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Serum Human Myoglobin in Acute Myocardial Infarction With and Without Streptokinase Therapy

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M. B. B. Ch.

Thesis

Submitted in partial fulfillment
of the *Master Degree* in *General Medicine*

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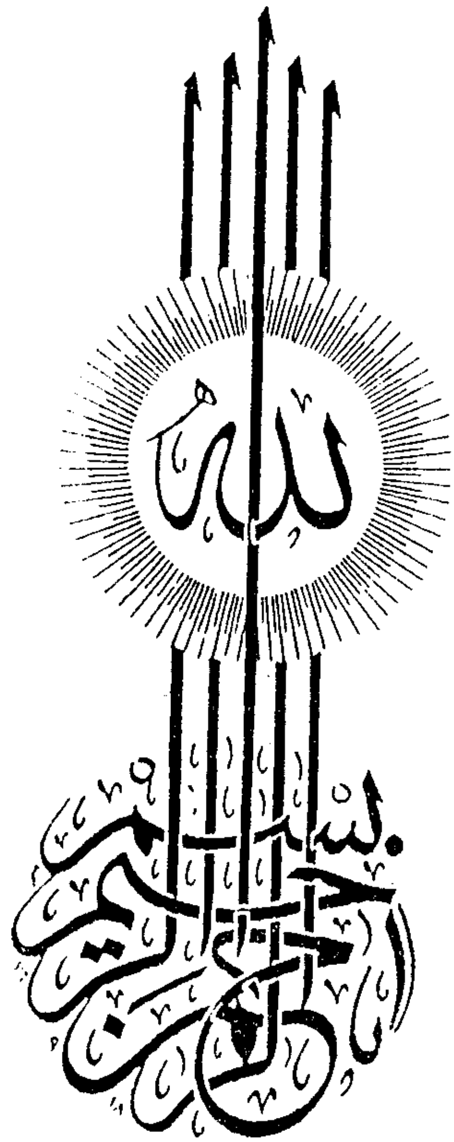
1994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ACKNOWLEDGMENT

FIRST AND FOREMAST, THANKS TO GOD.

I would like to express my sinerce thanks and deepest gratitude to my Professor **Dr. EKRAM M. El-ASUTI** professor of internal medicine and the head of internal medicine department, for his kind advice and centinuous help.

I appreciate too much the guidance offered by professor **Dr. ABD El-SHAFI M. TABL**, professor of Internal medicine for his continuous help and great aid during this work.

I am also deeply indebeted to **Dr. AlAA EL-DIN E. ABDALLA**, assistant professor of Internal Medicine.

Lastly, but not the last, special thanks to All Staff Members and all workers in the coronary care Unit in Zagazing and Mansoura University Hospitals who exteneted to me a helping hand during process of this work.

CONTENTS

LIST OF ABBREVIATIONS

AMI	:	acute myocardial infarction.
CPK (CK)	:	Creatine phosphokinase .
ECG	:	Electrocardiogram.
MB-CK	:	The creatine phosphokinase form predominantely present in the myocardium.
HBD	:	Hydroxybuturate dehydrogenase.
HDL	:	High density lipoprotein.
IU	:	International unit
IV	:	Iniravenous.
LDH	:	Lactic dehydrogenase
MI	:	Myocardial infarction.
mm/sec.	:	Millileter/second.
ng	:	Nilligram.
S4	:	Fourth heart sound.
SD	:	Standard deviation.
SE	:	Standard error
SGOT	:	Serum glutamic oxalacetic transaminase .
SK	:	Streptokinase.
μg	:	microgramn.
μl	:	microliter.
Σ	:	Summation of

INTRODUCTION AND
AIM OF THE WORK

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Apart from the patient who presents with the classic symptoms and typical electrocardiographic changes, the early detection of acute myocardial infarction based on conventional clinical and electrocardiographic findings alone is difficult *(Johannes et al., 1992)*. Most patients with acute myocardial infarction show some E.C.G changes, but these changes are diagnostic in only about one third of patients with acute myocardial infarction when they present at the emergency room *(Johannes et al., 1992)*. Creatine kinase and creatine kinase myoglobin activity which are the most frequently assayed biochemical markers in emergency laborataries do not rise in serum until the fourth to the eigths hour after the onset of chest pain and so, are not sensitive enough for diagnosis during the early stages of acute myocardial infarction *(Lee et al., 1986)*.

Most investigators who used intravenous thrombolytic agents have used ST segment elevation on the electrocardiogram as an inclusion criterion. The published data of the IST-2 trial found a reduced mortality in patients with and without S T segment elevation (ISIS-2 collaborative group 1988). Since the induction of a fibrinolytic state is associated with eased morbidity, additional testing would be useful in the early identification of patients with acute myocardial infarction. Raised serum concentrations of total creatine kinase and its MB fraction though very sensitive and specific tend to occur later than ST segment changes during the course of myocardial infarction. Rapid prediction of the outcome of therapeutic reperfusion, could become increasingly important by allowing the use of alternative invasive means of recanalization if initial non invasive attempts to restore reperfusion fail. Several studies have compared creatine kinase and creatine kinase MB with myoglobin during the early phase of myocardial infarction. In general, these studies have found that serum myoglobin has a higher diagnostic sensitivity than creatine kinase and its isoenzymes (*Sylven et al., 1978 and Noregard et al., 1980*). Serum concentrations of myoglobin above the normal range have been found as early as one hour after myocardial infarction, with peak activity in the range of 4-12 hours (*Stone and Willerson 1983*). Despite these impressive characteristics, serum myoglobin has not been used extensively for routine analysis in myocardial infarction. The main reason has been the very long assay time required (*Magnus et al., 1989*).

The hypothesis that rapid determination of serum myoglobin may improve and speed the accuracy of diagnosis in patients with suspected acute myocardial infarction deserves further evaluation.

The aim of the present study is to evaluate the value of serum myoglobin to diagnose acute myocardial infarction as well as its value for follow up of patients receiving thrombolytic therapy in the form of intravenous streptokinase using rapid assay of myoglobin by using the immunoturbidimetric assay (*Tuengler et al., 1988*) which gives quantitative results about a minute after the start of the assay.

REVIEW OF LITERATURE

ACUTE MYOCARDIAL INFARCTION

THE PATHOPHYSIOLOGY OF ACUTE MYOCARDIAL INFARCTION :

It has become increasingly clear that thrombus formation plays a major role in acute ischaemic heart attacks (*Falk, 1989*). A rational approach to the management of patients with acute ischaemic syndromes demands some knowledge of the fundamental mechanisms underlying the initiation, promotion and growth of a coronary thrombus and the dynamic changes occurring during the evolution of a thrombus.

Theoretically, the mechanisms underlying fatal and non fatal thrombus formation differ, but recent angiographic studies (*Nakawagaz et al., 1988*) revealed angiographic evidence of ruptured atheromatous plaque under the majority of coronary thrombi. So, plaque rupture seems to play a major role in both fatal and non fatal coronary thromboses. However, rupture of the plaque surface does not always cause luminal thrombosis : rupture may result in haemorrhage from the lumen into the plaque without gross evidence of thrombosis at the rupture site, alternatively, in addition to haemorrhage into the soft lipid material a non occlusive luminal thrombosis may occur with consquent rapid progression of the stenosis (*Davies and Thomas, 1985*).

Microscopic examination of coronary thrombi and ruptured plaues reveals that the degree of pre-existing stenosis at the rupture site is one of the main determinants of the outcome *(Falk, 1983)*. The greater the pre-existing stenosis at the rupture site, the greater the risk of thrombus formation when plaque rupture occurs. It seems reasonable to conclude that a stenosis causing rapid flow promotes arterial thrombus formation.

In addition to the severity of stenosis and the nature and extent of exposed thrombogenic material, the actual thrombotic thrombolytic equilibrium seems to be major determinant of coronary thrombosis. It is possible to modify this equilibrium with aspirin, heparin and plasminogen activators, with beneficial effect on prognosis *(Harrison et al., 1984 and wall et al., 1989)*.

Coronary spasm may contribute to the dynamic occlusion before, during and after infarction *(Hackett et al., 1987)*, but a recent study suggests that thrombus formation also plays a role in these cases *(Rapold et al., 1989)*.

The three main determinant of infarct size are : the size of the area at risk, the severity and duration of ischaemia and the vulnerability of the myocardium. The first tow factors are the most important. The extent of the at risk vascular bed depends on the coronary anatomy and the site of coronary occlusion (*Therout et al., 1988*). Risk areas associated with occlusion in the left anterior descending artery are usually greater than risk areas associated with occlusion in the circumflex artery or the right coronary artery. Consequently, the treatment gain may be much greater and more easily obtained in anterior infarction than in posterior or inferior infarction.

The main determinant of available collateral flow in cases of sudden coronary occlusion seems to be the degree of pre-existing atherosclerotic stenosis (*Cohen et al., 1989*). The greater this is, the greater the extent of collateral flow. The significance of this collateral flow is evident, 10% of patient with rest angina have a totally occluded coronary artery but no definite infarction because of well developed collateral circulation (*De-Zwaan et al., 1989*), 25% of patients with non Q wave infarction have a totally occluded artery at the time of acute angiography. increasing to 40% of patients in the course of a few days (*DeWood et al., 1986*), but due to parallel increase in collateral flow a considerable amount of moycardium is saved, in Q wave infarction many patients experience a relatively extensive infarction due to poor collateral circulation (*DeWood et al., 1986*). Thus collateral supply may preserve myocardium at risk.

CLINICAL PRESENTATION OF ACUTE MYOCARDIAL INFARCTION

Symptoms:

Unfortunately, by the time the patient calls the physician, he has usually been experiencing symptoms for several hours. In one study, a third of the patients who had an acute myocardial infarction made their own diagnosis prior to admission, another third interpreted the chest pain as indigestion and the remaining patients were unable to explain the nature of their chest pain (*Goldstein et al.*, *1972*). Prodromal symptoms may be present several weeks prior to the attack. In one study prodromal symptoms occured in about 65% of patients admitted to a coronary care unit with acute myocardial infarction (*Solmon et al.*, *1969*). Prodromal symptoms in that report usually were present 24 hours or longer before the occurrence of the definitive attack of acute myocardial infarction. Chest pain was the most common symptom, and it usually was progressive and recurrent. pain in 18% was associated with other symptoms such as dyspnoea, diaphoresis and light headedness. In 9%, pain was absent and symptoms included burning in the chest, dyspnoea, vertigo, weakness and fatigue. Patients with previous coronary artery disease may note a sudden change or return of anginal symptoms.

The majority of patients with acute myocardial infarction have chest pain that is similar in location and quality to that of angina. The distress is classically located in the retrosternal area with radiation to the inner aspect of the left arm, hypothenar eminence and fourth and fifth fingers. It may be referred to the neck and corresponding areas of the right arm, to both arms and to the lower Jaw. However, just as for angina, the distress may occur in only a segment of this distribution - it may be only in the chest, only in the interscapular area, only in the Jaw, only in the neck, only in the left or rigt arm and at times in only a part of the arms. It is usually described as a tight, squeezing, burning, indigestion, heavy, expanding or boring sensation. Seldom, it is sharp. It may begin as a sharpness and then develop into a heavy squeezing feeling. The pain of acute myocardial infarction differs from angina in that it lasts 30 minutes to several hours, also it frequently occurs at rest. Many patients have nausea and vomiting due to activation of vagal reflexes or to activation of cardiac receptors, namely in left ventricle, which initiate impulse that travel in the vagus to the vasomotor centres and inhibit sympathetic discharge (Bezold-Jarisch reflex), especially in patients with inferior infarction.

Various epidemiologic and autopsy studies have shown that 20 to 60% of all myocardial infarcts are unrecognised. Five years study reported an incidence of 39.8% unrecognized infarcts *(Medalie, 1976)*. Half of these subjects had no recollection of any symptoms or illnesses since their last examination, and their infarction were classified as silent. Another 42.3% had atypical attack. Among the remaining 7.7% the physicians thought that their symptoms might be related to infarcts, but the ECGs were not initially interprted as such. However the next survey examination of these subjects showed tracings interpreted as infarcts. American studies report 30% of all infarcts as unrecognized, half of which are classified as silent. While it is impossible to make an accurate prediction of the incidence of unrecognized infarcts, studies suggest that for every clinical infarct detected there is probably at least one that unrecognized in the same population group *(Margolis et al., 1973)*. The unrecognized infarction appears to be more frequent in patients with diabetes.

The patient with acute myocardial infarction may have evidence of some of the risk factors. Fundoscopic examination may reveal hypertensive, diabetic or lipaemic changes. Xanthomas may be noted around the eyelids, tendons, the bony tubercles, about the trunk, or legs in uncomplicated case, usually the patient appears sweaty. Hypertension may be present and sometimes hypertension during pain or apprehension even though the patient was not previously hypertensive. An abnormal cardiac apical pulsation or an ectopic precordial impulse may be detected even in the absence of cardiomegaly. An atrial (S4) gallop is present in the majority of patients with acute infarction. The first sound may become faint. Rarely there is paradoxical splitting of the second sound, unless left bundle branch block is present. The heart rate can be within normal range or sinus tachcardia or bradycardia (usually with an inferior infarction) may be present. Fever rarely more than 101° F but sometimes as high as 103°F usually develops after 24 hours, and a pericardial friction rub may appear after this period. The fever and pericardial rub can persist for several days.

In complicated cases, disturbances in the rhythm and conduction of the heart can be noted by physical examination. Final differentiation depends on the ECG. Major arrhythmias include persistent premature ventricular beats in excess of six beast per minute. Conduction disturbances can appear as A-V block or the development of right or left bundle branch block, left ventricular failure may present with persistent sinus tachcardia, pulmonary congestion, ventricular gallop or as frank pulmonary oedema. Later, right ventricular failure can develop. Cardiogenic shock should be differentiated from hypertension. Often the blood pressure may fall below 90 mm Hg, yet the patient is warm and has adequate urinary output.

An apical systolic murmur due to papillary muscle dysfunction may become audible.

Even though the left ventricle is usually damaged in acute myocardial infarction, it is now recognized that with acute inferior and posterior infarcts the right ventricle can be involved. This should be suspected in patients with such infarcts when there is an elevated systemic venous pressure without clinical or x-ray evidence of significant pulmonary congestion. Occasionally prominent systolic C-V waves can be seen in the jugular venous pulse as a result of tricuspid insufficiency due to infarction of the right papillary muscle. Other complications include pericarditis, thromboembolism and post myocardial infarction syndrome.

ELECTROCARADIOGRAPHIC FINDINGS IN ACUTE MYOCARDIAL INFARCTION:

Myocardial infarction may present with a typical clinical history, yet there may be no physical abnormalities or ECG changes. Unfortunately, during the early hours after the onset of myocardial infarction when the patient is more susceptible to primary ventricular fibrillation, the ECG may be normal. If serial tracings are obtained 90% of patients with infarction will show abnormal changes. It may take several days for changes to develop, especially in the nontransmural types. After an infarction, the tracing can return to normal. The site of infarction may be indicated as transmural (Q waves), intramural (ST depression) or subepicardial (ST elevation). The significance of reciprocal ST depression in an anterior or inferior infarction is still debated. However, most agree that myocardial infarction with ST segment depression in remote myocardial areas represent more extensive myocardial necrosis, and if such changes persist beyond the immediate peri-infarction period, more cardiac events are likely to occur because of multivessel coronary artery discease *(Becker and Alpert, 1988)*. Right ventricular infarction is most often noted with an inferior infarction. Therefore, findings of an inferior infarction will usually be present, and often in the right chest leads, especially in Rv4, the ST segment will be elevated. Surface mapping of RS-T segment (from 35 to 72 points on the chest) occasionally may be of value in diagnosis of acute myocardial infarction when the standard ECG is normal *(Reid et al., 1971)*. Recording over a wider area may reveal a site of infarction which is inaccessible to the standard tracing.

PLASMA ENZYME CHANGES IN ACUTE MYOCARDIAL INFARCTION :

Serum glutamic oxalacetic transamimase (SGOT), lactic dehydrogoenase (LDH) and isoenzymes, hydroxybuturate dehydrogoenase (HBD), and creatine kinase (CK or CPK) and isoenzymes are enzymes that are released from irreversibly injured myocardial cells into the circulation and can be measured. The SGOT was the first enzyme used clinically, but it is not used often today because it is nonspecific. The SGOT rises in 12 hours, peaks at 24 to 48 hours and returns to control by 3 to 4 days. *(Fisher et al., 1983).* The enzymes LDH and HBD exceed the normal range 24-48 hours after the onset of myocardial infarction, reaches a peak 3-6 days after the onset of myocardial infarction and returns to normal levels 8-14 days after the infarction. These are helpful in patient who does not seek aid until 48 hours after the episode of pain *(Lee and Goldman, 1986).* The enzyme CK rises in 4 to 8 hours, peaks by 5-58 hours and returns to normal in 72 hours *(Herlitz 1984).* The SGOT, LDH and HBD are often elevated because of non cardiac disorders, such as liver disease or liver congestion. The CK is not found in the liver but is prominent in skeletal muscle. The serum level can be elevated by any muscle trauma. It should be mentioned that the serum CK can rise after heart catherization, particularly selective coronary arteriography. However the other enzymes and isoenzymes do not exceed the accepted clinical range *(Peter ., 1990).* It must be recognized that infarction can be present without elevation of the enzymes if there is a small area of necrosis. The setting of chest pain and an enzyme elevation to peak and then decline is suggestive of infarction. Serum enzymes not only aid in diagnosis, but also give a rough idea of the size of infarction, which is related to their increase. *Coodley (1973)*, found a direct relationship between amplitude of enzyme rise and early mortality and morbidity. There was a mortality of 56% in patients with levels six times normal. If the level was four to five times the normal there was an increased incidence of arrhythmias, heart failure and cardiogenic shock.

Isoenzymes as MB fraction of CK and the ratio of LDH_1 to LDH_2 are more specific for myocardial infarction than total levels of CK and LDH. Isoenyemes of CK are dimers composed of muscle (M) or brain (B) subunits. The MM dimer predominates in skeletal muscle (MM) and the BB dimer in brain (BB) *(Verat and Mercer, 1975)*. With rare exceptions, marked elevation of plasma MB-CK reflects irreversible myocardial injury, LDH has five isoenzymes, which are numbered in the order of the rapidity of their migration toward the anode of an electric field. LDH_4, moves rapidly where LDH_5 is the slowest. The heart contains principally LDH_1, where liver and skeletal muscle contain primarily LDH_4 and LDH_5. Elevation of LDH and in the ratio of LDH_1 to total LDH occur in more than 95% of patients with acute myocardial infarction *(Weidner, 1982)*.

MYOGLOBIN :

Serum myoglobin has been found to increase significantly within four hours of acute myocardial infarction *(Stone et al., 1977)*. Several studies have compared creatine kinase and creatine kinase MB with myoglobin during the early phase of myocardial infarction. In general these studies have found that serum myoglobin has a higher diagnostic sensitivity than creatine kenase and its isoenzymes (*Sylven et al., 1978 and Norgard et al., 1980*). Serum concentration of myoglobin above the normal range have been found as early as one hour after myocardial infarction, with peak activity in the range of 4-12 hours (*Stone and Willerson., 1983*). Despite these impressive characteristics, serum myoglobin has not been used extensively for routine analysis in myocardial infarction. The main reason was the very long assay times required. More recently the rapid latex agglutination kit for myoglobin gives a result within 10 minutes and can be performed within the bed side (*Mair et al., 1991*).

The use of thrombolytic therapy for myocardial salvage reinforces the need for early and rapid identification and confirmation of acute myocardial infarction within four to six hours after the onset of the infarct related symptoms (*Timi study group,1985*). Creatine kinase and creatine kinase MB activity, which are the most frequently assayed biochemical markers in emergency laboratories do not rise in serum until the fourth to eighth hour after the onset of chest pain (*Lee and Goldman,1986*) and so are not sensitive enough for diagnosis during the early stages of acute myocardial infarction. Myoglobin, by contrast, is an early sensitive marker of myocardial cell injury (*Drexel et al.,1983 and Gibler et al., 1987*).

RADIONUCLIDE STUDIES IN ACUTE MYOCARDIAL INFARCTION :

Technetium $^{-99m}$ pyrophasphate is widely used for imaging in acute myocardial infarction, for this tracer will accumulate only in the infarcted area "hot spot". Such scans are positive in over 90% of patients with transmural infarction (*Dymond et al., 1978*), but nontransmural infarction are less reliably detected. The scans become positive in about 24 hours after the onset of pain and remain so for 5 to 7 days then gradually become negative (*Gazes, 1990*).

Technetium scans are most useful diagnostically in equivocal cases of acute chest pain, especially in the presence of left bundle branch block or an old myocardial infarction where ECG, after, is of limited value in detecting right ventricular infarction and estimating infarct size (*Gazes, 1990*). Abnormal scans can occur with unstable angina (*Walsh et al., 1976*) and ventricular aneurysm and after repeated high energy cardioversion (*Pugh et al., 1976*).

Thallium 201 distributes in the myocardium in proportion to regional blood flow. Therefore an infarcted area will show decreased uptake "Cold spot". It can be positive earlier than the techentium scan, usually within the first 24 hours (*Gazes, 1990*).

CARDIAC CATHERIZATION AND ANGIOCARDIOGRAPHY :

Such studies may become necessary in anticipation of angioplasty or surgery in the presence of acute myocardial infarction if the patient has continuing episodes of pain, septal rupture or papillary muscle rupture. It is also done after thrombolytic therapy (*Gazes, 1990*).

STREPTOKINASE :

Streptokinase is produced from cultures of lacefield group C beta haemolytic streptococci and has a molecular weight of 47.000 daltons (*Sherry and Gustafson, 1985*).

It is an indirect activator converting plasminogen to plasmin. It has no intrinsic enzymatic activity, but it forms a stable monocovalent 1:1 complex with plasminogen. This produces a conformational change that exposes the active site on plasminogen that cleaves arginine 560 on free plasminogen molecules to form free plasmin. As this process continues, the streptakinase plasminogen complex is gradually converted to streptakinase plasmin form, which can also activate and convert plasminogen (*Sharma et al., 1982*).

A loading dose of streptokinase (250,000 units; 2.5 mg) must be given intravenously to overcome plasma antibodies that are directed against the protein. These inactivating antibodies result from prior streptococcal infections (*Verstraete et al., 1966*).

Streptakinase was used in about 15 trials in a loading dose of 250.000 units followed by an infusion of 100.000 units perfusion for 12-24 hours with total dose of 320.000 units in the first 2 hours and total dose of 2.5 million units *(Marder and Francis, 1984)*. Attempts are naw underway to ensure high concentration of therapy in coronary circulation by high dose brief duration intravenous therapy *(Sherry and Gustafson, 1985)*.

PATIENT SELECTION :

History of recent chest pain less than 4-6 hours *(Laffel and Braunwald, 1984 b)* may be up to less than 12 hours *(Olson et al., 1986)* cunsistent with myocardial infarction, including persistent chest pain lasting more than 30 minutes accompanied by ST segment elevation is a major indication for thrombolytic therapy.

There has been debate about the relative benefit of thrombolytic therapy in inferior versus anterior myocardial infarction *(Braunwald, and Klener 1988)*. However, more careful analysis of data has shown that infarct size rather than location is the important factor, with no significant benefit in the smallest of infarcts, while the benefit increases with progressively larger infarcts *(Mauri et al., 1989)*.

Patients with cardiogenic shock also do not benefit much, although uncontrolled studies have suggested that thrombolysis can occasionally lead to dramatic improvement in this syndrome (*Marder, 1983*).

Thrombolytic therapy in unstable angina have been performed. There is no evidence that, they are more effective than anti platelet and/or anti coagulant therapy, which may be sufficient to inhibit further platelet-fibrin thrombosis in the coronary artery (*Gold, 1989 and Neri Serneri et al., 1990*).

CONTRAINDICATIONS TO THROMBOLYTIC THERAPY :

History of cerbrovascular accidents (*Zeleweski and Shi 1991*)

Severe or accelerated hypertension "diastolic pressure greater than 110 mmg Hg" (*Sherry and Gustafson, 1985*).

Recent surgery (within 6 weeks) in a non compressible area (*Glatter, 1990*).

Recent bleeding from the gastrointestinal or genitourinary tract (within 3 months)(*Glatter, 1990*).

Age over 70 years, significant valvular lesion, previous treatment with streptokinase (*Smalling and Gould, 1985*).

Diabetic hemorrhagic retinopathy or other hemorrhagic opthalmic conditions *(Zelewski and Shi, 1991)*.

Hemostatic defects including those secondary to severe hepatic or renal disease *(Zelewski and Shi, 1991)*.

Acute pericarditis and subacute bacterial endocarditis *(Zalewski and Shi, 1991)*.

Presence of a constitutional or acquired coagulation defect, severe liver disease or advanced uremia *(Sherry and Gustafson, 1985)*.

ADVERSE REACTIONS :

Bleeding complications are the most common and the most serious complications of streptokinase. Hemorrhagic complications have occured in about 5% of patients under therapy with streptokinase or urokinase *(Marder, 1979a)*.

Mild allergic reactions occur in about 15% of patients treated with streptokinase *(Bell and Meek , 1979)*.

Transient hypotension occured frequently during intra venous streptokinase infusion *(Ganz et al., 1984)*.

Reperfusion arrhythmia occured in 82% of patients with restoration of antegrade flow. These arrhythmias are usually not dangerous, well tolerated causing only a slight fall in blood pressure due to loss of arterial support mechanism

(Defeyter, 1984). Premature ventricular contractions are the most common arrhythmia noted at the time of reperfusion, an accelerated idioventricular rhythm and ventricular tachycardia are more common acutely after successful reperfusion than after failed therapy *(Cercek & and Horvat, 1985)*.

Ventricular fibrillation was reported in 6% in one series *(Kennedy et al., 1983)*.

MATERIAL & METHODS

MATERIAL AND METHODS

SELECTION OF CASES :

The present study comprised 30 patients with acute myocardial infarction presented to the coronary care unit in Zagazig and Mansoura University Hospitals within six hours from the onset of chest pain as a test group. Diagnosis of acute myocardial infarction was verified, according to the world health organization criteria, (world Health organization criteria for the diagnosis of acute myocardial infarction (1981)) when at least two of the following criteria were ppresent :

a) Typical prolonged chest pain : the duration between the onset of typical chest pain and admission to coronary care units was not more than 6 hours.

b) Characteristic electrocardiographic changes.

c) Elevation of serum level of relevant cardiac enzymes (CPK, SGOT). Intramsular injections were avoideed in all patients included in our study. Patients were excluded from this study if any of the following was present : Cardiogenic shock, renal failure, intramuscular injection, also patients receiving beta blockers and calcium channel blockers were excluded. As regards, streptokinase treated group, patients were excluded from the study, if they have advanced life threatening disease of other organ systems or contraindication to thrombolytic therapy.

Patients were divided into two groups, the first group comprised 15 patients (10 male and 5 females) with age ranging between 41 and 72 years and received the medical treatment of acute myocardial infarction without streptokinase. The second group compromised 15 patients (9 males and 6 females) with age ranging between 50 and 62 years. This group received intravenous streptokinase in addition to other medication of acute myocardial infarction. Streptokinase was given within 6 hours for these patients in a dose of 1.5 million IU in 100 ml of saline or glucose 5% infused I.V over 30-45 minutes. Hydrocortisone 100 mg bolus I.V, was given 2-3 minutes before streptokinase to prevent allergic reactions. Exclusion criteria for streptokinase were adopted before starting therapy.

Patients with arrhythmias were excluded from the study.

A control group (10) of age and sex matched subjects were also included in our study. (5 males and 5 females) with ages ranging between 40 and 53 were also included in our study. These subjects had no history of cardiovascular and were found to be normal by physical examination, ECG and Laboratory investigations.

ALL PATIENTS WERE SUBJECTED TO THE FOLLOWING :

(1) Full medical history and clinical examination with special stress on :-

a) History of :

- Ischaemic chest pain and its duration .
- Congestive lung symptoms and low cardiac output symptoms.
- Hypertension, smoking and diabetes mellitus .
- Family history of risk factors to coronary artery disease.

b) General examination:

- Pulse
- Blood presure
- Temperature
- Oedema of lower limb
- Neck veins.

c) Local examination :

- **Heart : heart sounds and murmurs, gallop**
- **Chest : signs of lung congestion.**

II- INVESTIGATIONS :

1- Electrocardiogram :

Standard 12 lead electrocardiograms were done to both non streptokinase and streptokinase treated patients and and six hours after intravenous infusion of streptokinase, to streptokinase treated patients.

THE ECGS WERE ANALYSED FOR :

Heart rate/minute

P-R interval

ST segment : elevation and depression.

QRS duration and axis degree

Site and extent of myocardial infarction

T wave changes

Observation for any arrhythmias or conduction defects if any.

(2) Laboratory tests :

Both the control group and patients with acute myocardial infarction were subjected to the following :

a) Estimation of serum myoglobin concentration after 6,12,24 hours from the onset of infarction then daily for seven days according to the method described by *Tuengler et al 1988).*

b) Estimation of serum creatine phosphokinase after 12 hours from the onset of infarction according to the method described by *(Szasz et al., 1977).*

c) Estimation of serum oxalacetic transamimase after 12 hours from the onset of infarction according to the method described by *(Reitman and Frankel 1957).*

d) Estimation of fasting and post prandial blood sugar according to the method described by *(Triender, 1969)*.

E) Estimation of serum total cholesterol (*Allain et al., 1974)*. serum triglycerides *(Fassati and Principe, 1982)*, serum high density lipoproteins *(Kostner, 1983)* and serum low density lipoproteins *(Fruchert and Revrt., 1982)*.

f) Estimation of serum creatinine, according to the method described by *(Bartels et al., 1982)*.

DETERMINATION OF SERUM MYOGLOBIN :

Serum myoglobin was estimated according to the method described by *(Tuengler et al., 1982)*.

PRINCIPLES OF THE METHOD :

In an immunochemical reaction, polystylene particles coated with anti-human myoglobin an antibody form agglutinates with myoglobin present in human serum. After mixing of reaction partners, the increasing turbidity is measured photometrically. Quantitative determination of the existing myoglobin concentration is accomplished by turbidimetric measurement of the maximum reaction velocity (Vmax).

REAGENTS:

materials provided :

Turbiquant myoglobin kits.

1x for 10 ml turbiquant myoglobin

1 x 10 ml Buffer for turbiquant myoglobin .

COMPOSITION :

Turbiquant myoglobin : is a lympholysate consisting of polystyrene particles coated with rabbit anti human myoglobin.

Buffer for turbiquant myoglobin : consists of a buffered sodium chloride solution containing detergents.

PREPARATION OF REAGENTS :

The lympholized turbiquant myoglobin reagent is suspended in 10 ml for turbiquant myoglobin. The contents of the 10 ml buffer bottle must be quantitatively transferred to the reagent bottle in this process.

SPECIMENS :

Serum samples stored for no more than 8 days at + 2 to + 8°C. Serum samples should have clotted completely and after centrifugation contain no particulate matter or traces of fibrin. Interference from icteric, hemolyzed and lipemic samples does not occur.

PROCEDURE :

1- allow the reagent to warm to room temperature .

2- Insert reagent bottle into the receptacle.

3- Pipette 50 μl of undiluted patient serum into a cuvette.

4- Insert the cuvette into the cuvette receptacle.

5- Pipette 500 μl of reagent into the cuvette when prompted to do so by the instrument .

6- Remove cuvette from the instrument when prompted to do so.

RESULTS :

The myoglobin determination result is automatically calculated by the turbitimer and printed in $\mu g/L$

SENSITIVITY AND MEASURING RANGE :

Turbiquant myoglobin kit is designed with measuring range of approximately 50 to 950 μg /litre using undiluted samples. The sensitivity is 50 μg/litre as defined by the lower limit of the measuring range.

SPECIFICITY :

The test is specific for myoglobin.

STATISTICAL ANALYSIS :

Statistical malysis of the results were carried out according to *(Fisher, 1946)* as follows :

1- Mean value $(x) = \frac{\Sigma X}{n}$

Where ΣX = sum of values.

n = Number of values .

2- Standard deviation (S.D) = $\sqrt{\frac{(\bar{X} - X)^2}{n - 1}}$

Where $(\bar{X}-X)^2$ is the sum of squares of the difference between each obseration (X) and the mean value of all observations.

3- "t" test for the significance of difference between two means, the following formula is used :

$$t = \frac{X_1 - X_2}{\sqrt{\frac{(S.D_1)^2}{n_1} \quad \frac{(S.D_2)2}{n_2}}}$$

Where :

X_1 = The mean value in samples 1

X_2 = The mean value in sample 2

n_1 = The number of cases in sample 1

n_2 = The number of cases in sample 2

SD_1 = Standard deviation in sample 1

SD2= Standard deviation in sample 2

SE = (standard Error) = $\frac{S.D}{\sqrt{n}}$

CORRELATION (r):

Sumple correlation is the relation between two variables. This relation is measured by the correlation coefficient *(Sperman, 1969)*:

$$r = 1 - \frac{\sum d^2}{n\ (n^2-1)}$$

n = number of values

d^2= summation of squares of the difference between two corresponding values.

It will have its value as + 1 for perfect positive correlation and − 1 for perfect negative correlation.

These tests were carried out using scientific calculator Casio (FX-140).

RESULTS

RESULTS

ANALYSIS OF RESULTS :

A) Clinical data :

The clinical data are summarised in appendix 1 & 2 They were 15 patients with acute myocardial infarction (11 males and 5 females) with age ranging from 41-72 years (mean 56.7±3.2) and compronise group I, which do not receive streptokinase. Group II comprised 15 patients (9 males & 62 Females) with age ranging from 50-62 years (Mean 59.8±8.9) and received I.V streptokinase. Group I was subdivided into subgroup Ia with acute anterior wall myocardial infarction compromising 8 males & 4 females & subgroup Ib comprising 2 males & 1 females, with inferior wall myocardial infarction. Similarly group II was subdivided into subgroup. IIa which compromised 6 males & 5 females with acute anterior wall myocardial infarction and subgroup IIb which compromised 3 males & 1 female with inferior wall myocardial infarction.

10 healthy persons were included in our study as control group with age ranging from 42 to 60 years and compromised group III.

B-SERUM MYOGLOBIN :

Comparative analysis of serum myoglobin level in studied groups showed :

a) group I (non streptokinase treated group) versus group III (control):

There was significant increase in serum myoglobin level of non streptokinase treated patients compared to control group, after 6 and 12 hours from the onset of symptoms ($P<0.01$) (Table 1). On the other hand, there was non significant change in serum myoglobin level after 24 hours from the onset of symptoms and up to the seventh day ($P>0.05$) (Table 1).

b) Group II (streptokinase treated group versus group III (control group):

There was significant increase of serum myoglobin level of streptokinase treated patients, compared to the control group, only after 6 hours from the onset of symptoms ($P<0.05$), on the other hand there was no significant change in serum myoglobin level after 12 hours and up to the seventh day from the onset of symptoms ($P>0.05$) Table (1).

c) group II (streptokinase treated) versus group I (non streptokinase treated):

There was significant increase in serum myoglobin level of streptokinase treated patients, compared to non streptokinase treated patients, only after 12 hours from the onset of symptoms ($P<0.05$). On the other hand, there was no significant change in serum myoglobin level of stroptokinase treated patients after 6 hours and from 24 hours up to the seventh day from the onset of symptoms (P>0.05) Table (2).

d) group Ia (Non streptokinase treated group with acute anterior myocardial infarction) versus group IIa (streptokinase treated group with anterior myocardial infarction); there was no significant change of serum myoglobin level of group Ia compared to group IIa (P>0.05) (Table 3).

e) group Ib (non streptokinase treated group with inferior myocardial infarction) versus group II b (streptokinase treated group with inferior myocardial infarction):

There was no significant change of serum myoglobin level of group II b compared to group Ib (P>0.05) table (4).

f) group Ia versus group Ib and group IIa versus group 2b: There was no significant change of serum myoglobin level of each of group Ia and IIa compared to group Ib and IIb respectively (Tables 5 and 6).

Serum creatine phosphokinase (CPK) and serum oxalacetic transaminase (SGOT):

There was no significant change in serum creatine phosphokinase or serum oxalacetic transaminase of non streptokinase treated group compared to streptokinase treated group (P>0.05 and P1>0.05) (Table 7).

C-CORRELATION STUDIES :

Pulse rate : there was weak correlation between serum myoglobin level after six hours from the onset of symptoms and pulse rate of both non streptokinase and streptokinase treated groups. (r=0.08 & 0.22, respectively),table (8) **Blood pressure:** there was weak correlation between serum myoglobin level, after six hours from the onset of symptoms and each of the systolic blood pressure and mean blood pressure of both the non streptokinase and streptokinase treated group (r= 0.08 & 0.o2, respectively), table (8). Also there was weak correlation between serum myoglobin level and age of each of non streptokinase treated and streptokinase treated group (r= 0.01 and 0.24 respectively), table (8).

Correlation between serum myoglobin level and creatine phosphokinase :

There was strong correlation between serum myoglobin level and serum creatine phosphokinase (CPK) level of both non streptokinase and streptokinase treated groups (r.0.82 & 0.89) respectively after 12 hours from the onset of symptoms table (9)

Correlation between serum myoglobin level and serum glutamic oxalacetic transaminase (SGOT) :

There was strong correlation between serum myoglobin level and serum oxalacetic transaminase (SGOT) level of both non streptokinase and streptokinase treated groups . (r 0.9 & 0.90, respectively), table (9).

D-ELECTROCARDIOGRAPHIC DATA :

1) Non streptokinase treated group : (Table 10)

Analysis of ECG findings of non streptokinase treated group revealed the following :

* Anterior myocardial infarction: cases (2, 3, 5, 6, 7, 9, 10, 11, 12 13,14,15) : evidenced by:

* S T segment elevation in leads v_2, v_3, v_4, v_5 & v_6 and pathological Q wave in leads v_1,v_2,v_3 (case 2) .

* S T segment elevation in leads v_1,v_2,v_3 and v_4 & pathological Q wave in lead v_1 & v_2, (case 3).

* S T segment elevation in leads v_1, v_2, v_3 v_4 v_5, and v_6 and - pathological Q wave in lead v_1,v_2,v_3 ,v_4 (case 5).

* ST segment elevation in leads v_1,v_2 and v_3 & pathological Q wave in leads v_2 ,v_3 v_4 (case 6).

* S T segment elevation in leads v_1,v_2,v_3 and v_4 and pathological Q wave in leads v_1,v_2,v_3 ,v_4 (case 9).

* S T segment elevation in leads v_1,v_2,v_3 and pathological Q wave in leads v_4 & v_5 R (case 10).

* S T segment elevation in leads v_1, v_2, v_3 and v_4 & pathological Q wave in leads v_1, v_2 and v_3 (case 11).

* S T segment elevation in leads v_1, v_2, v_3 and v_4 & pathological Q wave in leads v_1, v_2 , v_3 and v_4 (case 12).

* S T segment elevation in leads v_1, v_2, v_3 , v_4 & pathological Q wave in leads v_1, v_2 and v_3 (case 13).

* S T segment elevation in leads v_2, v_3 and v_4 & pathological Q wave in lead v_1, (case 14).

* S T segment elevation in leads v_2, v_3 and v_4 & pathological Q wave in lead v_1 (case 15).

Antero inferior myocardial infarction: Cases (1 and 7) evidenced by:

* ST segment elvation in leads II,III AVF, V_3, V_4, V_5 and V_6 and pathological Q wave in leads V_1, V_2, V_3 and V_4 (Case 1)

* ST segment elvation in leads II,III, AVF, V_2, V_3, V_4, V_5 and and pathological Q wave in leads V_1, V_2, V_3, V_5, V_6 (case 7)

Inferior myocardial infarction: Cases (8) evidenced by:

* ST segment elvation in leads II,III and AVF.

2- **STREPTOKINASE TREATED GROUP** :

a) **Before I.V streptokinase : (Table 11)**

Inferolateral myocardial infarction: ST segment elevation in leas II, III AVF, V5 and V6 - pathological Q wave in leads II, III AVF & V5 & V6 (case 1).

Inferior myocardial infarction: (case 4,5,8) evidenced by:

* S T segment elevation in leads II, III and AVF (case 4).
* S T segment elevation in leads II, III and AVF (case 5).
* S T segment elevation in leads III and AVF (case 8).
* Anteroinferior myocardial infarction (case 3): evidenced by:
* S T segment elevation in v_1, v_2 ,v_3, v_4, III, AVF and pathological Q wave in lead v_2 III and AVF .

Anterior myocardial infarction :

(case 2 , 6 , 7, 9, 10 , 12, 13 , 14) : evidenced by :

* S T segment elevation in leads v_1, v_2 ,v_3, v_4 and pathological Q wave in leads v_1, v_2 ,v_3, (case 2).

* S T segment elevation in leads v_1,v_2,v_3,v_4, and V_5 (case 6).

* S T segment elevation in leads v_1, v_2 and ,v_3, and pathological Q wave in leads v_1, v_2 ,v_3, (case 7).

* S T segment elevation in leads v_1, v_2 ,v_3, and v_4 & and pathological Q wave in leads v_1 and v_2 (case 9).

* S T segment elevation in leads v_1, v_2 ,v_3, and v_4 (case 10).

* S T segment elevation in leads v_1, v_2 and v_3, and pathological Q wave in leads v_1, v_2 ,v_3, and v_4(case 12).

* S T segment elevation in leads v_1, v_2 ,v_3, and v_4 and pathological Q wave in leads v_1, v_2 ,v_3 (case 13).
* S T segment elevation in leads v_1 and v_2 and pathological Q wave in leads v_2 and v_3 (case 14).

Anterolateral myocardial infarction :

evidenced by : S T segment elevation in lead I, AVL v_1,v_2 ,v_3, v_4, v_5, and pathological Q wave in leads v_3, (case 15).

b) After intravenous streptokinase : (Table 12)

* Revealed the same findings before intravenous streptokinase except :

* Decreased S T segment elevation in leads II,III and AVF (case 1)

* Return of S T segment to the isoelectric line in V_1 and decreased S T segment elevation in leads V_2,V_3 and V_4 (case 2).

* Decreased S T segment elevation in leads II,III, AVF, V_2, V_3, V_4 (case 3).

* Return of S T segment to the isoelectric line in lead AVF and decreased S T segment elevation in leads II and III. (case 4).

* Return of S T segment to the isoelectric line in leads II

and AVF and decreased S T segment elevation in lead III. (case 5).

* Return of S T segment to the isoelectric line in leads V_5 (case 6).

* Decreased S T segment elevation in lead V_1 (case 7).

* Decreased S T segment elevation in leads III and AVF (case 8).

* Decreased S T segment elevation in leads V_1,V_2, V_3 and V_4 and pathological Q wave in lead V_1,V_2 and V_3 (case 9).

* Return S T segment elevation to the iso electric line in leads V_1 and Decreased S T segment elevation in leads V_2, V_3 and V_4 and pathological Q wave in lead V_2 and V_3 (case 10).

* Pathological Q wave in leads V_2 and V_3(case 11).

* Pathological Q wave in lead V_4 (case 12).

* Pathological Q wave in leads $V_1$and V_4(case 13).

* Pathological Q wave in lead V_4 (case 14).

* Return of S-T segment to the isoelectric line in leads I and AVL (case 15).

Table (1): Comparative analysis of serum myoglobin findings of patients with acute myocardial infarction of non streptokinase treated group, streptokinase treated group and control group.

	Control Mean ± S.E. microgram /L	Non Streptokinase treated (mean±S.E.) microgram /L	Streptokinase treated (mean±S.E.) microgram /L
6 hours	70.8±7.83	486.93±95.43 $P < 0.05$	884.46±176.9 $P_1 < 0.05$
12 hours		377.06±105.89 $P < 0.05$	89.06±11.01 $P_1 > 0.05$
24 hours		182.4±101.5 $P > 0.05$	81.26±5.27 $P_1 > 0.05$
second day		96±24.91 $P > 0.05$	79.33±7.05 $P_1 > 0.05$
third day		83.46±5.16 $P > 0.05$	77.06±5.54 $P_1 > 0.05$
fourth day		82.93±4.94 $P > 0.05$	75.93±4.28 $P_1 > 0.05$
fifth day		80.26±4.99 $P > 0.05$	74.13±4.42 $P_1 > 0.05$
sixth day		78.2 ±6.20 $P > 0.05$	71.33±4.94 $P_1 > 0.05$
seventh day		74.93±4.83 $P > 0.05$	68.6±3.75 $P_1 > 0.05$

P : Significance of the difference between serum myoglobin level of patients with acute myocardial infarction of non streptokinase treated group and the control group.

P_1 : Significance of the difference between serum myoglobin level of streptokinase treated group and the control group.

Figure (1):

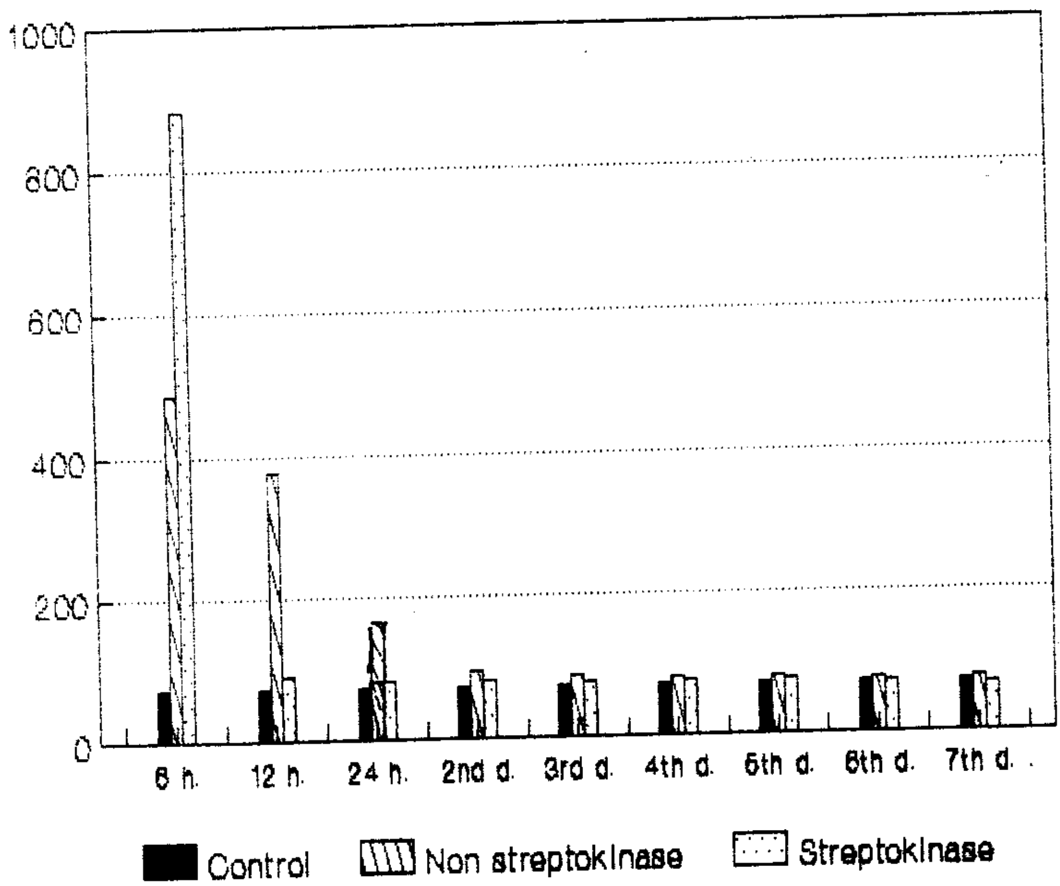

Fig. (1): Comparative analysis of serum myoglobin findings of patients with acute myocardial infarction of non streptokinase treated group, streptokinase treated group and control group.

Table (2): Comparative analysis of serum myoglobin findings of patients with acute myocardial infarction of non streptokinase treated group and streptokinase treated group.

	Non Streptokinase treated (mean±S.E.) microgram /L	Streptokinase treated (mean±S.E.) microgram /L
6 hours	486.93±95.43	884.46±176.9 $P > 0.05$
12 hours	377.06±105.89	89.06±11.01 $P < 0.05$
24 hours	182.4±121.5	81.26±5.27 $P > 0.05$
second day	96±24.91	79.33±7.05 $P > 0.05$
third day	83.46±5.16	77.06±5.54 $P > 0.05$
fourth day	82.93±4.94	75.93±4.28 $P > 0.05$
fifth day	80.26±4.99	74.13±4.42 $P > 0.05$
sixth day	78.2 ±6.20	71.33±4.94 $P > 0.05$
seventh day	74.93±4.83	68.6±3.75 $P > 0.05$

P : Significance of the difference between serum myoglobin level of patients with acute myocardial infarction of non streptokinase treated group and streptokinase treated group.

Figure (2):

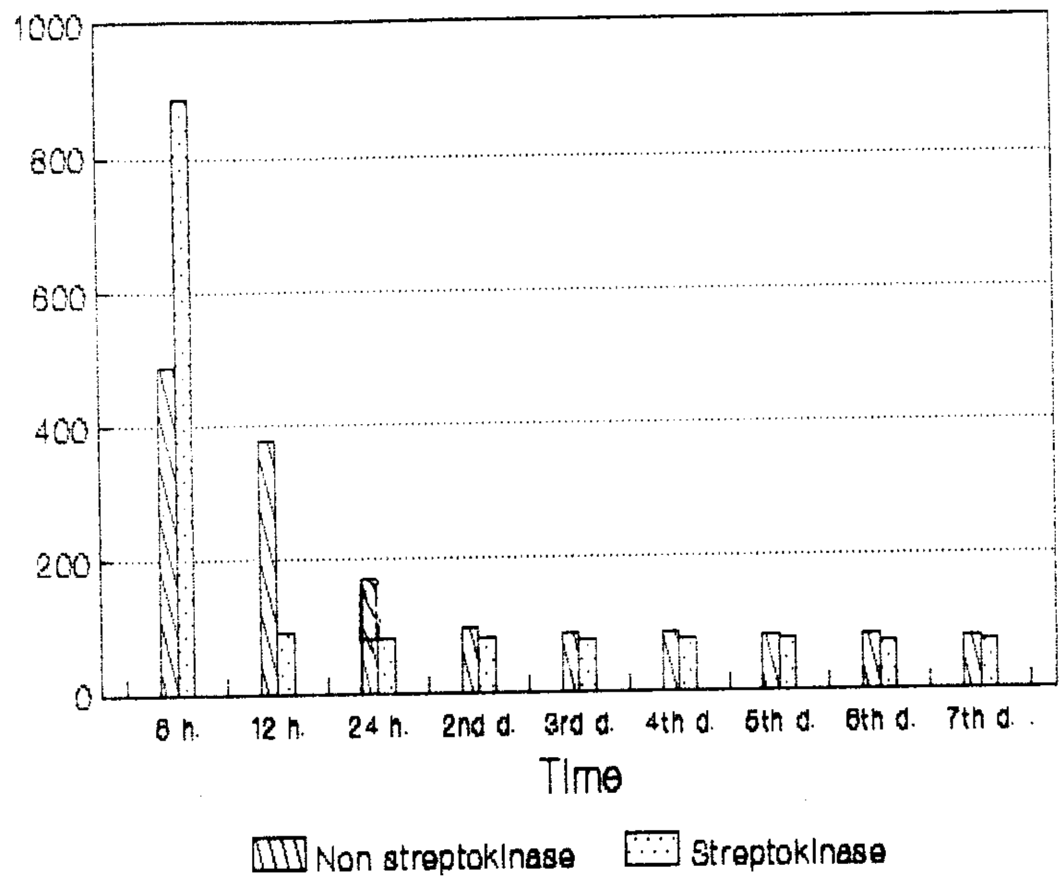

Fig. (2): Comparative analysis of serum myoglobin findings of patients with acute myocardial infarction of non streptokinase treated group and streptokinase treated group.

Table (3): Comparative analysis of serum myoglobin findings of patients with acute anterior myocardial infarction of non streptokinase treated group and patients with acute anterior myocardial infarction of streptokinase treated group.

	Non Streptokinase treated (mean±S.E.) microgram /L	Streptokinase treated (mean±S.E.) microgram /L
6 hours	481.16±106.56	862 ± 129.04 P < 0.05
12 hours	322.16±151.65	90 ± 10.7 P > 0.05
24 hours	173 ±104.64	81.6 ±5.58 P > 0.05
second day	98.5±27.24	80.55±8.09 P > 0.05
third day	83.4 ±5.61	78.33±4.69 P > 0.05
fourth day	82.91±5.42	77 ± 3.97 P > 0.05
fifth day	80.25±5.11	75.55±3.53 P > 0.05
sixth day	78.5 ±6.82	71.44±3.26 P > 0.05
seventh day	76 ± 4.04	68.11±4.12 P > 0.05

P : Significance of the difference between serum myoglobin level of non streptokinase treated group and streptokinase treated group.

Figure (3):

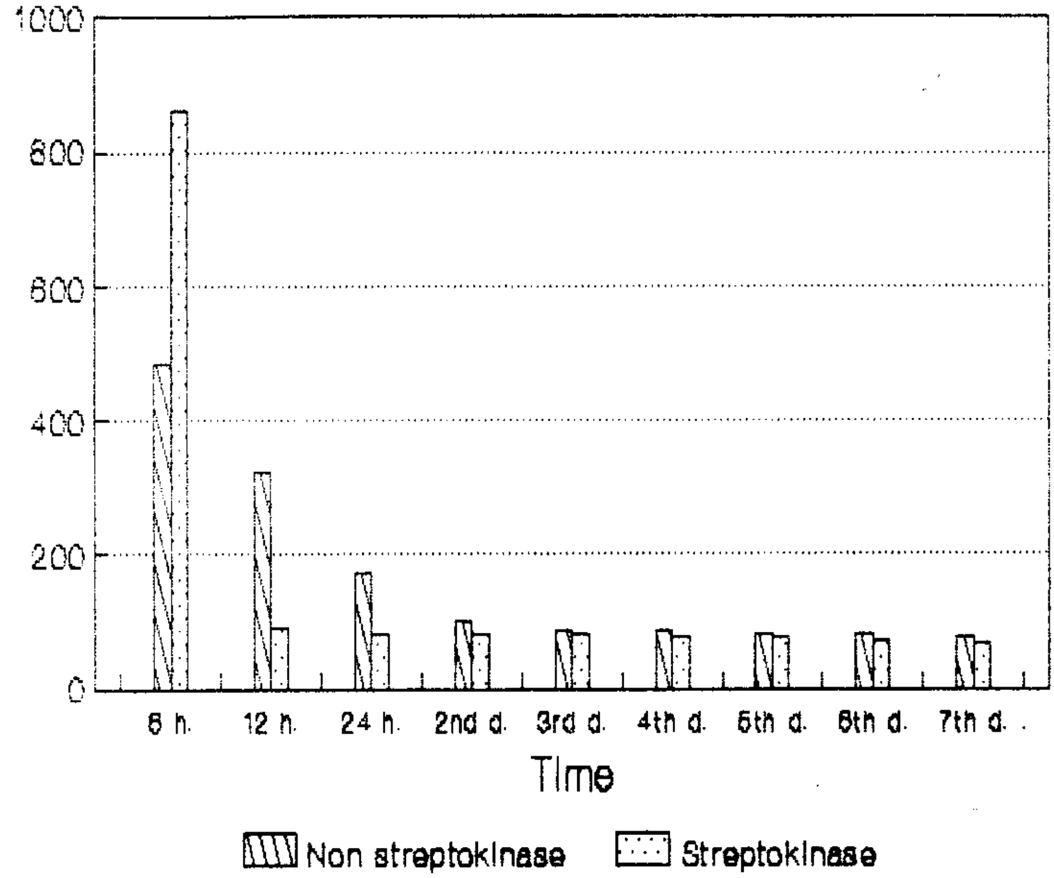

Fig. (3): Comparative analysis of serum myoglobin findings of patients with acute anterior myocardial infarction of non streptokinase treated group and patients with acute anterior myocardial infarction of streptokinase treated group.

Table (4): Comparative analysis of serum myoglobin findings of patients with acute inferior myocardial infarction of non streptokinase treated group and patients with acute inferior myocardial infarction of streptokinase treated group.

	Non Streptokinase treated (mean±S.E.) microgram /L	Streptokinase treated (mean±S.E.) microgram /L
6 hours	510 ±57.15	755.75±261.75 $P > 0.05$
12 hours	403.33±20.54	86.5 ± 11.36 $P < 0.01$
24 hours	220 ±74.83	82 ± 2.9 $P < 0.01$
second day	87 ±2.16	75.75± 4.6 $P > 0.05$
third day	83.66±2.62	73.5 ± 4.5 $P > 0.05$
fourth day	83 ± 2.16	73.5 ± 2.52 $P > 0.05$
fifth day	80.33±4.94	70.5 ±1.6 $P > 0.05$
sixth day	77 ±2.16	69.2 ± 2.5 $P > 0.05$
seventh day	70.66± 5.43	69 ± 0.7 $P > 0.05$

P : Significance of the difference between serum myoglobin level of non streptokinase treated group and streptokinase treated group.

(4):

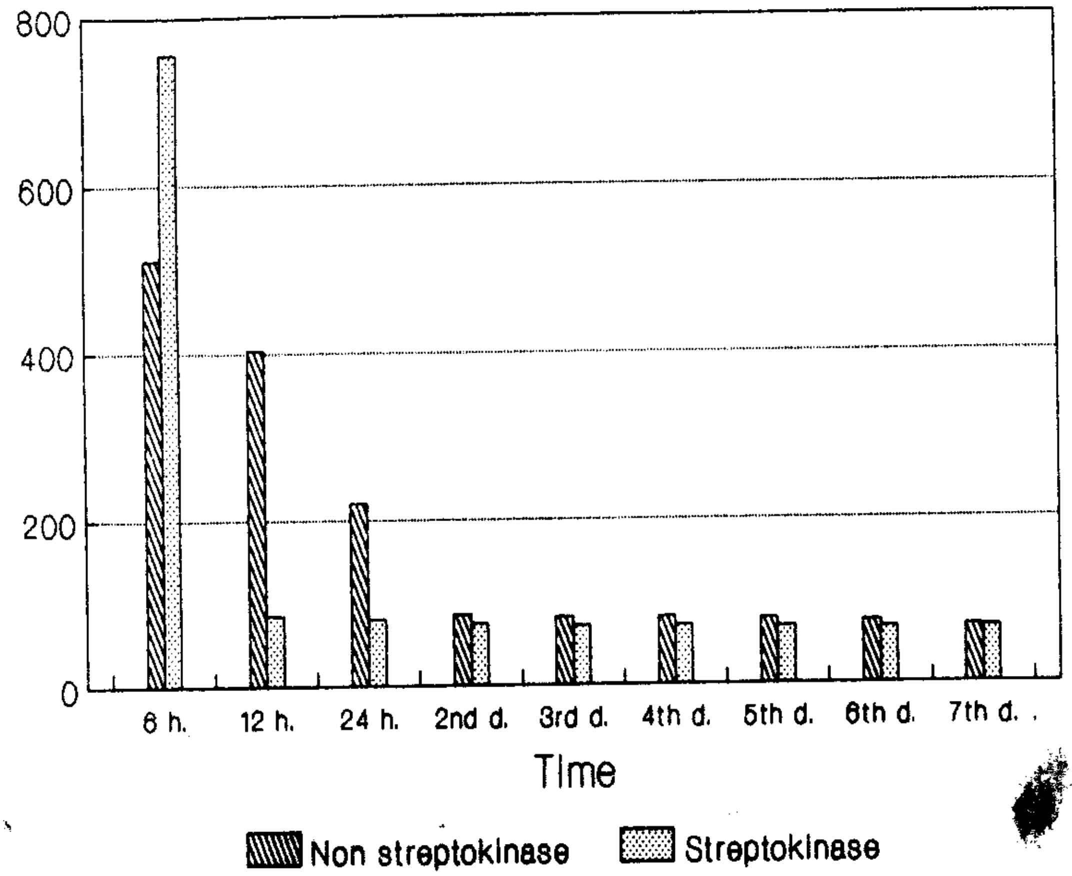

Fig. (4) : Comparative analysis of serum myoglobin findings of patients with acute inferior myocardial infarction of non streptokinase treated group and patients with acute inferior myocardial infarction of streptokinase treated group.

Table (5): Comparative analysis of serum myoglobin findings of patients with acute inferior myocardial infarction and patients with acute anterior myocardial infarction of non streptokinase treated group.

	Inferior myocardial infaraction (Mean ± SE.) microgram/L	Anterior myocardial infaraction (Mean ± SE.) microgram/L
6 hours	510 ±57.15	481.16±106.56 $P > 0.05$
12 hours	403.33±20.54	322.16±151.65 $P > 0.05$
24 hours	220 ±74.83	173 ±104.64 $P > 0.05$
second day	87 ±2.16	98.5 ±27.24 $P > 0.05$
third day	83.66±2.62	83.4 ±5.61 $P > 0.05$
fourth day	83 ± 2.16	82.91±5.42 $P > 0.05$
fifth day	80.33±4.94	80.25±5.11 $P > 0.05$
sixth day	77 ±2.16	78.5 ±6.83 $P > 0.05$
seventh day	70.66± 5.43	76 ±4.04 $P > 0.05$

P : Significance of the difference between serum myoglobin level of patients with acute inferior myocardial infarction and acute anterior myocardial infarction.

Figure (5):

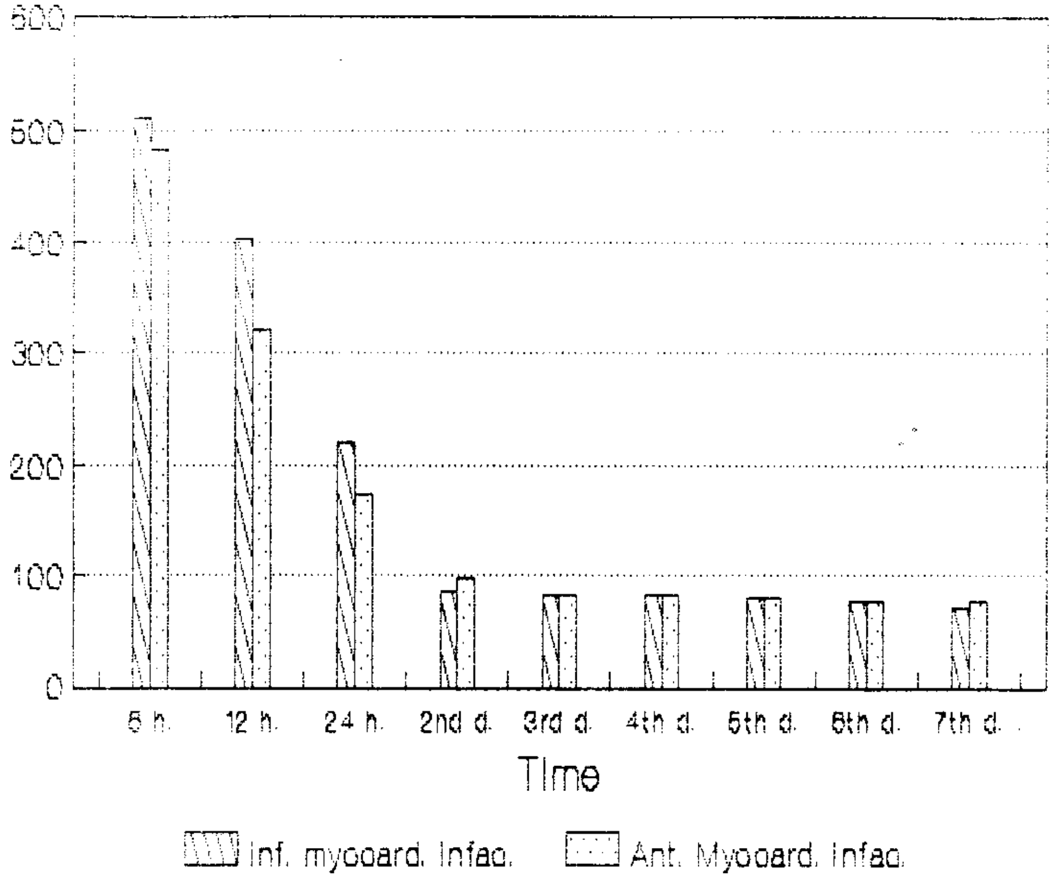

Fig. (5): Comparative analysis of serum myoglobin findings of patients with acute inferior myocardial infarction and patients with acute anterior myocardial infarction of non streptokinase treated group.

Table (6): Comparative analysis of serum myoglobin findings of patients with acute inferior myocardial infarction and patients with acute anterior myocardial infarction of streptokinase treated group.

	Inferior myocardial infaraction (Mean ± SE.) microgram/L	Anterior myocardial infaraction (Mean ± SE.) microgram/L
6 hours	795.75±261.3	862.18±129 P > 0.05
12 hours	86.5±11.36	174.33±72.31 P > 0.05
24 hours	82 ±2.9	90.88±11.52 P > 0.05
second day	75.75±4.6	80.55±8.09 P > 0.05
third day	73.5±4.5	78.33±4.69 P > 0.05
fourth day	73.5±2.5	77 ±3.97 P > 0.05
fifth day	70.5±1.6	75.55±3.53 P > 0.05
sixth day	69.2±2.5	71.44±3.26 P > 0.05
seventh day	69±0.7	68 ±4.12 P > 0.05

P : Significance of the difference between serum myoglobin level of patients with acute inferior myocardial infarction and patients with acute anterior myocardial infarction.

(6):

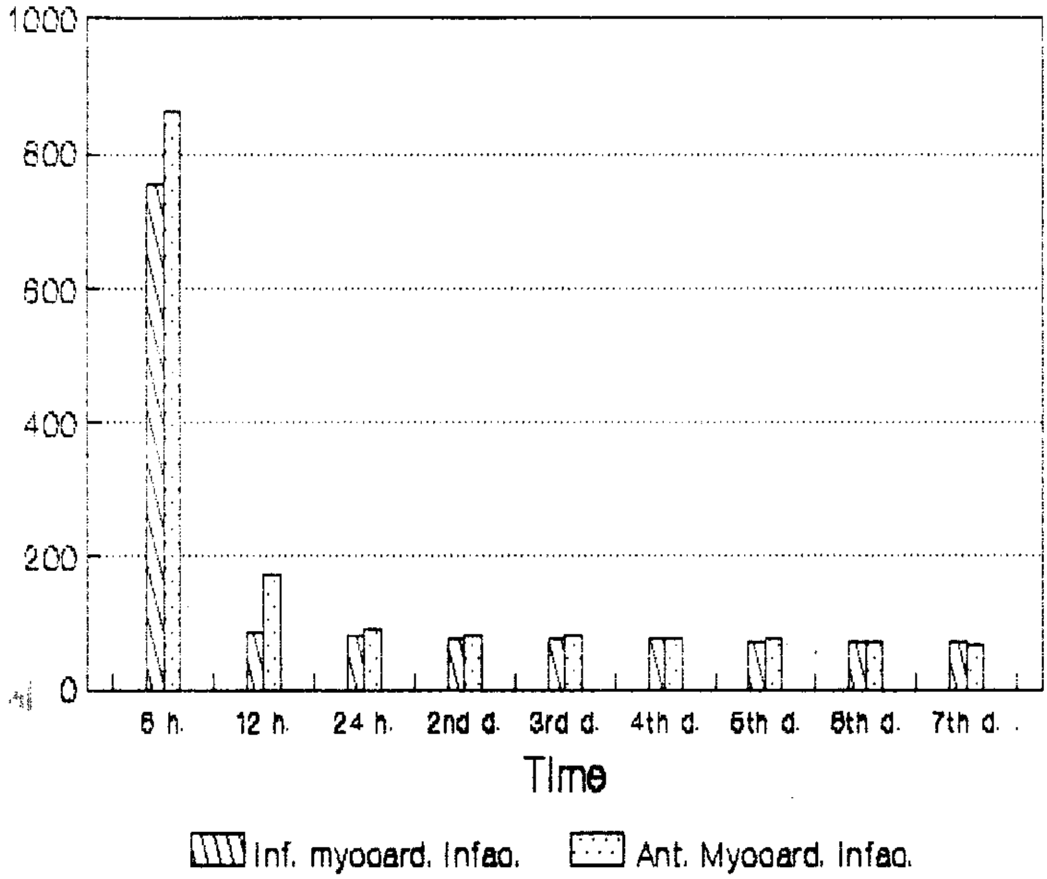

Fig. (6): Comparative analysis of serum myoglobin findings of patients with acute inferior myocardial infarction and patients with acute anterior myocardial infarction of streptokinase treated group.

Table (7): Comparative analysis of each of serum creatine phosphokinase (CPK) findings and serum oxalacetie transaminase (SGOT) findings of patients with acute myocardial infarction of non streptokinase treated group and streptokinase treated group .

	Non Streptokinase treated group (mean±S.E. Unit /L)	Streptokinase treated group (mean±S.E. Unit /L)
Serum Creatine phosphokinase	961.86±315.04	1059.93±649.60 $P > 0.05$
Serum oxalacetic transaminase	148.6±43.16	157.23±55 $P_1 > 0.05$

P : Significance of the difference between serum creatine phosphokinase level of non streptokinase treated group and streptokinase treated group.

P_1 : S ignificance of the difference between serum oxalacetic transaminase level of non streptokinase treated group and streptokinase treated group.

Table (8): Correlation between serum myoglobin level and some clinical data (age, pulse and blood pressure) of patients with acute myocardial infarction of non streptokinase treated group and streptokinase treated group .

	Non Streptakinase treated group	Streptakinase treated group
Age	r = 0.01	r = 0.24
Pulse	r = 0.08	r = 0.22
Blood pressure	r = 0.08	r = 0.02

Correlation (r): simple correlation is the relation between the two variables.

Table (9): Correlation between serum myoglobin level and each of serum creatine phasphokinase (CPK) level and serum oxalacetic transaminase (SGOT) level of non streptokinase and streptokinase treated group

	Non Streptakinase treated group	Streptakinase treated group
Serum creatine phosphokinase	r = 0.82	r = 0.89
Serum oxalacetic transaminase	r = 0.9	r = 0.9

Table (10): Electrocardiographic and radiologic data of non sterptokinase treated group.

No.	Heart rate	P.R. interval	Q R S axis	Q R S duration	Pathological Q wave	S - T Elevation	S - T Segment depression	T wave inversion	Arrhythmia	X ray chest, signs of pulmonary congestion
1	75	0.16	+ 75	0.16	V_1,V_2,V_3,V_4	II,III,AVF, V_3,V_4,V_5,V_6	V_1	AVR, V_1	-	-
2	100	0.12	+ 75	0.08	V_1,V_2,V_3	V_2,V_3,V_4,V_5,V_6	-	-	-	+
3	100	0.16	+ 15	0.08	V_1,V_2	V_1,V_2,V_3,V_4	-	AVR, V_4	-	+
4	75	0.20	- 15	0.08	——	V_4,V_5,V_6 II,III,AVF	-	AVR, AVL	-	-
5	100	0.16	- 60	0.08	V_1,V_2,V_3,V_4	V_1,V_2,V_3,V_4,V_5,V_6	-	AVR	-	+
6	60	0.12	- 30	0.08	V_2,V_3,V_4	V_1,V_2,V_3	-	I,AVL	-	-
7	72	0.20	+ 30	0.08	V_1,V_2,V_3,V_4,V_5,V_6	II,III,AVF V_2,V_3,V_4,V_5	-	AVR,V_1	-	-
8	85	0.16	+ 45	0.08	——	II,III,AVF	V_2,V_3,V_4,V_5,V_6	V_2,V_6	-	-
9	80	0.12	- 60	0.08	V_1,V_2,V_3,V_4	V_1,V_2,V_3,V_4	-	AVL,II	-	-
10	75	0.16	- 60	0.08	V_4R, V_5R	V_1,V_2,V_3	V_5	AVR,AVL	-	+
11	100	0.12	+ 75	0.08	V_1,V_2,V_3	V_1,V_2,V_3,V_4	-	AVR	-	-
12	100	0.16	+ 60	0.08	V_1,V_2,V_3,V_4	V_1,V_2,V_3,V_4	-	AVR,I	-	-
13	100	0.16	- 60	0.08	V_1,V_2,V_3	V_1,V_2,V_3,V_4	-	AVR	-	-
14	85	0.12	- 30	0.08	V_1	V_2,V_3,V_4	-	I,III,AVR AVL,V5	-	-
15	85	0.12	- 30	0.08	V_1	V_2,V_3,V_4	-	I,III,AVR AVL,V5	-	-

Table (11): Electrocardiographic and radiologic data of sterptokinase treated group before I.V. streptokinase.

No.	Heart rate	P-R. interval	Q R S axis	Q R S duration	Pathological Q wave	S - T Elevation	S - T Segment depression	T wave inversion	Arrhythmia	X ray chest, signs of pulmonary congesti
1	78	0.20	+ 15	0.08	II,III,AVF,v_5,v_6	II,III, AVF,v_5,v_6	AVL,v_1	AVR,AVL,v_4,v_5,v_6	–	–
2	100	0.20	– 45	0.08	v_1,v_2,v_3	v_1,v_2,v_3,v_4	II	AVR,AVL,I	–	–
3	75	0.16	0	0.08	v_2.III,AVF	V_1,V_2,V_3,V_4 II, III, AVF	–	v_2,v_3,v_4	–	–
4	56	0.16	+ 30	0.08	–	II,III,AVF	v_2,v_3,v_4,v_5	I,AVR,v_3,v_4,v_5	–	–
5	78	0.16	+ 30	0.07	–	II,III,AVF	v_1,v_2,v_3,v_4	AVR	–	–
6	75	0.12	+ 75	0.08	–	v_1,v_2,v_3,v_4,v_5		AVR	–	–
7	100	0.20	– 60	0.08	v_1,v_2,v_3	v_1,v_2,v_3	–	I,AVL	–	–
8	100	0.12	+ 15	0.08	–	III,AVF	v_1,v_2,v_3,v_4,v_5,v_6	–	–	–
9	120	0.16	+ 60	0.08	v_1,v_2	v_1,v_2,v_3,v_4	II,III	AVR	–	–
10	75	0.16	+ 10	0.06	–	v_1,v_2,v_3,v_4	–	AVR	–	–
11	75	0.16	+ 10	0.08	v_1	v_1,v_2,v_3	–	III,AVR	–	–
12	100	0.16	– 10	0.07	v_1,v_2,v_3,v_4	v_1,v_2,v_3,v_4	–	I,AVR,v_4,v_5	–	–
13	110	0.12	+ 15	0.07	v_1,v_2,v_3	v_1,v_2	–	I,AVR,AVL	–	–
14	100	0.12	– 10	0.08	v_2,v_3	v_1,v_2	–	I,AVL	–	–
15	75	0.16	+ 10	0.08	v_3	I,AVL,v_1,v_2,v_3,v_4,v_5	–	AVR	–	–

Table (12): Electrocarodiographic and radiologic data of strerptokinase treated group after I.V. streptokinase.

No.	Heart rate	PR. internal	Q R S axis	Q R S duration	Pathological Q wave	S-T segment Elevation	S-T segment depression	T wave inversion	Arrhythmia	X ray chest, signs of pulmonary congestio
1	100	0.16	+ 15	0.08	V_3, V_4, V_5, V_6	II, III, AVF, V_5, V_6	-	I, AVL, V_3, V_4, V_5, V_6	-	+
2	79	0.20	- 60	0.08	V_1, V_2, V_3	V_2, V_3, V_4	AVL		-	+
3	75	0.12	+ 15	0.08	V_2	II, III, AVF, V_2, V_3, V_4	-	AVR	-	-
4	78	0.16	- 10	0.08	-	II, III,	V_4, V_5	II, III, AVF	-	-
5	78	0.16	- 10	0.07	-	III	V_2, V_3, V_4	AVR	-	-
6	75	0.16	+ 60	0.08	-	V_2, V_3, V_4, V_5	-	AVR	-	-
7	86	0.16	- 60	0.08	V_1, V_2, V_3	V_1, V_2, V_3	-	AVL, V_3, V_4, V_5, V_6	-	-
8	100	0.16	+ 30	0.08	-		$V_1, V_2, V_3, V_4, V_5,$	-	-	-
9	300	0.16	+ 85	0.08	V_1, V_2, V_3	V_1, V_2, V_3, V_4	-	AVR	-	-
10	100	0.16	+ 30	0.08	V_2, V_3	V_2, V_3, V_4	-	AVR, V_4, V_5, V_6	-	-
11	100	0.12	+ 10	0.08	V_1, V_2, V_3	V_2, V_3	V_4, V_5, V_6	III, AVR, V_1	-	-
12	100	0.12	- 5	0.08	V_1, V_2, V_3	V_1, V_2, V_3	-	I, AVR, V_2, V_3	-	-
13	100	0.12	+ 15	0.08	V_1, V_2, V_3, V_4	V_2, V_3	-	I, AVR, AVL	-	-
14	75	0.12	+ 15	0.08	V_2, V_3, V_4	V_1, V_2	-	I, AVL, V_3, V_4, V_5, V_6	-	-
15	120	0.12	- 60	0.08	V_3	$V_1, V_2, V_3, V_4, V_5,$ 1 & AVL	-	I, AVR	-	-

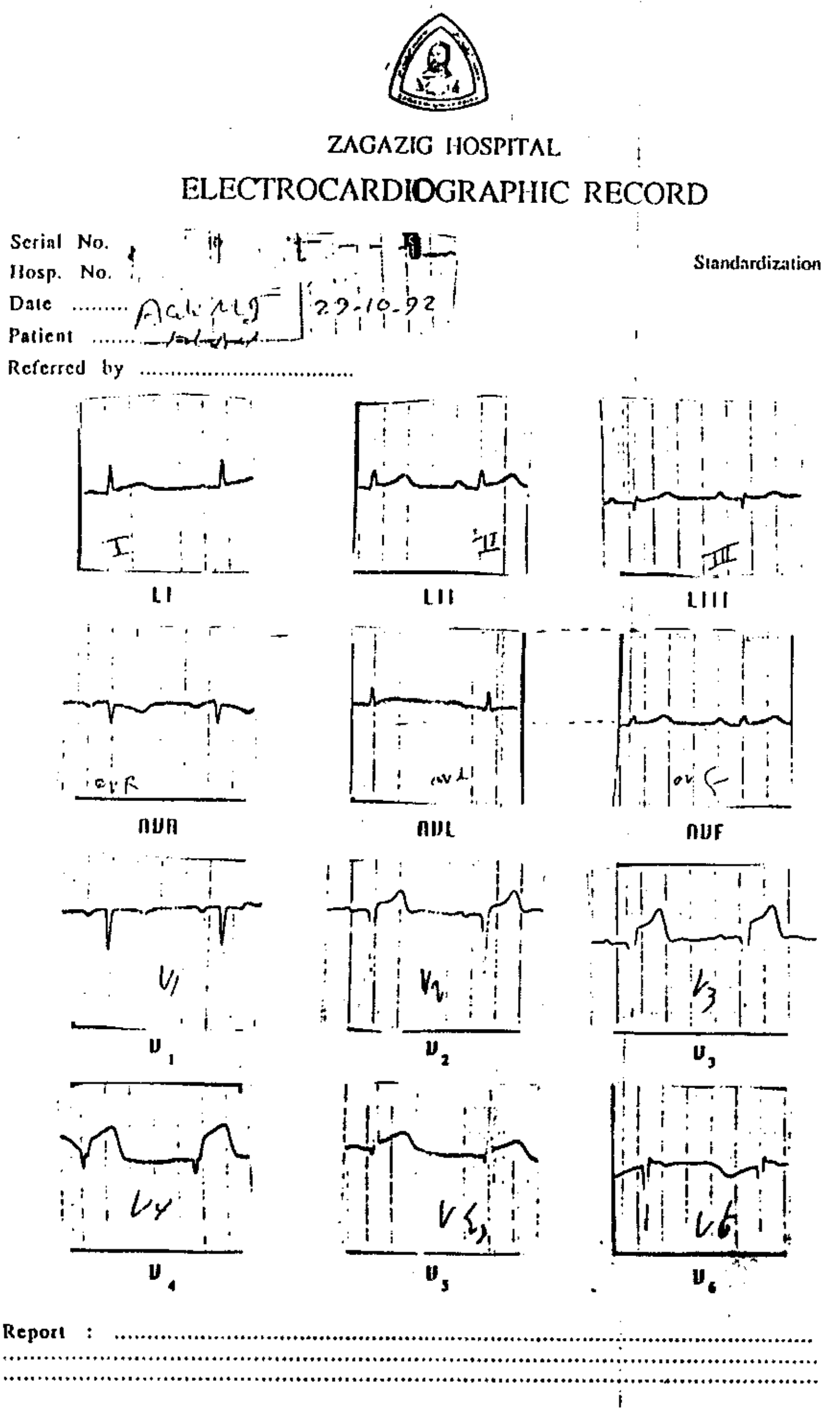

Fig. (7) : ECG tracing of the case No (7), non streptokinae treated, showing acute antero-inferior myocardial infarction with pathological Q wave in leads v_1, v_2, v_3, v_4 v_5, v_6 and ST segment elevation in leads II, III, AVF, v_2, v_3, v_4 and v_5.

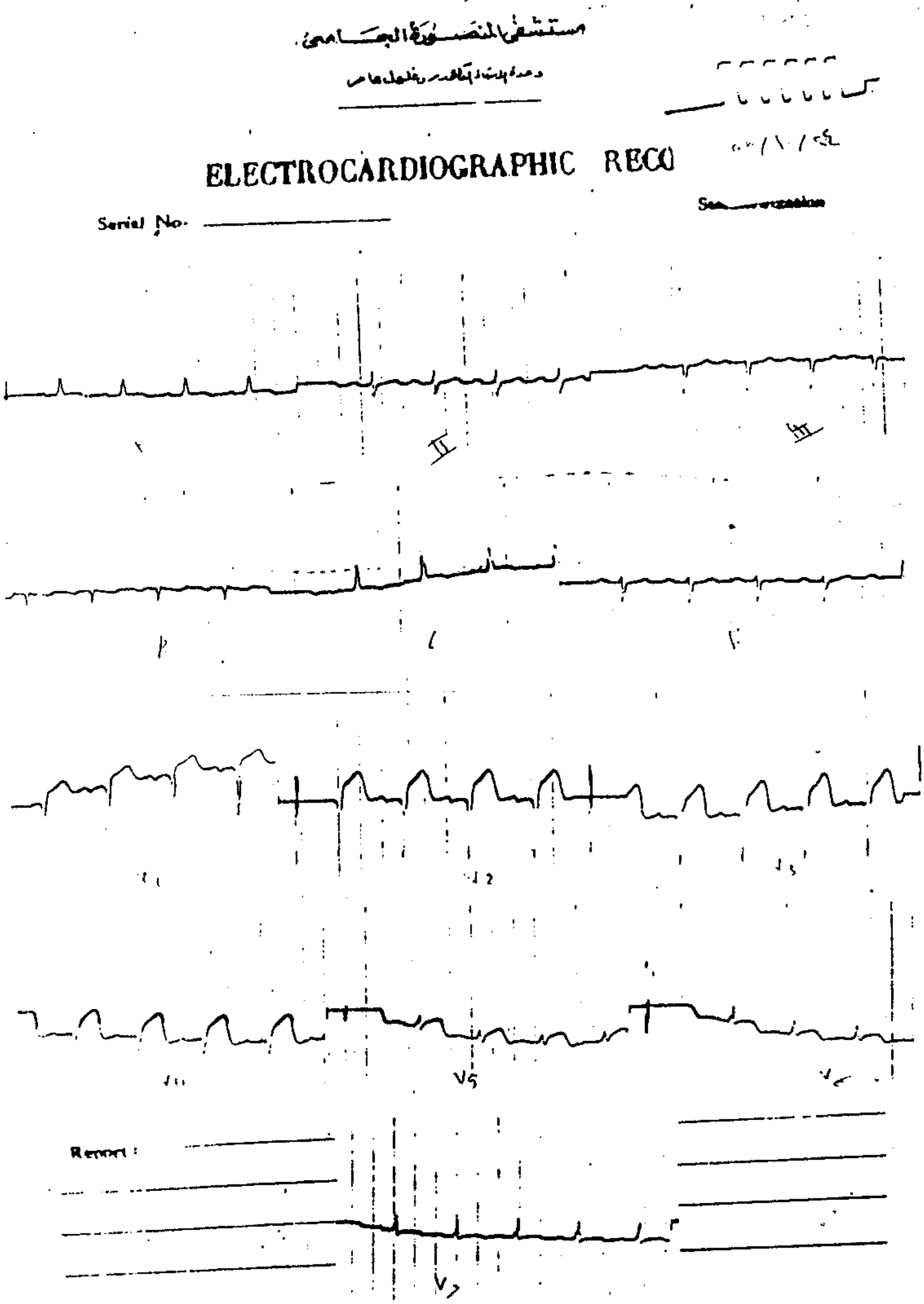

Fig. (8) : ECG tracing of the case NO (12), non streptokinase treated, showing acute anterior myocardial infarction with pathological Q wave in lead v_1, v_2, v_3, and v_4 and ST segment elevation in leads v_1, v_2, v_3 and v_4.

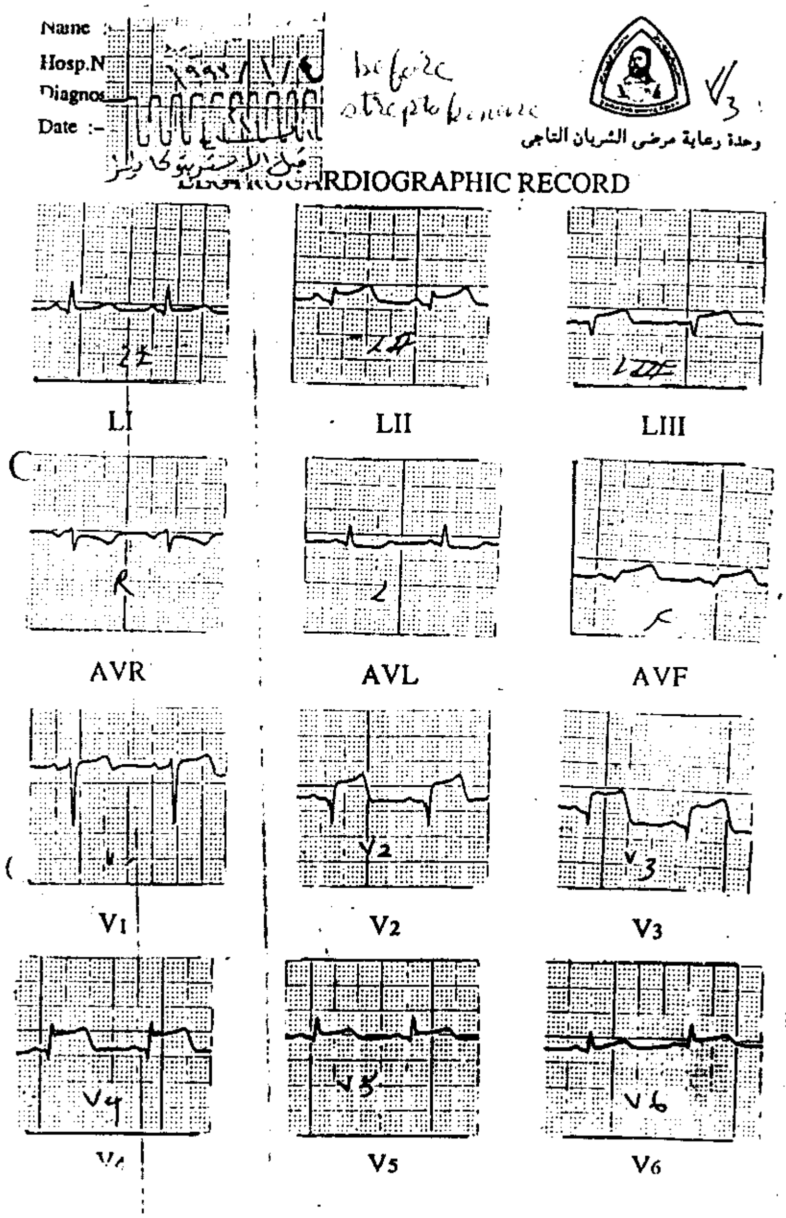

Fig. (9): ECG tracing of the case No (3) before I.V. streptokinase showing acute Antero - inferior myocardial infarction with pathological Q wave in leads III, AVF and v_2 and ST segment elevation in leads. II, III, AVF, v_1, v_2, v_3, and v_4.

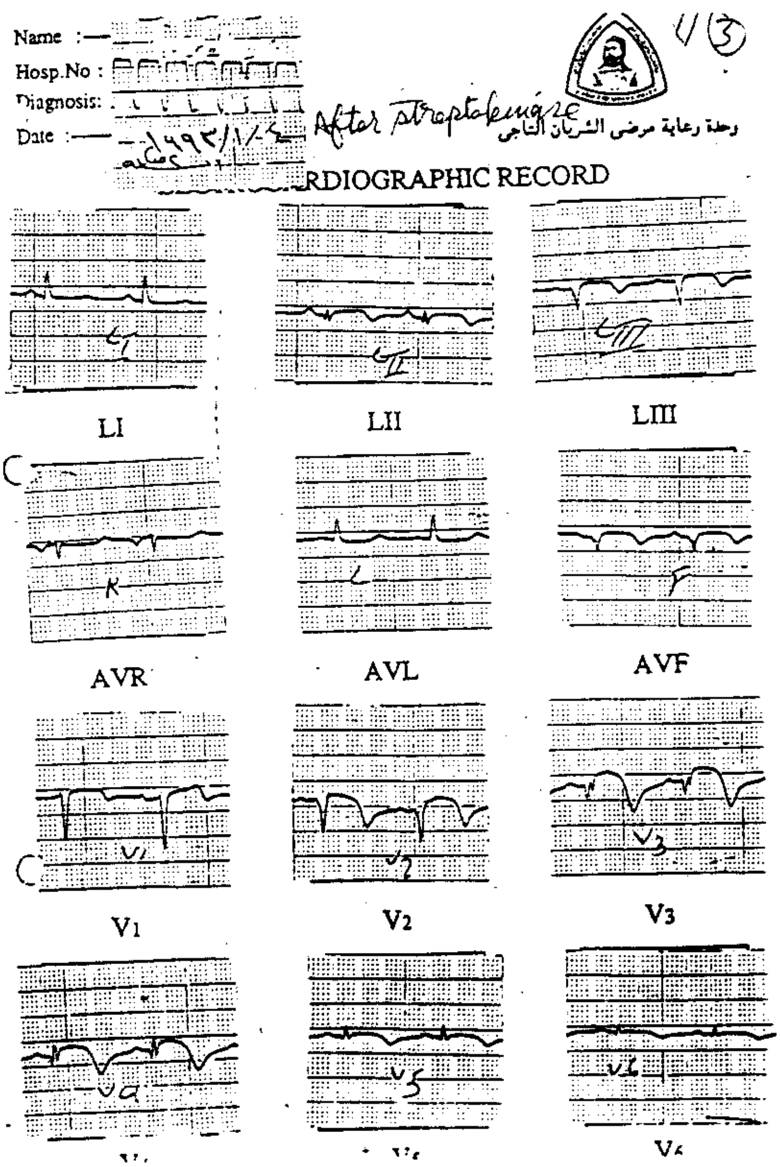

Fig. (10): ECG tracing of the case No (3) after I.V. streptokinase showing acute antero-inferior myocardial infarction with pathological Q wave in lead V_2 and decreased ST segment elevation in leads II, III , AVF , V_2, V_3, and V_4.

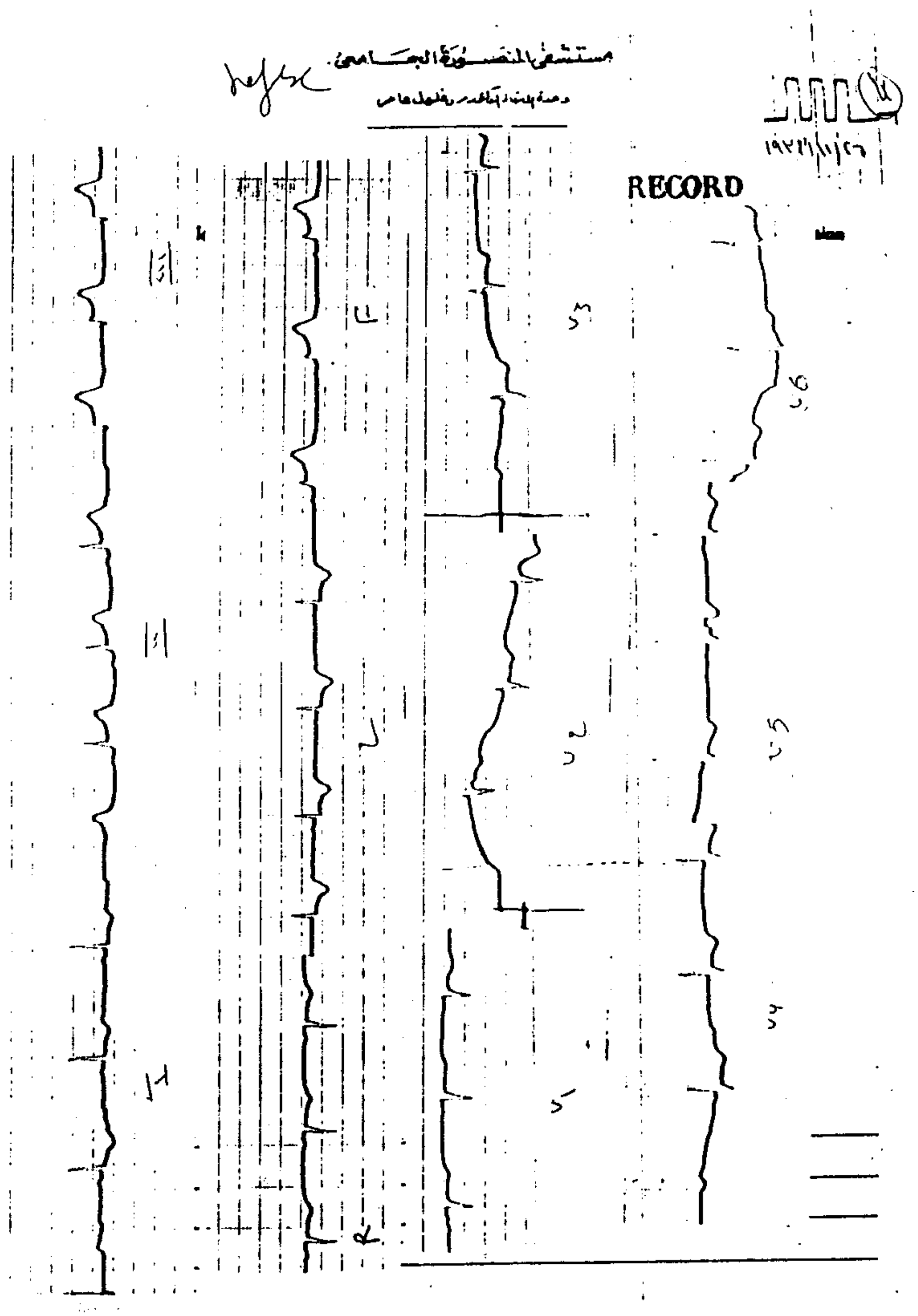

Fig. (11): ECG tracing of the case No (4) before I.V. streptokinase showing acute inferior myocardial infarction with ST segment elevation in leads II,IIIand AVF.

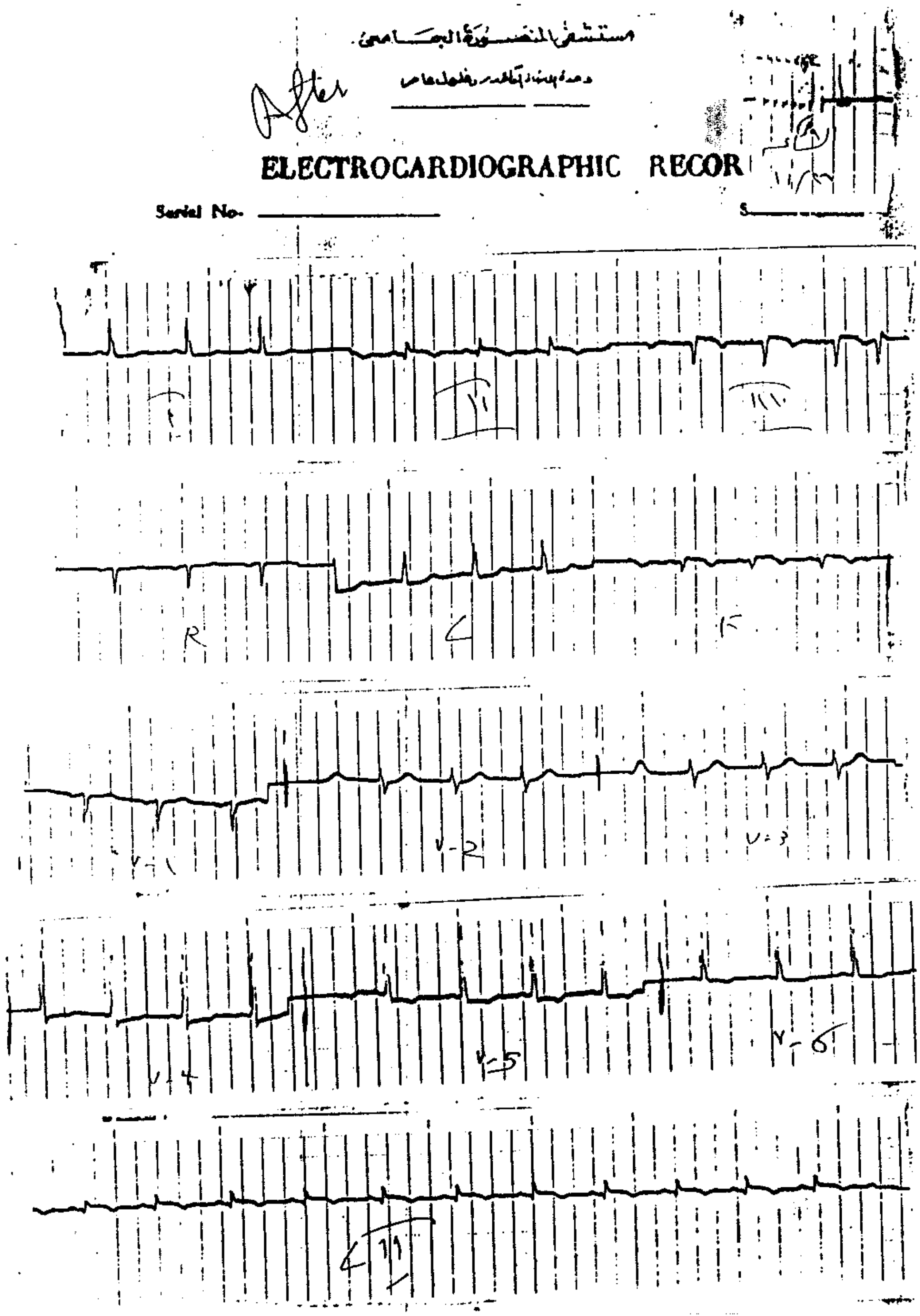

Fig. (12): ECG tracing of the case No (4) after I.V. streptokinase showing acute inferior myocardial infarction with decreased ST segment elevation in leads II, and III

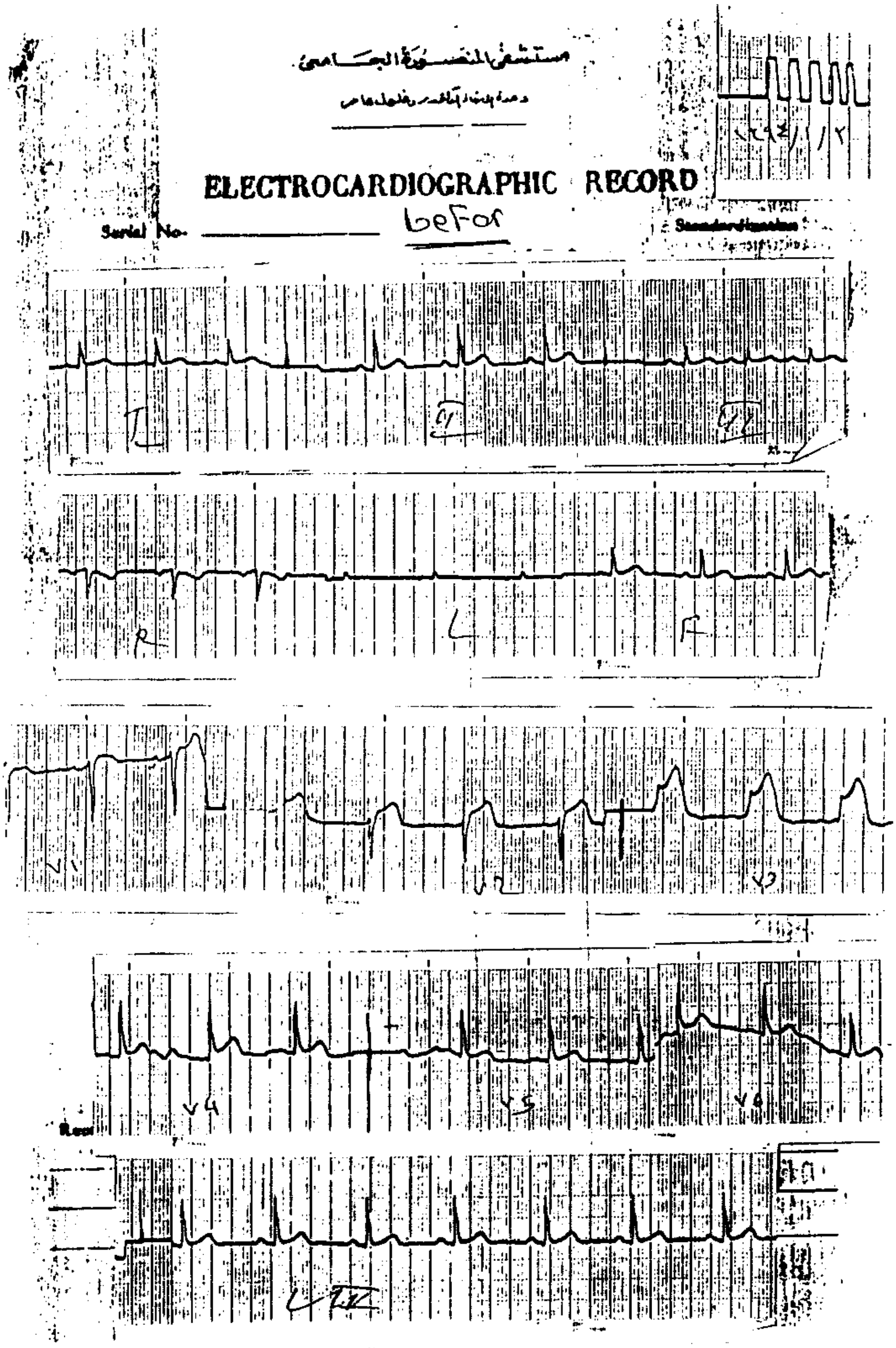

Fig. (13): ECG tracing of the case No. (10) before I.V streptokinase showing acute anterior myocardial infarction with ST segment elevation in leads v_1 ,v_2 $v_3$and v_4.

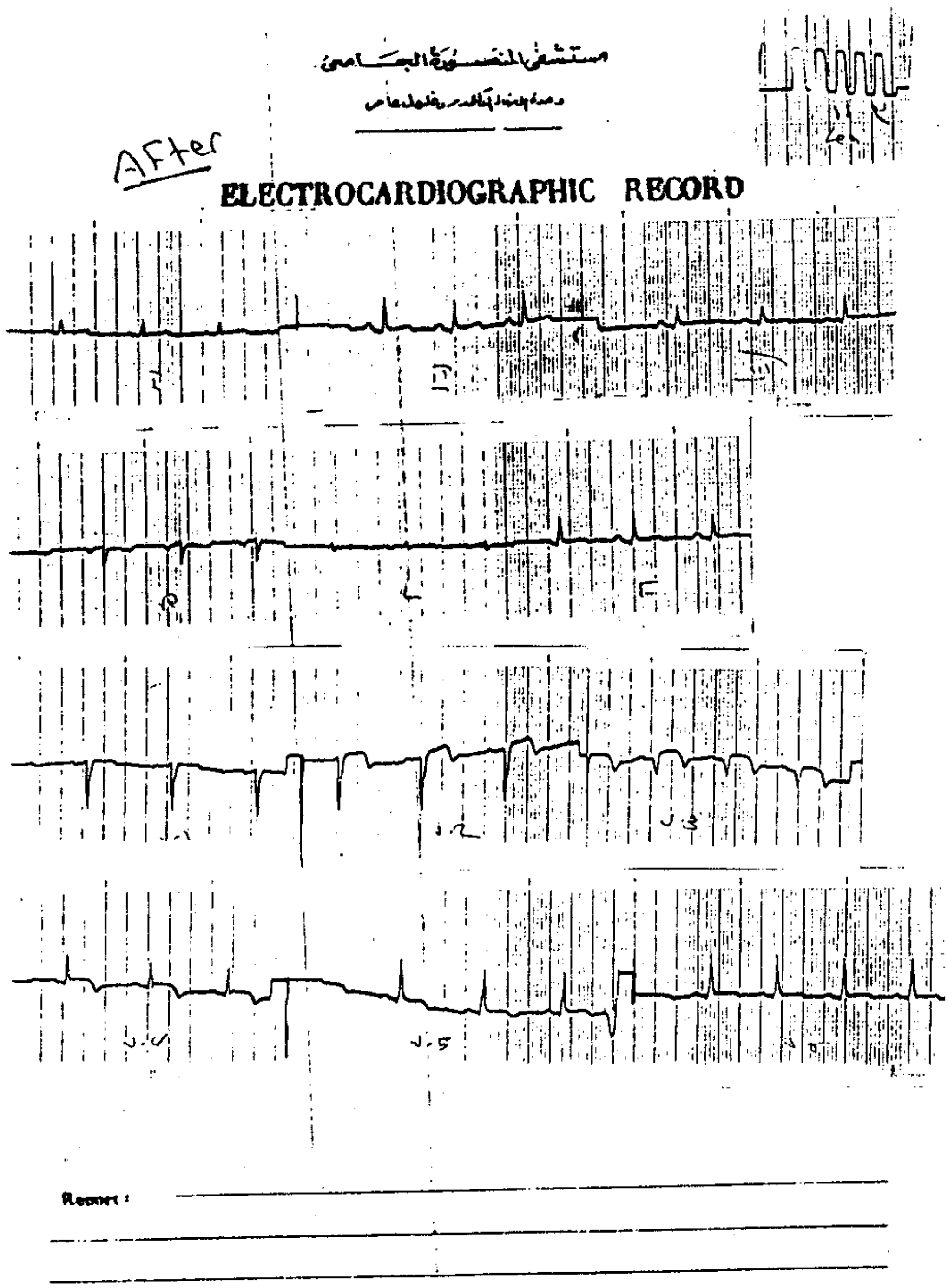

Fig. (14): ECG tracing of the case No (10) after I.V. streptokinase showing acute anterior myocardial infarction with pathological Q wave in leads V_2, and V_3 and decreased ST segment elevation in leads V_2, V_3 and V_4 and return of S T segment to iso electric line in lead V_1.

DISCUSSION

DISCUSSION

Several studies have compared serum creatine kinase and creatine kinase MB with myoglobin during the early phase of myocardial infarction. In general, these studies have found that serum myoglobin has a higher diagnostic sensitivity than creatine kinase and its isoenzymes (*Sylven et al., 1978*). Also it has been reported that myoglobin was a strong independent predictor of myocardial infarction in patients with symptoms of short duration (*Magnus et al., 1989*). Serum concentrations of myoglobin above the normal range have been found as early as one hour after myocardial infarction with peak activity in the range of 4-12 hours (*Stone and Willerson, 1983*). In particular 50% of patients with initially negative electrocardiograms and myocardial infarction were correctly diagnosed on the basis of their serum myoglobin (*Magnus et al., 1989*). Also myoglobin exhibits the most rapid concentration increase of all markers evaluated (*Markus et al., 1993*).

With increased use of thrombolytic therapy in the course of acute myocardial infarction there is increasing need to determine in individual patients whether reperfusion has been achieved. Several criteria have been suggested to allow non invasive determination of vessel patency after attempted thrombolysis : relief of chest discomfort, occurrence of reperfusion arrhythmiams, normalization of ST segment elevation *(Ganz et al., 1984)* and an early rapid rise *(Lewis et al., 1987)* and early peak in creatine kinase activity *(Shell et al., 1983 & Blanke et al., 1984)*. Although the first three parameters occur commonly after successful thrombolytic therapy, each parameter is relatively insensitive when considered seperately *(Golderb et al., 1983 & Rentrob et al., 1981)*. Although the predictive value is 100% if all the three findings are present, this occur only in less than 15% of reperfused patients *(Kircher et al., 1987)*. With regard to creatine kinase curves, successful reperfusion is associated with peak creatine kinase activity at 13 ± 6 hours compared with 22 ± 4 hours in patients in whom thrombolysis was unsuccessful *(Anderson et al., 1983)*. However, the large coefficients of variation (20-50%) indicate considerable variability and overlap among individual patients in these two groups. In addition to the large scatter and overlap of peak times, the need to collect frequent samples to determine the time of peak activity further limits this approach. The need to develop a non invasive marker of reperfusion is important in view of the large number of patients in whom intravenous thrombolysis is now likely to be used. Determination of blood myoglobin level early in the course of attempted coronary reperfusion is a relatively simple test that appears both sensitive and specific in providing information about the patency of the infarct related artery *(Avery et al., 1988)*. Also, it has been suggested that the infarct size could be estimated accurately within approximately 4 hours of reperfusion after thrombolytic therapy by calculating total myoglobin release from the injured myocardium into 1 ml of blood *(Tusminori et al., 1993)*.

The aim of our study is examination of the usefulness of blood myoglobin level determination for diagnosis of acute myocardial infarction with and without streptokinase therapy.

From table (1) we found a significant increase of serum myoglobin level in all patients of both non streptokinase treated and streptokinase treated groups. Our results are in agreement with the results of other studies. *Uji et al (1992)* reported that sera taken from 21 patients with definite acute myocardial infarction showed increased serum myoglobin levels (100-1200 ng/ml) upon admission and within 6 hours, using an immunoturbidimetric quantitation for serum myoglobin. Also, acute myocardial infarction was diagnosed in 33 patients at myoglobin levels of 130.3-459.7 ng/ml, in the first four hours of the disease *(Kirilov and Golikov, 1991)*. *Johannes et al (1992)*, recorded myoglobin concentration in the range of <50 μg /L to 2990 μg/L and reported that some patients with acute myocardial infarction had myoglobin concentrations below the detection limit (50 $\mu g/L$) of the immunoturbidimetric assay. The haemagglutination test was used to measure the myoglobin content in the blood serum in 92 patients suffering from coronary heart disease (myocardial infarction, unstable angina and stable angina pectoris). The myoglobin level turned out a safe indicator at testing of acute myocardial infarction. In patients with angina pectoris, the myoglobin level did not differ from control. However in patients with unstable angina pectoris, there was an increase of the myoglobin content after long term attacks (*Borovkov et al., 1991*). *Similarly Johannes et al (1992)* reported that serum myoglobin concentrations did not increase in patients with angina pectoris (ranged from <50 μg/litre to 72.6 μg/litre and were <50 μg/litre in 43 cases). On the other hand in their study, *Johannes et al (1992)* found that serum myoglobin levels were high in almost all patients who were admitted at 6 hours after the onset of symptoms of acute myocardial infarction who presented with non diagnostic electrocardiogram on admission.

VALUE OF MYOGLOBIN TO EVALUATE RESPONSE TO STREPTOKINASE :

From table (2), it is evident that peak serum myoglobin level, recorded after 6 hours from the onset of symptoms was higher in streptokinase treated group than non streptokinase treated group (884.46±176.9 μg/litre versus 486.93±95.43 μg/litre). On the other hand, in this study, patients receiving intravenous streptokinase therapy showed significantly low serum myoglobin levels than non streptokinase treated patients after 12 hours from the onset of symptoms (table 2). Also, myoglobin release stopped early in streptokinase treated patients than in non streptokinase treated patients, as control values were reached after 12 hours in contrast to 48 hours, respectively (tables 1 & 2) .

This higher peak and the early stop, of myoglobin release in streptokinase treated patients, can be explained by the fact that reperfusion alters the release kinetics of myoglobin and other cardiac cytoplasmic proteins by washout from injured or dead myocytes *(Vatiner et al., 1978 and Kwong et al., 1984).* A rapid increase and subsequent decrease in cardiac cytoplasmic proteins occur as perfusion is restored to the ischaemic region of the heart. From tables (1 & 2) it is apparent that myoglobin release continued for some time after initiation of thrombolytic therapy and probably after reperfusion. The cause of ongoing myoglobin release is presently unclear. Although large vessel opening probably occurs within a short period of time it is possible that reperfusion of the microcirculation is uneven and variable over time and myoglobin might be released from differnt areas within the occluded segment at different times depending when microcirculation reperfusion actually occurs *(Avery et al., 1985).* A number of studies that have examined ischaemia and reperfusion indicate that there could be considerable tissue injury associated with restoration of blood flow to an injured or infarcted area. This additional injury could also produce ongoing myocardial release *(Cobb et al., 1976)*. Consequently, such ongoing myoglobin release may reflect so called reperfusion injury, that is irreversible damage to ischaemic but viable tissue after restoration of blood flow after coronary occlusion *(Braunwald and Klener, 1985 & Nayler and Elz., 1986)*.

The findings, in this study are in agreement with the results of other workers. *Strandberg et al(1992)* reported that myoglobin release stopped after 5.5 ± 3.3 hour in streptokinase treated patients which was 11 hours earlier than in the control and they found that streptokinase resulted in accelerated and more uniform development of the infarct process early about 10 hours after the onset of therapy compared with 20-30 hours in the refrence group.

Measurement of serum myoglobin levels is useful and suitable for rapid judging on success or failure of reperfusion after thrombolytic therapy using its disappearance rate *(Katayama et al., 1991)*. It is now generally agreed that most of transmural myocardial infarctions are associated with fresh thrombi superimposed on atherosclerotic lesions of varying severity*(De Wood et al,1980)*. Although the thrombi can often be dissolved by lytic agents, it is frequently difficult to assess whether or not reperfusion has occured if an immediate angiographic examination is not performed *(Avery et al., 1985)*. *Johannes et al (1992)*. concluded that on the basis of recent findings, coronary angiography was performed only selectively usually several days after admission. So, the occurrence of reperfusion in Q wave infarction could not be observed directly by coronary angiography. It is well established both clinically *(Kwong et al., 1984, Ellis et al, 1988, Lewis et al 1987)* and experimentally *(Vatiner et al., 1978 and Ellis et al., 1985)*.that reperfusion in Q wave infarction results in more rapid increase in creatine kinase and myoglobin and an early peaks of both proteins. Early reperfusion was assumed to have occured, if there was both a myoglobin peak within seven hours and a creatine kinase peak within 16 hours after the onset of chest pain *(Katus et al., 1988)*; but elevated levels of myoglobin are often apparent before creatine kinase is detected *(Stone et al., 1977)*. Myoglobin is known to be cleared from the plasma with a half life of disappearance of less than 10 minutes *(Klocke et al., 1982)*. Because of this early appearance and usually rapid clearance, blood myoglobin concentration time pattern may provide a useful marker of entry of protien into the vascular space in situations such as myocardial infarction.

The rapid decrease in serum myoglobin level observed in our study in streptokinase treated patient extends other clinical studies that myoglobin concentration curves could be used as index of successful coronary reperfusion in acute myocardial infarction (*Ellis et al., 1985*). *Avery et al (1985)* found an increase in serum myoglobin level of at least 4.6 fold over the first 2 hours in 29 of 34 patients successfully reperfused after intravenous streptokinase, whereas the ratio was less than 4.6 in all six patients in whom therapy was unsuccessful and concluded that with the use of rapid human assay, myoglobin could potentiailly be assessed within 1-2 hours. Recently, sixty three patients with acute myocardial infarction treated with thrombolysis, has been studied for early assessment of coronary artery reperfusion. The thrombolytic agent used was prourokinase in 32 patients with recombinant tissue plasminogen activator in 20 patients and anisaylated plasmingen streptokinase activator complex (APSAC) in 31 patients. In their study, myoglobin peaked after 2.11±2.8 hours in patients with successful (peak value 2,361±1,824 ng/ml) compared with 4.9±4.2 hours in patients with failed reperfusion (*Markus et al., 1993*).

Also *Avery et al (1988)* in their study, they found in each of 14 patients receving intravenous streptokinase, in whom the infarct related artery was noted to be patent at the time of coronary arteriography, there was an early discrete peak in the myoglobin concentration curve time.

In addition to the early peaks in myoglobin, seen with successful reperfusion, a second and perhaps particularly more important feature is the consistently rapid rate of rise of myoglobin. The time required for myoglobin to increase from 25% to 100% of its peak value (T25-100) was calculated for some patients studied and it has been reported that rapid rise in myoglobin (corresponding to a short T25-100 of 71 ± 7.9 minutes) is present in all patients in whom reperfusion was successful *(Avery et al., 1988)*. Also *Avery et al(1985)*, found T25-100 of 48±27 (SD) minutes in successfully reperfused patients in contrast with that in unsuccessfully reperfused (T25-100=5.7 hours). These differences in T25-100 values suggest that this index can distinguish between the two groups without overlap.

MYOGLOBIN AND OTHER CARDIAC MARKERS :

From table (9) it is evident that there was strong correlation between serum myoglobin level and each of creatine phosphokinase and serum glutamic oxalacetie transaminase (SGOT) in both non streptokinase and streptokinase treated groups(r= 0.82 and 0.89 & 0.9 and 0.9 respectively) after 12 hours of the onset of symptoms of myocardial infarction. The use of thrombolytic therapy for myocardial salvage reinforces the need for early and rapid identification and confirmation of acute myocardial infarction within four to six hours after the onset of infarct related symptoms (Timi study group, 1985)

Creatine kinase and creatine kinase myoglobin activity, which are the most frequently assayed biochemical markers in emergency laboratories do not rise in serum until the fourth to the eighths hour after the onset of chest pain *(Lee and Goldman , 1986)* and so are not sensitive enough for diagnosis during the early stages of acute myocardial infarction. Myoglobin, by contrast is an early sensitive marker of myocardial cell injury *(Drexel et al., 1983 & Gibler et al., 1987)*. Many authors had tested the seneitivity of myoglobin, MB-creatine kinase and creatine kinase myoglobin in blood samples obtained before thrombolytic therapy of patients with acute myocardial infarction. Raised serum concentration of total creatine kinase and its myoglobin fraction though very sensitive tend to occur later than ST segment changes during the course of myocardial infarction. Several studies have compared creatine kinase and creatine kinase myoglobin during the early phase of myocardial infarction. In general, these studies have found that serum myoglobin has a higher diagnostic value than creatine kinase and its isoenzymes *(Sylven et al.,1978 & Norgard et al., 1980)*, though one study found no difference between the early rise of creatine kinase MB and myoglobin *(Freman et al., 1981)*. *Magnus et al (1989)* supported the superiority of myoglobin as an early marker of myocardial infarction since serum concentrations of myoglobin have been found as early as one hour after myocardial infarction. In addition it has been suggested that serum myoglobin mirrors the early course of myocardial necrosis by the temporal relation between myoglobin release and electrocardiographic changes (*Sedernholm and Sylven.,1983*). In particular 50% of patients with initially negative electrocardiograms and myocardial infarction were correctly diagnosed on the basis of their serum myoglobin concentrations without increase in the percentage of false positive results (*Magnus et al., 1989*).

The strong correlation between serum myoglobin level and serum creatine phosphokinase level, in our study, is in accordance with other clinical studies. *Johannes et al* (*1992*) found moderate correlation coefficient between peak values of myoglobin and creatine kinase. Also *Tommaso et al* (*1980*) reported that infart size as determined by CPK level correlated relatively well with the peak myoglobin level and suggested that myoglobin may be as good an indicator of infarction size as analysis of total C.P.K.. In their study; *Johannes et al* (*1992*), described the criteria of the release kinetics of myoglobin, creatine kinase and creatine kinase MB activities. An abnormal increase of the peak concentration and return to the refrence range occured significantly earlier for myoglobin than for creatine kinase and creatine kinase myoglobin activity. The median difference between the first appearance of myoglobin and creatine kinase and the first appearance of myoglobin and creatine kinase MB was one hour each. In these patients, the diagnostic sensitivity of myoglobin was significantly higher than that of both creatine kinase and creatine kinase MB activity at 3.5 and four hours after the onset of symptoms. The diagnostic sensitivity of myoglobin was 0.5 (50%) 3.4 hours after the onset of symptoms a value that creatine kinase activity only reached 4.8 hours and creatine kinase MB after five hours respectively. All patients had increased myoglobin concentrations (100%) at six hours and increased creatine kinase and creatine kinase myoglobin activities at 12 hours after the onset of chest pain. The magnitude of increase (calculated as the peak value divided by the cut off value) of myoglobin was significantly greater than for creatine kinase activity and creatine kinase MB activity. The correlation coefficient between peak values of myoglobin and creatine kinase was 0.69 and between myoglobin and creatine kinase MB was 0.68.

Recently, Markus et al (1993), in their study, compared the serum markers creatine kinase, creatine kinase MB, myoglobin and troponin T systematically regarding their value for non invasive prediction of reperfusion. Of all markers examined, myoglobin appeares to exhibit several advantages that make this protein particularly suitable for early non invasive prediction of patency of the infarct related vessel. First, analysis of the initial myoglobin concentration increase allows the best overall prediction of success or failure of thrombolytic therapy of all studied markers for analysis of both time to peak and early slopes. Additionally, the negative predictive value and specificity which reflect identification of patients with occluded vessels, were quiet high at 82% and 88%, respectively, numberes that are not reached by other markers examined. Second, a more rapid assay using turbidimetry has been introduced into clinical practice (*Ishi et al., 1991 & Mair et al., 1991*) which provides laboratory results after a short time of only 10 minutes. Third myoglobin conecntration exhibited the earliest rise of all serum markers examined after initiation of therapy. The usefulness of myoglobin for non invasive prediction of infarct-artery patency has been confirmed by other investigators (*Clemmensen et al., 1991*). However, *Clemmensen and Cowerhers (1991)* reported no comparison with other serodiagnostic markers in their study. *Markus et al (1993)*, reported that CK-MB and CK maximum yielded relatively good diagnostic information regarding coronary artery patency as well. However, Compared with the time elapsed to reach peak myoglobin levels, it took four to five times as long to reach maximal concentrations of these enzymes. This time fram would therefore significantly reduce the chances to salvage myocardium by means of invasive procedure. It has been shown previously that combined analysis of non invasive markers (i.e peak CK, resolution of ST segment elevation occurrence of reperfusion arrhythmias can improve assessment of success or failure of thrombolytic therapy (*Hohnloser et al., 1991*), but, in their study *Markus et al (1993)* reported that other reperfusion markers including troponin T did not add significantly to the predictive power of myoglobin analysis, indicating the particular strength of this marker.

Another importance of myoglobin is its value in estimation of infarct size. It is useful for risk stratification to estimate infarct size in patients with acute myocardial infarction because infarct size correlates closely with mortality (*Geltman et al., 1979*) and prognostic indexes as cardiac failure (*Kahn et al, 1977*) arrhythmias (*Roberts et al., 1975*) and ventricular function (*Rogers et al., 1977*). Infarct size can be estimated by electrocardiography, ecchocardiography, left ventriculography, radionuclide methods or cardiac enzymes. The ECG is conveninent for estimation of infarct size, however, because ST segment elevation and Q wave amplitudes change spontaneously within 24 hours of the onset of acute myocardial infarction and change rapidly after reperfusion (*Selwyn et al., 1977 & Essen et al., 1979*) the estimation of the infarct size from the ECG at the very early stage of myocardial infarction is unreliable. The motion of the postischaemic myocardium without the development of necrosis may remain depressed for several days *(Braunwald and Klener., 1982)*. Therefore the estimation of infarct size by ecchocardiography or left ventriculography at a very early stage is also of doubtful accuracy. Estimation of infarct size by measurement of intramyocardial protein released from the injured myocardium may be the most precise method. Creatine kinase, creatine kinase MB isoenzymes *(Sobel et al., 1978 & Jansen et al., 1985)* and cardiac myosin light chains *(Isope et al., 1987)* have been used for estimating infarct size. These markers are not appropriate for the early estimation of infarct size because 1-6 days is required. Conversely, myoglobin appears in the blood after infarction much earlier than creatine kinase *(Grenadier et al., 1983)* because of its lower molecular weight. In *1989, Ellis and Saran* reported that total myoglobin release into the blood, can be used as an index of infarct size in animal model. Recently, *Tsuminori et al (1993)* compared infarct size estimated from total myoglobin release with that estimated from total CK release and left ventriculograms and the total time required for total myoglobin release to be available with that obtained for total CK release and they suggested that infarct size can be estimated accurately with approximatlly 4 hours of reperfusion by calculating total myoglobin release from injured myocardium into 1 ml of blood samples collected at 15 minutes intervals in patients with acute myocardial infarction. *Similarly*

Maddison et al (1980) collected blood samples at 4 hour intervals in 29 patients and showed a significant correlation between $\sum$ myoglobin (total myoglobin) and $\sum$ CK.MB. *Groth et al (1984)* collected blood sample in 33 patients with acute myocardial infarction at 1-2 hour intervals after admission and found significant correlation between $\sum$ MB and $\sum$ CK-MB ($r = 0.7$). Further studies are necessary to clarify the usefulness of the estimation of infarct size from serial plasma myoglobin measurement in patients with reperfusion. In our study it is evident that intravenous streptokinase improved the electrocardiographic changes evidenced by reversal of S T segment elevation in some cases and decreased S T segment elevation in most cases within 6 hours after intravenous administration of streptokinase *Mc Cullough et al (1992)* reported that reperfusion after intravenous thrombolytic therapy was diagnosed if the measured ST segment elevation fell by greater than or equal to 50% at 2 hours post dosing and summated ST segment epsilon. ST segment elevations were significantly smaller in reperfused compared with non reperfused patients. Other investigators reported that ST segment changes after reperfusion therapy may evolve in two different ways, progressive, regression or accentuation of ST segment elevation *(Monassier et al., 1992)*.

SUMMARY
&
CONCLUSION

SUMMARY AND CONCLUSION

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 acute myocardial 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 with septal 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 (lipoqram, 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 oxalacetic transaminase (SGOT) 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 myocardial infarction:

3- Early non invasive prediction of coronary artery patency in the majority of patients after initiation of thrombolytic 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 soon after 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 skeletal muscle 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 myocardial infarction to be ruled out within a few minutes with a very high probability.

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APPENDIX

Appendix (1) : Clinical data of non-streptokinase treated group

No.	Age	Sex	MI by ECG	Risk Factor: Smoking	Risk Factor: Hypertension	Risk Factor: diabetes	Risk Factor: Family History	Pulse	Blood pressure: systolic	Blood pressure: diastolic	Blood pressure: mean	Oedema of lower limbs	Neck veins	Cardiac Examination	Bilateral Basal Rales
1	72	Male	Anter-inferior	+	−	−	−	75	120	70	87	−	−	−	−
2	58	Female	Anterior	−	+	+	−	100	110	70	83	−	−	−	+
3	51	Female	Anterior	−	+	−	−	100	160	90	113	−	−	S4	+
4	41	Male	Inferolater	−	+	−	−	80	120	80	93	−	−	−	−
5	61	Male	Anterior	+	+	−	−	100	120	90	100	−	−	−	−
6	58	Male	Anterior	+	−	−	−	60	100	70	80	−	−	−	−
7	63	Male	Antero-inferior	−	+	−	−	80	90	50	63	−	−	−	−
8	55	Female	Inferior	−	−	−	−	85	100	60	73	−	−	−	−
9	81	Male	Anterior	−	−	−	−	86	130	80	96	−	−	−	−
10	51	Male	Anterior	−	−	+	−	75	110	80	90	−	−	−	−
11	62	Female	Anterior	−	−	−	+	100	130	80	96	−	−	S4	−
12	64	Male	Anterior	−	+	−	−	100	120	80	93	−	−	−	−
13	56	Female	Anterior	+	+	−	−	100	150	80	103	−	−	−	−
14	65	Male	Anterior	+	+	−	−	85	115	90	98	−	−	−	−
15	59	Male	Anterior	+	+	−	−	100	150	90	110	−	−	S4	−

Age : Mean ± S.E. = 56.7 ± 3.2

Appendix (2) :Clinical data of streptokinase treated group

No.	Age	Sex	MI by ECG	Risk Factor Smoking	Hypertension	diabetes	Family History	Pulse	Blood pressure systolic	diastolic	mean	Oedema of lower limbs	Neck veins	Cardiac Examination	Bilateral Basal Rales
1	55	Male	Inferolateral	+	+	−	+	120	150	90	110	−	−	S4	+
2	56	Female	Anterior	−	+	+	+	100	110	80	90	−	−	−	−
3	57	Female	Anteriferior	−	−	+	−	75	100	70	80	−	−	S4	−
4	54	Male	Inferior	+	+	+	−	56	100	70	80	−	−	−	−
5	52	Male	Inferior	+	−	+	+	78	140	95	110	−	−	−	−
6	58	Female	Anterior	−	−	−	−	75	105	75	85	−	−	−	−
7	54	Male	Anterior	−	+	−	−	100	150	100	116	−	−	S4	−
8	50	Female	Inferior	−	+	+	−	100	150	95	102	−	−	−	−
9	61	Male	Anterior	−	−	+	−	120	100	70	80	−	−	−	−
10	56	Male	Anterior	−	+	+	−	75	140	95	110	−	−	−	−
11	58	Female	Antero - inferior	−	−	+	−	115	90	70	76	−	−	−	−
12	62	Male	Anterior	+	+	−	−	100	130	80	96	−	−	−	−
13	58	Female	Anterior	−	+	+	−	110	120	90	100	−	−	−	−
14	60	Male	Anterior	+	+	−	−	100	130	95	106	−	−	−	−
15	59	Male	Anterolaterl	+	+	−	−	75	120	90	100	−	−	−	−

Age : Mean ± S.E. = 59.8 ± 8.9

Appendix (3) : Serum myoglobin (microgram/ litre) of non streptokinase treated group.

No.	Control	Non streptokinase treated group after								
		6 hours	12 hours	24 hours	second day	third day	fourth day	fifth day	sixth day	seventh day
1	66	460	380	200	86	80	80	75	75	74
2	70	380	320	89	88	86	88	80	70	70
3	72	410	300	110	100	78	79	75	70	73
4	66	590	430	140	82	86	84	86	76	75
5	65	562	460	150	90	81	82	88	79	83
6	65	662	480	145	88	82	88	85	87	80
7	82	648	460	340	180	92	90	87	82	78
8	89	480	400	320	90	85	85	80	80	63
9	68	392	186	86	85	83	78	75	75	76
10		380	280	90	86	80	88	78	89	77
11		520	340	120	95	86	85	85	87	80
12		530	420	240	70	90	73	72	72	74
13		540	630	430	120	92	88	83	83	72
14		320	260	90	80	75	80	80	78	79
15		430	310	115	100	76	76	75	70	70

Appendix (4): Serum myoglobin (microgram / litre) of streptokinase treated group

No.	Control	streptokinase treated group after								
		6 hours	12 hours	24 hours	second day	third day	fourth day	fifth day	sixth day	seventh day
1	66	955	105	86	73	73	76	72	70	70
2	70	980	100	80	75	80	76	73	73	72
3	72	623	82	82	80	86	82	82	85	75
4	66	346	86	78	78	81	76	70	70	69
5	65	895	80	83	82	70	70	72	72	69
6	65	692	88	80	83	83	86	82	78	62
7	82	969	86	75	73	70	72	78	71	72
8	89	987	75	81	70	70	72	68	65	68
9	68	680	82	79	70	70	72	69	65	60
10		895	89	80	80	80	79	78	71	68
11		840	90	72	82	71	70	68	65	65
12		890	72	96	80	80	78	75	72	72
13		980	115	82	100	78	78	75	73	70
14		960	88	86	81	81	77	75	71	68
15		975	98	79	83	83	75	77	69	69

Appendix (5) : Laboratory data of non streptokinase treated group

No.	Creatine phosphokinase (CPK)	Plasma lipids				Serum oxalacetic transaminase (SGOT)	Blood suger		Serum ceratinine
		Total cholesterol	Triglycerides	High density lipoprotein	Low density lipoprotein		Fasting	Post Prandial	
1	960	170	83	43	111	170	100	150	0.9
2	760	190	180	34	120	145	140	230	0.9
3	690	260	182	86	138	220	110	135	0.8
4	1038	240	175	32	173	105	115	147	0.7
5	1964	180	145	46	105	190	100	130	0.9
6	760	225	160	65	128	205	105	140	0.9
7	1150	165	85	44	104	212	115	135	0.9
8	960	280	190	80	162	190	115	130	1.0
9	670	185	140	42	115	135	100	132	0.7
10	690	320	220	92	184	115	190	340	0.9
11	960	168	110	43	163	118	210	420	0.8
12	965	170	82	45	109	110	112	136	1.0
13	1000	260	152	35	195	95	100	120	0.7
14	582	226	140	60	138	112	108	129	0.9
15	690	180	90	38	124	105	115	138	0.9

Appendix (6) :Laboratory data of streptokinase treated group

No.	Creatine phosphokinase (CPK)	Plasma lipids				Serum oxalacetic transaminase (SGOT)	Blood suger		Serum ceratinine
		Total cholesterol	Triglycerides	High density lipoprotein	Low density lipoprotein		Fasting	Post Prandial	
1	1150	270	200	70	160	205	85	110	0.8
2	1964	190	135	42	121	115	100	190	0.9
3	540	235	100	63	152	100	180	220	0.9
4	490	290	91	30	241	150	138	205	1.0
5	870	200	170	30	136	165	100	147	0.9
6	980	170	110	35	113	195	190	350	0.8
7	1175	175	150	45	100	280	83	128	0.7
8	490	120	208	20	59	84	149	250	1.0
9	630	165	150	36	99	192	130	215	0.8
10	890	165	112	38	105	112	160	240	1.0
11	852	205	190	23	144	180	135	230	0.7
12	870	210	98	50	141	95	120	132	0.8
13	960	205	102	48	136.6	90	100	118	0.8
14	860	180	110	62	96	115	135	190	0.7
15	928	230	260	43	135	210	110	120	0.7

ARABIC SUMMARY

الملخص العربى

الهدف من البحث :

تهدف هذة الدراسة الى تقييم فاعلية تعين مستوى الميوجلوبين فى مصل الدم لتشخيص حالات أحتشاء القلب الحاد وكذلك فاعليتة فى متابعة المرضى الذين يتناولون عقار الأسترتبوكيناز المذيب للجلطة.

ملخص البحث :

تضمنت هذة الدراسة ثلاثين مريضا مصابون بأحتشاء حاد فى عضلة القلب كمجوعة أختبار وقد قسمت هذة المجموعة الى مجموعتين كل مجموعة تحتوى على خمسة عشر مريضا حيث شملت المجموعة الاولى عشرة ذكور وخمس إناث تراوحت أعمارهم من ٤١ الى ٧٢ سنة ولم تتناول عقار الأسترتبوكيناز • أما المجموعة الثانية فقد شملت تسعة ذكور وستة إناث تراوحت أعمارهم من ٥٠ الى ٦٢ سنة وتناولت عقار الأسترتبوكيناز بالحقن فى الوريد بالاضافة الى علاج الاحتشاء الحاد • تضمنت الدراسة أيضا مجموعة ضابطة مكونة من عشرة من الأصحاء فى نفس الأعمار السابقة تم أختيار المرضى من وحدة العناية المركزة لمرضى الشريان التاجي بمستشفى المنصورة والزقازيق الجامعى وأقتصرت الدراسة على الحالات التى لم يمضى عليها أكثر من ٦ ساعات من وقت حدوث الام الصدر النمطى للأحتشاء الحاد•

تم أستبعاد أى مريض مصاب بالصدمة القلبية أو هبوط بوظائف الكلى أو أى مريض أخذ حقن بالعضل وكذلك أى مريض تناول عقار مغلقات قنوات بيتا أو مغلقات قنوات الكالسيوم كما وضع فى الحسبان موانع أستعمال عقار الأسترتبوكيناز فى المرضى الذين تناولوا هذا العقار •

تم أجراء الفحوص الاتية لكل مريض :

١- فحص أكلينيكى دقيق وخاصة للقلب والاوعية الدموية

٢- عمل أشعة للرئتين والقلب •

٣- تعيين نسبة الميوجلوبين فى مصل المرضى فى خلال ٦ ساعات ،١٢ ساعة ، ٢٤ ساعة من حدوث أعراض الاحتشاء الحاد ثم يوميا لمدة ٧ أيام •

٤- قياس مستوى إنزيمات االقلب(الكرياتين فوسفوكيناز والأكسال أستيك ترانزإمينار) بعد ١٢ ساعة من وقت حدوث الم صدر •

٥- تخطيط قلب كهربائى تقليدى (ذو الاثنى عشر أتجاها) عند دخول المريض وكذلك بعد ٦ ساعات من إعطاء عقار الأسترتبوكيناز فى الوريد للمجموعة الثانية •

نتائج البحث :

١- من الناحية الأكلينيكية :

لم توجد علاقة أرتباطية بين مستوى الميوجلوبين فى المصل وكل من ضغط الدم أو النبض أو السن•

٢- بالنسبة لرسم القلب الكهربائى فقد وجد أنة قد حدث تحسن فى الرسم الكهربائى لمعظم المرضى الذين تناولوا عقار الأسترتبوكيناز•

٣- بالنسبة للميوجلبين : فقد وجد أنة قد حدثت زيادة في مستوى الميوجلوبين في مصل المرضى الذين لم يتناولوا عقار الأسترتبوكيناز وكذلك المرضى الذين تناولوا عقار الأسترتبوكيناز بعد ٦ ساعات من حدوث الألم وفي حين أنة قد وجدت زيادة ذات دلالة أحصائية في مستوى الميوجلوبين في المصل بعد ١٢ ساعة من الشعور بالألم في المرضى الذين لم يتناولوا عقار الأسترتبوكيناز فإنة لم توجد زيادة ذات دلالة احصائية في مستوى الميوجلوبين في مصل المرضى الذين تناولوا عقار الأسترتبوكيناز بعد ١٢ ساعة من الشعور بالألم ٠ وبعد ٢٤ ساعة من أعراض الأحتشاء الحاد لم توجد زيادة في مستوى الميوجلوين في مصل المرضى في المجموعتين وكذلك الى اليوم السابع لم توجد زيادة ٠ وبمقارنة مستوى الميوجلوين في مصل المرضى الذين لم يتناولوا عقار الأسترتبوكيناز والمرضى الذين تناولوا عقار الأسترتبوكيناز وجدت زيادة ذات دلالة إحصائية في نسبة الميوجلوين في مصل المرضى الذين لم يتناولوا عقار الأسترتبوكيناز بعد ١٢ ساعة فقط من حدوث الألم ٠

٤- وجدت علاقة ارتباطية موجبه بين مستوى الميوجلوبين في مصل المرضى المصابين بالاحتشاء البطيني الحاد الذين تناولوا عقار الاستربتوكيناز والذين لم يتناولوا عقار الاستربتوكيناز وبين كل من مستوى انزيم الكرياتين فوسفوكينار والاكسال استيك ترانزاميناز - بعد ١٢ ساعة من حدوث الالم ٠

الاستنتاج :

مما سبق يتضح أن تعيين الميوجلوبين في مصل المرضى المصابين باحتشاء القلب الحاد يعتبر وسيلة سريعة للتشخيص المبكر لحالات احتشاء القلب الحاد وكذلك يمكن استخدام الميوجلوبين لمتابعة استجابة مرضى احتشاء القلب الحاد للعلاج بعقار الاستربتوكيناز المذيب للجلطة ٠

جامعة الزقازيق
كلية طب بنها

الميوجلوبين فى مصل مرضى الإحتشاء البطينى الحاد تحت تأثير العلاج بالاستربتوكيناز وبدونه

رسالة مقدمة من الطبيب

محمد محمود الحديدى خريزة
بكالوريوس الطب والجراحة

كجزء من المتطلبات للحصول على
درجة الماجستير فى الباطنة العامة

المشرفون

الأستاذ الدكتور
إكرام مصطفى الأسيوطى
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بسم الله الرحمن الرحيم