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A very unusual headache

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Case presentation

A 43-year-old-builder developed a sudden-onset headache that radiated to the back of his head, while working on a roof in the afternoon. The pain was the worst that he had experienced and was associated with blurred vision, nausea and vomiting. The previous night he had consumed one bottle of wine. He admitted to taking cocaine at weekly intervals, the last time being 6 weeks previously. He used sildenafil to achieve erections, the last time being 3 weeks ago. His past medical history was unremarkable except for mild asthma and depression (treated with citalopram). He presented to the emergency department within the hour, where he was given morphine for analgesia.

Examination revealed no focal neurology; there was no neck stiffness and Kernig's sign was negative. Heart sounds were normal with no added sounds and the chest was clear to auscultation. Blood pressure was 107/57 mmHg, pulse 80 beats/min and oxygen saturations 96% on air, with a respiratory rate of 16 breaths/min. His electrocardiography (ECG) was unremarkable and bloods including inflammatory markers were normal. A computed tomography (CT) scan of the head was normal.

The working diagnosis at this stage was subarachnoid haemorrhage (SAH), although the admitting doctor noted that the features were atypical. A plan was made to observe the patient overnight and perform a lumbar puncture in the morning to look for xanthochromia. Ten hours after admission, the patient became increasingly unwell with worsening headaches and neck and chest pain. On examination, his oxygen saturation was 90% on air and his blood pressure was 108/60 mmHg in the left and 95/58 mmHg in the right arm. A new diastolic murmur consistent with aortic regurgitation was heard; his chest was clear and neurology intact.

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What was the differential diagnosis and the most likely diagnosis?

The sudden-onset headache and its severity suggested a possible SAH. However, this diagnosis would not explain the aortic regurgitant murmur or the low oxygen saturations. Pneumothorax was another possibility, but this would not be expected to cause a headache. Pulmonary embolism would cause hypoxia, but not a headache. Aortic dissection would cause chest pain which was present later in the admission. The severe neck pain associated with the new murmur was also highly suggestive of aortic dissection.[1]

What was the initial management?

The patient was given intravenous opiates and antiemetics**.** A chest radiograph showed a widened mediastinum (Fig 1) and a CT angiography (CTA) of the thoracic aorta was requested (Fig 2).

Case progression

CTA revealed a large dissecting flap in the ascending aorta, originating close to, but not involving, the aortic valve. It extended to the arch, terminating before the origin of the left subclavian artery. The dissection flap also involved the proximal aspect of the brachiocephalic artery. The descending aorta was of normal calibre. The cardiothoracic registrar saw the patient immediately and arranged transfer to the intensive care unit before surgery. In theatre, a valve-sparing aortic root replacement and interposition of ascending aortic graft was performed. Postoperatively, he developed pericardial effusions (which were drained) and atrial fibrillation, which was treated with amiodarone, as well as electrical cardioversion.

Discussion

Acute aortic dissection is an under-recognised and life-threatening medical emergency associated with a high

Key learning points.

- Sudden neck, jaw and occipital pain should alert the physician to the possibility of aortic dissection
- One-third of patients have a detectable murmur of aortic regurgitation
- A pulse deficit is seen in only 15% of patients with aortic dissection, but it is a significant finding in young patients
- Aortic dissection is linked to cocaine and possibly to sildenafil use

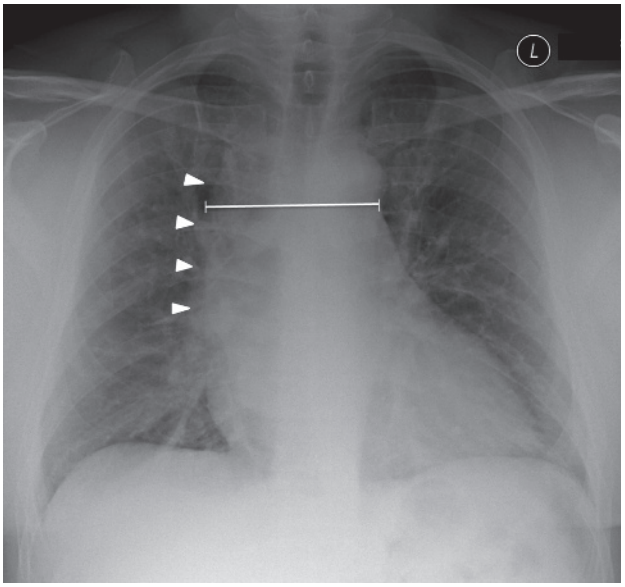

Fig 1. Erect anteroposterior chest radiograph showing non-traumatic mediastinal widening.

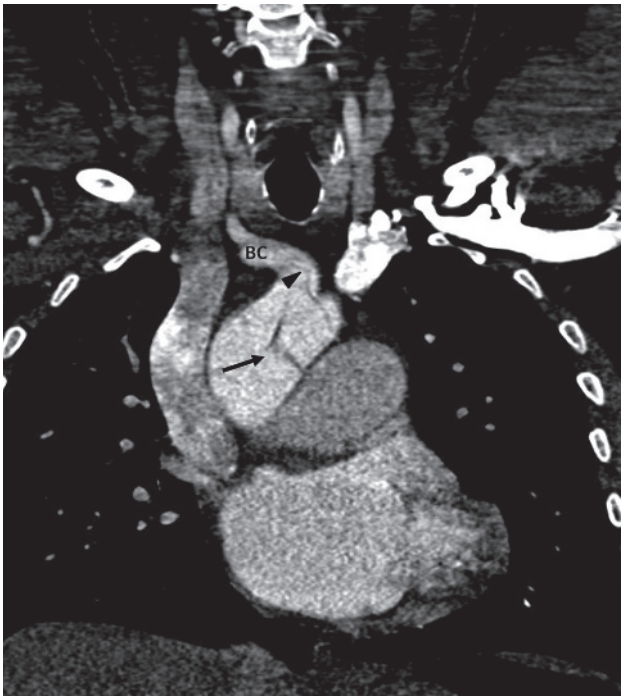

Fig 2. Computed tomography angiogram of chest showing main dissection flap (arrow) and the extension (arrowhead) into the origin of the brachiocephalic artery (BC).

mortality; the incidence is approximately 3 cases per 100,000 people per annum.[1] The Stanford classification is preferred by physicians and classifies aortic dissections as either type A, involving the ascending aorta, necessitating surgical intervention, or type B, in which the flap originates from the descending aorta and is usually treated medically. In type A aortic dissection early surgical intervention is crucial,[1] but diagnosis requires a high index of suspicion if it is to be made early and especially in young patients in whom atypical presentations are more common.[2]

Dissection is a dynamic process and the clinical spectrum of presentation is broad, can evolve rapidly over time and can mimic more common conditions, including myocardial ischaemia and stroke; as such diagnostic delay is common.[1,2]

Recognition of this condition is based on the identification of classic clinical features. According to registry data, the typical patient with an acute type A dissection is in his or her seventh decade, with a background of hypertension and presents with abrupt-onset chest pain.[2] A pulse deficit is seen only in 15% of cases and the murmur of aortic regurgitation in 32%.[2] Evolving symptomatology is useful in localising the dissection and is often predictable based on the affected anatomy, although the clinical picture can be confusing.[3] Involvement of the branches of the aorta result in organ malperfusion syndromes and complications.[1] Aortic dissection mimicking an SAH is a rare presentation and has been attributed to malperfusion of the carotid arteries.[3] In our patient, as the brachiocephalic artery was affected, there is likely to have been inadequate perfusion of the right common carotid artery, leading to headache.

Established risk factors for aortic dissection include: hypertension, age, atherosclerosis and previous aortic surgery; younger patients are more likely to have connective tissue disorders, Turner's syndrome or a bicuspid aortic valve.[1] Recreational drug use is another important, but under-recognised, risk factor in young patients. Tiryakioglu *et al*[4] report a case of a 28-year-old man with aortic dissection attributed to sildenafil used 2 hours previously, whereas Nusair *et al*[5] report a case of a 46-year-old man who presented with aortic dissection after using crack cocaine. It has been suggested that the catecholamine surge after cocaine use results in severe episodic hypertension which increases the shear stress on the aortic wall, resulting in an intimal tear.[5] This may be made worse by cocaine-induced atherosclerosis. Sildenafil decreases aortic stiffness in humans and it has been speculated that it can trigger intimal tearing,[4] although the case for sildenafil is less clear – the patient last used this 3 weeks previously.

Doctors are trained to look for certain 'classic' features when considering aortic dissection: retrosternal chest pain, pulse deficit and the murmur of aortic regurgitation. However, it is important to realise that atypical presentations such as headache occur, and acquired risk factors such as recreational drug use are important aspects of the clinical history, especially in younger patients.

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