ISSN: 2634 - 8861

Journal of Infectious Diseases & Case Reports

SCIENTIFIC Research and Community

Case Report

Open Access

Infected Abdominal Aortic Aneurysm

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Received: September 26, 2024; **Accepted:** October 02, 2024; **Published:** October 09, 2024

Introduction

Infected aortic aneurysms (AA) are rare with a prevalence of 0.7% - 3% of all AA but are associated with a significant mortality rate, which remains high at 22% - 36% despite advances in perioperative management and antimicrobial therapy [1]. Localized dilatation of the aorta occurs due to inflammation and destruction of the vessel wall by a bacterial or mycotic infection. The infection of the aortic wall can directly cause the development of an aneurysm, or a pre-existing AA becomes secondarily infected [2]. Risk factors for the development of infected AA are infective endocarditis, atherosclerosis, immunosuppression and age [3-5]. The most important differential diagnosis is inflammatory AA due to giant cell or Takayasu arteritis.

Pathogen inoculation into the vessel wall follows bacteremia, septic embolization (endocarditis), direct inoculation (postoperative) or by continuitatem spread of an adjacent infectious focus (especially abdominal infections). Staphylococcus aureus, S. epidermidis and Salmonella spp. have a higher affinity for the arterial wall and are therefore more likely to develop an infected AA. Coxiella burnetii, Streptococcus pneumoniae, Treponema pallidum, Mycobacterium tuberculosis and fungi (Candida spp. and Aspergillus spp.) are less common [6].

The classic presentation of infected abdominal AA combines a painful, pulsatile and size-progressive abdominal mass and systemic signs of inflammation such as fever and malaise. Peripheral embolization with limb ischemia is possible, but also per continuitatem spread to the spine with spondylodiscitis, osteomyelitis and epidural or prevertebral abscessation. Less common is gastrointestinal bleeding due to erosion of the intestinal wall with formation of an aorto-enteric fistula [7].

In addition to the clinical examination, the diagnosis includes taking blood cultures, which are positive in 50% - 85% of cases. It should be borne in mind that T. pallidum, C. burnetii and M. tuberculosis do not grow in normal blood cultures and require either serology or citrate blood cultures. If the patient undergoes surgery, tissue biopsies should also be examined microbiologically and histologically (culture, specific or eubacterial polymerase chain reaction (PCR) for non-culturable pathogens or in the case of antibiotic pre-treatment). Angio-computed tomography (CT) or magnetic resonance imaging (MRI) or FDG-PET/CT are useful for imaging [8].

Treatment of an infected AA involves a combination of surgery and antibiotic therapy, although no randomized studies exist [8]. Empirical antibiotic therapy should treat the most common pathogens (S. aureus, Salmonella spp.) and can, for example, consist of flucloxacillin i.v. plus ceftriaxone i.v. or piperacillin/tazobactam i.v. The combination with vancomycin i.v. is particularly indicated in cases of suspected methicillin-resistant S. aureus (MRSA). De-escalation should take place as soon as the pathogen has been identified. The duration of therapy is at least six weeks, but depends not least on the pathogen, the surgical therapy (e.g. implantation of foreign material) and complications (septic spread). Surgically, the focus is on open excision of the AA with debridement of the infected surrounding tissue and replacement with preferably biological material (e.g. pericardial prosthesis), but the creation of extra-anatomical bypasses can also be useful. Endovascular stenting should be discussed in patients with a prohibitively high surgical risk, even if this treatment is associated with a significantly higher recurrence rate, morbidity and mortality in the long term [9].

Case Report

Case History

The 81-year-old patient presented to the emergency department of a peripheral swiss hospital due to acutely aggravated back pain, malaise and an elevated temperature of almost 38°C, which had been present for four days. The patient had returned from Thailand two weeks previously. During the trip the patient was healthy, infectious diseases - especially diarrhea - were denied. Abdominal CT newly diagnosed abdominal AA. The personal history included a large ascending aortic aneurysm (46 mm) and a bilateral inguinal hernia operation.

Status

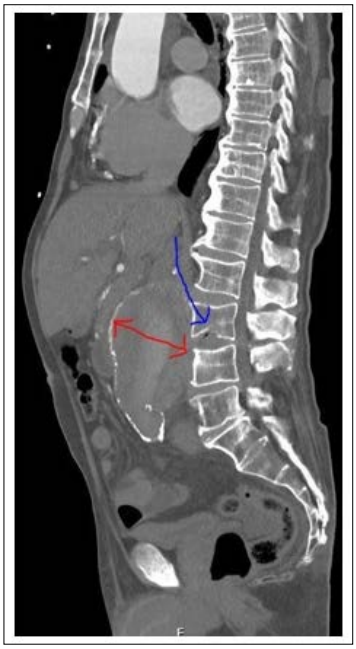
The awake and adequate patient was afebrile (35.9°C), centralized, barely normotensive (blood pressure 90/60 mmHg), tachycardic

(120/min), with a peripheral oxygen saturation of 94% under 6l O_2 by nasal cannula. There was a visual and palpatory impression of an abdominal pulsatile mass.

The foot pulses were symmetrically weakly palpable. The integument and joints were unremarkable, the spine was palpable.

Findings and Diagnosis

The laboratory tests revealed elevated inflammatory values (C-reactive protein (CRP) 163 mg/l, leukocytes 19 G/l), bicytopenia (hemoglobin 108 g/l, platelets 104 G/l) and acute renal insufficiency (creatinine 240 µmol/l) with accompanying metabolic and lactic acidosis in the venous blood gas analysis (pH 7. 30, pCO2 35 mmHg, HCO_3 17 mmol/l, BE -17, anion gap 16 mmol/l, lactate 5.4 mmol/l). The thoracoabdominal angio-CT revealed a two-bellied abdominal AA with a small juxtarenal AA on the left (up to 3.8 cm) and a large infrarenal AA (up to 8.1 cm) with circular wall thrombosis and an inflammatory surrounding reaction. In addition, there was a floor plate compression fracture of the lumbar vertebral body (L3) with gas in the intervertebral space L 3/4 (Figure). The known thoracic AA was unchanged. In summary of the findings, an infected infrarenal AA with dorsally covered rupture and per continuitatem spondylodiscitis L 3/4 was diagnosed.



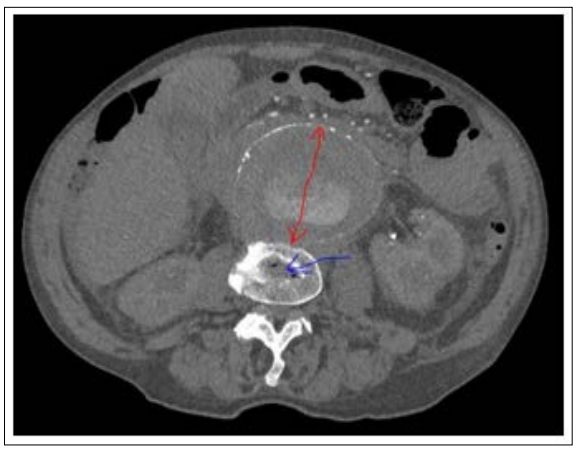


Figure 1,2: CT Sagittal and Axial

Therapy

Progressive somnolence, hypotension and hypoxaemia were followed by intubation, transfer to the intensive care unit and stabilization with crystalloids and vasoactive agents. After blood cultures were taken, empirical antibiotic therapy with piperacillin/tazobactam i.v., gentamicin i.v. and vancomycin i.v. was initiated (ratio: gram-negative resistance problems and suspected MRSA in a traveler returning to the hospital). The blood cultures showed a rapid positive result with detection of fully sensitive Salmonella enteritidis (Table 1), so that a de-escalation to high-dose amoxicillin i.v. took place. In a multidisciplinary meeting with infectiology, vascular surgery, spinal orthopaedics and intensive care medicine, an emergency abdominal aortic replacement using a pericardial prosthesis and debridement of the vertebral osteomyelitis and spondylodiscitis L 3/4 were decided in this septic patient with impending rupture of the huge abdominal AA.

Table 1: Antibiogram of Salmonella Enteritidis Antibiotic Sensitivity

Antibiotic	Sensitivity
Amoxicillin-Clavulanic acid	Sensitive
Ampicillin	Sensitive
Cefepime	Sensitive
Ceftazidime	Sensitive
Ceftriaxone	Sensitive
Ciprofloxacin	Sensitive
Ertapenem	Sensitive
Gentamicin	Sensitive
Meropenem	Sensitive
Piperacillin/Tazobactam	Sensitive
Trimethoprim-Sulfame	Sensitive

Intraoperatively, it was confirmed that the infrarenal AA had broken through dorsally with a covered rupture and that the lumbar spine had eroded. S. enteritidis was cultivated in the biopsies of the aorta and intervertebral disc. With prolonged bacteremia on the day of admission, no infective endocarditis was detected in a transesophageal echocardiography (TEE).

Apart from a functional single kidney with postoperative lack of flow detection of the left renal artery with dialysis requirement, the patient recovered very well, so that on day 11 the lumbar spine was repaired by debridement, abscess relief, corpectomy L 3 and dorsal fusion with spondylodesis L 2-5.

The intraoperative biopsies were still positive. On day 18, due to persistent inflammation and persistent periaortic and prevertebral abscessation on abdominal CT, a revision operation was performed with further debridement and vertebral body replacement with ventral interbody fusion L 2-4.

The patient remained on dialysis and low-dose catecholamines. Unfortunately, a small bowel ileus occurred on day 22 with small bowel volvulus with bowel ischemia and consecutive pneumatosis intestinalis and aeroportia. At this point, the patient did not want any further therapeutic measures with limited treatment options overall. He died two days later.

Discussion

Infected AA usually develop as a result of bacteremia, whereby bacteria adhere to pre-existing aneurysms or atherosclerotic

plaques, penetrate into deeper layers of the vessel wall and thereby infect the arterial wall. As the aorta is most frequently affected by atherosclerosis, the infected AA is the most common site of manifestation [10]. Infected aneurysms occur much less frequently in a healthy vessel, because the intima is very resistant to infection. Infection of the vessel wall can lead to localized perforations, pseudoaneurysms and abscesses. Abdominal AA was not previously known in our patient. However, due to the patient's age, we assume that infrarenal aortic plaques were pre- existing, to which S. enteritidis could adhere and lead to de novo formation of the AA. There was no evidence of a primary intra-abdominal focus of infection with per continuitatem infection of the aorta, and infective endocarditis could be excluded by TEE.

Along with staphylococci, Salmonella spp. are among the most common pathogens that cause an infected AA. The travel history of our patient made the case interesting. On the one hand, S. enteritidis and S. typhi could in principle be the cause, whereby both Salmonella spp. are ingested orally via contaminated food, but in Western Europe it is mainly S. enteritidis that occurs. On the other hand, the possible resistance problem of Salmonella acquired in Thailand had to be taken into account in empirical therapy.

Salmonella enteritidis causes the non-typhoidal salmonella infection. It is classically associated in 70% - 80% with acute gastroenteritis with diarrhea, which was absent in our patient. Enteritis can lead to intestinal translocation and Salmonella bacteremia with septic disseminated foci. Patients aged < 12 months or > 50 years, but also immunocompromised patients and patients with atherosclerosis, prosthetic heart valves or joints have an increased risk of a complicated Salmonella infection. The most common extraintestinal manifestation is endovascular infection. In patients at risk, antibiotic therapy should be considered for symptomatic S. enteritidis infection due to the increased complication rate, which is otherwise not recommended for uncomplicated courses as it promotes long-term excretion [11]. In Switzerland, there is a laboratory reporting requirement for S. enteriditis and a clinical and laboratory reporting requirement for S. typhi/paratyphi.

Empirical antibiotic therapy for Salmonella infection includes a third-generation cephalosporin, whereby risk factors for resistance problems must be taken into account. Fluoroquinolones should not be used empirically in critically ill patients due to the high resistance rate (28% in 2023 according to ANRESIS (Swiss Center for Antibiotic Resistance) [12]. In our case, once the antibiogram was available, it was possible to carry out resistance-appropriate treatment with i.v. amoxicillin. Across Switzerland, resistance to aminopenicillins was 11% in 2023 [12]. The resistance situation is very different in Thailand, where the majority of isolates are resistant to ampicillin (72.4%) and up to 52% are multiresistant [13]. With an inconspicuous environmental history, it ultimately remains speculative whether our patient became infected in Thailand or in Switzerland, although the lack of evidence of resistance makes an infection in Thailand less likely.

The duration of treatment for infected AA is not clearly defined, but should be at least six weeks in the case of a complicated endovascular infection [14]. The duration of treatment may be extended in the case of septic disseminated foci or embedded implants. Prolonged treatment was planned for our patient, as the implantation of the spondylodesis in an infected area resulted in an acute spondylodesis-associated infection. Ciprofloxacin exhibits biofilm activity in Gram-negative pathogens, so that a curative treatment concept with twelve weeks of treatment with ciprofloxacin was planned [15]. As a biological material, the pericardial prosthesis does not require prolonged therapy, in contrast to other vascular prostheses. In certain situations, suppressive antibiotic therapy may be indicated, especially if chronically infected implants cannot be removed or if there is resistance to ciprofloxacin and therefore no biofilm-active antibiotic therapy.

Conclusion

We present the clinical picture of infected abdominal AA due to *Salmonella enteritidis*, which occurred after a stay in Thailand. The patient presented classically with an abdominal pulsatile mass and back pain due to a covered dorsal rupture with arrosion of the lumbar spine and a per continuitatem abscessing spinal infection. Despite maximal treatment, which included targeted antibiotic therapy with i.v. amoxicillin, resection of the infected aneurysm with replacement by a pericardial prosthesis and spinal revision, the patient ultimately died of a small bowel ileus.

Take-Home Message

- Infected AA is a rare but serious infection with a mortality rate of between 22% and 36%.
- Pathogenetically, hematogenous infection of the aortic wall dominates in the context of bacteremia, whereby atherosclerotic plaques or pre-existing AA are usually present as predisposing factors.
- Bacteria with the highest affinity for the arterial wall and therefore the greatest probability of causing an infected AA are S. aureus, S. epidermidis and Salmonella spp.
- Classically, infected abdominal AA presents with a painful, pulsatile and size-progressive abdominal mass with systemic signs of inflammation such as fever and malaise.
- Diagnostically, blood cultures (positive in 50% - 86%), imaging (angio-CT or MRI) and intraoperative microbiological diagnostics (tissue biopsies of the aneurysm wall) are decisive.
- Therapeutically, a combination of surgical and antibiotic therapy is in the foreground.
- Empirically, the most common pathogens should be considered and treated, for example, with flucloxacillin i.v. and ceftriaxone i.v. or piperacillin/tazobactam i.v.. The combination with vancomycin i.v. is indicated in cases of high MRSA prevalence.
- Open surgical excision of the AA with debridement of the infected surrounding tissue and replacement with preferential biological material is the treatment of choice.

Acknowledgement(s)

We would like to thank our colleagues in the radiology and microbiology departments of the Cantonal Hospital Aarau for their radiological and microbiological support.

Ethics Statement

Informed consent was obtained from the patient involved.

Conflict of Interest Statement

The authors have declared that they have no potential conflicts of interest

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