RESEARCH ARTICLE

Early Diagnosis of Complications of ST-elevation Myocardial Infarction with 2D Echocardiography

Siddharth Mankar[1], Vijay Gaikwad[2], Astha Ganeriwal[3]

Abstract

Introduction: 2D echo is a non-invasive, rapid, investigation that enables us to visualize the heart directly in real-time using ultrasound and can help in such situations in diagnosing AMI by detecting any regional wall motion abnormality. The diagnosis of ST-elevation in myocardial infarction (STEMI) is usually based on patient's history and ECG findings, but it should be taken into account that patients may present with atypical symptoms, also it takes cardiac enzymes some time to elevate above the normal range after the onset of chest pain.

Materials and methods: A total of 100 diagnosed patients with the first episode of acute ST-elevation myocardial infarction who reported in the emergency were included in the study and who have given written informed consent. On admission, patients with typical or atypical ischemic symptoms and ECG changes of ST-segment elevation of >0.1 mV (1 mm) in leads 2, 3, avF, V4, V5, V6, 1, and avL, and in leads V2, V3 > 0.2 mV (2 mm) in males >40 years, >0.25 mV in males 0.15 mV in females, in 2 contiguous leads were diagnosed as acute STEMI.

Observation and results: In our study, mechanical complications of AMI were detected on 2D echo, of which, mitral regurgitation was found in 23 patients (23%), the ventricular septal rupture was found in 3 patients (3%), ventricular free wall rupture in 2%, papillary muscle rupture in 11%, pericardial effusion was found in 13%, and LV clot was found in 11% patients.

Conclusion: Acute myocardial infarction is seen more commonly in the age-group of 61–70 years and it is more common among males. Killip classification of patients has prognostic value and helps in accessing the severity of myocardial infarction. Mechanical complications of AMI can be detected by 2D echo and can aid accordingly in treatment.

Keywords: 2D echo, Complications, Early diagnosis, ST-elevation in myocardial infarction.

Journal of Mahatma Gandhi University of Medical Sciences & Technology (2021): 10.5005/jp-journals-10057-0160

Introduction

2D echo is a non-invasive, rapid, investigation that enables us to visualize the heart directly in real-time using ultrasound and can help in such situations in diagnosing AMI by detecting any regional wall motion abnormality. It is also useful for the amount of myocardium at risk and final infarct size after reperfusion therapy; evaluation of patients with unstable hemodynamic findings and detecting mechanical complications.

The diagnosis of ST-elevation in myocardial infarction (STEMI) is usually based on patient's history and ECG findings,[1] but it should be taken into account that patients may present with atypical symptoms, also it takes cardiac enzymes some time to elevate above the normal range after the onset of chest pain. Hence, 2D echo in patients with acute STEMI needs to be performed early.

The present observational study was carried out at a tertiary healthcare center with prior approval of institutional ethics committee for a study period of 2 years to find out early 2D echo findings in patients of acute ST-elevation myocardial infarction and to access the benefit of 2D echo within 24 hours of admission over the prognosis of acute STEMI.

Materials and Methods

A total of 100 diagnosed patients with the first episode of acute ST-elevation myocardial infarction who reported in the emergency were included in the study and who have given written informed consent. Patients <18 years of age, prior history of acute myocardial infarction, known case of valvular or congenital heart disease, known case of cardiomyopathy, patients undergoing cardiac surgery. Patients who were diagnosed as non-ST-elevation MI, and finally patients who did not give consent for the study were excluded.

[1]Department of Medicine, Govt Medical College, Aurangabad, Maharashtra, India

[2,3]Department of Medicine, Govt Medical College, Jalgaon, Maharashtra, India

Corresponding Author: Vijay Gaikwad, Department of Medicine, Govt Medical College, Jalgaon, Maharashtra, India, Phone: +91 9422274644, e-mail: drvijaygka@gmail.com

How to cite this article: Mankar S, Gaikwad V, Ganeriwal A. Early Diagnosis of Complications of ST-elevation Myocardial Infarction with 2D Echocardiography. J Mahatma Gandhi Univ Med Sci Tech 2021;6(1):17–20.

Source of support: Nil

Conflict of interest: None

On admission, patients with typical or atypical ischemic symptoms and ECG changes of ST-segment elevation of >0.1 mV (1 mm) in leads 2, 3, avF, V4, V5, V6, 1, and avL; and in leads V2, V3 >0.2 mV (2 mm) in males >40 years, >0.25 mV in males <40 years, and >0.15 mV in females; in 2 contiguous leads were diagnosed as acute STEMI.[2–8]

Of these patients, those patients who have had fulfilled all inclusion criteria and did not meet any exclusion criteria were then included in the study after taking written informed consent. Detailed clinical history was taken and clinical examination was done. These included patients were then classified according to ECG changes as follows and also based on KILLIP classification (Tables 1 and 2).

2D echo/Doppler was performed on all patients within 24 hours of admission to evaluate the overall performance with the help of

Table 1: Location of infarct, artery affected, and ECG features[9–12]

S. no.	*Site of infarct*	*ST-elevation*	*ST depression*
Anterior MI (AMI)			
1	Anteroseptal MI	v1–v4, qRBBB	2, 3, aVF
2	Anterolateral MI	1, avL, v2–v4	± v5 and v6
3	Anteroapical MI	v4–v6, occasionally 2, 3, aVF	avL
4	Extensive anterior wall MI	1, avL, v1–v6, ±avR, qRBBB	2, 3, aVF
5	Lateral wall MI	1, avL,± v5–v6	2, 3, aVF
6	Extensive anterolateral wall MI	1, avL, v1–v6, avR > v1	2, 3, aVF
Inferior MI (IMI)			
1	Inferoposterior	2, 3, aVF, v1, v3R, v4R	1, avL, ± v2–v3
		2, 3, aVF	±v1–v3
2	Inferolateral	2, 3, aVF, v5–v6, ± 1, avL	v1–v3, avR
Posterior MI (PMI)	Posterior MI	v7–v9	v1–v3
RVMI[142]	RVMI	v1, v4r–v6r	v2

Table 2: Killip classification[13]

Grades	*Clinical characteristics*	*Mortality (%)*
I	No clinical signs of heart failure	6
II	With rales in the lungs, third heart sound (S3), and elevated jugular venous pressure,	17
III	With acute pulmonary edema (rales > half of lung fields)	38
IV	With cardiogenic shock or arterial hypotension (measured as systolic blood pressure < 90 mm Hg), and evidence of peripheral vasoconstriction (oliguria, cyanosis, and diaphoresis)	81

the PHILIPS model HD11XE. 2D echo parameters used for examining the patient included left ventricular internal dimension at the end of systole (LVIDs), left ventricular internal dimension at end of diastole (LVIDd), left ventricular ejection fraction (LVEF), regional wall motion abnormality (RWMA). Depending on the wall motion pattern, patients were be classified as normokinetic, hypokinetic, akinetic, and dyskinetic. If more than one regional wall motion abnormality was detected, a predominant abnormality was assigned. Diastolic dysfunction, mitral regurgitation, papillary muscle rupture, free wall rupture, interventricular wall rupture, pericardial effusion, clots, or other mechanical complications of acute myocardial infarction were evaluated with 2D echo/Doppler studies.

Observations and Results

In our study, age of patients ranged from 27 to 86 years. Maximum, 29 (29%) patients were seen in the age-group of 61–70 years, followed by 27 patients (27%) in the age-group of 41–50 years, followed by 22 patients (22%) in the age-group of 51–60 years. The mean age was 56.65 (SD = 12.76). In a study of 132 patients of AMI conducted by Adhikari et al.,[14] out of 119 patients of STEMI, maximum patients 33 (27.72%) were found in the age-group of 61 to 70 years. Abraham et al.,[15] in a study of 100 patients of AMI, found that a total of 27 patients (27%) were in the age-group of 61–70 years, which was highest among all the age-groups studied. These above-mentioned studies have a common finding of maximum patients in the age-group of 61 to 70 years and comparable with the finding of our study (Fig. 1).

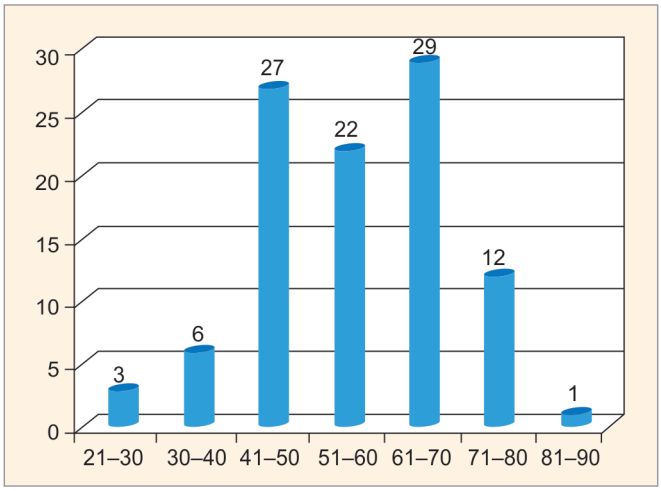

Fig. 1: Age distribution

Table 3: Gender distribution for the patients

Gender	*No of cases*	*% of cases*
Male	69	69
Female	31	31
Total	100	100

In our study, total males were 69 (69%) and total females were 31 (31%). Dr Hafiz Mughees Ather[16] in his study found 76% of males and 23% of females. Lerner et al.[17] had reported 60% of all coronary events were in male patients. Channamma et al.,[18] on analysis of gender distribution, found 92.5% were males and only 7.5% were females. Shabbir et al.[19] studied 250 patients out of which, 186 (74.4%) were males and 64 (25.6%) were females (Table 3).

In our study, we found that maximum patients, 55 (55%) were classified as anterior MI as per ECG changes, followed by the inferior MI category in which 21 patients (21%) were there. In a study carried by Shivpuje et al.,[20] 29 (58%) out of 50 patients had anterior wall AMI, 14 (28%) out of 50 patients had inferior wall AMI, and 7 (14%) out of 50 patients had Global MI. Jewitt et al.,[21] in their study of 222 patients of AMI, found that 124 cases (55.8%) had anterior wall AMI and 75 cases (33.8%) had inferior wall AMI. These studies had

a common finding that anterior MI was more common than other types (Table 4).

In our study, we found that LV clot formation as maximum with aneurysm (75%). It was detected on 2D echo in 5 out of 11 patients with dyskinesia (45.45%) with dyskinesia, In an akinetic group of 6 patients LV clot was found on 2D echo in 2 patients (33.33%), and least incidence was in the hypokinetic group, out of 79 patients 1 was having LV clot (1.26%). Kodilkar et al.[22] found that 3.3% of patients with hypokinesis, 26.3% with akinesis, and 66.7% with dyskinesis had LV thrombus. This is in correspondence with our study in that LV clot formation frequency increases as RWMA increases from hypokinesia to aneurysm. Lamas et al.[23] found that the frequency of LV thrombus goes on increasing with increasing wall motion abnormality. All the patients in our study who have had LV thrombus were having RWMA (Table 5).

In our study, the mean LVEF was 43.48 (SD = 13.43), we observed that mortality is much higher in the LVEF <40% group of patients than it is in LVEF >40% group. That is out of 34 patients with LVEF <40% 17 patients (50%) died when compared with only 3 deaths (4.68%) among 64 patients who have had LVEF >40%. Toth et al.[24] in their study found that ejection fractions of <40 were associated with increased mortality. We also observed that severe heart failure as represented by Killip class 3 or 4 is much more common, in patients with LVEF <40%, i.e., 47.05% of patients with LVEF <40% had Killip class 3 or 4 when compared with only 4.68% of patients of Killip class 3 or 4 with LVEF >40%. Darbar et al.[25] found that patients in which signs and symptoms of LVF (Killip class 3 or 4) are present have a mean LVEF of 40%. Dr Hafiz Mughees Ather[16] in his study found that patients with LVF have a mean LVEF of 37.13%. These findings are consistent with our findings frequency of LVF increases in the population having LVEF <40% than in patients of LVEF 40% (Table 6).

In our study, mechanical complications of AMI were detected on 2D echo, of which, mitral regurgitation was found in 23 patients (23%), the ventricular septal rupture was found in 3 patients (3%), ventricular free wall rupture in 2%, papillary muscle rupture in 11%, pericardial effusion was found in 13%, and LV clot was found in 11% patients. Kodilkar et al.[22] found in their study, mitral regurgitation in 11 of the total 55 patients studied (20%), ventricular septal defect in 1 patient(1.8%), and pericardial effusion in 2 patients (3.6%) (Table 7).

Thus, it can be concluded acute myocardial infarction is seen more commonly in the age-group of 61–70 years and it is more common among males. Killip classification of patients has prognostic value and helps in accessing the severity of myocardial infarction. Anterior MI is common than other types of acute myocardial infarction. LV clot formation is more common in patients with aneurysms and dyskinesia. As the severity of RWMA increases from hypokinesia to aneurysm frequency of LV clot formation also increases. Among AMI patients those with LVEF <40% are associated with more frequencies of LVF and mortality than those with LVEF >40%.

Mechanical complications of AMI can be detected by 2D echo and can aid accordingly in treatment. Thus, early 2D echo done within 24 hours of admission can predict patients at risk, can identify mechanical complications, and can aid in the treatment accordingly.

Table 4: Distribution of patients according to the type of AMI on ECG findings

Type of AMI	*Frequency*	*%*
Anterior MI (AMI)	55	55
Inferior MI (IMI)	21	21
Posterior MI (PMI)	2	2
RVMI	1	1
Combination of any of AMI, IMI, PMI, RVMI	21	21
Total	100	100

Table 5: Regional wall motion abnormality (RWMA) types and LV clot

Type of RWMA	*Frequency*	*LV clot formation*	*% of LV clot formation*
Hypokinesia	79	1	1.26
Akinesia	6	2	33.33
Dyskinesia	11	5	45.45

References

1. Kumar A, Cannon CP. Acute coronary syndromes: diagnosis and management, part ii. Mayo Clin Proc 2009;84(11):1021–1036. DOI: 10.1016/S0025-6196(11)60674-5.
2. Nikus K, Pahlm O, Wagner G, et al. Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-invasive

Table 6: LVEF and mortality and severe left ventricular failure (Killip class 3/4)

Ejection fraction	*Frequency*	*Patient died*	*% of patients died*	*Killip class 3/4*	*% of patients with Killip class 3/4*
<40%	34	17	50	16	47.05
>40%	64	3	4.68	3	4.68
Total	100	20	–	19	–

Table 7: Mechanical complications of AMI

Mechanical complications of AMI	*Frequency (yes)*	*Frequency (no)*	*Total*	*% of total patients*
Mitral regurgitation (MR)	23	77	100	23
Ventricular septal rupture (VSR)	3	97	100	3
Ventricular free wall rupture (VFWR)	2	98	100	2
Papillary muscle rupture (PMR)	11	89	100	11
Pericardial effusion (PE)	13	87	100	13
LV clot	11	89	100	11

Electrocardiology. J Electrocardiol 2010;43(2):91–103. DOI: 10.1016/j.jelectrocard.2009.07.009.
3. Schweitzer P, Keller S. The role of the initial 12-lead ECG in risk stratification of patients with acute coronary syndrome. Bratisl Lek Listy 2001;102(9):406–411.
4. Steg PG, James SK, Atar D, et al. ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 2012;33(20):2569–2619. DOI: 10.1093/eurheartj/ehs215.
5. Thygesen K, Alpert JS, White HD, et al. Universal definition of myocardial infarction: Kristian Thygesen, Joseph S. Alpert and Harvey D. White on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction Eur Heart J 2007;28(20):2525–2538. DOI: 10.1093/eurheartj/ehm355.
6. Bairey CN, Shah PK, Lew AS, et al. Electrocardiographic differentiation of occlusion of the left circumflex versus the right coronary artery as a cause of inferior acute myocardial infarction. Am J Cardiol 1987;60(7):456–459. DOI: 10.1016/0002-9149(87)90285-2.
7. Birnbaum Y, Drew BJ. The electrocardiogram in ST elevation acute myocardial infarction: correlation with coronary anatomy and prognosis. Postgrad Med J 2003;79(935):490–504. DOI: 10.1136/pmj.79.935.490.
8. Fuchs RM, Achuff SC, Grunwald L, et al. Electrocardiographic localization of coronary artery narrowings: studies during myocardial ischemia and infarction in patients with one-vessel disease. Circulation 1982;66(6):1168–1176. DOI: 10.1161/01.cir.66.6.1168.
9. Kanei Y, Sharma J, Diwan R, et al. ST-segment depression in aVR as a predictor of culprit artery and infarct size in acute inferior wall ST-segment elevation myocardial infarction. J Electrocardiol 2010;43(2):132–135. DOI: 10.1016/j.jelectrocard.2009.09.003.
10. Rhinehardt J, Brady WJ, Perron AD, et al. Electrocardiographic manifestations of Wellens' syndrome. Am J Emerg Med 2002;20(7):638–643. DOI: 10.1053/ajem.2002.34800.
11. Sadanandan S, Hochman JS, Kolodziej A, et al. Clinical and angiographic characteristics of patients with combined anterior and inferior ST-segment elevation on the initial electrocardiogram during acute myocardial infarction. Am Heart J 2003;146(4):653–661. DOI: 10.1016/S0002-8703(03)00369-7.
12. Tierala I, Nikus KC, Sclarovsky S, et al. Predicting the culprit artery in acute ST-elevation myocardial infarction and introducing a new algorithm to predict infarct-related artery in inferior ST-elevation myocardial infarction: correlation with coronary anatomy in the HAAMU Trial. J Electrocardiol 2009;42(2):120–127. DOI: 10.1016/j.jelectrocard.2008.12.009.
13. Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two-year experience with 250 patients. Am J Cardiol 1967;20(4):457–464. DOI: 10.1016/0002-9149(67)90023-9.
14. Adhikari G, Baral D. Clinical profile of patients presenting with acute myocardial infarction. Int J Adv Med 2018;5(2):228–233. DOI: 10.18203/2349-3933.ijam20181068.
15. Abraham MM, Reddi V, Periyasamy S, et al. Prescribing patterns of thrombolytics in acute MI and their outcome, (April 2017). 2018. 49–51.
16. Ather H. Left ventricular ejection fraction after acute myocardial infarction. 2008;15(2):234–239.
17. Lerner DJ, Kannel WB. Patterns of coronary heart disease morbidity and mortality in the sexes- A 26-year follow up of Framingham study. Am Heart J 1986;111(2):383–390. DOI: 10.1016/0002-8703(86)90155-9.
18. Channamma G. Age and gender distribution in patients with acute myocardial infarction. 2016;5(1):29–31.
19. Shabbir M, Kayani AM, Qureshi O, et al. Predictors of fatal outcome in acute myocardial infarction. J Ayub Med Coll Abbottabad 2008;20(3):14–16.
20. Shivpuje A. Echocardiographic assessment of left ventricular function in chronic alcoholics. J Assoc Physicians India 1997;45(10):769–770.
21. Jewitt DE. Incidence and management of supraventricular arrhythmias after acute myocardial infarction. Lancet 1967;2(7519):734–738. DOI: 10.1016/s0140-6736(67)91943-5.
22. Kodilkar J, Patil M, Chafekar N, et al. Role of early 2D echocardiography in patient with acute myocardial infarction in correlation with electrocardiography and clinical presentation. MVP J Med Sci 2014;1(July):51–55.
23. Lamas GA, Vaughan DE, Pfeffer MA. Left ventricular thrombus formation after first anterior wall AMI. AM J Cardiol 1988;62(1):31–35. DOI: 10.1016/0002-9149(88)91360-4.
24. Toth C, Csomos M, Vadnay I. Significance of early echocar-diography in acute myocardial infarct. Orv Hetil 1997;138(13):787–791.
25. Darbar D, Gillepsie N, Choy AM, et al. Diagnosing left ventricular dysfunction after myocardial infarction: the Dundee algorithm. QJM 1997;90(11):677–683. DOI: 10.1093/qjmed/90.11.677.