Case Report

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Acute Isolated Posterior Myocardial Infarction; Challenges in Recognition and Management in the Emergency Department

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Abstract

Posterior wall myocardial infarction (PWMI) accounts for about 15-20% of all STEMIs and is usually seen in the context of inferior and/or lateral wall MI [2]. Isolated posterior wall MI are much less common, of about only 3.3% of all myocardial infarcts [1].

The clinical presentation of PWMI may not be very specific and is confusing even for a cardiologist. Moreover the lack of ST elevation in a standard 12-lead ECG leads to missed or delayed diagnosis of a true PWMI. We are reporting a case of isolated PWMI in a 65 years old, previously healthy male patient, who presented with only gradual onset shortness of breath, who was later found to have 100% LCx stenosis. We have tried to emphasize some facts that may make the clinicians aware of a possible PWMI.

Keywords: ST Elevation Myocardial Infarction (STEMI); LCx; PWMI; Posterior ECG Leads V_7 V_8 V_9; Right Coronary Artery (RCA); Left Anterior Descending Artery (LAD); ST Depression; Dominant R Wave; Flip Test; Coronary Angiography (CAG); Troponin I; Percutaneous; Coronary Intervention (PCI); Stenting.

Introduction

PWMI is caused by necrosis of dorsal and infra-atrial part of left ventricle located beneath the atrioventricular sulcus [1].

The majority of PWMI are associated with occlusion of left circumflex artery (LCx) [3-5] but they sometimes may also be associated with right coronary artery (RCA) occlusion.

LCx is the dominant vessel in 10% population and is the least commonly infarcted coronary artery.

PWMI is usually associated with either inferior MI or with lateral wall MI or both where ST elevation can be seen in the respective leads in ECG but when this occurs in isolation ECG diagnosis becomes very difficult. When PWMI is associated with inferior or lateral MI, the area of infarction is very extensive and is associated with high mortality [11,12].

The risk factors for PWMI are same as that of other myocardial infarctions like diabetes, hypertension, hyperlipidemia, smoking etc.

True PWMI is difficult to recognise because the leads of the standard 12-lead electrocardiogram are not a direct representation of the area involved. Only with indirect changes in the precordial leads as such the diagnosis can be suspected.

As the posterior myocardium is not directly visualized in a standard 12-lead ECG, reciprocal changes are seen in the anteroseptal leads V_1-V_3 [2].

The ECG changes [2] of a true PWMI in a standard 12-lead ECG as seen in leads V_1-V_3 are as follows:

- Horizontal ST depression (more consistent finding)
- Tall and slightly broad R waves (30ms)
- Upright T waves
- Dominant R wave in V_2 (R/S>1).

However all of these changes may not be present and that makes the diagnosis even more difficult based on ECG alone.

Any patient with ischemic symptoms and horizontal ST depression in anteroseptal leads must be suspected to have a PWMI.

The anteroseptal leads are directed from the anterior precordium towards the internal surface of the posterior myocardial wall. Because posterior electrical activity is recorded from the anterior side of the heart, the typical injury pattern of ST elevation and Q waves becomes inverted; therefore the following changes occur [2].

- ST elevation becomes ST depression
- Q waves become R waves
- Terminal T-wave inversion becomes an upright T wave.

The addition of posterior leads V_7 to V_9 significantly increases the ability to detect posterior MI compared with the standard 12-lead ECG [6,7].

Posterior leads are placed at the following landmarks as shown below (figure 3).

Lead V_7 - at the level of lead V_6 at the posterior axillary line.

Lead V_8 - on the left side of the back at the tip of the scapula.

Lead V_9 - halfway between lead V_8 and the left paraspinal muscles.

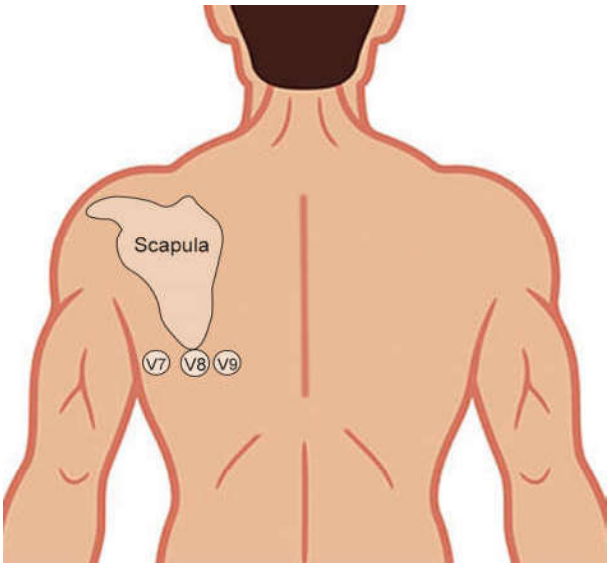

Fig. 1: Placement of posterior leads

When using posterior leads to diagnose PWMI, ST-segment elevation in leads V_7 through V_9 is defined as elevation of at least 0.5 mm in 2 or more of the leads on the basis of the increased distance between the posterior chest wall and the heart. Posterior ECG leads significantly improve sensitivity and specificity when identifying patients with isolated PWMI [7,8].

Many a Times A "Flip Test" [9] is Performed before doing the Posterior Leads ECG using the Following Steps

1. Get a standard 12 lead ECG
2. Turn it over 180 degrees to look at the back of the upside-down paper.
3. Aim the paper at a bright light source to enable seeing the "flipped" tracings.
4. ST elevation in these leads V1 – V3 with Q waves is consistent with posterior STEMI.

Other supporting investigations like cardiac markers and echocardiography can help in the diagnosis similar to any other types of myocardial infarction.

Case Study

A 65 years old male patient presented to ED at around 6am with c/o shortness of breath on and off since 2 weeks which got severely aggravated since 3am that woke him up from sleep.

There was no h/o chest pain, cough, nausea, vomiting, palpitations, syncope.

He did not give any h/o chronic illnesses nor was he on any regular medications. He was however an old chronic smoker.

He was taken to the monitored bed and initial evaluation done.

He was conscious, oriented but was tachycardic with PR = 108/min regular and tachypneic with RR = 26/min.

His oxygen saturation was 58% at room air which improved to 90% with oxygen supplementation @8LPM via face mask.

His BP was 150/90 mmHg and random blood sugar level was 263 mg/dl.

He did not have any pallor, cyanosis, icterus, jugular venous distension nor any peripheral edema.

Cardiac monitor showed sinus rhythm and the 12-lead ECG showed sinus rhythm with horizontal ST depression in V_1 to V_5.

Initial ECG of the patient is shown below (Figure 2).

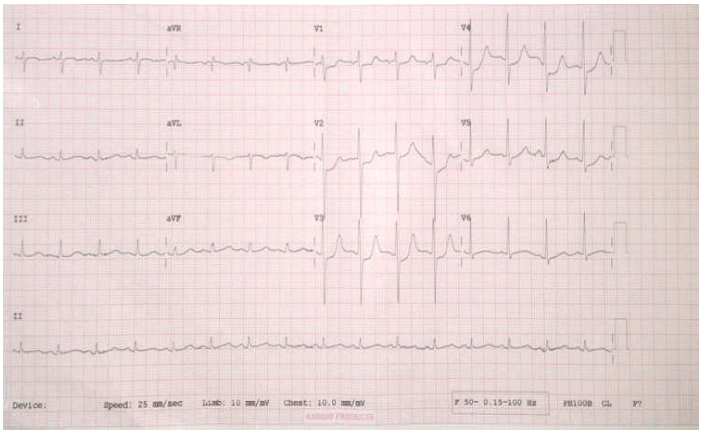

Fig. 2:

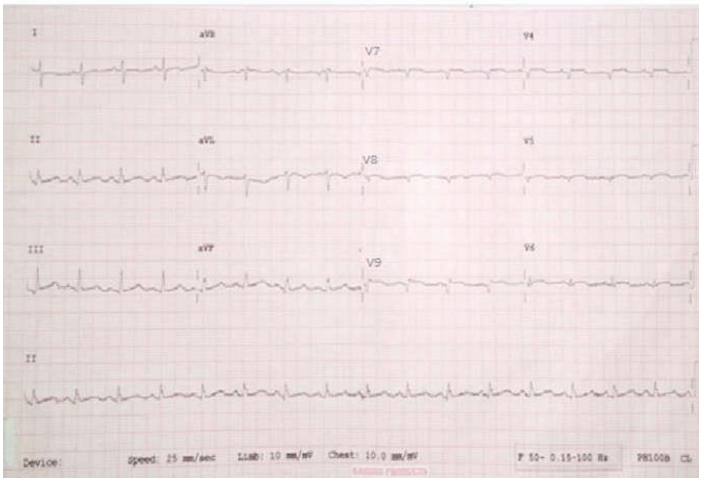

Fig. 3:

A large IV canula was inserted in left cubital vein and samples were taken for ABG, cardiac markers, D-dimers, and BNP.

Patient's ABG showed ph = 7.17, pO_2 = 75.2mmHg, Pco_2 = 50mmHg, HCO_3 = 17.6mmol/L, Na^+ = 134meq/L, K^+ = 4.5meq/L, Ca^{2+} = 1.16mmol/L

Chest X ray showed increased bronchovascular markings B/L. His systemic examination revealed minimal wheeze and basal crepitations on auscultation of lungs b/l and nothing else was significant.

Pt was initially evaluated by a junior Doctor in the ED and was treated in lines of acute exacerbation of COPD and the ECG was initially misinterpreted as either anterior wall ischemia or strain pattern of LVH.

He was given oxygen supplementation with BIPAP support, IV deriphylline, IV Hydrocortisone 200mg and IV Piperacillin +Tazobactum but his symptoms did not improve.

He was then reviewed by a senior ED doctor who after seeing the 1st ECG ordered a posterior leads ECG which is shown Above (Figure 3).

This ECG showed mild ST elevation in leads V_7 V_8 V_9 of >1mm and Q waves >2mm which strongly suggests posterior wall MI.

By then other laboratory tests showed CKMB 15.9 IU/L, raised myoglobin of 419ng/ml, raised tropinin-I of 17ng/ml, raised BNP of 1050pg/ml, and normal D-Dimer and normal urine ketone levels. 2-D echocardiography was done urgently in the ED which showed mild LVH with hypokinetic LCx territory and LVEF of 45% with moderate MR. A diagnosis of posterior wall myocardial infarction with LVF was made and he was given loading dose of Ecosprin 325, Ticagrelor 180 mg, Atorvastatin 80 mg and also was started on Furosemide infusion @ 5 mg/hour and NTG infusion @ 10mcg/min. He was then prepared and sent to cathlab for coronary angiography.

Course in the Hospital and Outcome

Coronary angiography revealed 100% occlusion in LCx and minimal blockage in RCA and LAD and a stent was placed in LCx after thrombosuction and tyrofiban injection resulting in good TIMI III flow.

He was kept in CCU for observation and was started on post-stenting medications.

His initial creatinine report was 1.2mg/dl but after the angiography it increased to 2.8mg/dl possible due to the contrast.

Nephrology consultation was requested and drug modification was done along with controlled fluid management and his creatinine came down to 1.1mg/dl on 5th day of hospitalization. His LVF also revolved after about 5 days of hospitalization.

He was then discharged in a stable condition after 1 week of hospitalization with Ecosprin 75mg HS, Clopidogrel 75mg BD, Rosuvastatin 40mg OD, Metoprolol 25mg BD, Nicorandil 5mg TDS, Furosemide 20mg BD, Ceftum 500mg BD, Alprazolam 0.25mg HS, Pantoprazole 40mg OD.

He was followed up in the cardiology OPD after 4 days and was found to be stable and symptom-free.

Discussion and Therapeutic Considerations

This case report illustrates a 65 years old male who had isolated PWMI. Coronary angiography (CAG) showed 100% LCx stenosis which was opened and a stent was inserted in the cathlab.

High suspicion by the ED doctor for a PWMI led to timely diagnosis and appropriate interventions to save the life of the patient. The patient's 1st ECG showed horizontal ST depression in V_1 to V_5 with tall R waves in V_2-V_6 with upright T waves in V_1-V_4 without any ST elevation in inferior or lateral leads.

On high suspicion for a PWMI, a posterior leads ECG was taken which showed ST elevation in V_7-V_9 that suggested PWMI. Troponin I was very high and echocardiography showed hypokinetic LCx territory which all confirmed high possibility of PWMI.

Lung crepitations, high BNP and low LVEF suggested left ventricular failure (LVF). Finally PWMI was confirmed in CAG and appropriate management was done with PCI (Percutaneous coronary intervention).

When PWMI is associated with either inferior or lateral wall MI, management is straightforward by giving anti-ischemic therapies and thrombolysis or PCI [10]. However the management of isolated PWMI is somewhat controversial [10]. One school of thought suggests the use of an approach similar to that used for NSTEMI; anti-ischemic, anti-platelet, anti-coagulation and then the patient is taken for CAG with or without PCI [10].

Others are of the opinion that isolated PWMI is an acute infarction and so the patient should undergo urgent PCI similar to management of STEMI; but there is not enough data to support this more aggressive management [10]. However the concept of opening the closed arteries as soon as possible thereby restoring perfusion to the damaged myocardium is likely the better option [10].

In our case it was a right decision to take the patient for urgent CAG+/-PCI; the procedure went uneventful and the patient recovered eventually.

Conclusion

Why should an emergency physician be aware of the challenges in recognition of an acute posterior wall MI!

This is because this is a STEMI and this requires urgent reperfusion of the myocardium but the diagnosis is often missed or delayed due to lack of typical symptoms and lack of the usual ST elevation of a standard 12-lead ECG.

High degree of suspicion and proper ECG knowledge of a PWMI and appropriate investigations are required for timely diagnosis and management for such a patient.

If there is unnecessary delay in identifying a PWMI due to lack of proper knowledge, there is high risk of ventricular dysfunction and death.

This report will highlight the electrocardiographic fine-tuned diagnosis of PWMI by using the posterior leads V_7 - V_9 leading to easier and faster recognition with consequences for treatment and improved prognosis.

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