

High Lateral Myocardial Infarction; the Smallest but the Most Painful Infarction – A Case Report

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ABSTRACT

Rationale: A high lateral ST-segment elevation myocardial infarction (STEMI) is well-defined small infarction. All types of MIs are mostly associated with chest pain. **Patient Concerns:** A 60-year-old married, officer, heavy smoker, Egyptian male patient presented with acute intolerable severe chest pain, and high lateral STEMI. **Diagnosis:** Hypertension and high lateral STEMI were the most probable diagnosis. **Interventions:** Electrocardiogram and streptokinase intravenous infusion. **Outcomes:** Dramatic response of acute high lateral STEMI to streptokinase. **Lessons:** The size of MI may not correlate with the severity of chest pain. A high lateral STEMI is considered the smallest one but accompanied by the highest chest pain.

Key words: High lateral myocardial infarction, lateral myocardial infarction, myocardial infarction, the smallest but the most painful infarction

INTRODUCTION

igh lateral ST-segment elevation myocardial infarction (STEMI) is a pattern of ST-segment elevation caused by acute occlusion of the first diagonal branch (D1) of the left anterior descending coronary artery (LAD-D1). The location of the most admirable ST-segment deviations looks like the shape of the South African flag.^[1-3] South African flag sign considered a teaching way for easier electrocardiogram (ECG) understanding of high lateral MI (LMI).^[3] Acute D1 artery occlusion is a distinctive pattern of ST-segment elevation in noncontiguous leads.[3] High lateral STEMI can present as ST-segment elevation involving lead I and aVL. Subtle ST elevation in V5, V6, and reciprocal changes in lead III and aVF may be present. A high LMI is mostly referred to as South African flag sign.^[4] Isolated LMI, similar to other acute MI, is caused by acute atherosclerotic plaque rupture with subsequent thrombus formation in left circumflex or one of its branches. More commonly, LAD is affected in the developing anteroLMI. In a case of recent drug-eluting stent, medication noncompliance can cause stent restenosis resulting in acute MI. On rare occasions, patients can present with MI with nonobstructive

coronary arteries (MINOCA). However, depending on a metaanalysis done in 2015, the prevalence of MINOCA is estimated approximately 6%.^[5] Some of the causes of MINOCA include coronary artery spasm, spontaneous coronary artery dissection, coronary artery embolism, autoimmune conditions such as Takayasu arteritis, and myocardial bridging.^[4] LMI manifested as STEMI should be managed promptly. Early reperfusion has demonstrated good benefits with improved the clinical outcomes.^[6] Percutaneous intervention (PCI) has shown the superior results if compared to fibrinolytic agents.^[7] ACC/ AHA guidelines for STEMI management motivate early PCI with preferable door to balloon time of fewer than 90 min at PCI capable facility and <120 min at non-PCI capable facility. ^[8] Antiplatelet therapy with aspirin and either P2Y12 inhibitor or glycoprotein IIb/IIIa inhibitor is advised in the peri-PCI time. Several studies have shown higher mortality benefits with both beta-antagonists and high-potency statins.^[9]

CASE REPORT

A 60-year-old married, officer, heavy smoker, Egyptian male patient presented with acute severe chest pain.

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Profuse sweating was the only associated symptom. The patient is a smoker for about 50 cigarettes per day for nearly 23 years. He denied the history of cardiovascular diseases, the same attack, drugs, or any other special habits. Chest pain was anginal, compressible, intolerable, and progressive. On general physical examination; generally, the patient was an anxious, severe sweaty, and had cold extremities, with an irregular heart rate of 120 bpm, blood pressure of 160/110 mmHg, respiratory rate of 18 bpm, the temperature of 36.9°C, and pulse oximeter of O₂ saturation of 97%. No more relevant clinical data were noted during the clinical examination. He was admitted to the intensive care unit as anginal chest pain and hypertension. Urgent ECG was done that showing acute high lateral (I, aVL, and V2) STEMI and reciprocal ST-depression changes in leads (III, aVF, and aVR) and AF [Figure 1a and b]. A high lateral STEMI was configured as Flag South Africa sign [Figure 2].

Hypertension and high lateral STEMI were the most probable diagnosis. A sublingual 5-dinitrate tablet was

given (5 mg). O_2 inhalation was given (100%, by nasal cannula, 5 L/min). Pethidine HCL 100 mg was given on intermittent IV doses. Intravenous nitroglycerin infusion (7.5 µg/min with intermittent titration) was given. Serial ECG tracings were taken. Blood pressure became normalized (130/70 mmHg). Aspirin four chewable, oral tablet (75 mg), clopidogrel four oral tablet (75 mg), and streptokinase IVI (1.5 million units over 60 min) were added after blood pressure control. Clinical improvement and gradual electrocardiographic ST-segment (whether elevations or reciprocal ST-depressions) resolution [Figure 3a-c] had happened. The chest pain starts to resolute within 15 min after ending streptokinase infusion. The measured random blood sugar was 147 mg/dl. The troponin test was positive (233 ng/L). Later echocardiography was mild high lateral hypokinesia with ejection fraction of 61%. No more workup was done. The patient was continued: Captopril tablet (25 mg twice daily), aspirin tablet (75 mg, once daily), clopidogrel tablet (75 mg, once daily), nitroglycerin retard capsule (2.5 mg twice daily), and atorvastatin (40 mg once daily) until discharged on the



Figure 1: Serial electrocardiogram tracings; (a) initial tracing showing high lateral (I, aVL, and V2) ST-segment elevation myocardial infarction (green arrows) and reciprocal ST-depression changes in leads (III, aVF, and aVR) (purple arrows), multiple premature ventricular complexes in V1-3 (blue arrows) and AF. (b) Tracing within 40 min of the intensive care unit admission showing high lateral (I, aVL, and V2) ST-segment elevation myocardial infarction (green arrows) and reciprocal ST-depression changes in leads (III, aVF, and aVR) (purple arrows) and AF.



Figure 2: Flag South Africa configuration for high lateral ST-segment elevation myocardial infarction



Figure 3: Serial electrocardiogram tracings; (a-c) tracings showing gradual electrocardiographic ST-segment (whether elevations or reciprocal ST-depressions) resolution

5th day. Planning for catheter revascularization was a future option.

DISCUSSION

- Overview: A 60-year-old heavy smoker male patient presented with intolerable chest pain with high lateral STEMI and severe hypertension
- I cannot compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison
- Study question here; how did very small MI inducing an intolerable chest pain?
- The primary objective for my case study was the presence of high lateral STEMI and hypertension
- The secondary objective for my case study was the initial treatment of hypertension to easy giving both fibrinolytic and thrombolytics.

Limitations of the study

There are no known limitations in the study.

Recommendations

- It is recommended to widening the research in clearing the neuronal interpret acute severe chest pain despite very small MI
- Furthermore, it is recommended to extend the researching in the impact of fibrinolytics and thrombolytics in reliving the chest pain.

CONCLUSION

• The author thinks that the size of MI may not correlate to the severity of chest pain

• A high lateral STEMI is considered the smallest one but accompanied by the highest chest pain.

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