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Case Report

Fatal Salmonella aortic infection presenting as Stanford type A aortic dissection

Ceren Sayarer, Senanur Duymaz, Gorkem Gokkaya, Sevket Baran Ugurlu

Dokuz Eylül University, Faculty of Medicine, Department of Cardiovascular Surgery, İzmir, Türkiye

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Abstract

Infectious aortitis is a rare but highly lethal condition characterized by rapid destruction of the aortic wall despite, timely surgical and medical treatment. Salmonella species are among the most aggressive causative pathogens. We report a 58-year-old female patient with inflammatory spondyloarthritis who exhibited prolonged fever and systemic inflammatory response, later diagnosed with a Stanford type A aortic dissection. Intraoperative findings revealed a chronic dissection with an adherent and infected-appearing pericardium. Ascending aortic replacement was performed successfully; however, the postoperative course was complicated by recurrent massive mediastinal bleeding requiring repeated surgical re-explorations. While the prosthetic graft remained intact, progressive destruction of the native aortic wall was observed, ultimately necessitating patch repair. Despite prolonged broad-spectrum antimicrobial therapy, systemic inflammation persisted, and the patient died suddenly, most likely due to acute rupture of another infected aortic segment with cardiac tamponade. This case highlights the aggressive and unpredictable nature of Salmonella aortitis and emphasizes that surgical intervention may not prevent fatal outcomes.

Keywords: Aortitis; Salmonella infections; aneurysm, infected; aortic dissection; aortic aneurysm, thoracic

INTRODUCTION

Infectious aortitis is a rare but life-threatening condition characterized by microbial invasion of the aortic wall, leading to rapid structural destruction, aneurysm formation, or dissection, with high reported mortality rates despite advances in surgical and medical therapy [1]. Among the causative pathogens, *Salmonella* species demonstrate a particular tropism for diseased or atherosclerotic aortic tissue and are associated with an especially aggressive clinical course [1,2].

Although *Salmonella* aortitis most commonly involves the abdominal aorta, thoracic and ascending aortic involvement has been reported and is frequently complicated by dissection or rupture [2,3]. Clinical presentation is often nonspecific, including prolonged fever, elevated inflammatory markers, and systemic symptoms, which may delay diagnosis and definitive treatment [1,4].

Urgent surgical repair combined with prolonged antimicrobial therapy is considered the cornerstone of management; however, postoperative outcomes remain poor due to persistent infection and progressive destruction of residual native aortic tissue [3,5]. Even after technically successful aortic replacement, late complications such as anastomotic dehiscence, recurrent hemorrhage, or rupture of previously uninvolved aortic segments may occur [1,5]. We present a fatal case of *Salmonella*-associated Stanford Type A aortic dissection illustrating the malignant postoperative course of this entity.

CITATION
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Corresponding Author: Ceren Sayarer, Dokuz Eylül University, Faculty of Medicine, Department of Cardiovascular Surgery, İzmir, Türkiye
Email: cerensayarer@gmail.com

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CASE REPORT

A 58-year-old woman with a history of hypertension and inflammatory spondyloarthritis was admitted with dyspnea, generalized fatigue, myalgia, arthralgia, diarrhea, and nausea persisting for approximately one month. She had experienced intermittent chest pain since 2016. Her regular medication included levothyroxine. In 2022, she underwent total hysterectomy with bilateral salpingo-oophorectomy. She had received an unspecified treatment for spondyloarthritis approximately ten years earlier but had no subsequent rheumatology follow-up and had not received any immunosuppressive or biological therapy for at least five years before presentation.

On admission, the patient had fever up to 39°C. Physical examination revealed decreased breath sounds. Laboratory evaluation demonstrated marked systemic inflammation, with a C-reactive protein level of 300 mg/L, erythrocyte sedimentation rate of 85 mm/h, and D-dimer level of 5500 ng/mL. Computed tomography angiography revealed a markedly aneurysmal ascending aorta (maximal diameter of 94 mm) with an intimal flap consistent with a Stanford Type A aortic dissection (Figure 1). The patient had been referred from an external centre with a presumptive diagnosis of acute type A aortic dissection and was urgently transferred to the cardiovascular surgery department for emergency intervention.

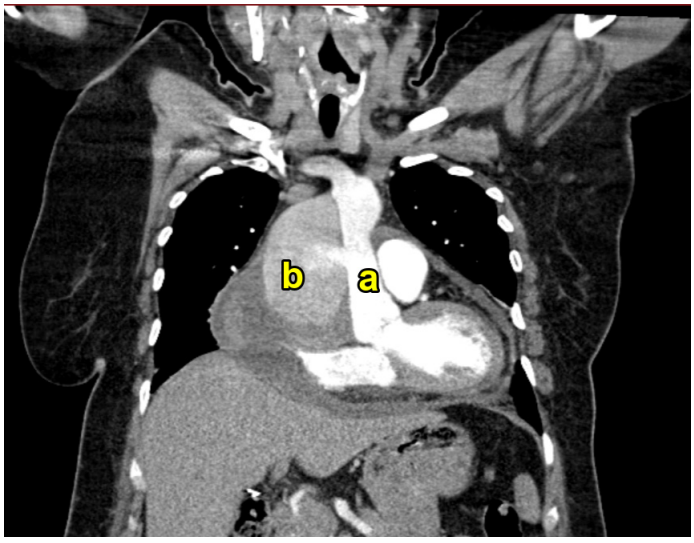


Figure 1. Preoperative computed tomography angiography (coronal view) demonstrating aneurysmal dilatation of the ascending aorta (maximal diameter 94 mm) with an intimal flap consistent with a Stanford type A aortic dissection. The true lumen (a) and false lumen (b) are indicated.

Transthoracic echocardiography showed normal left ventricular systolic function (ejection fraction 60%), no valvular insufficiency, and no pericardial effusion. Emergency surgery was performed. Because the patient presented as an acute aortic syndrome, infectious aortitis was not suspected preoperatively and a standard Dacron prosthetic graft was used. Intraoperatively, the dissection had a chronic appearance, and the pericardium was markedly adherent and appeared infected (Figure 2). Ascending aortic replacement was carried out. The patient was weaned from cardiopulmonary bypass without inotropic support and transferred to the intensive care unit in stable condition.

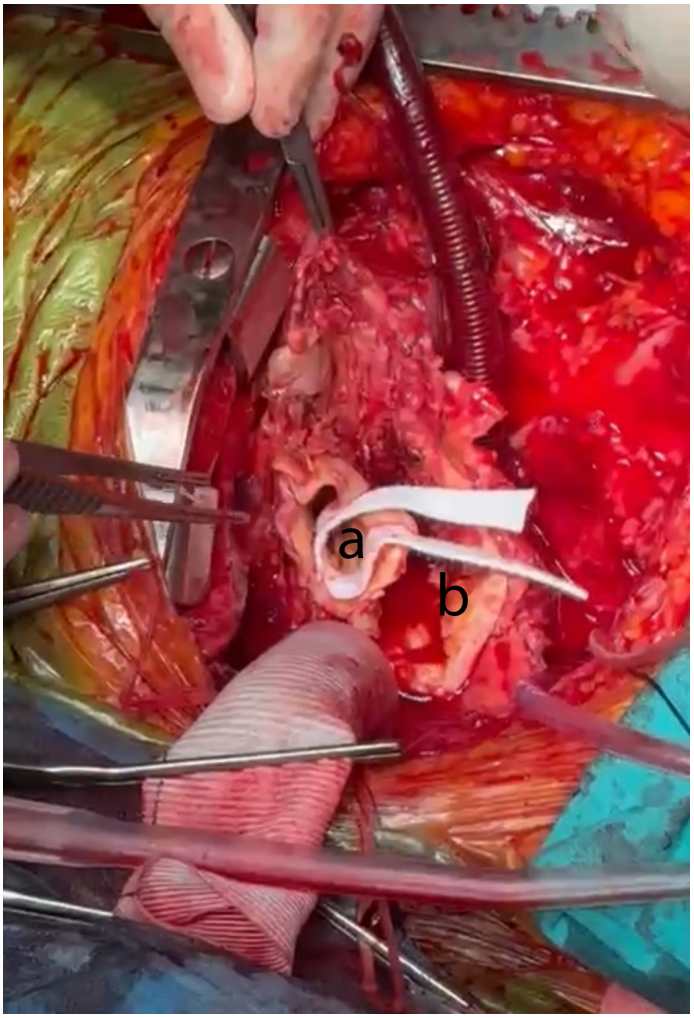


Figure 2. Intraoperative photograph taken during the initial operation, showing peri-aortic adhesions with an inflamed, infected-looking appearance surrounding the ascending aorta. The true lumen (a) and false lumen (b) of the chronic dissection are indicated; the braided material in the lower portion of the image (c) is the distal anastomosis of the prosthetic (Dacron) graft implanted during the same procedure.

Preoperative blood cultures grew *Salmonella* spp.; further serotyping was not performed. Antimicrobial susceptibility testing showed susceptibility to trimethoprim–sulfamethoxazole, cefotaxime and ampicillin, and resistance to ciprofloxacin. Empirical piperacillin–tazobactam plus amikacin (2 days) was changed, once the culture resulted, to ceftriaxone plus ciprofloxacin (18 days). Because fever and progressively rising acute-phase reactants persisted despite repeatedly negative

surveillance blood cultures, the regimen was escalated—under continuous infectious-diseases consultation—to meropenem (11 days), then meropenem plus amikacin (3 days), and finally ceftriaxone plus vancomycin (10 days); vancomycin was added empirically after the recurrent mediastinal bleeding episodes to cover possible gram-positive surgical-site superinfection. All blood cultures obtained over the following 42 days remained negative, and the total duration of antimicrobial therapy was approximately 44 days. No culture was obtained from the aortic wall intraoperatively at the initial operation because infectious aortitis was not suspected at that time; histopathological examination of the resected aortic wall demonstrated an elastic artery with medial degeneration, without direct demonstration of Salmonella in the tissue.

Approximately two weeks after the initial operation, the patient developed sudden massive bleeding from the previous mediastinal drain site while mobilizing. Emergency re-exploration revealed a defect at the proximal anastomosis, which was repaired. The prosthetic graft did not appear infected. The patient recovered uneventfully and returned to the ward.

Two weeks later, she experienced another episode of abrupt, profuse bleeding from the sternal incision. Emergency reoperation was performed. Due to uncontrollable hemorrhage, cardiopulmonary bypass and total circulatory arrest were instituted. Exploration revealed a 2 × 2 cm defect in the native aortic wall at the level of the non-coronary cusp, while the prosthetic graft remained intact. The defect was repaired using a bovine pericardial patch.

Postoperatively, the patient recovered without neurological complications and was extubated successfully.

Despite surgical control of bleeding, intermittent fever and progressive elevation of inflammatory markers continued. Postoperative computed tomography angiography (CTA) was performed, which demonstrated an intact aortic graft and patch repair, with no evidence of contrast extravasation, pseudoaneurysm formation, or radiological signs of ongoing infection. Two weeks later, the patient suddenly collapsed on the ward and suffered cardiac arrest. Cardiopulmonary resuscitation was unsuccessful. The presumed cause of death was acute rupture of another infected aortic segment leading to cardiac tamponade.

DISCUSSION

Salmonella aortitis is a rare but devastating condition with a well-documented propensity for rapid aortic wall destruction and fatal complications [1]. Persistent bacteremia and an intense inflammatory response contribute to progressive weakening of the aortic wall, even after surgical replacement of the affected segment.

An important consideration is the temporal relationship between the dissection and the Salmonella infection. The history of intermittent chest pain since 2016, the markedly aneurysmal ascending aorta (94 mm), the chronic intraoperative appearance of the dissection, and the histological finding of medial degeneration rather than acute suppurative aortitis together suggest that a pre-existing chronic aneurysm with dissection was most probably seeded by Salmonella during bacteraemia, consistent with the well-recognised tropism of Salmonella for diseased, aneurysmal or atherosclerotic aortic tissue [1,2]. A primary de novo infectious aortitis causing the dissection cannot be entirely excluded but appears less likely given these chronic features. It must be acknowledged that, as no aortic-wall culture was obtained and Salmonella was not directly demonstrated within the tissue, the diagnosis rests on the combination of Salmonella bacteraemia, the macroscopically infected-appearing peri-aortic tissue, and the aggressive destructive postoperative course rather than on direct histomicrobiological confirmation. Although immunosuppression is a recognised risk factor for Salmonella vascular infection [6], our patient had not received immunosuppressive or biological therapy for at least five years, indicating that the pre-existing aneurysmal aortic disease itself most likely constituted the principal predisposing factor.

A notable feature of the present case was the recurrence of catastrophic postoperative bleeding originating from the native aortic tissue rather than the prosthetic graft. This finding underscores that infection-related destruction may extend beyond the visibly involved aortic segment and continue postoperatively [3,5]. Similar reports have described late rupture or pseudoaneurysm formation despite technically successful surgery and prolonged antibiotic therapy [2,5].

The optimal antimicrobial regimen and duration for *Salmonella* aortitis remain uncertain. Although prolonged broad-spectrum therapy is generally recommended, persistent fever and inflammatory markers in this patient suggested ongoing infection and progressive tissue damage. Ultimately, neither repeated surgical intervention nor aggressive antimicrobial therapy was sufficient to prevent a fatal outcome.

The choice of emergency ascending aortic replacement with a standard Dacron prosthesis was dictated by the presentation as an acute aortic syndrome, before the infectious nature of the disease was recognised. Once Salmonella infection was confirmed, alternative strategies advocated for infectious aortic disease—radical debridement with in-situ reconstruction using cryopreserved arterial allografts or xenopericardial grafts, or rifampin-bonded prosthetic grafts—might have offered greater resistance to persistent infection. It should also be noted that cryopreserved arterial allografts and xenopericardial grafts are frequently difficult to procure in a timely manner—including at our institution and, more broadly, in our region—which, together with the emergency presentation, favoured the use of a readily available standard prosthetic graft. Systematic reviews report acceptable, though still substantial, morbidity and mortality with cryopreserved allografts [7] and xenopericardial reconstructions [8], and prolonged antimicrobial therapy has been associated with

lower rates of subsequent graft infection [9]. Persistent infection, as seen in this patient, is itself associated with substantially worse survival [10]. A staged strategy of initial antimicrobial infection control followed by delayed reconstruction may be considered when a chronic infected aneurysm is suspected in a haemodynamically stable patient; however, this was not applicable here given the acute presentation requiring emergency intervention. This case illustrates the diagnostic and therapeutic dilemma that arises when infectious aortitis is recognised only after conventional prosthetic repair.

CONCLUSION

This case demonstrates the highly aggressive and unpredictable nature of *Salmonella* aortitis. Even after timely surgical repair and prolonged antimicrobial therapy, progressive destruction of the native aortic wall may continue and lead to fatal complications. Surgeons should remain vigilant for recurrent bleeding and late rupture in patients with infectious aortic disease, as successful initial surgery does not guarantee long-term survival. Earlier recognition of the infectious aetiology might justify more aggressive upfront debridement with cryopreserved allograft or antibiotic-bonded grafts, and postoperative metabolic imaging such as 18F-FDG PET/CT may aid surveillance for residual infectious activity, potentially identifying threatened aortic segments before catastrophic rupture.

Patient Consent for Publication: Written informed consent for publication of this case report and accompanying images was obtained from the patient's legal next of kin.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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