CASE REPORT

Thoracic aortic dissection presenting as acute coronary syndrome

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SUMMARY

A patient presented with symptoms consistent with non-ST elevation myocardial infarction (NSTEMI) and was given appropriate antiplatelet and anticoagulant therapy. Despite treatment of the NSTEMI and fluid resuscitation the patient remained persistently hypotensive. Further imaging demonstrated an extensive Stanford type A dissection extending from the aortic root to the left common iliac artery. The patient was taken to theatre and had an emergency hemiarch replacement with repair of the aortic root. Post surgery the patient had an uneventful recovery. We present this case to highlight the importance of considering dissection as a differential in patients presenting with chest pain and the lack of symptoms a patient with such a severe dissection can display.

BACKGROUND

As a wise consultant told me if you hear the sound of hooves on the beach it will almost always be donkeys, but occasionally it will be a zebra. This case appeared to be a straightforward and commonly seen acute coronary syndrome, but on further investigation turned out to be one of the rarer causes of chest pain—a significant aortic dissection.

Aortic dissection is a life-threatening emergency with a 30% in-hospital mortality rate.¹ However, the incidence of aortic dissection is relatively low with the UK and the USA having an incidence of 3–4 cases/100 000 population/year.² This is compared to the incidence of out-of-hospital myocardial infarction, which in England is 110/100 000/year.

The most common presenting symptom of an acute dissection is chest pain; consequently the differential diagnosis includes many conditions which occur with greater frequency, leading to a risk of misdiagnosis.^{2–7}

There are multiple signs and symptoms associated with aortic dissection, but no one finding is both specific and sensitive. As a result, a high index of suspicion is required to correctly identify these patients. In an attempt to combat this, the *American Journal of Cardiology* has devised a risk assessment pathway to allow for the early identification of potential aortic dissections.⁸ In one patient experience group they found an initial misdiagnosis rate of 15.1% with almost half of these (47.1%) being diagnosed as acute coronary syndromes. The European Society of Cardiology found that up to 30% of patients who were diagnosed with aortic dissection were found to have been diagnosed with another condition such as acute coronary syndrome or pulmonary embolism, prior to the diagnosis of aortic dissection.⁹

Although the two conditions may present in a similar manner, the treatment of the two conditions is very different, with the treatment of ACS being potentially detrimental to the patient who has an aortic dissection. The National Institute for Health and Care Excellence (NICE) guidelines state that immediate treatment of a non-ST elevation myocardial infarction (NSTEMI) once diagnosed should be 300 mg aspirin, 300 mg clopidogrel and 2.5 mg fondaparinux. Treatment for thoracic aortic dissection depends on the type, but surgery is generally indicated for Stanford type A dissections.

CASE PRESENTATION

A 58-year-old man arrived by ambulance with hypotension and chest pain. He described a 5-min episode of a tight 'vice like' band around his chest with associated shortness of breath. There was no radiation of the pain, no vomiting, no sweating and he was now pain free.

He reported no previous episodes of chest pain. His only significant medical history was atrial fibrillation and below knee deep venous thrombosis over 10 years ago. He was a non-smoker, who drank infrequently.

On examination he remained hypotensive with a systolic of 80 mm Hg, his pulse was varying between 80 and 115 bpm and was irregularly irregular, and he had an ejection systolic murmur radiated to the carotids which had not previously been noted. His respiratory examination was unremarkable with oxygen saturations of 100% on room air.

There was no radial–radial or radial–femoral delay, and no difference in blood pressure between the arms.

INVESTIGATIONS

Initial ECGs showed atrial fibrillation with ST depression in leads V4–V6.

Chest X-ray showed no focal consolidation and no mediastinal widening.

Routine initial blood tests (urea and electrolytes, full blood count, liver function and cardiac troponin) were normal. Repeat blood tests after the CT showed a drop in haemoglobin of 2 g/dL and an increase in white cell count. Urea and creatinine levels on the repeat blood tests had not deteriorated.

Cautious fluid boluses did not improve the blood pressure. In discussion with the cardiology team CT aortogram was requested, with cardiologist performed bedside transthoracic echocardiography in the interim to assess for significant left ventricular impairment.

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The echocardiogram showed an ascending aortic dissection, but was unable to ascertain if the aortic root was involved.

Urgent CT aortogram and coronary angiogram showed a Stanford type A dissection from the aortic root to the origin of the left common iliac artery, with no involvement of the coronary arteries. The dissection flap did involve the left common carotid and inferior mesenteric arteries. The left renal artery was communicating with the false lumen, but was receiving a small amount of blood through the true lumen (figures 1 and 2).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for a 58-year-old man with transient chest pain is wide. The initial ECG changes were suggestive of acute coronary syndrome (NSTEMI or unstable angina). The final diagnosis was of Stanford type A aortic dissection. Pulmonary embolus, pneumonia, pericarditis and musculoskeletal chest pain are also important differentials.

TREATMENT

Our patient initially received the standard treatment for acute coronary syndrome (300 mg aspirin, 300 mg clopidogrel and 2.5 mg fondaparinux).

On close questioning after the CT was obtained he reported mild residual pain. At this point his systolic blood pressure had risen to 100 mm Hg and so small aliquots of morphine were given to completely eliminate the pain and reduce sympathetic drive. The target blood pressure of 90 mm Hg was sufficient to maintain adequate cerebral perfusion as evidenced by his remaining fully conscious and lucid.

OUTCOME AND FOLLOW-UP

Once the diagnosis was made, the cardiothoracic team was contacted for urgent review. The patient was taken to theatre later that evening for an emergency ascending aortic and hemiarch replacement with size 12 interposition Dacron tube graft. The surgery was successful, and the patient spent 5 days on the cardiac intensive care unit before being transferred to a general cardiothoracic ward and then home.

Figure 1 Coronal view of type A dissection—from the root of the aorta extending through the diaphragm and continuing downwards.

Figure 2 Transverse view of type A dissection at the level of the pulmonary veins in both the ascending and descending aorta.

DISCUSSION

As aortic dissection is a dynamic process and can occur in any part of the aorta, the symptoms and signs that it presents with are wide ranging and may be vague. There are multiple publications advising on how best not to miss a case, but as yet there is no single test other than CT aorta which can diagnose dissection consistently and accurately.

The underlying pathophysiology behind an aortic dissection is a tear in the intimal layer of the aorta which leads to the creation of a false lumen between the aortic intima and media. This tear can then propagate both proximally and distally. The consequences of dissection can include volume loss, organ ischaemia and subsequent infarction. Dissection can be classified as Stanford type A which involves the ascending aorta, and Stanford type B which involves only the descending aorta.¹⁰

The key predisposing factors for the development of dissection are hypertension and age. As patients age the walls of the aorta undergo degenerative changes which make them more likely to tear. During the normal cardiac cycle the aorta and heart move with the contractions of the ventricles. There are a number of points where the heart and aorta are fixed within the mediastinum. The junctions between fixed and mobile segments experience higher blood pressure during the cardiac cycle. Hypertension further increases the pressure within these weak points which makes an intimal tear and resultant dissection more likely.²

The classical presentation of a dissecting aneurysm is that of the sudden onset of 'tearing' chest pain which radiates to the back, with associated pulse or blood pressure deficit between the upper limbs, and an abnormal chest X-ray. These classical features are only present in one-third of patients who present with a dissection. It is of note that our patient presented with a vice-like anterior chest pain, no pulse deficit or blood pressure difference and a normal X-ray.¹¹

There was a new heart murmur present, but even this was not the classical aortic regurgitation murmur heard after disruption of the annulus by an ascending dissection.

Patients can present with a wide variety of symptoms based on where the false lumen arises, how far the dissection has spread, and whether vessels arise from the true or false lumen.

The initial management for a dissection is the same independent of type. The patient should be kept pain free and their

blood pressure controlled using intravenous β blockers (eg, Labetalol). Patients who have a type A dissection, if they are fit for surgery, will require emergency operative repair. Patients who have a type B dissection can be managed medically and monitored for the development of complications which will determine if surgical intervention is required.^{8 9}

The symptoms and signs associated with a dissection can easily fit a diagnosis of acute coronary syndrome particularly if there are supporting ECG changes. Indeed, a type A dissection can involve the coronary arteries causing cardiac ischaemia and infarction.¹² As the mantra 'time is muscle' applies to patients with acute coronary syndrome, the emphasis is on initiating treatment as soon as possible after the diagnosis is made. This case highlights the need to keep a broad differential in mind when assessing patients with chest pain, and in particular the importance of reassessing the possibilities if a patient does not respond to treatment in the expected manner.

Learning points

- ▶ Although aortic dissection is a rare cause of chest pain, it should always be included in your list of differentials and evidence for it actively sought.
- ▶ If a patient is not responding to the treatment for the initial diagnosis always consider that there could be another cause for their symptoms.
- ▶ If available, bedside echocardiography will sometimes allow a rapid diagnosis.
- ▶ Always take time to explain to the patient and their family in non-medical jargon what is happening—our patient went from a possible minor heart attack to life-threatening surgical emergency in under 2 h.

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