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| RESEARCH ARTICLE

A Deadly Mimic: Type A Aortic Dissection Presenting as Inferior STEMI

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| ABSTRACT

Acute Type A aortic dissection is a life threatening cardiovascular emergency that may present with clinical and electrocardiographic findings resembling acute myocardial infarction, creating a significant diagnostic challenge. We report the case of a 58 year old male with a history of hypertension who presented to the emergency department with sudden onset severe central chest pain radiating to the back, associated with diaphoresis, nausea, and vomiting. On arrival, he was tachycardic and relatively hypotensive but remained alert and neurologically intact. Initial electrocardiography demonstrated hyperacute T waves in the inferior leads with reciprocal ST segment depression in the anterior leads, representing an inferior STEMI equivalent pattern. High sensitivity troponin was elevated, further supporting an initial diagnosis of acute coronary syndrome. Despite treatment and preparation for urgent coronary angiography, persistent chest pain radiating to the interscapular region, mild mediastinal widening on chest radiography, and relative hypotension prompted further evaluation. Bedside transthoracic echocardiography demonstrated a mildly dilated ascending aorta, a small pericardial effusion, and findings suspicious for an intimal flap. Subsequent computed tomography angiography confirmed an extensive Stanford Type A aortic dissection involving the ascending aorta and aortic root with extension to the origin of the right coronary artery, resulting in coronary malperfusion. The patient underwent prompt medical stabilization with intravenous beta blockade and analgesia before transfer to a tertiary cardiothoracic center, where successful emergency surgical repair was performed. His postoperative recovery was uncomplicated, and he was discharged home in stable condition. This case highlights the importance of maintaining a broad differential diagnosis in patients presenting with apparent STEMI, particularly when atypical features such as abrupt pain onset, radiation to the back, hypotension, or mediastinal widening are present. Early recognition of aortic dissection and timely use of bedside imaging can prevent inappropriate antithrombotic therapy and facilitate life saving surgical intervention.

| KEYWORDS

Myocardial infarction, ST elevation, Aortic dissection, Chest Pain

| ARTICLE INFORMATION

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Introduction

Acute chest pain is one of the most common reasons for presentation to the emergency department and represents a significant diagnostic challenge for clinicians. Among the many potential causes, acute myocardial infarction remains a major concern because early recognition and prompt reperfusion therapy are strongly associated with improved outcomes. The widespread implementation of rapid electrocardiographic assessment and streamlined pathways for ST elevation myocardial infarction (STEMI) has significantly reduced treatment delays and improved survival. However, several life threatening conditions may present with clinical and electrocardiographic findings that closely resemble STEMI. Failure to recognize these alternative diagnoses can lead to inappropriate treatment and potentially catastrophic consequences. One of the most important of these conditions is acute aortic dissection. [10,11]

Acute aortic dissection is a medical and surgical emergency characterized by disruption of the aortic intima, allowing blood to enter the media and create a false lumen within the aortic wall. The condition belongs to the spectrum of acute aortic syndromes, which also includes intramural hematoma and penetrating aortic ulcer. Although less common than acute coronary syndrome, aortic dissection carries a substantially higher mortality when diagnosis or treatment is delayed. Historical data from the International Registry of Acute Aortic Dissection (IRAD) demonstrated that mortality increases rapidly during the first hours after symptom onset, emphasizing the importance of early recognition and urgent management. [13]

The Stanford classification remains the most widely used system for categorizing aortic dissections. Type A dissection involves the ascending aorta regardless of the site of the intimal tear, whereas Type B dissection is confined to the descending thoracic aorta distal to the origin of the left subclavian artery. This distinction is clinically important because Type A dissection usually requires urgent surgical intervention, while Type B dissection may often be managed medically or through endovascular approaches depending on the clinical situation. Among the two categories, Type A dissection is associated with a greater risk of complications, including cardiac tamponade, acute aortic regurgitation, myocardial ischemia, stroke, and sudden death. [10,12]

The incidence of acute aortic dissection is estimated to be relatively low compared with other cardiovascular emergencies. Nevertheless, because of its high mortality and varied presentation, it remains one of the most feared diagnoses in emergency and cardiovascular medicine. The condition most commonly affects older adults and is frequently associated with hypertension, which is considered the strongest modifiable risk factor. Other predisposing conditions include connective tissue disorders such as Marfan syndrome, bicuspid aortic valve, aortic aneurysm, prior cardiac surgery, inflammatory aortic disease, and a family history of aortic pathology. Despite these recognized risk factors, aortic dissection can occasionally occur in individuals without an obvious predisposing condition, making diagnosis even more challenging. [10,11,14]

Classically, patients with acute aortic dissection present with the sudden onset of severe chest or back pain that is often described as tearing, ripping, or sharp in nature. The pain may migrate as the dissection progresses along the aorta. Additional findings can include pulse deficits, blood pressure differences between extremities, neurological symptoms, syncope, and signs of end organ ischemia. However, clinical presentation is highly variable. Many patients do not exhibit the classic features described in textbooks, and some present with symptoms that strongly suggest other conditions. This variability contributes significantly to delayed or missed diagnosis. [11,13]

Among the most challenging presentations is acute Type A aortic dissection masquerading as STEMI. In these cases, patients may present with chest pain accompanied by electrocardiographic ST segment elevation and elevated cardiac biomarkers, creating a clinical picture that is virtually indistinguishable from acute coronary occlusion. Because current systems of care prioritize rapid reperfusion for STEMI, clinicians are often under considerable pressure to activate the catheterization laboratory and initiate antithrombotic therapy without delay. While this approach is appropriate for true myocardial infarction, it may be devastating in patients with an underlying aortic dissection. [1,2,6]

Coronary malperfusion is a recognized complication of Type A aortic dissection and represents the principal mechanism by which myocardial infarction occurs in these patients. Extension of the dissection flap into the coronary ostia may cause dynamic or fixed obstruction of coronary blood flow. Less commonly, compression of the true lumen or complete occlusion of a coronary artery can occur. The right coronary artery is most frequently affected because of its anatomical relationship to the ascending aorta. As a result, inferior wall myocardial infarction is the most common pattern observed when Type A dissection presents as STEMI. [9,10]

Inferior STEMI is typically characterized by ST segment elevation in leads II, III, and aVF, often accompanied by reciprocal changes in the lateral leads. In routine clinical practice, this pattern strongly suggests acute occlusion of the right coronary artery. Consequently, the diagnosis of acute coronary syndrome is usually made rapidly, particularly when accompanied by compatible symptoms and elevated troponin levels. However, in rare cases, the same electrocardiographic pattern may result from coronary involvement by an ascending aortic dissection rather than primary atherosclerotic plaque rupture. Distinguishing between these two entities at initial presentation can be extremely difficult. [1,6,9]

The overlap in presentation between acute coronary syndrome and aortic dissection has important therapeutic implications. Standard STEMI treatment often includes antiplatelet agents, anticoagulation, and urgent coronary intervention. In patients with aortic dissection, these therapies may worsen bleeding, increase the risk of aortic rupture, complicate subsequent surgery, and contribute to poor outcomes. Several case reports have described patients who initially received treatment for STEMI before the diagnosis of aortic dissection was recognized. These reports highlight the potential consequences of diagnostic error and reinforce the need for careful clinical assessment before initiating therapy when atypical features are present. [1,2,5]

Recognition of clues that suggest aortic dissection is therefore essential. Certain clinical findings should raise suspicion even when STEMI appears likely. These include abrupt onset of pain, pain radiating to the back, pulse asymmetry, unexplained hypotension, new diastolic murmurs suggestive of aortic regurgitation, focal neurological deficits, or evidence of ischemia involving multiple organ systems. While none of these findings is individually diagnostic, their presence should prompt consideration of alternative diagnoses and further evaluation before proceeding with standard STEMI treatment. [10,12]

Diagnostic imaging plays a central role in confirming or excluding acute aortic dissection. Computed tomography angiography remains the most widely used imaging modality because of its high sensitivity, broad availability, and ability to rapidly define the extent of disease. Transesophageal echocardiography is particularly useful in unstable patients and can provide valuable information regarding aortic valve involvement, pericardial effusion, and proximal aortic anatomy. Transthoracic echocardiography may also identify features suggestive of dissection, although its sensitivity is lower. Magnetic resonance imaging offers excellent diagnostic accuracy but is generally less practical in the acute setting. [10,12]

The challenge becomes even greater when patients proceed directly to coronary angiography because of presumed STEMI. In such situations, the diagnosis of aortic dissection may only become apparent during the procedure. Difficulty engaging the coronary arteries, visualization of an intimal flap, unusual coronary anatomy, or evidence of coronary compression may provide important clues. Recent reports have highlighted the role of intravascular ultrasound and advanced imaging techniques in identifying coronary involvement related to aortic dissection during cardiac catheterization. [1,5]

Management of Type A aortic dissection requires immediate stabilization followed by urgent surgical repair in most cases. Initial treatment focuses on reducing aortic wall stress through blood pressure and heart rate control while preparing for definitive intervention. Surgical management generally involves resection of the affected aortic segment with replacement using a vascular graft. Additional procedures may be required depending on the extent of disease, including aortic root reconstruction, valve repair, or coronary artery reimplantation. Despite advances in surgical techniques and perioperative care, mortality remains substantial, particularly when diagnosis is delayed or major complications are present. [10,14]

Coronary involvement represents one of the most serious complications of Type A dissection. Patients with myocardial ischemia or infarction often experience greater hemodynamic instability and may have worse outcomes compared with those without coronary malperfusion. In some cases, temporary coronary intervention may be performed to restore blood flow before definitive aortic surgery. However, management strategies must be individualized according to the patient's condition, the anatomy of the dissection, and the expertise available at the treating center. [1,6]

Recent case reports continue to demonstrate the diverse ways in which Type A aortic dissection can present. Several authors have described patients presenting with inferior STEMI due to right coronary artery involvement, while others have reported left main coronary artery obstruction resulting in extensive anterior infarction. These cases illustrate that aortic dissection should remain part of the differential diagnosis whenever the clinical picture appears unusual or when findings cannot be fully explained by conventional coronary artery disease alone. [1,2,3,6]

Although contemporary guidelines and improved imaging have enhanced diagnostic accuracy, missed or delayed diagnosis remains a persistent problem. The rarity of the condition compared with acute coronary syndrome, combined with the pressure to rapidly treat STEMI, creates an environment in which subtle warning signs may be overlooked. Increased awareness among emergency physicians, internists, cardiologists, and cardiac surgeons remains essential for improving outcomes. Maintaining a broad differential diagnosis is particularly important when evaluating patients with chest pain and electrocardiographic changes suggestive of myocardial infarction. [10,11]

The present case describes a patient with acute Type A aortic dissection presenting as inferior STEMI, a rare but clinically significant diagnostic challenge. This presentation highlights the close overlap between two cardiovascular emergencies that require fundamentally different treatment strategies. The case underscores the importance of careful clinical evaluation, early recognition of atypical features, and prompt use of appropriate imaging when suspicion for aortic dissection exists. By illustrating how a deadly aortic catastrophe can mimic a common coronary emergency, this report aims to reinforce awareness of this uncommon but potentially fatal presentation and to emphasize the need for vigilance in patients presenting with apparent STEMI.

Case Presentation

A 58 year old male with a history of hypertension presented to the emergency department complaining of sudden onset severe central chest pain that began approximately two hours prior to arrival. The pain started abruptly while he was at rest and was described as heavy and crushing in nature, with radiation to the back between the shoulder blades. It was associated with diaphoresis, nausea, and one episode of vomiting. He denied any recent trauma, fever, cough, or preceding illness. There was no previous history of ischemic heart disease, heart failure, or prior cardiac intervention.

On arrival, the patient appeared distressed and diaphoretic because of the severity of pain. Initial vital signs showed a blood pressure of 98/62 mmHg in the right arm, heart rate of 108 beats per minute, respiratory rate of 22 breaths per minute, temperature of 36.8°C, and oxygen saturation of 96% on room air. Physical examination revealed an alert but uncomfortable patient. Heart sounds were normal without an obvious murmur. Lung auscultation was clear bilaterally. Peripheral pulses were palpable in all extremities, although the left radial pulse appeared slightly weaker than the right. No focal neurological deficits were identified on examination.

An electrocardiogram obtained shortly after presentation demonstrated concerning ischemic changes. Interestingly, rather than established ST segment elevation, there were prominent hyperacute T waves in leads II, III, and aVF associated with reciprocal ST segment depression in the anterior leads, representing an inferior STEMI equivalent pattern (Figure 1). Given the patient's ongoing chest pain and electrocardiographic findings, acute coronary syndrome involving the right coronary artery was initially considered the most likely diagnosis.

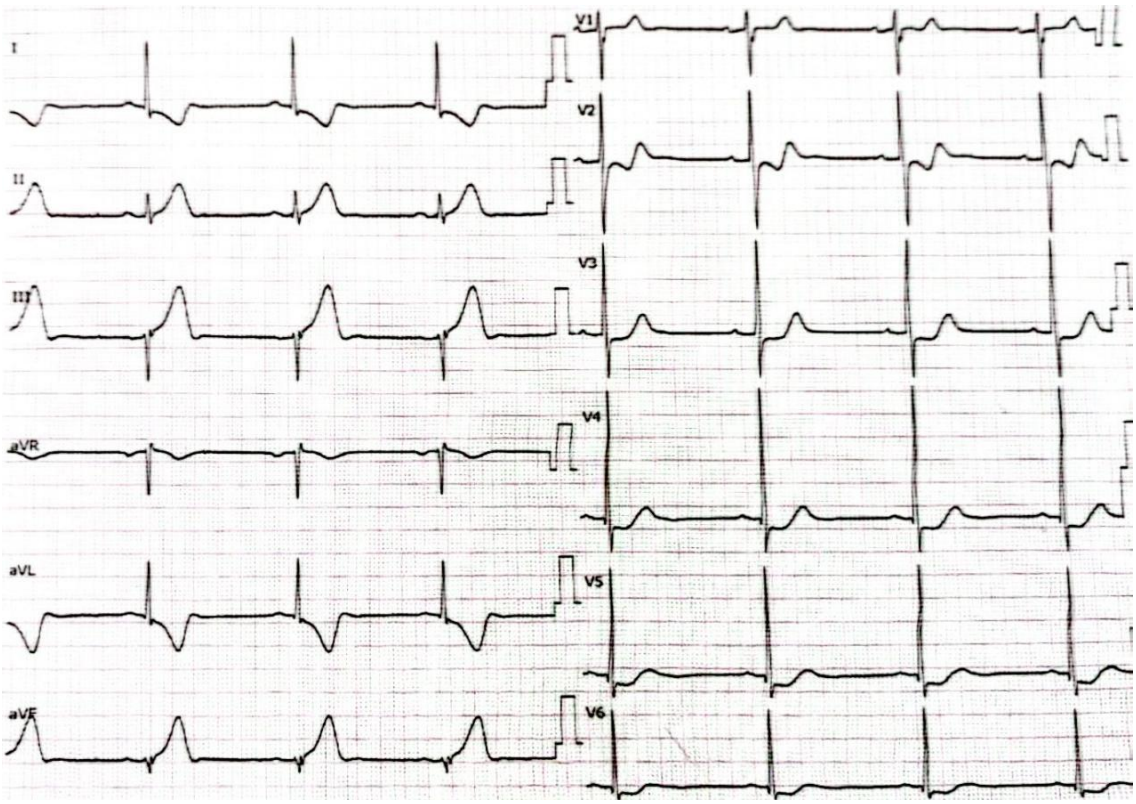

Figure 1: Twelve lead electrocardiogram demonstrating hyperacute T waves in the inferior leads (II, III, and aVF) with reciprocal ST segment depression in the anterior leads, consistent with an inferior STEMI equivalent pattern.

Initial laboratory investigations showed mild leukocytosis and an elevated high sensitivity troponin level of 145 ng/L. Renal and liver function tests were within normal limits. Venous blood gas analysis showed no significant metabolic abnormality. A portable chest radiograph demonstrated mild widening of the mediastinum, although this finding was initially considered nonspecific in the acute setting.

The patient received aspirin and was prepared for urgent coronary angiography. However, while awaiting transfer to the cardiac catheterization laboratory, he continued to report severe chest pain despite analgesia. The persistent radiation of pain to the back,

together with the mildly widened mediastinum on chest radiography and relative hypotension, raised concern for an alternative diagnosis. A bedside transthoracic echocardiogram was therefore performed before proceeding with invasive coronary assessment.

Focused echocardiography demonstrated a mildly dilated ascending aorta with a small circumferential pericardial effusion. Left ventricular systolic function was preserved, and there were no obvious regional wall motion abnormalities. Although image quality was suboptimal, a suspicious linear echogenic structure was noted within the proximal ascending aorta, raising concern for a possible intimal flap. Given these findings, the decision was made to obtain urgent computed tomography angiography of the chest before proceeding with coronary intervention.

Computed tomography angiography revealed an extensive Stanford Type A aortic dissection originating in the ascending aorta and extending into the aortic arch. The dissection flap involved the aortic root and extended to the origin of the right coronary artery, resulting in compromise of coronary blood flow. A small hemopericardium was also present, without evidence of overt cardiac tamponade. No rupture of the aorta was identified.

Following confirmation of the diagnosis, antithrombotic therapy was withheld, and cardiothoracic surgery was consulted immediately. The patient was transferred to the intensive care unit for close monitoring and initiation of intravenous antihypertensive therapy aimed at reducing aortic wall stress while awaiting definitive surgical management. Heart rate and blood pressure were carefully controlled using intravenous beta blockade.

The patient subsequently underwent emergency surgical repair. Intraoperative findings confirmed a Type A aortic dissection involving the ascending aorta with extension into the right coronary ostium. Replacement of the ascending aorta was performed using a vascular graft, and repair of the affected aortic root was undertaken. The right coronary artery was successfully preserved following restoration of normal anatomy.

The postoperative course was notable for transient vasopressor requirement during the first 24 hours but was otherwise uncomplicated. Serial cardiac biomarkers demonstrated a modest rise followed by gradual decline. Follow up echocardiography showed preserved left ventricular systolic function with no significant pericardial effusion and satisfactory appearance of the repaired ascending aorta. The patient was successfully extubated on postoperative day one and transferred from the intensive care unit several days later.

He continued to improve clinically throughout the remainder of his hospital stay. Repeat neurological examinations remained normal, and no evidence of renal dysfunction, limb ischemia, or other end organ complications developed. The patient was discharged home on postoperative day ten with antihypertensive therapy and arrangements for close follow up with both cardiology and cardiothoracic surgery services.

This case illustrates a potentially fatal presentation of acute Type A aortic dissection masquerading as inferior STEMI. The initial electrocardiographic findings, elevated cardiac biomarkers, and typical ischemic symptoms strongly suggested acute coronary syndrome. However, several subtle features, including persistent chest pain radiating to the back, relative hypotension, mild mediastinal widening, and suspicious findings on bedside echocardiography, prompted further investigation and ultimately led to the correct diagnosis. Early recognition prevented inappropriate catheter based intervention and allowed timely surgical repair of a life threatening condition.

Management course

Management began immediately upon the patient's arrival to the emergency department, where the initial working diagnosis was acute coronary syndrome with an inferior STEMI equivalent pattern on electrocardiography. Given the severity of his chest pain and concerning ECG findings, the patient was placed on continuous cardiac monitoring and pulse oximetry. Two large bore intravenous lines were secured, and blood samples were obtained for urgent laboratory investigations, including cardiac biomarkers, complete blood count, coagulation profile, renal function, liver function tests, and blood type and screen in anticipation of possible invasive intervention.

The patient was initially managed according to standard acute coronary syndrome protocols. Aspirin was administered, and preparations were made for urgent coronary angiography. Supplemental oxygen was not required because oxygen saturation remained within the normal range. Intravenous analgesia was provided to control ongoing severe chest pain, which remained one of the patient's most prominent symptoms. Despite treatment, the pain persisted and continued to radiate to the back, prompting further reassessment of the diagnosis.

During ongoing evaluation, several findings raised concern for an alternative cause of the patient's presentation. These included the abrupt onset of pain, radiation to the interscapular region, relative hypotension, and mild mediastinal widening on chest radiography. In view of these atypical features, the cardiology and emergency medicine teams elected to perform bedside transthoracic echocardiography before proceeding with invasive coronary intervention.

The echocardiographic findings of a mildly dilated ascending aorta, small pericardial effusion, and a possible intimal flap significantly increased suspicion for acute aortic dissection. At this stage, further antithrombotic therapy was withheld pending definitive imaging. The priority shifted from urgent coronary reperfusion to rapid exclusion of aortic pathology. An emergent computed tomography angiogram of the thoracic aorta was arranged and performed without delay.

Computed tomography angiography confirmed an extensive Stanford Type A aortic dissection involving the ascending aorta and aortic root, with extension toward the origin of the right coronary artery. A small hemopericardium was also identified. Following confirmation of the diagnosis, management priorities changed immediately. The patient was transferred to a high dependency monitored setting for close observation while arrangements were made for transfer to a tertiary cardiothoracic center.

Initial medical management focused on reducing aortic wall stress and preventing progression of the dissection. Intravenous beta blocker therapy was initiated with the goal of controlling heart rate and reducing the force of left ventricular contraction. Heart rate gradually decreased from approximately 108 beats per minute to the low 70s. Blood pressure was monitored closely through frequent noninvasive measurements, and additional antihypertensive therapy was administered as required to maintain safe hemodynamic targets while preserving adequate end organ perfusion.

Pain control formed an important component of management. Ongoing severe pain can stimulate sympathetic activation and lead to further increases in blood pressure and heart rate. Intravenous opioid analgesia was therefore administered in carefully titrated doses, resulting in significant symptomatic improvement. Following adequate analgesia and heart rate control, the patient appeared more comfortable and remained hemodynamically stable.

Serial clinical assessments were performed throughout his stay in the referring hospital. Repeated neurological examinations showed no evidence of stroke or spinal cord ischemia. Peripheral pulses remained palpable, and there were no signs of limb malperfusion. Urine output remained adequate, suggesting preserved renal perfusion. Serial laboratory investigations did not demonstrate significant deterioration in kidney function or evidence of major end organ ischemia during the stabilization period.

Given the confirmed diagnosis of acute Type A aortic dissection, the need for urgent surgical repair was recognized. However, cardiothoracic surgical services were not available at the presenting hospital. Following discussion between the emergency medicine, cardiology, intensive care, and cardiothoracic teams, arrangements were made for immediate transfer to a tertiary referral center with dedicated aortic surgery capabilities. The risks associated with delayed intervention were carefully considered, and transfer was prioritized as soon as the patient was sufficiently stabilized.

Prior to transfer, detailed handover information was communicated to the receiving center. This included the patient's clinical presentation, electrocardiographic findings suggestive of inferior STEMI, imaging results confirming Type A dissection, current hemodynamic status, medications administered, and the presence of a small hemopericardium. Copies of the CT angiogram and echocardiographic images accompanied the patient to facilitate rapid decision making upon arrival.

The anticipated management plan at the tertiary center consisted of urgent cardiothoracic surgical evaluation and operative repair of the ascending aorta. Based on the imaging findings, surgical intervention would likely involve replacement of the ascending aorta with a vascular graft and assessment of the aortic root and coronary artery involvement. Because the dissection appeared to extend toward the right coronary artery origin, intraoperative evaluation of coronary blood flow and possible repair or reconstruction would be expected if necessary.

Following transfer, the patient was admitted directly under the cardiothoracic surgery service and underwent further preoperative assessment. Repeat imaging and laboratory studies were performed to evaluate disease progression and surgical risk. Blood pressure and heart rate control were continued while preparations for surgery were finalized. The patient remained under close monitoring in a critical care setting because of the ongoing risk of aortic rupture, cardiac tamponade, coronary malperfusion, and sudden hemodynamic deterioration.

The patient subsequently underwent definitive surgical repair of the ascending aorta. The procedure was reported to be technically successful, with restoration of normal aortic anatomy and management of the affected aortic root. Postoperatively, he was monitored in the intensive care unit, where he remained hemodynamically stable without major complications. Serial examinations demonstrated no neurological deficits, and renal function remained preserved throughout recovery.

During the postoperative period, gradual mobilization and optimization of antihypertensive therapy were undertaken. Follow up imaging confirmed satisfactory repair with no immediate evidence of residual dissection involving the operated segment. The patient continued to improve clinically, with complete resolution of chest pain and stable cardiovascular parameters.

Prior to discharge, extensive counseling was provided regarding the nature of aortic dissection and the importance of long term blood pressure control. He was educated about the need for regular cardiovascular follow up and surveillance imaging to monitor the

remaining aorta. A structured outpatient follow up plan was arranged involving cardiology, cardiothoracic surgery, and primary care services.

This case demonstrates the importance of prompt stabilization and diagnostic reassessment when the clinical picture does not fully fit an initial diagnosis of acute coronary syndrome. Early recognition of atypical features, rapid imaging, careful blood pressure control, and timely referral to a specialized cardiothoracic center were critical steps in preventing potentially fatal complications and facilitating definitive treatment.

Discussion

Acute Type A aortic dissection is one of the most time critical emergencies encountered in cardiovascular medicine. Despite advances in imaging, emergency care pathways, and surgical techniques, it continues to carry substantial morbidity and mortality. One of the main reasons for adverse outcomes is the difficulty of diagnosis, particularly when the clinical presentation mimics more common conditions such as acute coronary syndrome. The present case illustrates a classic but uncommon diagnostic challenge in which an acute Type A aortic dissection presented with electrocardiographic findings suggestive of inferior STEMI. This overlap has important implications because the management of these two conditions differs fundamentally, and therapies that are beneficial in myocardial infarction may be harmful in aortic dissection. [10,11]

Acute aortic dissection occurs when a tear develops within the intimal layer of the aortic wall, allowing blood to enter the media and create a false lumen. Propagation of the dissection may occur both proximally and distally, resulting in involvement of branch vessels and multiple organ systems. The Stanford classification remains the most widely used system in clinical practice. Type A dissections involve the ascending aorta and require urgent surgical evaluation, whereas Type B dissections are confined to the descending aorta and are often managed medically unless complications develop. The distinction is clinically important because Type A dissection is associated with a significantly higher risk of death if left untreated. [10,12]

Data from the International Registry of Acute Aortic Dissection have provided valuable insight into the natural history of this disease. Historically, mortality for untreated Type A dissection was estimated to increase by approximately 1% to 2% per hour during the first 24 to 48 hours following symptom onset. Although modern management has improved outcomes, the condition remains associated with high mortality, particularly when diagnosis is delayed or complications such as cardiac tamponade, myocardial infarction, stroke, or aortic rupture occur. [13]

The incidence of acute aortic dissection is relatively low compared with acute coronary syndrome, which partly explains why it is often not the first diagnosis considered in patients presenting with chest pain. Nevertheless, its clinical importance is disproportionate to its incidence because of the potentially catastrophic consequences of a missed diagnosis. Current ACC/AHA guidelines emphasize that clinicians should maintain a high level of suspicion in patients presenting with abrupt onset severe chest, back, or abdominal pain, especially when associated with pulse deficits, neurological symptoms, unexplained hypotension, or mediastinal widening on imaging. [10]

One of the most important lessons from the present case is that aortic dissection can closely mimic myocardial infarction. Coronary malperfusion is a recognized complication of Type A dissection and occurs when the dissection flap extends into a coronary ostium or compresses coronary blood flow. Among the coronary arteries, the right coronary artery is affected more frequently than the left. Consequently, inferior wall ischemia and infarction represent the most common electrocardiographic pattern observed when aortic dissection presents as STEMI. [9,11]

Several mechanisms have been proposed to explain coronary involvement in acute dissection. Direct extension of the intimal flap into the coronary artery may create mechanical obstruction. Alternatively, expansion of the false lumen may compress the true lumen and reduce coronary perfusion. In some cases, circumferential detachment of the coronary ostium can occur, producing profound myocardial ischemia. The resulting clinical picture may be indistinguishable from primary coronary thrombosis, with chest pain, elevated cardiac biomarkers, and ischemic ECG changes. [1,4,6]

The electrocardiographic findings in our patient are particularly noteworthy. Rather than established ST segment elevation, the initial ECG demonstrated hyperacute T waves in the inferior leads accompanied by reciprocal ST depression in the anterior leads. Hyperacute T waves represent one of the earliest manifestations of transmural ischemia and are increasingly recognized as a STEMI equivalent. In many cases, this stage precedes the development of classic ST elevation. Recognition of this pattern is important because it may indicate ongoing coronary compromise despite the absence of traditional STEMI criteria. In the present case, involvement of the right coronary artery by the dissection likely produced early ischemic changes before complete infarction developed.

An important clinical question is whether there are clues that may help distinguish aortic dissection from acute coronary syndrome. Unfortunately, no single finding is sufficiently sensitive or specific to reliably exclude either diagnosis. The classic description of tearing chest pain radiating to the back is well known but not universally present. Data from IRAD demonstrated that many patients with

confirmed aortic dissection present with symptoms that overlap substantially with other cardiovascular conditions. Consequently, clinicians should avoid relying solely on textbook descriptions when evaluating patients with suspected dissection. [13]

Several features in this case should raise suspicion among experienced clinicians. First, the onset of pain was abrupt and maximal at presentation. Acute coronary syndrome often develops over minutes, whereas patients with dissection frequently describe a sudden explosive onset of symptoms. Second, radiation of pain to the interscapular region was present. Although not specific, this feature is more commonly associated with aortic pathology. Third, the patient demonstrated relative hypotension despite a history of hypertension. Hypotension in the setting of apparent STEMI should always prompt consideration of alternative diagnoses, including aortic dissection, mechanical complications of myocardial infarction, or massive pulmonary embolism. [10,12]

Another important clue was the chest radiograph demonstrating mediastinal widening. Although chest radiography lacks sufficient sensitivity to exclude aortic dissection, abnormal findings may provide valuable diagnostic direction. Mediastinal widening, abnormal aortic contour, pleural effusion, or displacement of calcified aortic intima may all be encountered. However, it should be emphasized that a normal chest radiograph does not exclude acute dissection, and further imaging is required whenever clinical suspicion remains significant. [10]

The role of bedside echocardiography deserves particular emphasis. In the present case, focused transthoracic echocardiography provided crucial information that altered the diagnostic pathway. Identification of ascending aortic dilation, a small pericardial effusion, and suspicion of an intimal flap prompted urgent computed tomography angiography before coronary catheterization. While transthoracic echocardiography is not sufficiently sensitive to rule out dissection, it can rapidly identify findings that support the diagnosis and guide further investigation. This is particularly valuable in emergency settings where time sensitive decisions must be made. [10,12]

Computed tomography angiography remains the diagnostic modality of choice in most stable patients with suspected acute aortic dissection. Modern multidetector scanners provide excellent spatial resolution and can rapidly define the location of the intimal tear, extent of disease, branch vessel involvement, and presence of complications. In our patient, CT angiography clearly demonstrated involvement of the ascending aorta and extension toward the right coronary artery, explaining the clinical and electrocardiographic findings. [10]

One of the greatest dangers in these cases is inadvertent administration of antithrombotic therapy before the diagnosis is recognized. Standard STEMI management frequently includes dual antiplatelet therapy, anticoagulation, and urgent coronary intervention. While appropriate for acute coronary occlusion, these treatments may significantly worsen outcomes in patients with aortic dissection. Increased bleeding risk, propagation of the dissection, hemopericardium, and difficulties during emergency surgery have all been reported following inappropriate antithrombotic treatment. Numerous case reports describing dissection initially mistaken for STEMI have highlighted this issue. [1,2,5,6]

This diagnostic dilemma creates a practical challenge in modern cardiovascular care. Current STEMI systems are designed to minimize door to balloon time, and clinicians are encouraged to activate reperfusion pathways rapidly. While these systems have improved outcomes for myocardial infarction, they also create a risk that uncommon but dangerous alternative diagnoses may be overlooked. The present case reinforces the importance of maintaining clinical vigilance even when ECG findings appear highly suggestive of STEMI.

Management priorities change dramatically once Type A dissection is identified. The immediate goal becomes reduction of aortic wall stress through control of heart rate and blood pressure. Current ACC/AHA guidelines recommend beta blockade as first line therapy because reducing the rate of left ventricular contraction decreases shear stress on the aortic wall. Additional vasodilator therapy may be considered after adequate heart rate control has been achieved. Effective analgesia is also important because uncontrolled pain can stimulate sympathetic activation and worsen hemodynamic stress. [10]

Definitive treatment of acute Type A dissection is surgical repair. The exact operative approach depends on the location and extent of disease, involvement of the aortic root, severity of aortic valve dysfunction, and presence of coronary involvement. In cases such as ours, where the right coronary artery appears compromised, surgical planning must include careful assessment of coronary perfusion and potential reconstruction if required. Advances in surgical techniques have substantially improved outcomes, although perioperative mortality remains significant due to the severity of the underlying disease. [10,14]

Coronary malperfusion has consistently been associated with worse outcomes in patients with Type A dissection. These patients often present with greater hemodynamic instability and may experience delays in diagnosis because the presentation mimics acute myocardial infarction. Some reports have described temporary coronary stenting before surgery as a bridge to definitive repair, although management remains individualized and dependent on local expertise and patient stability. [1,6]

The present case also highlights the value of reassessing the diagnosis when the clinical picture does not fully fit the presumed condition. Cognitive bias remains an important contributor to diagnostic error in emergency medicine. Once an ECG suggestive of

inferior STEMI is identified, there is a natural tendency to anchor on that diagnosis and interpret subsequent findings through the same lens. However, the persistence of severe back pain, relative hypotension, and mediastinal widening prompted reconsideration of the initial diagnosis and ultimately prevented potentially harmful intervention. Experienced clinicians often emphasize the importance of actively searching for findings that do not fit the presumed diagnosis, particularly in high risk situations.

From an educational perspective, several practical lessons emerge from this case. Hyperacute T waves in the inferior leads should not automatically be assumed to represent primary coronary thrombosis, especially when accompanied by atypical symptoms. Chest pain radiating to the back should always prompt consideration of aortic pathology. A mildly widened mediastinum may represent an important clue even when other findings suggest myocardial infarction. Bedside echocardiography can provide valuable information within minutes and should be considered when uncertainty exists. Most importantly, clinicians should remember that not every apparent STEMI is truly a STEMI.

Recent case reports continue to demonstrate the wide spectrum of presentations associated with Type A aortic dissection. Reports describing inferior STEMI, transient ST elevation, diffuse ST elevation resembling pericarditis, and left main coronary artery obstruction have all been published in recent years. Collectively, these cases reinforce a central message: aortic dissection remains one of the great imitators in cardiovascular medicine. [1,2,3,6,7]

In summary, this case illustrates a rare but clinically significant presentation of acute Type A aortic dissection masquerading as inferior STEMI. Coronary involvement, particularly of the right coronary artery, can produce ischemic symptoms, elevated troponin levels, and ECG findings that closely resemble acute myocardial infarction. Early recognition is essential because management differs fundamentally from standard STEMI treatment. Careful attention to atypical clinical features, appropriate use of bedside imaging, and timely confirmation with computed tomography angiography can facilitate accurate diagnosis and prevent potentially catastrophic consequences. This case serves as a reminder that even in the era of rapid reperfusion pathways, clinicians must remain alert to alternative diagnoses when evaluating patients with acute chest pain and apparent myocardial ischemia.

Conclusion

This case highlights one of the most dangerous diagnostic pitfalls in acute cardiovascular medicine: the assumption that all STEMI equivalent patterns represent primary coronary artery occlusion. Acute Type A aortic dissection can closely mimic acute myocardial infarction, particularly when the dissection extends into the coronary circulation and produces ischemic electrocardiographic changes. In such situations, the diagnosis may be easily missed, especially in modern emergency pathways where rapid reperfusion is appropriately prioritized. However, the consequences of overlooking aortic dissection can be catastrophic, as therapies routinely used for STEMI may significantly worsen outcomes in patients with an underlying aortic catastrophe.

Several important clinical lessons emerge from this case. First, the diagnosis of acute coronary syndrome should always be interpreted within the broader clinical context. Features such as abrupt onset chest pain, radiation to the back, unexplained hypotension, pulse asymmetry, or mediastinal widening should prompt consideration of acute aortic pathology, even when ECG findings appear highly suggestive of myocardial infarction. Second, hyperacute T waves and STEMI equivalent patterns are markers of myocardial ischemia but do not identify its cause. Clinicians must remain alert to alternative mechanisms of coronary compromise, including coronary malperfusion from aortic dissection.

This case also reinforces the value of bedside echocardiography and early cross sectional imaging when the presentation contains atypical features. A brief reassessment before proceeding with invasive coronary intervention can occasionally reveal findings that fundamentally change management and alter the patient's prognosis. In our patient, recognition of subtle clues led to timely diagnosis, avoidance of potentially harmful treatment, and successful referral for definitive surgical management.

Ultimately, the most important message is that acute Type A aortic dissection remains a master imitator. Maintaining diagnostic vigilance, questioning findings that do not fully fit the presumed diagnosis, and keeping a broad differential diagnosis can be lifesaving. In patients presenting with apparent inferior STEMI, remembering this rare but critical possibility may be the difference between a missed opportunity and a successful outcome.

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