Angiographic Slow/No Flow Predictors During Percutaneous Coronary Intervention For Acute Coronary Syndrome

Thesis

Submitted for Partial Fulfillment of Master Degree in Cardiology

Presented by

Ahmed Abdelmoneim El-Sayed Badra (M.B., B.Ch.)

Under Supervision of

Prof. Dr. Nabil Mahmoud Farag

Professor of Cardiology Faculty of Medicine - Ain Shams University

Dr. Ahmed Mohamed Onsy

Lecturer of Cardiology
Faculty of Medicine - Ain Shams University

Ain Shams University Faculty of Medicine 2012

List of Contents

Title	Page
♦ Introduction and Aim of the Work	1
• Review of Literature:	
• Chapter 1:	
Acute Coronary Syndrome	4
• Chapter 2:	
➤ Slow/No Reflow Phenomenon	49
♦ Patients and Methods	73
♦ Results	81
♦ Discussion	114
♦ Conclusion	129
♦ Recommendations	130
♦ Limitations	131
♦ Summary	132
♦ References	137
♦ Arabic Summary	

List of Table

Tab. No	Title	Page
Table (1)	ST-Segment Elevation or New or Presumably New LBBB: Evaluation for Reperfusion	20
Table (2)	Likelihood That Signs and Symptoms Represent As secondary to CAD	24
Table (3)	TIMI Risk Score for Patients with Unstable Angina and Non ST-Segment Elevation MI: Predictor Variables	26
Table (4)	Selection of Initial Treatment Strategy for Patients with Non-ST-Elevation ACS: Invasive Versus Conservative Strategy	31
Table (5)	Contraindications and Cautions for Fibrinolytic Use in STEMI from ACC/AHA 2004 Guideline Update	44
Table (6)	Incidence of Angiographic No-reflow in Various PCI Settings	50
Table (7)	Myocardial Blood Flow during Coronary Angiography	57
Table (8)	Predictors of Pathogenetic Components of No-reflow and Therapeutic Implication	66
Table (9)	Distribution of The Studied Cases As Regards Clinical and Demographic data	82
Table (10)	+ve Family History OF Study Patients	84
Table (11)	Dyslipidemia in Study Patients	85
Table (12)	Obesity in Study Patients	86
Table (13)	Hypertension in Study Patients	87
Table (14)	D.M in Study Patients	88
Table (15)	Smoking in Study Patients	89
Table (16)	Kidney Function Tests	90

List of Table (Cont.)

Tab. No	Title	Page
Table (17)	Liver Enzymes	91
Table (18)	Blood Glucose Level	
Table (19)	Lipid Profile in Blood	93
Table (20)	WBCs Count	94
Table (21)	Peak CK-Mb Level	95
Table (22)	Number of Diseased Vessels	96
Table (23)	Target Vessel for PCI	97
Table (24)	TIMI Flow Before PCI	98
Table (25)	MBG Grade Before PCI	99
Table (26)	Balloon to Artery Ratio	100
Table (27)	Number of Stents in Both Groups	101
Table (28)	Stent Length	102
Table (29)	Reference Vessel Diameter	103
Table (30)	Mean Diameter Stenosis Before PCI	104
Table (31)	Mean Diameter Stenosis After PCI	104
Table (32)	Minimal Lumen Diameter Before PCI	106
Table (33)	Minimal Lumen Diameter After PCI	106
Table (34)	Balloon Pressure	108
Table (34)	TIMI Flow After PCI	109
Table (36)	MBG Grade After PCI	110
Table (34)	Hospital Course of the Both Groups	111
Table (38)	MACE after 3 Months Follow-Up	111

List of Figures

Fig. No	Title	Page
Figure (1)	Acute Coronary Syndrome Algorithm	7
Figure (2)	Prehospital Fibrinolytic Checklist	14
Figure (3)	Multi-Factorial Causation of No-reflow and Slow-flow Following Percutaneous Coronary Intervention	51
Figure (4)	The No-reflow Phenomenon's Four Interacting Mechanisms	51
Figure (5)	Multiple Mechanisms Involved in the Pathogenesis of No-reflow that might be Targeted by Appropriate Therapy	61
Figure (6)	No-reflow As Assessed by Angiography (MBG), ECG, and Imaging Techniques	67
Figure (7)	Distribution of Study Patients	83
Figure (8)	Clinical Diagnosis of ACS in Study Patients	83
Figure (9)	+ve Family History in Both Groups	84
Figure (10)	Dyslipidemia in Both Groups	85
Figure (11)	Obesity in Both Groups	86
Figure (12)	Hypertension in Both Groups	87
Figure (13)	D.M in Both Groups	88
Figure (14)	Smoking in Both Groups	89
Figure (15)	Kidney Function Tests	90
Figure (16)	Liver Enzymes	91
Figure (17)	Blood Glucose Level	92
Figure (18)	Lipid Profile in Blood	93
Figure (19)	WBCs Count	94
Figure (20)	Peak CK-Mb	95

List of Figures (Cont.)

Fig. No	Title	Page
Figure (21)	Number of Diseased Coronary Arteries	96
Figure (22)	Target Vessel Analysis	
Figure (23)	Pre-PCI Angiographic TIMI Flow	
Figure (24)	Pre-PCI Angiographic MBG Grade	99
Figure (25)	Balloon to Vessel Ratio	100
Figure (26)	Number of Stents in Both Groups	101
Figure (27)	Stent Length	102
Figure (28)	Reference Vessel Diameter	103
Figure (29)	Mean Diameter Stenosis	105
Figure (30)	Minimal Lumen Diameter	107
Figure (31)	Balloon Pressure	108
Figure (32)	TIMI Flow After PCI	109
Figure (33)	MBG Grade After PCI	110
Figure (34)	MACE	112
Figure (35)	Right coronary Artery Injection Demonstrates Normal Epicardial Coronary Artery at Left Anterior Oblique View	113
Figure (36)	The Slow Progression of Dye in The Left Anterior Descending Coronary Artery at Right Anterior Oblique View	113
Figure (37)	No-reflow Occurring After Stenting of Mid-RCA	113

List of Abbreviations

A1c	Glycosylated hemoglobin
ABC	Airway breathing circulation
ACC	American college of cardiology
ACE	Angiotensine converting enzyme
ACS	Acute coronary syndrom
AHA	American heart association
AMI	Acute myocardial infarction
аРТТ	Activated partial thromboplastin time
ASA	Acetyle salicylis acid
ATP	Adenosine triphosphate
ATP	Adenosine triphosphate
AVM	Atriovenous malformation
BNP	B-Natriuretic peptide
CA++	Calcium
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
CCU	Coronary care unit
CHF	Congestive heart faliure
CK-MB	MB fraction of creatine kinase
CMR	Cardiac magnetic resonance
CPR	Cardiopulmonary resuscitation
CPU	Chest pain unit
CT	Computed tomography
cTFC	Corrected timi frame count
D	Diagonal artery
DBP	Diastolic blood pressure
ECG	Electrocardiogram
ED	Emergency department
EF	Ejection fraction
EMS	Emergency medical service
ESSENCE	Efficacy and safetyn of subcutaneous enoxaprin
ET	Endothelin
GIK	Glucose insulin potassium

List of Abbreviations (Cont.)

Gp	Glycoprotein receptors
GRACE	Global registry of acute coronary event
GREAT	Grampian region early anistreplase trial
Н	Hydrogen
HMG COA	Hydroxyl-methylglutaryl coenzyme a
IABP	Intra aortic balloon counter pulsation
INR	International normalized ratio
IR	Ischemia-reperfusion
IRA	Infarct-related artery
ISIS	International study infarct survival
IV	Intravenous access
K+	Potassium
LAD	Left anterior descending artery
LBBB	Left bundle branch block
LCX	Left circumflex artery
LDL	Low density lipo protein
LM	Left main artery
LMWH	Low molecular weight heparin
LOE	Level of evidence
LV	Left ventricle
LVF	Left ventricular failure
MACCE	Major adverse cardiac and cerebrovascular events
MACE	Major adverse cardiac events
MBG	Myocardial blush grade
MCE	Myocardial contrast echocardiography
MDCT	Multi detector computed tomography
MPS	Myocardial perfusion scintiography
m-PTP	Mitochondrial permeability transition pore
m-PTP	Mitochondrial permeability tension pore
MR	Mitral regurge
Na+	Sodium

List of Abbreviations (Cont.)

NSAIDS	Non steroidal anti-inflammatory drugs
N-STEMI	Non ST-elevation myocardial infarction
NVP	Negative predictive value
PAPP-A	Pregnancy associated plasma protein a
PDE-5	Phosphodistrase-5 inhibitors
PPCI	Primary percutaneous coronary intervention
RCA	Right coronary artery
RCT	Randomised controlled trial
rtPA	Tissue plasminogen activator
RVI	Right ventricular infarction
SBP	Systolic blood pressure
SC	Subcutaneous
SSA	Sulfosalycylic acid protein
protein	
STEMI	St-elevationmyocardial infarction
STR	ST-segment elevation resolution
TIA	Transient ischemic attack
TIMI	Thrombolysis in myocardial infarction
TxA2	Thromboxane-A2
UA	Unstable angina
UFH	Unfractionated heparin
URL	Upper reference limit
VF	Ventricular fibrillation
VT	Ventricular tachycardia

Acknowledgement

First and foremost, thanks to the gracious Allah. to Allah no words of thanks are sufficient.

I would like to express my deepest gratitude to **Prof. Dr. Nabil Farag**, Professor of Cardiology Department, Faculty of Medicine, Ain Shams University, for his masterful teaching, continuous support, enthusiastic encouragement and correction. I thank him beyond words can convey. I will always owe him so much for guiding me and teaching me the true meaning behind being a doctor. I hope I will be able to follow his footsteps.

I would like also to express my sincere gratitude to **Dr. Ahmed Onsy**, Lecturer of Cardiology, Faculty of Medicine, Ain Shams University, for his great help and faithful advice. His continuous encouragement was of great value and support to me. I have really learnt so much from his great support and stimulating views. His active, persistent guidance and overwhelming kindness have been of great help throughout this work. His precious advice, excellent supervision, his time and supreme effort are clear in every part of this work. He has taught me so much and to him I will be eternally grateful.

I also should thank My Seniors and Colleagues in my hospital Nasr City Insurance Hospital, Department of Cardiology for their kind help and continuous encouragement.

Special thanks to all patients, on whom and for whom this work has been achieved and without them it was nerve going to appear.

Last but definitely not least, I would like to add my love to My Parents and My Wife for always being there for me praying all the time, and for all the suffering and hardships I made them face. To them I owe my life.



Introduction

Ankle impingement is defined as a painful mechanical limitation of full ankle range of motion secondary to an osseous or soft-tissue abnormality (Sanders and Rathur, 2008) (Donovan and Rosenberg, 2010).

From both an anatomical and a clinical point of view these syndromes are classified as bone impingement, soft tissue impingement and entrapment neuropathy depending on which joint portion impinges on the others (Henderson and La Valette, 2004).

Soft tissue and osseous impingement syndromes of the ankle can be an important cause of chronic pain particularly in the professional athlete (*Rosenberg et al.*, 2000).

Depending on anatomical location and the structures involved, impingement syndromes are classified into anterolateral, anterior, posterior, posteromedial, and anteromedial types (*Robinson and White*, 2002) (*Donovan and Rosenberg*, 2010).

These conditions arise from initial ankle injuries which in the subacute or chronic situation lead to development of abnormal osseous and soft tissue thickening within the ankle joint (*Robinson and White*,2002).

Despite conventional radiography being usually the first imaging technique performed to assess any potential bone abnormalities, soft tissue affection usually escape and it has

disadvantages of improper cartilaginous, assessment of ligamentous and tendinous lesions (Dunfee et al., 2002).

US is a rapid, widely available and inexpensive modality for evaluation of pathologic conditions of the ankle but it need familiarity with normal anatomic variants as well as with sonographic artifacts and common pitfalls to increase the diagnostic accuracy of ankle and foot sonography (Fessel et al, *1999*).

CT and isotope bone scanning have been largely superseded by MR imaging (*Robinson and White*, 2002).

Magnetic resonance imaging has opened new horizon in diagnosis, hence treatment of most of ankle joint lesions. It enabled the detection of a wide varieties of bone changes and variety of soft tissue disorders such as ligaments, tendons and synovial membranes (Rosenberg et al., 2000).

MRI is particularly suited for evaluation of the complex bone and soft tissue anatomy of the foot and ankle because of its superior soft tissue contrast and the ability to image in multiple planes. In addition new fast scan techniques provide improved efficiency and allow dynamic studies to be performed. MR arthrography technique has improved significantly in recent years resulting in more routine use of this technique (Rosenberg et al., *2000*).



Aim of the Work

The aim of this study is to evaluate the role of magnetic resonance imaging in assessment of impingement syndromes of the ankle.



Anatomy of the Ankle Joint

The ankle is approximately a uniaxial hinge joint. It is composed of the lower end of the tibia and its medial malleolus, together with the lateral malleolus of the fibula and inferior transverse tibiofibular ligament, form a deep recess (mortise) for the body of the talus (*Davies*, 2005).

Anatomy of the ankle joint consists of:

I. Osseous Anatomy, include:

- 1. The distal end of tibia.
- 2. The distal end of fibula.
- 3. The talus.

II. Soft Tissue Anatomy, include:

- 1. Fibrous capsule.
- 2. Ligaments.
- 3. Retinacula.
- 4. The tendons of the ankle joint.

I. Osseous Anatomy:

1. The Distal End of Tibia

The lower end of the tibia is rectangular in cross section. *Anteriorly*, the bone is crossed by (from medial to lateral) tibialis anterior, extensor hallucis longus and extensor digitorum longus tendons, as well as anterior tibial neurovascular bundle.



Posteriorly, there is a groove behind the medial malleolus for tendon of the tibialis posterior (Fig. 1) (Sinnatamby, 2000).

2. The Distal End of Fibula

The distal end of the fibula or lateral malleolus, projects distally and posteriorly relative to the medial malleolus (Fig. 1&2) (Davies, 2005).

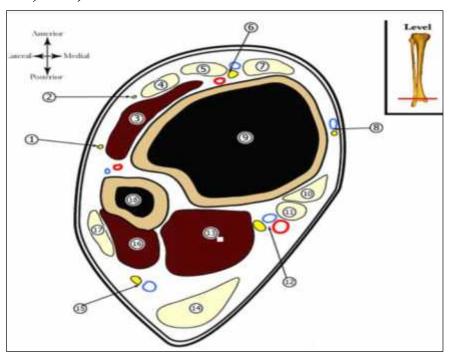


Fig. (1): Transverse section through the ankle joint (A) Intermediate Dorsal Cutaneous branch of superficial peroneal nerve 2.Medial Dorsal Cutaneous branch of superficial peroneal nerve 3. Peroneus tertius 4. Extensor Digitorum Longus 5. Extensor Hallucis Longus 6. Deep Peroneal Nerve and Anterior Tibial Vessels 7. Tibialis Anterior 8. Great saphenous vein and saphenous nerve9. Tibia 10. Tibialis Posterior11. Flexor digitorum longus (FDL) 12. Tibial Nerve and Posterior Tibial Vessels 13. Flexor Hallucis Longus 14. Achilles tendon 15. Sural Nerve and Lesser Saphenous Vein 16. Peroneus brevis 17. Peroneus longus 18. Fibula (Davies, 2005).