Journal of Cardiovascular Disease Research
ISSN:0975-3583,0976-2833 VOL14, ISSUE 05, 2023

Original research article

The effect of acute myocardial infarction on absolute cholesterol and triglyceride levels

[1]Dr. Vishwanath Nowbade, [2]Dr. Karan Nowbade

[1] MD (Internal Medicine) FICP, Professor and Head of ICCU and CCU MIMS, Vikarabad (retired) TS Director VN Hospital, Kalaburgi, Karnataka, India
[2]Assistant Professor, Department of Medicine, ESIC Medical College, Kalaburgi, Karnataka, India

Corresponding Author:
Dr. Karan Nowbade

Abstract

More than half of AMI related deaths occur before the stricken individual reaches the hospital. The in-hospital mortality rate after admission for AMI has declined from 10% to about 6% over the past decade. The 1-year mortality rate after AMI is about 15%. Mortality is approximately fourfold higher in elderly patients (over age 75) as compared with younger patients. Prior to conducting the study, approval was obtained from the Institutional Ethical Committee. Patients or their close relatives were approached during the hospitalization and informed consent was sought for enrolment into the study. Besides clinical examination and routine investigation, the serum lipid profile was measured within the first 24 hours of the onset of symptoms of myocardial infarction and again at day 4 post myocardial infarction. The serum total cholesterol, triglyceride levels were measured by colorimetric test and HDL cholesterol is measured by precipitation assay. On day 1 post myocardial infarction the mean total cholesterol value is 190.10. On day 4 post myocardial infarction, the mean total cholesterol value is 172.50. On day 1of post-myocardial infarction, the mean triglyceride value was 124.98. On day 4 of post-myocardial infarction, the mean triglyceride value was 143.22.
Keywords: Myocardial infarction, absolute cholesterol, triglyceride

Introduction

lschemic heart disease (IHD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium. It typically occurs when there is an imbalance between myocardial oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of anepicardial coronary artery (or arteries) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery [1].

Myocardial infarction is the death of cardiac myocytes that generally occurs with the abrupt decrease in coronary blood flow that follows a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis. Acute myocardial infarction (AMI) is one of the most common diagnoses in hospitalized patients in industrialized countries [2].

Patients with ischemic heart disease fall into two large groups: patients with chronic coronary artery disease (CAD) who most commonly present with stable angina and patients with acute coronary syndromes (ACSs). These include patients with acute myocardial infarction with ST-segment elevation (STEMI) on their presenting electrocardiogram and those with non-ST-segment elevation acute coronary syndrome (NSTE-ACS). The latter include patients with non-ST-segment elevation myocardial infarction (NSTEMI), who, by definition, have evidence of myocyte necrosis, and those with unstable angina (UA), who do not. The relative incidence of NSTEMI compared to STEMI appears to be increasing.

More than half of AMI related deaths occur before the stricken individual reaches the hospital. The in-hospital mortality rate after admission for AMI has declined from 10% to about 6% over the past decade. The 1-year mortality rate after AMI is about 15%. Mortality is approximately fourfold higher in elderly patients (over age 75) as compared with younger patients [3].

When patients with prolonged ischemic discomfort at rest are first seen, the working clinical diagnosis is that they are suffering from an acute coronary syndrome. The 12-lead electrocardiogram (ECG) is a pivotal diagnostic and triage tool because it is at the center of the decision pathway for management; it permits istinction of those patients presenting with ST-segment elevation from those presenting without ST-segment elevation. Serum cardiac biomarkers are obtained to distinguish unstable angina (UA) from non-ST-segment elevation myocardial infarction (NSTEMI) and to assess the magnitude of an ST-segment elevation myocardial infarction (STEMI) [4].

ISSN:0975-3583,0976-2833 VOL14, ISSUE 05, 2023

Methodology

Study design: A prospective observational study of patients satisfying the inclusion criteria was conducted at the intensive coronary care unit and medical wards.

Sample size

- Hundred patients who were admitted with a confirmed diagnosis of acute myocardial infarction were enrolled in the study.
- The diagnosis of acute myocardial infarction was made if patients had ischemic type chest pain for 2:30 minutes with evidence of ST-segment elevation of 2:1 mm in two anatomically contiguous leads on the ECG or the appearance of a new left bundle branch block.

Inclusion criteria

- Patients with symptoms suggestive of ACS presenting within 12 hours.
- ECG evidence of Ml.
- Increased biomarkers of myocardial infarction.

Exclusion criteria

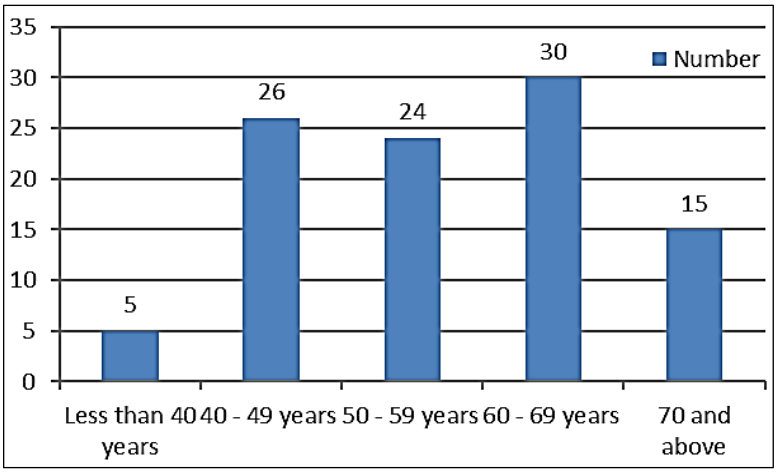
- Symptoms suggestive of acute myocardial infarction 2:12 hours.
- Hospital stays of <4 days.
- Already receiving lipid-lowering medications.
- All the patients were followed from the day of admission to the day of discharge.

Prior to conducting the study, approval was obtained from the Institutional Ethical Committee. Patients or their close relatives were approached during the hospitalization and informed consent was sought for enrolment into the study.

Besides clinical examination and routine investigation, the serum lipid profile was measured within the first 24 hours of the onset of symptoms of myocardial infarction and again at day 4 post myocardial infarction. The serum total cholesterol, triglyceride levels were measured by colorimetric test and HDL cholesterol is measured by precipitation assay.

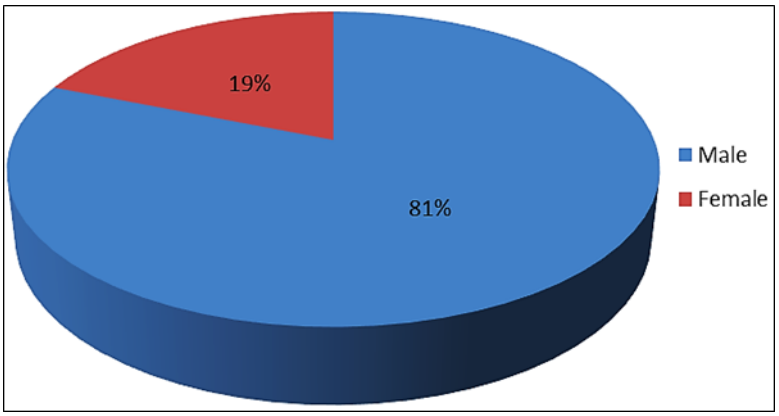
The LDL cholesterol value was calculated by using the Friedewald formula. LDL cholesterol = total cholesterol-HDL cholesterol-(triglyceride/5).

Results



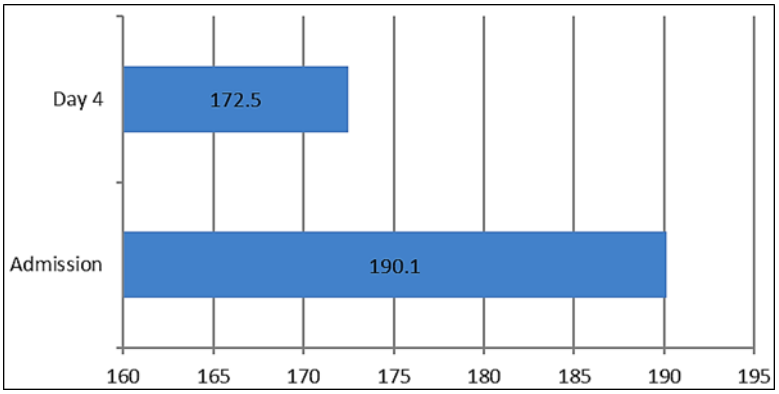
Graph 1: Age distribution

Maximum Number of Patients of study population are in the age distribution of 60-69.



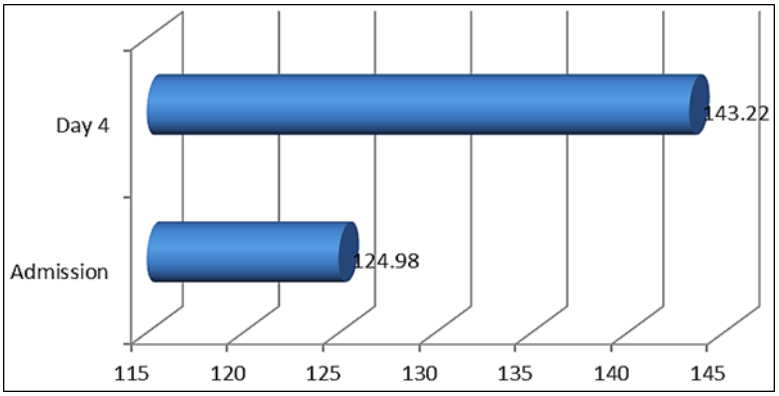
Graph 2: Gender

Eighty one percent (81%) of the patients studied were men.
On day 1 post myocardial infarction the mean total cholesterol value is 190.10. On day 4 post myocardial infarction, the mean total cholesterol value is 172.50.



Graph 3: Mean total Cholesterol

On day 1of post-myocardial infarction, the mean triglyceride value was 124.98. On day 4 of post-myocardial infarction, the mean triglyceride value was 143.22.



Graph 4: Mean triglyceride

Table 1: Comparison of the Serum Lipid Values and Ratios between Within 24 Hours of Myocardial Infarction and Day-4 Post Myocardial Infarction

Serum lipids	At Admission	Day-4	P Value
Total cholesterol (mg/di)	190.10 ± 25.75	172.50 ± 27.05	(0.00001)
Triglycerides (mg/di)	124.98 ± 38.35	153.22 ± 35.64	{<0.0001)

Discussion

Many studies in the past few decades have shown that acute myocardial infarction results in a significant decrease in the serum levels of total cholesterol, LDL cholesterol and HDL cholesterol 12.
The acceptable time for the measurement of plasma lipids after an acute myocardial infarction is within 24 hours after the onset of symptoms and the plasma lipid levels measured beyond 24 hours are mostly considered to be invalid [5].

ISSN:0975-3583,0976-2833 VOL14, ISSUE 05, 2023

The post myocardial infarction decline in serum cholesterol occurs because of the acute-phase response, and is of greatest extent by days 4 to day 5 post-myocardial infarction.

In present study, absolute values of cholesterol and cholesterol ratios are obtained on the day of admission and again on day 4 of hospital stay and comparison of values of 2 samples is done to determine the trends of lipid profile post myocardial infarction. The mean total cholesterol value decreased from 190.1 to 172.05 with p value being <0.05 meaning statistically significant drop has occurred from admission to day 4 of hospital stay. The mean triglyceride value has increased from 124.98 to 153.22 with p value being <0.05 and the variation is statistically significant [6].

While the serum cholesterol level decreases after an acute myocardial infarction, the serum triglyceride level increases [7]. This paradoxical rise in serum triglycerides is due to increase in serum-C reactive protein level which may increase to levels that are several hundred-fold higher than baseline 4 days after and myocardial infarction. The C-reactive protein binds selectively with very LDL and interferes with its catabolism thereby increasing the serum triglyceride concentration [8].

Conclusion

- Following acute myocardial infarction the total serum cholesterol level falls significantly and the triglyceride level rises significantly.
- Therefore, measurement of absolute levels of serum cholesterol and triglycerides following acute myocardial infarction are not valid in risk assessment 24 hours after infarction.

References

1. Castelli WP. Epidemiology of coronary heart disease; the Framingham study. Am J Med. 1984;27:4-12.
2. Smith SC Jr., Greenland P, Grundy SM. Identifying the high-risk patient for primary prevention; executive summary. Circulation. 2000;101:111-116.
3. Brugada R, Wenger NK, Jacobson TA, *et al*., Changes in Plasma cholesterol levels after hospitalization for acute coronary events. Cardiology. 1996;87:194-199.
4. Ryder RE, Hayes TM, Mulligan IP, *et al*., How soon after myocardial infarction should plasma lipid values be assessed? BMJ. 1984;289:1651-1653.
5. Chamsi-Pasha H, Taylor RJ, McDowell D, *et al*., Plasma lipids, when to measure after myocardial infarction? Br J Clin. Pract. 1989;43:447-450.
6. Jameson J, Fauci A, Kasper D, Hauser S, Longo D, Jameson J, *et al*., Harrison's Principles of Internal Medicine.
7. Farmer JA, Gotto AM Jr. Dyslipidemia and the vulnerable plaque. Prognosis of pardiovascular disease. 2002;44:415-528.
8. Bonow R, Braunwald E. Heart disease. Philadelphia: Elsevier I Saunders; c2012.