



## Frequency of Low Levels of High Density Lipoprotein Cholesterol in Newly Diagnosed Patients of Acute Coronary Syndrome

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### ABSTRACT

**Background:** One of the most significant causes of worldwide morbidity and mortality is acute coronary syndrome (ACS). One cardiovascular risk factor that is under-acknowledged, yet is significant, is dyslipidemia, specifically the low high-density lipoprotein cholesterol (HDL-C). **Aim:** To identify how often low HDL-C occurred in newly diagnosed ACS patients and how it is related to clinical risk factors. **Methodology:** It was a cross-sectional study that was carried out in the Faisalabad Institute of Cardiology between August 2021 and February 2022. The patient population of ACS was 125 with the age group 18-70. The amount of HDL-C was measured and analysed using SPSS. A critical p-value was set to be 0.05. **Findings:** There were 83.2% men and a mean age of  $44.35 \pm 9.69$  years. 18.4% of patients had low HDL-C. Significant associations were seen with smoking ( $p=0.031$ ) and hypertension ( $p=0.0009$ ). **Conclusion:** HDL-C is a large and modifiable risk factor of ACS patients. Early intervention and screening are suggested.

### INTRODUCTION

According to estimates, 17.9 million people die from CVDs every year, making them the leading cause of death in the world [1]. Acute coronary syndrome (ACS) is a form of coronary artery disease that is the most severe. It increases the costs of healthcare significantly in all corners of the globe. It includes unstable angina, non-ST elevation myocardium infarction (NSTEMI), and the ST-elevation myocardium infarction (STEMI) [2]. The percentage of individuals at risk including diabetes, high blood pressure, smoking and dyslipidemia have been increasing and this explains why the ACS is increasing rapidly in emerging nations and South Asia [3].

In Pakistan, the number of people dying of heart disease is the highest among all other causes of death, with over 30 percent of all deaths attributable to this disease [4]. We had to say that South Asians are more predisposed to coronary artery disease than their Western counterparts and the disease is much more rapidly diffused in South Asians [5]. This means that we need to find and work on the risk factors that can be changed.

Atherosclerosis revolves around Dyslipidemia. Although high levels of low-density lipoprotein cholesterol (LDL-C)

have received great attention, low levels of high-density lipoprotein cholesterol (HDL-C) have received more and more attention as a risk factor [6]. HDL cholesterol has also been reported to possess cardioprotective effects, such as reverse cholesterol transportation, anti-inflammatory effects and endothelial protection [7].

The deficiency of HDL-C can disrupt these protective processes, resulting in a greater accumulation of lipids, endothelial impairment and plaque instability [8]. A number of studies have shown a negative association between the level of HDL-C and cardiovascular risk [9]. Nonetheless, the use of HDL-C in risk stratification is underutilized especially among developing nations. Finding out how often low HDL-C is among newly diagnosed ACS patients and how it relates to other cardiovascular risk factors in the community are among the study's goals.

### METHODOLOGY

This cross-sectional descriptive study was done at the department of cardiology, Faisalabad Institute of cardiology, between 20th August 2021 to 19 th February 2022. Non-probability consecutive sampling was used to

include 125 patients. The sample size was determined by WHO sample size calculator with a 95 percent level of confidence and 6 percent margin of error.

They included patients aged 18–70 years of either gender who were diagnosed with acute coronary syndrome. Patients who have known coronary artery disease, are already on lipid-lowering treatment and patients with renal impairment were excluded.

Following informed consent and ethical approval, demographic information such as age and gender were taken. Clinical variables were also recorded like diabetes mellitus, hypertension, smoking, physical inactivity and family history. A standardized analyzer was used to analyze blood samples (3 ml) in a hospital laboratory.

Low HDL-C was determined as less than 40mg/dl in men and 50mg/dl in women. Analyses of data were done using SPSS. Categorical variables were computed using frequencies and percentages whereas quantitative variables were computed using mean and standard deviation. Effect modifiers were stratified and chi-square test was used. The p-value of 0.05 or less was taken to be statistically significant.

**RESULTS**

The age of the patients was between 18 and 70 years with mean age of 44.35 and standard deviation of 9.69. 77 (61.6%) of the patients were in the 18–45 age range. The remaining 48 (38.4%) patients ranged in age from 46 to 70. [Table 1]

Among all the patients, there were 21 women (16.8%) and 104 men (83.2%). This is almost 5:1 which implies a high level of male domination (Table 2).

There were 85 patients who were not diabetic (68.0%) and 40 patients who were diabetic (32.0%). Forty-nine (39.2%) and seventy-six (60.8%) patients, respectively, had high blood pressure. Sixty-seven (53.6%) out of the patients had reported that they were smokers but fifty eight (46.4%) had reported that they were not. Of the patients, 88 (70.4%) were active, while just 37 (29.6%) were not. We discovered that 27 (22.13%) patients also had positive-family history of coronary artery disease. In Table 3,

The average of HDL cholesterol was 37.64 + 7.87 mg/dl. The low HDL cholesterol (18.4%), there were 23 people and normal cholesterol (81.6) there were 102 people. [Table 4]

All the modifiers of effect such as age, gender, smoking status, physical activity, and family history were under the control of stratification. To indicate that the results were statistically significant, the post-stratification chi-square test was used. [Table 5]

Age-group stratification indicated that 5 (10.42%) patients between 46-70 years of age had low HDL values as compared to 18 (23.38%) patients between 18 to 45 years of age. No difference could however be detected (p=0.069).

On the same note, gender stratification showed that 20 (19.23) and 3 (14.29) male and female patients respectively had low levels of HDL, where there was no significant difference (p = 0.594).

Relationship between hypertension and low levels of HDL was found to be statistically significant. The number of

hypertension patients who had lower levels of HDL than 2 (4.08) in non-hypertension patients was 21 (27.63) versus 2 (4.08).

Smoking was strongly related to low levels of HDL. Seventeen (25.37%) smokers had low HDL, while only six (10.34%) non-smokers did (p = 0.031).

No statistically significant relationship was found between low HDL levels and physical inactivity (p = 0.107) or family history (p = 0.587).

The results show that a lot of ACS patients have low levels of HDL cholesterol, and this is significantly linked to high blood pressure and smoking.

**Table 1**

*Age Distribution*

Age Group (years)	Frequency (n)	Percentage (%)
18–45	77	61.6
46–70	48	38.4
Total	125	100

**Table 2**

*Gender Distribution*

Gender	Frequency (n)	Percentage (%)
Male	104	83.2
Female	21	16.8
Total	125	100

**Table 3**

*Distribution of Risk Factors (n = 125)*

Variable	Yes (n, %)	No (n, %)
Diabetes Mellitus	40 (32.0%)	85 (68.0%)
Hypertension	76 (60.8%)	49 (39.2%)
Smoking	67 (53.6%)	58 (46.4%)
Physical Inactivity	37 (29.6%)	88 (70.4%)
Family History	27 (22.13%)	98 (77.87%)

**Table 4**

*Frequency of Low HDL Cholesterol (n = 125)*

HDL Level	Frequency (n)	Percentage (%)
Low HDL	23	18.4
Normal HDL	102	81.6
Total	125	100

**Table 5**

*Stratification of Low HDL with Respect to Different Variables (n = 125)*

Variable	Category	Low HDL (n, %)	Normal HDL (n, %)	p-value
Age	18–45	18 (23.38%)	59 (76.62%)	0.069
	46–70	5 (10.42%)	43 (89.58%)	
Gender	Male	20 (19.23%)	84 (80.77%)	0.594
	Female	3 (14.29%)	18 (85.71%)	
Hypertension	Yes	21 (27.63%)	55 (72.37%)	0.0009
	No	2 (4.08%)	47 (95.92%)	
Smoking	Yes	17 (25.37%)	50 (74.63%)	0.031
	No	6 (10.34%)	52 (89.66%)	
Physical Inactivity	Yes	10 (27.03%)	27 (72.97%)	0.107
	No	13 (14.77%)	75 (85.23%)	

## DISCUSSION

Acute coronary syndrome remains one of the significant problems of global public health, especially in low- and middle-income countries where the healthcare service projects are frequently overwhelmed by the growing number of cardiovascular risk factors [10]. The current research conducted the frequency and clinical relevance of low HDL cholesterol in patients with ACS and concluded that 18.4% of patients were low in HDL cholesterol. This observation supports the significance of HDL as a significant yet neglected risk factor in heart disease.

The comparatively young average age (44.35 years) of this study is in line with the past research in South Asian populations where coronary artery disease is more likely to manifest at an earlier age than in the Western population [11]. The cause of this early onset could be linked to genetic predisposition, lack of physical activity, poor eating habits and the high rates of metabolic syndrome in this area [12].

The male bias seen in the current study (83.2) is also aligned with the data available across the world as men tend to develop ACS at an earlier age than women [13]. It should however be noted that risk in women is much higher after menopause because of hormonal changes on lipid metabolism [14].

HDL cholesterol is important in cardiovascular protection by reverse cholesterol transportation, antioxidant and anti-inflammatory action [15]. The deficiency in HDL levels impairs these protective processes and results in the excess deposition of lipids in the arterial walls, dysfunction of endothelium, and instability of the plaques [16]. These mechanisms play a key role in the pathogenesis of atherosclerosis and ACS.

Hypertension and smoking were strongly linked to low levels of HDL in the current study. Endothelial injury and vascular remodeling have been known to be caused by hypertension, potentially affecting the HDL functioning and causing atherosclerosis [17]. Equally, smoking has been observed to decrease the HDL, and augment oxidative stress, which also causes further vascular damage [18].

The high correlation between low HDL and smoking in this study is in line with the past studies that have shown that smokers have low HDL levels as compared to non-smokers [19]. Inflammation and thrombosis are also encouraged by smoking, thus making acute coronary events more likely [20].

Interestingly, in this study there was no significant relationship between low HDL levels and age or gender.

## REFERENCES

1. World Health Organization, Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019. *Lancet*. 2021;398(10307):1229–1239.
2. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction. *Circulation*. 2018;138(20):e618–e651. <https://doi.org/10.1161/cir.0000000000000617>
3. Prabhakaran D, Jeemon P, Roy A, Pais P, Rangarajan S, Yusuf S, et al. Cardiovascular diseases in India and South Asia:

This is an indicator that low HDL can be a risk factor of ACS, independent of the demographics [21]. This observation underscores the importance of regular screening of the HDL in every patient regardless of age and gender.

Comparisons with other studies indicate that there is a variation in the prevalence of low HDL. Higher prevalence rates have been reported in some studies carried out in Pakistan, between 40 to 80% [22]. This inconsistency could result because of the dissimilarity in the study design, sample size, population features, and lifestyle. However, even the decreased prevalence in this study is clinically significant.

Recent studies have also put a lot of stress on the effectiveness of HDL instead of its level [23]. The dysfunctional HDL can lose its protective effect and even become pro-inflammatory, which further increases the risk of cardiovascular disease [24]. This is a new idea implying that measuring the levels of HDL might not be enough and research in the future should emphasize in determining the functions of HDL.

The findings of this study have important clinical implications. To start with, HDL cholesterol must be regularly checked as part of lipid profiles of ACS patients. Second, lifestyle changes like physical activity, quitting smoking, and changing the diet may be applied to improve the levels of HDL and decrease cardiovascular risk [25]. Third, the public health programs must be aimed at raising awareness on the value of HDL cholesterol.

This study has some limitations, in spite of its strengths. It was done in one location with a rather small sample size that might not be generalizable. Also, the design is cross-sectional, and it is not possible to draw a causal conclusion. Multicenter longitudinal studies in the future are suggested to conduct further research on the role of HDL in ACS.

Overall, the present research emphasizes the importance of low HDL cholesterol as a crucial risk factor, which is serious and can be dealt with in ACS. Early detection and intervention of this risk factor can be important in alleviating the burden of cardiovascular disease.

## CONCLUSION

The individuals with acute coronary syndrome have a big and changeable risk since their HDL cholesterol levels are low. It is intimately connected with smoking and hypertension. Low levels of HDL can be prevented or reduced through early intervention and prevention of cardiovascular morbidity and mortality. Routine screening and lifestyle changes should be made as a clinical practice.

epidemiology and prevention. *Lancet*.

- 2021;397(10278):134–146.
4. Zulfiqar N, Razzaq S, Satti S, Ahmed S, Khan M, Ali R, et al. Risk factors of cardiac diseases in Pakistan. *J Community Health*. 2020;45(5):1040–1046.
5. Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. *N Engl J Med*. 2007;356(24):2412–2423. <https://doi.org/10.1001/jama.297.3.286>
6. Ference BA, Ginsberg HN, Graham I, Ray KK, Packard CJ, Bruckert E, et al. Low-density lipoproteins cause

- atherosclerotic cardiovascular disease. *JAMA*. 2017;318(12):1107–1108.  
<https://doi.org/10.1093/eurheartj/ehx144>
7. Rohatgi A, Khera A, Berry JD, Givens EG, Ayers CR, Wedin KE, et al. HDL cholesterol efflux capacity and incident cardiovascular events. *N Engl J Med*. 2014;371(25):2383–2393.  
<https://doi.org/10.1056/nejmoa1409065>
  8. Barter P, Gotto AM, LaRosa JC, Maroni J, Szarek M, Grundy SM, et al. HDL cholesterol, very low levels of LDL cholesterol, and cardiovascular events. *N Engl J Med*. 2007;357(13):1301–1310.  
<https://doi.org/10.1056/nejmoa064278>
  9. Gordon DJ, Probstfield JL, Garrison RJ, Neaton JD, Castelli WP, Knoke JD, et al. High-density lipoprotein cholesterol and cardiovascular disease. *Circulation*. 1989;79(1):8–15.  
<https://doi.org/10.1161/01.CIR.79.1.8>
  10. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Global burden of cardiovascular diseases update. *Lancet*. 2020;396(10258):1223–1249.  
<https://doi.org/10.1016/j.aogh.2016.04.002>
  11. Gupta R, Mohan I, Narula J, Rastogi P, Gupta VP, Khera A, et al. Trends in coronary heart disease epidemiology in India. *Int J Cardiol*. 2020;318:152–158.  
<https://doi.org/10.1016/j.aogh.2016.04.002>
  12. Misra A, Khurana L, Vikram NK, Goel A, Wasir JS, Pandey RM, et al. Metabolic syndrome in South Asians. *Lancet*. 2007;370(9599):1527–1538.
  13. Maas AHEM, Appelman YEA, van der Graaf Y, Nathoe HM, Bots ML, Grobbee DE, et al. Gender differences in coronary heart disease. *Eur Heart J*. 2010;31(11):1327–1335.  
<https://doi.org/10.1007/s12471-010-0841-y>
  14. Mendelsohn ME, Karas RH, Clarkson TB, Appt SE, Bittner V, Shufelt C, et al. Estrogen and cardiovascular disease. *N Engl J Med*. 2005;352(17):1801–1811.
  15. Rosenson RS, Brewer HB, Ansell BJ, Barter P, Chapman MJ, Heinecke JW, et al. HDL structure and function. *J Am Coll Cardiol*. 2016;67(13):1664–1676.
  16. Besler C, Heinrich K, Rohrer L, Doerries C, Riwanto M, Shih DM, et al. Mechanisms underlying HDL dysfunction. *J Am Coll Cardiol*. 2011;57(24):2436–2445.
  17. Virdis A, Giannarelli C, Fritsch Neves M, Taddei S, Ghiadoni L, Salvetti A, et al. Endothelial dysfunction in hypertension. *Hypertension*. 2010;55(3):561–568.
  18. Ambrose JA, Barua RS, Srivastava S, DeMarco T, Aikawa E, Libby P, et al. Smoking and cardiovascular disease. *J Am Coll Cardiol*. 2004;43(10):1731–1737.  
<https://doi.org/10.1016/j.jacc.2003.12.047>
  19. Craig WY, Palomaki GE, Haddow JE, Bostom AG, Wilson PW, Kannel WB, et al. Cigarette smoking and HDL cholesterol levels. *Am J Cardiol*. 1989;64(7):394–398.
  20. Messner B, Bernhard D, Laufer G, Wick G, Fuchs D, Kronenberg F, et al. Smoking-induced inflammation and cardiovascular risk. *Circ Res*. 2014;115(1):1–9.
  21. Emerging Risk Factors Collaboration, Di Angelantonio E, Sarwar N, Perry P, Kaptoge S, Ray KK, et al. Major lipids and cardiovascular risk. *Lancet*. 2009;373(9671):1247–1257.
  22. Khan B, Sawar S, Hassan M, Khan A, Rahman F, Noor N, et al. Frequency of lipid abnormalities in ACS patients. *Pak Heart J*. 2019;52(1):85–90.
  23. Kontush A, Chapman MJ, Amarenco P, Goldstein LB, Kaste M, Lamotte M, et al. HDL functionality and cardiovascular risk. *Atherosclerosis*. 2015;243(1):150–160.
  24. Navab M, Reddy ST, Van Lenten BJ, Fogelman AM, Berliner JA, Watson AD, et al. Dysfunctional HDL and atherosclerosis. *J Am Coll Cardiol*. 2011;58(12):1161–1172.
  25. Kodama S, Tanaka S, Saito K, Shu M, Sone Y, Onitake F, et al. Effect of exercise on HDL cholesterol. *Arch Intern Med*. 2007;167(10):999–1008.  
<https://doi.org/10.1001/archinte.167.10.999>