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Assessment of Silent Myocardial ischemia after primary PCI in anterior STEMI patients

Cardiology

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ABSTRACT

Background: Silent myocardial ischemia represents an important public health problem and to date the extent of this phenomenon is largely unknown. Since it was first recognized as a relevant part of the spectrum of ischemic heart disease in the early 20th century.

Aim of The Work: To estimate the prevalence and identify the main causes of silent myocardial ischemia in patients with anterior STEMI treated using primary PCI.

Patients and Methods: This prospective cross sectional observational study included 50 patients from the attendants of the cardiology department at Al Shiekh Zayed specialized hospital who were admitted with Anterior ST elevation myocardial infarction (STEMI) and treated with primary percutaneous intervention (1ry PCI) with complete revascularization of all lesions and have no residual coronary stenosis. Patients had exercise treadmill test (TMT) after 6 months of the event and classified into two subgroups, Group (A) included patients with TMT Positive, and Group (B) included patients with TMT Negative.

Results: Study findings revealed that 14 patients (28%) had a positive treadmill test at 6 months follow up and 36 patients (72%) were negative. From the 14 patients with positive treadmill test, (78.6%) were males and (21.4%) were females. In patients with negative exercise treadmill test, mean age was (62.50 ± 4.01) years, 12 patients (85.7%) had DM (p value <0.001), 8 patients (57.1%) were hypertensives, 13 patients (92.9 %) had Dyslipidemia, 3 patients (21.4%) were Smokers, 3 patients (21.4%) were Obese, zero patients (0%) had no Family-history of CAD.

Conclusion: SMI was detected more-than quarter of study population which proves that CAD is a major risk factor for further and even silent myocardial ischemia. Older age, diabetes mellitus and dyslipidemia are risk factors for silent myocardial ischemia.

Keywords: *Electrocardiogram; Percutaneous coronary intervention; Silent myocardial ischemia; ST-Elevation Myocardial Infraction; Treadmill testing.*

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INTRODUCTION

Silent myocardial ischemia (SMI) is the presence of objective evidence of ischemia without angina or equivalent symptoms. [1]

The objective evidence of SMI can be obtained from non-invasive diagnostic tests (exercise stress test, Holter-ECG, myocardial perfusion imaging, Echo-stress) or more recently by invasive tests during catheterization with the use of coronary pressure. [1]

Episodes of asymptomatic ischemia have been estimated to occur in a percentage ranging from 25% to 50% of the patients with ischemic heart disease and that this occurs, with respect to symptomatic episodes, with a ratio greater than 20:1. [2]

In silent myocardial ischemia (SMI), there are violations of perfusion, metabolism, function and electrical activity of the myocardium, which are not accompanied by clinical manifestations. [3]

SMI can be an independent form of coronary heart disease or be combined with other forms of CHD. In the presence of SMI, the risk of sudden cardiac death increases 10 times, cardiac arrhythmias - 2 times, myocardial infarction and congestive heart failure - 1-1.5 times. [4]

This study purposes to determine the incidence and to estimate the potential main causes of silent myocardial ischemia in anterior STEMI patient treated with PCI.

PATIENTS AND METHODS

This prospective cross sectional observational study included 50 patients from the attendants at Al Shiekh Zayed specialized hospital admitted with Anterior ST elevation myocardial infarction (STEMI) and received primary percutaneous intervention (1ry PCI)

with complete revascularization of all lesions and have no residual coronary stenosis.

All patients included in this study were presented within 12 hours from symptoms onset and had an ECG showing persistent ST-segment elevation myocardial infarction precisely anterior STEMI and treated with primary PCI.

STEMI was defined as ST elevation at the J point, in at least 2 contiguous leads, ≥ 2 mm (0.2 mV) in men ≥ 40 years, 2.5 mm (0.25 mV) in men < 40 years or ≥1.5 mm (0.15 mV) in women in leads V2–V3 and/or ≥ 1 mm (0.1mV) in other contiguous chest leads or the limb leads.

Patients managed by any method other than primary PCI as thrombolysis or coronary artery bypass grafting (CABG) surgery were excluded. We also excluded Patients with residual -angiographically-significant coronary stenosis after 1ry PCI, Patients with symptoms suggestive of CAD or documented ischemia at follow up and Patients with ECG abnormalities that interfere with the diagnosis of ischemia include: [184] Wolff-Parkinson-White pattern, Ventricular paced rhythm, Left Bundle Branch Block, Greater than 1 mm ST depression at rest and Left Ventricular Hypertrophy with ST-T abnormalities.

All enrolled patients were subjected to; medical history for diabetes mellitus and hypertension or if the patient currently on anti-diabetic and hypertensive medications. Family history of premature CAD, Smoking, Dyslipidemia, and Obesity.

Full assessment for general condition including; blood pressure, heart rate and rhythm, signs of pulmonary venous congestion. Additional heart sounds or murmurs. Lower limb edema, ascites, hepatomegaly and other signs of systemic venous congestion.

12 lead Electrocardiogram (ECG) was obtained at admission and follow up (1,3 and 6 months from discharge).

Laboratory investigations included serum cardiac biomarkers (troponin I, creatine kinase and myocardial band of creatine kinase. Lipid profile: at discharge and follow up (3 and 6 months from discharge).

Transthoracic Echocardiography (TTE) was done for all included patients before discharge and at follow up (1, 3 and 6 months after the event) using a Toshiba Ultrasound Machine according to the recommendations of American Society of Echocardiography to detect left ventricle size, left atrium size, left ventricular ejection fraction, any wall motion abnormalities or ischemic complications such as (LV systolic dysfunction, LV aneurysm, LV thrombus, Secondary mitral valve regurgitation).

Coronary Angiography and Primary PCI within time window suggested by current guidelines (if symptoms of ischaemia are less than 12 hours duration and persistently elevated ST segment). Coronary artery Stenosis is mild if narrowing is less than 50%, moderate stenosis is between 50% and 70%, and severe or significant for a stenosis of 70% or more. Non-culprit stenosis was considered angio-graphically significant if they cause less than 70% luminal stenosis of the vessel on naked eye estimation. Complete revazcularization of significant stenosis in non-Infarction Related arteries was done for all patients with multi-vessel disease.

Treadmill Stress Test (TMT) was done after 6 months of the event according to Bruce Protocol provided that patients had no symptoms suggestive of myocardial ischemia and no new documented ischemic events. The interpretation of the exercise ECG focuses on the presence or absence of ischemic ECG changes along with any induced arrhythmias. TMT was considered "positive" for ischemia when there is ≥1 mm horizontal or downsloping ST segment depression in one or more leads that persists at 80 milliseconds after the J point. Depression of the J point (end of the QRS and beginning of the ST segment) is normally seen with increasing heart rates during exercise.it is probably related to atrial repolarization that extends through the QRS rather than myocardial ischemia. In this circumstance, the ST segment rapidly (within 80 milliseconds) returns to baseline. Patients were classified into 2 groups, (A) patients with TMT Positive, and (B) patients with TMT Negative.

RESULTS

Mean age was 54.54 ± 8.96 years. Men represented 78% (39 patients) of our study population while women represented 22% (11 patients). 44% (22 patients) were hypertensive while 40% (20 patients) were diabetic and 38% (19 patients) were smokers. From the 50 patients,14 patients (28%)

had a positive exercise treadmill test (TMT) at 6 months follow up and 36 patients (72%) were negative. All patients were completely asymptomatic during period of follow up. In patients with positive TMT, 3 patients (21.4%) were Smoker while in those with negative TMT, only 16 patient

(44.4%) weren't Smokers which was statistically insignificant. (p value 0.132). In patients with positive TMT, 3 patients (21.4%) were Obese while in those with negative TMT, only 10 patients (27.8%) weren't Obese which was statistically insignificant. (p value p=0.734). In patients with positive TMT at follow up, zero patients (0%) had no Family history for CAD while in those with negative TMT, 7 patients (19.4%) had Family history for CAD which is statistically insignificant. (p value p=0.734) Figure (1).

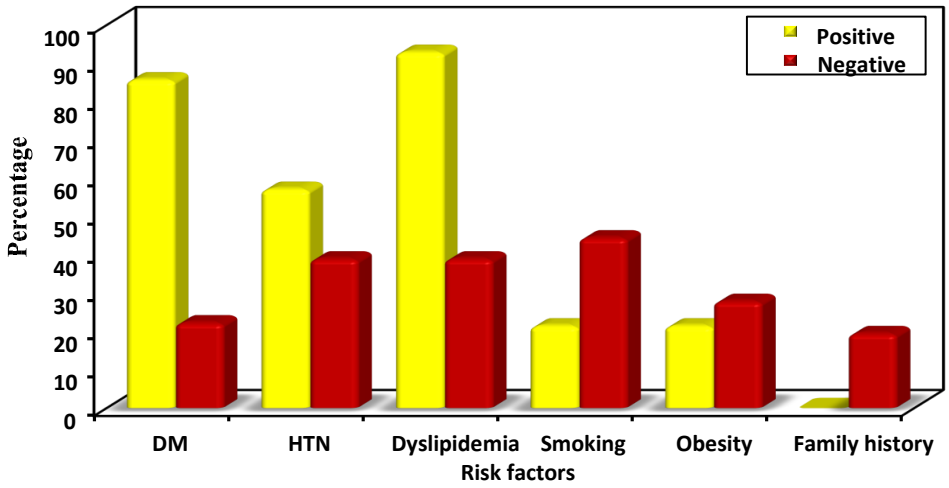

Fig. 1: the two studied groups were compared according to risk factors

From the 14 patients with positive TMT, 11 patients were males (78.6%) and 3 females (21.4%). while in those 36 patients with negative TMT, 28 patients were males (77.8%) and 8 females (22.2%). but this difference was not significant statistically. (p= 1.000). In patients with positive TMT, mean age was (62.50 ± 4.01) years, while in those with negative TMT mean age was (51.44 ± 8.44) years which has statistical significance. (p value <0.001). Table (1). According to risk factors; In patients with positive TMT, 12 patients (85.7%) had DM while in those with negative TMT, only 8 patients (22.2%) had DM which has statistical significance. (p value <0.001). In patients with positive TMT at follow up, 8 patients (57.1%) were hypertensives while in those with negative TMT, only 14 patients (38.9%) weren't hypertensives which was statistically insignificant. (p value 0.243). In patients with positive TMT at follow up, 13 patients (92.9 %) had Dyslipidemia while in those with negative TMT, only 14 patients (38.9%) were not dyslipidemics which has statistical significance. (p value <0.001) Table (2).

	Total (n = 50)		Exercise ECG				p
			Positive (n = 14)		Negative (n = 36)		
	No.	%	No.	%	No.	%	
Sex Male	39	78.0	11	78.6	28	77.8	FEp=
Female	11	22.0	3	21.4	8	22.2	1.000
Age (years) Min. – Max.	33– 71		57 – 71		33– 66		
Mean ± SD.	54.54 ± 8.96		62.50 ± 4.01		51.44 ± 8.44		<0.001*

Table 1: Comparison-between the included groups according to demographic data

Risk factors	Total (n = 50)		Exercise ECG				p
			Positive (n = 14)		Negative (n = 36)		
	No.	%	No.	%	No.	%	
DM	20	40.0	12	85.7	8	22.2	<0.001*
HTN	22	44.0	8	57.1	14	38.9	0.243
Dyslipidemia	27	54.0	13	92.9	14	38.9	0.001*
Smoking	19	38.0	3	21.4	16	44.4	0.132
Obesity	13	26.0	3	21.4	10	27.8	FEp=0.734
Family history	7	14.0	0	0.0	7	19.4	FEp=0.16 9

Table 2: the two studied groups were compared according to risk factors

Resting TTE echo	Total (n = 50)		Exercise ECG				p
			Positive (n = 14)		Negative (n = 36)		
	No.	%	No.	%	No.	%	
WMA							
No	42	84.0	11	78.6	31	86.1	^{FE}p=
Yes	8	16.0	3	21.4	5	13.9	0.670
LVH							
No	46	92.0	11	78.6	35	97.2	^{FE}p=
Yes	4	8.0	3	21.4	1	2.8	0.061
EF%							
Min. – Max.	47.0 – 69.0		48.0 – 64.0		47.0 – 69.0		0.261
Mean ± SD.	57.42 ± 4.69		56.21 ± 4.96		57.89 ± 4.56		
Mean ± SD.	5.03 ± 0.33		4.94 ± 0.32		5.06 ± 0.33		

Table 3: Comparison between the two studied groups according to resting TTE Echo

Clinical examination	Total (n = 50)	Exercise ECG		p
		Positive (n = 14)	Negative (n = 36)	
SBP (mmHg)				
Min. – Max.	100.0 – 150.0	110.0 – 150.0	100.0 – 150.0	0.055
Mean ± SD.	124.80 ± 11.99	130.0 ± 12.40	122.78 ± 11.37	
DBP (mmHg)				
Min. – Max.	60.0 – 100.0	70.0 – 100.0	60.0 – 100.0	0.710
Mean ± SD.	83.60 ± 8.02	84.29 ± 9.38	83.33 ± 7.56	

Table 4: Comparison between the two studied groups according to clinical examination:

LABS	Total (n = 50)	Exercise ECG		p
		Positive (n = 14)	Negative (n = 36)	
TC (mg/dl)				
Min. – Max.	112.0 – 389.0	180.0 – 365.0	112.0 – 389.0	0.035*
Mean ± SD.	218.4 ± 73.30	242.6 ± 67.87	209.0 ± 74.07	
TG (mg/dl)				
Min. – Max.	98.0 – 343.0	130.0 – 343.0	98.0 – 302.0	0.148
Mean ± SD.	165.6 ± 57.63	190.2 ± 82.50	156.1 ± 42.25	
LDL (mg/dl)				
Min. – Max.	67.0 – 273.0	88.0 – 273.0	67.0 – 211.0	<0.001*
Mean ± SD.	118.6 ± 53.69	163.9 ± 62.03	101.0 ± 38.33	
HDL (mg/dl)				
Min. – Max.	23.0 – 59.0	23.0 – 49.0	31.0 – 59.0	0.001*
Mean ± SD.	43.48 ± 7.88	37.71 ± 8.16	45.72 ± 6.61	
TCK (IU/L)				
Min. – Max.	844.0 – 2620.0	844.0 – 2620.0	850.0 – 1980.0	0.005*
Mean ± SD.	1276.2 ± 409.5	1586.4 ± 506.1	1155.6 ± 293.7	
CK-MB (IU/L)				
Min. – Max.	158.0 – 380.0	158.0 – 379.0	176.0 – 380.0	0.425
Mean ± SD.	273.6 ± 60.36	260.8 ± 74.22	278.6 ± 54.44	

Table 5: Comparison between the two studied groups according to LAB results:

PPCI	Total (n = 50)		Exercise ECG				p
			Positive (n = 14)		Negative (n = 36)		
	No.	%	No.	%	No.	%	
Stenting							
Direct	18	36.0	4	28.6	14	38.9	0.495
Predilation	32	64.0	10	71.4	22	61.1	
Number of stent/s							
Min. – Max.	1.0 – 3.0		1.0 – 2.0		1.0 – 3.0		0.070
Mean ± SD.	1.47 ± 0.62		1.21 ± 0.43		1.57 ± 0.65		
Median (IQR)	1.0 (1.0 – 2.0)		1.0 (1.0 – 1.0)		1.0 (1.0 – 2.0)		
Type of stent/s							
Biomatrix flex	3	6.0	0	0.0	3	8.3	$^{MC}p=$
Ultimaster	10	20.0	2	14.3	8	22.2	0.727
Ultimaster / Biomatrix flex	1	2.0	0	0.0	1	2.8	
Promus premier	1	2.0	1	7.1	0	0.0	
Resolute integrity	23	46.0	7	50.0	16	44.4	
Resolute onyx	5	10.0	2	14.3	3	8.3	
Xience xpedition	6	12.0	2	14.3	4	11.1	
Resolute onyx / Xience xpedition	1	2.0	0	0.0	1	2.8	
Atmospheric pressure							
Min. – Max.	10.0 – 18.0		12.0 – 18.0		10.0 – 18.0		0.960
Mean ± SD.	14.45 ± 1.79		14.43 ± 1.60		14.46 ± 1.88		
Median (IQR)	14.0 (14.0 – 16.0)		14.0 (14.0 – 14.0)		14.0 (14.0 – 16.0)		

Table 6: Comparison between the two studied groups according to PPCI.

DISCUSSION

Silent myocardial ischemia (SMI) which was defined as objective and documented evidence of myocardial ischemia in the absence of chest discomfort or other anginal equivalent symptoms is a relatively common, yet poorly understood clinical entity.[5]

Although the incidence of patients with silent ischemia who had undergone successful angioplasty and stenting is variable, patients with silent ischemia were more likely to have critical events than those without ischemia, but less likely than those with symptomatic ischemia. Patients with both silent and asymptomatic ischemia had a worse prognosis than those without ischemia, yet patients with silent ischemia had better event-free survival than those with symptomatic ischemia. [6]

Our study was a prospective cross sectional observational study involved 50 patients who were diagnosed as anterior ST elevation myocardial infarction (STEMI) and received primary PCI and followed up for 6 months to detect silent myocardial ischemia using clinical evaluation, ECG, echocardiography and treadmill test (TMT) and classified according to treadmill test into positive and negative silent myocardial ischemia.

Zellweger al. [6] studied 356 patients with silent ischemia who had undergone successful angioplasty and stenting and follow up with myocardial perfusion imaging for an average of 4.1 years. Eighty-one patients (23%) had evidence of target vessel ischemia, which was silent in 62% of patients.

In our study, 28% of total study patients had silent myocardial ischemia while 87.5% of diabetics had silent myocardial ischemia and this could be explained by the airable prevalence in literature according to different risk factors.

Our results are concordant with Taher al.[7] who showed that silent ischemia is more prevalent among diabetics representing 45% of the patients with silent ischemia. A Group of 26 patients (32.5%) showed stress ST depression without chest pain (silent ischemia), 18 patients of them (69.2%) were diabetics. Their study concluded that diabetics with coronary artery disease has a higher prevalence of silent myocardial ischemia than matched non-diabetic patients with coronary artery disease.

Also, with a study by Nisha Arenja al. [8] to evaluate the prevalence, extent, and independent predictors of silent myocardial ischemia. The prevalence of silent myocardial ischemia was 28.5% in diabetics and 21.5% in nondiabetics although different figures but more prevalent in diabetics.

And also, are concordant with a retrospective study by Kathiresan al. [9] to detect silent myocardial ischemia using 12-lead continuous electrocardiographic monitoring in patients who had PCI. Despite successful angiographic results, one third of the patients experienced silent myocardial ischemia during the postprocedural period.

Unlike our study, SMI was found less prevalent in the work of Tavkaeva.in which SMI was detected in only 6.6% after 6 months in the group of patients who underwent CA stenting. Although, this study used Holter ECG monitoring and variable risk factor than our study. [10]

However, SMI was found more prevalent in the ADORE2 trial (The Aggressive Diagnosis of Restenosis in high-risk patients). They found that among routine functional testing patients, 27.0% and 41.9% had a positive functional test at 1.5 and 6 months, respectively. [11]

We found that, out of 50 patients, 14 patients (28%) had a positive Exercise Treadmill Test at 6 months follow up and 36 patients (72%) were negative.

According to previous study when using SPECT MPI, Cristina Hernández, al. [12] stated that SMI was present in 21.9% of diabetics in a study of 41 type 2 diabetic patients.

Also, the DIAD study (Detection of silent myocardial Ischemia in Asymptomatic Diabetic Subjects) concluded that Silent myocardial ischemia occurs in greater than one in five asymptomatic patients with type 2 diabetes. [13]

In our study 57.1% of patients were hypertensives, only 38.9% of them had evidence of silent myocardial ischemia and this could and adherence to Medications in post myocardial infarction period. So controlled hypertension may be less linked to silent myocardial ischemia.

This is similar to a study by Lubaszewski et al[14] on Silent myocardial ischemia and the concomitant presence of diabetes mellitus and arterial hypertension who found silent myocardial ischemia more frequent in patients with both risk factors (hypertension and diabetes) than only hypertensives (29.3% vs 12.5%).

Also, In a study by Rodrigo Modolo, et al[15] on prevalence of myocardial ischemia in patients with HTN. Thirty-six (28%) patients had myocardial ischemia.

In our study half of dyslipidemic patients had almost silent myocardial ischemia. This is concordant with Ariane SULTAN, et al [16] study that concluded a significant association was found between Silent Myocardial Ischemia, LDL cholesterol, microangiopathy, and non-HDL cholesterol.

CONCLUSION

In this study of short term follow up with patients after acute ST elevation myocardial infarction treated with primary coronary intervention (PCI) and Silent myocardial ischemia (SMI) was detected in more than quarter of study population. This proves that CAD (especially patients recovering AMI) is a major risk factor for further and even silent myocardial ischemia. Older age, diabetes mellitus and dyslipidemia are risk factors for silent myocardial ischemia although other risk factors or mechanical issues have no role in SMI.

Conflict of interest : none

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