ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

Status of Emergent Reperfusion Therapy in Patients with Acute Coronary Syndrome Having ST Depression in Right Precordial Leads with Non-Reassuring ST Elevation in Posterior Leads

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ABSTRACT

Background: ST depression in right pre-cordial leads is the most common under-diagnosed and untreated infarction pattern of isolated posterior wall myocardial infarction (PMI). Guidelines emphasizes to treat above electrocardiographic changes in patients with acute coronary syndrome(ACS) with emergent reperfusion therapy, after confirming the same as posterior wall MI, detecting ST elevation (≥0.5 mm) in two or more posterior leads. However ST elevation may not be manifested in Posterior leads in some cases of PMI due to damping of electrical signals arising from posterior aspect of the heart by air of lungs.

Objective: To evaluate clinico-angiographic profile and outcomes of patients with ACS having ST depression in anterior leads with non-reassuring ST elevation in posterior leads

Methods: Total 32 hospitalised patients with Acute Coronary Syndrome having horizontal or down sloping ST-depression (≥0.5 mm in two or more leads) in leads V1,V2,V3 without ST elevation (≥ 0.5mm in two or more leads) in posterior leads were finalised as study participants. Data about clinical, echocardiographic and angiographic findings were studied.

Results: Prevalence of mitral regurgitation, left ventricular failure and mortality is very high i.e 71.8%, 43.7%, 12.4% among patients respectively. Regional wall motion abnormality in the posterior wall observed invariably in all the patients. All the patients found to have total or subtotal occlusion of either RCA or LCX representing PMI.

Conclusion: ST depression in right precordial lead in patients with ACS most often represents PMI even without significant ST elevation in posterior leads. Hence there is a need for suitable criteria for PMI based on electrocardiographic manifestations in anterior leads, so that early and accurate diagnosis can be established to prevent devastating complications, that arise from deprivation of emergent reperfusion therapy.

Key Words: LVF, MR, Occlusion, Posterior wall MI, posterior leads, Right anterior precordial leads.

Introduction

ST depression in right precordial leads is one of the most common under diagnosed and untreated infarction pattern in isolated posterior wall MI (PMI). A decade ago, around 99.6% patients with ST-depression in right precordial leads had been managed with non-emergent percutaneous coronary intervention(PCI).[1]Therefore ACCF/AHA STEMI guidelines of 2013 recommended a

ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

term "transmural posterior injury" for ST depression in V1-V4 to increase the awareness about above ECG manifestation of PMI among primary contact physicians.[2] ESC-2017 guideline and Universal definition of AMI (2018) also affirmed higher possibility of isolated posterior wall MI in patients with ACS (Acute Coronary Syndrome) having isolated ST depression in V1-V3, supporting AHA guideline.[3,4]Above guidelines also emphasized to treat acute coronary syndrome (ACS) patients with above ECG changes with emergent reperfusion therapy, after confirming the same as posterior wall MI, detecting ST elevation (≥0.5 mm) in two or more posterior leads(v7,v8,v9).[2,3,4] However posterior leads have their own inherent limitation. ST elevation may not be manifested in Posterior leads in some cases of PMI due to damping of electrical signals arising from posterior aspect of heart by the air of lungs. Around 12% of patients having PMI with anterior lead presentation do not manifest ST elevation (≥1 mm) in posterior leads .[5] The sensitivity of detecting PMI from posterior leads increases from 49% to 94%, only when ischemic ST threshold adjusted from 1 to 0.5 mm [6], which is close to upper normal value.[7] Therefore there is a chance of misdiagnosis of posterior wall MI as NSTEMI (Non ST Elevated MI) in the absence of ST elevation due to limitation of posterior leads. However none of the existing guideline provided a standard protocol regarding early diagnosis and prompt management of these cases having typical ECG finding of PMI in anterior leads without ST elevation in posterior leads. Present observational study will highlight clinical and angiographic outcomes of such rare subgroup of patients with PMI, who presented to the primary contact physicians within window period of 12 hours, but didn't get the benefit of reperfusion therapy in a timely fashion due to delay in diagnosis .

Materials & Methods

A cross-sectional study was conducted in the cardiology department of VIMSAR, Burla, considering total 312 patients hospitalized between Decemebr-2013 to December-2020 with Acute Coronary Syndrome (ACS) having the ECG findings of horizontal or down sloping ST-depression (≥0.5 mm) in two or more right precordial leads (V1,V2,V3) (figure I) were included in the study. Patients with ST depression in right precordial leads having ST elevation (≥ 0.5mm) in two or more posterior leads (V7, V8, V9) were excluded from the study. Patient with prior history of CAD, pulmonary hypertension, congenital or valvular heart disease, cardiomyopathy, chronic kidney disease were also excluded from the study. A total of 32 patients having horizontal or down sloping ST-depression (≥0.5 mm) in leads V1,V2,V3 without significant ST elevation (≥ 0.5mm) in two or more posterior leads were finalized as study participants (figure. II). Consent taken from all the patients enrolled. Baseline characteristics e.g., age, sex, traditional risk factors like diabetes mellitus, hypertension, smoking, dyslipidemia were evaluated. The time span from the symptom onset until presentation to the department and door-to-balloon time were recorded in each study subjects. Presenting clinical symptoms e.g., chest pain, dyspnoea, fatigability, syncope and signs e.g., s3, crepitation, elevated jugular venous pressure were noted in each patient. Notably the clinical signs (like S3, lung crepitation) among the study participants were classified based on KILLIP class at the time of presentation. Bed side chest X-ray was performed in all cases to evaluate presence of pulmonary venous hypertension. Serial ECG was done in all the patients to observe any arrhythmia or dynamic/fresh ECG changes. Urgent bedside echocardiography was conducted in all the study subjects to evaluate regional wall motion abnormality (RWMA),mitral regurgitation, left ventricular function and any other mechanical complication e.g., papillary muscle rupture or ventricular septal rupture etc. MR in patients was graded according to the severity. All the participants, who gave consent, were subjected to angiographic evaluation to find out the site, number, nature and severity of lesion as well as TIMI flow. Severity of lesions were further graded as total or sub-total occlusion, severe (>70% occlusion), borderline (50-70% occlusion), mild (<50% occlusion). Data with respect to baseline characteristics, clinical profile, electrocardiographic, echocardiographic and angiographic findings were collected and

ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

analyzed. Variables were presented as absolute numbers, percentage, proportions and mean with standard deviation.

FIGURE-I: Electrocardiographicpicture showing ST depression in right anterior precordial leads

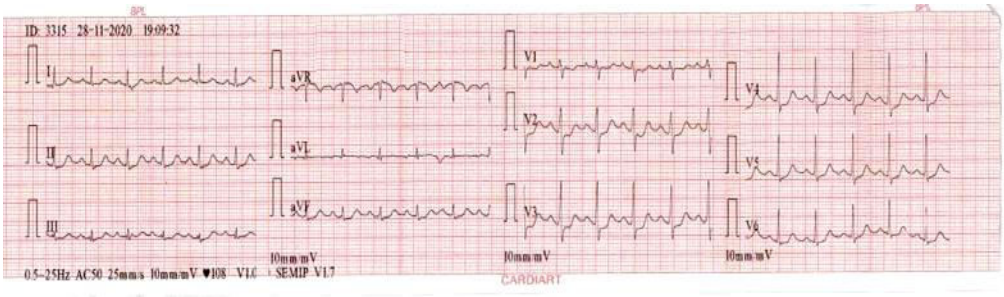
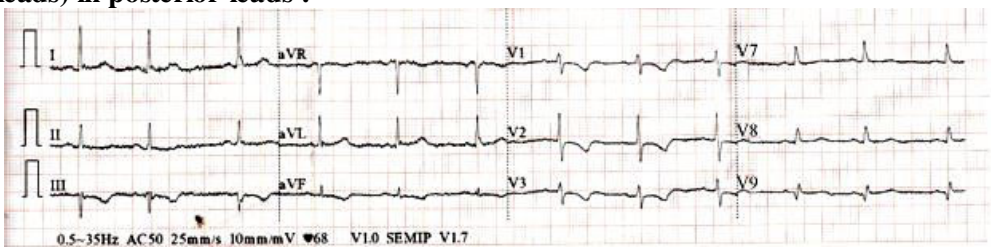


FIG-II: Electrocardiographic picture showing ST depression (≥0.5 mm in two or more leads) in right anterior precordial leads without having ST elevation (≥0.5 mm in two or more leads) in posterior leads .



Results

Amongst the 32 study participants, 21(65.6%) were males and 11(34.3%) patients were females. The mean age of the patients was 58 ± 12 years. Most common risk factor found among the patients was diabetes mellitus in 14 (43.7%) cases followed by HTN 12(37.5%) cases.

As none of the patients had additional ST elevation (≥0.5 mm in two or more leads) in posterior leads, patients were diagnosed as NSTEMI by primary contact physicians and referred to this institute for non-emergent PCI. Therefore all the patients reached to the cardiology department beyond the window period (>12 hours) due to delayed diagnosis. Mean time from symptom onset till presentation to this department was (1014 ± 1526) minutes. Mean door to balloon time in patients was (1800± 1584) minutes.

Chest pain was the most common clinical presentation observed in 15(46.9%) patients.14(43.75%) patients presented with dyspnea with LVF (left ventricular failure), out of which 7 (21.87%) patients were in frank pulmonary edema (KILLIP class-III) and one (3.12%) patient was in cardiogenic shock (KILLIP class-IV)(Figure 3,Table 1). Two (6.2%) patients developed cardiac arrest within 30 minutes of arrival to hospital and could not be revived even after resuscitation. 6 (18.75%) patients subsequently developed subtle ST elevation in the inferior leads later during hospitalization as depicted in figure-IV. However 3(9.37%) patients developed transient Chronic Heart block (CHB). Short episode of AF was observed in 1(3.12%) patient and another patient developed VT who was revived with cardio-version (table I). All the patients with left ventricular failure, hemodynamic instability, recurrent or on-going chest pain, dynamic ECG changes and malignant arrhythmia underwent urgent PCI. Rest of the patients planned for early angiography. 8(25%) patients underwent routine elective PCI after giving consent for the same. One patient, who underwent urgent PCI, died suddenly due to cardiac arrest on 2nd post PCI day and another patient died as a consequence of cardiogenic shock on 5th post PCI day.

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FIGURE III. Distribution of cases according to KILLIP class at the time of presentation

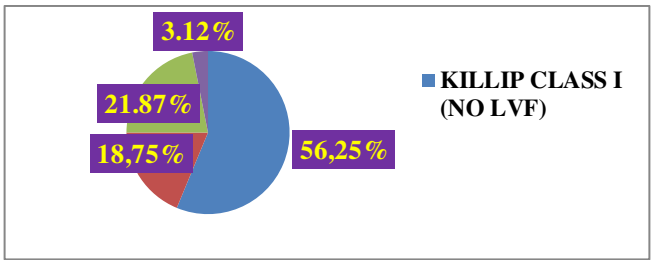

FIG-IV: Electrocardiographic picture of a patient with ACS showing subtle ST elevation in inferior leads with ST depression in-right precordial leads

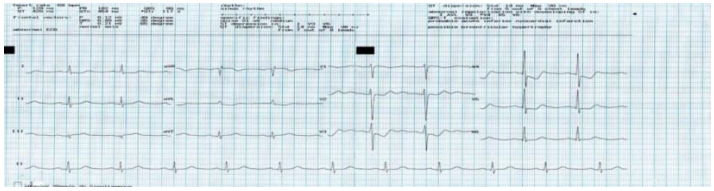

TABLE-I: Showing Clinical Spectrum of the Study Participants

SL No	CLINICAL SPECTRUM OF PATIENTS		Number of patients (N)	Proportion of patients (%)
1.	Left Ventricular Failure (LVF)	KILLIP CLASS-II (mild LVF)	6	18.75
		KILLIP CLASS-III (Pulmonary oedema)	7	21.87
		KILLIP CLASS-IV (cardiogenic shock)	1	3.12
2.	Mitral regurgitation	Mild	11	34.3
		Moderate	7	21.8
		Severe	5	15.6
3.	Electro-cardiographic abnormality	Dynamic ECG changes in inferior leads	6	18.7
		Atrial fibrillation	1	3.12
		Complete heart block	3	9.37
		Ventricular tachycardia	1	3.12
4.	Papillary muscle rupture		1	3.12
5	Pericardial effusion		1	3.12
6	Ventricular Septal rupture		0	0
7	Death		4	12.4

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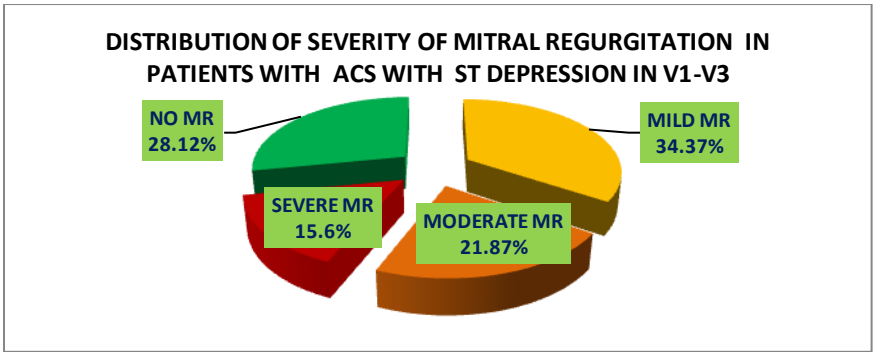
MR was observed in 23 (71.84%) patients, out of which 11 (34.37%), 7 (21.87%), and 5(15.6%) patients found to have mild, moderate and severe regurgitation respectively (figure V). One (3.12%) patient had Papillary muscle rupture (table I), for which the patient was referred to a Centre with cardio-thoracic-vascular surgery (CTVS) back up for coronary artery bypass surgery and mitral valve repair after stabilization, as the present Centre do not have CTVS back up. Left ventricular dysfunction was observed in 9 (28.1%) patients. Mean ejection fraction (EF) of the patients in percentage was (53 ± 9). Regional wall motion abnormality in the posterior wall observed in all patients during bedside echocardiography.

Figure V. Distribution of Mitral Regurgitation (MR) in terms of Severity in Patients



A total of 27 out of 32 patients underwent angiography after obtaining written formal consent. Three patients did not give consent for angiography. Two patients died within 30 minutes of arrival due to cardiac arrest even after resuscitation, therefore angiography could not be done. 21(77.8%) patients out of 27 patients having ST depression in V1-V3 (without any dynamic ECG changes) found to have total or subtotal occlusion of left circumflex artery (LCX) (figure VI, VII, Table II). Total LCX occlusion with grade-0 TIMI flow was observed in 16 (59.2%) patients and subtotal occlusion observed in 5(18.5%) patients. Amongst patients with subtotal LCX occlusion, 3 patients and 2 patients had grade-II & grade-I TIMI flow respectively. Mid portion of LCX was found to be the most common site of lesion in 15 (55.5%) patients followed by distal LCX in 6 (22.2%) patients. Whereas 6 out of 27 (22.2%) patients, developed ST elevation in inferior leads along with ST depression in V1-V3 later during hospitalization, found to have total or subtotal occlusion in mid or distal Right Coronary Artery (RCA) (figure VII, Table 2). Total RCA occlusion with grade-0 TIMI flow was observed in 5(18.5%) patients and sub-total RCA occlusion with grade-II TIMI flow observed in 1(3.7%) patient. 4 (14.8%) patients had lesion in mid RCA and rest two (7.4%) patients had lesion in distal RCA. Double vessel disease found in 7(21.8%) cases, triple vessel disease observed in 3(9.36%) cases (Table II

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TABLE: II- Case wise angiographic findings of patients with isolated or maximal ST depression V1-3

Case No.	LCX	RCA	LAD	Case No.	LCX	RCA	LAD
1	Mid total occlusion	Normal	Normal	15	Distal Subtotal occlusion	60% mid discrete	70% distal diffuse
2	Distal total occlusion	Normal	Normal	16	Mid total occlusion	40% distal diffuse	Normal
3	Mid Subtotal occlusion	30% distal diffuse	85% mid diffuse	17	Mid total occlusion	Minor irregularity mid	Normal
4	Mid total occlusion	Normal	Normal	18	Mid total occlusion	30% distal tubular	40% mid discrete
5	Distal total occlusion	40% proximal discrete	Minor irregularit y mid	19	Mid total occlusion	50% distal tubular	Normal
6	Mid subtotal occlusion	Normal	Normal	20	Mid total occlusion	60% mid diffuse	Normal
7	Mid Subtotal occlusion	Normal	60% Proximal discrete	21	Mid total occlusion	Minor irregularity mid	Normal
8	Distal total occlusion	Minor irregularit y mid	30% proximal discrete	Patients developed subtle inferior wall ST elevation along with ST depression in v1-v3			
9	Mid total occlusion	Normal	Normal	1	75% distal discrete	Distal total occlusion	Normal
10	Distal Subtotal occlusion	Minor irregularit y proximal	Normal	2	Normal	Mid total occlusion	50% proximal tubular
11	Mid total occlusion	Normal	30% proximal	3	Minor irregularity proximal	Mid total occlusion	Normal
12	Mid total occlusion	60% distal diffuse	75% distal diffuse	4	60% mid OM diffuse	Mid total occlusion	50% proximal diffuse
13	Mid total occlusion	Normal	Normal	5	30% mid diffuse	Distal Subtotal occlusion	Minor distal irregularity
14	Distal total occlusion	Normal	65% Distal discrete	6	Normal	Mid total occlusion	Minor distal irregularity

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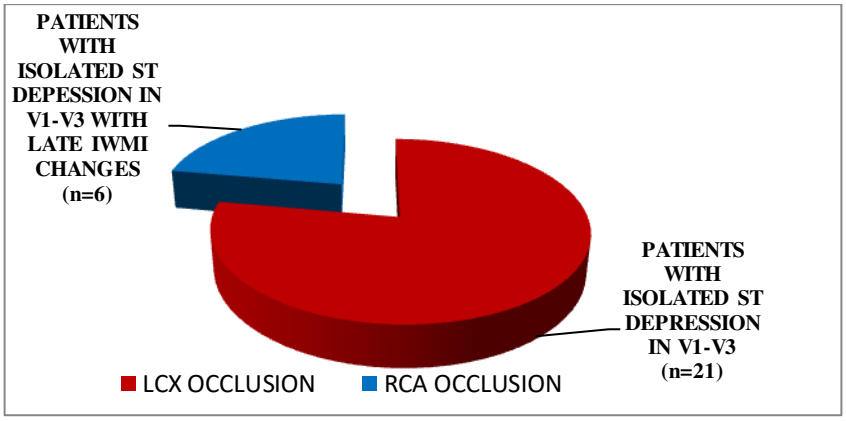
[LAD- left anterior descending, LCX-left circumflex artery, RCA- right coronary artery]

FIGURE VI: Angiographic picture of a patient with ACS having ST depression in right precordial leads showing left circumflex artery occlusion

FIGURE VII: Angiographic Profile of Patients with ACS having ST Depression V1-3



[IWMI-inferior wall myocardial infarction, LCX-left circumflex artery, RCA- right coronary artery]

DISCUSSION:

Acute coronary syndrome presenting with isolated ST segment depression in right precordial leads (V1-V3) represents a diagnostic challenge for the primary physicians. It is most often diagnosed as NSTEMI in the absence of ST elevation in posterior leads, though it represents an early electrocardiographic sign of acute posterior wall MI. Therefore patients with above ECG features, presents to primary contact physicians within window period of 12 hours, but do not get the benefit of emergent reperfusion therapy in a timely fashion and referred to higher tertiary center's for non-emergent PCI with a provisional diagnosis of NSTEMI. Due to delay in recognition of PMI, patients most often reach very late to the primary PCI capable center's presenting with life threatening mechanical complications like MR, LVF associated with PMI.

In the current study 14(43.7%) patients presented with LVF. 7 (21.8%) patients presented with frank pulmonary edema (KILLIP class-III). 1(3.1%) patient presented with cardiogenic shock (KILLIP class-IV) [figure-III, table I].4(12.4%) patients died due to complication of LVF. RWMA in postero-basal wall observed invariably in all patients during bed side echocardiography. MR was observed in 23(71.8 %) patients, out of which moderate to severe MR noticed in 12(37.5%) patients [figure V]. Higher prevalence of MR in isolated posterior wall MI can be explained with the facts as described below. Anatomically posterior wall is confined to

ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

small infero-basal segment on the diaphragmatic surface of heart,[8] but topographically posterior wall is closely related to posteromedial papillary muscle. [9] In contrast to Anterolateral Papillary muscle, Posteromedial papillary muscle is very thin and is supplied by single coronary artery either from RCA or LCX[10] and hence more susceptible to ischemia related mechanical complications like papillary muscle dysfunction or rupture resulting acute ischemic MR. Therefore despite borderline or normal LV ejection fraction in PMI, patients most often presents with lethal complications like LVF, pulmonary edema, cardiogenic shock, leading to higher mortality equivalent to that of anterior wall MI.[11]In the present study mortality among patients was around 12.4%. High mortality observed in current study is most probably due to high prevalence of moderate to severe MR, which developed after deprivation of emergent reperfusion therapy due to delayed diagnosis. Moderate to severe MR is a significant predictor for hospitalization which leads to HF and death, independent of age, EF.[12] Presence of even mild MR in patients with MI also carries an adverse prognosis.[13] So even if area of infarcted myocardium is small in PMI, prevalence of MR, LVF is very high among these groups of patients, leading to higher mortality equivalent to anterior wall MI.

In the present study while studying angiographic profile of patients, it was observed that, all the patients with ACS with ECG feature of ST depression in V1-V3,who underwent angiography, had total or subtotal occlusion of LCX, indicating isolated posterior wall MI (figure VI, figure VII, table I). LCX was also found to be the single most culprit vessel in isolated PMI in a study, while studying exclusively posterior lead for PMI.[14] In the current study total or subtotal occlusion of RCA was observed in all the patients who developed subtle ST elevation of inferior wall during hospitalization along with ST depression in V1-V3 (figure VII, table I). Similar dynamic ECG pattern also noticed in a study, while inflating RCA and LCX lesions with balloon during angioplasty in patients with single vessel disease. ST elevation in inferior leads was invariably observed in all the patients with RCA group, whereas ST elevation in posterior leads observed in all the patients with LCX group.[15] In present study, total or subtotal occlusion were observed either in mid or distal part of respective culprit artery in all the patients, but none of the patients had occlusion in the proximal part of the culprit artery, which can be explained with the fact below. As proximal RCA or dominant LCX occlusion is invariably associated with RV MI, therefore ST depression in anterior precordial leads in posterior wall MI most often neutralized by ST elevation in anterior precordial leads in RVMI.[16] Above discussion implies that, ST depression in right anterior precordial leads (V1-V3) in patients with ACS most often represents either mid or distal RCA or LCX occlusion indicating posterior wall MI. Also numerous studies concluded that ST depression in right anterior precordial leads is an electrocardiographic signs of PMI and not due to sub-endocardial ischemia.[16-21] Sub-endocardial ischemia due to LAD occlusion usually manifests as diffuse ST depression in anterior leads, usually deepest in V4-V6 leads[22-27], as overall average vector of depolarization of the entire left ventricle directs more towards the apex.[28] Whereas secondary ST depression in case of PMI most often remain localized to V2-V3 leads, as endocardial surface of posterior wall faces V2,V3.[28]ST depression in V2, V3 is also an important predictor of posterior wall MI and can differentiate occlusive MI form non occlusive ischemia with 96% specificity.[29] Secondly ST depression in both V2,V3 invariably observed in all cases of isolated of posterior wall MI in a retrospective study.[30] Therefore the diagnosis of acute posterior wall myocardial infarction can be based on the electrocardiographic criteria of ST depression in V1-V3.However under STEMI paradigms, ST elevation in posterior leads is the only necessary criteria for decision making regarding emergent reperfusion therapy in PMI. Around 1/4th of patients with acute coronary occlusion (most commonly in LCX), miss the opportunity of emergent reperfusion therapy, while strictly following ST elevation criteria for the diagnosis of AMI.[31] Around 2/3rd patients with acute LCX occlusion does not present ST elevation in ECG, may miss the opportunity for emergent reperfusion therapy.[29,32] Whereas recent OMI (Occlusive Myocardial infarction) paradigm does not consider the criteria of ST segment

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elevation for emergent reperfusion therapy, when an ECG pattern firmly indicates acute coronary occlusion. Hence there is a need to make a suitable criteria based on ECG manifestation in right anterior precordial leads, so that diagnosis of PMI can be confirmed by the primary contact physicians, by virtue of which important decisions about emergent reperfusion therapy can be under taken at an earliest.

LIMITATION OF STUDY: This is a single Centre, observational study. As the prevalence of such rare subtype of isolated posterior wall MI is low among MI patients, therefore a multi-centric study enrolling more number of study participants can provide much better idea about the scenario.

CONCLUSION: Recording of posterior leads may not always confirm posterior wall MI in patients with ACS with ST depression in V1-V3. Isolated or maximal ST depression in V1-V3 in patients with ACS most often represents posterior wall MI even without significant ST elevation in posterior leads. Hence there is a need for a suitable criteria for PMI based on electrocardiographic presentation on anterior lead, so that early and accurate diagnosis can be established by the primary contact physicians, to prevent devastating complications in patients, that arise from deprivation of emergent reperfusion therapy.

ACKNOWLEDGEMENTS

- Authors would like to thank our patients for their adherence and kind cooperation in this study.
- Funding: No funding sources
- Conflict of interest: None declared
- Ethical permission: taken from institutional authority

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