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An infected enlarging abdominal aortic aneurysm after acute cholecystitis☆

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ABSTRACT

INTRODUCTION: An abdominal aortic aneurysm (AAA) infection is rare and can be difficult to manage, with high morbidity and mortality. We present a patient who suffered an infected AAA after undergoing a laparoscopic cholecystectomy and discuss the surgical management options.
PRESENTATION OF CASE: A 69-year-old male presents with a rapidly enlarging AAA 4 weeks following laparoscopic cholecystectomy. He was managed with open debridement, washout and repair of the aneurysm, but suffered ongoing sequelae of *Escherichia coli* sepsis.
DISCUSSION: The options for surgical management of infected AAA include open, endovascular and combined approaches. Recent papers report successful use of endovascular repair of infected AAAs but this is an ongoing area of research.
CONCLUSION: Infection of an AAA is associated with high mortality and long-term morbidity and requires optimal treatment. Surgical options include open debridement and repair, endovascular aneurysm repair (EVAR) or a combined approach.

1. Introduction

Primary infection of an abdominal aortic aneurysm (AAA) is rare but can be difficult to manage. The cause is generally unknown, although in some patients, a preceding infective illness is identified. It is associated with high morbidity and mortality, and early recognition with appropriate vascular reconstruction can optimise patient outcome.[1]

2. Case history

A 69-year-old male presents to a tertiary referral hospital in the UK with a 1-week history of right upper quadrant pain and fever, on a background of known cholelithiasis. Past medical history included hypertension and lumbar disc degeneration and he was in the local surveillance programme for an AAA measuring 5.1 cm in maximal diameter. He was an ex-smoker and was independent for activities of daily living. On admission, the biochemical investigations revealed a white cell count (WCC) of $11 \times 10^9\,L^{-1}$, C-reactive protein (CRP) of 188 mg/L and normal liver function tests (LFTs), with ultrasound (US) of the abdomen showing features of acute cholecystitis.

The patient proceeded to an urgent laparoscopic cholecystectomy and intra-operative findings included severe inflammatory change with dense adhesions of adjacent omentum and colon to the gallbladder, and a large paracolic abscess. The gallbladder was eventually separated from surrounding colon and omentum and Calot's Triangle was dissected out and identified. An intra-operative cholangiogram showed normal biliary duct anatomy. Haemostasis of the gallbladder bed was achieved with diathermy and a subhepatic drain was inserted. Post-operative recovery was uneventful and the patient was discharged 2 days later with a week's course of oral antibiotics.

The patient represented 4 weeks post discharge with abdominal pain and distension associated with constipation (bowels not open for 10 days), nausea and vomiting and rigours. On presentation, his temperature was 39 °C with a WCC of $28.1 \times 10^9\,L^{-1}$ and CRP >320 mg/L with normal LFTs. An abdominal ultrasound showed a hepatic abscess measuring $6 \times 5 \times 5.5$ cm. Computed tomography (CT) guided drainage of the hepatic abscess was performed and incidentally, it was noted that the previously known AAA had increased in size to 6.5 cm with adjacent stranding and enhancement, suggesting an imminent rupture.

An urgent laparotomy was performed, which showed an oedematous aorta with gross inflammation of surrounding structures

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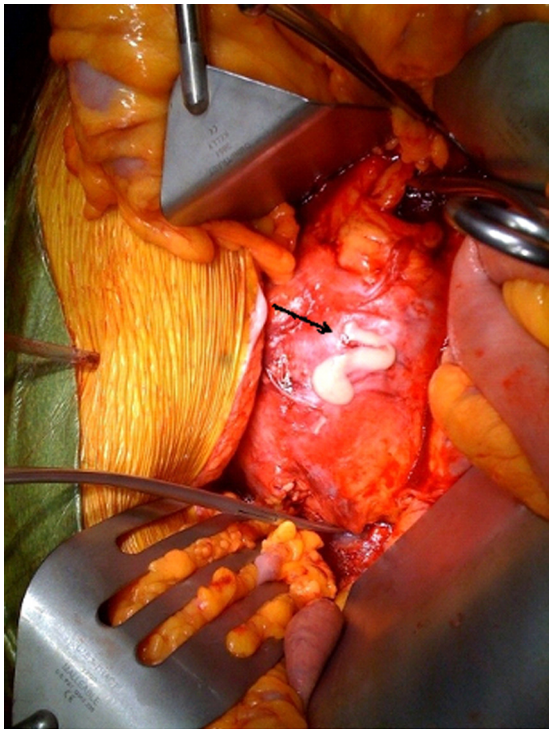

Fig. 1. Intra-operative picture demonstrating free pus (arrow) from the abdominal aortic aneurysm.

(Fig. 1). On opening the aorta, free pus was drained and microbiological cultures subsequently grew *Escherichia coli*. The infected tissue was debrided and the aortic neck was securely ligated with oversewing of the iliac orifices. A drain was left in the AAA sac and an axillo-bifemoral bypass graft soaked in betadine and rifampicin was performed. An intra-operative ultrasound of the liver was unremarkable at this stage.

Post-operatively, the patient continued to spike high fevers with nights sweats, associated with left hip and back pain. A repeat CT found an ongoing liver abscess, which was drained percutaneously (Fig. 2). This also grew *E. coli*. The patient received long term antibiotics in view of his aortic infection and metastatic sepsis and he was discharged to a rehabilitation unit after 5 weeks.

Seven months after initial presentation with acute cholecystitis, the patient complained of ongoing back pain. A MRI study located a discitis at the L3/L4 level and a CT guided biopsy grew *E. coli*, *Enterococcus* and anaerobes. The patient was treated with piperacillin, tazobactam and vancomycin. During this period, the patient unfortunately developed a deep vein thrombosis and was anti-coagulated.

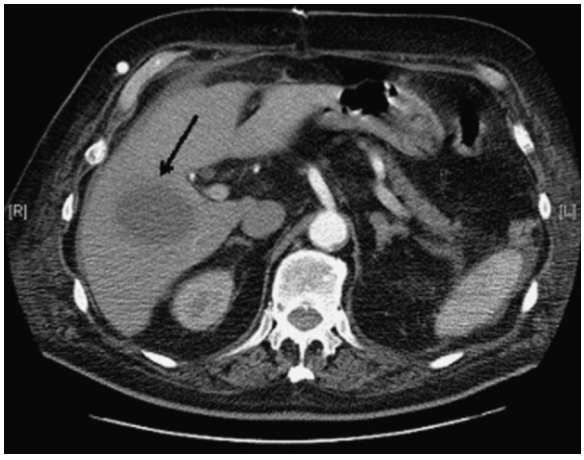

Fig. 2. Computed tomography demonstrating hepatic abscess (arrow).

The patient recovered from these episodes and was discharged home. At 15 months follow up, he is well with no symptoms or further sequelae of his illness.

3. Discussion

Primary infected AAA is relatively rare[2] but is associated with a poor prognosis with a mortality rate of 21–44% in published case series.[3] A preceding septic event is not always identifiable and the most common organisms isolated are *Salmonella*, *Streptococcus* and *Staphylococcus*.[2,4,5] In this patient, *E. coli* sepsis from acute cholecystitis with metastatic spread to the AAA, liver and spine was the most likely source of the prolonged illness.

Although *E. coli* sepsis originating from infections of the urinary or biliary tract is well-recognised, metastatic spread to vascular structures has rarely been reported in the literature with recent case reports commenting on such a phenomenon occurring in the ascending aorta, the subclavian and the internal carotid arteries.[6,7]

There are several surgical options for the treatment of the primarily infected AAA. The traditional open surgical approach involves complete debridement of the infected AAA and oversewing the aortic stump followed by an extra-anatomical bypass (such as the axillo-femoral bypass used in this case). A more aggressive approach would be to perform an in situ aortic bypass with a synthetic graft wrapped in omentum.[8] Both procedures require ongoing antibiotic cover post-operatively (which vary from 2 to 6 weeks) and the immediate to short-term outcome appears to be good with Maeda et al. and Ting et al. reporting no in-hospital deaths.[2,4] However, Fillmore and Valentine reported 4/10 in-hospital deaths and discuss that all 4 patients had suprarenal aneurysms and confirmed that they all had developed systemic inflammatory response syndrome (SIRS) pre-operatively.[3]

Recently, there have been reports of endovascular aneurysm repair (EVAR) of primarily infected AAA.[1,9] A case series by Kan et al. on 12 consecutive patients evaluated the surgical protocol of pre-operative culture before empirical antibiotics and either urgent or delayed (1–14 days of pre-treatment) EVAR, depending on patients' haemodynamic status. All patients were assessed at 1 week after EVAR with CT angiography and if there were ongoing signs of infection, CT-guided drainage or surgical debridement was performed. Bacteria specific intravenous antibiotics were given for at least 4 weeks post-operatively. This protocol caused no in-hospital mortality with three patients requiring CT-guided drainage and one patient requiring open debridement. Interestingly, there was no evidence of persistent or graft infection at mean follow-up of 24 months.

However EVAR for primarily infected AAA has the inherent risk of secondary infection of the graft, especially in the absence of adequate debridement of infected tissue.[9] Clough et al.[10] suggest that the optimal time for EVAR may be when the patient is afebrile. In the context of multiple septic foci as occurred in this patient, it is unclear as to whether EVAR would have been advisable, particularly with knowledge of preceding acute cholecystitis making it less likely that the causative organism would be *Salmonella*, which is the most common organism cultured in prior case series of primary infection of AAA.[9]

In summary, we present a patient with a primary infection of a pre-existing AAA, managed with intravenous antibiotics, complete surgical debridement and axillo-femoral bypass, which was complicated by multiple sequelae of *E. coli* sepsis.

Conflict of interest

None.

Funding

None.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contributions

Dr Sang Hwang has contributed in manuscript preparation, literature review and the write-up.

Dr James Clarke is the primary surgeon responsible for the patient, study design.

Dr Tjun Tang is the assisting surgeon for the patient, study design, data collection.

Key learning points

- Primary infection of AAA is associated with high mortality and long-term morbidity.
- Both open surgery and EVAR are potential options for infected AAA.
- Think of metastatic spread to other parts of the body.

References

1. Gibbons CP. What is the best treatment for primary infected aortic aneurysms? *European Journal of Vascular and Endovascular Surgery: The Official Journal of the European Society for Vascular Surgery* 2011;**42**:625–6.
2. Maeda H, Umezawa H, Goshima M, Hattori T, Nakamura T, Umeda T, et al. Primary infected abdominal aortic aneurysm: surgical procedures, early mortality rates, and a survey of the prevalence of infectious organisms over a 30-year period. *Surgery Today* 2011;**41**:346–51.
3. Fillmore AJ, Valentine RJ. Surgical mortality in patients with infected aortic aneurysms. *Journal of the American College of Surgeons* 2003;**196**:435–41.
4. Ting AC, Cheng SW, Ho P, Poon JT, Tsu JH. Surgical treatment of infected aneurysms and pseudoaneurysms of the thoracic and abdominal aorta. *American Journal of Surgery* 2005;**189**:150–4.
5. Hsu RB, Chang CI, Chan CY, Wu IH. Infected aneurysms of the suprarenal abdominal aorta. *Journal of Vascular Surgery* 2011;**54**:972–8.
6. McCann JF, Fareed A, Reddy S, Cheesbrough J, Woodford N, Lau S. Multi-resistant *Escherichia coli* and mycotic aneurysm: two case reports. *Journal of Medical Case Reports* 2009;**3**:6453.
7. Yano M, Hayase T, Furukawa K, Nakamura K. Mycotic pseudoaneurysm of the ascending aorta caused by *Escherichia coli*. *Interactive Cardiovascular and Thoracic Surgery* 2013;**16**:81–3.
8. Kwon TW, Kim HK, Moon KM, Cho YP, Park SJ. In situ polytetrafluoroethylene graft bypass for primary infected aneurysm of the infrarenal abdominal aorta. *World Journal of Surgery* 2010;**34**:1689–95.
9. Kan CD, Yen HT, Kan CB, Yang YJ. The feasibility of endovascular aortic repair strategy in treating infected aortic aneurysms. *Journal of Vascular Surgery* 2012;**55**:55–60.
10. Clough RE, Black SA, Lyons OT, Zayed HA, Bell RE, Carrell T, et al. Is endovascular repair of mycotic aortic aneurysms a durable treatment option? *European Journal of Vascular and Endovascular Surgery: The Official Journal of the European Society for Vascular Surgery* 2009;**37**:407–12.