



COMPARISON OF BMI, LIPID PARAMETERS, AST, AND LDH IN MYOCARDIAL INFARCTION PATIENTS STRATIFIED BY SMOKING STATUS: A CASE-CONTROL STUDY PAKISTAN

Fakhre Islam¹, Qismat Ullah², Farman Ullah³, Muhammad Bilal Shah¹, Ahmad Ehsan Rabani⁴, Faisal Nadeem^{1*}

¹Department of Community Medicine, Bannu Medical College, Medical Teaching Institution (MTI), Bannu, Khyber Pakhtunkhwa, Pakistan.

²Department of Medicine, Khalifa Gul Nawaz Teaching Hospital, Medical Teaching Institution (MTI), Bannu, Khyber Pakhtunkhwa, Pakistan.

³Department of Pathology and Curative Medicine, Ittehad Medical & Diagnostic Complex (IMDC), Karak, Khyber Pakhtunkhwa, Pakistan.

⁴Department of Community Medicine, HBS Medical & Dental College, Islamabad, Pakistan.

*Corresponding Author Email: ktkfaisal7@gmail.com

Abstract

Background: Smoking and dyslipidaemia contribute to cardiovascular disease. This study compared BMI, lipid parameters, AST, and LDH in Pakistani myocardial infarction (MI) patients and controls stratified by smoking status.

Methods: A hospital-based case-control study enrolled 150 MI patients and 150 age-comparable controls at Khalifa Gul Nawaz Teaching Hospital, Medical Teaching Institution (MTI), Bannu, Khyber Pakhtunkhwa, Pakistan, from 11 June 2025 to 20 October 2025. BMI and fasting serum biochemical parameters were assessed. Comparisons were stratified by smoking status and age. Exploratory correlations included 53 MI cases with complete data.

Results: BMI did not differ between MI patients and controls overall ($p = 0.857$). Total cholesterol and LDL were higher in MI patients across smoking strata. Among smokers, triglycerides were 215.50 ± 30.73 mg/dL in MI patients versus 155.92 ± 52.23 mg/dL in controls ($p < 0.001$), whereas the non-smoker comparison was not significant ($p = 0.401$). HDL was higher in MI patients overall and among non-smokers (both $p < 0.001$). LDH was higher in MI patients across strata (all $p < 0.001$). AST did not differ significantly between smoking cases and smoking controls ($p = 0.103$). In the complete-case subset, the HDL correlation among non-smokers met the nominal 0.05 threshold ($r = -0.376$, $p = 0.037$), whereas the LDH correlation among smokers did not ($r = 0.414$, $p = 0.055$).

Conclusion: MI patients showed differences in lipid and enzyme concentrations, with a marked triglyceride difference among smokers. These unadjusted comparisons do not establish independent smoking effects, causal mechanisms, or diagnostic accuracy. The unexpected HDL finding and exploratory correlations require cautious interpretation.

Keywords: myocardial infarction; smoking; dyslipidaemia; body mass index; aspartate aminotransferase; lactate dehydrogenase; Pakistan

Introduction

Cardiovascular disease remains a leading cause of mortality and disability worldwide, with substantial contributions from modifiable risk factors [1]. Pakistan faces a high cardiovascular burden and important challenges in prevention, access to care, and risk-factor management [2,3]. Dyslipidaemia is common internationally, supporting the importance of characterising lipid abnormalities in populations at cardiovascular risk [4]. Smoking may influence cardiovascular biology through multiple pathways. Cigarette smoke promotes reactive oxygen species formation and disturbs antioxidant defences, while smoke exposure can alter autonomic regulation [5,6]. Published analyses have linked smoking with higher triglyceride and total cholesterol concentrations and lower HDL concentrations [7], and more recent dose-response research in men has examined smoking exposure in relation to lipid profiles [8]. A cross-sectional study by Sultan and Lesloom assessed smoking alongside lipids, anthropometric measures, and cardiovascular biomarkers, illustrating the value of examining these domains together [9]. BMI, lipid concentrations, and enzyme activity describe different aspects of clinical status, and comparisons across disease and smoking strata may help characterise these patterns in a Pakistani hospital population. Nevertheless, AST and LDH require cautious interpretation because differences in their serum concentrations do not, by themselves, identify a myocardial source or demonstrate diagnostic performance. This study therefore aimed to compare BMI, lipid parameters, AST, and LDH between MI patients and age-comparable healthy controls, stratified by smoking status, and to describe age-stratified smoking comparisons. An additional complete-case analysis explored correlations within the MI group. Troponin-I was not measured.

Methods

This hospital-based case-control study was conducted at Khalifa Gul Nawaz Teaching Hospital, Medical Teaching Institution (MTI), Bannu, Khyber Pakhtunkhwa, Pakistan. from 11 June 2025 to 20 October 2025. The study enrolled 300 participants: 150 patients diagnosed with MI and 150 age-comparable healthy controls. Participants older than 30 years were eligible, and cases and controls were further categorized by smoking status.

Written informed consent was obtained from all participants. Smokers were defined as individuals who had smoked at least once weekly during the preceding year, while non-smokers were defined as individuals with no history of smoking. Individuals with major metabolic, renal, hepatic, or endocrine disorders, substance abuse, or relevant medication use were excluded according to the study protocol. After overnight fasting, venous blood samples were collected, serum was separated, and samples were stored at 4°C until biochemical analysis. Serum total cholesterol, triglycerides, HDL, AST, and LDH were measured using commercially available colorimetric assay kits (Abcam, USA) on a Microlab 200 biochemical analyser. LDL was calculated using the Friedewald formula: LDL cholesterol = total cholesterol – HDL cholesterol – triglycerides/5, with all lipid concentrations expressed in mg/dL [10]. BMI was calculated as weight in kilograms divided by height in metres squared [11]. Continuous variables are presented as mean ± standard deviation. The reported analyses used the Mann-Whitney U test, Kruskal-Wallis test, and analysis of variance, as appropriate. A nominal p-value below 0.05 was

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considered statistically significant. Comparisons were stratified by smoking status and age (<50, 50–60, and >60 years). Pearson correlation coefficients were reported for a complete-case subset of 53 MI patients, comprising 22 smokers and 31 non-smokers. Correlations are treated as exploratory; no adjusted effect estimates or multiplicity-adjusted p-values were reported.

Results

The study included 150 patients with MI and 150 age-comparable healthy controls. BMI did not differ between cases and controls overall (reported $p = 0.857$), among smokers ($p = 0.173$), or among non-smokers ($p = 0.415$). Total cholesterol and LDL concentrations were higher in MI patients in each smoking stratum. Among smokers, total cholesterol was 223.05 ± 43.56 mg/dL in MI patients and 205.99 ± 60.92 mg/dL in controls ($p = 0.007$), while LDL was 137.05 ± 34.70 mg/dL and 98.57 ± 22.89 mg/dL, respectively ($p < 0.001$).

Triglycerides were higher in MI patients overall ($p = 0.016$) and among smokers (215.50 ± 30.73 versus 155.92 ± 52.23 mg/dL; $p < 0.001$), but did not differ among non-smokers ($p = 0.401$). HDL was higher in MI patients overall and among non-smokers (both $p < 0.001$), with no statistically significant difference among smokers ($p = 0.348$). In age-stratified comparisons, smokers had lower BMI than non-smokers in all three age groups (all $p < 0.001$). Total cholesterol was higher among smokers aged <50 years (243.13 ± 78.36 versus 169.67 ± 17.35 mg/dL; $p = 0.005$), while differences in the 50–60-year and >60-year strata did not reach the nominal 0.05 threshold ($p = 0.257$ and $p = 0.066$, respectively). Triglycerides were higher among smokers aged <50 years ($p = 0.010$) and 50–60 years ($p = 0.021$), while the difference among those aged >60 years was not statistically significant ($p = 0.055$). HDL was lower in smokers aged >60 years (48.69 ± 6.56 versus 51.80 ± 6.99 mg/dL; $p = 0.009$). LDL did not differ significantly by smoking status in any age group.

AST was higher in MI patients overall and among non-smokers (both $p < 0.001$). Among smokers, the difference between MI patients and controls was not statistically significant (49.23 ± 7.22 versus 46.44 ± 6.42 U/L; $p = 0.103$). LDH was higher in MI patients overall and in both smoking strata (all $p < 0.001$). Among healthy controls, smokers had higher AST and LDH concentrations than non-smokers (both $p < 0.001$). In age-stratified comparisons, AST was higher in smokers in every age group (all $p < 0.001$), and LDH was also higher in smokers in each age group ($p = 0.030$ for <50 years and $p < 0.001$ for both older strata). Correlation analysis included 53 MI cases with complete data: 22 smokers and 31 non-smokers. The reported HDL correlation in non-smokers was negative ($r = -0.376$, adjusted $R^2 = 0.111$, $F = 4.760$, $p = 0.037$) and met the nominal 0.05 threshold. The LDH correlation in smokers was positive but did not reach that threshold ($r = 0.414$, adjusted $R^2 = 0.130$, $F = 4.139$, $p = 0.055$). No statistically significant correlations were reported for the remaining parameters. These findings are exploratory, and the paired variable was not identified in the available analysis specification, limiting interpretation.

Table 1. Cardiometabolic parameters and enzyme concentrations by MI status, smoking status,

A. Cardiometabolic profile by MI and smoking status

Parameter	Stratum	Controls Mean $\pm$ SD	MI patients Mean $\pm$ SD
BMI (kg/m ²)	Overall	23.79 $\pm$ 3.80	23.89 $\pm$ 4.02
BMI (kg/m ²)	Smokers	21.44 $\pm$ 3.96	20.71 $\pm$ 4.01
BMI (kg/m ²)	Non-smokers	25.84 $\pm$ 2.10	26.15 $\pm$ 2.01
Cholesterol (mg/dL)	Overall	188.83 $\pm$ 47.18	198.93 $\pm$ 40.13
Cholesterol (mg/dL)	Smokers	205.99 $\pm$ 60.92	223.05 $\pm$ 43.56
Cholesterol (mg/dL)	Non-smokers	173.95 $\pm$ 21.83	181.81 $\pm$ 27.10
Triglycerides (mg/dL)	Overall	140.49 $\pm$ 42.86	161.64 $\pm$ 53.90
Triglycerides (mg/dL)	Smokers	155.92 $\pm$ 52.23	215.50 $\pm$ 30.73
Triglycerides (mg/dL)	Non-smokers	127.11 $\pm$ 26.28	123.42 $\pm$ 27.16
HDL (mg/dL)	Overall	51.14 $\pm$ 7.09	57.83 $\pm$ 12.84
HDL (mg/dL)	Smokers	50.01 $\pm$ 7.06	51.55 $\pm$ 7.17
HDL (mg/dL)	Non-smokers	52.13 $\pm$ 7.00	62.29 $\pm$ 14.15
LDL (mg/dL)	Overall	97.63 $\pm$ 21.66	131.89 $\pm$ 33.43
LDL (mg/dL)	Smokers	98.57 $\pm$ 22.89	137.05 $\pm$ 34.70
LDL (mg/dL)	Non-smokers	96.82 $\pm$ 20.59	128.23 $\pm$ 32.57

B. Cardiometabolic profile by smoking and age

Parameter	Stratum	Smokers Mean $\pm$ SD	Non-smokers Mean $\pm$ SD
BMI (kg/m ²)	<50 years	22.06 $\pm$ 2.13	26.16 $\pm$ 2.10
BMI (kg/m ²)	50–60 years	23.09 $\pm$ 2.61	25.73 $\pm$ 2.70
BMI (kg/m ²)	>60 years	20.20 $\pm$ 4.76	25.82 $\pm$ 1.90
Cholesterol (mg/dL)	<50 years	243.13 $\pm$ 78.36	169.67 $\pm$ 17.35
Cholesterol (mg/dL)	50–60 years	189.78 $\pm$ 39.68	175.41 $\pm$ 26.59
Cholesterol (mg/dL)	>60 years	199.22 $\pm$ 56.79	174.23 $\pm$ 21.02
Triglycerides (mg/dL)	<50 years	183.38 $\pm$ 62.23	125.80 $\pm$ 24.87
Triglycerides (mg/dL)	50–60 years	152.59 $\pm$ 46.10	126.37 $\pm$ 23.92

Triglycerides (mg/dL)	>60 years	145.87 ± 47.36	127.57 ± 27.47
HDL (mg/dL)	<50 years	50.88 ± 6.94	53.87 ± 6.65
HDL (mg/dL)	50–60 years	51.63 ± 7.72	52.19 ± 7.32
HDL (mg/dL)	>60 years	48.69 ± 6.56	51.80 ± 6.99
LDL (mg/dL)	<50 years	96.04 ± 25.55	93.80 ± 11.84
LDL (mg/dL)	50–60 years	102.31 ± 23.39	98.82 ± 24.82
LDL (mg/dL)	>60 years	97.49 ± 21.52	96.72 ± 20.48

C. Enzyme concentrations by MI status, smoking, and age

Parameter	Stratum	Controls Mean ± SD	MI patients Mean ± SD
AST (U/L)	Overall	39.13 ± 12.12	47.66 ± 6.62
AST (U/L)	Smokers	46.44 ± 6.42	49.23 ± 7.22
AST (U/L)	Non-smokers	32.78 ± 12.33	46.55 ± 6.04
LDH (U/L)	Overall	137.80 ± 39.50	181.74 ± 12.77
LDH (U/L)	Smokers	170.21 ± 24.84	189.09 ± 7.26
LDH (U/L)	Non-smokers	109.70 ± 25.99	176.52 ± 13.33

Parameter	Age stratum	Smokers Mean ± SD	Non-smokers Mean ± SD
AST (U/L)	<50 years	47.13 ± 6.96	28.33 ± 7.18
AST (U/L)	50–60 years	45.81 ± 4.86	25.33 ± 9.81
AST (U/L)	>60 years	46.51 ± 7.03	35.90 ± 12.58
LDH (U/L)	<50 years	145.88 ± 26.33	126.27 ± 19.02
LDH (U/L)	50–60 years	170.06 ± 26.72	106.26 ± 27.72
LDH (U/L)	>60 years	180.91 ± 13.13	107.90 ± 25.67

Exploratory correlations in MI cases with complete data

Parameter	Cohort	df	Pearson r	Adj. R ²	F
BMI	Smokers	20	0.005	−0.050	<0.001
BMI	Non-smokers	29	0.119	−0.020	0.417
Cholesterol	Smokers	20	0.134	−0.031	0.367
Cholesterol	Non-smokers	29	−0.098	−0.025	0.283
Triglycerides	Smokers	20	0.172	−0.019	0.613
Triglycerides	Non-smokers	29	0.042	−0.033	0.052

HDL	Smokers	20	0.235	0.008	1.164
HDL	Non-smokers	29	-0.376	0.111	4.760
LDL	Smokers	20	-0.208	-0.005	0.905
LDL	Non-smokers	29	-0.099	-0.024	0.285
AST	Smokers	20	-0.083	-0.043	0.138
AST	Non-smokers	29	0.088	-0.026	0.227
LDH	Smokers	20	0.414	0.130	4.139
LDH	Non-smokers	29	0.073	-0.029	0.155

Discussion

This case-control study of 150 MI patients and 150 age-comparable controls identified higher total cholesterol and LDL concentrations in MI patients across smoking strata, while BMI did not differ significantly between cases and controls. Triglycerides were higher among MI patients who smoked, whereas HDL was unexpectedly higher in MI patients overall and among non-smokers. LDH was higher in MI patients across strata, while the AST difference was less consistent. These findings describe group differences at the time of measurement; the study design does not establish whether the measured biochemical differences preceded MI or resulted from the acute event, treatment, or other differences between participants.

The higher LDL concentrations in MI patients are consistent with the established role of LDL in atherosclerotic cardiovascular disease, supported by genetic, epidemiological, and intervention evidence [12]. In this cohort, the LDL difference was numerically larger among smokers: 137.05 ± 34.70 mg/dL in MI patients versus 98.57 ± 22.89 mg/dL in controls. However, the subgroup comparisons do not demonstrate that smoking modifies the association between LDL and MI, and that interpretation would require a formal interaction analysis and adjustment for relevant covariates. Likewise, the present data cannot establish an independent association between LDL and MI because adjusted effect estimates were not provided.

Triglycerides were substantially higher in MI patients who smoked than in smoking controls, but no significant case-control difference appeared among non-smokers. This pattern is compatible with published evidence linking smoking to an adverse lipid profile [7], yet the present comparisons do not isolate a smoking effect. Differences in diet, diabetes, alcohol use, medication exposure, fasting status, and smoking intensity could contribute to the observed pattern, and the absence of a significant triglyceride difference in non-smokers should not be interpreted as evidence that triglycerides have no relevance in that subgroup.

BMI did not distinguish MI patients from controls, although smokers had lower BMI than non-smokers in every age stratum. A non-significant difference in mean BMI does not exclude a contribution of adiposity to cardiovascular risk, and INTERHEART showed that waist-to-hip ratio had a stronger association with MI than BMI, illustrating the importance of fat distribution [13]. The lower BMI among smokers is also compatible with literature describing nicotine-related effects on energy expenditure and appetite [14], although these mechanisms remain possible explanations rather than demonstrated pathways in this cohort.

The age-stratified analyses showed higher cholesterol among smokers aged <50 years and higher triglycerides among smokers aged <50 and 50–60 years, while LDL did not differ significantly by smoking status within any age group. These findings should not be interpreted as showing that smoking-related lipid differences disappear with age, because a significant result in one stratum and a non-significant result in another does not, by itself, establish a difference between strata [15]. Unequal subgroup sizes and variability could affect precision, and the population represented by the age tables must be clarified before the findings can be attributed specifically to controls, MI patients, or the combined cohort.

The higher HDL concentrations in MI patients, particularly non-smokers, require careful interpretation because HDL concentration alone does not establish the functional properties of HDL particles. Voight and colleagues found that genetically higher HDL did not necessarily translate into lower MI risk [16], while Rohatgi and colleagues demonstrated an inverse association between cholesterol efflux capacity and incident cardiovascular events [17]. These studies support distinguishing HDL quantity from HDL function, but they do not prove that HDL was dysfunctional in the present patients; no functional HDL assessment was reported, and selection effects, medication use, sex distribution, sampling conditions, or measurement issues are alternative explanations that should be examined.

LDL was higher in MI patients across smoking strata, whereas AST did not differ significantly between smoking cases and smoking controls. Smokers among healthy controls also had higher AST and LDH than non-smokers. These results demonstrate biochemical differences but do not identify their tissue source or establish diagnostic accuracy. In particular, AST and LDH should not be presented as validated substitutes for cardiac troponin, because the Fourth Universal Definition of MI bases acute myocardial injury on cardiac troponin changes and requires evidence of myocardial ischaemia to diagnose MI [18]. Without assay-specific reference intervals, serial sampling, information on non-cardiac illness, and an assessment of haemolysis, the present enzyme findings cannot be attributed exclusively to myocardial injury.

The correlation findings provide limited additional evidence. Only 53 of the 150 MI cases contributed complete data, comprising 22 smokers and 31 non-smokers. HDL in non-smokers met the nominal significance threshold ($r = -0.376$, $p = 0.037$), whereas LDH in smokers did not ($r = 0.414$, $p = 0.055$), and the latter should not be described as a statistically significant relationship. Because several correlations were examined without a reported multiplicity adjustment, the isolated nominal finding may reflect chance and needs replication [19]. More fundamentally, the variable correlated with each biochemical parameter has not been identified, preventing biological interpretation of either direction.

Several limitations affect interpretation. The case-control design limits causal inference and is susceptible to selection and residual confounding, and age comparability alone does not account for sex, diabetes, hypertension, diet, alcohol use, smoking duration, or treatment differences. The timing of blood collection relative to symptom onset and lipid-lowering treatment is also important, and the LUNAR analysis specifically evaluated lipid measurements during the first four days after acute coronary syndromes, underscoring the need to describe the sampling window rather than assume that post-event measurements represent usual pre-event levels [20]. In addition, subgroup sample sizes, test selection, distributional assumptions, and confidence intervals were not supplied; these details, the correlation counterpart, and the handling of missing data should be reported in accordance with observational reporting guidance [21].

The reported LDL means also require reconciliation with the stated calculation method: because the

Friedewald equation is linear, group means based on the same participants should satisfy the equation apart from rounding, yet using the overall means gives approximately 109.59 mg/dL in controls and 108.77 mg/dL in MI patients, compared with the reported LDL means of 97.63 and 131.89 mg/dL [10]. The original records are needed to establish whether different denominators, another assay or calculation method, or a reporting error explains this discrepancy, so LDL-related interpretation remains provisional. Overall, the study describes higher LDL and total cholesterol concentrations in MI patients, a marked triglyceride difference among smokers, and higher LDH concentrations in cases. Similar BMI between cases and controls does not negate the possible relevance of adiposity, while the higher HDL finding remains unresolved. The results justify further investigation using clearly defined populations, standardized sampling, verified analyses, and adjusted estimates with confidence intervals, but they do not establish smoking-specific mechanisms, predictive performance, or a new diagnostic role for AST or LDH. Despite these limitations, smoking cessation remains an evidence-based component of cardiovascular prevention, and a Cochrane review found lower risks of subsequent cardiovascular events among people who stopped smoking compared with those who continued [22].

Conclusion

MI patients had higher reported total cholesterol, LDL, and LDH concentrations than controls across smoking strata, while BMI was similar. The case-control triglyceride difference was marked among smokers, and the higher HDL concentration in MI patients remains unexplained. The findings are descriptive and do not establish independent effects of smoking, age interactions, or a diagnostic role for AST and LDH. LDL results require reconciliation with the stated calculation method, and the complete-case correlations remain exploratory.

Recommendations

Smoking-cessation support should form part of cardiovascular prevention and follow-up care in line with the wider evidence base [22]. Future studies should quantify smoking exposure, define case ascertainment and sampling times clearly, include appropriate cardiac troponin assessment, and report adjusted estimates with confidence intervals. Larger prospective studies are needed before considering population-specific thresholds or predictive applications, and the present data do not justify new smoking-adjusted lipid or enzyme cut-offs.

Authors' contributions:

Fakhre Islam contributed to conceptualization, study design, methodology, data collection, data curation, and original manuscript drafting. Qismat Ullah contributed to patient recruitment, clinical assessment, data collection, and manuscript review. Farman Ullah contributed to biochemical investigations, laboratory analysis, quality control, and interpretation of laboratory findings. Muhammad Bilal Shah contributed to methodology, research supervision, data validation, and critical manuscript revision. Ahmad Ehsan Rabani contributed to data interpretation, scientific review, manuscript editing, and Faisal Nadeem Correspondence, overall supervision critical revision.

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