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CAT BITE LEADING TO THORACIC AORTIC ENDOGRAFT INFECTION AND FATAL HAEMOPTYSIS

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ABSTRACT

Thoracic aortic graft infection is a rare but life-threatening complication of aortic repair, associated with high mortality due to sepsis, aortic rupture and fistulisation. Typical causative bacteria include *Staphylococcus spp.*, *Enterobacteriaceae* and *Pseudomonas spp.* However, atypical microorganisms may be implicated.

A 53-year-old patient with a history of thoracic aortic endovascular repair following dissection presented with fever, cough and dyspnoea, initially attributed to non-specific viral illness that led to discharge. Blood cultures later grew *Capnocytophaga canimorsus*.

Subsequent history revealed a recent cat bite, and a fluorine-18 -fluorodeoxyglucose positron emission tomography scan suggested thoracic aortic graft infection. Despite antibiotic therapy and endovascular intervention following a single episode of haemoptysis, fatal haemoptysis occurred.

This case highlights the potential for a minor cat bite to cause bacteraemia and catastrophic vascular graft infection. Thorough evaluation, including animal exposure history, and a high index of clinical suspicion are essential in patients with aortic grafts presenting with an infection of unclear origin.

KEYWORDS

Aortic graft infection, *Capnocytophaga canimorsus*

LEARNING POINTS

- Aortic graft infections carry a high risk of a fatal outcome, making early identification and multidisciplinary management critical.
- Early fluorine-18-fluorodeoxyglucose positron emission tomography/computed tomography scan should be considered when aortic graft infection is suspected despite inconclusive initial imaging.
- *Capnocytophaga canimorsus* bacteraemia following a cat bite can cause life-threatening infections, even in immunocompetent patients.

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INTRODUCTION

Aortic graft infection is an uncommon condition occurring in 1% to 6% of patients with aortic grafts[1], with a reported in-hospital mortality rate of 20% to 48%[2,3]. Major complications include sepsis, aortic rupture, and fistulisation to the aerodigestive tract[2,3].

Late graft infections (>2 months) may arise from delayed perioperative contamination, graft-enteric erosion or fistulisation, contiguous spread, and haematogenous seeding[4]. The main causative bacteria include *Staphylococcus spp.*, *Enterobacteriaceae*, and *Pseudomonas spp*.[4].

Zoonotic infections of aortic grafts are a rare occurrence[5], with no established prevalence data in the literature and most evidence presented as case reports or case series. Cat and dog bites are common, with cat bites carrying a higher risk of infection due to deeper puncture wounds with bacterial inoculation[6].

Capnocytophaga canimorsus, a Gram-negative, slow-growing bacillus of the commensal oral flora of cats and dogs, has been associated with severe infections and significant mortality (31% mortality in a series of 39 cases with sepsis)[7]. *C. canimorsus* causes severe infection predominantly among immunocompromised individuals but is usually sensitive to beta-lactam/beta-lactamase inhibitors[6,8].

The combination of virulent zoonotic bacteraemia and endovascular prosthetic material represents a high-risk scenario for aortic graft infection and requires prompt recognition with multidisciplinary care.

CASE DESCRIPTION

A 53-year-old man presented to the emergency department with a 3-day history of cough, fever, chills, and dyspnoea.

His medical history was significant for arterial hypertension, active smoking, and a type B aortic dissection in 2021 treated with two thoracic aortic endoprostheses, prosthetic carotid–carotid (right-to-left) and carotid–subclavian (left-to-left) bypasses. Retrograde descending aortic false lumen perfusion was recently identified and was awaiting elective endovascular intervention.

At initial presentation, temperature was 38.2 °C with otherwise unremarkable examination. Laboratory investigations showed elevated inflammatory markers (white blood cells 17 G/l, C-reactive protein 32 mg/l). Chest radiography, urinalysis, and viral nasopharyngeal swab were negative for infection. Blood cultures were taken and the patient was discharged with a presumptive diagnosis of non-specific viral respiratory infection.

Three days later, blood cultures grew *C. canimorsus*. The patient was contacted and returned to the emergency department the following day. On further questioning, he reported a cat bite to his right thumb approximately 2 weeks prior to symptom onset. The wound was minor and had properly healed.

A thoracoabdominal computed tomography (CT) scan demonstrated a small left pleural effusion and mediastinohilar lymphadenopathy, but no definitive evidence of vascular graft infection. Intravenous amoxicillin–clavulanate (2.2 g every 6 hours) was initiated, and the patient was admitted to internal medicine. Repeat blood cultures remained sterile and transthoracic echocardiography showed no signs of endocarditis.

Following multidisciplinary discussion with cardiology, vascular surgery and infectious diseases fluorine-18-fluorodeoxyglucose ([18F]-FDG) positron emission tomography (PET)/CT scan was planned to assess for endoprosthesis infection and elective surgery was postponed until infection resolution. However, the patient self-discharged after 5 days, with a prescription for oral amoxicillin–clavulanate (1g four times a day) and outpatient follow-up.

One week later, he re-presented with persistent fever and chills and reported poor adherence to antibiotic therapy. He was readmitted, and intravenous amoxicillin–clavulanate was resumed.

The FDG-PET scan demonstrated multiple intense FDG-avid perigraft foci along the endoprosthesis, with maximum standardized uptake value (SUVmax) of 14.0, highly suggestive of aortic endograft infection *(Fig. 1)*.

The following day, the patient developed haemoptysis. Contrast-enhanced thoracic CT scan showed newly

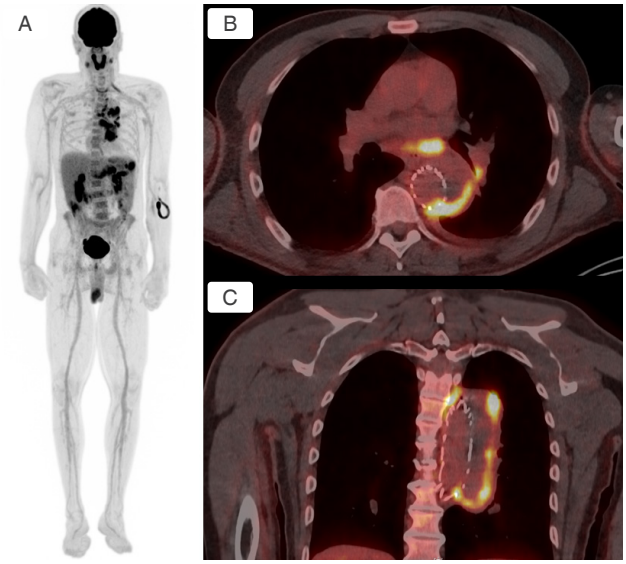


Figure 1. *Fluorine-18- fluorodeoxyglucose positron emission tomography/computed tomography ([18F]-FDG PET/CT) findings suggestive of thoracic aortic endograft infection after thoracic endovascular aortic repair for type B aortic dissection. A) Whole-body maximum intensity projection image showing abnormal focal FDG uptake in the thoracic aortic region; B) Axial fused PET/CT image demonstrating intense perigraft FDG uptake along the thoracic aortic endoprosthesis; C) Coronal fused PET/CT image confirming multiple hypermetabolic perigraft foci, with a maximum standardized uptake value (SUVmax) of 14.0, consistent with suspected aortic endograft infection.*

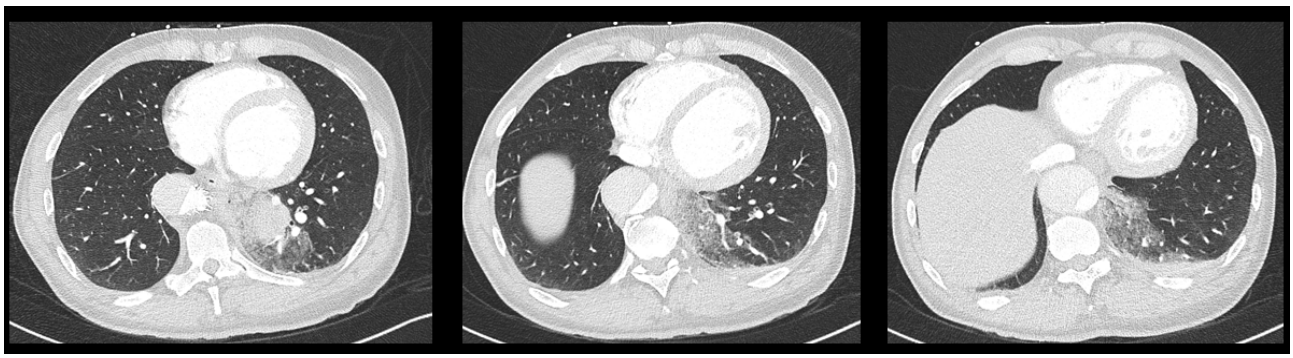


Figure 2. Contrast-enhanced chest computed tomography (CT) scan. Representative axial CT images showing newly developed ground-glass opacities in the left posterobasal lung, adjacent to the thoracic aortic false lumen. These findings were interpreted as focal alveolar hemorrhage and raised suspicion for an aortopulmonary fistula.

developed ground-glass opacities in the left posterobasal lung, adjacent to the patent aortic false lumen, consistent with focal alveolar haemorrhage *(Fig. 2)*. An aortopulmonary fistula was strongly suspected, although no direct fistulous tract was demonstrated on imaging.

Vascular surgery team proceeded with urgent endovascular intervention, placed an additional thoracic endoprosthesis and occluded the false lumen using a "candy-plug" device. In anticipation of new prosthetic material implantation, antimicrobial therapy was escalated preoperatively to intravenous meropenem (2 g every 8 hours) and clindamycin (600 mg every 8 hours). Metagenomic next-generation sequencing of perioperative blood samples adjacent to endograft were positive for *Capnocytophaga spp.* deoxyribonucleic acid.

In the early postoperative hours, the patient developed massive haemoptysis followed by cardiac arrest and died despite prolonged resuscitation efforts.

DISCUSSION

Aortic graft infections with zoonotic pathogens are atypical. Even though case reports exist, including *C. canimorsus*, their clinical significance is not well defined in the literature and may be underrecognized[5,9]. Haematogenous seeding is less common in late graft infections due to graft endothelialisation[4].

Symptoms of late infection may be insidious and physical examination inconclusive[4,10]. Imaging techniques are thus essential for diagnosis. Computed tomography angiography (CTA) is the primary tool for suspected infection; nevertheless, its sensitivity (0.67) and specificity (0.63) are limited[4,10]. [18F] FDG-PET scan is a complementary and more sensitive (0.95) modality than CTA, yet its use may be limited by availability and early postoperative changes[4,10].

Treatment of graft infections consists of antibiotic therapy and, if possible, open surgical graft explantation (complete or partial), debridement, microbiological analysis and vascular reconstruction[11]. Conservative treatment with antibiotic therapy alone is associated with worse outcomes[1,11], and is less likely to be effective in the presence of large purulent infections, vascular discontinuity and fistulisation. However, it may be considered for patients unfit for, or denying, surgery and requires prolonged therapy[11].

Our patient presented with symptoms mimicking respiratory viral infection, with unreported animal exposure. Initial discharge seemed reasonable, but incomplete exposure questioning limited risk stratification. This initial presentation led to a delay in treatment of *C. canimorsus* bacteraemia, compounded by early self-discharge and limited adherence to treatment. [18F] FDG-PET scan and metagenomic next-generation sequencing of perioperative blood samples were compatible with *C. canimorsus* graft infection.

The patient, though immunocompetent, had a history of complex aortic repair, rendering him a poor candidate for complete graft explantation due to a technically difficult and high-risk intervention. Moreover, the perfused false lumen may have been more susceptible to bacterial seeding and for fistula formation than the endothelialized graft. Alveolar haemorrhage signs on CT scan were notably close to the perfused false lumen.

After an episode of haemoptysis, an endovascular prosthesis was placed. This strategy can be used in infected, bleeding patients to cover the bleeding source as a temporising measure for open surgical graft excision and vascular repair. It is not recommended as a definitive therapy and prosthesis placement in infected terrain is itself at high risk of infection[11].

Despite endovascular salvage procedure, our patient developed fatal haemoptysis, though whether it arose from aorto-pulmonary fistulisation due to infection or to a postoperative complication remains subject to speculation. Intraoperative control showed effective false lumen occlusion.

Bacteraemia with a virulent microorganism, combined with a high mortality infection, limited surgical possibilities and poor treatment adherence likely contributed to a fatal outcome. Earlier infection identification might not have altered this issue.

This case underscores the need for animal exposure history in patients with aortic grafts and infection of unknown origin, and supports early use of [18F] FDG-

PET scan when initial imaging is non-conclusive for graft infection. It also highlights that zoonotic bacteraemia, even when rare, can cause devastating infections with the presence of vascular prosthetic material in immunocompetent patients.

Aortic graft infection outcomes are often sombre once complications have developed, thus, early recognition, rapid escalation to advanced imaging and multidisciplinary care are essential for its management.

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