

Low HDL levels as a major risk factor of acute myocardial infarction

Abstract

Introduction: Cardiovascular ~~illness~~ disease (CVD) is the main source of death around the world, which has turned into an overall general medical condition. Intense myocardial dead tissue (AMI) is a typical clinical basic disease. **Objectives:** The basic aim of the study is to analyse the low high density lipoprotein (HDL) levels as a major risk factor of acute myocardial infarction in Pakistan. **Material and methods:** This cross sectional study was conducted in Ghazi Khan Medical College and DHQ Hafizabad between during June 2021 and to November 2021. ~~The study was conducted~~ according to the ethical committee of the hospital. The data was collected from 100 patients of both genders. **Results:** ~~The information was gathered from 100 patients of the two sexes.~~ The mean age was 45 ± 5.46 years. The extent of male subjects was higher in bunches with high TG levels, while the distinction in age was not measurably critical. **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 to.....

Comment [NT1]: You did not write the full meaning of AMI before you put in the bracket.

Comment [NT2]: Write in words before you abbreviate since this is the first time you are mentioning this term

Comment [NT3]: It will be good if the author can mention the age of respondents recruited for this study in the abstract. Since the main objective is to analyse the low HDL, it will be good if the author mentioned here what was got for HDL for both male and female and how it has contributed to MI. The author should please stick to either sex or gender, rather than using it interchangeably

Introduction

Myocardial infarction (MI) remains a leading cause of death worldwide. An acute MI happens when myocardial ischemia surpasses a basic edge, normally because of an intense plaque burst in the coronary courses, and the cell course of occasions overpowers myocardial cell fix systems prompting myocardial cell harm. Myocardial ischemia happens because of

Comment [NT4]: It will be better to reference this

Comment [NT5]: This sentence is not very clear

plaque develop in the coronary veins, an infection officially known as atherosclerosis or coronary supply route illness (CAD) [1]. Breaking of weak atherosclerotic plaque follows a time of ceaseless plaque destabilization or potentially plaque development due to different patho-organic cycles. Plaque substances are encased inside a settling sinewy cap that forestalls openness of the thrombogenic center to the circulatory system, and debilitating of this cap can hence prompt plaque crack and MI [2].

Comment [NT6]: Atherosclerosis is not an infection, it is the build-up of fats, cholesterol and other substances in the arterial walls although infection has been reported to predispose to atherosclerosis

Comment [NT7]: Please is coronary supply route illness synonymous to CAD?

Cardiovascular illness (CVD) is the main source of death around the world, which has turned into an overall general medical condition. Intense myocardial dead tissue (AMI) is a typical clinical basic disease. In the beyond couple of many years, huge headway has been made in understanding, forestalling and controlling this sickness [3]. Specifically, the ascent of reperfusion treatment essentially decreased mortality and worked on the anticipation of AMI. As of late, the job of low thickness lipoprotein thickness (LDL-C) in the pathogenesis of atherosclerosis (AS) has drawn in much consideration [4]. Nonetheless, an ever increasing number of clinical preliminaries have uncovered that in the wake of controlling for deterministic gamble factors like LDL-C, the gamble for coronary illness (CHD) stayed, while the expansion in fatty oils (TG) was essentially related with the expansion in mortality, the rate of myocardial localized necrosis (MI) and the repeat pace of coronary course illness [5].

Comment [NT8]: Reference please

Comment [NT9]: The statement is not clear

Comment [NT10]: Low density lipoprotein cholesterol

Comment [NT11]: There is no linkage of how low HDL causes AMI in this write-up

Aims and objectives

Comment [NT12]: There is only one aim, no 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 Ghazi Khan Medical College and DHQ Hafizabad ~~between during~~ June 2021 ~~and to~~ November 2021. The review was led by the moral

advisory group of the medical clinic. The information was gathered from 100 patients of the two sexes. ~~The information was gathered from those patients~~ who visited the OPD of the medical clinic routinely. TC level in serum was estimated ~~using~~utilizing the endpoint test technique. HDL-C and LDL-C were estimated utilizing the immediate test technique. TG was estimated utilizing the GPO strategy. Non-HDL-C not set in stone by deducting serum HDL-C from serum TC.

Comment [NT13]: Write in words before the abbreviation

Comment [NT14]: What is GPO Strategy?

~~Data~~Information were entered into ~~were gathered utilizing the product~~ Epidata 3.0 and analysed using Statistical Package for Social Science ~~—examined utilizing measurable programming~~ (SPSS) version 20.0. Estimation information were communicated as mean \pm standard deviation (SD). Intergroup examination was led utilizing investigation of fluctuation.

Results

The information was gathered from 100 patients of the two sexes. The mean age was 45 ± 5.46 years. The extent of male subjects was higher in bunches with high TG levels, while the distinction in age was not measurably critical. With the expansion in TG level, the extent of individuals with a background marked by smoking expanded, weight file (BMI), SBP, DBP, FBG, UA and the pace of MI expanded, while HDL-C level step by step diminished, and the distinctions were all measurably huge. Contrasts in TC and LDL-C levels were not genuinely huge.

Comment [NT15]: All these should have been mentioned in the methods as part of the information the author sought

Table 1: Logistic regression analysis to identify predictors of periprocedural myocardial infarction

Comment [NT16]: It will be better for Table 1 to show the socio-demographic characteristics of the patients, then the no of them that smoke, has Hypertension or Diabetes and also the BMI
Another table to show the predictors of Periprocedural MI before the logistic regression of the significant predictors

Variables	Univariate		Multivariate	
	OR (95% CI)	<i>p</i> value	OR (95% CI)	<i>p</i> 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

In a broadly referred to meta-examination of four enormous investigations (absolute number of people contemplated: 15,252), a 1 mg/dL increment of HDL-C levels was accounted for to be related with a 2%-3% diminished CVD risk [6]. Niacin, by and by endorsed with a statin, is one of the most regularly involved pharmacological treatment pointed toward bringing HDL-C focuses up in patients with such dangers. At a pharmacological portion of ~1.5-2 g each day, Niacin is perhaps the most intense specialist accessible for this reason. Niacin likewise decreases all proatherogenic lipids and lipoproteins, including absolute cholesterol, TGs, exceptionally low-thickness lipoprotein, LDL, and lipoprotein [7]. Notwithstanding its prevalence, the viability of niacin has come into question in ongoing examinations. Two particular examinations, 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-thickness Lipoprotein to Reduce the Incidence of Vascular Events (HPS2-THRIVE) were pointed toward assessing whether adding the cutting edge, stretched out discharge niacin details to statin treatment gives steady advantage over

Comment [NT17]: The pharmacological treatment should have been mentioned earlier in the introduction

statin treatment alone as far as cardiovascular essential occasions in patients with laid out CAD [8]. These clinical preliminaries concentrated on explicit populaces of stable ischemic coronary illness patients, barring patients with MI or those with critical remaining blended dyslipidemia not treated with ideal portions of serious statin treatment [9]. Both the AIM-HIGH and HPS2-THRIVE clinical preliminaries were halted rashly because of an absence of gainful impacts and a failure to meet essential endpoints of decreased cardiovascular illness 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

Comment [NT18]: How this conclusion was derived from this study was not clear

References

1. Schwartz GG, Olsson AG, Abt M, Ballantyne CM, Barter PJ, Brumm J, et al. Effects of dalcetrapib in patients with a recent acute coronary syndrome. *N Engl J Med*. 2012;367:2089–2099.
2. Lincoff AM, Nicholls SJ, Riesmeyer JS, Barter PJ, Brewer HB, Fox KAA, et al. Evacetrapib and cardiovascular outcomes in high-risk vascular disease. *N Engl J Med*. 2017;376:1933–1942.
3. Ginsberg HN, Elam MB, Lovato LC, Crouse JR, Leiter LA, Linz P, et al. Effects of combination lipid therapy in type 2 diabetes mellitus. *N Engl J Med*. 2010;362:1563–1574.
4. Kontush A, Chapman MJ. Functionally defective high-density lipoprotein: a new therapeutic target at the crossroads of dyslipidemia, inflammation, and atherosclerosis. *Pharmacol Rev*. 2006;58:342–374.

5. Kimura S, Sugiyama T, Hishikari K, Yamakami Y, Sagawa Y, Kojima K, et al. Association of Intravascular Ultrasound- and Optical Coherence Tomography-Assessed Coronary Plaque Morphology with Periprocedural Myocardial Injury in patients with stable angina pectoris. *Circ J*. 2015;79:1944–1953.
6. Boden WE, Probstfield JL, Anderson T, Chaitman BR, Desvignes-Nickens P, Koprowicz K, et al. Niacin in patients with low HDL cholesterol levels receiving intensive statin therapy. *N Engl J Med*. 2011;365:2255–2267.
7. Landray MJ, Haynes R, Hopewell JC, Parish S, Aung T, Tomson J, et al. Effects of extended-release niacin with laropirant in high-risk patients. *N Engl J Med*. 2014;371:203–212.
8. Barter PJ, Caulfield M, Eriksson M, Grundy SM, Kastelein JJ, Komajda M, et al. Effects of torcetrapib in patients at high risk for coronary events. *N Engl J Med*. 2007;357:2109–2122.
9. Rosenson RS, Brewer HB, Jr, Chapman MJ, Fazio S, Hussain MM, Kontush A, et al. HDL measures, particle heterogeneity, proposed nomenclature, and relation to atherosclerotic cardiovascular events. *Clin Chem*. 2011;57:392–410.
10. Sharrett AR, Ballantyne CM, Coady SA, Heiss G, Sorlie PD, Catellier D, et al. Coronary heart disease prediction from lipoprotein cholesterol levels, triglycerides, lipoprotein(a), apolipoproteins A-I and B, and HDL density subfractions: the atherosclerosis risk in communities (ARIC) study. *Circulation*. 2001;104:1108–1113.