

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

Adhikari KC¹, Bist A², Verma AK¹, Mahato BK¹, Shah R³

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

Authors:

1. Dr. Krishna Chandra Adhikari
2. Dr. Aayush Bist
3. Dr. Awadesh Kumar Verma
4. Dr. Bijay Kumar Mahato
5. Mrs. Rojina Shah

¹Cardiology, Department of Medicine, Nepalgunj Medical College and Teaching Hospital, Kohalpur, Banke

²Department of Surgery, Nepalgunj Medical College and Teaching Hospital, Kohalpur, Banke

³Usha Cardio Care, Dhangadhi, Kailali

Address for Correspondence:

Dr. Krishna Chandra Adhikari
 Cardiology, Department of Medicine
 Nepalgunj Medical College and Teaching Hospital
 Kohalpur, Banke
 Email: drkrishnacadhikari@gmail.com
 ORCID ID. No.: 0000-0001-9781-1405

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 AWTI, 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 ob-

served 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.

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