



شبكة المعلومات الجامعية
التوثيق الإلكتروني والميكروفيلم

بسم الله الرحمن الرحيم



MONA MAGHRABY



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شبكة المعلومات الجامعية التوثيق الإلكتروني والميكروفيلم



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التوثيق الإلكتروني والميكروفيلم

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

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of Master Degree in **Cardiology**

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالَ

لَسْبَدَانِكَ لَا عِلْمَ لَنَا
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْعَظِيمُ

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List of Contents

Title	Page No.
List of Tables.....	5
List of Figures	6
List of Abbreviations.....	8
Introduction	- 1 -
Aim of the Work	3
Review of Literature	
▪ Myocardial Injury	4
▪ Bradycardia	8
▪ Permanent Pacemakers	20
Patients and Methods.....	41
Results.....	44
Discussion	57
Conclusion and Recommendations.....	63
Summary.....	64
References	66
Arabic Summary	

List of Tables

Table No.	Title	Page No.
Table 1:	Reasons for the elevation of cardiac troponin values because of myocardial injury	6
Table 2:	Guidelines for choice of pacemaker generator in selected indications for pacing.....	29
Table 3:	Descriptive data of age and sex.....	45
Table 4:	Descriptive of demographic data.....	45
Table 5:	Descriptive of procedural details.....	46
Table 6:	Descriptive of procedural details.....	46
Table 7:	Relationship between demographic data and troponin before	47
Table 8:	Relationship between demographic data and troponin after	48
Table 9:	Relationship between demographic data and troponin increase fold	49
Table 10:	Relationship of troponin in before, after and increase fold	49
Table 11:	Relationship between pacemaker implantation regardless site of RV lead in both groups and incidence of myocardial injury.....	50
Table 12:	Relationship between site of RV lead regarding troponin after.....	52
Table 13:	Relationship between site of RV lead regarding troponin increase fold	53
Table 14:	Correlation between number of screwing and extent of myocardial injury.....	54
Table 15:	Logistic regression analysis for predictors of Septum	56

List of Figures

Fig. No.	Title	Page No.
Figure 1:	Spectrum of myocardial injury, ranging from no injury to myocardial infarction	7
Figure 2:	Conduction system of the heart.....	10
Figure 3:	Parasympathetic and sympathetic innervation of the heart: anatomy	12
Figure 4:	An ECG strip showing sinus bradycardia reproduced from	15
Figure 5:	An ECG strip showing sinus pause.....	15
Figure 6:	Electrocardiogram of lead II showing first degree atrioventricular block with a prolonged PR interval of 0.30 sec	15
Figure 7:	Single lead electrocardiogram (ECG) showing Mobitz type I (Wenckebach) second degree AV block.....	16
Figure 8:	Single lead ECG showing Mobitz type II heart block with 2:1 conduction.....	16
Figure 9:	A single lead ECG showing complete heart block, notice the complete A-V dissociation	17
Figure 10:	Electrocardiographic (ECG) and electrophysiologic features of Mobitz type II second degree atrioventricular (AV) block.....	18
Figure 11:	Chest X-Ray of a patient with ILR implanted at lt pectoral region	19
Figure 12:	Frontal chest radiograph shows a single-chamber pacemaker with a single lead in the right ventricle (arrow)	21
Figure 13:	Frontal chest radiograph shows a dual-chamber pacemaker, which has leads in the right atrium (arrowhead) and right ventricle (arrow).....	22

List of Figures cont...

Fig. No.	Title	Page No.
Figure 14:	Frontal chest radiograph shows a biventricular pacemaker with one right ventricular lead (arrowhead) and one left ventricular lead (arrow).....	23
Figure 15:	Three different types of cardiac pacemakers.....	24
Figure 16:	Lead fixation	25
Figure 17:	Terminal connector pin.....	25
Figure 18:	Chronic SND management algorithm.....	32
Figure 19:	Management of bradycardia or pauses attributable to chronic atrioventricular block algorithm	36
Figure 20:	Management of conduction disorders algorithm	38
Figure 21:	Relationship between pacemaker implantation regardless site of RV lead in both groups and incidence of myocardial injury	51
Figure 22:	Relationship between site of RV lead regarding troponin after	52
Figure 23:	Relationship between site of RV lead regarding troponin increase fold	53
Figure 24:	Correlation between number of screwing and troponin after	55
Figure 25:	Correlation between number of screwing and troponin increase fold	55

List of Abbreviations

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

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*).