Detection of myocardial dysfunction in sepsis using the cardiac specific troponin I as a diagnostic and prognostic marker

Thesis

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By

Ahmed Fathi Mohammed

M.B.B.ch

Supervised by

Dr. Hossam Mostafa Sherif

Assistant Professor of Critical Care Medicine

Faculty of Medicine - Cairo University

Dr. Mohammed Magdy M. Abbas

Lecturer of Critical Care Medicine

Faculty of Medicine - Cairo University

Dr. Amr Mohammed Essmat

Lecturer of Critical Care Medicine

Faculty of Medicine - Fayoum University

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قَالُوا۟ سُبْحَٰنَكَ لَا عِلْمَ لَنَآ إِلَّا مَا عَلَّمْتَنَآ ۖ إِنَّكَ أَنتَ ٱلْعَلِيمُ ٱلْحَكِيمُ ﴿٣٢﴾

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Abstract

Introduction:

Cardiac troponins I (cTnI) and T (cTnT) are cardiospecific markers of prognostic value in acute myocardial infarction (AMI), unstable angina, acute chest pain, myocarditis, cardiac trauma, and perioperative cardiac complications .Therefore, cTnI measurement may be a method that can identify myocardial injury in sepsis in a simple and practical way.

Aim of work:

Detection of myocardial dysfunction in sepsis using the cardiac specific troponin I

Patients and methods:

Our study included 30 patients with different degree of sepsis, all patients and controls were subjected to; cardiac specific troponin I,echocardiography and APACHE II scoring at day1 and day 5 of admission.

Results:

Patients were divded into cardiac troponin I positive and cardiac troponin I negative, 18 (60%) patients had elevated serum cTnI.These cardiac troponin I positive patients had higher need for MV and inotropic support; there was a statistically highly significant difference between the two groups as regards the need for for mechanical ventilation and inotropes , it shows that cTnI positivity is associated with more need for MV [55.6% in cTnI+ve vs 25% in cTnI-ve] *(P<0.05)* and more need for inotopes [61.1% in cTnI+ve vs 25% in cTnI-ve] *(P<0.05)*.Higher APACHE II score in cardiac troponin I positive patients [18.4 ± 3.8 in cTnI+ve vs 8.9 ± 2.8 in cTnI-ve)] *(P<0.05)*, lower ejection fraction [44.4% of cTnI+ve patients vs. 16.7% of cTnI-ve patients] *(P<0.05)* and higher mortality [55.6 %in cTnI+ve vs 25% in cTnI-ve] *(P<0.05,*. compared to normal cTnI patients. serum cTnI and APACHE II score were predictor of death and length of stay in intensive care unit. Serum cTnI was a good predictor of need for inotropic/vasopressor support. Receiver-operating characteristics of serum cTnI as a predictor of death in septic shock were significant. The elevated serum level of cTnI correlated with the lower left ventricular ejection fraction ($p < 0.001$).

Conclusion:

Myocardial injury can be detected in patients with sepsis severe sepsis or septic shock by serum cTnI. Serum cTnI concentration correlates with myocardial dysfunction in septic shock. Serum cTnI can predict increased severity of sepsis and higher mortality.

(Key Words): **Myocarial dysfunction, Sepsis, Troponin**

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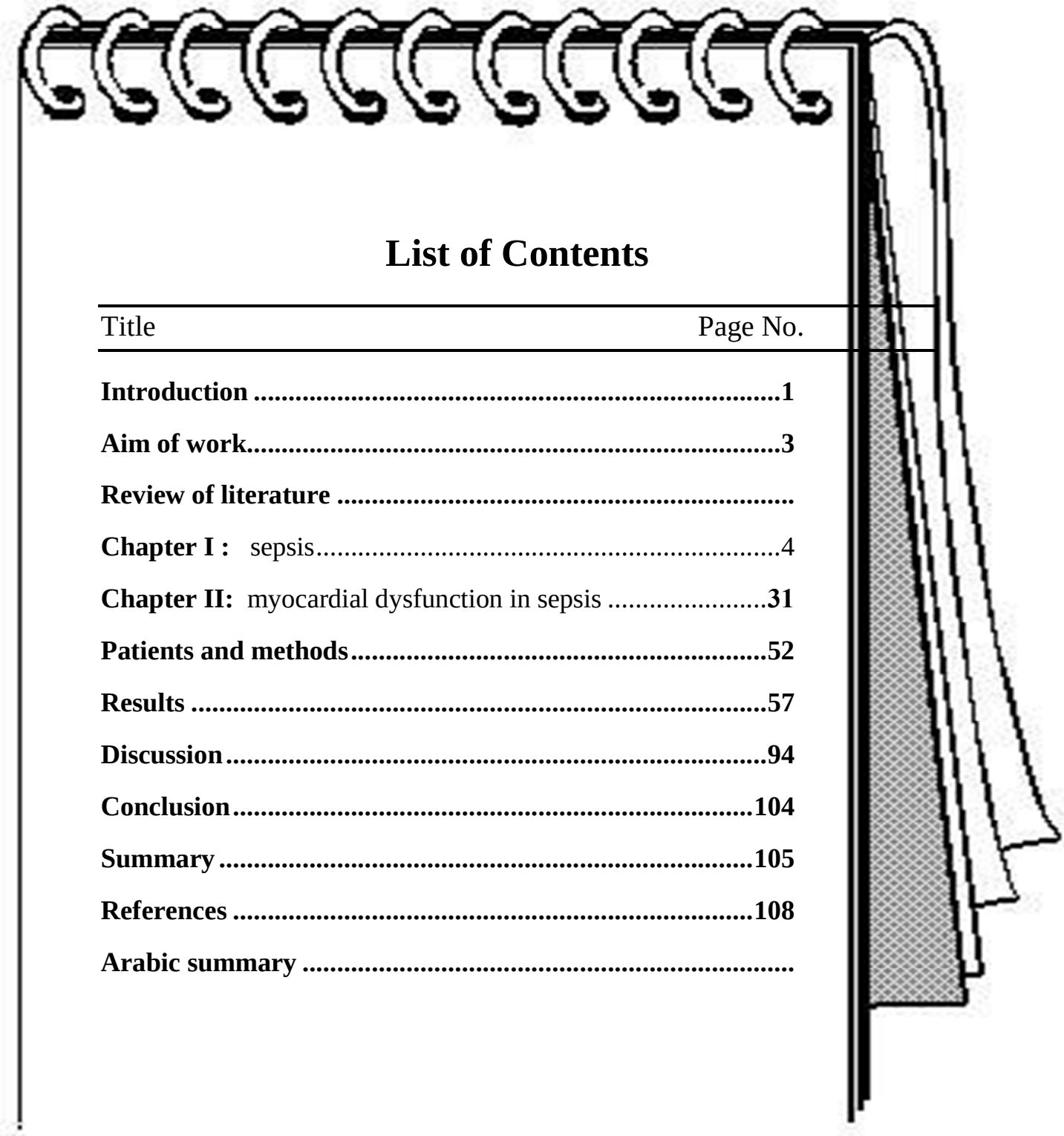
List Of Abbreviations

ADP	**Adenosine Diphosphate**
APACHE II	Acute Physiology and Chronic Health Evaluation
ATP	Adenosine Triphosphate
Ca^2	Calcium
cAMP	Cyclic Adenosine Monophosphate
CI	Cardiac Index
CK	Creatine Kinase
CK-MB	Creatine Kinase MB Fraction
CO	Cardiac Output
cTnC	Cardiac Troponin C
cTnI	Cardiac troponin I
CVA	Cerebrovascular Accident
DNA	Deoxyribonucleic acid
ECG	Electrocardiography
ESC/ACC	European Society of Cardiology/American College of Cardiology Committee
ICU	Intensive Care Unite
IL	Interleukin

List of abbreviations

LV	Left Ventricle
LVEF	Left Ventricular Ejection Fraction
MEIA	Micro-particle enzyme immunoassay
MI	Myocardial Infarction
MICU	Medical Intensive Care Unite
NADPH	Nicotinamide Adenine Dinucleotide Phosphate.
NO	Nitric Oxide
NOS	Nitric Oxide Synthase
PAC	Pulmonary Artery Catheter
PAWP	Pulmonary Artery Wedge Pressure
SIMD	Sepsis Induced Myocardial Dysfunction
PAWP	Pulmonary Artery Wedge Pressure
SIMD	Sepsis Induced Myocardial Dysfunction
TEE	Transesophageal Echocardiograph
TNFα	Tumor Necrosis Factor α
TTE	Transthoracic Echocardiography
-ve	Negative
+ve	Positive
MODS	multiorgan dysfunction syndrome



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Intoduction and aim of work

Introduction

Inadequate myocardial performance, characterized by left ventricular systolic depression and diastolic dilatation, is a common and early complication of septic shock **(Grocott-Mason et al.,2005)**. Studies in humans **(Hinshaw et al.,2004)** strongly argue against an ischemic origin of sepsis-induced cardiac injury. However, a dysfunctional microcirculation that produces regional flow disturbances and abnormal tissue oxygenation is a hallmark of septic shock, which may cause relative ischemia in various organs, including the heart. Moreover, regional myocardial ischemia may well be present in septic patients with identifiable coronary risk factors or coexistent coronary artery disease.

The mechanism of action of the Myocardial depressant substances was recently shown to be attributable to synergistic effect of tumor necrosis factor and interleukin 1 at the subcellar level these cytokines produce depressant activity in a nitric oxide mediated mechanism (**Margaret et al.,1999**). Measurement of Cardiac specific contractile protein troponin I have been shown to be superior to measurement of creatine kinase Mb (CKmb) for the detection of minor Myocardial injury. it is the inhibitory subunit of the tropnin complex. Unlike the B form of the CK it is never expressed in fetal or adult skeletal muscles. It has been shown to be highly specific for cardiac injury in postoperative patients and in patients with myopathesis (**Adams et al.,1993**). It has been shown to accurately detect cardiac contusions with a greater sensitivity than transthoracic echocardiography and it can detect myocardial injury in the absence of diagnostic electrocardiography changes (**Adams et al,.1996**).

Introduction& Aim of work

Cardiac troponins I (cTnI) and T (cTnT) are cardiospecific markers of prognostic value in acute myocardial infarction (AMI) **(Wu AHB et al 1994)**, unstable angina **(Galvani et al.1997)**, acute chest pain **(Polanczyk et al.,1998)**, myocarditis **(Smith SC et al.2oo3)**, cardiac trauma **(Adams et al.,1999)**, and perioperative cardiac complications **(Adams JH,et al.1999)**. Also, cTn positivity has been documented in patients with heart failure of nonmyocardial ischemic origin **(La Vecchia et al.1997)**, and in a heterogeneous population of critically ill patients in medical **(Guest TM,et al.1995)**, surgical **(Kollef et al.1998)**, and pediatric intensive care units (ICUs) **(Hirsch et al.1999)**.

Therefore, cTnI measurement may be a method that can identify myocardial injury in sepsis in a simple and practical way.

Aim of work

1. Assessment of myocardial dysfunction in sepsis for administration of optimal therapy.
2. Analysis of the elevation of cardiac specific troponin I in patient with sepsis, severe sepsis and septic shock.
3. Evaluation of the relationship between cardiac troponin I and left ventricular ejection fraction.
4. Assessment of prognostic value of cardiac specific troponin I in patient with systemic sepsis.