Incidence of Ventricular Arrhythmias in Patients with Fragmented QRS Post ST Elevated Myocardial Infarction

Thesis Submitted For Partial Fulfillment of

Master Degree in Cardiovascular Medicine

Bv

Ahmed Soliman Mohammed Youssif
(M.B.B.Ch)
Ain Shams University

Under Supervision of

Doctor/Hayam Mohamed El-Damanhory

Professor of Cardiovascular Medicine Faculty of Medicine Ain Shams University

Doctor / Osama Ali Dyab

Assistant Professor of Cardiovascular Medicine Faculty of Medicine Ain Shams University

Faculty of Medicine
Ain Shams University
2012



First and foremost, I thank Allah for helping and guiding me in accomplishing this work.

I would like to express my sincere gratitude to **Professor Dr.**Hyam El-Damanhory, Professor of Cardiology, Ain Shams University, for her great support and overwhelming kindness and guiding my way till this work came to light.

I must extend my warmest gratitude to **Dr. Osama Deiab**, Assistant professor of Cardiology, Ain Shams University, for his great help and faithful advice.

I would also like to thank my mother and my wife for supporting me in every step till this work has been accomplished. And I pray for my father as he is the one who showed me the way to succeed.

And I like to thank all my chiefs and colleagues in Egypt Air Hospital for their total support and back up all through our days together.



Ahmed Soliman Mohammed Youssif

List of contents

List of Abbreviations List of Tables List of Figures	i iii V
Introduction	1
Aim of the work	4
Review of literature	
Chapter 1: ST elevated myocardial infarction	5
Chapter 2: Ventricular Arrhythmias	28
Chapter 3 : QRS morphology changes in ST	
elevated myocardial infarction	47
Patients and methods	62
Results	72
Discussion	96
Summary	103
Conclusion	105
Recommendation	106
References	108
Master table	i
Arabic summary	

List of Abbreviations

ACC : American college of cardiology.

ACS : Acute coronary syndrome.
ADA : American diabetes association.
AHA : American heart association.

AIVR : Accelerated idioventricular rhythm

BBB : Bundle branch block.

CABG : Coronary arteries bypass grafting.

CAMIAT: Canadian Myocardial Infarction Amiodarone

Trial

CHD : Coronary artery disease.

CK : Creatine kinase.
CRP : C-reactive protein.

CTnI : Cardiac specific troponin I CTnT : Cardiac specific troponin T

EMIAT : European Myocardial Infarction Amiodarone

Trial

ESC : European society of cardiology.

ESVEM : Electrophysiologic Study Versus

Electrocardiographic Monitoring.

FH : Family history. fQRS : Fragmented QRS.

FU : Follow up.

HDL : High density lipoprotein.

ICD : Implantable cardioverter/defibrillatorIVCD : intraventricular conduction delay.

JNC : Joint national committee. LDL : Low-density lipoprotein. LMCA : Left main coronary artery.

LVEF : Left ventricular ejection fraction.

List of Abbreviations (Cont.)

MADIT II : Multicenter Automatic Defibrillator Implantation

Trial II

MI : Myocardial infarction.

MILIS: Multicenter Investigation of the Limitation of

Infarct Size.

MUSTT : Multicenter UnSustained Tachycardia.

NCEP-ATP: National cholesterol education program, adult

treatment panel.

NHLBIFHS: National heart, lung and blood institute

Framingham heart study

NSTE-ACS: Non ST elevation acute coronary syndrome.
 NSTEMI: Non ST elevation myocardial infarction
 NSVT: Non sustained ventricular tachycardia.
 STEMI: ST elevation myocardial infarction
 TIMI: Thrombolysis in myocardial infarction.

UA : Unstable angina.

VF : Ventricular fibrillation VT : Ventricular tachycardia.

WPW : Wolf Parkinson White syndrome

List of tables

Table no	Title	Page
1	Universal definition of myocardial	6
	infarction according to different	
	techniques	
2	Universal definition of myocardial infarction	6
3	Classification of myocardial infarction	7
4	Classification of ventricular arrhythmias	29
5	Ventricular arrhythmias mechanism and clinical features	34
6	Sensitivity and specificity of fQRS in detection of previous myocardial infarction.	57
7	General Demographic and echocardiographic and Follow up duration of all patients.	73
8	Difference between Group I and Group II.	75
9	Relation between LVEF and fQRS.	77
10	Relation between LVIDd and fQRS	78
11	Relation between LVIDs and fQRS	79
12	Description of VT incidence	84
13	Survival time between Group I (fQRS) and Group II	85
14	Comparison between patients with and without VT/VF	91
15	Multivariate analysis of the predictors of VT/VF	92

List of tables (Cont.)

Table no	Title	Page
16	Different relations of no. of leads with	93
	fQRS and other risk predictors	
17	Different relations of no. of akinetic	93
	segments and other risk predictors	

List of figures

Fig no	Title	Page
1	Annual rate of 1 st heart attacks by age, sex	11
	and race	
2	Difference between STEMI and Non-	13
	STEMI	
3	Hyperacute T wave and ST segment	22
	elevation in anterolateral myocardial	
	infarction	
4	Kinetics of release of CK-MB and	25
	Troponin in STEMI	
5	Multiform PVCs	30
6	Non sustained VT	30
7	Monomorphic sustained VT	31
8	Different shapes of VF	31
9	Torsade de Points	32
10	Sequence of depolarization and	48
	repolarization changes with acute STEMI	
11	Acute inferior STEMI with fQRS and	56
	thallium study	
12	Universal advanced life support (ALS)	68
	algorithm	
13	Relation between LVEF and fQRS	77
14	Relation between LVIDd and fQRS	78
15	Relation between LVIDs and fQRS	79
16	Relation between the presence of Akinesia	80
	and the presence of fQRS	
17	Relation between No. of akinetic	80
	segments and fQRS	
18	Comparison between viability between the	81
	two groups	

List of figures (Cont.)

Fig no	Title	Page
19	Kaplan Mayer survival curve that shows total mortality of all patients	82
20	Difference between the two groups in the incidence of VT/SCD	84
21	Kaplan Meyer survival curve between the two Groups as regard survival time and follow up (FU) duration	85
22	ECG with inferior and FQRS with cut section shows fragmentation seen down on the left and episode of sus. VT seen on the right	86
23	Picture of Non sust. VT on DC strip in Patient with fragmentation seen in V2-3	87
24	ECG shows inferior and high lateral fQRs with cut section on fragmentation and episode of sustained VT	88
25	FQRS in pericardial leads (V4-5) with episode of sustained VT	89
26	Picture of sustained VT with FQRS seen in inferior and anterolateral leads	90
27	linear correlation between no. of leads and LVEF by echocardiography and no. of leads with fQRS	94
28	Linear correlation between viability by thallium and no. of leads with fQRS	94
29	Thallium Scan at rest for patient no.13 that shows anterior and anteroseptal scar	95
30	ECG of the same patient no. 13 with fragmentation in V1-3	95

Introduction

Despite impressive advances in the diagnosis and management over the last four decades, ST segment elevation myocardial infarction (STEMI) continues to be a major public health problem in the industrialized world and had become increasingly important problem in developing countries. (*Luepker et al, 2003*).

ST segment elevation myocardial infarction (STEMI) is a fatal event in approximately one-third of patients, with about half of the deaths occurring within one hour of the event from ventricular tachyarrhythmias because ST segment elevation myocardial infarction can strike an individual during the most productive years, it may have profound deleterious psychosocial and economic ramifications (*Alpert et al.2000*).

Observations of both post-MI patients (*Volpi A et al.*, 1989) and survivors of cardiac arrest that occurred during the acute phase of transmural MI (*Liberthson RR et al.*, 1974) suggest that life-threatening ventricular tachyarrhythmias occurring during the first 24 to 48 hours of MI do not imply continuing risk over time.

Fragmented QRS complexes (fQRS) are defined as various RSR' patterns with or without Q waves on a 12-lead resting ECG. Based on their duration, they are sub-classified into fQRS complexes (QRS duration <120 ms) and fragmented wide-QRS complexes (f-wQRS; QRS duration >120 ms). Various RSR' patterns include an additional R wave (R') or notching in the nadir of the S wave, or the presence of >1 R' (fragmentation) in 2 contiguous leads, corresponding to a major coronary artery territory. (*Das et al.*, 2006)

Introduction and Aim of the work

Recently, fragmented QRS (fQRS) has been shown to predict cardiac events in several populations. Pathophysiologically, fragmented QRS has been associated with regional myocardial scar, which is believed to be a substrate for ventricular arrhythmia. (*Varriale et al, 1992*)

Clinically, fQRS has been associated with an increased mortality risk in patients with acute myocardial infarction. (*Das et al, 2009*)

Slurring and notching of the QRS complexes similar to fQRS has long been associated with post myocardial infarction (MI) cardiac scars. However, fQRS has been proven to be a sign of a scar from a remote MI as detected by single photon emission computed tomography (SPECT); it shows greater sensitivity in detecting regional perfusion abnormalities than Q waves alone and of increasing the sensitivity in detecting MI scars when combined with Q waves. (*Michael et al*, 2007)

In a population referred for stress testing, fQRS was associated with higher rates of cardiac events. (*Das et al*, 2007)

More recent studies, in patients with a wide QRS (≥ 120 ms), fQRS was found to be an independent predictor of all cause mortality. (*Das et al, 2008*)

In patients with left ventricular systolic dysfunction, fQRS has been shown to be a predictor of arrhythmic events but not mortality. (*Das et al, 2010*)

Various prior studies have suggested that the region of a myocardial scar is associated with alteration in QRS morphology, leading to a terminal conduction delay or a fragmentation of QRS complexes on the 12-lead ECG. (*Das et al, 2009*)

Introduction and Aim of the work

Fragmentation of QRS complexes is thought to be associated with ventricular tachyarrhythmias. Where the fragmentation of the QRS complex was quantified using a fragmentation index, was found a direct correlation between fQRS complexes and sustained, monomorphic ventricular tachycardia (V-tach) (*Oeff*, *G`odde et al,1997*)

Aim of the Work

To study the incidence of malignant ventricular arrhythmias (VT/VF/SCD) in post ST elevated myocardial infarction (STEMI) patients with fragmented QRS (fQRS) complex in resting electrocardiogram (ECG).

To study the presence or obscene of myocardial viability in areas with fQRS in the same group of patients compared to the other group with no fQRS.

Chapter (1) ST elevation myocardial infarction.

Definition

Acute myocardial infarction can be defined from a number of different perspectives related to clinical, electrocardiographic (ECG), biochemical, and pathological characteristics. The present guidelines pertain to patients presenting with ischemic symptoms and persistent ST-segment elevation on the ECG (STEMI). The great majority of these patients will show a typical rise of biomarkers of myocardial necrosis and progress to Q-wave myocardial infarction. (*Thygesen et al*, 2007)

The pathological diagnosis of myocardial infarction (MI) requires evidence of myocyte cell death as a consequence of prolonged ischemia. Characteristic findings include coagulation necrosis and contraction band necrosis, often with patchy areas of myocytolysis at the periphery of the infarct. During the acute phase of MI, the majority of myocyte loss in the infarct zone occurs via coagulation necrosis and proceeds to inflammation, phagocytosis of necrotic myocytes, and repair eventuating in scar formation. (*Topol text book*, 2007)

The clinical diagnosis of MI requires an integrated assessment of the history with some combination of indirect evidence of myocardial necrosis using biochemical, electrocardiographic, and imaging modalities. The sensitivity and specificity of the clinical tools for diagnosing MI vary considerably and change at varying times after the onset of the infarction. (*Topol text book*, 2007)

Aspects of Diagnosis of Myocardial Infarction by Different Techniques:

Table (1) Universal definition of myocardial infarction according to different techniques

TECHNIQUE	FEATURES		
Pathology	Myocardial cell death		
Biochemistry	Markers of myocardial cell death recovered from blood samples		
Electrocardiography	Evidence of myocardial ischemia (ST and T wave abnormalities); evidence of loss of electrically functioning cardiac tissue (Q waves)		
Imaging	Reduction or loss of tissue perfusion; cardiac wall motion abnormalities		

(Thygesen K et al., 2007).

Table (2) Universal definition of myocardial infarction according to criteria

accor uniş	g to criteria
Crite	eria for Acute, Evolving, or Recent MI
	er of the following criteria satisfies the diagnosis for acute, recent MI:
	Typical rise and/or fall of biochemical markers of myocardial s with at least one of the following:
a	Ischemic symptoms
b	Development of pathologic Q waves in the ECG
c seg	Electrocardiographic changes indicative of ischemia (ST- gment elevation or depression)
d nev	Imaging evidence of new loss of viable myocardium or w regional wall motion abnormality
2 I	Pathologic findings of an acute myocardial infarction

(Thygesen K et al., 2007)