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Presence of metabolic abnormalities in patients with acute myocardial infarction: at the onset and after recovery. A pilot study.

Abstract

Objective: To explore the interrelationship, if any, between acute myocardial infarction and metabolic abnormalities.

Methodology: Forty six patients with acute myocardial infarction were selected and their blood was analysed for some key biochemical parameters. These parameters were analysed again after three weeks in twenty-two patients who recovered and were discharged from the hospital. For comparison, twenty adult individuals were selected as normal controls.

Results: Altered glucose and lipid parameters, along with raised serum uric acid & calcium and reduced levels of serum magnesium & potassium, were found during acute myocardial infarction. In the recovery phase, the metabolic changes did not improve in spite of significant clinical improvements. Out of all the parameters, triglyceride/high density lipoprotein cholesterol ratio was found to be a stable biomarker for acute myocardial infarction. Patients with acute myocardial infarction with elevated TG/HDL-C ratio could represent a high-risk group requiring aggressive intervention.

Conclusion: Our pilot study indicates that a larger epidemiological study should be conducted in patients with acute myocardial infarction using TG/HDL-C ratio as a biomarker of interest, and finding an appropriate cut-off value for the same.

Keywords: Acute myocardial infarction; lipoproteins; hyperglycaemia; triglyceride; magnesium; uric acid.

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Abbreviations: TG, Triglyceride; CH, Cholesterol; HDL-C, High density lipoprotein cholesterol; LDL-C, Low density lipoprotein cholesterol; AST, Aspartate transaminase; CK, Creatinine kinase; LDH, Lactic dehydrogenase; AMI, Acute Myocardial Infarction

Introduction

The diagnosis of acute myocardial infarction is traditionally made based on characteristic clinical picture with typical electrocardiographic and serum enzymes findings. However, these findings could not give us any insight into the disease processes and the metabolic abnormalities associated with AMI. A tendency to hyperglycaemia, for which the lack of insulin was blamed,[1] and the possibility of a link with hyperuricemia have been reported.[2,3] Several studies reported a close association between high serum lipids and AMI, although a cause and effect relationship remain controversial.[4,5] Much emphasis had been given to the role of mineral metabolism when it was reported that cities with the hardest water had a lower mortality rate from coronary heart disease compared to places with soft water.[6,7] However, the literature on follow-up studies in acute myocardial infarction is meagre. It was, therefore, our interest to study, some relevant biochemical parameters in this disorder at the onset and three weeks after the event to find out the presence of any association. Search for a stable and dependable parameter which is most positively correlated with AMI was also sought.

Materials and Methods

Forty-six male patients aged between 45 to 57 years, weighing between 60 to 75kg, admitted to the hospital with acute myocardial infarction, were selected for the study. The diagnosis of AMI in these cases was based on standardized international guideline. Utmost care was taken in the selection of the patients so that they did not have any previous history of diabetic mellitus, hepatic & renal disorders, gout, and enlargement of prostrate. Twenty healthy male subjects with the same baseline parameters, without any past or present illness, who attended the hospital out patient's department for routine check-up, were selected as normal controls. Venous blood samples were collected from the patients after an overnight fast of 12 hours but within 24 hours of onset of symptoms and immediately analysed for glucose, urea, uric acid, potassium, calcium, magnesium, total protein and albumin, CK, CK-MB, LDH, AST, CH, LDL-C, HDL-C and TG. The same protocol was repeated at 3-weeks follow up in the 22 patients who recovered from MI and were discharged from the hospital. The biochemical parameters were estimated by standard enzymatic methods with the help of an auto-analyser, Cobas Integra 400 plus, as per method manual supplied by Roche Diagnostic.[8] Written consents from all the subjects along with ethical committee approval were obtained.

Results

In acute myocardial infarction cohort, fasting blood glucose was found to be increased ($p < 0.001$) and remained elevated in recovery phase (3-weeks post AMI). ($p<0.001$) Blood urea and uric acid were elevated during AMI ($p <.001$); and decreased in recovery phase although they did not come down to baseline levels ($p<.001$) (Table 1).

Table 1 Biochemical Parameters of Normal Controls, Patients Suffering from Acute Myocardial Infarction and Patients Recovered after the Attack of Acute Myocardial Infarction

Parameters with Units	Normal Controls(20)	Patients Suffering from acute Myocardial Infarction (46)	Patients Recovered from Acute Myocardial Infarction (22)
Plasma Glucose, mg/dl	97±9.1	152±31.4	119±12
Serum Urea, mg/dl	20±4.7	38±14.3	24±7.1
Serum Uric Acid, mg/dl	3.8±0.6	5.8±1.4	4.3±1.1
Serum Potassium, mEq /dl	4.4±0.21	3.9±0.4	4.6±0.32
Serum Magnesium, mg/dl	2.0±0.32	1.58±0.27	1.82±0.3
Serum Calcium, mg/dl	9.2±0.43	10.8±0.5	9.7±0.45
Serum Total Protein, mg/dl	7.3±0.81	6.9±0.64	7.0±0.68
Serum Albumin, gm/dl	4.2±0.52	4.0±0.43	3.8±0.6
Serum CK Activity, IU/L	72±12	517±61	89±23
Serum CK MB Activity, IU/L	14±6.2	115±37	19±3.7
Serum LDH Activity, IU/L	158±24	462±219	214±39
Serum AST Activity, IU/L	19±4.3	136±42	24±8.1
Serum CH, mg/dl	208±18	180±25	171±20
Serum HDL – C, mg/dl	48±13	42±17	39±15
Serum LDL –C, mg/dl	115±22	120±26	106±19
Serum TG, mg/dl	104±19	138±47	145±31
Serum TG/HDL–C Ratio (molar ratio)	0.95	1.44	1.62

Calcium was high ($p<0.01$) and remained somewhat elevated in the recovered phase ($p<0.01$). Magnesium level decreased ($p<0.01$) and recovered almost up to the normal level in the recovery phase ($p<0.01$). Potassium level was low ($p<0.05$) and came back to normal level during the recovery phase. No appreciable changes were observed in total protein and albumin.

CK, CK-MB, LDH and AST were all elevated in acute myocardial infarction patients ($p<0.001$); reducing in the recovery phase, with the exception of LDH and CK which remained elevated ($p<.001$)

TG was found to be elevated ($p<0.001$) and remained elevated during the recovery phase ($p<0.001$). Both CH and HDL – C were found to be low ($p<.001$) and remained low in the recovery stage ($p<0.01$). TG/HDL –C ratio was found to be very high in the patients with AMI ($p<.001$) and remained high during the recovery stage ($p<0.001$).

Discussion

None of the patients had any past history of diabetes mellitus. The persistent hyperglycaemia in acute myocardial infarction could be attributed to high counter-regulatory hormone levels in shock, to functional changes in the hypothalamus or to unmasking of pre-existing diabetes. Adrenaline and adrenocorsteroids have been found to be increased in response to stress following acute myocardial infarction.[9,10] The persistence of hyperglycaemia even in recovery stage could be due to persistent insulin resistance leading to defective glucose utilization. Reduction in renal blood flow following shock leading to renal tubular damage might be an important factor in causing high serum calcium, urea and uric acid levels, extending into the recovery phase. Uric acid level increased along with the higher Killip classification in acute myocardial infarction.[11,12] A striking relationship between hypomagnesaemia and incidence of serious dysrhythmia e.g. multifocal atrial tachycardia, ventricular fibrillation has been reported, and magnesium therapy has been suggested to improve the condition.[13,14] Further hypomagnesaemia has been found to be strongly associated with hypokalaemia leading to reduction in membrane ATPase activity with a loss of intracellular potassium and subsequent urinary loss.[15] Several studies have reported that hypokalaemia is associated with increased risk of ventricular arrhythmia leading to poor prognosis in acute myocardial infarction, although the exact mechanism is still unknown. From the present study it seems that altered electrolyte balance was associated with the clinical condition of the patients which improved sufficiently in the recovery stage after suitable treatment.[16]

The persistence of raised cardiac enzymes even in the recovery stage was probably due to their enhanced release from sustained cellular destruction, and sluggish rate of degradation or through altered membrane permeability. They indicate extensive myocardial tissue damage and help in the diagnosis and prognosis of the disease. However, no change in protein metabolism was observed during the study period.

Although several epidemiological studies had demonstrated a positive correlation between serum lipids level and incidence of atherosclerosis,[1] it was suggested that the incidence of coronary heart disease was linked more intimately with the serum TG concentration than serum cholesterol.[17] The abnormal carbohydrate metabolism associated with acute myocardial infarction might stimulate the

mobilization of free fatty acids from adipose tissues, which in turn might influence the endogenous synthesis of TG in the liver of these patients. The abnormal lipoprotein pattern could be related to this overproduction of TG. The increase in TG level could also be attributed to an increased level of catecholamine as encountered in stress-related conditions thereby producing an increase in circulating free fatty acids.[18,19] The plasma lipoprotein profile has been an important determinant for cardiovascular disorders with decreased concentration of HDL and increased level of LDL being significant risk factors.[20,21] While VLDL – C is often calculated on the basis of TG values, it is obvious that its rise and fall fully depends on TG. In the present study CH, HDL-C and LDL-C were all found to be lowered during AMI and furthered lowered in the recovery phase in contrast to conventional wisdom and other epidemiological studies reporting otherwise. Recently, oxidized LDL has been implicated for the vascular pathological changes. The importance of TG to HDL-C has also been stressed as it may be related to the processes involved in LDL size which is relevant with regard to the risk of clinical vascular diseases. LDL size correlates negatively with plasma TG (R^2=0.52) and positively with HDL –C (R^2=0.14).[22] However, an inverse correlation between the TG to HDL-C molar ratio and LDL size was even stronger. (R^2=0.59). This ratio was found to be more than 1.33 in 90 % of the patients with small LDL particles. A cut off point of 1.33 for the TG to HDL-C ratio helps identifying small LDL-C.[23] In our study, the TG to HDL-C molar ratio was found to be markedly increased which remained elevated even in the recovery stage. Thus, this ratio is not only suitable as a stable biomarker in patients with vascular complications but also found to be suitable for the selections of the patients needing an earlier aggressive treatment for lipid abnormalities.

A detailed population study in this sub-continent is needed to find a new cut off value for this ratio, if any, so that a rigid treatment schedule could be started well ahead to combat acute myocardial infarction.

Acknowledgment

None

Conflicts of interest

The author declares that there is no conflicts interest.

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