

Acute aortic dissection in sepsis with *Staphylococcus* bacteraemia

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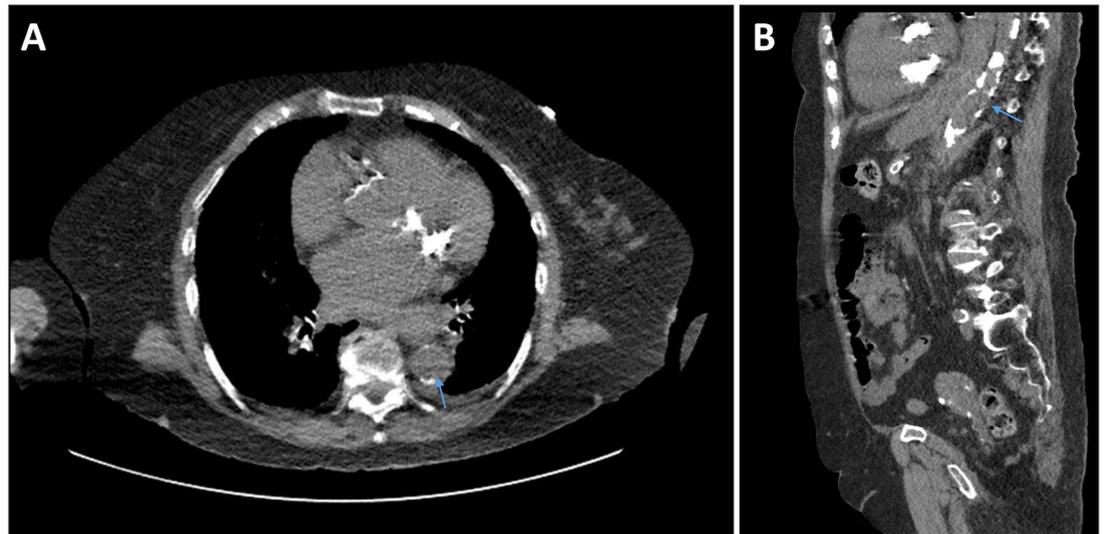


Figure 1 CT abdomen and pelvis performed on admission with portovenous phase contrast (A) axial image at T8 level and (B) sagittal image of a heavily atherosclerotic thoracic aorta but no surrounding collection.

DESCRIPTION

Acute aortic dissection is a clinical emergency. The most common causes are uncontrolled hypertension and atherosclerosis.¹ We present a case of an acute aortic dissection diagnosed following persistent *Staphylococcus aureus* bacteraemia sepsis.

A 98-year-old woman was transferred to hospital after an unwitnessed fall at her aged care facility and was found to be febrile on arrival. Relevant medical history included previous stroke without significant neurologic deficit, hypertension and congestive heart failure. Medications included verapamil slow-release 240 mg two times per day, perindopril 5 mg in morning, indapamide 2.5 mg in morning,

frusemide 20 mg in morning and pravastatin 20 mg at night. She was a non-smoker and ambulated well with a four-wheel frame.

On presentation to the emergency department, she denied chest pain or presyncope. Her only localising infective symptom was a productive cough for 1 week and mild lower abdominal pain without dysuria. On examination, her blood pressure was 130/60 mm Hg and heart rate 80 bpm. She had left basal inspiratory chest crepitations and mild suprapubic tenderness. There was no focal neurology. Initial inflammatory markers included C-reactive protein 324 mg/L and white cell count $12.8 \times 10^9/L$. Her chest X-ray

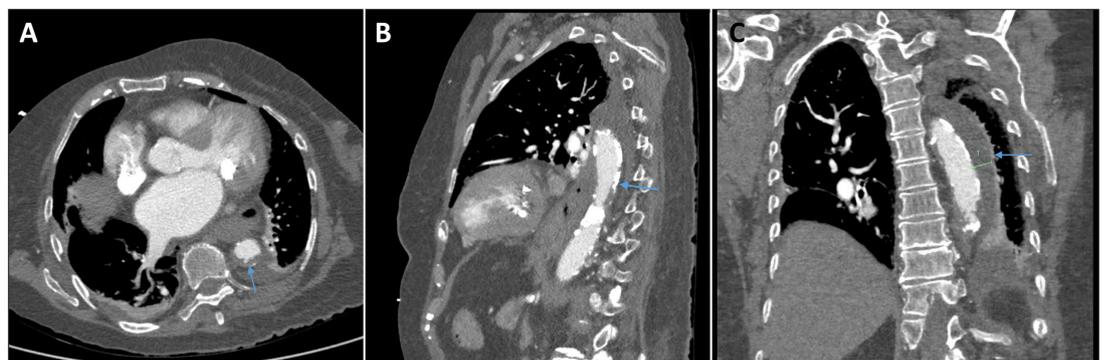


Figure 2 CT chest performed with arterial phase contrast for investigation of back pain on day 4 of admission. (A) Axial slice at the T8 level, (B) sagittal slice and (C) coronal slice demonstrated a large haematoma surrounding the descending thoracic aorta with 15 Hounsfield units. The collection appears contiguous with the posterior aortic wall. No thickening of the aortic wall or fat-stranding to suggest aortitis.



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demonstrated left lower lobe consolidation. CT abdomen and pelvis revealed no identifiable cause for her abdominal pain (figure 1). She was commenced on intravenous antibiotic therapy for community-acquired pneumonia. On day 1 of admission, blood cultures flagged positive for methicillin-sensitive *Staphylococcus* species (MSSA) and antibiotics were rationalised appropriately. Subsequent daily blood cultures continued to grow MSSA for three further consecutive days.

On the third day of admission, the patient complained of new onset thoracolumbar back pain. She underwent a CT chest which revealed a Stanford type-B aortic dissection with a moderate haematoma surrounding the descending thoracic aorta (figure 2). This was not present on CT abdomen/pelvis performed 4 days prior. The thoracic aorta was calcified with an extensive haematoma but no active contrast extravasation to suggest ongoing bleed. In discussion with the vascular surgery team, the patient and her family, goals-of-care were shifted towards a palliative approach.

This case highlights a potential temporal relationship between sepsis and aortic dissection. Sepsis reduces vessel wall shear stress leading to microcirculatory dysfunction, loss of anticoagulation and increased local blood viscosity.² Additionally, sepsis has been shown to increase inflammatory cytokine expression localised to the aortic lumen of mice even during its early presentation (measured at 72 hours and 120 hours) with resultant increased atheroma at 5 months follow-up.³ Existing

atheroma (due to atherosclerosis) through intimal injury can become nidus for infection in sepsis and predisposes to aortitis. Furthermore, bacterial seeding from intimal tear can predispose to full thickness infection and ischaemia from septic emboli in vasa vasorum.⁴ Collectively, it appears plausible that a combination of these mechanisms could explain the endothelial damage with subsequent intimal wall tear observed in our case, exacerbated by the patient's predisposing risk factors including older age, hypertension and hypercholesterolaemia. Although our case is rare and there remains limited treatment options given her age and comorbidities, it is one of the first documented clinical examples of haemodynamic alterations in sepsis preceding an acute vascular injury.

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Learning points

- ▶ Aortic dissection is a potential sequelae of sepsis.
- ▶ Sepsis causes reduced shear stress leading to increased thrombotic, coagulopathic and inflammatory activity that could predispose to vascular intimal wall tear.

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