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Identification of patient affected by mitral regurgitation with unhealthy controls

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Abstract

This study included patients suffering from Mitral regurgitation caused by papillary muscle rupture.it is frequency of mitral regurgitation after myocardial infarction and it is determined by echocardiography and clinical examination . myocardial infarction one of the important mechanical complication is mitral regurgitation. The total number was 66 subjects subdivided into 36 patient suffering from mitral regurgitation And 30 unhealthy control.

Keywords: Mitral regurgitation, papillary muscle rupture

INTRODUCTION

Ischemic mitral regurgitation (MR) is frequently seen after the acute ST **elevation** myocardial infarction (MI) and is an independent predictive factor for long-term cardiovascular morbidity and mortality after MI. Timing and severity of MR after MI is relevant to the type and extent of MI **(1).**

Mitral regurgitation (MR), also known as mitral insufficiency or mitral incompetence, is the reflux of blood from the left ventricle into the left atrium during cardiac systole. The functional competence of the mitral valve relies on the coordinated interaction of the mitral annulus and leaflets, chordae tendineae, papillary muscles, left atrium and left ventricle (LV). Figure 1 illustrates the different components of the mitral apparatus **(2)**. MR can result from failure of one or more of these components. The clinical presentation of MR may vary from an asymptomatic patient with MR noticed on an echocardiogram done for post-myocardial infarction risk stratification, to a patient who presents in cardiogenic shock due to acute severe MR **(3).** On clinical examination a pan-systolic murmur is audible; the grade of the murmur does not correspond to severity. The symptoms and signs of heart failure may be associated with MR when it is hemodynamically significant. The time period over which MR develops dictates the degree to which the patient is able to compensate. Severe MR due to rupture of a papillary muscle trunk leads to acute circulatory collapse, whereas severe MR due to progressive degenerative disease **(4).**

MATERIAL AND METHODS

This subject were included in this study, patient with mitral regurgitation And unhealthy control group. The study was conducted in Marjan teaching hospital in Hilla city. This study lasted from 2017 to 2018.The total number of subjects involved in this study were 66 Subjects. They included (patient 36, unhealthy control 30).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**.** It was demonstrated that troponin concentration displays a strong correlation with infarct size**.**

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 guidelines of the European Society of Hypertension**.** DM was diagnosed by random plasma glucose ≥ 200 mg /dl (11.1 mmol/l), Fasting plasma glucose ≥ 126 mg /dl (7.0 mmol). Or oral glucose tolerance test (OGTT) more than 200 mg**.** A positive family history was defined as the presence of at least one first-degree relative who had developed coronary artery disease before the age of 55 years for men and 65 years for women**.**

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

C-The method of lipid Profile laboratory analysis: After fasting 12 –14 hours, a the blood was centrifuged and collected serum was investigated for serum cholesterol, serum triglyceride, and serum high density lipoprotein HDL by direct method**.** The serum very low density lipoprotein VLDL = Serum triglyceride/5. LDL was calculated by use Friedewald formula as follow: Total cholesterol = HDL +LDL +VLDL[17].

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**.** BMI 18.5 to <25 kg/m^2 were considered normal; 25 to <30 kg/m^2 over weight; >30 kg/m^2 obese**.**

Data Analysis

Statistical analysis was carried out using SPSS version 17. Categorical variables were presented as frequencies and percentages. Continuous variables were presented as (Means ± SD). Student t-test was used to compare means between two groups. Pearson's chi square (X^2) and fisher-exact test were used to find the association between categorical variables. A *p*-value of ≤ 0.05 was considered as significant**(5).**

RESULT

In this study the Association between Study Groups and Study Variables The mean differences of age between study groups Table (1).

Table **(2)** shows the association between studies groups including (patients with MR and control group) and study variables including (gender, smoking habit, BMI, history of DM and blood pressure measurement). There was significant association between presence of MR with BMI, blood pressure measurement and history of DM; while there was no significant association between presence of MR and gender, smoking habit. Mean Differences of Lipid Profile Elements between Patients with Mitral Regurgitation and Control Group. Table **(3)** shows mean differences of lipid profile elements including (cholesterol, triglyceride, HDL, LDL and VLDL) between patients with mitral regurgitation and control group. There were significant differences. patients with mitral regurgitation and control group. There were significant differences between means of EF, troponin level, WBC count and potassium level by study groups, while there were no significant differences between means of LFED by study groups table **(4).**

Table 1: The mean differences of age between study groups.

Variable	Study groups	N	Mean ± SD	t-test	P-value
Age (years)	Patients with MR	36	58.41 ± 6.76	1.789	0.078
	Control group	30	55.03 ± 8.60		

Table 2: Association between study groups and study variables

Study variables	Study groups		χ^2	P-value
	MR	Control group		
Gender				
Male	26 (72.2)	16 (53.3)	2.523	0.112
Female	10 (27.8)	14 (46.7)		
Smoking habit				
Smoker	18 (50.0)	10 (33.3)	1.861	0.173
Non-smoker	18 (50.0)	20 (66.7)		
Body mass index				
Normal (18.5-24.9)	6 (16.7)	14 (46.7)		**0.025** [f]
Pre-obese (25-29.9)	25 (69.4)	15 (50.0)		
Obese (≥ 30)	5 (13.9)	1 (3.3)		
History of DM				
Present	19 (52.8)	8 (26.7)	**4.615**	**0.032**
Absent	17 (47.2)	22 (73.3)		
Blood pressure measurement				
Hypertensive (≥140 or ≥ 90)	29 (80.6)	2 (6.7)	**35.867**	**<0.001***
Normotensive or pre-hypertensive	7 (19.4)	28 (93.3)		

Table 3: The mean differences of lipid profile elements between study groups.

Study variables	Study groups	N	Mean ± SD	t-test	P-value
Total serum cholesterol (mmol/l)	MR	36	5.57 ± 0.62	11.658	< 0.001*
	Control	30	3.68 ± 0.69		
Triglyceride (mmol/l)	MR	36	2.78 ± 0.32	6.606	< 0.001*
	Control	30	1.96 ± 0.65		
HDL (mmol/l)	MR	36	1.88 ± 0.78	-9.756	< 0.001*
	Control	30	3.68 ± 0.69		
LDL (mmol/l)	MR	36	4.31 ± 0.75	19.995	< 0.001*
	Control	30	0.81 ± 0.64		
VLDL(mmol/l)	MR	36	0.56 ± 0.10	5.797	< 0.001*
	Control	30	0.39 ± 0.13		

Table 4: The mean differences of study variables according to mitral regurgitation

Study variables	Study groups	N	Mean ± SD	t-test	P-value
Ejection fraction (%)	MR	36	49.25 ± 11.23	-4.217	<0.001*
	Control	30	58.53 ± 6.33		
LFED	MR	36	48.33 ± 4.85	1.859	0.068
	Control	30	46.26 ± 4.02		
Troponin level (ug/l)	MR	36	15.47 ± 10.17	9.097	<0.001*
	Control	30	0.04 ± 0.03		
WBC count (x 10^9/l)	MR	36	14.55 ± 2.14	14.785	<0.001*
	Control	30	7.60 ± 1.67		
Potassium level (years)	MR	36	5.33 ± 0.82	11.759	<0.001*
	Control	30	2.76 ± 0.95		

DISCUSSION

There was significant differences in ejection fraction between control and mitral regurgitation because patients who have myocardial infarction (MI), proven by numerous investigation, reduce left ventricular ejection fraction (LVEF) significantly **(6).** There was significant differences in troponin level between normal persons and mitral regurgitation patients because of troponin level increased in non S wave elevation (NSTE) **(7).** There was significant differences in WBC count between controls and mitral regurgitation patients**.** Because of the coronary atherosclerosis is an inflammatory process. Leucocytes are major mediators of inflammation. Leucocytosis is common in acute STEMI, it result from inflammatory response **(8)**. There was significant differences in potassium level between controls and mitral regurgitation patients in this study , because there was higher level of potassium observed in > 50 years age group of AMI than controls. **(9)**. There was significant differences in lipid profile (TC, serum triglyceride, LDL, VLDL, HDL-c) between control and mitral regurgitation patients**,** because HDL-c starting falling from day 2 onwards, serum triglyceride showed an increasing after with a significant increase on day 3after MI and pre discharge (P value < 0.001). the mechanism may be due to elevated of fatty acid and impaired removal of VLDL from plasma. Total cholesterol and LDL show significant change in 24 hours of acute episode**. (10)** observed significant higher TC, TG, level and lower HDL-c level in AMI patients.

CONCLUSION:

1- Frequency of mitral regurgitation among patients with unhealthy controls
2- Potassium level, WBCs count, BP, and lipid profile except HDL increased significantly in MR comparison to controls.
3- Ejection fraction decreased significantly comparison to controls.
4- Early detection of risk factors restrict cigarette smoking decrease body mass index by suitable exercise.

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