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Case Report

CASE REPORT

Aortitis causing rapid growth of a mycotic aortic aneurysm

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Abstract

Mycotic infrarenal aortic aneurysms are rare and often masquerade as other abdominal pathology. We present a case where serial imaging made the diagnosis and provided an insight into the pathophysiology of mycotic aneurysm. A 71-year-old man presents with abdominal pain, rigours and dysuria. Computed tomography reveals an irregular, thickened ectatic abdominal aorta, but cholescintigraphy suggests acalculous cholecystitis. Deterioration prompts repeat radiographical assessment, which demonstrates an increase in the size of the aorta over 10 days. The patient was treated emergently with an open aortic ligation, debridement and extra-anatomical bypass. Infections account for up to 2% of abdominal aortic aneurysms. The rate of growth of mycotic aneurysms is sparsely discussed in the literature and to our knowledge, there are no reports with serial single-modality imaging. The most significant finding was rapid expansion in aneurysm size. While mycotic aneurysm requires urgent treatment, diagnosis can be delayed and difficult.

INTRODUCTION

Mycotic aortic aneurysms are rare and often masquerade as other acute abdominal pathology, delaying diagnosis. We present a case where serial imaging made the diagnosis of mycotic aneurysm. To our knowledge, this case is the first in the literature to describe serial scanning with the same imaging modality.

CASE REPORT

A 71-year-old man presented to the emergency department after a 2-week history of cramping peri-umbilical pain and rigours. Pre-admission computed tomography (CT) demonstrated irregular thickening of the infrarenal aorta, which measured 3.3 cm in its maximal diameter. His background was significant for ischaemic heart disease, hypertension, gout, hypercholesterolaemia and an admission for small bowel obstruction which was treated conservatively. There was no history of diabetes, intravenous drug use, or recent medical or dental procedure. He was an ex-smoker.

On examination, his blood pressure was 104/64 mmHg and he was afebrile. The abdomen was centrally tender, without palpable or pulsatile mass, and all lower limb pulses were normal. Investigations revealed new atrial fibrillation, a pH of 7.44, mildly derranged liver enzymes (aspartate transaminase 51 U/l, alanine transaminase 58 U/l, alkaline phosphatase 136 U/l, gamma-glutamyl transpeptidase 271 U/l and bilirubin 14 µmol/l), haemoglobin of 131×10^{12}/l and white cell count of 11.6×10^{9}/l with neutrophilic change (8.1×10^{9}/l). Platelet count was 471×10^{9}/l, erythrocyte sedimentation rate (ESR) 72 mm/h and C-reactive protein 49.8 mg/l. Troponins and lipase were normal. Blood and urine cultures yielded no growth. Transthoracic echocardiography and labelled white cell studies were normal. A single dose of prednisolone was administered in the emergency department based on the ESR. Hepatobiliary iminidiacetic acid scan with cholecystokinin and abdominal ultrasound were suggestive of chronic acalculous cholecystitis.

As such, intravenous ampicillin and gentamicin was commenced for acalculous cholecystits. The patient had ongoing

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pain and developed intermittent pyrexia up to 38.4°C. Blood cultures remained negative and the white cell count reached 13.6×10^9/l on the fourth admission day. A repeat CT scan demonstrated a more inflamed and aneurysmal aorta involving the origin of the left renal artery with intraluminal thrombus ulceration. The aneurysm measured 5 cm transversely, demonstrating 1.7 cm growth in 10 days.

The patient underwent a left axillo-uni-femoral bypass with a rifampicin-soaked Dacron graft and debridement of mycotic aneurysm the following day. The aorta was ligated infrarenally and above the iliac bifurcation. The proximal stump was covered with an omental patch. Peri- and postoperative vancomycin and meropenem were provided as advised by the infectious diseases team. From the debrided tissues, polymerase chain reaction showed a 100% match for *Streptococcus* and no syphilis was detected.

Postoperatively, recovery included a 13-day intensive care admission with renal impairment, gastroparesis and pneumonia. Blood cultures remained negative and repeat CT revealed a patent graft with no collection or phlegmon. The patient was discharged on the 49th postoperative day on warfarin, intravenous meropenem and vancomycin for 6 weeks. Follow-up duplex at 2 years described a widely patent graft. There were no new surgical issues other than a reducible incisional hernia.

DISCUSSION

Infections account for up to 2% of abdominal aortic aneurysms (AAAs) [1]. Infected aneurysms may occur in any artery and often have a delayed diagnosis. There are four types, in order of increasing mortality, including post-traumatic infected false aneurysms, mycotic aneurysms, microbial arteritis and infection of an existing aneurysm. The latter carries a mortality rate of 90% [2]. *Salmonella* spp. and *Staphylococcus* spp. account for 50% of infected aortoiliac aneurysms [3]. Patients may be culture negative in up to 20% of cases [3].

This patient presented with abdominal pain and mild leucocytosis, present in 92 and 69% of infected aortoiliac aneurysms, respectively, and later pyrexia. Other features may include positive blood cultures and palpable abdominal mass [3]. The absence of clinical features and the presence of distracting signs made for a challenging diagnosis in this case.

Neither the rate of growth, nor a change of morphology (new thrombus ulceration) of mycotic aneurysms, has been discussed in the literature; the consensus is that all infected aneurysms should be treated as surgical emergencies, regardless of size and shape [4]. Rapid expansion of mycotic aneurysm has been described; however, there are no published cases with serial scanning using a single imaging modality available for reference. The expansion of a mycotic thoracic aortic aneurysm over 3 weeks has been described [5]. Another group describes a sudden 4-cm growth of a long-standing AAA over 10 days in a patient with community-acquired pneumonia [6].

While ultrasound accurately determines the size and character of an AAA, it cannot reliably comment on infection as an aetiology. CT angiography is effective in determining the size and character of aortoiliac aneurysms. Soft tissue inflammation, para-aortic mass, eccentrically shaped aneurysms and narrow aneurysm neck may suggest an infective cause [3]. When rapid expansion on serial CT is observed, a mycotic aneurysm has to be considered.

Radio-labelled white cell scans may demonstrate para-aortic inflammation, but have also been negative in cases of proven abdominal aortitis [7].

Treatment options for mycotic infrarenal AAAs include extra-anatomic bypass with ligation, resection and debridement or *in situ* graft placement following extensive debridement. The risks include reinfection and a mortality rate of up to 42% [3]. Extra-anatomic bypass carries an 8–19% risk of aortic stump rupture [3]. Implanted antibiotic releasing carriers have been used in conjunction with open surgery in the past [8]. Endovascular repair for mycotic AAAs has been reported again with a mortality rate of 42% [9]. Long-term antibiotics are always indicated; however, their use and duration postoperatively has not been extensively studied [10]. Often, 6 weeks of intravenous followed by 6 weeks of oral antibiotics with appropriate sensitivities is advised.

In summary, we present a case of a 71-year-old gentleman with a peculiar clinical picture of a mycotic aortic aneurysm masquerading as acute acalculous cholecystitis. The most significant clue in his work-up was rapid change in the character of the aneurysm. Despite extensive investigation and seeking expert opinion, mycotic aneurysm can be missed.

CONFLICT OF INTEREST STATEMENT

None declared.

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