Serum Human Myoglobin in Acute Myocardial in Farction with and without streptokinase Therapy

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The material of this study comprised 30 patients with acute myocardial infarction as a test group, which were divided into 2 groups each of 15 patients. The first group (10 males & 5 females) with ages ranging between 41 to 72 did not receive streptokinase as thrombolytic therapy. The second group 9 males & 6 females with ages ranging between 50 to 62 years received I.V streptokinase as thrombolytic therapy. A control group (10) of age & sex matched subjects were also included in our study. Patients suffering from acutemyocardial infarction were selected from the inpatients admitted to coronary care units of Zagazing and Mansoura University Hospitals.All patients had an admission ECG. According to ECG location of infarction, the first group (non streptokinase treated group) was divided into 2 subgroups, subgroup Ia comprising 12 patients with either anterior myocardial infarction or anterior myocardial infarction with septal or-lateral extension and subgroup Ib comprising 3 patients with inferior myocardial infarction. Similarly, the second group (streptokinase treated group) was divided into 2 subgroups, subgroup IIa comprising 11 patients with either anterior myocardial infarction or anterior myocardial infarction withseptal or lateral extension and subgroup IIb comprising 4 patients with inferior myocardial infarction. Both groups I & II received the usual treatment of acute myocardial infarction, in addition group II, received IV streptokinase.Group III (control group) included 10 normal subjects with age ranging from (40 - 53 years). selected on the basis of having adequate ECG recording of normal electrocardigraphic findings. These patients had no history of cardiovascular or any other medical disease and were found to be normal by physical examination, ECG and laboratory investigations.All patients were subjected to thorough history clinical examination, serum myoglobin determination, serum CPK & SGOT determination and other laboratory investigations (lipogram, fasting and past prandial blood sugar and serum creatinine). A twelve lead ECG was done for every patient on admission. In streptokinase treated group, another 12 lead ECG was done for every patient 6 hours after streptokinase therapy. Chest x-ray was done for every patient to detect signs of pulmonary congestion or cardiac enlargment.There was decrease in ST segment elevation in most cases, after intravenous administration of streptokinase. Also in some cases, after intravenous administration of streptokinase, the ST segment returned to the base line.There was significant increase in serum myoglobin level of both non streptokinase and streptokinase treated groups 6 hours, from the onset of sympyoms.Also there was

significant increase in serum myoglobin level in non streptokinase treated group after 12 hours from the onset of symptoms. On the other hand, there was no significant change in serum myoglobin level after 24 hours from the onset of symptoms in non streptokinase treated group and no significant change in serum myoglobin level in streptokinase treated group after 12 hours from the onset of sympyoms there was significant increase in serum myoglobin level of non streptokinase treated group compared to streptokinase treated group, only after 12 hours from the onset of symptoms.There was no significant change in serum myoglobin level of patients with acute anterior myocardial infarction and inferior myocardial infarction of non streptokinase treated group, compared to streptokinase treated group.There was strong correlation between serum myoglobin level and serum creatine phosphokinase level of both non streptokinase treated and streptokinase treated groups after 12 hours from the onset of symptoms. Also, there was strong correlation between serum myoglobin level and serum oxalacetictransaminase (SCOT) level of both non streptokinase and streptokinase treated groups. CONCLUSION AND RECOMMENDATIONS:1-This study illustrates the value of serum myoglobin measurment for diagnosis of acute myocardial infarction and follow up of thrombolytic therapy in patients with acute myocardial infarction.2-It has been shown that myoglobin, is one of the earliest appearing markers of acute myocardial infarction. Our results support this and indicates that myoglobin is a sensitive test for early diagnosis of acute myocardialinfarction:3-Early non invasive prediction of coronary artery patencyin the majority of patients after initiation ofthrombolytic therapy can be estimated by early slope analysis of myoglobin, which is superior to the other serum markers examined, in addition, myoglobin exhibits the most rapid concentration increase of all markers evaluated.4-As a test to confirm diagnosis of acute myocardial infarction myoglobin has two limitations first myoglobin shows earlier peak and return to normal values than creatine kinase and creatine kinase myoglobin after myocardial infarction; myoglobin should be measured soonafter a heart attack . If hospital admission is delayed for 12-24 hours or more, myoglobin concentration will have fallen sharply and may be within the refrence range. Thus, if chest pain is recurrent, myoglobin concentration may be a better indicator of further tissue necrosis than creatine kinase and creatine kinase myoglobin. Second, myoglobin lacks specificity for cardiac muscle. When the test result is positive, the clinical setting must be taken into account. In the absence concomitant damage to skeletalmuscle or severly impaired renal function, a positive myoglobin test result predicts myocardial infarction with a very high probability. A negative myoglobin assay result in the 4-12 hours period after the onset of infarct related symptoms, on the other hand allows acute myocardialinfarction to be ruled out within a few minutes with a very high probability.