

Plasminogen Activator Inhibitor-1 Antigen in Acute Myocardial Infarction Patients

Thesis

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Abstract

Background: Coronary heart disease (CHD) and myocardial infarction (MI) have a significant impact on morbidity and mortality in developed countries. Coronary artery disease results from progressive atherosclerotic plaque development and subsequent thrombus formation.

Aims: To determine plasminogen activator inhibitor -1 antigen level in acute myocardial infarction patients and to evaluate its prognostic impact.

Subjects and Methods: This study was conducted on 30 patients, at the Cardiac intensive care unit of Ain-Shams University Hospitals and the National Heart institute.

Results: AMI who are candidates for PCI '*Group 1*' and 30 healthy individuals as control group '*Group 2*' of matched age and sex. Informed consent was taken from both groups in order to use their data in the study.

Conclusion: PAI-1 increase during the acute phase of AMI. Also PAI-1 level was found to increase in diabetic MI patients dramatically more than non-diabetic MI patients. These levels were found to decrease 6 weeks after MI but not reaching the level of control group, indicating that these patients are still in a hypercoagulable state. PAI-1 could be considered a poor prognostic marker in AMI as it is associated with recurrence of MI.

Recommendations Further clinical studies on wider scale and larger number of cases are recommended. Serial evaluation of PAI-1 level within first 24 hours to study its course.

Keywords: Plasminogen Activator, Inhibitor-1 Antigen, Acute Myocardial Infarction.



“ No! Worship Allah and be among the thankful.” (AzZumar; 66)

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List of Abbreviations

ACCF	The American College of Cardiology Foundation
AHA	American heart association
AHA/NHLBI	American Heart Association/ National Heart, Lung, and Blood Institute
AMI	Acute Myocardial Infarction
BNP	B-type natriuretic peptide
BP	Blood pressure
CAD	Coronary artery disease
CHD	Coronary heart disease
CK	Creatine kinase
CK-MB	MB iso-enzyme of CK
CRP	C-reactive protein
CVDs	Cardiovascular diseases
DIC	Disseminated intravascular coagulation
DM	Diabetes Mellitus
ECG	Electrocardiography
ECM	Extracellular matrix
ELISA	Enzyme Linked Immuno-Sorbent Assay method
ESC	The European Society of Cardiology
ESC/ACC	European Society of radiology/ American College of Cardiology
FMC	First Medical Contact
H&E	Hematoxylin and eosin

List of Abbreviations

HDL	High-density lipoprotein cholesterol
HIV	Human immunodeficiency virus infection
IL	Interleukin
LAD	Left anterior descending
LDH	Lactate dehydrogenase
LDL-C	Low-density lipoprotein cholesterol
MCP-1	Matrix cellular protein-1
MI	Myocardial Infarction
MMP	Matrix metalloproteinases
NCEP-ATP III	National Cholesterol Education Program and Adult Treatment Panel III
NSTEMI	Non-ST-elevation Myocardial Infarction
PAI-1	Plasminogen Activator Inhibitor-1
PCI	Percutaneous coronary intervention
RCL	Reactive center loop
SERPIN	Serine protease inhibitor
STEMI	ST-elevation Myocardial Infarction
tn	Troponin
TNF	Tumor necrosis factor
t-PA	Tissue-type plasminogen activator
UA	Unstable angina
u-PA	Urokinase-type plasminogen activator
VLDL	Very low-density lipoprotein
WHO	World health organization

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Introduction

Coronary heart disease (CHD) and myocardial infarction (MI) have a significant impact on morbidity and mortality in developed countries (**Gruzdeva et al., 2013**).

Coronary artery disease results from progressive atherosclerotic plaque development and subsequent thrombus formation (**Soeki et al., 2000**). Plaque disruption and thrombus formation in coronary arteries lead to variable degrees of luminal obstruction to the blood flow and can present clinically as unstable angina (UA) or acute myocardial infarction (AMI) and lead to sudden death (**Fuster and Lewis, 1994**).

Primary percutaneous coronary intervention (PCI) is a reperfusion strategy used in patients with acute ST-segment elevation myocardial infarction (STEMI), to prevent progression of myocardial necrosis (**Seifollah et al., 2015**). Primary PCI performed in a timely fashion [< 90 min of first medical contact (FMC)-device time in PCI-capable hospital, and < 120 min of FMC-device time in non-PCI-capable hospital] is the preferred strategy for the treatment of STEMI patients with symptom onset < 12 h (**Feng et al., 2015**).