Review Article

# Diagnostic challenges during recognition of isolated posterior wall myocardial infarction

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## Abstract

**Introduction:**Patients with isolated PMI is most often misdiagnosed as anterior subendocardial ischemia and get deprived of emergent reperfusion therapy. Therefore patients with PMI often presents with dreadful complications like acute ischemic MR,LVF,leading to higher mortality equivalent to anterior wall MI despite normal or borderline EF. Therefore a qualitative analytic study was conducted after collecting and compiling related literatures and guidelines to derive a conclusion about early and accurate diagnosis of isolated PMI. During analysis we obtained useful information as described below. Prominent R wave in V1-V2 takes averagely 33 hours to develop after onset of symptom, therefore it is unobserved in early golden hour of PMI. However it was included as a sign of acute myocardial ischemia in a recent guideline.As precordial ST depression is the one and only important electrocardiographic signs in early golden hours of PMI,therefore above ECG changes most often misdiagnosed as anterior subendocardial ischemia. Most of the important guidelines considers only isolated pattern ST depression (≥ 0.5 mm) as isolated posterior wall MI. Whereas diffuse pattern(v1-v6) is the common pattern in isolated PMI, therefore such pattern most often misdiagnosed as subendocardial ischemia. As ST depression in V2,V3 is a specific marker of PMI, therefore diffuse patterns with maximal ST depression in V2,V3 may be considered as a criteria for PMI .Guidelines suggests recording of posterior leads in patients with ACS with ST depression (≥0.5 mm) or non-diagnostic ECG changes in right anterior precordial leads in an attempt to fit PMI into ST elevation criteria of STEMI paradigm , whereas the same was not practiced most often by training or resident physicians due to lack of knowledge and awareness. However recent OMI paradigm,which is superior to STEMI paradigm in terms of accuracy,does not strictly considers ST elevation criteria for emergent reperfusion therapy, when ECG entity firmly concludes acute coronary occlusion. **Conclusion**: Presence of R wave in V1,V2 is a late evolved electrocardiographic sign of PMI and it doesn't warrants emergent reperfusion therapy.Isolated (v1-v3 ) or diffuse (v1-v6) pattern with maximal ST depression (≥0.5 mm) in V2,V3 is a specific electrocardiographic sign of acute or hyper-acute posterior wall MI and seeks emergent reperfusion therapy . Whereas non diagnostic ECG like minuscule ST depression (< 0.5 mm ) in anterior precordial leads can represent isolated posterior wall MI and may require further confirmation by screening echocardiography or recording posterior leads .

**Keywords**: Isolated posterior wall MI, R wave in v1,v2,ST depression in v2,v3,posterior leads



## Introduction

Around seven decades ago in the pre-imaging era, an eminent pioneer of heart Perloff conveyed an excellent insight about its electrocardio-graphic manifestation of posterior myocardial infarction. He considered that as there is no corresponding lead that overlies the posterior wall and heart is sitting in a strictly anterior posterior position,therefore vector of necrosis in posterior wall travels from the back to the front to manifest mirror image electrocardiographic changes in anterior precordial leads , as a reflection of electrical events registered in posterior wall .Therefore PMI presents electrocardiographically as reciprocal ST depression on the standard anterior leads along with prominent R wave in V1,V2 secondary toST elevation ,Q wave registered in posterior leads respectively. Perloff further emphasized that,R wave V1-V2 is a confirmatory sign of PMI ,which can differentiate PMI from anterior wall ischemia[1].Recognition of isolated posterior wall MI most specifically during early golden hours represents a diagnostic challenge for physicians.Isolated ST depression in right anterior precordial leads is the predominant electrocardiographic manifestation during acute or hyper acute stages of PMI .[2] As per a study, 99.6% patients with isolated ST-depression in right precordial leads were treated with non-emergent PCI due to misdiagnosis of PMI as anterior subendocardial ischemia .[3] It has been also noticed that, patients with PMI often reach late to primary PCI capable centres with mean DTB (door to balloon time) of around 30 hours due to delay in diagnosis of PMI .[4] As a result patients do not get the benefit of emergent reperfusion therapy and develop life threatening mechanical complications associated with PMI. Although area of infarction in posterior wall MI restricted to a small inferobasal segment of heart,[5] however clinical presentation in isolated posterior wall MI is very devastating due to associated complications like mitral regurgitation arising from posterior papillary muscle dysfunction and rupture.Posterolateral papillary muscle is topographically closely related to inferobasal segment of heart [6] and supplied by a single blood vessel either LCX or RCA without having any collateral.[7]On the contrary its counterpart anteromedial papillary muscle is supplied by both LAD and LCX . [7] Therefore chances of development of ischemia related

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complication like papillary muscle dysfunction is around 3 times higher in patients in posterior wall MI as compared to its counterpart anterior wall MI.[8]Around 2/3rd of patients with isolated posterior wall MI develops MR during hospitalisation , out of which around half of the patients develops moderate to severe MR.[9,10]Therefore despite borderline or normal LV ejection fraction in PMI,patients most often presents with lethal complications like LVF , pulmonary oedema ,cardiogenic shock , leading to higher mortality (=21%) comparable to that of anterior wall MI.[11]Prevalence of true posterior wall MI is 15-21 % of among all STEMI.[12] As per a recent report, prevalence of posterior wall MI increases to 38%, when bed side echocardiography included for evaluation of MI .[11] Posterior wall MI most often accompanies with inferior wall MI or lateral MI.[13,14] Whereas it is very difficult to diagnose isolated posterior wall MI from standard ECG leads more specifically in acute or hyper acute stages of MI . As per literature, prevalence of isolated posterior wall MI is 3.3-11% patients with acute MI .[15,16] However as isolated posterior wall most often underdiagnosed electrocardiographically, therefore actual prevalence may be much higher than it was estimated .

As patients with isolated PMI is most commonly underdiagnosed and undertreated due to either misdiagnosis or delayed diagnosis and presents with dreadful complications like acute ischemic MR,LVF , leading to higher mortality equivalent to anterior wall MI,therefore early and accurate diagnosis of isolated posterior wall is extremely essential to prevent short and long term morbidity and mortality ,that result from deprivation of emergent reperfusion therapy .Hence a qualitative analytic study conducted after collecting and compiling related literatures and guidelines to derive a conclusion about early and accurate diagnosis of PMI.

**Methodology:** All the literatures and guidelines related to diagnosis of posterior wall MI were collected and compiled. Each components of diagnostic criteria of PMI were analysed separately and collectively to derive strength and limitations of componentsby comparing different literatures and guidelines.Limitations of each components which can lead to misdiagnosis and late diagnosis of PMI,were further studied extensively .Issues arising from limitations of each components were analysed. Alternative solutions and ideas to prevent the issues related to misdiagnosis or delayed diagnosis of PMI, was further derived from recent updated literatures.

**Guidelines based diagnosis of isolated posterior wall MI**

Guidelines provides a recent updates in the diagnosis of diseases. Over decades together different literatures highlighted PMI as most common underdiagnosed and undertreated infarction among AMI, however most of the updates were done in the field of diagnosis of PMI over last decade only, which were mentioned below.

1. European Society of Cardiology (ESC) 2017 guidelines defined posterior wall MI as "isolated ST-segment depression (≥0.5 mm)in leads V1-V3 with ST elevation (≥0.5 mm ) in leads V7-V9".It recommended mandatory recording of posterior leads for confirmation of diagnosis of PMI in patients with ACS with isolated ST depression in v1-v3.Guideline further mentions that ,isolated posterior wall MI often corresponds to the left circumflex territory.Isolated ST-segment depression ≥ 0.5mm in leads V1–V3 is the dominant electrocardiographic finding in isolated posterior wall MI.[17]
2. The 2013 ACCF/AHA STEMI guidelines suggest that "ST depression in 2 or more leads of V1-4 may indicate transmural posterior injury" .[18]
3. AHA/ACC 2017 Clinical Performance and Quality Measures for Adults With ST-Elevation and Non–ST-Elevation Myocardial Infarction suggesteda term"STEMI equivalent" for isolated PMI in view of emergent reperfusion therapy.[19]
4. The Fourth Universal Definition of MI in 2018 defines acute myocardial ischemia as "new horizontal or downsloping ST-depression 0.5 mm or greater in two contiguous leads and/or T inversion >1 mm in 2 contiguous leads with prominent R wave or R/S ratio >1". Guideline mentions that "Isolated ST-segment depression ≥0.5 mm in leads V1–V3 may indicate left circumflex occlusionand suggestive of inferobasal myocardial ischemia (previously termed posterior infarction), especially when the terminal T wave is positive (ST-elevation equivalent); however, this is nonspecific."It strongly recommends recording of posterior leads in patients with ST-segment depression in leads V1–V3 .Guideline fixes higher cut-off point (i.e≥1 mm)for ST elevation in posterior leads specifically in men <40 years old patients to increase the specificity during diagnosis of PMI .[20]
5. The 2009 Standardization of ECG Interpretation Guidelines defines isolated PMI as 0.05 mV of abnormal J-point elevation in leads V7-V9 or "abnormal J-point depression of -0.05 mV in V1-V3 or -0.1 mV in all other leads" without qualification of what indicates abnormal J-point depression.[21]
6. The National Cardiovascular Data Registry (NCDR) defines isolated PMI as "ST elevations in leads V7-V9 or ST depressions maximal in V1-V3, without ST elevation in other leads is considered a STEMI equivalent and qualifies for emergent reperfusion."[22]

**Challenges during recognition of isolated posterior wall MI**

PMI diagnosed primarily based on three important electrocardio-graphic components included in guideline criteria's, i.e a) prominent R wave in V1,V2 , b) ST depression in anterior precordial leads c)ST elevation in posterior leads. Each components has own limitations and strengths. Strength of each components enhances accuracy of diagnosis, whereas limitations of each components leads to misdiagnosis or late diagnosis creating challenges during recognition of PMI.

*R wave in v1,v2*

1.Universal definitions of AMI (2018) included typical perloffs sign of PMI i.e prominent R wave in V1,V2 as a component in the diagnostic criteria of "acute myocardial ischemia ", though guideline not clarified whether the definition is for posterior wall MI or lateral wall MI .[20] Whereas recent AHA consensus with evidence of CMR imaging confirms that, the vector of inferobasal segment ( that confines to posterior wall) directs towards v3 instead of V1,V2 due to slightly oblique position of heart in thorax .[5] Also the inferobasal segment depolarises around 40 msec later than other parts of ventricular myocardium.[23] So there is hardly any possibility to develop R wave in V1,V2 in posterior wall MI. Whereas lateral wall MI may register R waves in leads V1 and V2 because the lateral wall infarction vector faces these leads.[5] Although there is increased awareness about this concept among cardiologists , the concept yet to be included in guidelines,so that clarity about PMI among primary physician can be ascertained.

2. R wave in V1,V2 is a mirror image of Q wave registered in posterior wall,[1] is a sign of late evolved AMI.[24] Therefore R wave in V1,V2 hardly seen in hyper acute and acute stage of AMI. As per a literature it takes averagely 33 hours to develop after onset of symptoms.[2]In above context it seems not reasonable to include R wave in V1,V2 as a component in the diagnostic criteria of acute myocardial ischemia ,as patients with R wave in v1,v2 representing late evolved MI, can receive emergent reperfusion therapy, which is not beneficial rather detrimental to them.

3.As RBBB is a marker of ACS and can manifest secondary ST depression in anterior leads,therefore there is chance of misinterpretation of isolated RBBB with isolated posterior wall MI and vice versa.[25]However in patients with isolated RBBB , T waves are usually asymmetrical ,whereas patients with posterior wall MI T wave are usually symmetrical. [24] Bedside screening echocardio-graphy or recording of posterior lead can provide a clue to differentiate isolated PMI from isolated RBBB.[11]

As prominent R wave also seen other cardiac diseases like primary/ secondary pulmonary arterial hypertension , congenital heart disease like pulmonary stenosis, atrial septal defect ,therefore chances of

misdiagnosis likely in patients with isolated PMI in late stage also . [12].Therefore it becomes very important in the part of emergency physician to distinguish isolated PMI from other cardiac diseases. Recoding of posterior leads can only help in differentiating isolated posterior wall MI from other cardiac diseases . However as recording of posterior lead can't assess EF and severity of MR in PMI , therefore it is preferable to do a screening echocardiography, if it is available , so thatseverity of MR can be assessed to prognosticate the patients with PMI.

*ST depression in anterior leads*

1. Different guidelines defined PMI based on criteria of ST depression in anterior leads, however uniformity not maintained among guidelines while defining pattern of precordial ST depression in PMI.Majority of guide line recommended v1-v3 leads for diagnostic criteria of PMI,[17,20-22]whereas AHA recommended v1-v4 leads for the same .[18] One group of guidelines suggested isolated pattern of ST depression in right precordial leads for the diagnosis of PMI,[17,20] whereas other group of guidelines suggested diffuse or maximal ST depression pattern for the same . [21-22]Most of the guidelines considered ST depression (≥ 0.5mm) in right precordial lead as a most possible criteria rather than confirmatory criteria for PMI and recommended recording of posterior leads for confirmation. [17-18,20] Whereas other guidelines suggested that ST depression (≥ 0.5mm) in right anterior precordial lead as confirmatory criteria for PMI.[21-22]Lack of uniformity among guidelines, created a complexity during diagnosis of PMI. So it becomes difficult in the part of physicians to choose a pattern to follow for the diagnosis of PMI. Therefore a uniform and universal criteriafor the diagnosis of posterior wall MI is extremely essential for prompt and accurate diagnosis of PMI.

2.Most of the important guidelines consideredisolated ST depression (≥ 0.5mm) in right precordial leads (v1-v3) as a most probable criteriafor acute PMI and recommended recording of unconventional posterior leads for confirmation.[17,20]However numerous studies evaluated different patterns of ST depression right anterior leads like v1-v3, v2-v3 ,v1-v4,v2-v4etc and concluded that ST depression in right anterior precordial lead is an electrocardiographic signs of PMI and not a sign of subendocardial ischemia .[2,13,26-30] Subendocardial ischemia due to LAD occlusion usually manifests as diffuse ST depression in anterior lead, usually deepest in V4-V6 leads,[31-36] as overall average vector of depolarization of the entire left ventricle directs more towards the apex .[37] Whereas secondary ST depression in case of PMI most often remain localised to v2-v3 leads , as endocardial surface of posterior wall faces v2,v3 .[37] In a retrospective study, ST depression (≥ 0.5mm) in both v2,v3leads was observed invariably in all cases of posterior wall MI[4]. ST depression in v2,v3 is also a marker of LCX occlusion indicating posterior wall MI with 96% specificity[28] Therefore isolated pattern of ST depressions in right precordial leads (v1-v3) with maximal depression in v2,v3 can be considered as a most specific criteria for isolated posterior wall MI.

3.Most of guidelines considered only isolated pattern of ST depression for diagnosis of isolated posterior wall MI,[17,20] whereas diffuse patterns were common in isolated posterior wall MI. [4] Therefore diffuse pattern with isolated posterior wall MI most often misdiagnosed as subendocardial ischemia.While studying involvement of leads in posterior wall MI, it has been observed in a study that, most of the pattern were diffuse with involving leads V4 ,V5 and V6 in 86%,64%, 36% cases respectively .[4] As ST depression in v2,v3is specific and invariably observed in all cases isolated posterior wall MI ,[4,28] therefore diffuse pattern with maximal ST depression in v2,v3 leads can differentiate isolated posterior wall MI from anterior subendocardial ischemia in case of diffuse involvement of anterior leads.National Cardiovascular Data Registry also recommends maximal ST depression in V1-V3 as a criteria for PMI .[22]

Around half of patients with ACS presents to the ED with truly diagnostic ECG. [38-41] The other half have non diagnostic ECG, which can be in the form of either electrocardiographic signs of ischemia or nonspecific ST-T changes, or a completely normal ECG .[38-41] Non diagnostic minuscule ST depression (< 0.5 mm ) in anterior precordial leads can represent isolated posterior wall MI. Therefore guidelines recommends recording of posterior leads in patients with ACS with non-diagnostic suspicious ECG finding. [17-20]

*ST elevation in posterior lead*

Under STEMI paradigms, ST elevation is the only necessary criteria for decision making regarding emergent reperfusion therapy. In order to fit PMI in to STEMI criteria, many guidelines recommended mandatory recording of posterior lead in patients with ACS with ST depression in right precordial leads for confirmation of PMI.[17-20] On the process, sensitivity of detecting PMI was increased after adjusting cut off value for ST elevation in posterior leads.[42] However in many studies, it has been observed that,posterior leads are not recorded routinely in most of the health centres.ED staff even in cardiac centres do not record posterior leads either due to cumbersome procedure or lacks the knowledge to do so in PMI .[43] Only around 10% cardiologists and emergency physicians routinely practice posterior leads.[44] Less than 40% of physicians advise recording of posterior leads in hospitalized patients with ACS.[45] Around 80% doctors(most of them are resident physicians) don't have an idea about correct positioning of posterior leads and diagnosis of PMI based on ST criteria in posterior leads.[46] It implies that,although Guidelines and literatures made an attempt to fit isolated posterior wall MI into criteria of STEMI paradigm in order to increase the sensitivity of detection of PMI, however the attempt not produced any dynamic impact in the perception and recognition of isolated posterior wall MI except in the small groups of clinicians who have special interest in such literatures. Therefore guidelines need to be updated in the formal books of medical graduates,so that training or resident physicians will become aware of diagnosis of PMI from ST elevation in posterior leads instead of prominent R wave in V1-V2.

Although many studies enumerated usefulness of posterior leads in the diagnosis of isolated posterior wall MI,[9,47] however it is very essential to have an idea about its limitations ,as described below. Posterior leads may not record ST elevation in some cases of PMI due to damping of electrical signals arising from posterior aspect of heart by air of lungs ,left atrium, pulmonary vein . Around 12% patient of PMI with anterior lead presentation does not show significant (≥ 1mm)ST elevation in posterior leads.[48]The sensitivity of detecting PMI from posterior lead increases from 49% to 94% , only when ischemic ST threshold adjusted from 1 to 0.5 mm, [42] which is close to upper normal value.[49] Therefore if diagnosis of PMI is obvious from the ECG findings in the anterior leads meeting ST depression criteria of guidelines, then recording of the posterior leads can be falsely reassuring. Guidelines recommends recording of posterior leads to detect ST segment elevation for confirmation of PMI.[17-20] Guidelines also mentioned that, isolated posterior wall MI most often corresponds to LCX territory.[17,20] However ST-segment elevation is an insensitive marker of left circumflex artery occlusion .[50,51] Only around 1/3 rd of patients with ACS with LCX occlusion manifest ST elevation in corresponding leads facing ischemic zone .[28] Therefore there may be chance of missing PMI, while strictly following posterior lead ST elevation criteria, when ECG finding in anterior leads strongly suggestive of PMI.

As a consequences,Patients with PMI presents to the emergency department during golden hours ,most often misdiagnosed as NSTEMI and do not get the benefit of emergent reperfusion therapy and develops devastating complications like LVF, pulmonary oedema, cardiogenic shock and finally death . Whereas patients with late evolved MI may receive emergent reperfusion therapy like

thrombolysis or PCI, which is not beneficial, rather detrimental for them. [4]

Emerging Trends In The Field Of Diagnosis Of Posterior Myocardial Infarction (Older Vs Recent)

STEMI paradigm strictly considersST elevation criteria as requisitefor emergent reperfusion therapy. On the contrary 36% patients meeting ST elevation criteria on ECG do not presents withacute coronary occlusion when referred for primary PCI .[52]Around 25-30% patients with acute coronary occlusion (most commonly in LCX), miss the opportunity of emergent reperfusion therapy, due to misassumption of STEMI as NSTEMI.[53]Patients with isolated posterior wall MI are not an exception to it. In an attempt to justify emergent reperfusion therapy in PMI,different terminologies like "Transmural posterior ischemia" and "STEMI equivalent" had been suggested for PMIby guidelines .[18-19]In order to fit ST depression in v1-v3/v1-v4 into ST elevation criteria of STEMI paradigm ,cut off point for ST elevation in unconventional and unpredictable posterior leads was also adjusted close to upper normal value .[49] However as PMI does not confines to dominant STEMI paradigm, therefore above efforts have not produced any dynamic impact in perception or recognition of acute PMI among physicians.Whereas recent OMI paradigm which is superior to STEMI paradigm in terms of accuracy for the ECG diagnosis of Occlusion MI ,does not strictly considers ST segment elevation as a requisite criteriafor emergent reperfusion therapy,when an ECG entity firmly concludes acute coronary occlusion. As ST depression (isolated/diffuse) in anterior lead with maximal depression in v2,v3 firmly concludes LCX occlusion ,it should be considered as a confirmatory criteria for isolated PMI. OMI Paradigm also emphasizes expert opinion and echocardiography to diagnose PMI .In a recent study blinded expert ECG interpretation found to be superior to STEMI criteria for the ECG diagnosis of Occlusion MI.[50,51] Echocardiographic diagnosis of PMI was found to be more accurate than electrocardiographic diagnosis of PMI.[11] Newer advanced technique like strain imaging echocardiography can detect occlusive myocardial infarction in patients with ECG features mimicking NSTEMI.Therefore patients with ACS withnon-diagnostic ECG changes like minuscule ST depression (< 0.5 mm ) in anterior leads should undergo screening echocardiography to confirm PMI .If echocardiography is not available ,posterior leads can be recorded for confirmation. Expert opinions should be taken in such patients, while taking important decisions about emergent reperfusion therapy.

**Conclusion**

Presence of R wave in V1,V2 is a late evolved electrocardiographic sign of PMI and it does not warrants emergent reperfusion therapy . Isolated (v1-v3) or diffuse (v1-v6) patterns with maximal ST depression(≥0.5 mm ) in V2,V3 is a specific electrocardiographic sign of acute or hyperacute posterior wall MI and seeks emergent reperfusion therapy.Whereas non diagnostic minuscule ST depression (< 0.5 mm ) in right anterior precordial leads may require further confirmation by screening echocardiography or recording posterior leads . Expert opinions should be taken in patients with ACS with above non diagnostic ECG changes, while taking important decisions about emergent reperfusion therapy.

**Abbreviations**: PMI-posterior wall MI, NSTEMI-Non ST elevated myocardial infarction ,ECG- electrocardiogram, STEMI-ST elevated myocardial infarction, LAD-left anterior descending artery, LVF-left ventricular failure, OMI-occlusive myocardial infarction, MI-myocardial infarction, MI-myocardial infarction, EF-ejection fraction, PCI-percutaneous coronary intervention, AHA-American heart association,ESC-european society of cardiology,

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