CASE REPORT

Abdominal Aortitis due to *Streptococcus pneumoniae* and *Enterobacter aerogenes*

A Case Report and Review

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Endovascular infections are 1 cause of fever of unknown origin. We describe a diagnostically challenging case of cryptogenic abdominal aortitis from *Streptococcus pneumoniae* and *Enterobacter aerogenes*. A 72-year-old male presented with epigastric pain, fevers, and chills. A computed tomography scan demonstrated enlargement and ulceration of the distal abdominal aorta, prompting urgent vascular surgery. Intraoperative tissue cultures grew *S. pneumoniae* and *E. aerogenes* and gatifloxacin was administered for 6 weeks. Spontaneous abdominal aortitis is uncommon and usually due to a single pathogen. This is the second reported case of polymicrobial infectious aortitis and to date, *Enterobacter* has only been reported in infected aortic grafts. Clinicians should maintain a high index of suspicion for infectious aortitis as the mortality, if only treated medically, approaches 100%.

KEY WORDS: aortitis; *Streptococcus pneumoniae*; *Enterobacter aerogenes*; infection.

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Infectious aortitis occurs with endocarditis, bacteremia,¹ preexisting aneurysms, and other infections,^{2,3} and is 1 cause of fever of unknown origin (FUO). Other cases occur with central catheters and medical devices (pacemakers, defibrillators) or are idiopathic.⁴ Common pathogens include *Staphylococcus aureus*, *Salmonella*, *Proteus*, and *Escherichia coli*.¹ We describe a diagnostically challenging case of cryptogenic abdominal aortitis from *Streptococcus pneumoniae* and *Enterobacter aerogenes*.

CASE REPORT

A 72-year-old male presented with 2 weeks of epigastric pain, fevers, and chills. He denied cough, back pain, migraines, diarrhea, urological symptoms or past urinary tract infections, upper respiratory tract infections, sinusitis, or otitis. His immunizations, including the pneumococcal and influenza vaccine, were current. He had a history of coronary artery bypass grafting and peripheral vascular disease with iliac angioplasty 10 years back. He had a 25 pack-year smoking history. He denied intravenous drug use, recent central catheters, surgery, trauma, or the use of ergot-alkaloids.

In the previous week, he had received 4 days of empiric antibiotics (ampicillin-sulbactam), last given the day before his transfer to our facility. Three sets of blood cultures were drawn before administration of antibiotics; all remained without growth at 5 days.

On admission, his temperature was 37.9°C, blood pressure was 156/84, heart rate was 66, and respiratory rate was 24. His heart was regular with a II/VI systolic murmur. His abdomen was distended with diffuse epigastric tenderness without pulsatile masses, peritoneal signs, organomegaly, or flank tenderness. He had no skin lesions or rashes, joint abnormalities, or neurologic deficits. The white blood cell count was 13.2×10^9 cells/ μ L. The erythrocyte sedimentation rate (ESR) was 107 mm/h. Blood and urine cultures obtained without antibiotic treatment were without growth for more than 5 days. A chest radiograph was normal.

On admission, he was not continued on antibiotics but became febrile to 40°C. An abdominal computed tomography (CT) with CT angiogram (CTA) demonstrated extensive peri-aortic inflammation and ulceration without aneurysm from the superior mesenteric artery to the aortic bifurcation (Fig. 1A). A transesophageal echocardiogram was without vegetations. An Indium¹¹¹-labeled white blood cell scan was without evidence of infection. Antibiotics were not empirically started, and a presumptive diagnosis of idiopathic retroperitoneal fibrosis was made. Prednisone was begun with rapid improvement in his abdominal pain and resolution of his fevers.

Three weeks later, he developed severe abdominal pain radiating to his back and was readmitted. A repeat abdominal CTA demonstrated enlargement and ulceration of the distal abdominal aorta to 4 cm (Fig. 1B). During urgent abdominal surgery, the aneurysm was resected and a rifampin-soaked Dacron graft was placed. Grossly, the aortic tissue was inflamed and necrotic, pale gray, and thickened to 0.3 cm.

Tissue gram stain showed 4 Gram-positive cocci in chains that grew *S. pneumoniae* with intermediate resistance to penicillin (MIC = 1.0 mg/L) but susceptible to gatifloxacin (MIC = 0.25 mg/L) and vancomycin (MIC = 0.25 mg/L). Tissue cultures also grew *E. aerogenes* resistant to cefuroxime (MIC > 96.0 mg/L) but susceptible to gatifloxacin (MIC = 0.25 mg/L). Tissue cultures for anaerobic and fungal organisms, and acid-fast bacteria were negative. Postoperative blood cultures remained negative for > 72 hours. Postoperatively, he was treated with gatifloxacin 400 mg daily for 6 weeks. He clinically responded, was discharged, and a year later, is well without recurrence.

DISCUSSION

Bacteremia is the most common mechanism by which the aorta becomes infected, but extension from contiguous sites

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FIGURE 1. (A) Initial abdominal computed tomography angiogram (CTA) demonstrating a large aortic ulceration without aneurysm (arrow). (B) Repeat reconstructed abdominal CTA demonstrating marked enlargement and ulceration of the infrarenal abdominal aorta (arrow).

of infection also occurs. How pathogens invade the aorta is unknown but may involve attachment to the aortic intima, perhaps by seeding an atheromatous plaque, or invade the aortic wall via the vasa vasorum. In the preantibiotic era, 90% of aortic infections occurred with endocarditis,⁵ while recent data suggest that the association with endocarditis is less common. In 37 cases of *pneumococcal* aortitis, only 5 occurred with endocarditis; the majority of cases (41%) were cryptogenic.⁶

Infectious aortitis is uncommon, with an incidence of 0.3% to 0.4% in large necropsy studies^{7,8} but, as with other endovascular infections, should be included in the differential diagnosis of FUO. The classic triad of fever, abdominal pain, and a palpable, pulsatile abdominal mass is uncommon. In retrospective analyses of infectious aortitis due to *Salmonella*, the incidence of fever, abdominal pain, and a palpable pulsatile mass was 79%, 42%, and 42%, respectively.^{9,10} If the aorta is not aneurysmal, detecting a pulsatile abdominal mass may be impossible. Thus, with a low rate of disease occurrence and poor sensitivity and specificity of signs and symptoms, diagnosing infectious aortitis can be challenging.

Imaging studies may confirm the diagnosis. Computed tomography angiogram may show peri-aortic inflammation, aortic aneurysms,¹¹ or progressive aortic dilatation.^{11,12} Other CT findings include peri-aortic soft-tissue masses or gas, fat stranding, and fluid collections.¹¹ The utility of leukocyte scintigraphic imaging is variable. In 1 report, an abnormal study led to a confirmed tissue diagnosis in >80% of studies.¹¹

Many pathogens may cause infectious aortitis. In a series of 21 patients, the most common organisms isolated were *Salmonella* (38%), *Staphylococci* (19%), and *E. coli* (14%).¹² Other organisms include *Streptococcal* species, *Candida*, *Aspergillus*, and *Listeria monocytogenes*.^{6,13–15}

In this case of infectious aortitis, 2 organisms were isolated: *S. pneumoniae* and *E. aerogenes*. Upper respiratory colonization with transient bacteremia or an undiagnosed *pneumococcal* pneumonia with resulting bacteremia may have seeded an atherosclerotic aneurysm. The source of *E. aerogenes* is unclear but may have been from an undiagnosed urinary tract infection or pneumonia. To our knowledge, this is only the second report in the literature noting multiple organisms in infectious aortitis.¹² Additionally, *Enterobacter* has on-

ly been reported as a cause of infectious aortitis in the setting of aortic grafts.^{16,17} This is the first time that *Enterobacter* has been reported in cryptogenic infectious aortitis.

The diagnosis of infectious arteritis in this case was elusive for a number of reasons. Early empiric antibiotic therapy may have limited the yield of subsequent blood cultures. There was no aneurysmal dilatation of the aorta and a palpable, pulsatile mass could not be detected on physical examination. The combination of a periaortic mass on CT, fever, leukocytosis, abdominal pain, elevated ESR, and negative blood cultures suggested a diagnosis of idiopathic retroperitoneal fibrosis.

Although a presumptive diagnosis of retroperitoneal fibrosis was made, other diagnoses including lymphoma, vasculitis, solid tumor malignancy, and occult infection were considered. However, given his normal peripheral smear and lack of lymphadenopathy, a negative vasculitis evaluation, the lack of masses on CT imaging, and the extensive work-up for infection (which was negative at this time), a diagnosis of retroperitoneal fibrosis was consistent with his symptoms and CT findings. Although our patient had a moderate initial response to corticosteroids, his clinical status ultimately deteriorated, suggesting that steroids worsened his disease, altered the natural history of the infection, or both.

The diagnosis ultimately was made when repeat CTA showed an enlarging aorta. Surgical treatment was essential as the mortality in those treated medically alone approaches 100%. In those treated surgically and medically, the mortality is between 20% and 60%.⁹

CONCLUSION

Infections of the aorta are rare, are caused by various pathogens, and are difficult to diagnose. Patients may present with insensitive and nonspecific clinical findings including fever and abdominal pain (and a palpable, pulsatile abdominal mass if the aorta is aneurysmal). Blood cultures, if positive, can be helpful, as can be CTA, and leukocyte scintigraphic imaging. A high index of suspicion is necessary as the mortality, if only treated medically, approaches 100%. Combined surgical and medical therapy is superior, reducing the mortality rate to between 20% and 60%.

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