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From MI to Aortic crisis; A Diagnostic Challenge: Case report

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Abstract

Aortic dissection (AD) is a life-threatening condition that can mimic acute myocardial infarction (MI), highlighting the importance of maintaining a broad differential diagnosis in acute chest pain**.** We present a case of a 43-year-old male with acute chest pain and electrocardiographic findings consistent with ST-segment elevation myocardial infarction (STEMI), including ST elevations in leads V1-V4. Despite elevated troponin levels, classical signs of AD—such as mediastinal widening on imaging and discrepancies in pulse or blood pressure—were absent. Coronary angiography revealed no significant coronary obstruction, but an immediate aortogram identified a dissecting flap at the root of the ascending aorta, just distal to the right coronary artery (RCA) ostium. The patient underwent emergent surgical repair with an uncomplicated postoperative course. This case highlights the critical need for vigilance in atypical presentations of MI and reinforces the importance of incorporating advanced imaging early in the diagnostic process. Prompt recognition of AD in such scenarios is vital, as delays can result in catastrophic outcomes. This report underscores the educational value of recognizing nuanced presentations and integrating multidisciplinary approaches to optimize patient care.

Keywords: Aortic; Dissection; STEMI; Chest pain; Case report

Abbreviations: AD: Aortic dissection; MI: Myocardial infarction; STEMI: ST-elevation myocardial infarction; EKG: Electrocardiogram; RCA: Right coronary artery; LAD: Left anterior descending; EF: Ejection fraction; TTE: Transthoracic echocardiography; CTA: Computed Tomography angiography

Introduction

Aortic dissection, a life-threatening condition, occurs when elevated blood pressure leads to the separation of layers within the aortic media. This division creates a false lumen within the aortic wall [1,2]. Distinguishing between Stanford type A aortic dissection and myocardial infarction (MI) with ST segment elevation can be challenging. However, it is crucial for physicians to do so, as anticoagulation therapy, a key component of MI management, is contraindicated in patients with aortic dissection [3]. Approximately 0.1% of patients experiencing acute myocardial infarction (MI) and 7.5% of those with aortic dissection are diagnosed with both conditions simultaneously [4]. Additionally, around 2% of aortic dissection cases present with occlusive myocardial infarction (OMI), with the right coronary artery being more frequently involved compared to the left main coronary artery [5]. Recent studies underscore the significance of biomarkers and advanced imaging techniques in differentiating between these conditions [4]. Timely recognition of both typical and atypical presentations is essential, given that approximately

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30% of patients with aortic dissection succumb to the condition in the emergency department [6]. In this case report, we present an adult male who initially presented with acute chest pain and classic MI picture, including ST changes on ECG and elevated troponin levels. He was sent for catheterization, but patent coronary arteries were observed. However, further investigation revealed an unexpected diagnosis of aortic dissection, emergency surgical repair was performed, and the patient was successfully treated.

Case Presentation

43 years old male with past medical history of hepatitis B carrier: HBV DNA viral load by Quantitative PCR on 11/Jun/2014: NOT DETECTED. HBsAg was positive in Nov 2010. HBsAg and HBeAbs were positive in March 2008, hypertension (few months prior to presentation was diagnosed) and a strong family history of cardiac disease (Brother has CAD who is 48-year-old, and his cousin died at 42 years of age from an ACS event. Two uncles had CAD at an early age) presented to the emergency department complaining of chest pain. The pain was described as oppressive that started during the night radiating to the neck, jaw and left upper extremity associated with shortness of breath.

Initial vital signs showed blood pressure of 90/54 mmHg in right arm and 89/54 mmHg in left arm, Heart rate of 88 beat per minute, respiratory rate was 18 breath per minute, temperature of 37.6 degree C and oxygen saturation was 100% breathing ambient air.

Physical examination was significant for good peripheral pulses in all extremities and cardiac exam showed Regular S1S2 with no murmurs. S3 and S4 were not present.

Initial lab results showed a white blood cell count of 13,500 /cu.mm, hemoglobin of 16.1 g/dL, platelets of 231,000 /cu.mm, creatinine of 1.67 mg/dL, initial set of troponin T was 0.03 ng/mL. Twelve lead electrocardiogram (EKG) showed ST changes in the anterior leads. (Figure 1).

The patient received a loading dose of Ticagrelor 180 mg and Aspirin 300 mg and Heparin 4000 IU IV once (Ticagrelor provides faster, more potent, and consistent platelet inhibition, reduces cardiovascular mortality, and is not affected by genetic variability in metabolism according to latest data). Chest X-ray showed cardiac silhouette is normal in size and no widened mediastinum (Figure 2).

Emergent coronary angiography was done and revealed: Near normal coronary angiogram. Mid LAD focal tubular stenosis seen only in 2 cine loops, likely spasm (Figure 3).

Since the patient had a previous TTE that showed mildly aortic root dilatation and the coronary angiography was nonrevealing, we opted to do an aortic root angiogram which showed: Dissecting flap identified at the root of the ascending aorta, just distal to the RCA ostium (Figure 5).

A bedside transthoracic echocardiogram was also performed after cardiac catheterization and showed an EF of 60-65% and mild aortic insufficiency (Figure 4).

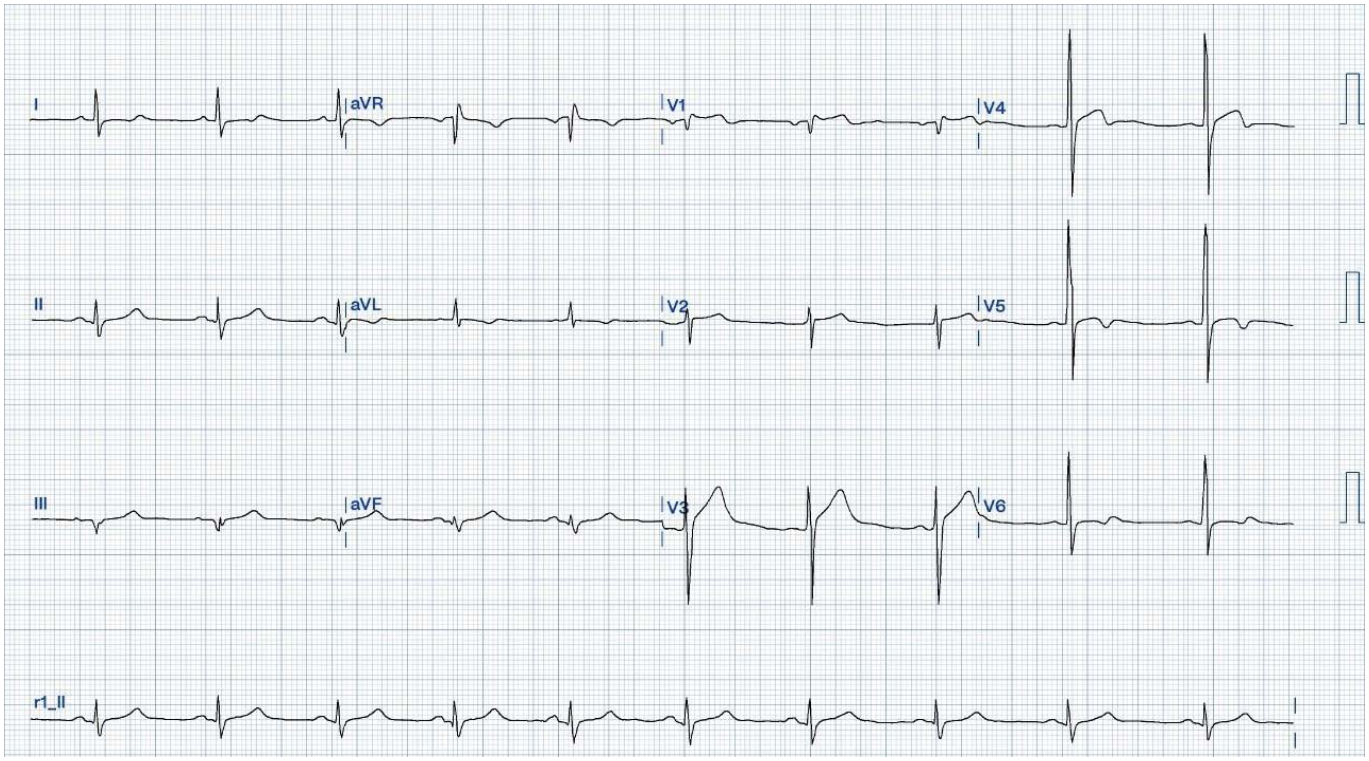

Figure 1: ECG on admission showing ST-changes in the anterior leads suggestive of acute ischemia.

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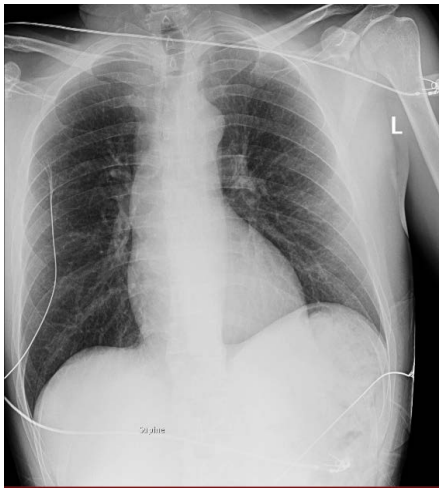

Figure 2: No focal consolidation. No pleural effusion. No pneumothorax. The cardiac silhouette is normal in size.

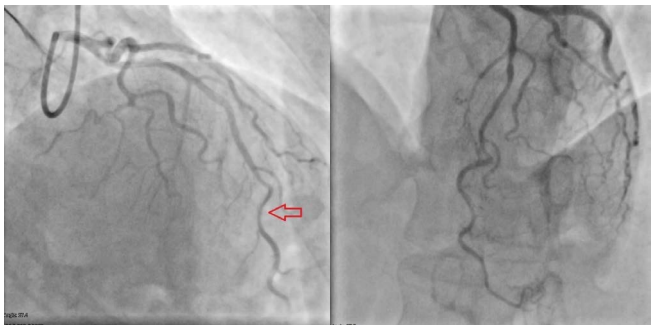

Figure 3: The left anterior descending coronary artery is free of stenosis in its proximal and distal segments. It has a small narrowing in its mid-segment that may represent spasm.

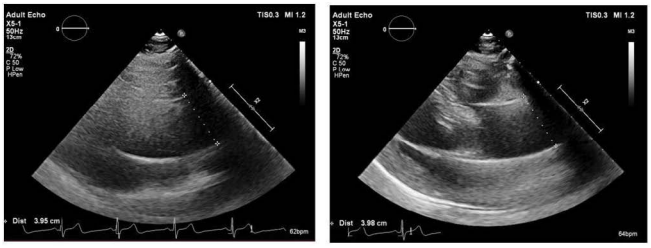

Figure 4: Mildly dilated ascending aorta measures 4 cm, sinus of Valsalva 3.7 cm with the absence of clear evidence of an intimal flap.

Vascular surgery team was immediately consulted, and a computed tomography with angiography (CT) scan was done and revealed an aortic dissection (Figure 6).

After the diagnosis of ascending aortic dissection Type A, the decision for immediate lifesaving surgical repair was made.

Intraoperative findings showed tear beyond the left subclavian

The patient underwent Resuspension of the aortic valve Ascending replacement with a 28 mm strait Dacron conduit graft (post Ascending Aorta graft) and the procedure was done successfully without complications.

The patient had an uncomplicated postoperative course and was discharged home.

Followed up in clinic 10 days with the cardiothoracic team for possible acute complications with a TTE showing normal ascending aortic graft with mild aortic regurgitation. One month later the patient had follow-up with the cardiology team, the patient is doing well with no long term sequalae.

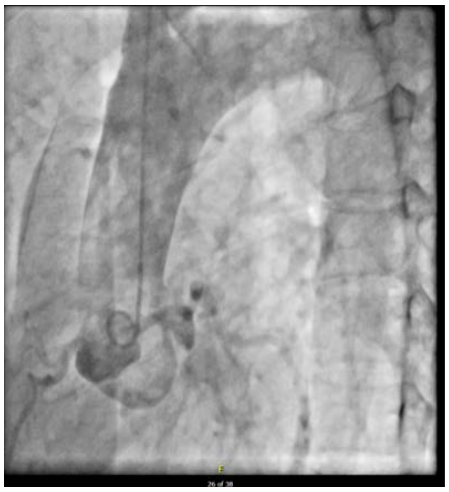

Figure 5: Aortogram performed in the RAO position showed dissecting flap identified at the root of the ascending aorta, just distal to the RCA ostium.

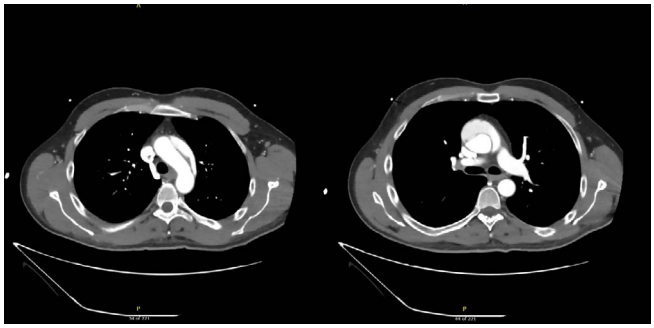

Figure 6: Intimal flap seen arising just distal to the origin of the right coronary artery, extending to the ascending thoracic aorta, aortic arch and terminating at 1.5 cm beyond the left subclavian artery.

Discussion

Aortic dissection (AD) is characterized by a separation of the aortic wall layers and subsequent creation of a pseudo-lumen that may compress the true aortic lumen. Predisposing factors mediate their risk by either increasing tension on the wall (e.g. hypertension, atherosclerosis, etc.) or by causing structural degeneration (e.g. connective tissue diseases). AD can be categorized based on anatomical location using either

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the Stanford or DeBakey classification [7]. Our reported patient had acute AD of Stanford type A, and DeBakey type I. AD has an estimated incidence rate of 5-30:1,000,000 cases annually. Type A dissection has a high mortality rate of 58%, especially without surgical intervention, while those managed surgically was 26%. Surgical management is preferred in type A and is a surgical emergency [8]. The symptoms of AD may be variable and can mimic other more common conditions such as myocardial ischemia (MI) and is hence sometimes referred to as the "great masquerader". Patients typically present with chest or back pain, of sudden onset and usually described as sharp or tearing pain. Almost 25% present without chest pain [9]. An abnormal aortic contour or widening of the aortic silhouette (seen in 86.5%), EKG changes (seen in 68.7%), aortic regurgitation (seen only in 31.6%), and a discrepancy in the pulse or blood pressure in the upper extremities (seen only in 15.1%) are all characteristic features of AD [8]. Additionally, Patients may present with complications from AD progression or disrupted blood flow, including heart failure, neurological deficits, syncope, myocardial ischemia, visceral ischemia, or limb ischemia. [7]. More recent studies show that low D-dimer levels are being increasingly used for the early exclusion of AD in patients with low pre-test probability. This is especially effective when combined with clinical scoring tools like the Aortic Dissection Detection Risk Score (ADD-RS), with sensitivity reaching 99% [10]. Point-of-care ultrasound (POCUS) is increasingly valuable in the rapid evaluation of suspected AD, especially in emergency settings. POCUS allows for visualization of key findings, such as an intimal flap within the aorta, pericardial effusion indicating potential rupture, or aortic root dilation. Parasternal long axis and subcostal views are commonly used for this purpose. Its real-time imaging capability facilitates immediate clinical decisions, especially when advanced imaging is delayed. However, POCUS requires operator expertise and is generally less sensitive than CT angiography [11]. Contrast enhanced CT scanning, magnetic resonance and TEE are all superior and highly accurate techniques useful for the confirmation or exclusion of the diagnosis [3].

Given that the management of primary STEMI is potentially lethal if given to a patient with AD (possible rupture, expansion, or uncontrolled bleeding), it is essential to distinguish them early on. Despite that, unfortunately a handful of cases, such as this one, are diagnosed with AD only during cardiac catheterization. Coronary angiography was normal. This rendered other differential diagnosis of acute chest pain more likely in our patient, including AD. Given his history of mild aortic root dilation, an aortogram identified a dissecting flap at the root of the ascending aorta, just distal to the right coronary artery (RCA) ostium, prompting a vascular surgery consultation, CTA, and then a life-saving surgical repair. Here, the aortogram played an instrumental role and lead to prompt diagnosis and expedited surgical intervention, underscoring its importance as a complementary diagnostic tool. Administering antiplatelets such as aspirin and ticagrelor in suspected AD could be detrimental, as it may worsen bleeding in the event of aortic rupture. The decision to proceed with coronary catheterization is also risky, as it might delay the diagnosis of AD, possibly leading to unwanted complications. Ideally, aortic dissection should be ruled out before administering antiplatelet therapy or performing coronary angiography. However, given the higher prevalence of MI compared to AD, combined with elevated troponin, ST elevations on EKG, a strong family history, and a clinical presentation strongly suggesting MI, further delay to obtain imaging for a less likely diagnosis of AD poses significant risks. Although two other case reports with minimal AD signs & symptoms avoided the use of antiplatelets, their presentation was clearer, with TTE showing evidence of intimal tear, unlike ours [1,3]. In the case of acute MI mimicking aortic dissection, the pathophysiological mechanisms of coronary Mal perfusion can be attributed to two factors. First (static mechanism), the expanding hematoma narrows the affected vessel, compromising blood flow. Second (dynamic mechanism), the dissection flap may partially block the ostium of the coronary artery, potentially leading to coronary thrombosis and subsequent MI. Additionally, dissection can directly extend into the coronary arteries, with the RCA being more frequently affected than the left artery. This higher incidence of RCA involvement is attributed to dissections originating from the right anterior aspect of the ascending aorta, just above the right coronary sinus [3], which is almost the case in our patient. In a recent study highlighting the impact of AD misdiagnosis, it was found that the absence of severe intensity pain, less urgent triage category, and absence of POCUS ultrasound exam were independent factors associated with misdiagnosis of AD [5], which lead to administration of antithrombotic agents in 39% of cases, leading to more major bleeding and in hospital mortality [12]. However, in our case, POCUS (bedside TTE) was performed, the patient had severe intensity chest pain, and was triaged as urgent, and yet AD was misdiagnosed at first due to more atypical presentation and a higher likelihood of MI. A post-catheterization (and prior to aortic surgical repair) bedside TTE was repeated and showed a new onset of mild aortic regurgitation as compared to none upon presentation. This was likely secondary to active propagation of the dissection. Interestingly, a follow up TTE 10 days post-operation (latest TTE available as of the writing) showed stable mild aortic regurgitation, further supporting the notion that if this patient's aortic dissection was caught at its very early stages. It is important to educate patients with aortic root dilation about the symptoms of AD, which would help catch them early on and markedly reduce

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mortality, as was the case with our patient. Paradoxically, the EKG findings (leads V1-V4 ST segment elevations) are not entirely explained by the location of the AD (just distal to the RCA ostium), which in theory should correspond to leads II, III, and AVF. Further studies pertaining to this finding should be implemented. Clinicians should maintain a high index of suspicion and always include AD in the differential diagnosis of patients presenting with acute chest pain, particularly for those with prior history of aortic root dilation. Notably, low ADD-RS values combined with low D-dimer levels can provide clinicians with greater confidence in administering antiplatelet agents in patients with a strong clinical suspicion of acute coronary syndrome. Machine learning could enable real-time analysis of Electronic Health Records (EHR) data to flag patients at higher risk for AD during triage, even before definitive imaging is performed [13]. For example, systems could analyze risk factors such as a history of aortic root dilation, uncontrolled hypertension, ECG changes, and subtle imaging findings (e.g. mediastinal widening) [11]. Triage categorization helps reduce misdiagnosis of AD [12].

Conclusion

This is a case of an atypical presentation of AD masquerading as a typical STEMI. High index of suspicion for AD should be kept in every patient with history of dilated ascending aorta. Until ruled out, it is best not to load with DAPT as it might effect the management and cause complications in AD. ADD-RS value combined with low D-dimer level can be used to rule out AD before giving DAPT. Future perspectives would include enhanced machine learning to enable real-time analysis of electronic health records (EHR) data to flag patients at higher risk of AD during triage, even before definitive imaging is performed [13]. More awareness should be directed toward clinicians dealing with these patients and to educate patients about AD warning signs upon the diagnosis of dilated aorta.

Reporting statement: This Case report has been reported in line with the CARE 2013 criteria [14].

Availability of data and materials

Not applicable.

Ethics declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Competing interest

The authors declare no conflict of interest in the research, authorship and publication of this article.

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