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Frequency of functional mitral regurgitation in patients with acute myocardial infarction in Babylon province.

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Abstract

Acute myocardial infarction (AMI) still causes morbidity in wide area of word, death cells of heart due to cut of oxygen. Frequency of mitral regurgitation (MR) after myocardial infarction (MI) varies from 11 - 59 %, which determined by echocardiography.

Key words: Electrocardiography ,echocardiography, biochemical changes.

INTRODUCTION

The AMI causes of morbidity and mortality in wide of world. It is the death of cardiac cells that occurs following prolonged oxygen cut. The main risk factors were; disturbance of lipids, taking tobacco, high blood pressure, diabetes, obesity, psychosocial disturbance, drinking of alcohol and little physic in both sexes and at all ages in all regions[1].

Two main types of AMI are ST elevated (STEMI) and non ST elevation (NSTEMI) when ischemia present mainly in internal layer of heart. Myocardial injury is observed when troponin I or creatine kinase (CK-MB) are increased many times of normal level. Cardiac troponin I is components of the contractile apparatus of cardiac cells. These markers reflect injury of cardiac cells[2].

The mitral regurgitation (MR) is frequently seen after the acute ST elevation of myocardial infarction (STEMI). The morbidity and mortality after MI are depending to the type and severity of MI[3].

Incidence of mitral regurgitation after myocardial infarction:

Mitral regurgitation as a complication of MI is a poor prognosis. It is about severity is 13% to 45%. Mitral regurgitation caused by papillary muscle rupture (figure 1). Fibrinolysis decreases the incidence of rupture; rupture was reported to occur after some days without fibrinolysis. Papillary muscle rupture is found 7% of patients after acute MI and mortality 5% [4,5]. The percentage of mitral regurgitation in MI varies from 11% to 59%[6].

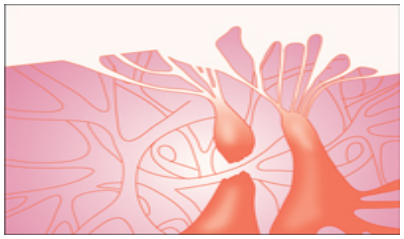

Figure (1): Rupture of papillary muscle.

Pathophysiology of MR in MI:

Mitral regurgitation occurs by number of mechanisms[5]:

1- Mitral valve annular dilatation due to left ventricle (LV) dilatation.

2- Papillary muscle dysfunction due to ischemia.

3- Partial or complete rupture of the chordae tendineae of papillary muscle.

Papillary muscle rupture is common with an inferior MI because posteromedial papillary muscle is most often involved due to single blood supply through the posterior descending coronary artery[7].

Mitral valve regurgitation (MR) is a frequent finding in patients of acute myocardial infarction (AMI). Reported risk factors include old age, high lipid, and recurrent ischemia. Doppler echocardiography is diagnostic tool for detecting MR[6]. High cholesterol was defined as: low-density lipoprotein cholesterol (LDL-c) >160 mg/dl (4.1 mmol/l) on a fasting sample, total cholesterol ≥200 mg/dl (5.2 mmol/l). [18].

SUBJECTS AND METHODS

1-Subjects:

The study was conducted in Marjan teaching hospital in Hilla city. This study lasted from April /2015 to July / 2015.The total number of subjects involved in this study was 100 Subjects. They included (male 55, female 45). The age distribution of study sample ranged from 40-60 years.

All patients admitted to CCU have been referred to the echocardiography in the hospital before 4th day from admission . All patients were subjected to echocardiography study by the same echocardiographs. Patients were sent for serum cardiac biomarkers (cardiac troponin I, & CK-MB) during first 12-24 hours of signs and symptoms of MI[8,9]. It was demonstrated that troponin concentration displays a strong correlation with infarct size[10, 11].

2- Methods:

A-Patient history: Complete history of hypertension, diabetes mellitus, and previous attack of disease were obtained. Patients were considered hypertensive when they were already on antihypertensive treatments or their blood pressure at rest was > 140 / 90 mmHg according to the European Society of Hypertension, 2009[12]. DM was diagnosed by fasting plasma glucose ≥ 126 mg⁄dl (7.0 mmol) [13]. A positive family history was defined as the presence of at least one first-degree relative who had developed coronary artery disease [14].

B-Blood sample: Five milliliter of venous blood was taken from all subjects for lipid profile, serum potassium, White blood cells count, and troponin I. White blood cells count was mentioned in Dacie and Lewis practical hematology[15].

C-The lipid Profile laboratory analysis: After fasting 12 –14 hours, the blood was centrifuged and collected serum was investigated for serum cholesterol, serum triglyceride, and serum high density lipoprotein HDL by direct method[16].

Very low density lipoprotein VLDL = Serum triglyceride/5.

Low density lipoprotein (LDL) was calculated by use Friedewald formula as follow:

Total cholesterol = HDL +LDL +VLDL[17]. HDL : High density lipoprotein.

D-Body mass index (BMI) measurements:

Weights and height of patients and were measured by the use of well calibrated digital weight and height scale measuring device, BMI was calculated by dividing weight in kilograms by the square of the height in meters [18].

Data Analysis

Statistical analysis was carried out using SPSS version 17. Continuous variables were presented as (Means ± SD). Student t-test was used to compare means between two groups. A *p*-value of ≤ 0.05 was considered as significant[19].

RESULTS

1- The Incidence of MR among Patients with MI:

Figure 3.6 shows the incidence of mitral regurgitation among patients with myocardial infarction. The incidence of mitral regurgitation among patients with myocardial infarction was 36% figure .

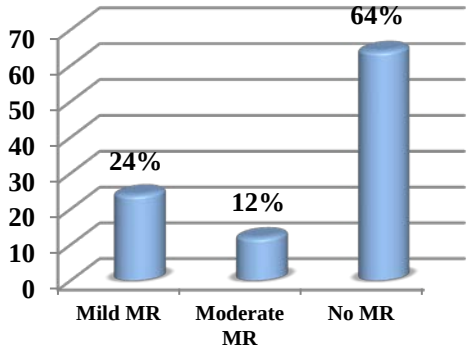

Figure (2): Incidence of MR among patients with MI.

2- The Association between Study Groups and Study Variables

Table 1: shows the association between studies groups including (patients with MR and patients without MR) and study variables including (gender, smoking habit, BMI, history of DM and blood pressure measurement). There was no significant association between MR with BMI and history of DM; while there was significant association between MR and gender, smoking habit, site of MI and hypertension.

Table 2 shows mean differences of age (years) according to mitral regurgitation. There were no significant differences between means of age by study groups.

Table 3 shows mean differences of study variables including (ejection fraction, LFED, troponin level, WBC count and potassium level) according to mitral regurgitation. There were significant differences between means of LFED level and potassium level by study groups, while there were no significant differences between means of other study variables by study groups.

Table 1: Association between study groups and study variables.

Study variables	MR		χ^2	P-value
	Present	Absent		
Gender				
Male	26 (72.2)	29 (45.3)	**6.741**	**0.009***
Female	10 (27.8)	35 (54.7)		
Smoking habit				
Smoker	18 (50.0)	49 (76.6)	**7.352**	**0.007***
Non-smoker	18 (50.0)	15 (23.4)		
Body mass index				
Normal (18.5-24.9)	6 (16.7)	14 (21.9)	0.53	0.767
Pre-obese (25-29.9)	25 (69.4)	40 (62.5)		
Obese (≥ 30)	5 (13.9)	10 (15.6)		
Site of MI				
Anterior	10 (27.8)	24 (37.5)		
Inferior	26 (72.2)	13 (20.3)	31.747	<0.001*
Posterior	0 (0.0)	15 (23.4)		
Extensive	0 (0.0)	12 (18.8)		
History of DM				
Present	19 (52.8)	41 (64.1)	1.223	0.269
Absent	17 (47.2)	23 (35.9)		
Blood pressure measurement				
Hypertensive	29 (80.6)	25 (39.1)	15.969	<0.001*
Non hypertensive	7 (19.4)	39 (60.9)		

*P value < 0.05.

3- Mean Differences of Age according to Mitral Regurgitation:

Table (2): The mean differences of age according to mitral regurgitation

Study variables	MR	N	Mean ± SD	t-test	P-value
Ejection fraction (%)	Present	36	49.25 ± 11.23	0.64	0.524
	Absent	64	47.78 ± 10.90		
LFED	Present	36	48.33 ± 4.85	2.142	0.035
	Absent	64	50.68 ± 5.49		
Troponin level (ug/l)	Present	36	15.47 ± 10.17	1.162	0.248
	Absent	64	13.00 ± 10.21		
High WBC count (x 10^9/l)	Present	36	14.55 ± 2.14	-0.762	0.448
	Absent	64	14.88 ± 2.07		
High Potassium level	Present	36	5.33 ± 0.82	2.003	0.048
	Absent	64	4.97 ± 0.90		

4- Mean Differences of study variables according to Mitral Regurgitation

Table (3): The mean differences of study variables according to mitral regurgitation.

Variable	MR	N	Mean ± SD	t-test	P-value
Age (years)	Present	36	58.41 ± 6.76	1.718	0.089*
	Absent	64	55.78 ± 7.67		

*p value ≤ 0.05 was significant

DISCUSSION:

Percentage of mitral regurgitation in patients with AMI was 36 %. This result was agree with result of other study by Birnhaum et al., 2002[6] who mentioned that the prevalence of mitral regurgitation varies from 11% to 59%. As shown in figure1 . Smoker patients were significant (P value was 0.007) to MR than non-smoker as shown in table1, because ischemic mitral regurgitation following acute coronary syndrome more likely in elderly diabetics and hypertensive smokers.[20]

Hypertension > 140/90 was prone to MR in MI significantly (P < 0.001) as shown in table1. Because ischemic mitral regurgitation following acute coronary syndrome more likely in elderly diabetics and hypertensive smokers.[20]

Old age patients mean (58.41 ± 6.76 years) with MI was prone to develop MR more than patients with mean ages (55.78 ± 7.67 years) but not significant as shown in table 3. This result not agree with **Deroyer et al., 2015[21]** who said that advanced age in myocardial infarction was significant prone to MR.

Potassium level and gender was significant (P- value 0.004, 0.001) respectively difference between moderate and mild MR as shown in Table 3 and 1.

Inferior site, elderly more likely to female than male, LVEDD and smoker and non-smoker of MI showed significant (P- value <0.001, 0.003, 0.035, 0.007) respectively difference between MR patients and MI without MR patients as shown in table 1. Because Deroyer et al., 2015 [21] found that inferior site of MI, elder patient more likely to be female, left ventricle end diastolic diameter in mm (LVEDD), smoker patients were significant to prone MR in MI patients.

CONCLUSION:

1-The percentage of mitral regurgitation in patients with AMI was 36%.

2- Male, smoker, inferior MI, hypertension, and diabetes were prone to mitral regurgitation in myocardial infarction.

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