CASE REPORT

Ascending aortic dissection presented as inferior myocardial infarction: a clinical and diagnostic mimicry

Ali A Alsaad,¹ Olufunso W Odunukan,² John Norman Patton²

¹Department of Internal Medicine, Mayo Clinic Florida, Jacksonville, Florida, USA

²Department of Cardiovascular Diseases, Mayo Clinic, Jacksonville, Florida, USA

Correspondence to
Dr Ali A Alsaad,
alsaad.ali@mayo.edu

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SUMMARY

Acute ST segment elevation myocardial infarction (STEMI) is typically associated with acute coronary thrombosis or plaque rupture. Rarely, STEMI can be associated with ascending aortic dissection, which represents the majority of acute aortic syndrome aetiologies and carries dreadful outcomes. Routine cardiac intervention with emergent cardiac catheterisation may lead to a higher mortality rate in this group of patients. We present a case of painless inferior STEMI in the setting of ascending aortic dissection. The patient had an inferior STEMI due to the involvement of the right coronary artery as an extension of the ascending aortic dissection. In this era of protocol-driven practice and the pressure to fulfil quality measures, we aim to alert emergency physicians, cardiologists and interventionalists of the possible presentation of painless ascending aortic dissection as an STEMI. The two pathologies characterise by crucial differences in their initial and ultimate management.

BACKGROUND

Cardiovascular disease represents the vast majority of mortality causes in the USA and all over the world.¹ Acute ST segment elevation myocardial infarction (STEMI) is a readily treatable cardiovascular diseases if the appropriate intervention occurs within a timely manner.² However, not all STEMI events should undergo cardiac catheterisation. Other superimposing aetiologies should be considered first; in particular, catheterisation of an ascending aortic dissection (AAD) or a dissecting coronary artery, as that might impose risk for rupture, expansion of the dissection and death.³

CASE PRESENTATION

A 77-year-old woman with a 45-pack-years smoking history and obstructive pulmonary disease presented to the emergency department with 3 days of severe dyspnoea, left arm numbness and tingling with no reported chest or back pain. She denied fever, orthopnoea, paroxysmal nocturnal dyspnoea or lower extremities swelling. Other than 'not feeling well', she did not report any other symptoms. Medical history included chronic obstructive lung disease and hypertension that were treated with inhaled β_2 agonists/anticholinergic and losartan, respectively. She had no history of diabetes mellitus or neuropathy and her last haemoglobin A1C level was found to be 5.1%.

At time of presentation, she was afebrile, blood pressure was 88/55 mm Hg in the right arm and 80/51 mm Hg in the left arm, heart rate was 109 bpm in sinus rhythm and respiratory rate was 23 breaths per minute. Physical examination revealed moderate respiratory distress. Peripheral pulses were weak with minimal bilateral pedal oedema. Carotid upstroke was weaker on the right side with normal down stroke. She had normal S1 and S2. There was no jugular venous distension, murmur, S3 or S4 heart sounds.

INVESTIGATIONS

Complete blood count and electrolytes were within normal limits. Arterial blood gases showed respiratory acidosis with a pH of 7.21, PaCO₂ of 59.9 mm Hg and PaO₂ of 120 mm Hg on 4 L of oxygen. High-sensitivity C reactive protein was elevated at 114 mg/L (normal reference ≤ 8.0 mg/L), and venous lactate was 4.4 mmol/L (normal reference 0.9–1.7 mmol/L); troponin T was 0.05 ng/mL (normal reference <0.01 ng/mL); B-natriuretic peptide was 256 pg/mL (normal reference <38 pg/mL) and d-dimer was 5000 ng/mL DDU (normal reference ≤ 250 ng/mL DDU).

Electrocardiography showed ST segment elevation in the inferior leads II, III and aVF (figure 1). A portable chest radiograph showed a widened superior mediastinum (figure 2). An urgent bedside transthoracic echocardiogram revealed a dilated ascending aorta with a false lumen and possibly a bicuspid aortic valve (figure 3). This picture was suggestive of an acute aortic dissection. A multidetector computerised axial tomography angiography (CTA) of the chest demonstrated a 5.0 cm diameter ascending aorta with a Stanford type-A acute dissection arising from the level of the aortic annulus (figure 4). The right coronary artery (RCA), unlike the left main coronary artery, was arising from the false lumen created by the dissection (figure 4 and 5).

DIFFERENTIAL DIAGNOSIS

The diagnosis of painless aortic dissection can be challenging and critical especially when it presents with ST segment elevation on electrocardiogram (EKG). Distinguishing aortic dissection from STEMI is extremely important due to the fundamental differences in the management of the two conditions.

TREATMENT

The patient was immediately taken to the operation room (ACS). The emergency surgery confirmed the

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Figure 1 Initial electrocardiography obtained immediately at time of arrival to the emergency department showing prominent ST segment elevation in the inferior leads (II, III and aVF) (red arrows). Reciprocal changes represented by ST segment depression in septal leads (V1 and V2) and aVL (blue arrows). Also, an unexplained ST segment elevation was noted in anterior leads V3 and V4, which theoretically could be related to the massive inferior wall ischaemia. Findings are suggestive of acute inferior myocardial infarction and right coronary artery disease.

Figure 2 Chest radiograph showing a widened superior mediastinum and prominently enlarged ascending aorta (red arrows). Hyperinflated lungs are noted secondary to the history of chronic obstructive airway disease.

diagnosis of ascending aortic aneurysm with acute dissection, bicuspid AV and a right ventricular infarction.

OUTCOME AND FOLLOW-UP

The patient underwent surgical repair of the aortic dissection, resuspension of the AV and repair of the aortic arch.

However, her intraoperative course was complicated by cardiac arrest with subsequent cardiorespiratory failure. She underwent prolonged hospitalisation in the critical care unit before she died from septic shock related to healthcare-associated pneumonia.

DISCUSSION

Acute aortic syndrome (AAS) is a constellation of pathological subtypes that involve the major aortic trunk.⁴ It ranges from AAD, aortic aneurysm and intramural haematoma to a ruptured aortic atheromatous plaque and penetrating aortic ulcer.^{5 6} The estimated incidence of AAD is 5–30 cases per million with a man:woman ratio of 2:1.^{4 5} Although all subtypes of AAS carry high morbidity and mortality rates, thoracic AAD carries the worst prognosis.^{7 8} The rate of mortality from AAD is 1–2% in the first few hours from presentation.^{8 9}

A wide variety of clinical presentations can be encountered secondary to thoracic AAD. Severe tearing chest pain, back pain or abdominal pain are the most classical presentation and present in 12–31% of patients.¹⁰ Neurological manifestations, such as numbness, tingling, focal neurological signs and Ortnier syndrome, can occasionally be the presenting symptoms.^{5 11} Ortnier syndrome or cardiovascular syndrome is described as compression of the recurrent laryngeal nerve secondary to cardiovascular disease causing hoarseness.¹¹ Painless dissection is reported in nearly 6–10% of the thoracic AAD.^{11 12} It is usually associated with neurological manifestations, syncope, stroke and heart failure.^{12 13} Mortality can reach 30% when pain is not the characteristic presentation in AAD.^{12 14 15}

The classification of the AAD can be conducted using two reliable classification systems: DeBakey (I, II and III) and Stanford (A and B).¹⁶ Our patient had a DeBakey type I, Stanford type-A AAD with the RCA uniquely involved in the dissection process. The involvement of RCA in the dissection

Figure 3 Transthoracic echocardiography. (A) An enlarged image of a parasternal long-axis view showing the dissection flap of the ascending aorta originated from the right coronary cusp (red arrow). (B) Parasternal long-axis view with Doppler showing the dissection flap in the ascending aorta (red arrow) and thick calcified aortic valve. (C) Doppler image in the parasternal long-axis view showing findings suggestive of possible bicuspid aortic valve with significant aortic valve insufficiency (blue arrows). (D) Parasternal short-axis view (image is not completely clear due to a technical difficulty because of emphysema) showing aortic valve with only two leaflets suggestive of bicuspid aortic valve. Ao, ascending aorta; RV, right ventricle; LV, left ventricle; RA, right atrium; LA, left atrium.

Figure 4 Sagittal sections of the computerised axial tomography angiography of the chest. (A) A 5-cm diameter ascending Stanford type-A aortic, DeBakey type I aortic dissection arising from the level of the aortic annulus and creating a false lumen (red asterisk). The false lumen resolves in the descending thoracic aorta at the level of the diaphragm (red arrow). (B) The right coronary artery arises from the false lumen (yellow arrows). The dissection flap extending into the innominate artery bifurcates into right common carotid and right subclavian arteries. (C) Right coronary artery arises from the false lumen (yellow arrow). No aneurysmal dilation of the abdominal aorta. (D) The left common carotid and left subclavian arteries arise from the true lumen of the aorta (yellow arrow).

Figure 5 (A) Computerised tomography angiography axial section of the chest showing the pseudo-lumens (red bold arrows). (B) Computerised tomography angiography coronal section of the chest showing the left main coronary artery arising from the true lumen of the left sinus of Valsalva (blue arrow).

process is quite rare and requires a high index of suspicion to establish the diagnosis.

In one case report, Stanford type-A AAD was shown to extend and involve the ostium of the RCA, causing a presentation mimicking inferior wall myocardial infarction.¹⁷ However, our case is different because we were able to diagnose and recognise the AAD early, prior to taking the patient to the catheterisation laboratory table. Instead, our patient was appropriately triaged to the operating room due to that early diagnosis.

Generally, the EKG findings in AAD are variable and might be an important distractor leading to inappropriate diagnosis and treatment of this serious disease. One study on Stanford type-A AAD found that ~50% and 37% of those patients have acute and chronic EKG changes, respectively.¹⁸ However, 27% of those patients have normal EKG, and only 8% have ST segment elevation (STE). Those with STE usually have direct coronary artery involvement.¹⁸

This concomitant AAD and RCA involvement potentially leads to poor outcome and delayed intervention.¹² Increased mortality in the setting of cardiac catheterisation is attributable to two main reasons: possible extension of the dissection or rupture by direct manipulation during catheterisation and anticoagulation. Therefore, distinguishing RCA territory involvement in AAD from acute coronary syndrome (ACS) due to plaque rupture or thrombosis is crucially important prior to the decision of management, particularly starting anticoagulation and dual antiplatelet therapy.

Obtaining an aortic CTA in this scenario is extremely helpful to confirm the suspicion and establish the diagnosis of aortic dissection. However, it may take a long time to obtain the CTA, jeopardising the time to an emergent surgical intervention, which is necessary for this condition. In our institution, we are generally able to obtain a very quick CTA (within 5–8 min) and during that time, the operation room is prepped to receive the patient as soon as the CTA is completed. In other institutions, surgeons might accept the results of the bedside echocardiography, if the results are compelling.

AV involvement in patients with AAD is not uncommon and usually results in aortic regurgitation (AR).⁶ Four main mechanistic forms of AV involvement have been established in patients with AAD: (1) eccentric AV leaflet prolapse and distortion caused by the dissecting aortic flap (the most common form), (2) central AR due to incomplete evulsion of the aortic leaflets from the aortic root, (3) circumferential intimal flap and (4) pre-existing AR from root dilation or bicuspid AV (BAV). Our patient had a BAV which was likely the trigger for her aortic dissection. The relationship between BAV and AAD is physiologically explained by wider aortic root dilation.^{6 19} Those patients have a nearly 10 times increased risk of AAD than the general population.¹⁹ In the BAV, aortic dilation can involve the aortic root, the sinotubular junction or the ascending aortic segment.¹⁹ Therefore, appropriate monitoring and timely intervention are warranted in patients with BAV.¹⁹ The other possible complication of Stanford type-A AAD is pericardial haematoma and tamponade, and they are usually associated with ST/T segment changes.¹⁸

Few studies have shown the role of d-dimer in the diagnosis of AAD. One study found that a d-dimer level of ≥ 1600 ng/dL within 6 hours of the beginning of the chest pain has a high and significant positive likelihood ratio of 12.8, which suggests a high probability of AAD.^{20 21} However, the exact role of biomarkers has not been established in the ACCF/AHA guidelines and algorithms yet. Our patient had a high d-dimer level (5000 ng/dL), which raised the suspicion for AAD.

Learning points

- ▶ Acute myocardial infarction can be a huge distractor when it complicates an acute ascending aorta dissection.
- ▶ Involvement of the right coronary artery can occur in the setting of painless acute aortic dissections.
- ▶ A high index of suspicion by using the general clinical picture is needed to establish the diagnosis.
- ▶ Imaging becomes critical in the early diagnosis of acute aortic dissections, especially in the absence of classical chest pain.

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