Probable zoonotic aortitis due to group C streptococcal infection

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Human infections due to group C streptococcus (SGC) are unusual. Among them, vascular compromise, especially aortic involvement, is extremely rare. A case of microbial aortitis with aneurysm formation, likely secondary to a SGC soft tissue infection, in a 61-year-old patient who was caring for a purulent leg wound of his horse, is presented. He was successfully treated with antibiotics and in situ aortic replacement with a prosthetic graft and an omental wrap. He remains well almost 2 years after surgical intervention. Aortic infection caused by SGC is a rare condition that can be successfully treated with in situ prosthetic graft replacement. (J Vasc Surg 2007;46:1039-43.)

β-hemolytic streptococcus Lancefield group C is a worldwide causative agent of many forms of horse and foal infections and has also been recognized as a zoonotic agent causing human disease, including aortitis. We present a probable case of zoonotic aortitis due to these bacteria. The related literature is also analyzed.

CASE REPORT

A 61-year-old man presented to another hospital with fever and chills associated with abdominal and lower back pain. His illness started 4 weeks previously, when he developed an open wound in his right hand. The patient had been caring for an infected leg wound of his horse. His workup demonstrated blood cultures positive for β-hemolytic streptococcus Lancefield group C (SGC). Computed tomography (CT) scan of the abdomen revealed aortic inflammation associated with a 2.5-cm irregular distal aorta, consistent with bacterial aortitis (Fig 1). The patient was treated with intravenous (IV) ceftriaxone, metronidazole, and tobramycin, which were given through a peripherally inserted central catheter. He received these antibiotics for 2 weeks, with minimal clinical improvement. He continued to have back pain and intermittent fever. Re-examination with a CT scan confirmed that the aorta had increased to 3 cm, thus suggesting an infected aneurysm with a contained rupture (Fig 2).

The patient was transferred to the vascular service at University Medical Center. On presentation, he complained of low-grade fever and back pain. His examination was unremarkable except for a temperature of 37.3°C and slight abdominal tenderness to palpation. The patient was taken to the operating room for repair of his aneurysm. Infrarenal aortic inflammation with dense adhesion to the vena cava was found. After aortic control, the aneurysm was opened, and a 2.5-cm disruption of the aortic wall at the level of the inferior mesenteric artery level was evident. There was no purulence. The necrotic aorta and surrounding tissue were debrided until only viable tissue remained. A 16-mm expanded polytetrafluoroethylene interposition graft was used for aortic replacement, and it was wrapped with omentum (Fig 3). Culture of the necrotic aorta grew no organisms. The patient's perioperative recovery was unremarkable. An infectious disease consultation was obtained in addition for combined management given the rarity of the bacteria that were isolated. He received a 6-week course of IV vancomycin. An abdominal CT scan 18 months after intervention revealed an infrarenal aortic graft with no evidence of complications, and he remains well almost 2 years later (Fig 4).

The horse was treated with IV antibiotics and local wound care, with improvement. Nasal swab cultures were obtained from the animal, with negative results.

DISCUSSION

Aortic aneurysm infections were initially described in 1885 by Osler.[1] Since his description, newer terminology has arisen as our understanding of the various presentations of infectious aortitis has grown. Nomenclature for native arterial infections is a matter of confusion, because most clinicians agree that multiple forms exist. The case here presented likely belongs to a rare case of microbial arteritis with aneurysm formation, according to the Reddy and Ernst classification.[2] This entity includes patients with bacteremia that seed normal or atherosclerotic arteries, weakening the arterial wall and resulting in an aneurysm. In these cases, the source of bacteria can be multiple, including pneumonia,[3] appendicitis,[4] intra-arterial drug injection by abusers,[5] osteomyelitis,[6] or typhoid fever,[7] among others. The most common bacteria associated with aortic infections are salmonella and staphylococcus, whereas streptococcal infections are rare.[8] This report is one of the five cases reported in the literature of a native aortic zoonotic infection due to SGC (Table).[9-11] Two other reports were found of SGC infections affecting aortic grafts.[12,13] We do not know with certainty whether the aortic infection developed at an area of pre-existing aortic disease or in a normal aortic wall. Abdominal CT scans before this patient's current admission were not available. Moreover, during the operation, there was no identifiable aorta at the level of the inferior mesenteric artery. Minimal thrombus was present, and the blood flow was contained by surrounding friable tissue.

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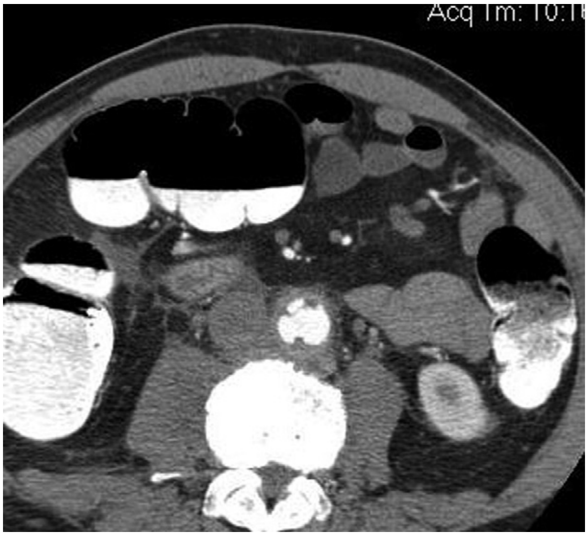

Fig 1. Initial computed tomographic scan, obtained on admission at the referring institution, interpreted at that time as aortic inflammation associated with intimal irregularities, consistent with bacterial aortitis. The aorta was measured in its maximum diameter as 2.5 cm.

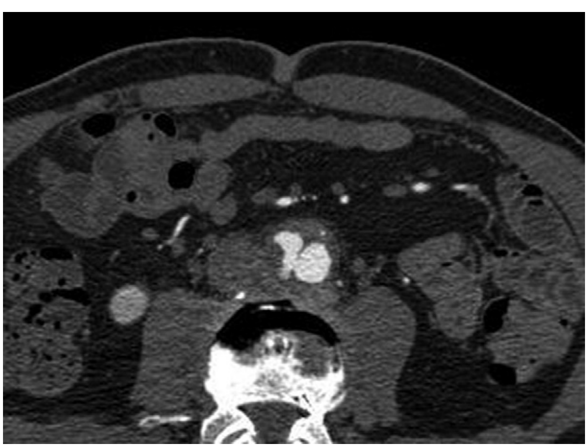

Fig 2. Repeated abdominal computed tomographic scan 2 weeks after the initial study, revealing worsening of the periaortic inflammation. The aorta now measured 3 cm in maximum diameter. This prompted surgical consultation and, eventually, surgical intervention.

Fig 3. Repeated abdominal postoperative computed tomographic scan, showing a tube aortic prosthetic graft and resolution of periaortic inflammation. Omentum was used to wrap the newly placed prosthetic graft.

Fig 4. Follow-up computed tomographic scan 18 months after surgical intervention reveals a stable appearance of the infrarenal aortic prosthetic graft with no evidence of complications.

Large-colony Lancefield SGC is a β-hemolytic organism commonly found in mammals, including domestic animals and horses. It is by far the most common cause of wound infections and causes respiratory tract infections of foals and young horses.[14] SGCs are subdivided into *S dysgalactiae* subsp *equisimilis, S dysgalactiae* subsp *dysgalactiae, S equi* subsp *equi*, and *S equi* subsp *zooepidemicus.*[15]

SGC can be found as part of normal flora in humans, populating the skin, upper respiratory, gastrointestinal, and genitourinary tracts.[16] Even though they are thought to be very infrequent pathogens in humans, several reports exist of ocular,[17] respiratory,[16] vascular,[9-13] systemic,[18] or central nervous system[19] infections. Furthermore, a carrier state in humans has also been described.[20] A review of 150,000 human blood cultures obtained at the Mayo Clinic over 10 years found only 8 cases of SGC bacteremia.[18] Similarly, 28 cases of SGC bacteriemia were found among

Table. Zoonotic aortic infections due to streptococcus group C

Study	*Age (y)*	*Sex*	*Bacteria*	*Presentation*	*Therapy*	*Follow-up*
This report	61	Male	Beta-hemolytic SGC	3-cm AAA	ePTFE tube graft + omental wrap	Did well; 19 mo
Albarracin[9]	73	Male	SGC subsp *zooepidemicus*	6.3-cm AAA	Aortoiliac graft	Did well; 14 mo
Wong[10]	65	Male	SGC subsp *zooepidemicus*	8-cm AAA	Dacron* aortobifemoral graft	Did well; 12 mo
Wong[10]	62	Male	SGC subsp *zooepidemicus*	12-cm AAA	Dacron aortobifemoral graft	Died 10 d after surgery
Perkins[11]	59	Male	Beta-hemolytic SGC	Infrarenal saccular AAA	Exploratory laparotomy ("unresectable")	Died 2 wk after surgery

SGC, Streptococcus group C; *AAA,* abdominal aortic aneurysm; *ePTFE,* expanded polytetrafluoroethylene.
*DuPont (Wilmington, Del).

74,126 blood cultures in a 5-year period at a Spanish teaching hospital.[21]

Horse owners have often considerable close contact with their animals, including their mucous secretions, such in the case of our patient. Horses colonized with SGC are a large reservoir for a potential human infection. Among patients with streptococcal bacteriemia, 23.9% of them reported exposure to animals or their products.[22] This could readily explain the transmission of SGC resulting in colonization, infection, or both. The subspecies *zooepidemicus* and *equisimilis* are mostly associated with human infections.[23] *Streptococcus zooepidemicus* is considered the most virulent microorganism of the SGC species, with a higher associated mortality than the others. Most patients (73.9%) affected with this infection have a serious underlying disease, such as cardiovascular disease, malignancy, or immunosupression.[22]

Bradley and colleagues[22] reported in 1991 probably the most comprehensive analysis of patients with SGC bacteriemia. In their work, fever (67.1%), chills (30%), and fatigue (22.9%), as well as gastrointestinal complaints (61.4%), were the most common presenting symptoms among 88 patients. These were precisely the symptoms that our patient initially developed. The port of entry was likely the open wound in his hand. A cutaneous source of infection was seen in 17.1% of the patients in the Bradley series.[22] The respiratory and gastrointestinal routes were slightly more often reported as the port of entry (20.5% and 18.2%, respectively). However, the source in the vast majority (38.6%) was not identified. Finally, agreeing with our literature review, their group found vascular involvement of patients with SGC bacteriemia in only 3 of 88 patients (2 mycotic aneurysms and 1 fistula infection), again speaking to a very rare occurrence.[22]

Although aortography was once the gold standard for confirming aortic involvement, other invasive[24-26] and noninvasive[27,28] imaging studies are increasingly used. Pathologic examinations can assist in the diagnosis when tissue is available. However, the diagnosis of aortitis is difficult because of nonspecific clinical, pathologic, and aortographic findings. Furthermore, the infectious or inflammatory nature of an aortitis is difficult to assert because the microbiological results are often negative. In our case, animal cultures were sent after IV antibiotics were initiated in the horse, the results being, unfortunately, negative. Therefore, we do not have absolute proof of a zoonotic infection, and our case remains as a probable one. However, circumstantial evidence is strong supporting this as the source of this patient's aortitis. Very few cases report the original source of infection with isolation of SGC from the natural host.[11]

Early diagnosis and aggressive therapy are needed to avoid life-threatening complications. SGC typically shows high sensitivity to penicillins, cephalosporins, chloramphenicol, clindamycin, and erythromycin,[22] which are the antibiotics of choice. In fact, the sensitivity of the SGC affecting our patient indicated a good response to penicillin. However, he reported a severe allergy to penicillins, and this was why this antibiotic was not used from the beginning. The patient was under the care of a general surgeon at the referring institution, and our suspicion is that his initial attempt was to sterilize the aorta with medical therapy. When the patient arrived at our institution, we believed that ceftriaxone provided good coverage for streptococci, and given the length of therapy that had already been administered (more than a month), we did not consider any different targeted antibiotic regimen.

Resistance has been shown to penicillin,[21] tetracycline, sulfonamides, and aminoglycosides.[22] The combination of sensitivity-directed antibiotic treatment and surgical intervention is the best way to approach this entity. Surgery is often needed to treat associated complications. Factors such as patients' comorbidities, bacterial virulence, and grade of contamination of the surgical field are important at the time to choose the surgical procedure. Surgical strategies for infectious aortitis include extra-anatomic grafting followed by staged excision of the infected aortic segment,[29] in situ reconstruction using omental wraps[30] or rifampin-bonded grafts,[31] autogenous tissues,[32] or cryopreserved arterial[33] or venous (unpublished data) allografts. Endovascular techniques[34,35] have also been used. They are generally considered as a bridge therapy before a more permanent repair is undertaken. However, the patient on arrival was stable and not ill enough for us to consider endografting as a temporizing maneuver.

We chose an in situ replacement over other more conventional repair techniques because our patient was relatively young and because of the existence of literature supporting the use of omental wraps in this situation,[30] our own institutional positive experience with this technique, and also the lack of controlled studies comparing these methods. A higher reported patency of in situ replacement over extra-anatomic bypasses, as well as similar reinfection rates reported between in situ repair with an omental wrap and extra-anatomic bypasses for cases of aortic graft infections, further support our approach.[29,30,36-38]

Six weeks of antibiotic treatment, directed by the minimum inhibitory and bactericidal concentrations of these agents for the causative organisms, has been suggested.[39] The lifelong duration of oral antibiotics for this patient can be seen as an exaggeration. However, there are no available data to support the benefit of shorter courses in cases of these rare streptococcal aortic infections.

CONCLUSIONS

Zoonotic infections of abdominal aortic aneurysms due to SGC have been reported in only four instances before our report. The rarity of this situation does not allow for meaningful analysis or guidelines. However, aortic infections due to these bacteria seem to follow the same principles of infections due to other more prevalent microorganisms. Their diagnosis is difficult because of the vague, nonspecific symptomatology. Findings of symptomatic periaortic inflammation, with or without the presence of an aortic aneurysm in a patient with a history of animal contact, should raise the possibility of a zoonotic infection. Therapy is associated with significant mortality, especially if the presentation is of rupture or is symptomatic, such as in our patient. On the basis of our case and our review of the literature, in situ prosthetic graft replacement seems to be a reasonable alternative under certain circumstances, such as when minimal contamination is found and when the involved bacteria have low virulence.

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