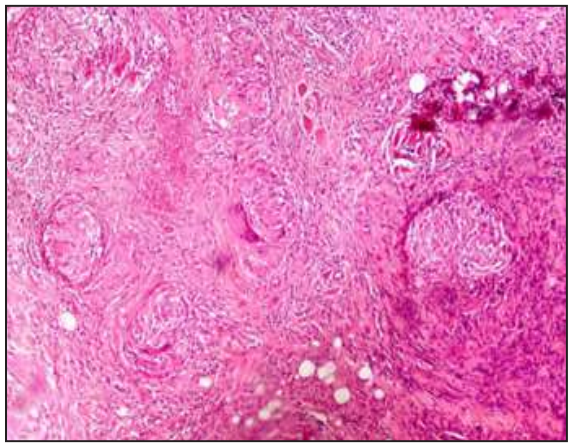


Figure 5: Non -Neoplastic Lesions



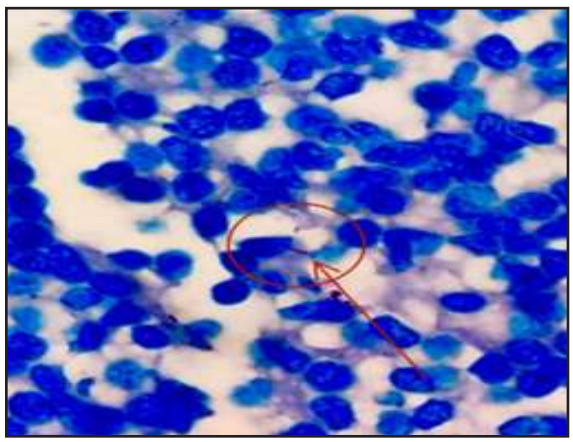


Photo 3: Tuberculosis of Buccal Mucosa, H & E, 400x with corresponding ZN Stain demonstrating TB bacilli

The study population was divided into two groups: Never tobacco consumers and Ever tobacco consumers, who have consumed tobacco in any forms viz tobacoo chewing, cigaratte smoking, bidi, tamakhu, gutkha. Two out of 35 never tobacco consumers were oral cancer positive whereas 21 of the 40 ever tobacco consumers were cancer positive and the association of tobacco with oral cancer was statistically significant with a P value of 0.001. (Table I

Characteristic	Categories	Malignancy		P value	Remark
		Positive	Negative		
Tobacco	Never consumer	2	33	0.001	S *
	Ever consumer	21	19		

**** Fisher's Exact test applied***

Table I: Association of tobacco with Oral cancer

DISCUSSION

A total 75 cases of biopsy specimen were studied that were submitted from the Department of Oral and maxillofacial surgery during the study period. All types of biopsies ranging from small incisonal biopsies, wedge resections as well as complex resection biopsies were included in our study. With a very well equipped department and provision of two oral and maxillofacial surgeons, NGMC serves as a referral site for complex surgeries of the Orofacial region. Moreover, the health insurance program that has been instituted by the Government of Nepal in this institution, has led to overall increase in the burden of cases and complex surgeries are being done on a routine basis.

Mean age of the patients in our study was 41 years, age of the patients ranging from 10 to 75 years. Majority of the cases were male (61%) and female comprised (39%) of the study population. Study done by Gaire et al in TUTH showed mean age of the patients as 44 years and study by Sakpal RY et al shows male: female ration of 1.8:1, results of which are similar to our study.[2,3]

Our study showed Buccal Mucosa as the most common site of lesion followed by mandible and palate. Studies done by Sakpal RY et al, Mishra V et al, Karki A et al, Mehta NV et al, Gupta et al and Bajracharya D et al also showed Buccal mucosa as the commonest site for lesions in oro-facial region.[3,6-10]

Neoplastic lesions accounted for 69% of the cases, exceeding the 31% represented by non-neoplastic lesions. Benign neoplastic lesions of the orofacial region were the most frequently observed, with 29 cases, followed by 23 cases each of malignant neoplasms and non-neoplastic lesions. This finding aligns with the results reported by Mehta NV et al, Agrawal R et al, and Bajracharya D et al.[8-10]

Of the malignant lesion detected by histopathology, 83%(19 cases) were Squamous cell Carcinoma. Two cases of Mucoepidermoid carcinoma and one each cases of Adenoid Cystic Carcinoma and Sarcoma were also detected in the study. Most of the studies done globally show primary Squamous cell carcinoma as the most common malignant lesion detected in the oral cavity. These SCC cases were seen in the buccal mucosa, gingivobucal sulcus, palate and lip. Salivary gland neoplasms (MEC and AdCC) arose from the minor salivary glands present in the oral cavity. A single case of sarcoma(fibrosarcoma) arose from the mesenchymal tissue in the palate.[1-6,9,10]

Benign Neoplastic lesions formed the bulk of the lesions occuring in the orofacial lesion and the common entities were fibroma, Ameloblastoma, Pleomorphic Adneoma and odontogenic cysts of the jaw. These findings show near similar results compared with the studies done by Garibay F et al, Agrawal R et al, Kalavati CL et al.[11,12,13] Leucoplakia and oral submucosal fibrosis were the common premalignant lesions in our studies, as described in other studies as well.[6-11] These lesions are best described as precursor lesions to the development of Squamous cell Carcinoma however their exact nature as a neoplastic entity is not established. Hence they are grouped in non neoplastic category along with other non neoplastic infective and inflammatory lesions.

Three cases of oral Tuberculosis were diagnosed, all of which presented with ulceration of the mucosa. Though an uncommon entity in oral cavity, studies have sporadically found few cases of TB of oral cavity across the globe.[11,14] Correct diagnosis of TB of oral cavity can only be established by histopathological evaluation of the lesion so that the patient can be enrolled to ATT with 100% therapeutic benefit.

An attempt to establish the association of tobacco consumption with oral cancers was made in this study. For this, patient who have ever consumed tobacoo in any forms (Tobacoo chewing, cigaratte smoking, bidi, tamakhu, gutkha) were taken into a group and the association of these with causation of oral cancer was studied. Statistically significant association of tobacoo was seen with oral cavity cancers in our study (p value- 0.001). Our study finding is in concordance to the globally established and postulated findings of tobacoo in causation of oral mucosal cancers. Results similar to ours were seen in studies done by Pudasaini et al, Ali M et al, Pathak A et al and Mathur A et al which showed strong association of tobacco in causing oral cavity cancers.[15-21]

LIMITATIONS

The major limitation of our study is the relatively small sample size, which may limit the representativeness and reproducibility of the findings. A larger-scale study would offer a more comprehensive understanding of the lesion patterns. Furthermore, oral lesion cases managed by the ENT department were not included, resulting in the omission of some relevant biopsy cases from the study.

CONCLUSION

Oral cavity lesions exhibit considerable heterogeneity, including a wide array of both neoplastic and non-neoplastic conditions. Our study showed buccal cavity as the commonest site of oral lesions with benign tumors accounting for most of the cases. Squamous cell carcinoma was commonest malignancy seen in our study. The distribution pattern observed in our center closely mirrors those documented in the most commonest malignancy previous studies. Histopathological evaluation is vital for accurate diagnosis, enabling clinicians to make informed decisions and implement appropriate interventions for improved patient outcomes.

REFRENCES

1. Leigh O, Akinyamoju A, Ogun G, Okoje V. Spectrum of oral and maxillofacial tissue biopsies at the foremost tertiary institution in The Gambia: A retrospective review. J West African Coll Surg. 2023;13(3):1.
2. Gaire D, Pant AD, Maharjan D, Manandhar U. Spectrum of Oral Cavity Lesions and its Clinico-Histopathological Correlation. Nepal J Heal Sci. 2021;1(2):42–7.
3. Sakpal RY, Warpe BM, Joshi-Warpe S. Spectrum of Histopathological Diagnosis of Oral Lesions in a Tertiary Care Hospital at Miraj in Maharashtra State, India. Natl J Lab Med. 2021;1–6.
4. Shrestha B, Subedi S, Poudel S, Ranabhat S, Gurung G. Histopathological Spectrum of Oral Mucosal Lesions in a Tertiary Care Hospital. J Nepal Health Res Counc. 2021;19(3):424–9.
5. Ahern J, Toner M, O'Regan E, Nunn J. The spectrum of histological findings in oral biopsies. Ir Med J. 2019;112(10):1-8
6. Misra V, Singh PA, Lal N, Agarwal P, Singh M. Changing pattern of oral cavity lesions and personal habits over a decade: Hospital based record analysis from Allahabad. Indian J Community Med 2009;34(4):321–5.
7. Karki A, Manandhar V, Maharjan R, Maharjan A. Oral Mucosal Lesions in Patients Attending Dermatology Outpatient Department of a Tertiary Care Center: A Descriptive Cross-sectional Study. J Nepal Med Assoc. 2024;62(274):387–91.
8. Bajracharya D, Gupta S, Ojha B, Baral R. Prevalence of oral mucosal lesions in a tertiary care Dental Hospital of Kathmandu. J Nepal Med Assoc 2017;56(207):362–6.
9. Gupta I, Rani R, Suri J. Histopathological spectrum of oral cavity lesions – A tertiary care experience. Indian J Pathol Oncol. 2021;8(3):364–8.
10. Nikunj V. Mehta, Kalpana K. Dave, R.N.Gonsai, H.M.Goswami, Purvi S. Patel TBK. Histopathological study of oral cavity lesions: a study on 100 cases. ijcrr. 2013;05:110–6.
11. Agrawal R, Chauhan A, Kumar P. Spectrum of oral lesions in a tertiary care hospital. J Clin Diagnostic Res. 2015;19(6):11–3.
12. Kalavathi L, Chaitanya K, Venkata V. A bird's-eye view of pathologist over diagnostic confusion of oral cavity lesions. J Oral Maxillofac Pathol. 2023;27(2):266–74.
13. Fierro-Garibay C, Almendros-Marqués N, Berini-Aytés L, Gay-Escoda C. Prevalence of biopsied oral lesions in a Department of Oral Surgery (2007 - 2009). J Clin Exp Dent. 2011;3(2):2–6.
14. Krawiecka E, Szponar E. Review paper Tuberculosis of the oral cavity: an uncommon but still a live issue. Adv Dermatology Allergol. 2015;4:302–6.
15. Pudasaini S, Baral R. Oral cavity lesions: A study of 21 cases. J Pathol Nepal. 2011;1(1):49–51.
16. Ali M, Sundaram D. Biopsied oral soft tissue lesions in Kuwait: A six-year retrospective analysis. Med Princ Pract. 2012;21(6):569–75.
17. Pathak A, Verma S. Association of Oral Cancer With Consumption of Tobacco , Smoking and Alcohol : a Prospective Cohort Study of. 2023;5(2):1420–8.
18. Mathur A, Jain M, Shiva M, Navlakha M, Kulkarni S. Tobacco Habits and Risk of Oral Cancer: A Retrospective Study in India. Iran J Blood Cancer |. 2009;1(3):111–6.
19. Gray JL, Al Maghlouth A, Al Hussain H, Al Sheef M. Impact of oral and oropharyngeal cancer diagnosis on smoking cessation patients and cohabiting smokers. Tob Induc Dis. 2019;17(11):1–10.
20. Schmidt BL, Dierks EJ, Homer L, Potter B. Tobacco smoking history and presentation of oral squamous cell carcinoma. J Oral Maxillofac Surg. 2004 Sep 1;62(9):1055–8.
21. Jiang X, Wu J, Wang J, Huang R. Tobacco and oral squamous cell carc

Study of Renal Function Test in Pregnant Women with Preeclampsia: A Hospital Based Cross-Sectional Study

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ABSTRACT

Introduction: The multifactorial disease known as preeclampsia presents significant hazards throughout pregnancy and often results in renal impairment. **Aims:** To evaluate renal function tests in pregnant women with preeclampsia compared to normotensive pregnant women. **Methods:** A Hospital based comparative cross-sectional study was carried out for a period of six months from December 2024 to June 2025, involving 80 participants (40 with Pre-eclamptic pregnant women and 40 normotensive pregnant women) aged 18 to 45 years at gestational ages after 30 weeks. Serum urea, creatinine, and uric acid levels were estimated, and statistical analysis was performed using SPSS version 25. **Results:** The median age was 27.5 years and 25 years in the pre-eclamptic and normotensive group respectively. The pre-eclamptic group manifested significant increases in serum urea (21.7 mg/dl vs. 15.8 mg/dl, $p < 0.001$), creatinine (0.70 mg/dl vs. 0.62 mg/dl, $p = 0.039$), and uric acid (7.08 mg/dl vs. 5.5 mg/dl, $p < 0.001$) compared to normotensive group. The pre-eclamptic group systolic and diastolic blood pressure readings were 150.0 mmHg and 100.0 mmHg respectively which vary significantly compared to the normotensive group measurements which were 110.0 mmHg and 70.0 mmHg. **Conclusion:** Our study found that preeclampsia had a negative impact on renal function, as evidenced by elevated levels of serum urea, creatinine and uric acid. These results highlight the importance of monitoring renal function in pregnant women with hypertension since these measurements are crucial indicators of renal impairment in preeclampsia.

***Keywords*:** Creatinine, Preeclampsia, Urea & Uric acid

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INTRODUCTION

Preeclampsia (PE) and eclampsia is a multifactorial syndrome which is a severe complication during pregnancy.[1] Globally, World Health Organization (WHO), reported the incidence of PE ranges between 2% to 10% of pregnancies[2] and is the second leading cause of direct maternal and fetal deaths.[3] PE complicates nearly 3% of pregnancies[4] and the incidence of PE is reported to be 2.3% and 8-10% in United states and India respectively.[5,6] Whereas in Nepal, according to the result of a Meta-Analysis the prevalence of PE and eclampsia were 2.6% and 0.5%.[7] PE is a multisystem disorder causing damage to many maternal organs mainly the kidney and liver.[8] PE causes broad endothelial dysfunction, which has a special impact on the renal system.[9] In normal pregnancies, physiologic vasodilation increases renal plasma flow and glomerular filtration rate (GFR) by 50%, lowering blood creatinine and urea levels. At the same time, in PE these adaptations are

compromised and glomerular endotheliosis, a hallmark lesion, results in decreased renal perfusion, decreased GFR, and proteinuria.[9] According to some studies, serum urea, creatinine and uric acid were significantly increased in pre-eclamptic women.[8,10] However, in another study, serum urea and serum creatinine were increased but were insignificant in PE.[6] Since the alteration of renal function in PE is yet unclear and there is a lack of similar studies from the western region of Nepal. So, the current study was conducted with an aim to evaluate alterations of renal function tests in pregnant women with preeclampsia and compare them with those of normotensive pregnant women in a tertiary care center at Kohalpur.

METHODS

A hospital-based comparative cross-sectional study was conducted at the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynaecology of Nepalgunj Medical College Teaching Hospital (NGMCTH), Kohalpur, for a period of six months from December 2024 to June 2025. Ethical approval for the study was obtained from the Institutional Review Committee of NGMCTH (Ref: 35/081-082 dated December 2024). Prior to enrollment oral and written consent were obtained from all the participants. The participants were pregnant women, being primigravida/ multigravida, with an age range of 18 to 45 years and gestational age greater than 30 weeks, admitted to the labor room, Department of Obstetrics and Gynaecology, NGMCTH were included in our study. Participants with proteinuria caused by conditions other than PE, participants with pre-existing hypertension, and participants who refused consent were excluded from the study. Using the following formula, a convenient sampling technique was employed to determine the sample size.[7]

Prevalence of PE (P): 2.6%[7]

q = 100 - p

Margin of error (d)= 5

Z= 1.96 at confidence interval 95%

Sample size (n) = Z^2pq/d^2

= $(1.96)^2$ (2.6) (97.4) / $(5)^2$

= 3.8 x 2.6 x 97.4 / 25

=39

The study participants were divided into two groups: pre-eclamptic group and normotensive group. A total of 80 participants were enrolled in the study. Out of which 40 pregnant women clinically diagnosed with preeclampsia were enrolled into the pre-eclamptic group and an equal number of age and gestational age-matched normotensive pregnant women were enrolled into the normotensive group. PE is diagnosed as gestational hypertension (systolic blood pressure > 140 mmHg and/or diastolic blood pressure > 90 mmHg) measured on two occasions separated by at least 6 hours and proteinuria (≥ 300 mg/ 24-hour urine or urine protein/ creatinine ratio (PCR) ≥ 0.3 mg/mg or qualitative >1+) or other maternal organ dysfunction after 20 weeks of gestation.[11] Three ml of venous blood was collected in a gel tube under sterile conditions and was subjected to centrifugation for 5 minutes at 3500 revolutions per minute (RPM) to separate serum, which was processed for serum urea, creatinine and uric acid by Mindray BS 430 with wet chemistry principle. Serum urea, creatinine and uric acid were measured by urease-glutamate Dehydrogenase, UV method, Sarcosine-oxidase method and Uricase-peroxidase method, respectively. At the same time, urine protein was detected by using the urine dipstick method.

Statistical analysis: Data were inputted into a Microsoft Excel spreadsheet and analysed using the Statistical Package for the Social Sciences (SPSS) version 25. The normality of the data was judged using the Shapiro-Wilk test. Since the data of both pre-eclamptic and normotensive group were not normally distributed. So, the analysis was done using the non-parametric tests. Results were expressed as a median with an interquartile range. The mean ranks between the pre-eclamptic and normotensive groups was compared using the Maan- Whitney U test. A p-value <0.05 was considered statistically significant.

RESULTS

In the current study, the majority of participants were ≤25 years in both the pre-eclamptic and normotensive groups.

Parameters	Median (Interquartile range)		
	Total Participants	Pre-eclamptic group	Normotensive group
Age (years)	27.0 (22.2 – 32.0)	27.5 (23.25 – 32.0)	25.0 (22.0 – 31.75)
Urea (mg/dl)	17.7 (15.0 – 25.0)	21.7 (16.29 – 28.10)	15.8 (14.6 – 18.3)
Creatinine (mg/dl)	0.64 (0.57 – 0.76)	0.70 (0.58 – 0.97)	0.62 (0.51 – 0.70)
Uric acid (mg/dl)	6.15 (5.40) – 7.37	7.08 (6.05 – 7.96)	5.5 (4.62 – 6.42)
POG (weeks)	37.0 (34.25 – 39.0)	36.5 (33.0 – 39.0)	37.0 (35.25 – 39.0)
SBP (mm Hg)	130.0 (110.0 – 150.0)	150.0 (140.0 – 160.0)	110.0 (100 – 120.0)
DBP (mm Hg)	90.0 (70.0 – 100.0)	100.0 (100.0 – 107.5)	70.0 (60.0 -80.0)

POG: Period of gestation, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Table I: Baseline characteristics of the Pre-eclamptic and Normotensive group

Table I shows that serum urea, creatinine, uric acid, SBP and DBP increase in the pre-eclamptic group compared to the normotensive group.

	Age group (years)	n (%)
Pre-eclamptic group	≤25	17 (42.5)
	26 - 30	9 (22.5)
	31 - 35	11 (27.5)
	36 - 40	3 (7.5)
Normotensive group	≤25	21 (52.5)
	26 - 30	7 (17.5)
	31 - 35	8 (20.0)
	36 - 40	4 (10.0)

n: Number of participants, %: percentage of participants

Table II: Age distribution of Pre-eclamptic and Normotensive groups

Table II shows that the majority of participants in the Pre-eclamptic and Normotensive groups were ≤25 years, accounting for roughly 42.5% and 52.5%, respectively.

Parameters	Median (Interquartile range)		
	Pre-eclamptic group	Normotensive group	P value[a]
Age (years)	27.5 (23.25 – 32.0)	25.0 (22.0 – 31.75)	0.542
Urea (mg/dl)	21.7 (16.29 – 28.10)	15.8 (14.6 – 18.3)	0.001
Creatinine (mg/dl)	0.70 (0.58 – 0.97)	0.62 (0.51 – 0.70)	0.039
Uric acid (mg/dl)	7.08 (6.05 – 7.96)	5.5 (4.62 – 6.42)	0.001
POG (weeks)	36.5 (33.0 – 39.0)	37.0 (35.25 – 39.0)	0.317
SBP (mm Hg)	150.0 (140.0 – 160.0)	110.0 (100 – 120.0)	0.001
DBP (mm Hg)	100.0 (100.0 – 107.5)	70.0 (60.0 -80.0)	0.001

POG: Period of gestation, SBP: Systolic blood pressure, DBP: Diastolic blood pressure a:Mann-Whitney U test, P < 0.05 was considered statistically significant and was indicated in bold type

Table III: Comparison of parameters between the pre-eclamptic group and normotensive group

Table III shows that serum urea, creatinine, uric acid, SBP and DBP were significantly increased in the pre-eclamptic group compared to the normotensive group.

DISCUSSION

Preeclampsia-related renal impairment has been linked to several factors, most likely glomerular endotheliosis, hemodynamic alterations, and podocyte destruction.[12] Although the renal impairment is typically not noticeable throughout the prenatal period, pre-eclamptic patients are more likely to experience it, and if renal impairment is not identified promptly, it may develop into renal failure and eventually cause other vascular diseases.[12] So, the current study was conducted to study the renal function test in pregnant women with preeclampsia visiting tertiary care hospital in Western Nepal.

The present study enrolled 80 participants, including 40 pregnant women diagnosed with preeclampsia into the preeclamptic group and an equal number of normotensive healthy pregnant women into the normotensive group. The median age of participants was 27.5 and 25 years in the pre-eclamptic and normotensive groups respectively and the majority of participants belonged to the age group ≤25 years in both the pre-eclamptic (n=17) and normotensive groups (n=21).

The present study represents that serum urea was increased in the pre-eclamptic group [21.7 (16.29 - 28.10)] compared to the normotensive group [15.8 (14.6 – 18.3)] and was statistically significant (p = 0.001). The findings of our study were in line with the study done by Abdelrahman R et al[10] and Hamed S et al.[13] In contrast to our findings, a study done by Manjareeka M et al[14] reported that serum urea was slightly increased in preeclamptic women (28.07 ± 4.97) compared to normotensive pregnant women (26.46 ± 3.55) but was statistically insignificant (p = 0.068). Our current study also showed that serum creatinine was increased in the pre-eclamptic group [0.70 (0.58 - 0.97)] compared to the normotensive group [0.62 (0.51- 0.70)] and was statistically significant (p = 0.03). The findings in our study were in accordance with the study done by Ambad DRS et al[6] while the study done by Hamed S et al[13] reported that serum creatinine was increased in pre-ecalmptic women (0.84 ± 0.34) in comparision to normotensive pregnant women (0.79 ± 0.31) but was insignificant (p = 0.508). Placental tissue from preeclamptic women has decreased monoamine oxidase activity and increased serotonin levels than placental tissue from normal pregnant women. These factors decrease renal perfusion, lowering GFR in preeclamptic women compared to normal pregnant women, which in turn leads to increased urea and creatinine levels in blood.[13] In addition, the micro-angiopathic hemolysis generated by maternal endothelial dysfunction also contributes to the increase in blood urea levels by increasing the synthesis of urea.[13]

In our study, serum uric acid was higher in pre-eclamptic women [7.08 (6.05 – 7.96)] than in normotensive pregnant women [5.5 (4.62 – 6.42)] and the difference was statistically significant (p = 0.001). The findings of our study were in agreement with the study done by Jumaah M et al[8], Niraula

A et al[12] and Dhungana A et al.[15] A study by Adebisi OO et al[9] reported that serum uric acid was increased in the pre-eclamptic group (1.60 ± 0.49) compared to the control group (1.29 ± 0.20) but the difference was not significant (p = 0.135). During normal pregnancy, uric acid concentration decreases in blood by 25% and is due to increased renal plasma flow and GFR causing an increased in uric acid clearance from 6 to 12 mL/min to 12 to 20 mL/min while in PE these adjustment are compromised causing hyperuricemia in PE.[9,15] In PE, hyperuricemia is multifaceted and is caused by increased reabsorption of uric acid and decreased renal excretion, as well as increased oxidative stress from placental ischemia and increased xanthine oxidase activity since uric acid is a byproduct of purine catabolism.[6] The small sample size of our hospital-based study is a limitation. Clarifying the renal function alteration can be made easier with a larger sample size and a general population investigation. It would be more helpful if urinary measurements of uric acid, creatinine, and various oxidative stress-inducing substances were performed.

CONCLUSION

Our current study concludes that renal function is negatively affected by preeclampsia, highlighting the necessity to evaluate renal function tests, particularly serum uric acid, for all pregnant women with high blood pressure, as it is the first indicator of how renal function is influenced in preeclamptic women.

REFERENCES

1. Meena R, Pachori P, Chaudhary S, et al. Level of serum uric acid in patients with preeclampsia compared to controls and its relation to feto-maternal outcome. Int J Reprod Contracept Obstet Gynecol. 2019;8:2471-4.
2. Khan B, Allah Yar R, Khakwani A, et al. Preeclampsia Incidence and Its Maternal and Neonatal Outcomes With Associated Risk Factors. Cureus. 2022; 14(11): e31143. DOI 10.7759/cureus.31143
3. Agrawal S, Walia GK. Prevalence and Risk Factors for Symptoms Suggestive of Pre-Eclampsia in Indian Women. J Womens Health, Issues Care. 2014; 3:(6): 2-9
4. Ariana Traub, Apoorva Sharma, Maria Carolina Gongora, Hypertensive Disorders of Pregnancy: A Literature Review – Pathophysiology, Current Management, Future Perspectives, and Healthcare Disparities, US Cardiology Review. 2024;18:e03.
5. Abalos E, Cuesta C, Grosso AL, et al. Global and regional estimates of preeclampsia and eclampsia: a systematic review. Eur J Obstet Gynecol Reprod Biol. 2013 Sep;170(1):1-7. doi: 10.1016/j.ejogrb.2013.05.005. Epub 2013 Jun 7. PMID: 23746796.
6. Ambad DRS, Dhok DA. The role of serum urea, creatinine, uric acid in diagnosis of pre-eclampsia and eclampsia. Int J Med Biomed Studies. 2019; 3(9):77-80
7. Shrestha DB, Budhathoki P, Malbul K, et al. Prevalence, Risk Factors and Outcome of Pregnancy induced Hypertension in Nepal: A Meta-Analysis of Prevalence Studies. J Nepal Health Res Counc. 2021 Sep 6;19(2):221-29. doi: 10.33314/jnhrc.v19i2.3589. PMID: 34601508.
8. Jumaah M , Israa A. Estimation of uric acid ,urea, creatinine and creatinine clearance in the serum of preeclamptic women. Kerbala J Pharmaceutical Sci. 2012;4(4): 183-9.
9. Adebisi OO, Bakare AK, Vaughan AT, Faponle AE, Ahmed KA, Odeyemi A, Okunola OO. Kidney Function in Normotensive and Preeclamptic Pregnancies: A Comparative Cross-Sectional Study in Abeokuta, Nigeria. The Nigerian Health Journal 2025; 25(2): 816 – 23.
10. Abdelrahman, R. , Zaroog, M. , Abdalla, B. , Hamza, M. and Mohamed, M. Renal Function in Preeclamptics versus Normal Pregnant Women. Journal of Biosciences and Medicines, 202:10; 169-78. doi: 10.4236/jbm.2022.105015.
11. Brown MA, Magee LA, Kenny LC, Karumanchi SA, McCarthy FP, Saito S, Hall DR, Warren CE, Adoyi G, Ishaku S Hypertensive Disorders of Pregnancy: ISSHP Classification, Diagnosis, and Management Recommendations for International Practice. Hypertension. 2018;72:24–43
12. Niraula A, Lamsal M, Baral N. Cystatin-C a s a Marker for Renal Impairment in Preeclampsia. J Biomark. 2017; 2017: 7406959. doi: 10.1155/2017.;7406959.
13. Hamed S, Khalifa T, Mekal F, Ali M. Evaluation of Changes in Renal Function of Pregnant Women with Preeclampsia in Al-Jabal Al-Akhdar. Alq J Med App Sci. 2022;5(1):56-64.
14. Manjareeka M, Nanda S. Elevated levels of serum uric acid, creatinine or urea in preeclamptic women. Int J Med Sci Public Health. 2013 Jan 1;2(1):43-7.
15. Dhungana A, Bharati A, Manandhar R, Karki C. A comparative study of serum uric acid, glucose, calcium and magnesium in pre-eclampsia and normal pregnancy. Journal of Pathology of Nepal. 2017 Sep 1;7(2):1155-61

ORIGINAL ARTICLE

Bleeding Time in Different Blood Groups of the ABO System- A Descriptive Cross-Sectional Study

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ABSTRACT

Introduction: ABO blood grouping depends on the presence of specific antigens on red blood cell membranes: Type A, B, AB, and O have antigens a, b, both a and b, and neither a nor b, respectively. Bleeding time is the time between the puncture of vessels and to stoppage of bleeding. Certain disease seems more prevalent in specific ABO blood groups. Likewise, many studies found that blood group O and females exhibit a higher bleeding tendency. **Aims:** To study the bleeding time in different ABO blood groups and genders. **Method:** A descriptive cross-sectional study was conducted among 139 medical students of first and second year after Ethical approval from the Institutional Review Committee of Nepalgunj Medical College Teaching Hospital, during practical hours from June to October 2025. **Results:** Blood group O was the most common (n=63), followed by A and B (n=28 each), and AB (n=20). Among the ABO and Rhesus factors, O-positive was most prevalent (41%), followed by A-positive and B-positive (18% each), AB-positive (12.2%), O-negative (4.3%), and the least was A-negative, B-negative, and AB-negative (2.2%). We found more Rh-positive students (90.6%) than Rh-negative (9.4%). The mean bleeding time was 2.698 minutes, with a range of 1.5 to 6.5 minutes. Non-O blood group students (n=76) consisting of Type A, B, and AB, had a shorter bleeding time compared to the O blood group, which was statistically significant. Males (n=70, mean: 2.771±.8710) had a longer bleeding time than females (n=69, mean: 2.623±.8762). **Conclusion:** Blood group O was predominant among the ABO group, and with the Rhesus factor, O positive was predominant. Bleeding time was prolonged in the O blood group than in any other group. Bleeding time was prolonged among males than among females.

Keywords: ABO grouping, Bleeding time (BT), Gender, Non-O blood group

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INTRODUCTION

ABO blood grouping is based on the presence of an antigen on the surface of RBCs. Type A, B, AB, and O have the antigen a, b, both a and b, and neither of these, respectively, on the membrane; consequently, these typed individuals' plasma contains anti-B, anti-A, neither, or both anti-A and anti-B, respectively. [1-3] Bleeding time (BT) is the time from the onset of bleeding to the formation of a temporary hemostatic plug that stops bleeding. BT assesses the integrity of platelets.[4-6] Research consistently shows that Type O individuals exhibit a higher bleeding tendency due to significantly lower levels of von Willebrand factor (vWF).[7,8] Additionally, it has been noted that the absence of ABO antigens may expedite the clearance of vWF from plasma.[9] In contrast, a retrospective study done by B. Mahapatra and N. Mishra (2019) on 740 medical students found that BT was significantly higher in AB group than in persons with blood group O.[10] The relationship between blood groups and BT varies across populations, but it is clear that there is a notable lack of research on gender-based variations within blood groups, especially in the Nepalese context. The interaction between ABO group and gender regarding BT is still insufficiently explored. However, it is well-documented that gender differences in BT exist, with females typically exhibiting longer BT than males. [11-14]

Hence, this study was conducted to find out the predominant ABO blood group and its relation with BT, and also gender variation in BT.

METHODS

The descriptive cross-sectional study was conducted in the Department of Physiology, Nepalgunj Medical College, from June to October 2025 on 139 students, including male and female medical students, between the age group of 18 and 26 years. The blood group and bleeding time of the students were determined. Comparison of bleeding time between males and females was performed. Moreover, any difference in bleeding time between O and non-O blood groups was determined, as well as effect of the Rh factor on bleeding time was assessed. Information regarding the history of bleeding disorder and drug intake (NSAIDS) or any recent trauma, surgery, or ongoing infection was obtained through a questionnaire to the students, and categorized as the exclusion criteria. All students participated, as none of them fell under the exclusion criteria. Inclusion criteria include the healthy physically and mentally fitted students of MBBS and BDS between 18-26 years old, irrespective of gender.

Formula used for the calculation of sample size:

n = N / [1 + N × e^2] Where,

n = the sample size

N = Target population = 210

e = the margin error in the calculation, i.e., 5%

All the data were collected after approval from NGMC IRC (ref. 68/081-082) and determined during practical time in the physiology laboratory. Blood samples were collected by finger prick with a sterile lancet after cleaning the puncture site with spirit. The sample blood was mixed with anti-A, anti-B, and anti-D serum. Blood groups were determined on the basis of the presence or absence of agglutination. Agglutination was confirmed by observing under a low-power objective of a compound microscope.[15]

Bleeding Time was determined by Duke's Filter paper method. A deep skin puncture was made, and the length of the time required for bleeding to stop was recorded by blotting the drop of blood coming out of the incision every 30 seconds using blotting paper. Bleeding Time was calculated by multiplying the number of drops on the filter paper and 30 seconds. The normal bleeding time by Duke's Filter paper method is usually in the range of 1-5 minutes.[15]

Statistical analysis: One- way ANOVA and independent t-test were used in SPSS software to analyze the data.

RESULTS

The sample size of a total of 139 medical students was analyzed. There was an almost homogeneous age group (17-26 years) as all belonged to the first and second year MBBS students and the BDS students. Out of 139 students, 70 were male, and 69 were female (Fig. 1, Table IV). We found that blood group O was predominant (n=63), followed by an equal number of A and B (n=28), and least in the AB blood group (n=20) among students. The percentage distribution of ABO blood group was in order of O (45.3%)> A (20.1%) = B (20.1%)> AB (14.4%) [Table V]. With Rh factor, in ABO blood group, we found O+ blood group in a more predominant number(41%), followed by A+(18%), B+ (18%), AB+(12.2%), O-(4.3%), A-(2.2%), B-(2.2%) and AB-(2.2%), displayed in Table I.

The mean value BT was 2.698 min, with 1.5 min and 6.5 min as the lowest and highest BT values in the study, respectively. In our study, there were more Rh-positive students (90.6%) than Rh-negative students (9.4%). Table I and II shows the distribution of ABO blood groups and the Rh blood groups. There were more non-O blood groups than the O blood group among students [Table III].

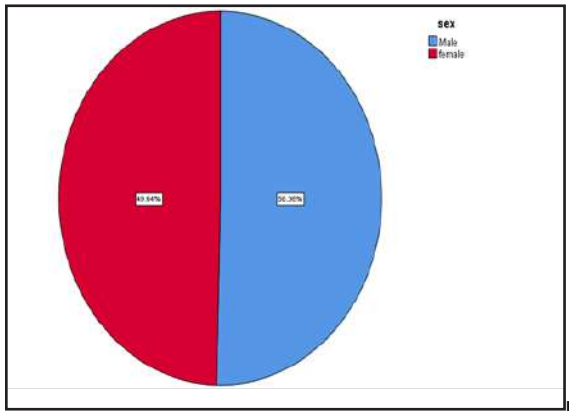

Figure 1: Gender distribution among students

Blood group	Frequency	Percent
A+	25	18.0
A-	3	2.2
B+	25	18.0
B-	3	2.2
AB+	17	12.2
AB-	3	2.2
O+	57	41.0
O-	6	4.3
Total	139	100.0

Table I: Showing the distribution and frequency of blood group among students

Rh blood group	Frequency	Percent
Negative	13	9.4
Positive	126	90.6
Total	139	100.0

Table II: Showing distribution and frequency of Rh blood group among students

Blood group	Frequency	Percent
Non-O	76	54.7
O	63	45.3
Total	139	100.0

Table III: Showing distribution and frequency of blood groups among students

Sex	Bleeding time				Significance
	≥ 3.5 min		< 3.5 min		
	No.	%	No.	%	
Male	15	10.8	55	39.6	F=0.003
Female	11	7.9	58	41.7	P=.319
Total	26	18.7	113	81.3	
	Male	Female	Total		
Number	70	69	139		
Mean ± SD	2.771± .8710	2.623± .8762			

Table IV: Gender wise distribution of bleeding time with an independent t-test

BT was prolonged more than or equal to 3.5 minutes among males (10.8 %) and among females (7.9%). Analyzing the data with an independent t-test did not show any significant difference BT of gender groups (p=.319) (Table IV).

Blood group	Bleeding time				Significance
	≥ 3.5 min		< 3.5 min		
	No.	%	No.	%	
A	4	2.9	24	17.3	
B	4	2.9	24	17.3	
AB	5	3.6	15	10.8	
O	13	9.4	50	36.0	
Total	26	18.7	113	81.3	F=2.295
Blood group	Number	Mean± SD			p =.081
A	28	2.482 ± .6869			
B	28	2.482 ± .8765			
AB	20	2.675 ± .6340			
O	63	2.897 ± .9762			
Total	139	2.698 ± .8736			

Table V: Distribution of bleeding time on various blood groups with one-way ANOVA analysis

BT prolonged more than or equal to 3.5 minutes among O (9.4%), followed by AB (3.6%), A (2.9%), and B (2.9%). Also from the table above, we found O-blood group students have prolonged BT than any other blood groups, whereas blood group A and B have equal but the least BT than others. Although there was a difference in BT of ABO blood groups but it was not found to be statistically significant (p=.081) (Table V).

Blood group	Bleeding time				Significance
	≥ 3.5 min		< 3.5 min		
	No.	%	No.	%	
O	13	9.4	50	36	
Non-O	13	9.4	63	45.3	F=1.638
Total	26	18.7	113	81.3	p = .014
	O	Non-O	Total		
number	63	76	139		
Mean ±SD	2.897± .9762	2.533± .7454			

Table VI: Distribution of bleeding time on O and Non-O blood group with independent t-test analysis

While considering O and Non-O blood groups, an equal number (9.4%) of both groups show BT prolonged more than or equal to 3.5 minutes. Analyzing with an independent t-test, data shows a statistically significant difference in BT of O and Non-O blood groups (p=.014) (Table VI).

In our study, we found that more than 90 percent of students were Rh positive; being disproportionate in size among Rh blood groups, it may be inappropriate to generalize the BT difference between the groups.

DISCUSSION

In this study conducted on 139 students, the blood group O showed predominance (45.3%) in the percentage distribution of ABO blood groups, followed by A (20.1%), B (20.1%), and least AB (14.4%). Asian trend of prevalence of blood groups O>B>A>AB, with predominant O and least AB blood group, has been reported in many research studies, which is similar to our study.[10,16,17,18] Contrary to our study, a different trend of prevalence of blood groups B>O>A>AB, with the B blood group having a predominance, was observed in various research studies.[1, 19, 20] In our study, we found that there is a significant difference in mean BT between O and non-O blood groups, with a longer time in O than in non-O blood groups. Comparatively, we found a larger number of O blood group students having BT more than 3.5 minutes than any other blood group student. Similar to ours, many studies in the past have found BT prolongation in blood group O, and they also described it due to less expression of vWF.[13,21,22,23] Also in our study, we found O-blood group students have prolonged BT, followed by AB, and then blood group A and B having the least and equal BT, but the difference was not statistically significant.

In contrast, one study BT is found to be prolonged in blood group B, followed by blood group O.[24] Moreover, there is one retrospective study done by B. Mahapatra and N. Mishra in 2019 on 740 medical students, where clotting time was prolonged in blood group B and bleeding time was prolonged in blood group AB than in other blood groups, and the difference was statistically significant.[10]

vWF is important for hemostasis, which plays an important role in platelet adhesion and aggregation. Research indicates that the ABO gene locus, chromosome number 9, accounts for about 30% of the genetic factors affecting vWF levels, suggesting that the ABO blood group influences plasma vWF. And some studies suggest that the absence of ABO antigens may expedite the clearance of vWF from plasma. Both studies explain the lower levels of vWF in individuals with blood group O. Therefore, one can say that a lack of A and B antigens may lead to lower plasma vWF levels, explaining the increased bleeding tendencies associated with O blood groups.[9,25]

We found that in our study mean BT of males is greater than females, but it is not statistically significant; a larger number of male has BT more than 3.5 min compared to females. One study shows prolonged BT in males than females similar to us, but their difference was statistically significant, unlike ours.[24] Also, a similar result was found in a study done by Benjamin and Bagavad showing BT prolonged in males as compared to females.[26] Like ours, in some studies, no such significant difference in BT was observed between male and female.[2,10] It was reported in one study that testosterone inhibits platelet aggregation, and this effect was dependent on endothelial nitric oxide synthesis, which somehow favors our study.[27] In contrast, one study reported that thromboxane A2 synthesis increased by testosterone and this steroid hormone also facilitates platelet aggregation.[28] However, in some studies, it is well-documented that gender differences in bleeding time exist, with females typically exhibiting longer bleeding times than males, and it is thought to be due to the high estrogen, which may suppress platelet functions and also causes fibrinogen decrement in blood.[3, 11, 12, 13, 14, 29, 30]

LIMITATIONS

The studied sample size was small; therefore, the findings may vary from those of other studies. A bigger multicentric study is suggested to verify the above-mentioned findings.

CONCLUSION

In the present study population, the O blood group was predominant among other ABO blood groups. We found that bleeding time was prolonged in cases with blood group O than in other blood groups. Moreover, we found that the mean bleeding time of males is greater than females, and also a larger number of male has BT more than 3.5 compared to females, but the difference was not significant.

REFERENCES

1. Roy B, Banerjee I, Sathian B, Mittal A, Baboo NS, Jha N. Blood group distribution and its relationship with bleeding time and clotting time: a medical school-based observational study among Nepali, Indian and Sri Lankan students. Nepal J Epidemiol. 2011;1(4):135–40. doi:10.3126/nje.v1i4.5755. Available from: https://doi.org/10.3126/nje.v1i4.5755
2. Kaur M, Singh A, Bassi R, Kaur D. Blood group distribution and its relationship with bleeding time and clotting time. Natl J Physiol Pharm Pharmacol. 2015;5(3):253–57. doi:10.5455/njppp.2015.5.2609201433. Available from:https://doi.org/10.5455/njppp.2015.5.2609201433
3. Manandhar Adhikari S, Amatya A. Variation of bleeding time and clotting time in the ABO blood groups. J Physiol Soc Nepal. 2020;1(2):19–23. Available from: pdf
4. Pal GK, Pal P. Comprehensive textbook of medical physiology. Vol. 1. 1st ed. New Delhi: Jaypee Brothers Medical Publishers; 2017. ISBN:9789386056979. doi:10.5005/jp/books/12960. Available from:https://doi.org/10.5005/jp/books/12960
5. Pal GK, Pal P. Textbook of practical physiology. 2nd ed. Manipal: Universal Press; 2005. p.107–09.
6. Adhana R, Chaurasiya R, Verma A. Comparison of bleeding time and clotting time between males and females. Natl J Physiol Pharm Pharmacol. 2018;8(10):1388–90. doi:10.5455/ijmsph.2018.06201417062018. Available from: https://www.njppp.com/fulltext/28-1528542228.pdf
7. O'Donnell J, Laffan MA. The relationship between ABO histo-blood group, factor VIII and von Willebrand factor. Transfus Med. 2001;11:343–51. Available from: https://doi.org/10.1046/j.1365-3148.2001.00315.x
8. Franchini M, Mannucci PM. ABO blood group and thrombotic vascular disease. Thromb Haemost. 2014;112(6):1103–09.doi:10.1160/TH14-05-0457. Available from: https://doi.org/10.1160/TH14-05-0457
9. Gallinaro L, Cattini MG, Sztukowska M, Padrini R, Sartorello F, Pontara E, et al. A shorter von Willebrand factor survival in O blood group subjects explains how ABO determinants influence plasma von Willebrand factor. Blood. 2008;111(7):3540–45.doi:10.1182/blood-2007-11-122945.Available from:https://doi.org/10.1182/blood-2007-11-122945
10. Mahapatra B, Mishra N. Comparison of bleeding time and clotting time in different blood groups. Am J Infect Dis. 2009;5(2):106–08 Available from: ResearchGate
11. Kumar S, VK J, George J, Mukkadan J. Bleeding time and clotting time in healthy male and female college students of Karukutty Village, Kerala. Health Prospect. 2013;12(1):7–9. doi:10.3126/hprospect.v12i1.8720. Available from: Health Prospect
12. Verma A, Chaurasia R, Adhana R, Ballabh J, Kaur J. Interdependence of major blood group with bleeding time and clotting time. MedPulse Int J Physiol. 2019; 11:34-37.
13. Waghmare RV, Muniyappanavar NS. Influence of blood groups on bleeding and clotting time. Int Physiol. 2018;6(3):200–204. doi:10.21088/ip.2347.1506.6318.6. Available from: https://dx.doi.org/10.21088/ip.2347.1506.6318.6
14. Gupta SP, Dutta P, Anand S, Kanchan RK. Correlation of bleeding time and clotting time with ABO blood grouping among first year medical students. Natl J Physiol Pharm Pharmacol. 2021;11(5):525–529. doi:10.5455/njppp.2021.11.12371202018012021. Available from: http://dx.doi.org/10.5455/njppp.2021.11.12371202018012021
15. Pal GK, Pal P. Textbook of practical physiology. 3rd ed. New Delhi: Universities Press (India) Pvt Ltd; 2010. P.100–01.
16. Thenmozhi S, Neelambikai N, Aruna P. Comparison of bleeding time and clotting time in different ABO blood groups.

Natl J Physiol. 2013;1(1):19-24.

17. Kohli PG, Kaur H, Maini S. Relationship of bleeding time and clotting time with blood groups. Res J Pharm Biol Chem Sci. 2014 Jan 1;5(2):1780-3.Available from:https://www.rjpbcs.com/pdf/2014_5(2)/%5B210%5D.pdf

18. Sasekala M, Saikumar P. Relationship between bleeding time and clotting time among gender difference and varying blood groups in UG medical students. IOSR J Dent Med Sci. 2013;10(6):40–43. Available from: https://www.iosrjournals.org/iosr-jdms/papers/Vol10-issue6/I01064043.pdf

19. Abhishekh B, Mayadevi S, Meena D, Usha KC. Distribution of ABO and Rhesus-D blood groups in and around Thiruvananthapuram. Kerala Med J. 2011; 1:28–29. Available from: ResearchGate

20. Talib HV. Handbook of Medical Laboratory Technology. 2nd ed. New Delhi: CSB Publishers; 1991. P.205-10.

21. Jha RK, Kushwaha MS, Kushwaha DK, Tiwari S, Bhandari A, Nepal O. Blood group distribution and its relationship with bleeding time and clotting time in medical undergraduate students. Int J Res Rev [Internet]. 2017;4(9):10–15. Available from: IJRR003.pdf

22. Gavit S, Bhorania S. An observational study: association of blood groups with bleeding time and clotting time. Asian J Pharm Clin Res. 2022;15(7):119–22. doi:10.22159/ajpcr.2022.v15i7.44266 Available from: https://doi.org/10.22159/ajpcr.2022.v15i7.44266

23. Chinara A, Purohit P, Mahapatra B. No association of bleeding time and clotting time with four ABO blood groups in healthy young adults: an observational study. Natl J Physiol Pharm Pharmacol 2019;9(12):1193–97. doi:10.5455/njppp.2019.9.0931620092019. Available from: https://www.ejmanager.com/mnstemps/28/28-1568556156.pdf?t=1765625413

24. Adhikari B, Maharjan N. Blood group distribution and its association with bleeding time and clotting time among medical students. Asian J Med Sci 2024 Aug 1. doi:10.71152/ajms.v15i8.1389. Available from: https://doi.org/10.71152/ajms.v15i8.1389

25. Franchini M, Capra F, Targher G, Montagnana M, Lippi G. Relationship between ABO blood group and von Willebrand factor levels: from biology to clinical implications. Thromb J . 2007;5:14. doi:10.1186/1477-9560-5-14. Available from: https://doi.org/10.1186/1477-9560-5-14

26. Benjamin JJ, Geetha MB. Study of association of bleeding and clotting time with blood group among young adults. Indian J Clin Anat Physiol. 2020; 7(4):350-53. doi:10.18231/j.ijcap.2020.074. Available from:Indian Journal of Clinical Anatomy and Physiology

27. Borchgrevink CF. Platelet adhesion in vivo in patients with bleeding disorders. Acta Med Scand. 1961;170:231–43. doi:10.1111/j.0954-6820.1961.tb00234.x Available from: https://doi.org/10.1111/j.0954-6820.1961.tb00234.x

28. Campelo AE, Cutini PH, Massheimer VL. Testosterone modulates platelet aggregation and endothelial cell growth through nitric oxide pathway. J Endocrinol.2012;213(1):77–87.doi:10.1530/JOE-11-0441 Available from: https://doi.org/10.1530/JOE-11-0441

29. Adhana R, Chaurasia R, Verma A. Comparison of bleeding time and clotting time between males and females. Natl J Physiol Pharm Pharmacol.2018;8(10):1388-90.doi.org/10.5455/ijmsph.2018.06201417062018 Available from: https://doi.org/10.5455/ijmsph.2018.06201417062018

30. Aleem A, Wahid M. Correlation of blood groups, bleeding time and clotting time in male and female students: An observational study. Pak J Pharm Res. 2016;2(2):121-26.doi: 10.22200/pjpr.20162121-126 Available from: Researchgate

Clinicopathological Analysis of Eyelid Masses: A Cross-sectional Study

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ABSTRACT

Introduction: Eye lid masses are common presentations in ophthalmic practices, ranging from benign lesions to potentially life-threatening malignancies. While the clinical examination provides the initial diagnosis, histopathological evaluation remains the gold standard for accurate identification and management. Limited studies in Nepal have explored the clinicopathological correlation of eyelid tumors. **Aims:** To analyze the demographic profile, clinical presentation, and histopathological findings of benign and malignant eyelid masses, and to assess the correlation between clinical and histopathological diagnosis. **Methods:** This hospital based cross sectional study was conducted from May 2022 to May 2023 in the department of oculoplasty, Lumbini eye institute and research Centre. Total 53 patients presenting with eye lid masses underwent detailed ocular examination, surgical excision and histopathological analysis. Data were analyzed using SPSS version 22.0. **Results:** Among 53 patients, 30(56%) were female and 23(43%) male in both benign and malignant eyelid masses. Left upper lid was predominantly affected. The predominant clinical presentation was eye lid mass 33(62.26%) followed by pigmentary changes 8(15.09%). Ulcerated wounds and mechanical ptosis were equally observed in about 5(9.4%) cases each, while mechanical ectropion was the least common presentation, seen in 2(3.7%) cases. Histopathological analysis revealed 44(83%) as benign lesion and 9(17%) as malignant. The most common type of benign lesion was cyst (epidermal, inclusion, and sebaceous types) accounting for 19(43.8%) cases, followed by Nevi (simple, compound and intradermal) and papilloma ecompromising 8(18.8%)cases each. Among malignancies, Basal cell carcinoma 4 (44.44%) was most common followed by sebaceous cell carcinoma 3(33.33%). The overall clinical diagnostic accuracy compared to histopathological diagnosis was 75%. **Conclusion:** Benign eyelid lesions were more prevalent than malignant ones, with cysts being the most common benign lesion and basal cell carcinoma the most frequent malignancy. Only clinical examination may not be sufficient for accurate diagnosis; therefore, histopathological evaluation is essential for guiding appropriate management

Keywords: Basal cell carcinoma, Benign, Eyelid tumors, Histopathology, Malignant, Sebaceous carcinoma

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INTRODUCTION

Eyelid masses are commonly encountered in ophthalmic practice. The incidence of skin tumor is mainly due to the environmental factor, exposure to sunlight, ultraviolet rays. Approximately 10% of all skin tumors occurs in eyelid.[3] The relative frequencies of eyelid lesions are different in various parts of the world as reported in literature. The Benign tumor of eyelid is more common as compared to the malignant tumor.[2] The main malignant tumors affecting the eyelids are basal cell carcinoma, sebaceous carcinoma, squamous cell carcinoma, malignant melanoma. Basal cell carcinoma has the highest incidence among the malignant tumor in the western part of the world whereas sebaceous carcinoma is reported to have the highest incidence in Asians.[6] Although all the eyelid masses are diagnosed clinically at first, histopathological examination give us the definitive diagnosis which play the major role in further treatment. There are limited studies done in Nepal regarding eyelid tumor with clinicopathological correlation. This study analyzed the demography, clinical presentation, gross and histopathological findings to support the clinical diagnosis of different benign and malignant eye lid masses. This study aims to analyze the demographic profile, clinical presentation, and histopathological findings of benign and malignant eyelid masses, and to assess the correlation between clinical and histopathological diagnosis.

METHODS

This was a hospital based cross sectional study done of all con-

secutive cases of eye lid mass presenting to the department of oculoplasty, Lumbini eye institute and research Centre, over one year from May 2022 to May 2023. The eyelid masses were examined in ambient room light and photograph were taken in some cases. The eye lid masses presented undergo surgical excision with histopathological examination at National Pathology laboratory. The sample size was 53.

Sample size

$n = Z^2 \times p \times q/d^2$ where

P= Prevalence of eyelid mass (36% = 0.36)

Z= 1.96 at 95% confidence interval

q= 1-p=1-0.36=0.64

d= maximum tolerable error=10% = 0.1

$n= (1.96)^2 \times 0.36 \times 0.64/ (0.1)^2 = 53.02$

Sample size=53

Inclusion criteria

1. Patients with eyelid masses who underwent surgery and histopathological examination.

2. All patients ready to enroll in our study.

Exclusion Criteria

1. The patient presenting with infective or inflammatory eyelid masses.

2. Recurrent eyelid tumor.

3. Eyelid masses without surgery or histopathological examination.

4. The patient not willing to enroll in the study .

5. Evidence of systemic involvement as there may be evidence of metastasis to the adjacent structures and other systemic organ.

A comprehensive history of each patient presenting with eyelid mass was recorded. Particular emphasis was placed on the onset, laterality, progression, duration of the lesion, and time of initial presentation. Any evidence of lymph node involvement or systemic metastasis at presentation was documented. Potential risk factors, including tobacco use, smoking, prolonged sun exposure, history of radiation exposure and relevant family history were also noted. Systemic history was taken with specific reference to chronic illnesses and known case of metastatic carcinoma. Initial visual acuity assessment was performed using the Snellen visual acuity chart. All patients underwent detailed ocular examination using slit -lamp bio-microscopy conducted by an oculoplastic surgeon. Special attention was given to evaluating the eyelid mass, including its anatomical location, laterality, and dimensions (measured in millimeters). The mass was assessed for characteristics such as site, shape, form, mobility, compressibility, consistency, color, depth and surface features (e.g. regularity, elevation and margin definition). Additional features such as bleeding points, vascularization, and any other notable morphological traits were carefully recorded. To aid in clinical diagnosis, relevant imaging and laboratory investigations were performed. These included ultrasonography, Xray, computed tomography (CT), Magnetic resonance imaging (MRI), and routine blood tests. Based on clinical and radiological evaluation, a decision was made to proceed with excisional biopsy under local anesthesia (LA) or general anesthesia (GA), as deemed appropriate. Surgical excision of the eyelid mass was performed with a 4-5 mm margin of healthy tissue. Depending on the size and location of the resulting defect, appropriate reconstructive procedures were selected. These included direct closure, modified Hughes procedure, Cutler-Beard flap, or combinations of local flaps and grafts to restore functional and cosmetic integrity. The surgically resected specimens will be fixed in 10% formalin and will be sent to the department of pathology at National pathology lab for histopathological diagnosis by consultant pathologist Dr. Jeevan Adhikari.

The specimen sections will be stained by eosin stain and hematoxylin then to routine paraffin embedding in all cases. From different areas of the specimen, four to five sections 2-3 mm thick were taken and processed in automatic tissue processor. After trimming of blocks sections of 5-7 um thick were cut with help of rotatory microtome. Sections were floated on water at temperature of 45 degree and were taken on albuminized slides. Special stain such as PAS stain was used whenever required. Clinical records of patients were entered and verified by the specialist performing the clinical examination and intervention procedure. Collected data was entered on computer case sheet for statistical analysis.

Statistical analysis

Analysis was done with statistical package for social science (SPSS) 22.0. Flow diagrams, bar diagrams, histograms were used as needed. Interim analysis of the data was done after one month of completion of study followed by final analysis by computer. Statistician was consulted whenever necessary.

RESULTS

Out of 53 cases 30 (56%) were female and 23 (43%) were male. The most common age of presentation was 40-59 yrs. Elderly (>60 yrs) made up 35.8%. Among malignant eyelid lesion a higher proportion was seen in patients above 60 years, indicating age related increase in malignancy (Table I).

Age group in years	Total number of patients	Percentage
<20	4	7.5
20-39	12	22.6
40-59	18	34.0
60-79	15	28.3
>80	4	7.5
Total	53	100

Table I: Demographic profile of patients

Left upper lid was seen predominantly affected in both benign and malignant cases accounting for 19(35.85%) cases. Right lower lid was least affected and seen in 7(13.21%) cases. (Table II)

Lesion locations	Total number of patients	Percentage
Left upper lid	19	35.85
Right upper lid	15	28.30
Left lower lid	12	22.64
Right lower lid	7	13.21
Total	53	100

Table II : Anatomical locations of eye lid masses

The majority of the patients presented with an eyelid mass, 33(62.26%) cases. Ulcerated wounds and mechanical ptosis were equally observed in about 5(9.4%) cases each. Mechanical ptosis was due to the size of the mass in upper eye lid. Discoloration of the skin or pigmentary changes were noted in 8(15.09%) patients, while mechanical ectropion was the least common presentation, seen in 2(3.7%) cases. Discoloration of skin or pigmentary changes were seen mostly in benign lesions as the primary symptoms. (Table III)

Presenting symptoms	Total number of patients	Percentage
Eyelid mass	33	62.26
Ulcerated wound	5	9.4
Mechanical ptosis	5	9.4
Discoloration /pigmentation of the skin	8	15.09
Mechanical Ectropion	2	3.7
Total	53	100

Table III: Clinical presentations of eye lid masses

Out of 54 cases, 44(83%) were identified as benign lesions after histopathological examination, and 9(17%) identified as malignant. The most common type was cystic lesions (epidermal, inclusion, and sebaceous type) accounting for 19 cases (43.8%). Nevi (simple, compound and intradermal) and papilloma were the next most frequent, each compromising 8 cases (18.8%). Less common lesions included xanthelasma, hemangioma, seborrheic dermatitis, and dermolipoma, 2 cases (4.55%) each. The least frequent lesion observed was neurofibroma, 1 case (2.27 %). (Table IV)

Benign lesions	Total number of patients	Percentage
Cyst (epidermal, Inclusion, sebaceous)	19	43.8
Nevus	5	9.4
(simple, compound, intradermal)	8	18.8
Papilloma	8	18.8
Xanthelasma	2	4.55
Hemangioma	2	4.55
Seborrheic dermatitis	2	4.55
Dermolipoma	2	4.55
Neurofibroma	1	2.27
Total	44	100

Table IV: Histopathological spectrum of benign eyelid masses

Among 9 cases diagnosed as malignant after histopathological examination, the most common was Basal cell carcinoma 4 (44.44%). Sebaceous cell carcinoma accounted for 3(33.33%) cases, while squamous cell carcinoma and Non-Hodkins lymphoma accounted for 1 (11.11%) each. (Table V)

Malignant lesions	Total number of patients	Percentage
Basal Cell Carcinoma	4	44.44
Sebaceous cell Carcinoma	3	33.33
Squamous cell carcinoma	1	11.11
Non-Hodkins Lymphoma	1	11.11
Total	9	100

Table V: Histopathological spectrum of malignant eyelid masses

Among 53 patients, 75% of the clinical accuracy was seen while correlating clinical and histopathological diagnosis of all the cases. (Figure 1)

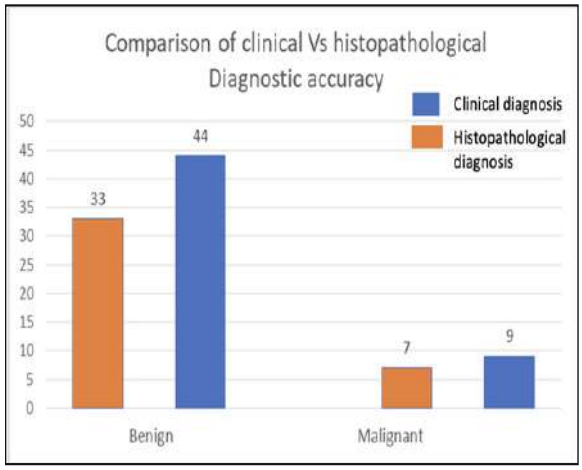

Figure 1 : Comparison of clinical vs histopathological diagnostic accuracy of eye lid masses

DISCUSSION

Eyelid masses are commonly encountered in ophthalmic practice. The incidence of skin tumor is mainly due to the environmental factor, exposure to sunlight, ultraviolet rays. Approximately 10% of all skin tumors occurs in eyelid.[3] various studies have been done regarding the eyelid mass tumor in various part of the world. These tumors may show geographical and ethnic variation. The data of our study shows the demography,

various modes of presentations of the eyelid tumor presenting in the tertiary eye care center of the western part of Nepal. The data will serve as the reference for this geographical region and guide in planning resources for screening as well as in proper management of the eyelid tumors. In our study benign eye lid lesions 44(81%) were far common than malignant lesion 9(17%) in accordance with previous other studies worldwide that ranged from (71% -91%).[2,6,7] Female preponderance was observed in our study 30 (56%) were female and 23(43%) patient were male similar to the study conducted in another center. The most common age of the presentation was 40-59yrs. Elderly (>60 yrs) make up to 35.8% important for malignancy trend.[2,1] Eye lid malignancies are more common in elderly patients due to cumulative sun damage, age related immune decline, DNA repair and chronic skin changes over time .[3,6] Left upper lid was predominantly affected 35.85%.

A total of 44(81%) benign cases were analyzed. we found the cystic lesion including epidermal, inclusion and sebaceous cyst 19(43.8%)as the most common benign histologic diagnosis. This finding is consistent with previous studies , which also report a high prevalence of these lesions due to obstruction of pilosebaceous units and their tendency to enlarge gradually drawing clinical attention.[1,2,18] The superficial location and cosmetic prominence of these cysts, especially in the periocular region, often prompt early presentation and surgical excision .[18] The melanocytic nevus simple , compound and intradermal and papilloma were the next most common, each compromising 8(18.8%). Their frequent detection may be attributed to their visibility and ease of excision especially in cosmetically sensitive areas such as eyelids.[2,5,18] Papilloma, often associated with viral etiology or chronic irritation, also represent a significant proportion of benign eyelid lesions. Less common lesions included were xanthelasma, hemangioma, seborrheic dermatitis, and dermolipoma, each with 2(4.55%) cases. It may be due to the unrecognizable clinical appearance, leading to non-surgical management in many cases unless cosmetic concerns or diagnostic uncertainty necessitate biopsy.[7] For example, Xanthelasma is usually diagnosed clinically and may be treated with laser or topical modalities rather than excision.[8] The least frequent lesion observed was neurofibroma, with 1 (2.27 %) case. solitary neurofibromas in the periocular area are rare, and the diagnosis often warrants investigation for underlying neurofibromatosis in younger patients, their low prevalence in the routine eyelid histopathology has been similarly reported in other studies.[10]

Among 9 cases diagnosed as malignant after histopathological examination, the most common was Basal cell carcinoma compromising 4(44.44%) of the cases. this finding is consistent with the global epidemiological trends where BCC is reported as the most frequent malignant eyelid tumor, particularly in fair skinned elderly populations with significant cumulative ultraviolet UV exposure.[6,16,18] The predilection of BCC for the periocular region , especially the lower eyelid and medial canthus, is likely due to greater sun exposure and thinner skin in these areas, making them more susceptible to UV induced DNA damage.[6,16,18] Sebaceous cell carcinoma was the second most common malignancy in our study, accounting for 3(33.33%) of cases, While relatively rare in western populations, sebaceous carcinoma is frequently reported in Asian populations , including those from south and east Asia, where it often presents a diagnostic challenge due to its ability to mimic benign conditions such as chalazion or chronic blepheritis.[9,10,18] The aggressive nature of sebaceous carcinoma, coupled with its tendency for pagetoid spread and regional metastasis, underscores the importance of early diagnosis and promptsurgical management.[6,17] Squamous cell carcinoma and Non-Hodgkin lymphoma were the least common malignancies in our cohort , each representing 1(11.11%) of cases. SCC of eyelid is known to be less common than BCC but has a higher potential for local invasion and metastasis.[6,18] Risk factors include chronic sun exposure, immunosuppression, and preexisting actinic keratoses. The single case of Non-Hodgkin Lymphoma likely represents a primary cutaneous or conjunctival lymphoma, which although rare should be considered in the differential diagnosis of persistent eyelid swelling or mass particularly in older adults.[6] The distribution of malignant lesions in our study highlights the predominance of BCC, followed by sebaceous cell carcinoma, which is in line with patterns seen in South Asian populations .[6,17] It reinforces the necessity for histopathological evaluation in all excised lesions, especially in older patients and those with recurrent or atypical presentations, to ensure timely and accurate diagnosis.

In the present study, the overall clinical diagnostic accuracy was found to be 75% when comparing clinical impressions with histopathological diagnosis. This suggests that while clinical evaluation plays a vital role in the initial assessment of periocular lesions, it may not always be sufficient to definitively distinguish between benign and malignant conditions. Factors contributing to diagnostic discrepancies may include overlapping clinical features, atypical presentations, or coexisting pathologies that mimic other lesions.[18] Although a reasonably high accuracy was achieved clinically, histopathology remains the gold standard for definitive diagnosis, especially in suspicious or ambiguous cases.

LIMITATIONS

The limitation of the study arises from the relatively small sample size, which confined the detailed subgroup analysis particularly for less common eyelid lesions. As only surgical excision with biopsy cases were included, benign lesions managed conservatively were not included introducing selection bias. Larger studies with longer follow up should be conducted to identify the accurate clinicopathological feature, diagnosis and further management of eye lid masses.

CONCLUSION

Benign eyelid lesions were more prevalent than malignant ones, with cysts being the most common benign lesion and basal cell carcinoma the most frequent malignancy. Only clinical examination may not be sufficient for accurate diagnosis; therefore, histopathological evaluation is essential for guiding appropriate management.

REFERENCES

1. Kafle PA, Hamal D, Sahu S, Poudyal P, Kafle SU. Clinico-pathological Analysis of Malignant Eyelid and Adnexal Tumors Presenting to a Tertiary Eye Hospital Of Eastern Nepal. Birat J. Health Sci. 2020;4(3):840-4.
2. Shrestha R, Sayami G. Study of histomorphological spectrum of eyelid lesions. J Pathol Nep. 2021; 11:1790-1802
3. Gundogan Fatih Cakir et al, Clinicopathological Evaluation of eye lid tumors. Asian pac. J. cancer prev. 2015;16(10):4265-269.
4. Huang Y Y ,Liang WY ,Tsai CC, Kao SC,Yu Wk, kau Hc. Comparison of the clinical characteristics and outcome of beign and Malignant eyelid tumors: Analysis of 4521eyelid tumors in tertiary medical centre clinicopathological study. Biomed Res Int. 2015;2015: 453091.
5. Bagheri A ,Tavakoli M, Kanaani A, Beheshti Zavareh R,Esfandiari H,Aletaha M et al . Clinicopathoogical evaluation of eyelid malignancies. Middle East Afr J Ophthalmo. 2013;20(3):187-92.
6. Tesluk GC. Eyelid lesions: incidence and comparison of benign and malignant lesions. Ann opthalmol.1985;17(11): 704-707 .
7. Deprez M and Uffer S, Clinicopathological features of eyelid skin tumors. A retrospective study of 5504 cases and review of literature. Am J Dermatopathol.2009;31(3):256-62 .
8. Gautam P, Adhikari RK, Sharma BR. A profile of eye-lid conditions requiringreconstruction among the patients attending an oculoplasty clinic in Mid-Western region of Nepal. Nepal J Ophthalmol. 2011;3(1):49-51.
9. Takamura H, Yamashita H. Clinicopathological Analysis of Malignant Eyelid Tumor Cases at Yamagata University Hospital: Statistical Comparison of Tumor Incidence in Japan and in other Countries. Jpn J Ophthalmol 2005; 49: 349-54.
10. Gosai J, Mehta D, Pherwani K, Bha_ R, Agrawal K, Tandel D. Clinical study of lid tumors in adults patients in western region of India. J Evol Med Dent Sci.2014;3(73):364-373.
11. Karan S, Nathani M, Khan T, Sen S, khader A. Clinicopathological study of eyelid tumors in Hyderabad- A review of 57 cases. J Med Allied Sci 2016;6(2):211-15.
12. Kale SM, Patil SB, Khare N, Math M, Jain A, Jaiswal S. Clinicopathological analysis of eyelid malignancies – a review of 85 cases. Indian J PlastSurg 2012; 45:22–28.
13. Wang CJ, Zhang HN, Wu H, Shi X, Xie JJ, He JJ, Kook KH, Lee SY, Ye J. Clinicopathologic features and prognostic factors of malignant eyelid tumors. Int journal of ophthalmol 2013;6 40:442-47
14. Sotiropoulos G , Gartzios C, Raggos Vet al., "Eyelid tumors at the university eye clinic of Ioannina, Greece: a 30-year retrospective study." Middle East Afr J Ophthalmol.2015;22(2):230-232.
15. Farhat F, Jamal Q, Saeed M, Ghaffar Z. Evalua_on of Eyelid Lesions at a Tertiary Care Hospital, Jinnah Postgraduate Medical Centre (JPMC),Karachi. Pak J Ophthalmology 2010; 26: 83-6.
16. Rugo CE, Waltz K. Basal cell carcinoma of the eyelid and periocular skin. Surv ophthalmo1993;38(2):169-92.
17. Ni C, Searl SS, Kuo PK, Chu FR, Chong CS, Albert DM. Sebaceous cell carcinomas of the ocular adnexa. Int ophthalmo clin 1982; 22(1):23-61
18. Deprez, M, Uffer Sylvie . American J Dermatopathol 2009;31(3)256-262 .

ORIGINAL ARTICLE

A Study on the Determinants of Uterovaginal Prolapse in Tertiary Hospital, Nepal

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ABSTRACT

Introduction: Pelvic organ prolapse is the descent of the pelvic organ from its normal position. Although few genetic and idiopathic causes have been associated with it, it most commonly follows difficult and repeated child births, making it one of the most common morbidities in developing countries like Nepal. **Aims:** To identify determinants of utero-vaginal prolapse (UVP) among women attending gynecologic department. **Methods:** A hospital-based case control study was carried out from March 2023 to March 2025 among women attending Department of Gynaecology and Obstetrics at Nepalgunj Medical College.Cases were women with utero-vaginal prolapse while controls were women free from utero-vaginal prolapse but with other gynecologic disease during the same period as of cases. Descriptive analysis along with bivariate and multivariate logistic regressions was performed in Statistical Package for Social Sciences (SPSS). Adjusted odds ratio with 95% confidence interval was used and statistical significance was considered at $p \leq 0.05$. **Results:** Out of 226 cases, only 3(4.1%) of the cases compared to 148(65.5 %) of the controls had attended delivery at health facility. Only 8(10.8%) of cases and 8(5.3%) of the controls had ever used family planning. Only 4(5.4%) of the cases and no controls had history of hysterectomy. Only 15(20.3%) of the cases and 25(16.4%) of the controls had medical problems (history of chronic cough, had history of carrying heavy objects, hypertension, diabetes mellitus and chronic constipation) This study revealed, age ≥ 40 years (AOR = 10.49; 95%CI: 4.03, 27.35), duration of labor ≥ 24 hours (AOR = 8.32; 95%CI: 3.58, 19.33), instrumental delivery (AOR = 7.40; 95%CI: 1.21, 45.28), non- utilization of family planning (AOR = 3.14; 95%CI: 1.32, 7.47) were found to have statistical significance. **Conclusion:** Age ≥ 40 years, prolonged labor, instrumental delivery, non-utilization of family planning were identified as determinant factors of utero-vaginal prolapse. Thus, family planning service utilization and appropriate obstetric care are advisable.

Keywords: Determinants, Pelvic floor repair, Uterovaginal prolapse, Vaginal hysterectomy

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INTRODUCTION

Genital prolapse affects the quality of life of many women during their pre-menopausal and post menopausal years.[1] Uterine Prolapse is a condition related to reproductive health that has been inadequately addressed in accordance with its prevalence in our country.[2] A study identified there are about 6 00,000 women with uterovaginal prolapse who need to be treated.[3] In spite of existence of safe motherhood programme and application of different strategies for so many years, the care seeking behaviour in relation to UVP is low in Nepal. To improve maternal health, strategy on the management of UVP at health institutions should be there including by potential Skilled Birth Attendants (SBAs).[4,5] Data about uterine prolapse in Nepal is little. Some studies on Non-reproductive Risk Factors of Uterovaginal Prolapse have shown the association associations between family history of prolapse and underweight sta-

tus as risk factors, with smokers also showing increased risk.[6] The most important factors those are associated with UVP are increase intra-abdominal pressure, difficult labor and delivery, malnutrition, old age, connective tissue disorders, heavy exercise, and pelvic trauma.[6-8] AANG JHARNE is the typical Nepali terminology used for the pelvic organ prolapse especially the uterovaginal prolapse illnesses.[9] Maternity health care like treatment seeking behavior of UVP is a choice and depends on individuals but literature suggests that choices are limited especially in the mountain and hill areas of Nepal.[10] The objectives of the study was to identify the determinants of UVP .

METHODS

This is a hospital based unmatched case control study conducted among women attending Outpatient Department (OPD) and Inpatient Department (IPD) of Gynaecology and Obstetrics at the Nepalgunj Medical College, Teaching Hospital Nepal, between March 2023 to March 2025. Ethical clearance was obtained. Data were retrieved from gynecological admission register, case files and medical record section, who were treated for uterovaginal prolapse as well as the control cases. All women attending gynecological and obstetrics OPD and IPD during the study period were the study unit. The sites of data collection were the department of gynecology and obstetrics and medical record section.

Cases were women with utero-vaginal prolapse and controls were those women free from utero-vaginal prolapse but with some other gynecologic disease (like pelvic inflammatory disease, uterine cancer, cancer of cervix, syphilis, gonorrhea, other sexually transmitted diseases) during the same study period.

The records of women aged more than 18 years attending OPD and IPD during the study period were included into the study. However, critically ill women and women with mental problems were excluded from the study.

Sample Size Determination

The prevalence of uterine prolapse (UP) in previous study was 21.3 %.[11] Sample size was calculated using Epi-info software version 7 using sample size determination for unmatched case–control studies. The parameters that were used to calculate sample size were; Confidence level 95%, power 80%, control to case ratio of 2: 1 and Odd Ratio of 2.1.[11] The final sample size was found to be 74 cases and 152 controls.

Data Collection Procedures

All the records of the patients during the study period were studied for the following parameters: age, caste, education, occupation, parity, obstetric factors, gynecologic history and child bearing.[12] The completeness of the data was checked by the principal investigator. Shaw's classification system of utero vaginal prolapse was used for grading of the disease in the case files. In this classification system, descent is classified into four grades; the first grade is descent of cervix into the vagina, second grade is descent of cervix into introitus, third grade is descent of the cervix outside introitus, and the fourth grade is when the whole of the uterus is outside introitus.[13]

Statistical analysis

The data were analyzed by descriptive and inferential statistics using the statistical package for social science (SPSS) version 20. Logistic regression was applied. Statistical significance was set at p-value < 0.05.

RESULTS

Socio-Demographic Characteristics of study participants

Out of the total 226 respondents, 74 cases and 152 controls participated in the study with the response rate of 100 %. The proportion of older age women (age≥ 40) was found to be higher among cases 70(94.6%) compared to controls 4(2.64%). Hindu religion was 63(85.1%) and 115(75.7%) among cases and controls respectively. Of the respondents, 71(46.7%) of controls were literate while only 5(6.8%). Majority of participants among cases 70(94.6%) and controls 147(96.7%) were housewife. Majority 70(94.6%) of cases had gravida ≥4 compared to 144(94.7%)of the controls, followed by parity of ≥ 4 among cases 55(74.3 %) compared to 23(15.1%) among controls (TableI).

Variables	Cases Number (%)	Controls Number (%)	Subtotal Number (%)	P value
Age				
≥40	70 (94.6)	4 (2.64)	74 (33.2)	0.01
<40	4 (5.4)	148 (97.36)	152 (68.8)	
Education				
Literate	5 (6.8)	71 (46.7)	76 (33.6)	0.01
Illeterate	69 (93.2)	81 (53.3)	150 (66.4)	
Ethnicity				
Dalit	46 (62.16)	69 (45.4)	115 (50.9)	0.003
Janjati	11 (14.86)	51 (33.6)	62 (27.4)	
Brahmin/ Chhetry	7 (9.45)	23 (15.1)	30 (13.3)	
Other	10 (13.51)	9 (5.91)	19 (8.4)	
Religion				
Hindu	63 (85.1)	115 (75.7)	178 (78.8)	.102

Other religion	11 (14.9)	37 (24.5)	48 (21.2)	
Occupation				
Housewife	70 (94.6)	147 (96.7)	217 (96)	0.445
Labour	4 (5.4)	5 (3.3)	9 (4)	
Gravida				
>4	70 (94.6)	144 (94.7)	214 (94.7)	0.964
4 or less than 4	4 (5.4)	8 (5.3)	12 (5.3)	
Parity				
4 >/4	55 (74.3)	23 (15.1)	78 (34.5)	0.001
<4	19 (25.7)	129 (84.9)	148 (65.5)	

Table I: Socio demographic characteristics and obstetric history of women

Variables	Cases	Controls	Subtotal	P value
History of abortion				
Yes	19 (25.7)	38 (25.2)	57 (25.3)	0.998
No	55 (74.3)	113 (74.8)	168 (74.7)	
Mode of delivery				
Vaginal delivery	63 (85.1)	105 (69.1)	168 (74.3)	0.001
Instrumental delivery	8 (10.8)	0	8 (3.5)	
Cesarean section delivery	3 (4.1)	47 (30.3)	50 (22.1)	
Place of delivery				
Home	71 (95.9)	7 (4.6)	78 (34.5)	0.001
Health facility	3 (4.1)	148 (65.5)	148 (65.5)	
Vaginal tear during last child birth				
Yes	5 (6.8)	32 (21.1)	37 (16.4)	0.006
No	69 (93.2)	120 (78.9)	189 (83.6)	
Duration of labor (labor for great or equal to 24 hours)				
=>24 hours	2 (2.7)	1 (0.7)	3 (1.3)	0.208
<24 hours	72 (97.3)	151 (99.3)	223 (98.7)	
Episiotomy during last child birth				
Yes	0	53 (34.9)	53 (23.5)	0.000
No	74 (100)	99 (65.1)	173 (76.5)	

Table II: Obstetric history of women attending gynecologic OPD among cases and controls

Variables	Cases	Controls	Subtotal	P value
Ever used family planning				
Yes	8 (10.8)	8 (5.3)	16 (7.1)	0.127
No	66 (89.2)	144 (94.7)	210 (92.9)	
Duration of rest after delivery (for the last child)				
=<42 days	64 (86.5)	105 (69.1)	169 (74.8)	0.004
>42 days	10 (13.5)	47 (30.9)	57 (25.2)	
Menopausal status				
Regular	6 (8.1)	151 (99.3)	157 (69.5)	0.000
Post menopausal	68 (91.9)	1 (0.7)	69 (30.5)	
Smoking history				
Yes	33 (44.6)	2 (1.3)	35 (15.5)	0.04
No	41 (55.4)	150 (98.7)	191 (84.5)	
History of hysterectomy				
Yes	4 (5.4)	0	4 (1.8)	0.004
No	70 (94.6)	150 (100)	222 (98.2)	
Previous prolapse surgery				
Yes	1 (1.4)	0	1 (0.4)	0.000
No	73 (98.6)	152 (100)	225 (99.6)	

Significant at 5 percent level of significance (p<0.05) Note: Values in parenthesis indicate percentage

Table III: Obstetric and other history of women attending gynecologic OPD among cases and controls

From the total study participants, 19 (25.7%) of the cases had history of abortion compared to 38(25.2%) of the controls,

63(85.1%) of cases and 105(69.1%) of the controls had vaginal delivery as mode of delivery,71(95.9%) and 7(4.6%) controls gave birth at home. Proportion of women who experienced vaginal tear during last delivery were only 5(6.8%) among cases and 32(21.1%) among controls .Majority of cases 72(97.35%) and controls 151(99.3%) had experienced less than 24 hours of duration of labor.

Majority of cases 74(100%) and control 99(65.1%) had not experienced episiotomy during last child birth. Only 8(10.8%) of cases and 8(5.3%) of the controls had ever used family planning. Duration of rest after delivery (for the last child) was ≤42 days for 64(86.5%) of the cases and 105(69.1%) of the controls. Menopausal status was found regular among only 6(8.1%) of the cases and 15 (99.3% of the controls. Only 20(27%) of the cases and no controls had family history of UVP. Only 4(5.4%) of the cases and no controls had history of hysterectomy. Only 1(1.4%) of the cases and no controls had previous prolapse surgery.

Only 15(20.3%) of the cases and 25(16.4%) of the controls, had medical problems (history of chronic cough, had history of carrying heavy objects, hypertension, diabetes mellitus and chronic constipation)(Table II & III).

Characteristics of respondents	Cases Number (%)	Controls Number (%)	Crude OR(95%CI	Adjusted OR(95%CI)
Age				
≥40	71 (94.7)	4 (20.6)	20.39 (10.76,38.63)	10.49 (4.03,27.35)*
<40	3 (4.1)	148 (97.6)		
Education				
Literate	5 (6.8)	71 (46.7)		
Illeterate	69 (93.2)	81 (53.3)	9.3 (5.23,18)	12 (5.23,28)*
Gravida				
>4	70 (94.6)	144 (94.7)	4.85 (2.61,8.99)	1.29 (0.32,5.26)
4 or less than 4	4 (5.4)	8 (5.3)		
Parity				
4 >/4	55 (74.3)	23 (15.1)	5.17 (2.93,9.12)	2.12 (0.88,5.04)
<4	19 (25.7)	129 (84.9)		
Mode of delivery				
Vaginal delivery	63 (85.1)	105 (69.1)		
Instrumental delivery	8 (10.8)	0		
Cesarean section delivery	3 (4.1)	47 (30.3)	4.18 (1.13,15.42)	7.40 (1.21,45.28)*
Place of delivery				
Home	71 (95.9)	7 (4.6)	5.86 (3.31,10.35)	1.43 (0.49,4.11)
Health facility	3 (4.1)	148 (65.5)		

Table IV: Determinants of uterovaginal prolapse among study participants

Characteristics of respondents	Cases Number (%)	Controls Number (%)	Crude OR(95%CI	Adjusted OR(95%CI)
Duration of labor(≥24 hours)				
=>24 hours	2 (2.7)	1 (0.7)	5.96 (3.43,10.38)	8.32 (3.58,19.33)*
<24 hours	72 (97.3)	151 (99.3)		
Ever used family planning				
Yes	8 (10.8)	8 (5.3)		
No	66 (89.2)	144 (94.7)	4.67 (2.56,8.52))	3.14 (1.32,7.47)*
Menopausal status				
Regular	6 (8.1)	151 (99.3)		
Post menopausal	68 (91.9)	1 (0.7)	15.69 (8.29.43)	1.77 (0.44,7.17
Family history of UVP				
Yes	20 (27)	0	4.63 (2.14,10.02)	3.77 (1.10,12.88)*
No	54 (73)	152 (100)		
Medical problems				
Yes	15 (20.3)	25 (16.4)	3.97 (2.29,6.90))	1.94 (0.82,4.60)
No	59 (79.7)	127 (83.6)		

TableV : Determinants of uterovaginal prolapse among study participants

Determinants of uterovaginal prolapse among study participants

Logistic regression results revealed the odds of developing UVP. Accordingly age, education, gravida, parity, place of delivery, duration of labor, mode of delivery, family planning use, family history, menopausal status and medical problems

predictor variables were significantly associated with UVP (p-value < 0.05) (Table IV & V) and after adjusting variables remained significant were age, education, mode of delivery ,family planning ever used, duration of labor and family history of UVP (Table IV & V).

Multivariable logistic regression analysis indicated that the odds of being age≥40 were 10.49 times higher among cases than controls (AOR=10.49; 95%CI: 4.03, 27.35). The odds of developing UVP were 8.32 times higher among women who had duration of labor great or equal to 24 hours during the last childbirth among cases than controls with (AOR=8.32; 95%CI: 3.58, 19.33). Women who gave birth by instrumental delivery were7.40 times (AOR=7.40; 95%CI: 1.21, 45.28) more likely to develop UVP in cases as compared to controls. The odds of having a UVP among women who did not ever used family planning was 3.14 (AOR=3.14; 95%CI: 1.32, 7.47) times higher among cases as compared to controls. Similarly, the odds of positive family history of UVP was 3.77 higher among cases than controls (AOR=3.77; 95%CI: 1.10,12.88)

However, the effect of episiotomy during last child birth, gravidity, parity, place of delivery, menopausal status, medical conditions (history of chronic cough, chronic constipation, and carrying heavy objects) disappeared after adjusting.

DISCUSSION

This study has identified determinants of UVP and identified the relationship with UVP with sociodemographic variables and explored the determinants of UVP. The study contradicts with the study conducted in Kaski district of Nepal which found the prevalence of UVP to be 11.7 %.[15] This study revealed that women aged ≥ 40 years of age were 10.49 times more likely to have had UVP as compared to women aged < 40 years. This might be due to age related weakening of pelvic supportive structures, decreased level of estrogen, and high parity in this age group. Study conducted in Lebanon and Ethiopia documented consistent results.[16,17] Results of systematic review also revealed consistent finding.[18] Duration of labor great or equal to 24 hours was significantly associated the development of UVP and the results are consistent with results from India[19], Nepal[20], Nigeria[21] and Ethiopia.[16]

Women who ever not used family planning were 3.14 times more likely to develop UVP as compared to women used family planning at least once. This finding is similar with the study conducted at Wolaita Sodo University Referral Hospital [22] this is because mothers who do not use family planning have repeated deliveries during which cumulative effects of pushing down pain damage the pelvic supportive structures.

A study done in Italy found that the risk of UVP was higher in women with family history of prolapse as compared to women without family history of prolapse.[23] This could be due to the presence of congenital connective tissue disorders in these families. Joseph N conducted a similar survey in a center in south India and found 76.8% of study population have third degree UVP, commonly associated with cystocele i.e. in 74.6%. Most of them underwent the surgical treatment of vaginal hysterectomy, even though they were prescribed ring pressaries to another significant lot. The scenario presented in the studies is congruent to our set up as well, where maximum of the cases have UV prolapse only or associated cystocele. The treatment options provided are similar as well.[24]

Teenage pregnancy and too many pregnancies contributed to the occurrences of UVP. Another reason was that most of the women delivered their babies at home assisted by untrained persons, and most of the parturient mothers or delivering women resumed work soon after delivery and had very poor nutrition.[25]

LIMITATIONS

This study relies on women self-reporting obstetric and medical histories (such as age at first delivery, birth spacing, and delivery conditions) over long periods and this may have led to recall bias while taking the history by a clinician. This study is a retrospective study. Small size is also a limitation.

CONCLUSION

The study revealed that age ≥ 40 years, prolonged labor, instrumental delivery, non-utilization of family planning were identified as determinant factors of UVP. Thus, family planning service utilization and appropriate obstetric care are advisable.

REFERENCES

1. Robinson D. Urogenital Prolapse, In: Luesley DM, Baker PN (eds). Obstetrics and Gyaecology: An evidence-based text for MRCOG 1st Edition London, Anorld 2004; 661-70.
2.. Booklet on Uterine Prolapse, UNFPA & Sancharika Samuha,April 2007
3. Schaaf JM, Dongol A, Vander HL. Follow -up of prolapse Surgery in Rural Nepal, Int Urogynecol J Pelvic Floor Disfunct 2008;19(6):851-5.
4. Bonetti TR, Erpelding A, Pathak LR. Listening to"felt needs": investigating genital prolapse in western Nepal. Reprod Health Matters 2004;12(23):166–175
5 DoHS/FHD. Safe delivery incentive strategy guideline. 2062
6. Menur A, Segni H. Pelvic Organ Prolapsein Jimma University Specialized Hospital, Southwest Ethiopia. Ethiop J Heal Sci. 2012;22(2):85–92.
7. Asresie A, Admassu E, Setegn T. Determinants of pelvic organ prolapseamong gynecologic patients in Bahir Dar, North West Ethiopia: A case–control study. Int J Womens Health. 2016;8:713–9.
8. ElejeG, Udegbunam O, OfojebeC, AdichieC. Determinants and management outcomes of pelvic organ prolapsein a low resourcesetting. Ann Med Health Sci Res. 2014;4(5):796.
9. Genital Prolapse. Women's Health Queensland wide; [cited 2011 18 May]; Available from: http://www.womhealth.org.au/factsheets/genital_prolapse.htm

10. Baral Y R , Lyons K ,Skinner J,Van Teijlingen ER "determinants of skilled birth attendants for delivery in Nepal"KUMS journal, 2010;8(31):325-32

11. Ying-Shuang,Gen-Den Chen,Soo-Cheen Ng "Prevalence of and risk factors for pelvic organ prolapse and lower urinary tract symptoms among women in rural Nepal" , International Journal of Gynecology & Obstetrics ,2012;119(2):185-8 DOI: 10.1016/j.ijgo.2012.05.031

12. Asresie A, Admassu E, Setegn T. Determinants of pelvic organ prolapse among gynecologic patients in Bahir Dar, North West Ethiopia: A case–control study. Int J Womens Health. 2016;8:713–9

13. Howkins and B. Shaw's Textbook of Gynaecology. 16th editi. 2015. 351 – 53 p.

14. WHO/SEARO. South-East Asia local Workshop on circle of relatives planning, STIs/RTIs and professional birth Attendants. 2005; web page 25.

15. Shah S.Urawn S, "Sociodemographic profile of uterine prolapse cases in Nepalgunj Medical College,Teaching hospital" JNGMC,December 2013,Vol 11 No. 2

16. Menur A, Segni H. Pelvic Organ Prolapsein Jimma University Specialized Hospital, Southwest Ethiopia. Ethiop J Heal Sci. 2012;22(2):85–92.

17. Awwad J, Sayegh R, Yeretzian J, Deeb ME. Prevalence, risk factors, and predictors of pelvic organ prolapse: A community-based study. J North Am MenopauseSoc. 2012;19(11):1235–41.

18. Vergeldt TFM, Weemhoff M, Inthout J, Kluivers KB. Risk factors for pelvic organ prolapseand its recurrence: a systematic review ,IntUrogynecolJ. 2015 Nov;26(11):1559-73. doi: 10.1007/s00192-015-2695-8.

19. Sujindra E, Himabindu N, Sabita P, Bupathy A. Determinants and treatment modalities of uterovaginal prolapse: A retrospectivestudy. Indian J Health Sci. 2015;8(1):36.

20. Thapa S, AngdembeM, Chauhan D, Joshi R. Determinants of pelvic organ prolapseamong the women of thewestern part of Nepal: A case-control study. J Obstet Gynaecol Res. 2014;40(2):515– 20.

21. ElejeG, Udegbunam O, OfojebeC, AdichieC. Determinants and management outcomes of pelvic organ prolapsein a low resourcesetting. Ann Med Health Sci Res. 2014;4(5):796

22. Zinash Lema Y, BerhaneMM. Determinants of Pelvic Organ Prolapseamong Gynecological Cases in Wolaita Sodo University Referral Teaching Hospital, Southern Ethiopia : A CaseControl Study. 2015;5(21):1–10.

23. Chiaffarino F, Chatenoud L, Dindelli M, Meschia M, Buonaguidi A, Amicarelli F, et al. Reproductive factors, family history, occupation and risk of urogenital prolapse. Eur J Obstet Gynecol Reprod Biol. 1999;82(1):63–7.

24. Joseph N, Krishnan C, Reddy BA, Adnan NA, Han LM, Min YJ. Clinical Profile of Uterine Prolapse Cases in South India. J Obstet Gynecol India. 2015 Oct 16;66(S1):428- 34 DOI: 10.1007/s13

25. Shrestha Ava Darshan,Lakhaey Bamala Sharma Jyoti/ Singh Meeta ,Prevalence of Uterine Prolapse amongst Gynecology OPD Patients in Tribhuvan University Teaching Hospital in Nepal and its Socio-Cultural Determinants, Kuala Lumpur, Malaysia: The Asian-Pacific Resource & Research Centre for Women, 2014• https://doi.org/10.14431/aw.2014.03.30.1.81

When Yellow Isn't Jaundice: Palmar and Plantar Discoloration in Carotenoderma – A Case Report

Aryal A, Poudel RS, Karmacharya H, Shris N

ABSTRACT

Carotenoderma is characterized by a yellow-orange discoloration of the skin, typically resulting from the excessive consumption of carotene-rich fruits and vegetables. We report a case involving a patient who developed yellow pigmentation of the palms and soles in the absence of jaundice, secondary to a high intake of carrots, pumpkin, papaya, and green leafy vegetables. Although this condition is benign and does not require medical treatment, it is essential to distinguish it from pathological causes of yellow skin, such as jaundice.

Keywords: β-carotene; Carotenoderma; Jaundice

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INTRODUCTION

Carotenoderma is a phenomenon characterized by yellow-orange pigmentation of the skin that results from carotene deposition in the skin, mainly in the stratum corneum. It is associated with a high blood β-carotene value, and is regarded as a significant physical finding, but is a harmless condition.[1] As it is a benign condition, it seldom requires further investigations. Carotenoderma is not an uncommon presentation, despite the prevalence data being scarce.[2] Awareness of carotenoderma is necessary to avoid confusion with jaundice and unnecessary diagnostic tests.[3]

CASE REPORT

A 36-year-old female presented to the Dermatology Outpatient Department with complaints of yellowish discoloration of palms and soles for 4 months. She initially noticed yellowish discoloration of her palms followed by her soles. She denied pruritus, darkening of the urine, and pale stools. She also gave history of generalized weakness in the past, for which her diet had predominantly consisted of carrots, pumpkin, papaya, and green leafy vegetables. Upon further evaluation, the patient revealed that her dietary pattern had been self-adopted in response to a persistent sense of generalized weakness, which she had attributed to a nutritional deficiency. Subsequent consultation with a psychiatrist led to a diagnosis of moderate depression, which was considered to be the cause of her generalized weakness. There was no history of diabetes mellitus, thyroid disorder, and liver disorder in the past. The general condition of the patient was fair. The patient was conscious, oriented to time, place, and person. All her vital signs were normal. Local examination revealed yellow-orange discolorations on the palmar aspect of both hands, and the plantar aspects of both feet. Her sclera was anicteric. There were no changes detected in the scalp, nail, and mucosa. No significant abnormalities were detected on systemic examination.The patient was sent for a complete blood count, random blood sugar, thyroid function test, and liver function test, which were unremarkable. However, serum β-carotene levels could not be assessed due to the unavailability of necessary testing facilities. The patient was advised to cut down on foods rich in carotenoids, and pigmentation is expected to improve slowly over time.

DISCUSSION

Carotenoderma is a benign and reversible condition characterized by yellow discoloration of the skin and elevated levels of β-carotene in the blood, which occurs secondary to excessive and prolonged ingestion of carotene-rich foods. Carotene serves as the primary precursor of vitamin A in human beings. However, hypervitaminosis A does not occur with excess carotene ingestion as the body converts only a limited quantity of carotene to vitamin A daily.[4,5] A detailed history should be taken, along with dietary history. The focus should be on the history of foods consumption that have high carotene levels,

with the estimation of the amount taken and duration. In addition, screening should be done for other conditions that could present with carotenoderma, such as diabetes, anorexia, hypothyroidism, and liver and kidney diseases.[2] Pigmentation usually involves the palms, dorsum of hands, soles, forehead, tip of the nose and nasolabial folds, but spares the sclera and mucous membranes. A typical sign of carotenoderma is its enhanced appearance under artificial light.[6]

The diagnosis of diet-induced carotenoderma is usually made clinically, and there is typically no need for laboratory confirmation. The mainstay of treatment is the reduction of the amount of carotene in the diet. This ultimately leads to the progressive disappearance of yellow skin coloration. However, the pigmentation could persist for several months, even after carotene levels return to normal due to the lipophilic nature of carotenoids.[7] Reassurance should be given to patients that diet-induced carotenoderma is a reasonably benign condition, and requires no treatment.

Our case highlights the importance of recognizing its clinical presentation, particularly the yellow-orange discoloration of the skin with sparing of the sclera, to differentiate it from more serious conditions like jaundice. Increased awareness among clinicians can help prevent unnecessary investigations and provide reassurance to patients through appropriate dietary counselling.

Figure 1: Carotenoderma of the Palms

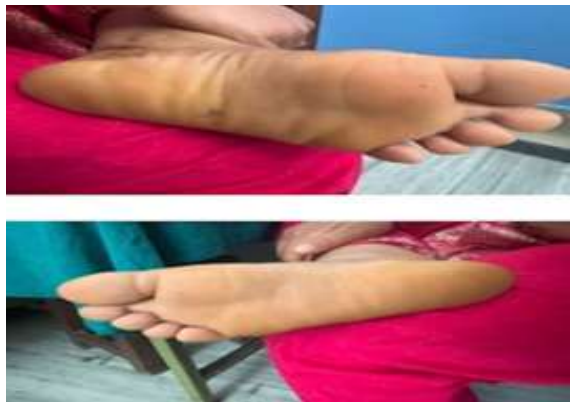

Figure 2: Carotenoderma of the Soles

REFERENCES

1. Maharshak N, Shapiro J, Trau H. Carotenoderma — a review of the current literature. Int J Dermatol. 2003;42(3):178–81. DOI:10.1046/j.1365-4362.2003.01657.x
2. Al Nasser Y, Jamal Z, Albugeaey M. Carotenemia [Updated 2023 Jun 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. Available from: https://www.ncbi.nlm.nih.gov/books/NBK534878/
3. Lascari AD. Carotenemia: a review. Clin Pediatr (Phila). 1981;20(1):25–9. DOI:10.1177/000992288102000103
4. Gandhi M, Walton S, Wyatt EH. Hypercarotenaemia in a tomato soup faddist. BMJ. 1988;297(6664):1635. DOI:10.1136/bmj.297.6664.1635
5. Chhabra C, Dogra P, Rijhwani M. Carotenemia: Post Hemi - Thyroidectomy. International Journal of Science and Research (IJSR). 2024 Jul 5;13(7):506–7
6. Leonard JW. Carotenemia vs jaundice. JAMA. 1976;236(23):2603
7. Edigin E, Asemota IR, Olisa E, et al. Carotenemia: a case report. Cureus. 2019 Jul 23;11(7):e5218. DOI:10.7759/cureus.5218

CASE REPORT

Dermoid Cyst on the Temporal Surface of Skull - An Unusual Case Report

Singh SK[1], Mahat AK[2], Jha BC[3]

ABSTRACT

A dermoid cyst is a subcutaneous mass derived from both ectodermal and mesodermal tissues. Dermoid cysts of the temporal region involving the bone are rare. We present a case of a child with an enlarging, palpable mass in the temporal region of the scalp.Parents of a 4-year-old male child presented with complain of a swelling on the right lateral side of the head, above the ear. Physical examination revealed a subcutaneous, cystic mass on the right lateral side of the head, above the ear in the temporal region, approximately 1.5 cm in diameter. The CT scan revealed a bony depression in the skull near the fronto-temporo-parietal-sphenoidal junction. Enucleation of cyst done with hemicoronal approach. Histopathological examination confirmed the diagnosis of a dermoid cyst. Early removal is recommended not only to establish a definitive diagnosis but also to prevent chronic inflammation, enlargement, invasion, or rupture of the cyst.

Keywords: Bony depression, Demoid cyst, Hemicoronal approach, Rare, Temporal cyst

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INTRODUCTION

A dermoid cyst is a subcutaneous mass derived from both ectodermal and mesodermal tissues.[1] Dermoid cysts have been reported in various locations, including the head and neck region, which accounts for approximately 7% of cases. In this region, they are often asymptomatic.[2] Some patients may present with symptoms such as yellow discharge, tenderness on palpation, headache, swelling, or pruritus. In rare instances, more severe manifestations have been documented, including sudden onset of oculomotor nerve palsy, blindness, chronic otitis media, and other neurological signs and symptoms.[3] Congenital dermoid cysts of the temporal region involving the bone are rare, with only scattered case reports available in the literature. In 1937, New and Erich reported a single temporal dermoid in their review of 103 head and neck dermoid cases.[4,5] We present a case of a child with an enlarging, palpable mass in the temporal region of the scalp.

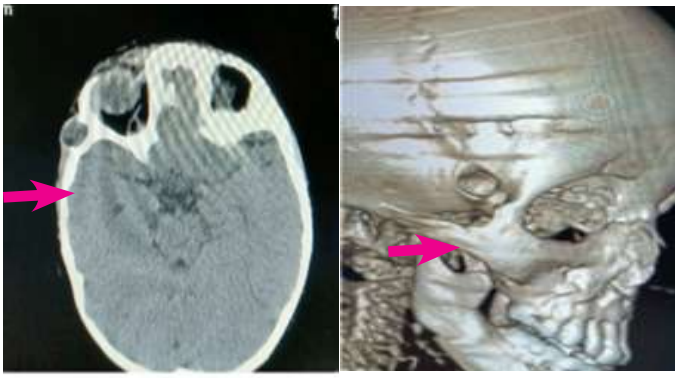
CASE REPORT

Parents of a 4-year-old male child presented to us at Chhinnamasta Hospital, Rajbiraj, Saptari, a rural hospital of Nepal, complaining of a swelling on the right lateral side of the head, above the ear. They attributed the swelling to a road traffic accident that occurred six months prior. The accident was minor and did not require hospital admission. The parents noticed the swelling three to four months after the accident. They did not see such a type of lesion before trauma. The child's caregiver denied any history of infection or changes in the size of the swelling.

The patient was otherwise asymptomatic, with no visual impairment or any neurological signs or symptoms. There was no other relevant past medical history. Physical examination revealed a subcutaneous, cystic mass on the right lateral side of the head, above the ear, and behind the orbit in the temporal region. The lesion measured approximately 1.5 cm in diameter. The lesson was not mobile from base, but the overlying scalp/ skin is mobile. It was noncompressible and nontender, with no evidence of an associated sinus tract, skin dimpling, discoloration, or communication with adjacent structures. The rest of the head and neck examination was unremarkable.

A computed tomography (CT) scan was performed due to the unusual presentation of the mass in the temporal region. The CT scan revealed a bony depression in the skull near the fronto-

temporo-parietal-sphenoidal junction (Pterion region) (Figure 1). It also confirmed that the subcutaneous mass was not in communication with adjacent structures, specifically showing no intraorbital or intracranial involvement.



A B

Figure 1: CT scan reveals a bony depression in the skull near the fronto-temporo-parietal-sphenoidal junction. A: Axial view, B: 3D view, Arrow: showing lesion

Given the unsatisfactory appearance of the mass, the parents' concern about potential growth, and the possibility of cranial involvement, surgical enucleation of the mass was planned under general anesthesia. Various surgical approaches, including endoscopic surgery, were discussed with parents. Enucleation of the cyst via the hemicoronal approach was chosen for surgery, and then verbal and written consent was taken for the procedure.Hairline Hemicoronal incision extending into the preauricular area was given. A scalp and temporal skin flap was elevated to expose the temporalis fascia (Figure 2). Hemostasis was achieved with bipolar cautery and fine hemostats. The cyst was then exposed by incising the temporalis fascia and temporalis muscle (Figure 3). Complete enucleation of the cyst was performed carefully.

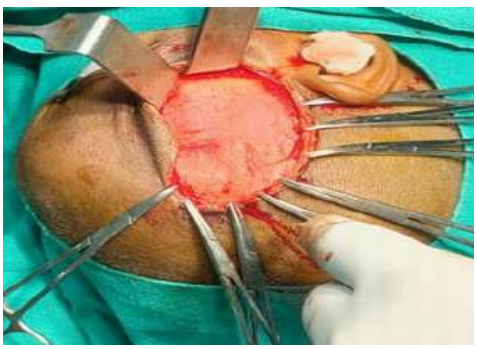


Figure 2: Hemicoronal incision (expose the temporalis fascia)

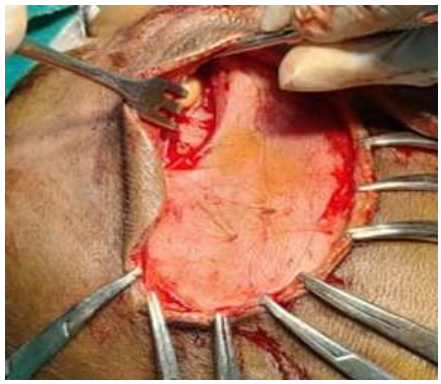


Figure 3: The Temporalis muscle is dissected vertically, exposing the cyst

A bony depression approximately 1.5 cm in diameter was noted near the pterion region, with intact bony walls on all sides; no underlying cranial defect was observed (Figure 4). Peripheral ostectomy of the bone cavity was performed after enucleation using round burs, accompanied by thorough saline irrigation.

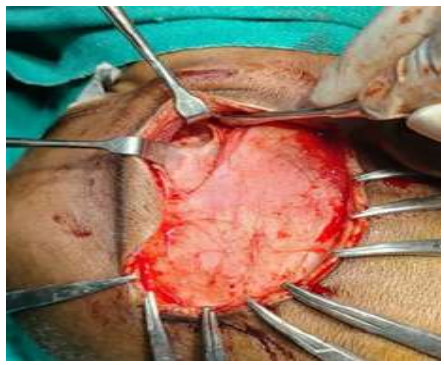


Figure 4: Bony depression after cyst enucleation and peripheral osteotomy

Final hemostasis was confirmed, the temporalis fascia and temporalis muscle was closed with 4-0 Vicryl suture (Figure 5). Skin closure was done with 4-0 Prolene sutures (Figure 6). The patient was discharged on the second postoperative day with an uneventful recovery. The incisions healed well without any complications.

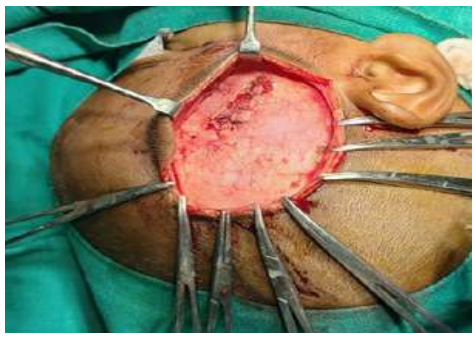


Figure 5: The temporalis fascia and temporalis muscle was closed with 4-0 Vicryl suture

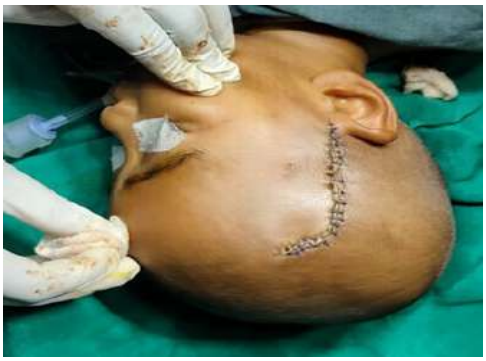
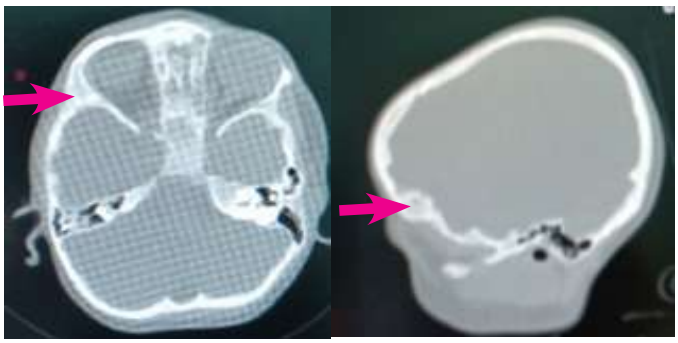


Figure 6: Skin closure done with 4-0 Prolene

Histopathological examination confirmed the diagnosis of a dermoid cyst. Regular follow-up was maintained every three months for one year. At one year post-operation, a follow-up CT scan showed normal healing of the lesion site (Figure 7). The incision scar was minimally noticeable, with mild alopecia around the incision line (Figure 8). Follow-up continued for an additional two years, during which there was no evidence of recurrence or adverse effects.



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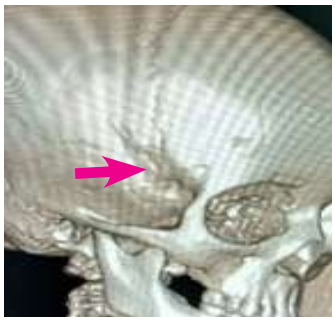

C

(A: Axial, B: Sagittal, C: 3D View, Arrow shows site of healing)

Figure 7: Normal bony healing in 1-year-follow-up

Figure 8: Normal healing with mild alopecia noted in the incision line

DISCUSSION

Dermoid tumors were first described by Parsons in 1742.[6] Dermoid cysts, also known as "oil cysts," are non-neoplastic, congenital ectodermal inclusion cysts that contain varying amounts of ectodermal derivatives, including apocrine, sweat, and sebaceous glands, as well as hair follicles, squamous epithelium, and sometimes teeth. They should not be confused with epidermoid cysts, which contain only squamous epithelium. Although similar in some respects, teratomas are a distinct entity; they are true neoplasms composed of tissues derived from all three embryonic germ layers.[1,7]

When evaluating a mass in the temporal region, it is important to consider a broad differential diagnosis, including capillary hemangioma, hemorrhagic lymphangioma, and non-Hodgkin lymphoma. Although dermoid cysts are relatively uncommon in the temporal region, they should still be included in the differential diagnosis.[8] Imaging with thin-section CT scans and/or MRI is valuable because these cysts may extend beyond the bone into the orbit or intracranially. In some cases, a biopsy may be necessary for definitive diagnosis, treatment planning, and monitoring.[8,9]

The treatment of choice for dermoid cysts is complete surgical excision. Early removal is recommended not only to establish a definitive diagnosis but also to prevent chronic inflammation, enlargement, invasion, or rupture of the cyst. Long-standing dermoid cysts may cause bone erosion and posterior migration, complicating surgical management. Careful dissection is essential to avoid rupture of the cyst capsule, as spillage of its contents can lead to recurrence. Lastly, the risk of malignant transformation, especially into squamous cell carcinoma, must be taken into account, though it is rare.[8-10]

Various types of incisions can be used for the removal of temporal dermoid cysts, including bicoronal and hemicoronal incisions. The bicoronal incision provides wide exposure but carries a higher risk of bleeding and a larger area of alopecia. In contrast, the hemicoronal incision offers excellent exposure while achieving a more cosmetically satisfactory result.[10] Needle aspiration can be employed to drain the cyst to facilitate surgical removal, especially when en bloc excision would require extensive dissection or risk injury to adjacent

vulnerable structures. Endoscopic removal is another option for temporal dermoid cysts. Advantages include a smaller, concealed incision within the hairline, improved visualization with magnification of the surgical field, and reduced risk of injury to critical anatomical structures.[5] However, this technique may be costly and demands a steep learning curve for surgeons. Regardless of the chosen method, complete removal of the cyst remains the primary surgical goal.

CONCLUSION

We describe a relatively rare case of a temporal dermoid cyst presenting as a swelling in the temporal region, with CT imaging revealing bony involvement. Based on this case and supporting literature, we recommend that any suspected congenital mass in the temporal region, with or without an associated sinus tract, should undergo preoperative radiological evaluation using CT or MRI to assess the extent of the lesion. In cases where bony invasion is evident, a more aggressive surgical approach is warranted. This should include complete enucleation of the cyst and peripheral ostectomy, ideally involving at least the outer table of the cranium, to minimize the risk of recurrence.

REFERENCES

1. Ray MJ, Barnett DW, Snipes GJ, Layton KF, Opatowsky MJ. Ruptured intracranial dermoid cyst. InBaylor University Medical Center Proceedings 2012 Jan 1 (Vol. 25, No. 1, pp. 23-25). Taylor & Francis.
2. Dutta M, Saha J, Biswas G, Chattopadhyay S, Sen I, Sinha R. Epidermoid cysts in head and neck: our experiences, with review of literature. Indian Journal of Otolaryngology and Head & Neck Surgery. 2013 Jul;65:14-21.
3. Adulkar NG, Arunkumar MJ, Kumar SM, Kim U. Unusual case of temporal dermoid cyst presenting as oculomotor nerve palsy. Indian Journal of Ophthalmology. 2014 Oct;62(10):1032.
4. New GB, Erich JB. Dermoid cyst of the head and neck. Surg Gynecol Obstet 1937;65:48 – 56.
5. Guerrissi JO. Endoscopic excision of frontozygomatic dermoid cysts. Journal of Craniofacial Surgery. 2004 Jul 1;15(4):618-22.
6. Elahi MM, Glat PM. Bilateral frontozygomatic dermoid cysts. Annals of plastic surgery. 2003 Nov 1;51(5):509-12.
7. Hirschberg J. Oil cyst of orbit. Arch Ophthalmic. 1879;8:373-5.
8. Sadeghi Y, Obéric A, Hamédani M. Different locations of dermoid cysts in the orbital region. Klinische Monatsblätter für Augenheilkunde. 2015 Apr;232(04):489-92.
9. Scolozzi P, Lombardi T, Jaques B. Congenital intracranial frontotemporal dermoid cyst presenting as a cutaneous fistula. Head & Neck: Journal for the Sciences and Specialties of the Head and Neck. 2005 May;27(5):429-32.
10. Parag P, Prakash PJ, Zachariah N. Temporal dermoid–an unusual presentation. Pediatric surgery international. 2001 Feb;17:77-9.

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