Abstract:

Isolated lateral STEMI is less common, but may be produced by occlusion of smaller branch arteries that supply the lateral wall, e.g. the first diagonal branch (D1) of the LAD, the obtuse marginal branch (OM) of the LCx, or the ramus intermedius.

Recognition of a lateral STEMI:

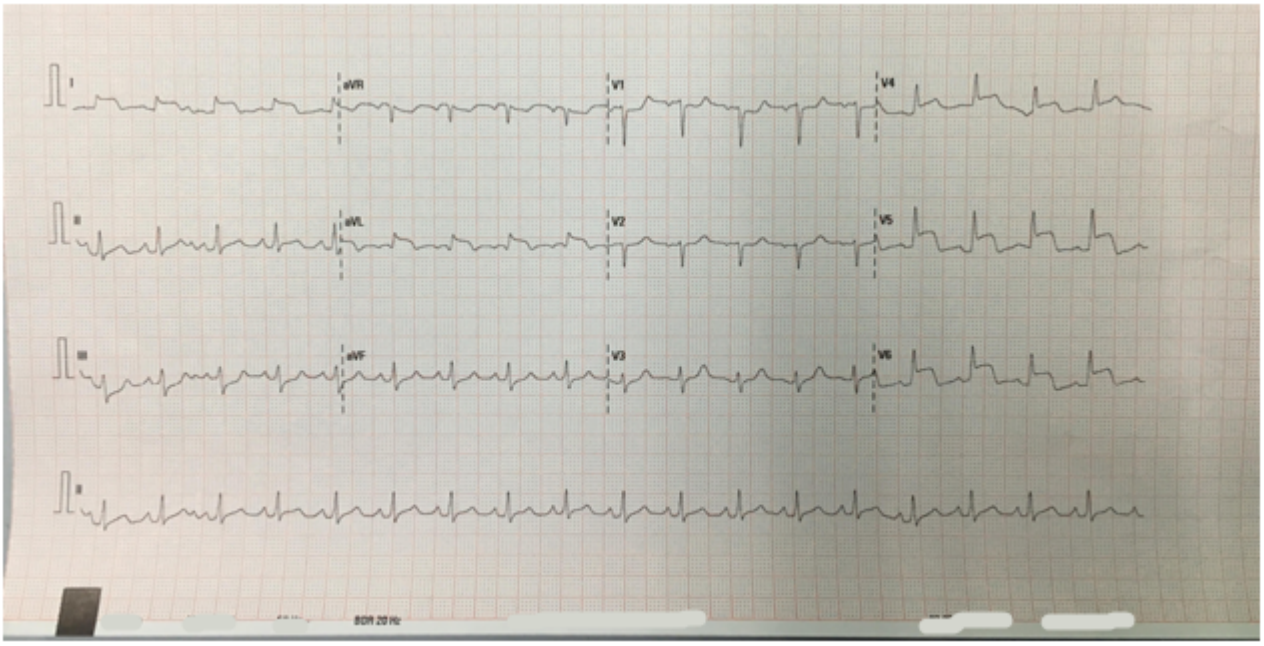
- ST elevation in the lateral leads (I, aVL, V5-6).
- Reciprocal ST depression in the inferior leads (III and aVF).
- ST elevation primarily localised to leads I and aVL is referred to as a **high lateral STEMI.**

CASE REPORT:

A 46yr old male patient with primary complaints of Fever since last 4 days for which he took self medication from a local pharmacy. After 3 days at around10am patient developed heaviness of chest with mild pain which was radiating to back, patient went to the same pharmacy and took inj.pantoprazole(i.v) after which symptoms apparently subsided. The next morning patient again developed similar symptoms and was taken to a local hospital where after doing ECG he was diagnosed to have ACS-STEMI. Patient condition deteoriated in the hospital and CPR was given(according to patient party) after ?Asystole. ROSC was achieved and the patient was immediately reffered to NH for further management.

Upon arrival in ER patient was still complaining of severe left sided chest pain. ECG showed Lateral Wall MI(ST elevation in the lateral leads – I, aVL, V5-6) and the patient was in Shock (? Cardiogenic / Vasopressors started immediately). Blood sample was taken for Trop-I and Cardiologist was informed immediately. Bed side 2D-Echo was done which showed Cardiac Tamponade and Transmural involvement was suspected. CTVS opinion was taken later and poor prognosis was explained to the patient party. In the mean time Trop-I report showed 8.56ng/ml.

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Discussion:

All patients suspected of having LMI should have a thorough history and physical exam performed. Details regarding the nature of chest pain including onset, aggravating/relieving factors and radiation can help make an important clinical decision. Patients should be examined for signs of excessive lipid accumulation such as xanthoma and xanthelasma. Physical findings such as diaphoresis, extra heart sounds, heart murmurs, and elevated jugular venous pressure can guide toward the diagnosis of LMI.[1] Patients suspected of having acute LMI should have a prompt evaluation with an electrocardiogram (ECG) and measurements of serial cardiac troponins.[2] Recognizing distinct ST-T involvement pattern can aid in early diagnosis of MI.

ECG Findings:

- *ST-Elevated LMI:* ST elevation in lead I, aVL, V5, and V6; Reciprocal ST depression in inferior lead III and aVF
- *High lateral STEMI:* High lateral STEMI can present as ST elevation involving lead I and aVL. Subtle ST elevation in V5, V6 and reciprocal changes in lead III and avF may be present. This is usually caused by occlusion of the first diagonal branch of LAD and is sometimes referred to as South African flag sign.
- *Old LMI presents with deep and broad Q waves I leads I and aVL*

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- *Inferolateral STEMI presents with ST-elevation involving lateral (I, aVL, V5, V6) and inferior leads (II, III, aVF):*This is usually seen with occlusion of the proximal LCx artery.
- *Anterolateral STEMI presents with ST-elevation involving lateral (I, aVL, V5, V6) and anterior leads (V1, V2, V3):*This is highly indicative of proximal LAD occlusion.

In patients with normal troponin and non-diagnostic ECG, a quick evaluation with echocardiogram can be done. Echocardiogram has high sensitivity and low specificity when diagnosing MI. [3]Severe ischemia produces regional wall motion abnormalities (RWMA) which can be visualized on echocardiogram. However, it is difficult to differentiate acute ischemia from old infarct based on RWMA. Patients with normal echocardiogram but having moderate pre-test probability should be evaluated with a stress test.[4]

LMI presenting as STEMI should be treated immediately. Early reperfusion has shown benefits with improved clinical outcomes.[5] Percutaneous intervention (PCI) has demonstrated superior results when compared to fibrinolytic therapy.[6] ACC/AHA guidelines for STEMI management recommend early PCI with preferable door to balloon time of fewer than 90 minutes at PCI capable facility and less than 120 minutes at non-PCI capable facility.[7] Antiplatelet therapy with aspirin and either P2Y12 inhibitor or Glycoprotein IIb/IIIa inhibitor is recommended before and after PCI. Multiple studies have demonstrated mortality benefits with beta-blockers and high-intensity high potency statins.[8]

LMI patients presenting as NSTEMI should receive initial medical therapy with oxygen (if hypoxic), beta-blocker, and a statin. Antithrombotic therapy including antiplatelet medications and anticoagulants such as unfractionated heparin should be initiated as soon as possible. NSTEMI patients have also shown better outcomes with early reperfusion strategy.[9]

LMI has overall favorable outcomes. Long-term outcomes in patients with STEMI and NSTEMI have improved over the last decade.[10]

Prognosis tends to be worse in patients presenting with life-threatening complications such as arrhythmias, sudden cardiac arrest, free wall/papillary muscle rupture and cardiogenic shock. Risk stratification using TIMI scores can help identify patients with increased in-hospital mortality.[11]

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Conclusion:

Widespread misinformation and ignorance of common men about Chest Pain/Heaviness which sometimes have similar symptoms as Gastritis have led to worst outcome for many. As medical intervension/attension usually gets delayed in such cases resulting in poor prognosis and sometimes even death.

Our patient was having chest pain/heaviness almost 24-hour before seeking medical help, he was taking PPI`s thinking he is having gastritis. By the time he came to see a doctor in his locality it was already very late and the time taken by him to reach our hospital after referral led to more damage.

It can finally be concluded that,“In any kind of Chest Dyscomfort medical attention should be taken immediately.“

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