Escherichia coli and mycotic thoracic aortic aneurysm: a case report

Rajesh Rajendran\textsuperscript{a}, Humayoun Malik\textsuperscript{b} and Tristan Richardson\textsuperscript{c}

\textsuperscript{a}Diabetes Centre, Queen Alexandra Hospital, Cosham, Portsmouth, PO6 3LY, UK; \textsuperscript{b}West Suffolk Hospital, Bury St Edmunds, Suffolk, IP33 2QZ, UK; \textsuperscript{c}Bournemouth Diabetes and Endocrine Centre, Royal Bournemouth Hospital, Bournemouth, BH7 7DW, UK

Corresponding address: Rajesh Rajendran, Diabetes Centre, Queen Alexandra Hospital, Cosham, Portsmouth, PO6 3LY, UK.

Email: rajeshmarthar@yahoo.com

Date accepted for publication 15 May 2012

Abstract

We report a case of an 85-year-old lady with repeated hospital admissions secondary to presumed urosepsis with blood cultures positive for \textit{Escherichia coli}. Chest radiographs during the final admission had changed dramatically and computed tomography scan of the aorta confirmed mycotic thoracic aortic aneurysm.

Keywords

Mycotic; thoracic; aortic; aneurysm; aortitis; \textit{Escherichia coli}.

Case report

An 85-year-old woman was admitted via the emergency department with a 3-day history of intermittent left-sided chest pain and dysphagia of several weeks. She was being treated for a presumed urinary tract infection by her general practitioner with trimethoprim. There was a history of a 10-kg weight loss in the preceding 6 weeks. Her past medical history included hypertension, stage 4 chronic kidney disease, bio-prosthetic aortic valve replacement and a permanent cardiac pacemaker for sinoatrial node disease. She had had three previous hospital admissions in the last 6 months due to presumed but unconfirmed recurrent urinary tract infections.

One month previously, she had presented with confusion, vomiting and abdominal pain. There were no respiratory tract symptoms and her admission chest radiograph (Fig. 1) was unremarkable except for the presence of a pacemaker. She had a raised white cell count of 21,000/l (reference range 4000–110,000/l) and blood cultures isolated \textit{Escherichia coli}. She was treated with intravenous Tazocin (piperacillin with tazobactam) for a presumptive diagnosis of urosepsis for 1 week and trimethoprim for 1 week, but urine cultures were repeatedly negative. In view of her intermittent pyrexia, a computed tomography (CT) scan of the abdomen and pelvis was performed which did not demonstrate any intra-abdominal or pelvic collections. A source for her sepsicaemia was not identified and she was discharged home after she had remained afebrile for 48 h.
On her latest admission, she had a temperature of 37.4°C, blood pressure 124/72 mm Hg, pulse rate 103 beats/min, respiratory rate of 17 breaths/min, and oxygen saturation of 98% on air. Her chest was clear to auscultation and there were no murmurs. Her inflammatory markers were elevated with a white cell count of 15,100/l, and a C-reactive protein of 310 mg/l (<5 mg/l). Her electrocardiogram showed sinus tachycardia and troponin I was negative. Chest radiographs (Fig. 2) revealed a 9-cm mediastinal mass.

CT imaging with three-dimensional reconstruction of the heart and aorta (Figs. 3 and 4) showed a large mycotic aneurysm arising off the arch of the aorta, distal to the left subclavian artery. Blood cultures once again isolated *Escherichia coli*. A diagnosis of mycotic or infectious thoracic aortic aneurysm was made. In view of her co-morbidities, she was considered unfit for surgical or endovascular therapy. She was commenced on intravenous Tazocin. The patient died a day after admission after sudden cardiovascular collapse due to presumed rupture of the mycotic aortic aneurysm. A postmortem was not performed.
Diagnosis
Mycotic/ infectious thoracic aortic aneurysm.

Discussion
A mycotic aneurysm is a rare but serious clinical condition with high morbidity and mortality that develops either when a new aneurysm is produced by infection of the arterial wall or when a pre-existing aneurysm becomes secondarily infected. Despite the name coined by Osler in 1885 to denote the appearance of “fresh fungus-like vegetations”, the majority of mycotic aneurysms are caused by bacteria and rarely by fungus. They may either be true or false aneurysms, involving all layers or only a portion of the arterial wall. Mycotic aneurysms are exceedingly rare and represent
only 2.6% of all aortic aneurysms, with the thoracic aorta being the least common site of occurrence\(^1\). More recently the term infectious aortitis has been commonly used to also include patients who have not yet developed an aneurysm.

It usually affects patients with prior atherosclerotic disease with or without infective endocarditis\(^1\). A number of other predisposing factors have been described such as arterial trauma, local infection, impaired immunity, cancer, diabetes, alcoholism, and old age\(^1\). Therefore, infectious aortitis should be suspected among older patients with atherosclerotic disease and septicemia where the source is not clear.

Gram-positive bacteria such as *Staphylococcal species*, *Enterococcus* species and *Streptococcus pneumoniae* are the most common culprits and are responsible for nearly two-thirds of cases. Gram-negative bacilli (especially *Salmonella* species) are more prevalent in causing infectious abdominal aortitis\(^1\). *Escherichia coli* has been described as a very rare cause for infectious aortitis including thoracic aortitis\(^2\). Aortic infection with *Mycobacterium tuberculosis* has also been described in developing countries.

Clinical presentation is often non-specific making diagnosis difficult and symptoms depend on the site of infection, the cause and aneurysm formation. Therefore, in the early stages, a high index of suspicion is required to make this diagnosis. The most frequent symptoms are fever, thoracic and dorsal pain, abdominal pain and rigors\(^1\). Compressive symptoms such as dysphagia, dyspnoea, hoarseness of voice, cough and superior vena cava compression syndrome can occur with thoracic aortitis\(^1\). Leucocytosis and neutrophilia are present in more than three-quarters of cases\(^1\). C-reactive protein and erythrocyte sedimentation rate are elevated in almost all patients\(^1\). Blood cultures are positive in 50–85% of patients\(^1\).

**CT with contrast enhancement** is the imaging technique of choice\(^{3,4}\). It can also exclude other aortic pathologies such as aortic dissection, intramural hematoma, and penetrating atherosclerotic ulcer\(^1\). Magnetic resonance imaging (MRI) with gadolinium contrast enhancement is emerging as an alternate imaging modality for aortitis as it provides an entire aortic image with high resolution. However, MRI is contraindicated in patients with certain implanted devices such as pacemakers, which are present more frequently in this group of patients. Both MRI and CT scan can precisely define disease extension and can help plan surgical intervention\(^{3,4}\). The use of \(^{18}\text{F}\)fluorodeoxyglucose-positron emission tomography either alone or in combination with contrast-enhanced CT or MR angiography, has been described as being useful in giant cell aortitis and Takayasu arteritis\(^3\), and may be useful in infectious aortitis as well.

There are no randomized controlled studies to guide the management of infectious aortitis and there is a paucity of literature regarding the optimal management strategy. The broad consensus includes the importance of early detection, with a combination strategy of prolonged courses of intravenous broad-spectrum antibiotics and surgical excision of the infected aorta. The most up-to-date surgical method of revascularization involves resection of the infected segment with in situ prosthetic graft\(^5\). Recently, endovascular techniques to treat infectious aortitis have shown promising results but further studies are needed with longer follow-up periods to define their role in the management of infectious aortitis\(^5\).

Possible complications of infectious aortitis include rupture, bleeding, thrombosis, septic embolization, and aortic insufficiency\(^4,5\). A combination of surgical and medical therapy can lead to a survival rates of 75–100% before aneurysm formation\(^1\), but only 62% after aneurysm formation. With medical treatment alone, the mortality rate is \(\sim\)90%.

Our patient presented with recurrent episodes of sepsis secondary to *Escherichia coli*. She had several risk factors for infectious aortitis such as atherosclerosis, bio-prosthetic aortic valve replacement, old age and impaired immunity secondary to chronic kidney disease. We postulate that she may have had either an original urinary tract infection (but negative urine cultures which could be attributable to pretreatment in the community with antibiotics) leading to septicemia that predisposed her to the development of infectious aortitis. Alternatively, the recurrent septicemia may have been due to infectious aortitis itself in association with an underlying subacute bacterial endocarditis of her bio-prosthetic aortic valve. The presence of pacing wires from the pacemaker could have also possibly acted as a source of persistent infection. Eventually the *Escherichia coli* infected aortitis led to presumed fatal thoracic aneurysm formation.

---

**Teaching points**

1. Infectious aortitis should be suspected among older patients with atherosclerotic disease and septicemia of unclear origin.
2. Early detection of mycotic aneurysm is crucial because of the high risk of rupture if treatment is delayed.
3. Septicaemia due to *Escherichia coli* does not always reflect a urinary source and alternative sources should be sought if urine cultures are negative.

**References**