Differences in Physician Compliance with Guideline on Lipid Profile Determination within 24 h after Acute Myocardial Infarction

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Key Words

Acute myocardial infarction · Total cholesterol ·
Coronary heart disease · Coronary risk factor ·
Lipoprotein cholesterol · Triglycerides

Abstract

Objective: To evaluate the American College of Cardiology/American Heart Association guidelines on blood lipid testing within 24 h of the onset of chest pain in patients with myocardial infarction. **Subjects and Methods:** This is a cross-sectional observational study on 83 patients (77 male, 6 female) admitted into the Coronary Care Units of the Al-Amiri and Mubarak Al-Kabeer Hospitals, Kuwait with myocardial infarction. The lipid profiles were obtained within 24 h of the onset of chest pain. Twenty patients were on treatment with statins prior to admission. Diagnosis of myocardial infarction in all patients was based on standard criteria. Total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, and triglycerides (Tg) were measured and low-density lipoprotein (LDL) cholesterol was calculated. **Results:** Twenty-three patients had normal cardiac markers on admission but later developed increased serum markers and ECG changes of acute myocardial infarction. Mean (95% con-

fidence interval) TC, HDL, Tg and LDL were 5.1 (4.8–5.4); 0.93 (0.88–0.98); 1.85 (1.56–2.14), and 3.39 (3.13–3.65) mmol/l, respectively. 70% of the patients had normal or only mild elevations of LDL with low HDL and poor HDL:TC ratio (<20%). Thirty-eight patients had low HDL (<0.9 mmol/l) and only 22 (27%) patients met the National Cholesterol Education Program guideline of target LDL <2.6 mmol/l. Fifty-six patients were classified as having the metabolic syndrome according to the criteria of the WHO. **Conclusion:** The findings indicate that HDL appears to be the main lipid risk factor in patients presenting with AMI in Kuwait, therefore primary prevention strategies should focus on treatment modalities that increase HDL. We recommend that the lipid profile should be done within 24 h of admission and lipid-lowering therapy initiated as part of secondary prevention strategy.

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Introduction

Elevated serum cholesterol concentration is a significant risk factor for development, progression and recurrence of coronary heart disease (CHD) [1–3]. Epidemio-

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logic studies and several clinical trials have provided convincing evidence that treatment with cholesterol-lowering drugs reduces the risk for CHD-related morbidity and mortality [4, 5]. Despite the convincing evidence and guidelines, treatment of hyperlipidaemia has been less than optimal, especially in patients who have had myocardial infarction. Several studies have shown that a variable proportion of patients who have had acute myocardial infarction (AMI) had cholesterol-lowering interventions [6–8]. The problem is compounded by the controversy on the appropriate timing of lipid measurement after AMI [9–12]. Some studies have shown that AMI causes an acute-phase response, which includes increased cholesterol synthesis as well as concomitant increase in low-density lipoprotein cholesterol (LDL-C) receptor activity which results in reduction of LDL-C [13–16]. Therefore it is often assumed that lipid testing is not useful in a patient presenting with AMI.

Initial recommendations that suggested delaying assessment and treatment of lipid levels until at least 6 weeks after AMI [11, 12] have been revised [13]. Postponing the measurement of serum lipid levels in patients with acute coronary events usually leads to delay in the treatment of this important CHD risk factor. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines for lipid analysis after myocardial infarction now specify that lipid analysis be performed within 24 h of onset of chest pain [13], but physician compliance with this guideline is variable. The aims of this cohort study were to evaluate the ACC/AHA guidelines and determine the proportion of patients in whom lipid profiles were requested during hospitalisation after AMI and the lipid profile and other traditional risk factors in patients presenting with AMI.

Materials and Methods

The study included 83 patients (77 male, 6 female) admitted to the Coronary Care Units of the Al-Amiri (n = 35) and Mubarak Al-Kabeer Hospitals (n = 48) with myocardial infarction. Myocardial infarction was confirmed in all patients by positive serum cardiac markers (creatinine kinase-MB and/or cardiac-specific troponin I) with ECG changes diagnostic of myocardial infarction. Information about demographic data, past medical history, family history, smoking status, clinical characteristics and the use of various hypolipidaemic drugs before hospitalisation were obtained from the patients or from their medical records. Review of past medical history showed that all the patients included in this study were having myocardial infarction for the first time. Severely ill patients were not recruited to the study. Weight and height were recorded for all patients, but only 20 patients (all male) gave informed consent to have their waist circumferences

measured. Body mass index (BMI) was calculated according to the formula: weight in kilograms divided by the square of the height in metres.

Blood samples were collected within 24 h of the onset of chest pain. For the samples collected at Al-Amiri Hospital, lipid profile was routinely requested and determined there, but for samples collected at Mubarak Al-Kabeer, lipid profile was determined at the Department of Pathology, Faculty of Medicine. Total cholesterol, high-density lipoprotein cholesterol (HDL-C), and triglycerides were measured with the Beckman LX20 automated analyser (Beckman, Brea, Calif., USA). LDL-C was calculated with the formula of Friedewald et al. [17]. Fasting serum glucose was determined in all the patients. Features of the metabolic syndrome were assessed in patients according to the guidelines of the World Health Organization (WHO) [18]. Evidence of insulin resistance was defined as the presence of type 2 diabetes or impaired fasting glucose. Further special testing of glucose status could not be performed in 10 patients with fasting glucose <6.1 mmol/l.

Statistical Analyses

All the statistical analyses were performed using computer software (SPSS, version 11.5; SPSS, Chicago, Ill., USA). Continuous variables were expressed as the mean $\pm$ SD, and the categorical variables were expressed as a percentage. Comparison of categorical variables was done with the Pearson χ^2 test. A two-tailed p value of <0.05 was considered to be significant.

Results

Requests for lipid profile determination after AMI varied markedly between the Mubarak Al-Kabeer and Al-Amiri Hospitals – all the patients at Al-Amiri Hospitals had their lipid profile assessed routinely during the first 24 h of hospitalisation for AMI whereas no patient at Mubarak Al-Kabeer Hospital had lipid estimation. All the attending physicians questioned on prescription of hypolipidaemic drugs agreed with the principle of lipid lowering as a secondary preventive measure in patients with AMI.

The mean age of all the patients was 54 ± 13 years. However, 61 male patients (79%) were older than 45 years while 3 female (50%) were older than 55 years. Thirty-two patients (39%) were smokers, and 39 (47%) had a family history of CHD. Mean BMI was 32.69 ± 5.86 . Forty-two patients (51%) had evidence of insulin resistance and 31 (32%) had impaired fasting glucose. BMI was $>30 \text{ kg/m}^2$ in 67 (80.72%) patients (fig 1); hypertension was present in 39 patients (47%), fasting triglycerides were $>1.7 \text{ mmol/l}$ in 35 (42%) and 50 patients (65%) had $\text{HDL-C} <0.9 \text{ mmol/l}$. Overall, 56 patients (68%) could be classified as having the metabolic syndrome according to the WHO criteria, although the syndrome could not be fully excluded in the 10 patients with fasting glucose

<6.1 mmol/l. HDL-C was significantly lower in patients with BMI >25 kg/m² compared to patients with BMI <25 kg/m² (p = 0.02) (fig. 1). Although 20 patients (24%) were on treatment with 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins), 13 (65%) had not reached the target LDL-C of <2.5 mmol/l as set by the National Cholesterol Education Program [3] at the time of hospitalisation for AMI. Table 1 summarises the lipid profile in the patients.

Discussion

This study reports a variable use of lipid profile testing in patients hospitalised for AMI at the Al-Amiri and Mubarak Al-Kabeer Hospitals, Kuwait. The results show wide differences in practice of requesting for lipid profile during hospitalisation for AMI between the two hospitals. The confusion about the validity of the determination of the lipid profile during the first few hours after AMI may play a role in the observed differences in practice. Several studies have shown significant decrease in the serum concentrations of total cholesterol, LDL-C, and HDL-C after AMI [9–16]. The decrease occurs as a result of the infarction and accompanying tissue injury which initiate various local and systemic reactions that include release of various cytokines and increase in various acute-phase reactants [14–16]. The accompanying acute-phase response results in lower LDL-C due to increased catabolism. However, several studies have shown that the decrease in lipid concentrations occurs during the later stages and lipid profiles obtained within 24 h after the onset of symptoms are valid estimates that could be used for the institution of dietary or therapeutic intervention [3, 13]. The lipid profile obtained in the patients in this study (table 1) confirmed the need for secondary prevention efforts to reduce the risk of subsequent coronary events.

The recently released third report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III) emphasised the need for intensive lipid-lowering treatment in patients with CHD. In these patients, if LDL-C level is >2.59 mmol/l, then initiation of therapy is recommended. Therefore, 63 of the patients in this study required immediate therapeutic intervention. Evidence suggests that the in-patient initiation of lifestyle interventions and lipid-lowering medication result in better long-term compliance and more lasting rates of success [19, 20]. Therefore, the immediate assessment of the lipid profile in patients with AMI on

Fig. 1. Box and whisker plots of the relationship between BMI and HDL-C. The box represents the interquartile range (25th–75th), the line across the box represents the median value, the range lines extend from the highest to lowest values excluding an outlier, which is shown as an open circle.

Table 1. Lipid profile in patients presenting with AMI

Variable	Mean $\pm$ SD	NCEP recommended target, mmol/l
Total cholesterol	5.12 $\pm$ 1.38	<5.2
Triglyceride	1.85 $\pm$ 1.35	<1.7
HDL-C	0.93 $\pm$ 0.24	>1.1
LDL-C	3.39 $\pm$ 1.14	<2.6

NCEP = National Cholesterol Education Program.

admission and early initiation of therapeutic options and goals of therapy should be carried out [20]. As recommended by the ACC/AHA, failure to perform lipid analysis within the first 24 h after admission could result in delays in diagnosis, treatment and patients being lost to follow-up. Barriers to the acceptance of the ACC/AHA guidelines on lipid testing after AMI need to be identified and overcome so that secondary preventive measures could be introduced to reduce the associated morbidity and mortality.

Our study has some limitations. The first relates to the lack of data on the lipid levels of patients who were not on lipid-lowering medication prior to admission. Similarly the lack of data on the lipid profile of the 20 patients who were receiving statins prior to admission for myocardial infarction is also a limitation. However, these limitations should be interpreted in the context of the fact that only 7 patients (7 of 20 of the treated group) achieved the target LDL-C of <2.59 mmol/l. Therefore, one could argue that all the patients studied should have had retesting of their lipid profile within 24 h after onset of symptoms.

Although the initiation of lipid-lowering therapy is occurring in increasing proportions of patients with CHD who have hyperlipidaemia, it still occurs in a minority of eligible subjects because of delays in evaluation after AMI [3, 5]. Furthermore, as target cholesterol levels differ in patients with and without CHD, the modest increase in LDL-C found in the patients who were not on lipid-lowering medication suggests that there is a need to refine primary prevention guidelines to improve identification of at-risk individuals before they develop AMI. Our data have broad implications for the selection of patients for more aggressive therapy. The finding that a majority of the patients have three or more features of the criteria for the metabolic syndrome suggests the need for adherence to the ATP III recommendation that stresses the significance of weight reduction and exercise [3]. This is particu-

larly important in Kuwait where economic wealth has resulted in a sharp increase in physical inactivity, obesity and incidence of diabetes of epidemic proportions [21]. Specifically, our results show that more consideration should be given to low HDL-C, which was found in association with higher BMI (fig. 1) in 80.72% of the patients, and achievement of the target LDL-C values in those who are receiving treatment including patients with conditions that are CHD-equivalent such as diabetes mellitus.

Conclusion

The findings of this study indicate that HDL appears to be the main lipid risk factor in patients presenting with AMI in Kuwait, therefore primary prevention strategies should focus on treatment modalities that increase HDL. We recommend that the lipid profile should be done within 24 h of admission and lipid-lowering therapy initiated as part of a secondary prevention strategy.

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