بسم الله الرحمن الرحيم

MONA MAGHRABY

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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MONA MAGHRABY

Assessment of Extent of Myocardial Injury in Patients Undergoing Transvenous Implantation of Permanent Pacemaker using Cardiac Troponin I (cTnI) as a Marker of Structural Heart Damage and it's Relation to Different Sites of RV Implantation

Thesis

Submitted for Partial Fulfillment
of Master Degree in **Cardiology**

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Mohamed Mahmoud Ali Mohamed Ramadan

List of Contents

List of Tables

Table No.	Title	Page No.

List of Figures

List of Figures cont...

List of Abbreviations

Abb.	Full term
AAI	*Single-chamber atrial*
AAIR	*Rate response available if desired*
AV	*Atrioventricular*
AV	*Atrioventricular node*
BBB	*Bundle Branch Block*
BPEG	*British Pacing and Electrophysiology Group*
CK-MB	*Creatine kinase MB isoform*
CRT	*Cardiac resynchronization therapy*
cTn	*Cardiac troponin values*
cTnI	*Cardiac troponin I*
DDD	*Dual-chamber*
DDDR	*Rate response available if desired*
ECG	*Electrocardiogram*
hs	*High-sensitivity*
ILR	*Insertable loop recorder*
NASPE	*North American Society of Pacing and Electrophysiology*
SA	*Sinoatrial*
SND	*Sinus Node Dysfunction*
URL	*Upper reference limit*
VDD	*Single-lead, atrial-sensing ventricular*
VVI	*Single-chamber ventricular*
VVIR	*Rate response available if desired*

INTRODUCTION

The term myocardial injury should be used when there is evidence of elevated cardiac troponin values (cTn) with at least one value above the 99th percentile upper reference limit (URL). The myocardial injury is considered acute if there is a rise and/or fall of cTn values ***(Thygesen et al., 2007).***

The cardiac troponin I (cTnI) is a part of the cardiac contractile apparatus, the troponin-tropomyosin complex. It is a very sensitive laboratory marker of myocardial cell necrosis and one of the gold standard measurements in detecting myocardial injury. Elevated cTnI levels maybe associated with a variety of clinical conditions like myocardial infarction, acute pulmonary edema, ventricular tachycardia, shock, acute renal impairment ***(Thygesen et al., 2010).***

Transvenous insertion of endocardial leads for permanent pacing is accompanied by troponin elevation compatible with myocardial damage, secondary to the direct myocardial trauma elicited by pacing leads ***(Boos et al., 2004).***

The RV apex has been the preferred site for RV lead placement because of the ease of implantation and low risk of lead dislodgement. With the development of active fixation leads, alternative RV pacing sites have been explored, including the RV outflow tract, the RV septum, and the His bundle region. Pacing from these sites is thought to be more

physiological, engaging the Purkinje network earlier than apical pacing thus reducing or preventing the electric and mechanical dyssynchrony associated with RV apical pacing. Some data from acute or short-term randomized studies support this hypothesis ***(Shimony et al., 2012).***

Aim of the Work

The aim of the study is to assess the extent of myocardial injury in patients undergoing trans-venous implantation of permanent pacemaker using cardiac troponin I (cTnI) as a marker of myocardial injury and it's relation to different sites of RV pacing and number of trials of screwing the RV lead into the myocardium.

Chapter 1

MYOCARDIAL INJURY

The term myocardial injury should be used when there is evidence of elevated cardiac troponin values (cTn) with at least one value above the 99th percentile upper reference limit (URL). The myocardial injury is considered acute if there is a rise and/or fall of cTn values ***(Thygesen et al., 2007).***

Biomarker detection of myocardial injury and infarction:

Cardiac troponin I (cTnI) and T (cTnT) are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the heart ***(Thygesen et al., 2012).***

Increase in cTnI values have not been reported to occur following injury to non-cardiac tissues. The situation is more complex for cTnT. Biochemical data indicate that injured skeletal muscle expresses proteins that are detected by the cTnT assay leading to some situations where elevations of cTnT could emanate from skeletal muscle ***(Mair et al., 2017).***

Recent data suggest that the frequency of such elevations in the absence of ischaemic heart disease may be higher than originally thought ***(Schmid et al., 2018).***

cTnI and cTnT are the preferred biomarkers for the evaluation of myocardial injury and high-sensitivity (hs)-cTn assays are recommended for routine clinical use ***(Apple et al., 2015).***