

**CLINICAL SIGNIFICANCE OF STRESS INDUCED
TRANSIENT LEFT VENTRICULAR DILATATION DURING
MYOCARDIAL PERFUSION IMAGING IN PATIENTS WITH
NORMAL OR MILDLY POSITIVE MYOCARDIAL
PERFUSION SCANS AND NEGATIVE STRESS ECG**

Thesis

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بسم الله الرحمن الرحيم

اقرأ باسم ربك
الذي خلق ﴿١﴾ خلق
الإنسان من
علق ﴿٢﴾ اقرأ
وربك
الأكرم ﴿٣﴾ الذي
علم بالقلم ﴿٤﴾ علم
الإنسان ما لم
يعلم ﴿٥﴾

صدق الله العظيم

سورة العلق



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List of Contents

Title	Page No.
<i>Introduction</i>	<i>1</i>
<i>Aim of the Work</i>	<i>4</i>
<i>Review of Literature</i>	
○ <i>Assessment of Myocardial Ischemia.....</i>	<i>5</i>
○ <i>Causes of transient dilatation of the Left Ventricle during Myocardial Perfusion Imaging</i>	<i>34</i>
<i>Patients and Methods.....</i>	<i>43</i>
<i>Results</i>	<i>53</i>
<i>Discussion.....</i>	<i>68</i>
<i>Summary</i>	<i>82</i>
<i>Conclusion.....</i>	<i>86</i>
<i>Recommendations</i>	<i>87</i>
<i>References</i>	<i>88</i>
<i>Arabic Summary</i>	

List of Tables

Table No.	Title	Page No.
Table (1):	Summed stress score according to Cedars-Sinai scoring system.	50
Table (2):	Age and sex of the studied patients	54
Table (3):	Risk factors of the studied patients.....	56
Table (4):	Clinical characteristics of the studied patients	57
Table (5):	Number of vessels affected in patients with CAD.	59
Table (6):	Vessels affected in patients with CAD.....	61
Table (7):	Number of risk factors in reference to coronary angiography.....	64
Table (8):	Risk factors in reference to coronary angiography.....	65

List of Figures

Fig. No.	Title	Page No.
Fig. (1):	Short –axis summed images show dilated cavity on stress due to hypoperfusion of the subendocardium.....	20
Fig. (2):	Transient ischemic dilatation predicts extensive CAD	22
Fig. (3):	TID in patient with hypertension normal myocardial perfusion imaging and normal coronary angiography.	36
Fig. (4):	TID in patient with hypertension normal myocardial perfusion imaging and coronary artery disease.....	38
Fig. (5):	TID in patient with myocardial perfusion defect and coronary artery disease.....	41
Fig. (6):	Classification of the 17 segments of the left ventricle in bull's-eye view, echocardiographic parasternal short axis slices, and apical 4- and 2-chamber views	49
Fig. (7):	Sex of the studied patients.....	54
Fig. (8):	Clinical characteristics of the studied patients	57
Fig. (9):	Number of vessels affected in patients with CAD.	59
Fig. (10):	Vessels affected in patients with CAD.....	61
Fig. (11):	Risk factors in reference to coronary anatomy.	67

List of Figures (Cont...)

Fig. No.	Title	Page No.
Fig. (12):	Stress technetium for patient number 7. Showing minimal defect in the anterior wall during stress images.	77
Fig. (13):	Coronary angiography for patient number 7. Subtotal proximal 90% lesion in D1 with normal other coronaries.	77
Fig. (14):	Stress technetium for patient number 1. Normal stress MPI	79
Fig. (15):	Coronary angiography for patient number 1. 90% proximal LAD and 90% proximal PDA.....	79
Fig. (16):	Stress technetium for patient number 4. Normal MPI.....	80
Fig. (17):	Coronary angiography for patient number 4. Normal coronary angiography.....	80

List of Abbreviations

Abb.	Full term
BP	Blood pressure
CAD	Coronary artery disease
D	Diagonal
DM	Diabetes mellitus
E+SF	Ectasia + slow flow
ECG	Electrocardiography
HTN	Hypertension
IHD	Ischemic heart disease
IVS	Inter-ventricular septum
LAD	Left anterior descending
LCX	Left circumflex
LM	Left main
LV	Left ventricle
LVEDD	Left ventricular end diastolic diameter
LVESD	Left ventricular end systolic diameter
LVH	Left ventricular hypertrophy
MPI	Myocardial perfusion imaging
OM	Obtuse marginal
PDA	Posterior descending artery
PET	Positron emission tomography
PL	Postero-lateral branch
RCA	Right coronary artery
SPECT	Single photon emission computerized tomography
Tc	Technitium
TID	Transient ischemic dilatation

INTRODUCTION

Myocardial perfusion imaging sometimes produce false negative results in patients with multi-vessel CAD .when multiple arteries are occluded to an equivalent degree i.e (balanced hypo-perfusion) the perfusion defect that occur during stress images may be generally uniform in severity throughout the myocardium. As a result the radiotracer uptake will be also uniform, leading to false negative examination (*Duarte et al., 2003; Burerell and Mac Donald, 2006*).

A finding of transient ischemic dilatation (TID) on stress MPI (either exercise or pharmacologic) is valuable in such situation as it enables clinicians to identify patients with multi-vessel CAD despite otherwise normal MPI (*Kinoshita et al., 2002; Abidov et al., 2003*).

It can also be used for risk stratification by serving as a clinical useful marker for severe and extensive disease (*Mazzanti et al., 1996*) and may be a predictor of future cardiac events (*Abidov et al., 2003; McClelan et al., 1997*).

TID is found to be present when the diameter of the left ventricle (LV) appears larger in post stress images as compared with rest images. It is almost likely due to stress induced sub-endocardial ischemia (*Doukky and Hendel, 2004*). Hypo-perfusion of the sub- endocardial region causes LV's enlarged appearance rather than actual dilatation.

TID may also results from physical dilatation(true dilatation of the LV through the cardiac cycle, also caused by ischemia) (*McLaughlin and Danias 2002; Wiess et al., 1987*) or from systolic dysfunction (in which the LV stunning decreases the ejection fraction at stress, thereby increasing the ventricular size) (*Hung et al., 2005*) but even if the underlying cause of TID is not completely understood, the finding of TID has been shown to be a clinically useful marker for severe and extreme CAD (*Mazzanti et al., 1996*).

Abnormal TID has been correlated better with the presence of multi- vessel CAD that have multiple perfusion abnormalities and visual summed stress score (*Mazzanti et al., 1996; Weiss et al., 1987; Abidov et al., 2003*). It's a more sensitive and specific marker for multi-vessel CAD than other common markers during stress testing, such as patient limited ability to exercise and severe ST- depression (more than 2 mm) (*Wiess et al., 1987*) and patients with TID are more likely to have a cardiac event (non fatal myocardial infarction or cardiac death) than those without TID (*McClellan et al., 1997*).

TID is relatively easy to detect, The size of LV is measured at stress and again at rest often with automated software and a ratio of the two measurements is computed, TID occurs when the ratio falls above a normal limit that has ranged from 1.12 to 1.36 in various studies (*Mazzanti et al., 1996; Wiess et al., 1987; Abidov et al., 2003*).

Certain patients characteristics can signal a higher likelihood of TID, like HTN, DM. and old age.

Sometimes transient LV dilatation occurs in the absence of chest pain and ECG changes during the stress ECG preceeding the nuclear imaging, with normal MPI, or mild ischemia at a single coronary territory that can't explain this TID.

AIM OF THE WORK

The aim of this study was to assess the clinical significance of the TID that occurs during stress MPI in patient with negative stress ECG and normal or mildly positive myocardial perfusion scans.

ASSESSMENT OF MYOCARDIAL ISCHEMIA

1- Stress ECG

Stress testing with electrocardiogram (ECG) monitoring is the original form of noninvasive cardiac testing. In the last decade it has become clear that ST-segment changes during stress have low sensitivity and specificity in the evaluation of coronary artery disease, and are poor predictors of risk. This may be partially due to the fact that stable obstructive plaques, which typically result in exercise mediated ischemia, are less relevant to myocardial infarction and sudden cardiac death than non obstructive plaques. Although the bulk of obstructive coronary artery disease screening has now shifted to echocardiography or nuclear assessment immediately following pharmacologic or exercise testing, evaluation of functional capacity, heart rate changes, burden of ectopy, and dynamic ECG changes during after exercise have emerged as powerful prognostic indicators. As such, the main uses of exercise ECG testing should be evaluation of prognosis and a gateway to other imaging modalities (*Kligfield et al., 2006*).

Exercise stress testing can be accomplished by either treadmill or cycle ergometry, though treadmills are predominantly used. There are several standard treadmill protocols that can be customized for patients to reach the endpoint of 85% to 100% maximum predicted heart rate. Among them, the Bruce protocol is the most popular. This

protocol consists of a maximum of eight 3-minute stages with incremental increases in speed and incline (*McElroy et al., 1992*).

The patient is continuously monitored via ECG while heart rate and blood pressure are closely observed. Testing is stopped when the patient reaches the target heart rate (a percentage of the predicted maximum heart rate). Indications to stop testing (other than reaching the target heart rate) are as follows: decrease in blood pressure of more than 10 mm Hg from baseline, sustained ventricular tachycardia, ST elevation greater than or equal to 1 mm in leads without q waves, moderate to severe angina, or severe dizziness (*Detry et al., 1977*).

Horizontal or downsloping ST depression greater than or equal to 1 mm for at least 60–80 ms is commonly considered a positive stress test. However, the capacity of a stress test to localize ischemia based on leads demonstrating ischemic changes is poor. An inappropriately slow heart rate or a decrease in blood pressure compared with baseline is also indicative of ischemia. (*Gibbons et al., 1997*).

Patients with equivocal or non-diagnostic stress test results may be recommended for radionuclide or echocardiographic stress testing before a final decision regarding coronary angiography is made. Baseline ECG abnormalities make interpretation of exercise ECG difficult,