Inferior ST-Elevation Myocardial Infarction Secondary to Aortic Mass: A Rare Case Report

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Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Consent for publication

Written consent form for publication of his details was obtained from the patient.

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The authors declare that they have no competing interests.

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Data curation

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Funding acquisition

Investigation

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Formal analysis

Validation

Visualization

Key clinical message

Inferior STEMI from an aortic mass demands multidisciplinary care for accurate diagnosis and optimal outcomes. Timely recognition and surgical intervention are crucial for favourable results.

Abstract

Inferior ST-elevation myocardial infarction (STEMI) secondary to an aortic mass presents a rare and complex clinical scenario requiring heightened diagnostic vigilance. We report a case of a 22-year-old female presenting with recurrent chest pain, ultimately diagnosed with inferior STEMI secondary to an aortic mass compressing the coronary arteries. Advanced imaging modalities and a multidisciplinary approach were crucial for accurate diagnosis and optimal management. Surgical intervention to address the aortic mass, coupled with myocardial reperfusion strategies, led to favourable outcomes. This case underscores the importance of recognizing the intricate interplay between cardiac and vascular pathologies to guide tailored therapeutic interventions and optimize patient care.

Keywords

Emergency medicine, cardiology, cardiothoracic surgery, Radiology & Imaging

Introduction

The convergence of cardiovascular pathology poses unique challenges in clinical scenarios. The emergence of Inferior ST-elevation myocardial infarction (STEMI) secondary to an aortic mass represents a compelling intersection of cardiac and vascular abnormalities (1,2). While Inferior STEMIs typically implicate the coronary artery system, the involvement of an aortic mass introduces a distinctive dimension, necessitating a comprehensive understanding of the intricate relationship between aortic pathologies and myocardial ischemia (2). This intricate interplay underscores the importance of a multidisciplinary approach, blending the expertise of cardiologists, cardiac surgeons, and vascular specialists. This introduction sets the stage for exploring the nuanced intricacies of how an aortic mass can contribute to myocardial infarction, shaping discussions around diagnosis, treatment modalities, and the broader implications for patient care (3,4). As medical understanding advances, delving into specific instances like Inferior STEMI associated with aortic masses becomes pivotal, offering valuable insights into refining clinical strategies and enhancing outcomes for individuals facing this intricate amalgamation of cardiac and vascular disorders (5).

Case presentation

A 22-year-old newly married female presented to our emergency department with recurrent intermittent attacks of retrosternal stabbing chest pain for a one-month duration. The pain typically lasted for a few minutes, radiated to the back and both shoulders, and was associated with nausea. She reported an increased awareness of heartbeats with physical activity and relief upon sitting down or lying flat.

Despite three previous visits in the last 10 days, where she received IV fluids, paracetamol, and omeprazole, along with reassurance that the pain was musculoskeletal, it persisted ranging from mild to severe, prompting her return.

Her past medical history is unremarkable, and she is a non-smoker and non-drinker. There is a family history of maternal hypertension and paternal type two diabetes, with no similar conditions or history of sudden cardiac death.

On examination, she appeared conscious, oriented, mildly dyspnoeic but not tachypnic, and anxious due to severe pain. Physical findings included normal chest auscultation, an ejection systolic murmur loudest at the left sternal border, and no abdominal abnormalities. Vital signs were as follows: Spo2 = 96% on room air, PR = 88 beats/min, BP = 120/70 mmHg, Temp = 37.9°C, RR = 22 cycles/min.

Deferential diagnosis, Investigations and management

An emergency ECG was conducted, revealing inferior ST-segment elevation myocardial infarction (STEMI) as shown in Figure 1, despite normal serum troponin I levels.

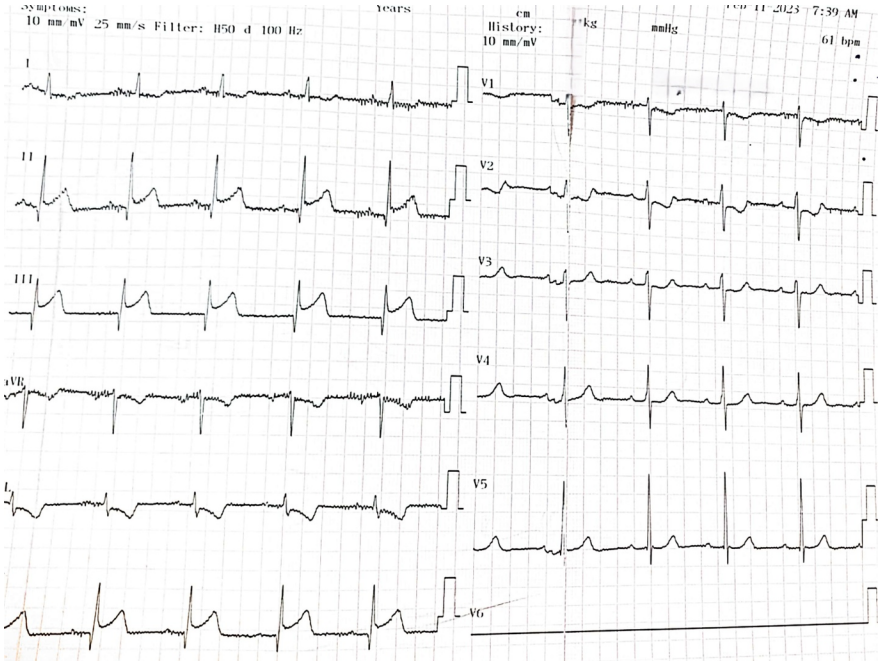


Figure 1. Emergency ECG showing ST segments elevation at leads I, II and AvF.

The catheter laboratory was notified, and the patient was promptly referred to a tertiary cardiac hospital by ambulance. Upon arrival, a follow-up ECG after 30 minutes depicted normal sinus rhythm without ischemic changes (Figure 2), coinciding with the patient being pain-free and clinically stable.

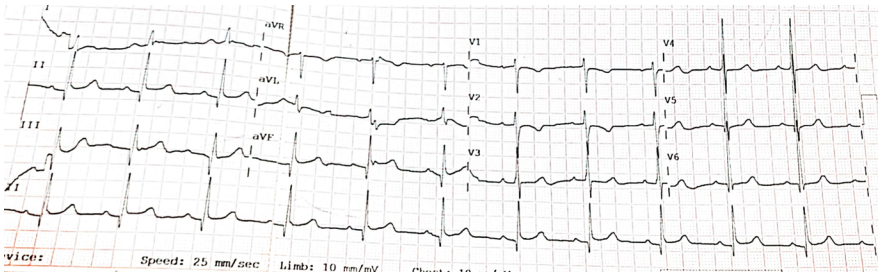
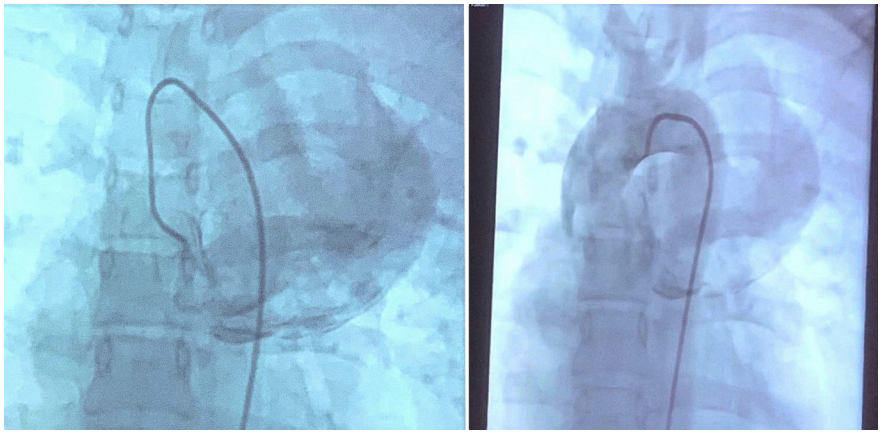


Figure 2. ECG 30 min after presentation at the tertiary cardiac hospital revealing normal ST segment and sinus rhythm.

A decision was made to proceed with invasive coronary intervention due to the nature of the pain and ECG changes. The result revealed normal blood flow in the coronary arteries. However, a sac was observed behind the ascending aorta, aortic arch, and descending aorta, compressing the coronary arteries, mainly the right coronary artery (RCA), resulting in inferior STEMI (Figure 3)



Fi gure 3: Aortic injection using a Judkins left (JL) catheter shows a sac behind the ascending aorta, aortic arch, and descending aorta.

The patient was transferred to the medical ward for further evaluation and follow-up. A chest X-ray was conducted, revealing a large mediastinal mass (Figure 4). Her complete blood count and renal function test were normal.

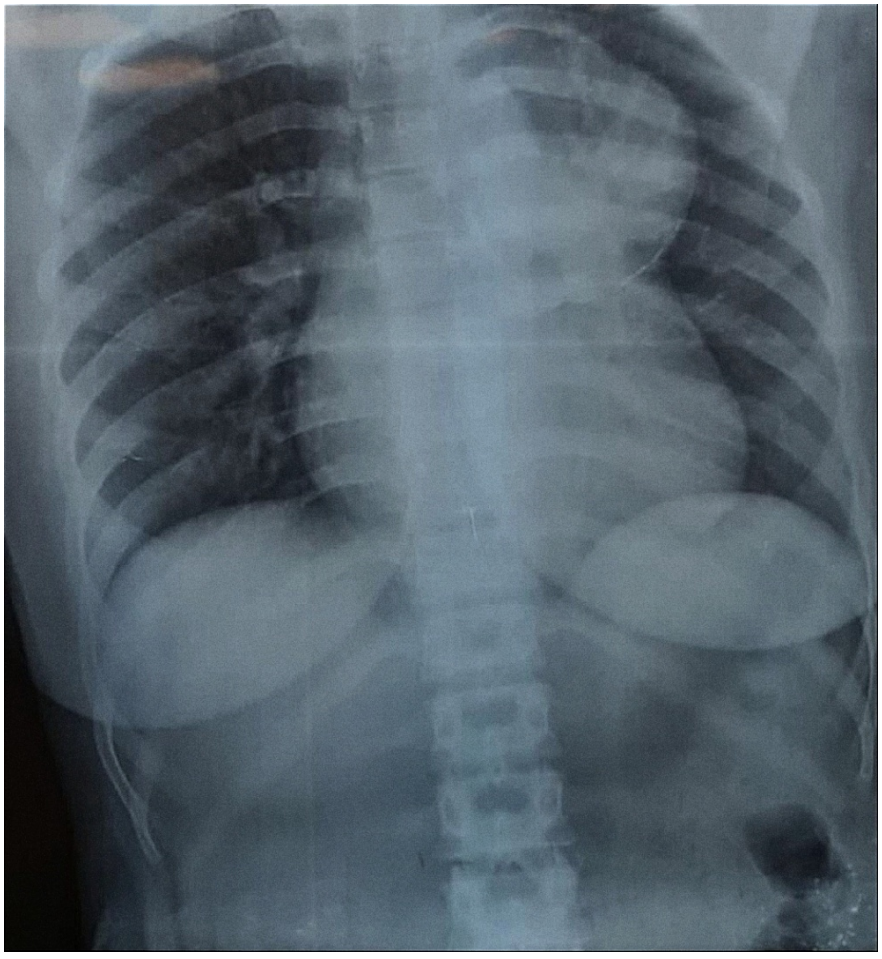


Figure 4.chest X ray PA view showing large mediastinal mass.

Results and follow up

We proceeded further with a chest CT angiogram, which revealed a well-defined anterior mediastinal soft tissue density mass lesion measuring about 84.9x67.2mm, seen at the aorto-pulmonary window. The mass exhibited a mass effect, compressing the main pulmonary artery and proximal ascending aorta. The mass showed a calcified wall with enhancement after IV contrast, with two different densities within. The mass showed close contact, almost inseparable from the ascending aorta. A definite relation with the aorta could not be established, whether it originated from or was invading it (Figure 5).

Figure 5. Chest CT angiogram revealing well-defined anterior mediastinal soft tissue density mass lesion of about 84.9x67.2mm seen at aorto-pulmonary window with mass effect compression of the main pulmonary artery and Proximal ascending aorta(red arrows and cross).

After a multidisciplinary team meeting involving a cardiothoracic surgeon, anaesthesiologist, cardiologist, and the patient with her family, it was decided to proceed with open heart surgery.

During the surgery, an aneurysm in the aortic wall near the sinus of Valsalva was discovered (see figures 6 and 7).

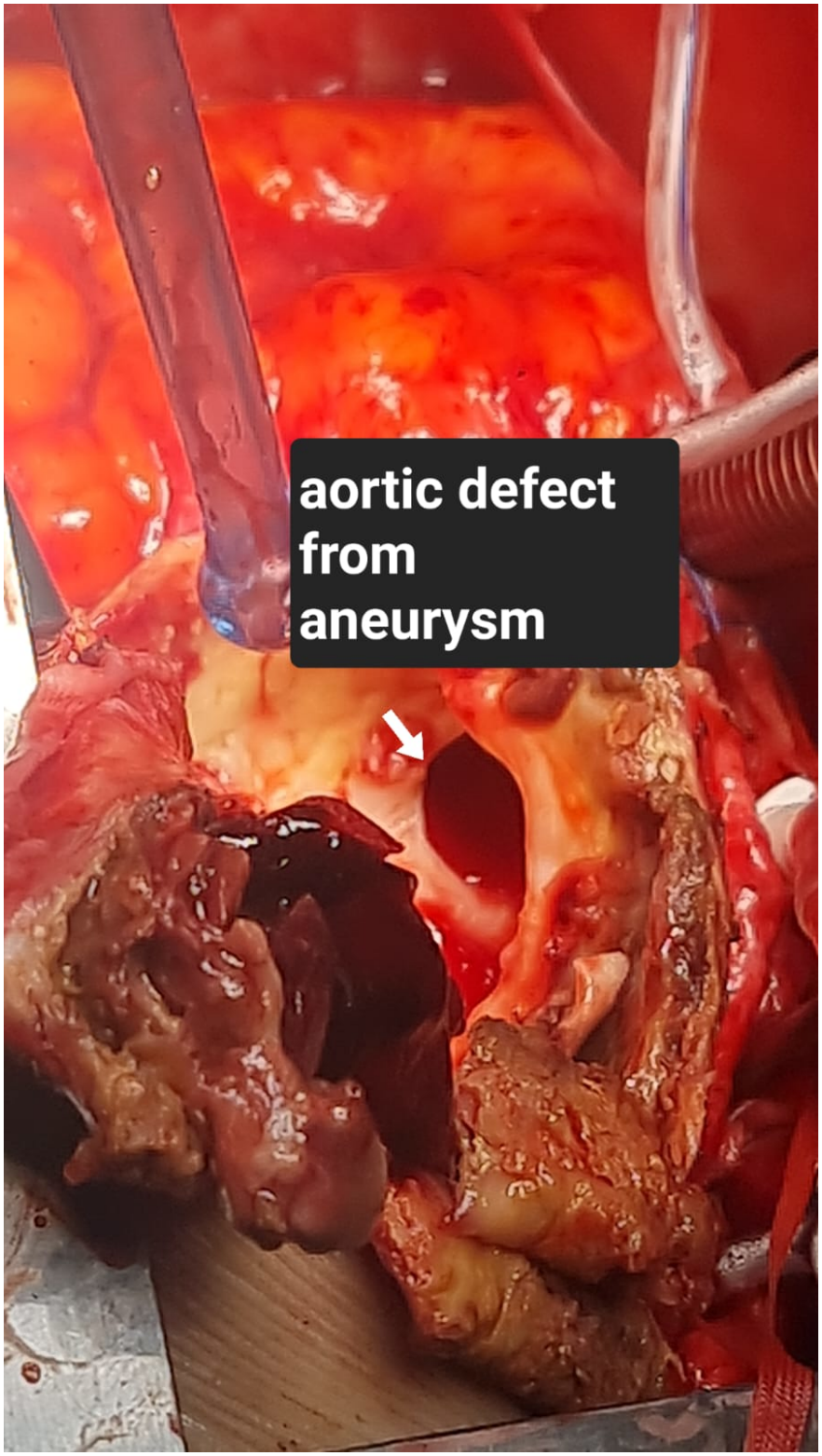


Figure 6. Aortic defect near sinus of Valsalva after aneurysm resection

Figure 7. Aneurysm tissue sample after resection

The surgical procedure involved resection of the aneurysm and interposition graft placement (figure 7)

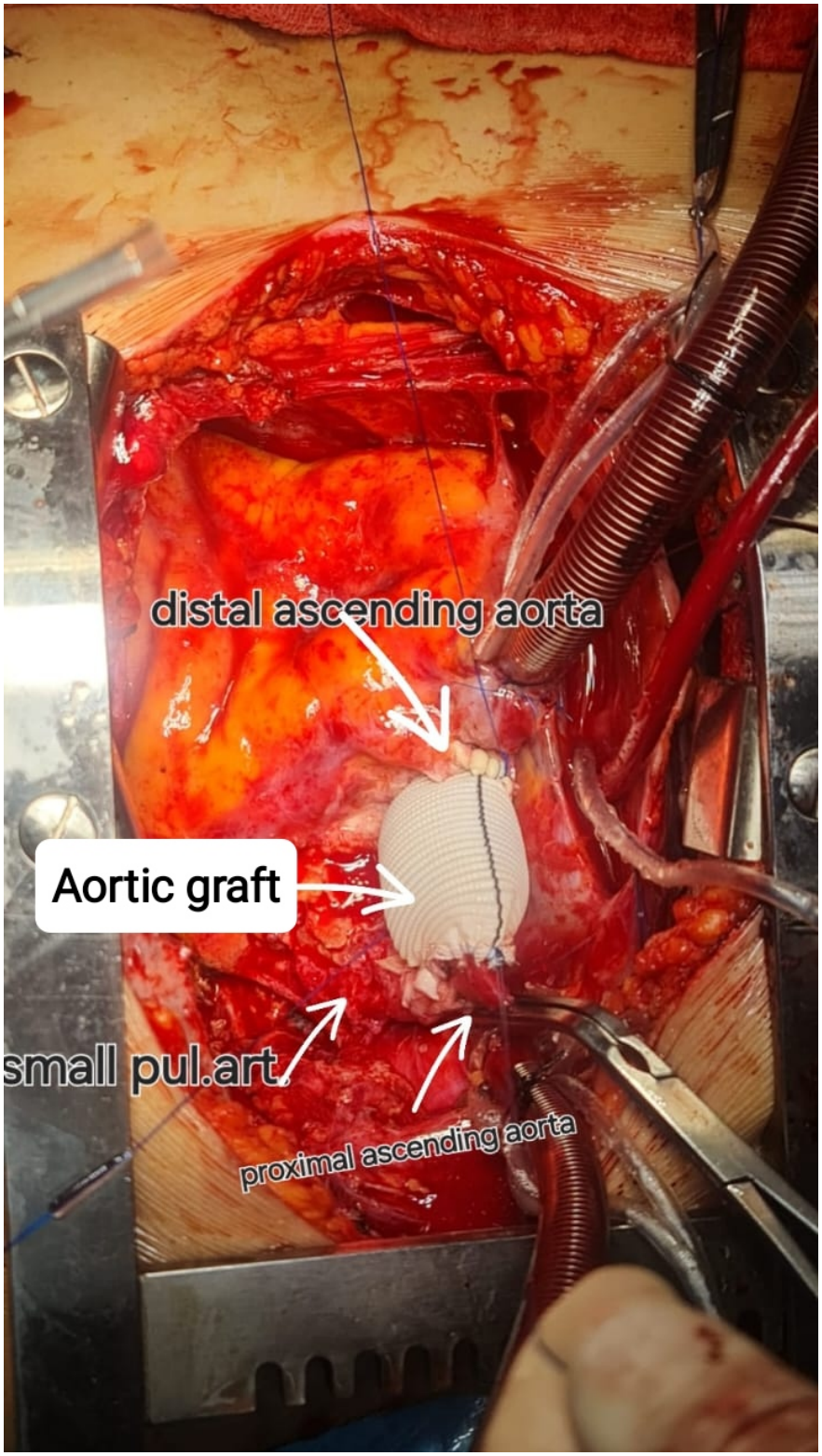


figure 8. Aortic Interposition graft placement

A sample of the aneurysm tissue was sent for histopathological analysis, revealing fibrosis, hyalinization, necrosis, and calcification. The patient's condition improved post-surgery, and she was discharged home with monthly follow-up appointments. Subsequent echocardiography showed normal results, and she later conceived and delivered a single female fetus via normal vaginal delivery.

Discussion

Inferior ST-elevation myocardial infarction (STEMI) secondary to an aortic mass is a rare and intricate clinical scenario that demands heightened diagnostic vigilance (6). The aorta, a vital conduit for systemic circulation, can harbor various pathologies, including tumors or thrombi, compromising coronary blood flow (7).

The challenges arise from the potential for embolic events originating from the aortic mass, leading to downstream coronary artery occlusion and subsequent myocardial infarction. The inferior distribution is a common manifestation due to the proximity of the coronary ostia, complicating the electrocardiographic interpretation (8).

Accurate diagnosis relies on a comprehensive approach. Advanced imaging modalities, particularly transesophageal echocardiography (TEE) and computed tomography angiography (CTA), offer insights into the nature and extent of the aortic mass (9). Timely recognition of this unique etiology is crucial to guide appropriate management.

Therapeutic strategies include addressing both the aortic mass and myocardial infarction. Surgical intervention may be warranted for the aortic mass, aiming to prevent embolic events and restore normal coronary perfusion. Simultaneously, myocardial reperfusion strategies, such as percutaneous coronary intervention (PCI) or thrombolytic therapy, are deployed to salvage ischemic myocardium (11).

Post-intervention, close monitoring for potential embolic complications, arrhythmias, or recurrent infarctions is essential. Anticoagulation may be indicated to mitigate the risk of further embolism from the aortic mass (12).

Conclusion

Inferior STEMI secondary to an aortic mass underscores the intricate interplay between cardiac and vascular pathologies. A multidisciplinary approach, integrating advanced imaging and targeted interventions, is imperative for accurate diagnosis and optimal patient outcomes in this rare and complex clinical scenario.

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