بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــسـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
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يجب أن

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15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

EARLY DIAGNOSIS OF ACUTE MYOCARDIAL INFARCTION

Thesis submitted for the partial fulfillment

of the Master Degree In Clinical Biochemistry

By

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NAD	Nicotinamide adenine dinucleotide
SR	Sarcoplasmic reticulum
TnT	Troponin T

INTRODUCTION

The diagnosis of an acute myocardial infarction (AMI) has traditionally been based on the triad of characteristic chest pain, specific electrocardiographic abnormalities and increased serum enzymes. [Johannes, 1991] Thrombolytic therapy requires an early detection of a suspected AMI, because the success rate of recanalization decreases proportionally to the time elapsed between attack and treatment [TIMI study group, 1985]. In addition, rapid and reliable diagnosis will be increasingly important for correct triage of patients into various hospital areas for appropriate degree of care. However, because the differential diagnosis value of chest pain is limited [Lee And Rouan, 1987] and the electrocardiography changes have varying degrees of sensitivity [McQueen, 1983] and specificity [Fulton And Mariott, 1963], measurement of serum enzymes as a reflection of myocardial cell damage play an important role in the diagnosis of an AMI in patients with a nondiagnostic electrocardiogram. [Johannes, 1991]

CK-MB determination are generally regarded as the reference standard for the diagnostic tests for AMI. However, the presence of atypical CK forms and adenylate kinase activities in serum may lead to false positive results, moreover

CK-MB activity in serum dose not increase until the fourth to eighth hour after the onset of chest pain therefore, lacks the desirable degree of sensitivity during the early stages of AMI.

In practice, physicians are sometimes left with patients in whom a definitive diagnosis of AMI cannot be made by CK-MB measurement and an alternative test of even greater sensitivity and specificity is needed. [Lee, 1986]

Troponin-T belongs to the proteins of the contractile apparatus that are unique in their primary structure for cardiac muscle. [katus, 1989]

Troponin T was found in the serum samples of patients with AMI from 3.5 hours to more than 10 days after onset of pain. [katus, 1989]

Troponin T has higher sensitivity and specificity compared with CK-MB measurement. [Hugo, 1991]

AIM OF THE WORK

The aim of this work is to study the sensitivity and diagnostic efficiency of serum Troponin T in diagnosis of acute myocardial infarction compared with other chemical tests especially measurement of MB isoenzyme of creatine kinase.

REVIEW

ISCHEMIA AND MYOCARDIAL FUNCTIONS.

METABOLIC & BIOCHEMICAL CHANGES

Under physiological condition, myocardium derives most of its energy from oxidative phosphorylation. when oxygen availability is limited, the rate of ATP synthesis declines, regeneration of ATP from ADP and phosphocreatine decreases, and ultimately, high-energy phosphate stores decline. The ratio of phospho-creatine to creatine is an index of energy reserve. [Schaefer, 1989]. The energy production and consumption remains balanced when contractile reserve is taxed. [Marshall, 1987]. The diminution of contractility induced by ischemia reflects a limited turnover of high-energy phosphate stores [Bittl, 1987] rather than reduction of total cellular content of ATP itself, until and unless ischemia is profound and sustained. [Asimakis, 1990]. However, even intermittent ischemia depletes the mitochondria of adenine nucleotides and may therefore limit oxidative metabolic reserve. [Guth, 1987].

When ATP content is reduced below 20 percent of control values, cells become unable to regenerate high-energy phosphate, to maintain physiological

ionic gradient, and to control their volume. The combination of reduced myocardial high-energy phosphate stores, cell swelling, and sarcolemmal damage appears to play a key role in cell death with ischemia or reperfusion. When tissue is only reversibly injured by ischemia (ie., when its viability can still be maintained by reperfusion), ATP stores are usually greater than 60 percent of control and electronmicroscopy may reveal only glycogen loss, nuclear chromatin clumping, intermyofibrillar edema, and mitochondrial swelling but no sarcolemmal damage or accumulation of amorphous dense bodies in the mitochondria. Reduction of ATP below 30 percent is usually associated with visible sarcolemmal damage and irreversible injury (ie., the tissue is not viable despite reperfusion). ATP but not CK content has been shown to correlate with return of contractile function after reperfusion. [Flaherty, 1982].

An abrupt closure of a coronary artery eliminates the flow of oxygenated blood in the myocardial region that the artery supplies. Within 8 to 10 seconds oxygen trapped in the region will be consumed, leading to significant functional and metabolic changes that encompass cessation of contraction, electrocardiographic changes, and cellular injury leading to eventual myocardial necrosis. [Fuster, 1992]