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ANALYSIS OF LOW HDL LEVELS AS A MAJOR RISK FACTOR OF ACUTE MYOCARDIAL INFARCTION IN PAKISTAN

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Abstract:

Introduction: *Myocardial infarction (MI) remains a leading cause of death worldwide. Myocardial ischemia occurs as a result of plaque build up in the coronary arteries, a disease formally known as atherosclerosis or coronary artery disease (CAD).*

Aims and objectives: *The basic aim of the study is to analyse the low HDL levels as a major risk factor of acute myocardial infarction in Pakistan.*

Material and methods: *This cross sectional study was conducted in DHQ teaching hospital Sahiwal during 2018 to 2019. The study was conducted according to the ethical committee of the hospital. The data was collected from 100 patients of both genders. The data was collected from those patients who visited the OPD of the hospital regularly. TC level in serum was measured using the endpoint test method.*

Results: *The data was collected from 100 patients of both genders. The mean age was 45±5.46 years. The proportion of male subjects was higher in groups with high TG levels, while the difference in age was not statistically significant. With the increase in TG level, the proportion of people with a history of smoking increased, body mass index (BMI), SBP, DBP, FBG, UA and the rate of MI increased, while HDL-C level gradually decreased, and the differences were all statistically significant. Differences in TC and LDL-C levels were not statistically significant.*

Conclusion: *It is concluded that low HDL level is noted to be present in a high percentage of acute myocardial infarction patients and can be a major risk contributor.*

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INTRODUCTION:

Myocardial infarction (MI) remains a leading cause of death worldwide. An acute MI occurs when myocardial ischemia exceeds a critical threshold, usually due to an acute plaque rupture in the coronary arteries, and the cellular cascade of events overwhelms myocardial cellular repair mechanisms leading to myocardial cell damage. Myocardial ischemia occurs as a result of plaque build up in the coronary arteries, a disease formally known as atherosclerosis or coronary artery disease (CAD) [1]. Rupturing of vulnerable atherosclerotic plaque follows a period of continual plaque destabilization and/or plaque growth due to various patho-biological processes. Plaque contents are enclosed within a stabilizing fibrous cap that prevents exposure of the thrombogenic core to the bloodstream, and weakening of this cap can therefore lead to plaque rupture and MI [2].

Cardiovascular disease (CVD) is the leading cause of death worldwide, which has become a worldwide public health problem. Acute myocardial infarction (AMI) is a common clinical critical illness. In the past few decades, significant progress has been made in understanding, preventing and controlling this disease [3]. In particular, the rise of reperfusion therapy significantly reduced mortality and improved the prognosis of AMI. In recent years, the role of low density lipoprotein density (LDL-C) in the pathogenesis of atherosclerosis (AS) has attracted much attention [4]. However, more and more clinical trials have revealed that after controlling for deterministic risk factors such as LDL-C, the risk for coronary heart disease (CHD) remained, while the increase in triglycerides (TG) was significantly correlated with the increase in mortality, the incidence of myocardial infarction (MI) and the recurrence rate of coronary artery disease [5].

Aims and objectives:

The basic aim of the study is to analyse the low HDL levels as a major risk factor of acute myocardial infarction in Pakistan.

MATERIAL AND METHODS:

This cross sectional study was conducted in DHQ teaching hospital Sahiwal during 2018 to 2019. The study was conducted according to the ethical committee of the hospital. The data was collected from 100 patients of both genders. The data was collected from those patients who visited the OPD of the hospital regularly. TC level in serum was measured using the endpoint test method. HDL-C and LDL-C were measured using the direct test method. TG was measured using the GPO method. Non-HDL-C level was determined by subtracting serum HDL-C from serum TC.

Statistical analysis:

Data were collected using the software Epidata 3.0 and analyzed using statistical software SPSS 18.0. Measurement data were expressed as mean ± standard deviation (SD). Intergroup comparison was conducted using analysis of variance.

RESULTS:

The data was collected from 100 patients of both genders. The mean age was 45±5.46 years. The proportion of male subjects was higher in groups with high TG levels, while the difference in age was not statistically significant. With the increase in TG level, the proportion of people with a history of smoking increased, body mass index (BMI), SBP, DBP, FBG, UA and the rate of MI increased, while HDL-C level gradually decreased, and the differences were all statistically significant. Differences in TC and LDL-C levels were not statistically significant.

Table 01: Logistic regression analysis to identify predictors of periprocedural myocardial infarction

Variables	Univariate		Multivariate	
	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Age	1.01 (0.97–1.04)	0.75	1.01 (0.96–1.05)	0.79
Sex, male	0.66 (0.26–1.67)	0.38		
Body mass index	0.98 (0.89–1.09)	0.73		
Current smoking	1.63 (0.70–3.82)	0.26	2.46 (0.87–6.95)	0.090
Hypertension	0.77 (0.34–1.75)	0.53		
Diabetes mellitus	0.77 (0.37–1.58)	0.48	0.65 (0.27–1.58)	0.34
eGFR	0.99 (0.97–1.01)	0.48	0.98 (0.96–1.01)	0.22
LDL cholesterol	1.00 (0.99–1.02)	0.63		
HDL cholesterol	0.97 (0.93–1.00)	0.058		
HDL2 cholesterol	0.97 (0.92–1.02)	0.26		
HDL3 cholesterol	0.86 (0.76–0.98)	0.018	0.86 (0.74–0.99)	0.038
Triglyceride	1.00 (0.99–1.01)	0.16		

C-reactive protein	1.18 (0.90–1.54)	0.22		
Total stent length	1.06 (1.01–1.10)	0.011	1.04 (0.99–1.09)	0.16
Total inflation time	1.01 (1.00–1.01)	0.014	1.00 (0.99–1.01)	0.22

DISCUSSION:

The well-known "HDL hypothesis" suggests that therapies aimed at raising HDL-C concentrations will lower the risk of CAD and MI. In a widely cited meta-analysis of four large studies (total number of individuals studied: 15,252), a 1 mg/dL increase of HDL-C levels was reported to be associated with a 2%–3% decreased CVD risk [6]. Niacin, presently prescribed with a statin, is one of the most commonly used pharmacological therapy aimed at raising HDL-C concentrations in patients with such risks. At a pharmacological dose of ~1.5–2 g per day, Niacin is one of the most potent agents available for this purpose. Niacin also reduces all proatherogenic lipids and lipoproteins, including total cholesterol, TGs, very-low-density lipoprotein, LDL, and lipoprotein [7]. Despite its popularity, the efficacy of niacin has come into question in recent studies. Two distinct studies, Atherosclerosis Intervention in Metabolic Syndrome with Low HDL/High Triglycerides and Impact on Global Health Outcomes (AIM-HIGH) and Heart Protection Study 2 – Treatment of High-density Lipoprotein to Reduce the Incidence of Vascular Events (HPS2-THRIVE) were aimed at evaluating whether adding the modern, extended-release niacin formulations to statin therapy provides incremental benefit over statin therapy alone in terms of cardiovascular primary events in patients with established CAD [8]. These clinical trials studied specific populations of stable ischemic heart disease patients, excluding patients with MI or those with significant residual mixed dyslipidemia not treated with optimal doses of intensive statin therapy [9]. Both the AIM-HIGH and HPS2-THRIVE clinical trials were stopped prematurely due to a lack of beneficial effects and an inability to meet primary endpoints of reduced cardiovascular disease and MI risk [10].

CONCLUSION:

It is concluded that low HDL level is noted to be present in a high percentage of acute myocardial infarction patients and can be a major risk contributor

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