Myocardial infarction: Analysis and diagnosis

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ABSTRACT

Aims: Acute Myocardial infarction (AMI) occurs during the period when circulation to a region of the heart is obstructed and necrosis ensues. The objective of this study was to analyze the various risk factors associated with AMI and estimate the levels of serum myoglobin and enzymes as cardiac marker for diagnosis of early myocardial Infarction.

Methods: This study was conducted at Cardiac Care Center, wherein 183 patients suffering from chest pain were admitted and were taken immediately to Coronary Care Unit (CCU). Twenty five apparently healthy subjects were enrolled as control. The age ranged between 39-60 years. ECG, blood biochemistry and medical history of all the subjects under study was recorded including age, level of education, diabetes, hypertension, mental stress, physical exertion, psychological condition, patient history of MI, family history of MI and sudden death due to MI, time of onset of chest pain, smoking habits and ECG findings. Blood samples were collected for the estimation of myoglobin using fully automatic immunoanalyzer and cardiac marker enzymes were measured using spectrophotometer.

Results: It was found that the major risk factor for AMI was smoking (74% of patients), followed by hypertension (60%) and diabetes mellitus (56%). Medical history of the patient’s family had an effect of upto 54% of the total enrolled patients for AMI. Physical exertion was noticed as one of the causes for AMI (46%). The incidence of myocardial infarction declined with increasing level of education over weight (70-90 kgs) was yet another contributing factor for AMI. Persons in the age group of 51-60 showed an increased incidence of AMI. Lipid profile including cholesterol, TAG, LDL-cholesterol was higher in the patients with myocardial infarction and HDL-cholesterol was much lower in patients as compared to control. Among the biochemical parameters myoglobin showed a marked increase within six hours after onset of MI, followed by CK-MB and LDH, whereas AST and CK were not of diagnostic importance.

Conclusion: This study indicated that smoking is the major contributing factor for AMI. Myoglobin and CK-MB are more sensitive cardiac markers compared to total CK, LDH and AST for the diagnosis of Myocardial Infarction.

Key words: Acute myocardial infarction, myoglobin, Creatine kinase, Lactate dehydrogenase, Aspartate transaminase.
INTRODUCTION

The heart is a muscular organ responsible for moving blood through a series of vessels to all parts of the body. Acute Myocardial Infarction (AMI) occurs during the period when circulation to a region of the heart is obstructed and necrosis ensues. AMI is characterized by severe pain (angina pectoris), frequently associated with pallor, perspiration, nausea, shortness of breath, and dizziness. A precursor state of AMI is myocardial ischemia, in which obstruction of a coronary artery leads to severe oxygen deprivation of the myocardium prior to necrosis. Angina may be pronounced during myocardial ischemia.

Studies suggest that between 40 and 50% of non fatal AMI are unrecognized by the patient and are discovered only on subsequent routine ECG or postmortem examinations [1]. Of this unrecognized infarction, approximately half are truly silent, with the patient unable to recall any symptoms whatsoever. The other half of patients with so-called silent infarction can recall an event characterized by symptoms compatible with acute infarction when leading questions are posed after the abnormal ECG is read, unrecognized or silent infarction occurs more commonly in patients without angina, and is more common in patient with diabetes and hypertension.

The diagnosis of AMI, as formally established by the world health organization, requires at least two of the following criteria: a history of chest pain, evolutionary changes on the ECG, and elevation of serial cardiac enzymes (proteins). Often, the examining physician is fairly certain after obtaining a patients history and completing a physical examination and performing ECG that an MI has occurred. When the ECG fails to demonstrate an AMI, the cardiac markers must be used.

In most patients with AMI, no precipitating factors can be identified. Studies have noted the following patient’s activities at the onset of AMI: heavy physical exertion, 13%; modest or usual exertion, 18%; surgical procedure, 6%; rest, 51%; and sleep, 8%. The sever exertion that preceded an infarction was often performed at times when the patient was fatigued or emotionally stressed. Exertion before infarction is somewhat more common among patients who have had history of angina [2].

There is a pronounced periodicity for the time of onset of AMI. Often, an AMI occurs in the morning hours soon after arising, a period of increasing adrenergic activity, increased plasma fibrinogen levels, and increased platelet adhesiveness. The diagnosis of AMI, as formally established by the world health organization, requires at least two of the following criteria: a history of chest pain, evolutionary changes on the ECG, and elevation of serial cardiac enzymes (proteins). Often, the examining physician is fairly certain after obtaining a patients history and completing a physical examination and performing ECG that an MI has occurred. When the ECG fails to demonstrate an AMI, the cardiac markers must be used.

One of the most valuable contributions of the ECG is in the diagnosis of AMI. It is usually the first test performed, and, when appropriately interpreted, it is the cornerstone of the diagnosis. The initial ECG is diagnostic of AMI in slightly more than 50% of AMI patients. In about 15% of AMIs, there are no changes on the initial ECG tracing. Various cardiac markers have been proposed to date some of them are - creatine kinase (CK); lactate dehydrogenase (LD); cardiac troponin I and T, myoglobin; cholesterol; triglycerides; low density lipoprotein (LDL); high-density lipoprotein (HDL). An initial creatine kinase isoenzyme-2 (CK-2) rise takes 4 to 6 hrs to increase above the upper reference limit. Peak levels occur at approximately 24 hrs. Return to normal takes 48 to 72 hrs. Factors that might affect the classic pattern include size of infarction, CK-2 composition in the myocardium, concomitant skeletal muscle injury,
and reperfusion (spontaneous; following thrombolytics, or following angioplasty). For patients having an Acute Myocardial Infarction (AMI), serum total Lactate Dehydrogenase (LD) values become elevated at 12 to 18 hrs after onset of symptoms, peak at 48 to 72 hrs, and return to below the upper reference limit after 6 to 10 days [3]. LD-1 (the isoenzyme enriched in the heart) rises within 10 to 12 hrs, peaks at 72 to 144 hrs, and returns to normal in approximately 10 days after AMI, paralleling total LD. The elevation patterns of LD-1 and total LD contrast with the elevation patterns of total CK and CK-2, which peak at 24 hrs, and return to below the upper reference limit with 72h after the onset of AMI [4]. Because of its prolonged half-life, LD-1 is a clinically sensitive (90%) marker for infarction when used more than 24 hrs after occurrence. Studies have shown that myoglobin is a very sensitive marker (90-100%) for AMI. Serum concentrations of myoglobin rise above the reference interval as early as 1 hr after MI, with peak activity in the range of 4 to 12 hrs, suggesting that serum myoglobin reflects the early course of myocardial necrosis. 

Myoglobin is rapidly cleared and thus has a substantially reduced clinical sensitivity after 12 hrs. If myoglobin is to have a role in detecting AMI, it must be within the first 0-4 hrs, the time period in which CK-2 (CK-MB) is still within its reference interval. Also, the overall cumulative release of myoglobin has been correlated with infarct size. However, despite these findings, the measurement of serum myoglobin has not been extensively utilized in clinical laboratories for the routine analysis in AMI. The main reason has been the poor clinical specificity (60-95%) of the protein, caused by the large quantities of myoglobin found in skeletal muscle. Other studies that have compared patterns of myoglobin to CK-3 isoforms and total CK activity release have confirmed that myoglobin can be used very early (within 90 min of thrombolytic therapy) for non invasive detection of reperfusion, with high specificity (>80%) [5]. 

Finally, it has been shown that a myoglobin to total CK activity ratio of greater than 5.0 is indicative of reperfusion, with a clinical sensitivity of 75% and a clinical specificity of 96%. Thus, it has been concluded that reperfusion status might be satisfactorily predicted by a single sample obtained either at the time of admission, to assess spontaneous reperfusion, or very early (90 min) after initiation of thrombolytic therapy.

Although every living organism has been found to contain sterols, cholesterol is found almost exclusively in animals, in which it is also the main sterol. Virtually all cells and body fluid contain some cholesterol. Animal products, specially meat, egg yolk, seafood, and whole-fat dairy product, provide the bulk of dietary cholesterol. Increased cholesterol is a causative factor in the etiology of atherosclerotic diseases. As early as 1910, Windaus described cholesterol in the lesion of diseased arteries. Subsequently, many studies have confirmed that free and esterified cholesterol accumulates in the aorta, coronary arteries, and cerebral vessels and that the rate of accumulation varies among individuals. The association between serum cholesterol and atherosclerosis in human was first suggested in 1938, when Thanhauser and Muller each demonstrated familial aggregation of hyper cholesterololemia and Coronary Heart Disease (CHD). Further studies showed that when the total cholesterol concentration is high, the incidence and prevalence of CHD are also high. Cholesterol can be assayed in whole plasma (or serum) and lipoprotein fraction by either chemical or enzymatic methods. In general, enzymatic procedures have replaced chemical methods of analysis, although the later are still regarded as the “gold standard” and are used to assay reference sera. Abell-Kendall method, which involves solvent extraction of lipids, alkaline hydrolysis of esterified cholesterol and quantization of total cholesterol by the Lieberman-Burchard reaction, the colored end product of which is measured spectrophotometrically [6]. Enzymatic methods involve the conversion of cholesterol ester to free cholesterol.
Triglycerides, or triacylglycerols, are fatty acid esters of glycerol, usually containing a mixture of two or three different fatty acids. The role of fasting hypertriglyceridaemia as a risk factor is more controversial than the role of hypercholesterolaemia. The association between raised triglycerides and CHD simply reflects the relationship between low HDL cholesterol and CHD [7]. Low density lipoprotein LDL (α-lipoprotein) is the major cholesterol – carrying particle in plasma and differs from its precursor VLDL in its much lower triglyceride content and in retaining only one at the various apoproteins found in VLDL, namely apoB100. The correlation between total cholesterol and CHD coronary heart diseases is almost entirely due to the correlation between the latter and the concentration of LDL in plasma, whether expressed as the mass of Sf 0-20 particles or the concentration of LDL cholesterol. This correlation persists after middle age and is present in both men and women. The Framingham study showed a strong, inverse correlation between HDL cholesterol and CHD in both sexes, which was present on both univariate and multivariate analysis. The apparent protective effect at high concentrations at HDL cholesterol remained evident until the age of 80 years [8]. The presence of hypertension more than doubles the risk of CHD at any given level of serum cholesterol and a similar but smaller increment is attributable to cigarette smoking. The absolute risk of Cardiac Heart Disease (CHD) rises with age and is much greater at 65 than 35 years. However, the relative risk increases with increasing serum cholesterol more steeply in 35-years-old than in 65-years-old. At any given level of serum cholesterol risk of CHD in men in roughly three times of women of comparable age [9]. This study was undertaken to re-evaluate and analyse the factors contributing to the AMI and the early diagnostic tools that can be used for MI.

**MATERIALS AND METHODS**

This study was conducted at a Cardiac Care Center, where in 183 patients suffering from chest pain and 25 apparently healthy subjects (the control group without present or past history of Myocardial Infarction), who visited the hospital for a routine check-up to rule out any cardiac problem were enrolled. ECG, blood biochemistry and medical history of all the subjects under study was recorded including age, level of education, diabetic, hypertension, mental stress, physical exertion, psychological condition, patient history of MI, family history of MI and sudden death due to MI, time of onset of chest pain, smoking habits and ECG findings. The estimation of the blood biochemical components was taken up in the hospital laboratory by use of 4 ml of venous blood samples that was collected immediately after hospital admission from each patient and control using disposable syringes for estimation of cardiac enzymes, lipid profile and myoglobin. All blood samples were allowed to clot at room temperature and then centrifuged at 4000 r.p.m. to obtain the serum. The clear serum was taken immediately for analysis or stored at 2 - 8°C and the estimation done within 24 hours. Fully automatic immunoanalyzer MAGIA 7000 was used for the measurement of Myoglobin. Manual spectrophotometer was used for analysis of the cardiac enzymes and lipid profile. All kits and reagents were procured from sigma chemical company.
Estimation of Myoglobin
Serum was incubated simultaneously with the following reagents: Magnetic particles coated with a monoclonal anti-Mgb antibody, a second monoclonal anti-Mgb antibody, which differs from the first and which is conjugated with alkaline phosphatase (AP conjugate). After formation of a complex of particles/Mgb/AP conjugate excess conjugate is removed by washing steps. The remaining enzyme activity bound to the particles is directly proportional to the amount of Mgb in the sample [10].

Estimation of Lactate dehydrogenase (LDH)
Lactate dehydrogenase (LD or LDH) catalyzes the reduction of pyruvate by NADH to form lactate and NAD+. The catalytic concentration is determined from the rate of decrease of NADH measured at 340 nm. The standard methods used for measuring LDH are kinetic methods [11,12].

Estimation of Creatine kinase (CK)
Creatine Kinase (CK) catalyzes the phosphorylation of ADP, in the presence of creatine phosphate, to form ATP and creatine. The catalytic concentration is determined from the rate of NADPH formation, measured at 340 nm, by means of the hexokinase (HK) and glucose-6- phosphate dehydrogenase (G6P-DH) coupled reactions [13].

Estimation of Creatine Kinase-MB (CK-MB)
A specific antibody inhibits CK-M subunits but it does not affect to the CK-B subunits. CK-B catalytic concentration, which corresponds to half of CK-MB concentration, is determined from the rate of NADPH formation, measured at 340 nm, by means of the hexokinase (HK) and glucose-6- phosphate dehydrogenase (G6P-DH) coupled reaction [13,14].

Estimation of Aspartate aminotransferase (AST/GOT)
Aspartate aminotransferase (AST/GOT) catalyzes the transfer of the amino group from aspartate to 2-oxoglutarate, forming oxalacetate and glutamate. The catalytic concentration is determined from the rate of decrease of NADH measured at 340 nm, by means of the malate dehydrogenase (MDH) coupled reaction [13,15].

Estimation of Cholesterol
Free and esterified cholesterol is measured by means of a coupled reaction with cholesterase and peroxidase enzymes producing Quinoneimine, a colored complex that can be measured by spectrophotometry [16].

Estimation of High Density Lipoprotein (HDL) Cholesterol
Very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) in the sample precipitate with phosphotungstate and magnesium ions. The supernatant contains high-density lipoproteins (HDL). The HDL-cholesterol is then spectrophotometrically measured by means of the coupled reactions as described previously for cholesterol.

Estimation of Low Density Lipoprotein (LDL) Cholesterol
Low-density lipoprotein (LDL) in the sample precipitate with polyvinyl sulphate. Their
concentration is calculated from the difference between the serum total cholesterol in the supernatant after centrifugation. The cholesterol is spectrophotometrically measured by means of the coupled reactions as earlier [17].

**Estimation of Triglycerides**

Triglycerides in the sample are measured by means of the sequential reactions by lipase, glycerol kinase, Glycerol-3-phosphate oxidase and peroxidase producing Quinoneimine, a colored complex that can be measured by spectrophotometry [18].

**RESULTS**

The percentage of patients experiencing Myocardial Infarction (MI) in relation to various physiological factors like age, weight, level of education, physical and emotional condition and medical risk factors viz. diabetes and hypertension are presented in table No. 1. Further the percentage of patients experiencing Myocardial Infarction (MI) in relation to other diverse features like patient’s history of MI, family history of MI, family history of sudden death due to MI, time of chest pain onset, smoking habits and ECG findings are presented in table No. 2. Moreover, table No. 3 depicts the lipid profile in the study group with Myocardial Infarction. The levels of lactate dehydrogenase, creatine kinase, creatine kinase-MB and aspartate transaminase, myoglobin as the biomarkers for myocardial infarction immediately and 24 hours following commencement of chest pain are presented in table No. 4 and the same have been outlined in figure No. 2.

It was observed that the maximum incidence of myocardial infarction occurred in the age group of 51 to 60 and in persons weighing between 70 and 90 kgs. The occurrence of MI decreased with increase in the educational status of the subjects. In relation to the physical and emotional condition of the patients, it was observed that physical exertion had the maximum effect on the frequency of MI. Furthermore diabetics and hypertension has a greater impact on the onset of MI as compared to non-diabetics and non-hypertensive individuals. About 74% of the cases recorded for MI were due to heavy smoking. Majority of these patients had a second attack of MI (59%) and many had family history of MI (54%). However scrutiny of the family history of sudden

<table>
<thead>
<tr>
<th>Age groups</th>
<th>Weight (Kgs)</th>
<th>Level of education</th>
<th>Physical and emotional condition</th>
<th>Risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 40</td>
<td>9</td>
<td>&lt; 50</td>
<td>0</td>
<td>Primary 37</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>[Mental Stress 29</td>
<td>Diabetic 56</td>
</tr>
<tr>
<td>41-50</td>
<td>23</td>
<td>50-70</td>
<td>28 Physical exertion 28</td>
<td>Non diabetic 44</td>
</tr>
<tr>
<td>51-60</td>
<td>47</td>
<td>71-90</td>
<td>University 23 Psychological 14</td>
<td>Hypertensive 60</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>21</td>
<td>&gt; 90</td>
<td>Post-graduate 12 Non-specific 11</td>
<td>Non hypertensive 40</td>
</tr>
</tbody>
</table>

Table 1: Percentage of patients experiencing Myocardial Infarction (MI) in relation to various physiological and medical factors

<table>
<thead>
<tr>
<th>Patient history of MI</th>
<th>Family history of MI</th>
<th>Family history of Sudden death due to MI</th>
<th>Time of chest pain onset</th>
<th>Smoking Habits</th>
<th>ECG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>First attack</td>
<td>41</td>
<td>Had</td>
<td>Had</td>
<td>Morning 69</td>
<td>Smoker 74</td>
</tr>
<tr>
<td>Second attack</td>
<td>59</td>
<td>No</td>
<td>Didn’t</td>
<td>Evening 31</td>
<td>Non smoker 36</td>
</tr>
</tbody>
</table>

Table 2: Percentage of patients experiencing Myocardial Infarction (MI) in relation to diverse features
Table 3: Lipid profile (mg/dl) in the study group with Myocardial Infarction

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Patients</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>233.6 ± 108.5</td>
<td>159.17 ± 78.4</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>191.6 ± 96.3</td>
<td>122.0 ± 63.7</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>132.25 ± 88.2</td>
<td>92.5 ± 50.6</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>33.6 ± 18.1</td>
<td>41 ± 22.5</td>
</tr>
</tbody>
</table>

Table 4: Showing mean values of various biochemical parameters at the time of myocardial infarction and after 24 hours of infarction

<table>
<thead>
<tr>
<th>BIOCHEMICAL PARAMETER</th>
<th>Normal Value</th>
<th>MI patients (N=120)</th>
<th>Control (N=25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate dehydrogenase (LDH)</td>
<td>235 – 417 U/L</td>
<td>326 ± 96.4</td>
<td>266 ± 46.7</td>
</tr>
<tr>
<td>Creatine kinase (CK)</td>
<td>25 - 195 U/L</td>
<td>1259 ± 789.6</td>
<td>274 ± 32.2</td>
</tr>
<tr>
<td>Creatine kinase – MB (CK-MB)</td>
<td>up to 24 U/L</td>
<td>899 ± 223.2</td>
<td>63 ± 14.01</td>
</tr>
<tr>
<td>Aspartate Transaminase (AST/GOT)</td>
<td>&lt; 42 U/L</td>
<td>21 ± 2.9</td>
<td>12 ± 2.6</td>
</tr>
<tr>
<td>Myoglobin (Mgb)</td>
<td>&lt; 72 ng/ml</td>
<td>49 ± 12.98</td>
<td>22 ± 2.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>105 ± 25.0</td>
<td>25 ± 2.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>51 ± 19.21</td>
<td>39 ± 5.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>412 ± 103.6</td>
<td>38 ± 5.6</td>
</tr>
</tbody>
</table>

Figure 1: Showing the percentage of patients in relation to various risk factors causing myocardial infarction

Figure 2: Showing the pattern of various biochemical parameters at the time of myocardial infarction and after 24 hours of infarction
death due to MI resulted in only a few (44%) having such a history. Most of the cases of MI were reported in the morning hours (69%) and only 55% of these cases showed significant ECG findings, hence were advised for blood biochemical markers for MI.

**Myoglobin**

It can be seen that within 6 hrs of onset of chest pain the level of myoglobin was significantly elevated to about 8 fold of normal value and about 16 fold of control value, being 642 ng/ml and 38 ng/ml for patients and control respectively. This level remained higher even after 24 hrs of admission (412 ng/ml). The same trend was also noticed in other groups of patients admitted to hospital at 10, 20, 24 hrs following onset of chest pain (Figure 2). The myoglobin level was much reduced in patients admitted to the hospital after two days of the inception of chest pain, but was still significantly higher than normal value or control, and continued to be elevated even after 24 hrs of admission. Patients admitted to the hospital after three days of commencement of chest pain, have shown considerable low levels of myoglobin. Concentration reduced to about normal value 24 hrs after that.

**Creatine kinase (CK)**

Creatine kinase dramatically elevated within the first 24 hrs in all patients admitted to hospital during 6, 10, 20, 24 and 48 hrs following commencement of chest pain. The CK began to fall towards normal values after 24 hrs in patients admitted after 48 hrs of chest pain (figure 2).

**Creatine kinase MB (CK-MB)**

CK-MB gradually elevated in patients admitted 6, 10, 20, 24 and 48 hrs from chest pain attack, reaching a peak after 24 hrs of chest pain attack, then CK-MB levels declined to normal values after 48 hrs.

**Lactate dehydrogenase (LDH)**

The LDH level exhibited increase levels in patients admitted 6, 10, 20, 24 and 48 hrs following chest pain attack, and the levels persisted all through 24 hrs irrespective of the time of hospital admission.

**Aspartate transaminase (AST, GOT)**

AST level was not elevated after 6 hrs or 10 hrs following the chest pain attack, but a dramatic elevation was seen after 24 hrs following hospital admission, however increased levels of AST was noticed in patients admitted to hospital after 24 or 48 hrs following the attack. The levels were within the normal value in patients admitted after 48 hrs of the chest pain attack, and remained so following 24 hrs of admission. It is worth mentioning here that the levels of myoglobin, CK, CK-MB, LDH and AST were within the normal range in about 28% of the patients admitted hospital following chest pain attack.

**Lipid profile**

There was a significant increase in total cholesterol level in patients with MI as compared to control at the time of admission to the hospital. A significant increase in triglyceride concentration over control was also seen. A significant increase in LDL-
cholesterol over the control value was also seen. No significant difference in HDL-cholesterol levels between chest patients and controls.

**DISCUSSION**

The preliminary findings obtained from this study revealed that clinical assessment and ECG examinations showed that 72% of the admitted patients were diagnosed as having myocardial infarction (MI) while 28% were found to suffer from chest pain. Cardiac markers were estimated as soon as the patients suffering from chest pain attack were admitted to the Hospital. It appeared that myoglobin concentration was immediately elevated during the first six hours of admission, and remain to be so until 24 hours, this pattern of elevation occurred in all patients with chest pain admitted to CCU at 6 hours, 10 hours, 20 hours and 24 hours of chest pain onset, but patients admitted to CCU after 2 days of the onset of chest pain have shown lower value of myoglobin.

In all patients, the level of myoglobin decreased to normal value, these findings are in agreement with the results of Mair, et al (1995) [19]. Myoglobin and CK-MB are more sensitive in the first hours of attack and this is in agreement with BIOMACS study that reported on patients with chest pain and a nondiagnostic ECG presenting up to 12 hrs from the onset of symptoms. Using half-hourly blood samples for the first 3 hrs from admission, it was found that a combination of myoglobin and CK-MB reached a sensitivity of 92% after 2 hrs and 98% after 6 hrs with the specificity reaching 93% [20].

On the other hand, creatine kinase (CK) and CK-MB gradually elevated in all patients admitted at 6, 10, 20, 24 and 48 hours, and both of them reached peak after 24 hours of chest pain attack. This result is also in line with the work of Ravkilde, et al (1995) [21].

Lactate dehydrogenase, showed increased levels in patients admitted to CCU at 24 and 48 hours from the onset of the chest pain attack and persisted at high levels till after 72 hours.

Aspartate transferase (AST), also showed elevated values but after 24 hours of chest pain attack, and peak was noticed in patients admitted the hospital after 24 and 48 hours of onset of chest pain, but patients admitted to hospital 24 hours after the chest pain have shown normal values of AST.

Blood samples taken at the moment of hospital admission of patients suffering chest pain attack, and who had a well defined onset of chest pain, showed that during the first 6 hours period, Myoglobin was most significantly sensitive than CK, CK-MB, LDH and GOT. During the 10 – 12 hours of chest pain, the sensitivity of CK and CK-MB were higher than other enzymes, then LDH and GOT sensitivity appear in those, who came after 24 hr of attack.

Our study revealed that, within the 6 – 10 hours after the onset of chest pain, the sensitivity of CK, CK-MB, LDH and GOT (AST) were too low to justify their measurements. The sensitivity of myoglobin was greater after successful early reperfusion. Myoglobin becomes more sensitive during the first 6 hours, while CK, CK-MB and GOT are not sensitive enough during 6 – 10 hours from the onset of chest pain. These findings are in agreement with the results of Mair et al, (1995) [19] and Bakker et al (1993) [22].

As a decrease of myoglobin concentration was seen after 24 hours of chest pain attack, CK and CK-MB activities raised rapidly in patients admitted to hospital 24 hours after the onset of chest pain. An increase of about 879% and 554% over the upper reference limit value were seen for CK and CK-MB respectively. These findings are in agreement with the results of Ravkilde (1995) [21] and William (1996) [23] who reported that after myocardial infarction (MI), serum value of CK, was found to increase after about 6 hours, reaches a peak level in 24–30 hours and returns to normal in 2–4 days.
On the other hand, markers such as LDH and GOT remain unchanged, e.g. within normal values at the first 6 hours of chest pain attack in patients admitted CCU but serum levels raised sharply after 24 hours reaching an increase of about 263% and 588% over the upper reference limit for LDH and GOT respectively. These findings agree with the results of Newby, et al (1995) [27] who reported that in acute MI serum activity of GOT raised sharply within the first 12 hours, with a peak level at 24 hours or over and return to normal within 3–5 days, while LDH activity raised within 12–24 hours, reached peak at 48 hours (2 – 4 days) and then return gradually to normal from 8th to 14th day.

CONCLUSION

This study indicated that Myoglobin and CK-MB are more sensitive cardiac markers compared to total CK, LDH and AST for the diagnosis of Myocardial Infarction. Measuring myoglobin level could be of great help in confirming the diagnosis of Myocardial Infarction especially in the first few hours following the onset of chest pain. Recent cardiac marker such as and myoglobin is very useful in the diagnosis of acute myocardial infarction in the first hour of the attack and more sensitive and specific than cardiac enzymes recommended to introduce this test in our laboratories. Myoglobin, cardiac Toponin I or T should replace CK and CK-MB during the early period following the onset of chest pain.

REFERENCES