

STUDY OF CORRELATION BETWEEN SEVERITY OF ISCHEMIC MITRAL VALVE REGURGITATION BY ECHO DOPPLER AND ECG CHANGES IN ACUTE MYOCARDIAL INFARCTION

By

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ABSTRACT

Background: Myocardial infarction (MI) accompanies with different mechanical complications including ischemic mitral regurgitation (IMR) which is correlated with increased risk of mortality and heart failure. IMR existence and its severity had significant prognostic effect on patients' mortality following acute myocardial infarction (AMI). Echocardiography is the choice imaging modality for the diagnosis and assessment of IMR and establishing its etiology. IMR usually results from papillary muscle displacement, ventricular dilation and remodeling with reduced closing forces.

Objective: Assessment of the correlation between echocardiographic severity of ischemic mitral valve regurgitation following acute myocardial infarction and its electrocardiographic location.

Patients and methods: This was a prospective study that included 100 patients admitted to CCU OF Beni-Suief General Hospital in the period from October 2019 to May 2020 . Those patients were divided on two equal groups: Group I: patients with STEMI and no, trivial or mild MR and Group II: patients with STEMI and moderate or severe MR.

Results: Mean age of group I involved in the study was 55.44 ± 7.23 years and 70% of them were males. Mean age of group II was 58.38 ± 7.8 and 56% were males. 70 % of the group II had inferolateral STEMI compared to group I who had more anterior STEMI. Group II were more smokers with hyperlipidemia. Group II was with higher previous stable and unstable angina, with less streptokinase treatment, with higher abnormal chest radiogrgh and higher killip class. Group II had significantly lower EF and higher LVEDD, LVESD, LA diameter, RV diameter, MV A velocity, tricuspid valve excursion TAPSI and pulmonary artery pressure and more diastolic dysfunction. Mechanism of IMR in case group was mono leaflet tethering in 43 cases, both leaflet tethering in 7 cases and ring dilation in 30 cases. MR jet direction was medial commissure posterior in 31 (62%), lateral commissure anterior in 12 (24%), and both commissures central in 7 (14%) cases.

Conclusion: Mitral regurgitation is common after acute myocardial infarction, especially in cases with inferior mitral regurgitation. The echocardiographic findings are indicative of left ventricular remodeling and abnormality of mitral valve apparatus.

Keywords: Ischemic Mitral Valve Regurgitation, Echo Doppler, ECG, Myocardial Infection.

INTRODUCTION

Ischemic mitral regurgitation (IMR) is one of the most important coronary artery disease complications and, in particular, myocardial infarction (MI). The risk stratification of AMI patients during the early stage can identify high-risk patients, who require more advanced treatment and whose outcome can be improved through early intervention (*Fazlinezhad et al., 2014*).

Ischemic mitral regurgitation (IMR) is a subtype of secondary mitral regurgitation (MR), which is a complication of ischemic heart disease. Valvular involvement can be primary (organic) or secondary (functional). Primary occurs after the rupture of the mitral subvalvular apparatus in the context of an AMI. Secondary is when the valve leaflets and chordae are structurally normal and MR results from an imbalance between closing and tethering forces on the valve secondary to alterations in the left ventricle (LV) geometry (*Lancellotti et al., 2013*).

Ischemic mitral regurge is characterized mechanistically by incomplete mitral leaflet closure, namely displacement of the leaflet coaptation apically within the LV cavity. Although a spectrum of anatomic abnormalities of both LV and PMs exists, evidence points to a predominant role of “tethering” as the final common pathway in inducing IMR. Post- infarct, outward displacement of PMs Leads to stretching of the chordae tendinae and increased tethering forces on the mitral leaflets, which causes the apical coaptation and restricted closure. Annular dilatation may also contribute by

stretching leaflets and causing incomplete closure. Practitioners should mindful not to classify as IMR those patients with mitral regurgitation and comorbid ischemic heart disease if there is any intrinsic mitral valve apparatus abnormality and/or there has not been a history of myocardial infarction (*Nishimura et al., 2014*).

However, functional ischemic MR is a highly dynamic lesion that may improve over time after coronary revascularization or impair because of progressive adverse left ventricular (LV) remodeling and tethering of the mitral leaflets (*Chua et al., 2010*). Furthermore, ischemic MR, contributing to LV volume overload, can exacerbate the adverse LV remodeling after STEMI, promoting further LV dilatation and, as in a vicious circle, further MR (*Uddin et al., 2012*).

Echocardiography is the choice imaging modality for the diagnosis and assessment of IMR and establishing its etiology (*Lancellotti et al., 2013*). IMR usually results from papillary muscle displacement, ventricular dilation and remodeling with reduced closing forces (*Boyd, 2013*).

Left ventricular (LV) remodelling after myocardial infarction (MI) can lead to apical and posterior displacement of the papillary muscles with resulting malcoaptation of the mitral leaflets and valvular insufficiency (*Meris et al., 2012*).

The present work aimed to assess the correlation between echocardiographic severity of ischemic mitral valve regurgitation following acute myocardial infarction and its electrocardiographic location.

PATIENTS AND METHODS

This was a prospective study that included 100 patients admitted to CCU OF Beni-Suief General Hospital in the period from October 2019 to May 2020. These patients were divided on two equal groups: **Group I** patients with STEMI and no, trivial or mild MR and **Group II:** patients with STEMI and moderate or severe MR.

Inclusion criteria: Patients presented with acute STEMI with: No previous history of MI, no previous percutaneous coronary intervention (PCI), not performing PCI before echocardiography during the admission, no previous coronary artery bypass surgery (CABG), no history of valvular or congenital heart disease and no echocardiographic finding of organic valve lesion.

Exclusion criteria: Patients presented with acute STEMI and history of previous MI, history of previous percutaneous coronary intervention (PCI), history of performing PCI before echocardiography during the admission, history of previous coronary artery bypass surgery (CABG), history of valvular or congenital heart disease and echocardiographic finding of organic valve lesion.

All patients were subjected to:

I. Full history taking that included:

Age, gender diabetes mellitus, hypertension, Cigarette smoking, dyslipidemia, previous stable angina, previous unstable angina, exertional dyspnea, treatment with streptokinase during admission.

II. Clinical examination: Examination included the estimation of heart rate, rhythm, pulse volume, respiratory rate, blood pressure, temperature, additional heart sounds, murmurs, pericardial rub and pulmonary rales.

Killip class was determined: Class I includes individuals with no clinical signs of heart failure. Class II includes individuals with rales or crackles in the lungs, an S3, and elevated jugular venous pressure. Class III describes individuals with frank acute pulmonary edema. Class IV describes individuals in cardiogenic shock or hypotension.

III- Investigations:

A. 12-lead electrocardiograms: 12-lead electrocardiogram was recorded at admission to diagnose STEMI and determine its site (inferior, anterior, lateral, posterior, and right) by determining the leads exhibiting ST segment elevation.

B. Chest X-ray: Chest radiograph was done searching for congestion, pulmonary oedema, cardiomegaly or other causes of chest pain.

C. Transthoracic echocardiogram: Transthoracic two-dimensional imaging and pulsed wave Doppler echocardiographic studies were obtained in all patients using a commercially available ATL HDI 5000 echocardiographic machine with a broadband 2.5 – 4 MHz phased array transducer.

Patients were examined in the supine and left lateral positions to obtain adequate images in the parasternal long and short axis views, the four, five and two chamber apical

views, the apical long axis as well as in the subcostal views. Transthoracic echocardiography was performed at rest during the first 5 days after AMI.

Echocardiographic:

- Left ventricular ejection fraction (LVEF).
- Right ventricular function.
- Regional wall motion abnormality (RWMA).
- Mitral inflow.
- Left atrium, left ventricle, right ventricle size and aortic root diameter.
- Pulmonary artery pressure from tricuspid valve regurge velocity.

When the valve structure intrinsically was normal and the regurgitation was caused as a result of the myocardial infarction and the ischemic MR was diagnosed. Other parameters for assessing MR were measured to classify MR as mild, moderate or severe MR as:

- LV annular diameter,
- Leaflet tethering and tethering depth,
- Direction of MR jet,
- Vena contracta width,
- Effective Regurgitant orifice area,
- PISA radius,

- MR regurgitant volume.

MR severity and correlation to the site of MI in the ECG was determined.

D. Laboratory investigations: Routine blood tests like CBC& kidney function tests, lipid profile and cardiac troponin were done.

Ethical consideration: Participation in this study was voluntary and gave written consents to share in this work. The study was approved from ethical committee of AL-Azhar University hospital.

Statistical Analysis:

All data were subjected to revision and validation then description and analysis on IBM-compatible PC by using SPSS (Statistical Package for the Social Sciences) program version 22.0.0, Microsoft Office Excel 2007, and GraphPad Prism 6. Descriptive statistics were performed for all studied parameters in the three studied groups and were presented in the form of mean, standard deviation (SD), minimum, maximum, range, and percentages. Analytical comparison between different groups was done by using student t test for comparing parametric data when normally distributed. $P < 0.05$ was considered significant.

RESULTS

Mean age of group I involved in the study was 55.44 ± 7.23 years. 70% of them were males.

Mean age of group II was 58.38 ± 7.8 , 56% were males. Clinical risk factors among the study

populations were investigated and we found that group II was more smokers with hyperlipidemia. Comparison of the ECG between the 2 groups showed statistically significant difference according to the site of MI where 70 % of group II had inferolateral STEMI compared to the group I who had more anterior STEMI. By clinical history, examination and investigations there was statistically significant difference

found between two groups regarding and treatment with Streptokinase, Normal chest x-ray and Killip class. But there was no statistically significant difference found between two groups regarding Previous Stable Angina and Previous Un Stable Angina. Group II was with higher previous stable and unstable angina, with less streptokinase treatment, with higher abnormal chest radiograph and higher Killip class (Table 1)

Table (1): Comparison between the two studied groups according to demographic data, risk factors, MI type and clinical data

Parameters		Group I (n = 50)		Group II (n = 50)		P
		No.	%	No.	%	
Sex						
Male		35.0	70.0	28.0	56.0	0.147
Female		15.0	30.0	22.0	44.0	
Age (years)						0.054
Min. – Max.		41.0 – 75.0		42.0 – 75.0		
Mean ± SD.		55.44 ± 7.23		58.38 ± 7.8		
Diabetes Mellitus (DM)	No	38	76.0%	33	66.0%	0.271
	Yes	12	24.0%	17	34.0%	
Hypertension (HTN)	No	26	52.0%	22	44.0%	0.423
	Yes	24	48.0%	28	56.0%	
Hyperlipidemia	No	36	72.0%	40	80.0%	0.349
	Yes	14	28.0%	10	20.0%	
Smoking	No	33	66.0%	32	64.0%	0.834
	Yes	17	34.0%	18	36.0%	
M.I Type	Anterior	27	54.0%	15	30.0%	0.015
	Inferolateral	23	46.0%	35	70.0%	
Previous Stable Angina	No	45	90.0%	38	76.0%	0.062
	Yes	5	10.0%	12	24.0%	
Previous UnStable Angina	No	47	94.0%	42	84.0%	0.110
	Yes	3	6.0%	8	16.0%	
Normal chest x ray	No	14	28.0%	27	54.0%	0.001
	Yes	36	72.0%	23	46.0%	
Killip class	I	43	86.0%	24	48.0%	0.001
	II	7	14.0%	18	36.0%	
	III	0	0.0%	8	16.0%	
Treatment With Streptokinase	No	11	22.0%	23	46.0%	0.011
	Yes	39	78.0%	27	54.0%	

By echocardiography group II had significantly lower Ejection Fraction (EF%) and higher LVEDD (cm), LVESD

(cm), LA Diameter (cm), Aortic root Diameter (cm), RV Diameter (cm), MV A velocity (m/sec), tricuspid valve excursion

TAPSI (cm) and pulmonary artery pressure PAP (mmHg). Tissue Doppler findings were also significantly different between the two groups. Group II had RWMA more in RCA territory while group I more in LAD territory.

Mechanism of IMR in case group was mono leaflet tethering in 43 cases, both

leaflet tethering in 7 cases and ring dilatation in 30 cases. MR jet direction was medial commissure posterior in 31 (62%), lateral commissure anterior in 12 (24%) and both commissures central in 7 (14%). **Table 2).**

Table (2): Comparison between the two studied groups according to echocardiographic data

Parameters	Groups	Group I	Group II	P-value
		No.= 50	No.= 50	
EF (%)	Mean ±SD	51.1 ±8.76	45.92 ± 8.49	0.003
LVEDD (cm)	Mean ±SD	4 ± 0.49	5.04 ± 0.63	0.001
LVESD (cm)	Mean ±SD	2.91 ±0.47	3.8 ± 0.51	0.001
Posterior wall thickness (cm)	Mean ±SD	0.87 ±0.13	0.88 ± 0.14	0.651
Septal Wall Thickness (cm)	Mean ±SD	0.92 ±0.13	0.92 ± 0.15	0.889
LA Diameter (cm)	Mean ±SD	2.57 ±0.36	3.89 ± 0.35	0.001
Aortic root Diameter (cm)	Mean ± SD	2.6 ± 0.36	3.11 ± 0.29	0.001
RV Diameter (cm)	Mean ±SD	2.74 ±0.26	2.92 ± 0.16	0.001
MV A velocity (m/sec)	Mean ±SD	0.8±0.14	0.97±0.26	0.001
MV E velocity (m/sec)	Median(IQR)	0.90(40 –60)	1(40 – 60)	0.016
E/A	Mean ±SD	1.15 ±0.29	1.1 ± 0.28	0.005
MAPSI (cm)	Mean ± SD	1.01 ± 0.18	0.91 ± 0.17	0.005
RWMA	RCA	23(46%)	35(70%)	0.015
	LAD	27(54%)	15(30%)	
TAPSI (cm)	Mean ± SD	1.77 ± 0.21	1.9 ± 0.22	0.003
PAP (mmHg)	Median(IQR)	25(40 – 60)	48 (40 – 60)	0.001
EROA (CM2)	Mean ±SD	-	20.40 ± 1.98	-
MV Regurgitant volume (ML)	Mean±SD	-	27.86 ± 5.32	-
Vena Contracta (mm)	Mean±SD	-	6.28 ± 1.9	-
PISA diameter (ml)	Mean±SD	-	1.04 ± 0.24	-
Tenting Area (cm2)	Mean±SD	-	1.59 ± 0.34	-
MV Annulus diameter (cm)	Mean ±SD	-	3.97 ± 0.32	-
Leaflet Tethering	Both	-	7 (14.0%)	-
	Mono	-	43 (86.0%)	
Jet Direction	Anterior	-	12 (24.0%)	-
	Central	-	7 (14.0%)	-
	Posterior	-	31 (62.0%)	-

MVE & PAP are not normally distributed. So, we used another test (Mann-Whitney test) and found group II median higher than group I regarding PAP & MVE.

DISCUSSION

In our study, we observed that cases with IMR are more male, older and smoker. Being male, smoker and old age,

these are risk factors to IHD and its complication. Males were more dominant in cases of IMR(70%) in the study of Clinical implications of early mitral regurgitation in patients with reperfused acute myocardial infarction (*Carrabba et al., 2010*) .male was the dominant sex regarding frequency (72%) in a study about the frequency of IMR after first time acute MI and its relation to infarct location and in-hospital mortality (*Fazlinezhad et al., 2014*).

Valuckienė et al. (2015) had found that more cases were females and smokers when studied the relation between IMR and acute MI. *MacHaalany et al. (2014)*, had also found that more cases with IMR were females and smokers when they studied predictors and prognosis of IMR in the era of PCI.

The differences between the groups regarding the IMR severity are related to different baseline characteristics, coronary artery risk factors, and time of performing echocardiography (*Fazlinezhad et al., 2014*).

In our study, we observed that 70 % of the Group II had inferolateral STEMI compared to the other group who had more anterior STEMI. So, moderate to severe IMR was more common with inferior MI, the occurrence and severity of the IMR was affected by the location of MI. The mechanism producing IMR was different in anterior compared with inferior STEMI. Although LV remodeling and global LV dysfunction was greater in anterior MI, IMR was more severe in inferior STEMI due to the increased tethering force of the posteromedial papillary muscle near the site of the infarction. Posteromedial papillary muscle

has a single blood supply, while anterolateral papillary muscle has a dual blood supply so posteromedial papillary muscle is easily affected by occlusion of its artery in inferior MI.

Valuckienė et al. (2015), in the study of IMR in acute phase of MI, also reported that IMR is more common in inferior MI. *Mentias et al. (2016)*, in the study of outcomes of ischemic mitral regurgitation in anterior versus inferior ST elevation myocardial infarction, reported that IMR is more common in inferior MI.

Chua et al. (2011) have found that factors associated with a significantly higher risk of positive IMR included advanced age, Killip class ≥ 3 , increased left atrial diameter, increased LV end diastolic volume, and isolated inferior MI. When MI type (isolated inferior over isolated anterior) was used as a covariate, patients with an isolated inferior MI were 2.37 times more likely to have moderate or severe IMR than those with an anterior MI. *Fazlinezhad et al. (2014)* had reported that anterior and anterolateral walls were the most common territories of MI in patients with or without IMR.

In our study, patients in group II were more smokers with hyperlipidemia and there was no statistical difference regarding diabetes mellitus and hypertension. Smoking, hyperlipidemia, diabetes mellitus and hypertension are major risk factors for coronary artery disease and can influence its course and complications, but in our study we did not find any statistically difference regarding diabetes and hypertension and this may be due to small number of patients in our study.

Pant et al. (2011) reported that ischemic mitral regurgitation and diabetes or hypertension was not significantly related. Also, clinical significance of mitral regurgitation after acute myocardial infarction, had reported that IMR and diabetes or hypertension was not significantly related. They did not find a significant association of functional ischemic MR with age, sex or any of the conventional risk factors alone.

In our study, Group II was with higher previous stable and unstable angina, with less streptokinase treatment, with higher abnormal chest radiograph and higher Killip class. We found that previous history of stable and unstable angina was more with cases of moderate to severe IMR. We also found that streptokinase treatment was less in cases of moderate or severe IMR. So, treatment with streptokinase may decrease the severity of IMR. This was supported by *Poh et al. (2012)* as they found that both thrombolytic therapy and primary PCI were associated with decreased incidences of IMR following inferior posterior STEMI. It was also supported by *Mehrnoush et al. (2018)* who found that cases of moderate or severe IMR are less frequently treated with streptokinase.

We also found that cases with moderate or severe IMR had higher abnormal chest radiograph and higher Killip class pointing to their more susceptibility to heart failure and its complication with bad prognosis. *Fazlinezhad et al. (2014)* observed higher rate of LVEF <35% in the patients with IMR. *MacHaalany et al. (2014)* also reported Lower LVEF in the cases with moderate to severe IMR. This was supported by *Mehrnoush et al. (2018)*

who reported that cases with IMR had significantly higher Killip class and lower LVEF.

In our study, group II had significantly lower EF and higher LVEDD, LVESD, LA diameter, RV diameter, MV A velocity, tricuspid valve excursion TAPSI and pulmonary artery pressure and more diastolic dysfunction.

Different echocardiographic findings are reported in cases with moderate to severe IMR. *Fazlinezhad et al. (2014)* reported higher grade of diastolic dysfunction, end-diastolic LV pressure in IMR cases. They also had higher Systolic Pulmonary Artery Pressure (SPAP) which was related directly to the severity of MR. *MacHaalany et al. (2014)* also showed higher prevalence of PAP, LVESD and LA size in moderate to severe IMR. It was reported that moderate and severe MR in acute MI is related to increased left ventricular diastolic dimensions (*Valuckienė et al., 2015*). *Mehrnoush et al. (2018)* also reported that LVEDV, LVEDVI, LVESV and LVESVI were mild to moderately enlarged which are predictable considering the local remodeling as a consequence of RWMA and papillary muscle displacement in inferior MI with IMR.

In our study, mechanism of IMR in group II was mono leaflet tethering in 43 cases, both leaflet tethering in 7 cases and ring dilatation in 30 cases.

The concept of PM dysfunction was based on clinical observations that ischemic MR occurred after inferior myocardial infarction and secondary dysfunction of the medial PM. Historically, the mechanism of chronic IMR was attributed to papillary muscle

dysfunction. However, further studies demonstrated that ischemia of papillary muscles themselves fails to produce significant MR without damage of the underlying myocardial wall (*Thomas et al., 2010*). Our study was supported by *Mehrnoush et al. (2018)* who demonstrated that mono leaflet tethering and ring dilatation were the main contributors in IMR. MR jet direction was medial commissure posterior in 31 (62%), lateral commissure anterior in 12 (24%) and both commissures central in 7 (14%). This was supported by *Mehrnoush et al. (2018)* who demonstrated that MR jet direction was posterior in most cases of moderate and severe IMR.

CONCLUSION

Ischemic mitral regurgitation (IMR) was common after acute myocardial infarction which has a great impact on the survival and development of post-MI heart failure. Its existence was associated with a higher mortality compared to those patients who do not develop MR, being directly related to its severity. There was a correlation between severity of ischemic of IMR and the site of myocardial infarction. This study showed that the incidence of IMR in inferior MI is higher than other types. The echocardiographic findings were indicative of left ventricular remodeling and abnormality of mitral valve apparatus.

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دراسة العلاقة بين درجة إرتجاع الصمام الميترالى الناتج عن جلطة القلب الحادة بالموجات الصوتية على القلب ورسم القلب الكهربائي

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خلفية البحث: إن إحتشاء عضلة القلب الحاد قد يكون مصاحب بمضاعفات ميكانيكية عديدة ومن أهمها إرتجاع الصمام الميترالى. فوجود إرتجاع الصمام الميترالى الناتج عن جلطة القلب الحادة له تأثير تنبئى كبير بحالة المريض والذى يزيد من نسبة الوفيات و حدوث ضعف عضلة القلب وهنا يأتى دور الموجات الصوتية على القلب والدوبلر فى تشخيص هذا الإرتجاع وتحديد درجة شدته.

الهدف من البحث: دراسة العلاقة بين درجة إرتجاع الصمام الميترالى الناتج عن جلطة القلب الحادة بالموجات الصوتية على القلب ورسم القلب الكهربائي.

المرضى وطرق البحث: تم إجراء هذه الدراسة على 100 شخص بوحدة عناية القلب بمستشفى بنى سويف التخصصى فى الفتره من اكتوبر 2019 وحتى مايو 2020 وتم تقسيمهم إلي مجموعتين متساويتين:

المجموعه الاولى: ليس لديهم إرتجاع بالصمام الميترالى نتيجة جلطة القلب أو إرتجاع طفيف.

المجموعه الثانيه: لديهم إرتجاع متوسط أو شديد بالصمام الميترالى نتيجة جلطة القلب.

وقد تم عمل الآتى لجميع المرضى: التاريخ المرضى لكل مريض. الفحص الطبى الشامل لكل مريض. تم عمل بعض التحاليل الأساسيه وتشمل صورة الدم وتحاليل دهون بالدم وإنزيمات القلب. رسم القلب الكهربائي لتحديد مكان جلطة القلب. أشعة عادية على الصدر. موجات صوتيه علي القلب لتحديد وجود الإرتجاع بالصمام الميترالى ودرجة شدته.

نتائج البحث: في حالات الإرتجاع المتوسط والشديد: 70% منهم لديه جلطة بالجزء السفلى في القلب, بينما الحالات ذوات الارتجاع الطفيف معظمهم لديه جلطة بالجزء الأمامي. وكان لديهم تاريخ مرضى أكثر بالتعرض لنذبة صدرية مستقرة أو غير مستقرة. وكان لديهم تاريخ مرضى أقل بالنسبة لحقنة إذابة الجلطة (إستربتوكينيز). كما كانوا أكثر عرضة للنهجان مع وجود علامات إحتقان بالرئة في الأشعة العادية على الصدر. وبالنسبة لفحص الموجات الصوتية كان لديهم متوسط كفاءة عضلة القلب أقل بالنسبة للمجموعة الأخرى. ومتوسط حجم البطين الأيسر الإنقباضى والإنساطى وحجم البطين الأيمن والأذيين الأيسر ومتوسط ضغط الشريان الرئوى أعلى من المجموعة الأخرى. وكان سبب الإرتجاع بالصمام الميترالى قوة الشد فى إحدى وريقات الصمام فى 43 حالة, وقوة الشد فى الوريقتين فى 7 حالات, وإتساع حلقة الصمام فى 30 حالة. وكان إتجاه تيار إرتجاع الصمام الميترالى خلفى فى 31 حالة: أمامى فى 12 حالة ومركزى فى 7 حالات.

الإستنتاج: الإرتجاع الميترالى شائع بعد إحتشاء عضلة القلب الحاد وخاصة في جلطات القلب في الجزء السفلي وتشير نتائج تخطيط صدى القلب إلى إعادة تشكيل البطين الأيسر وشذوذ في جهاز الصمام التاجي.

الكلمات الدالة : إرتجاع الصمام المترالى – جلطة القلب الحاد – الموجات فوق الصوتية .