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CASE REPORT

Group B Streptococcus Mycotic Aneurysm of the Abdominal Aorta: Report of a Case and Review of the Literature

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Mycotic aneurysm of the aorta is an uncommon condition, and Group B Streptococcus (GBS†) is exceedingly rare in this setting. We present the first reported case of a GBS-infected abdominal aortic aneurysm (AAA) in North America. Key clinical and imaging findings and pathologic correlation are highlighted. A relevant review of the literature is discussed, which will bring the reader up to date with this specific disease entity.

INTRODUCTION

Focal dilatation of any artery is termed as an aneurysm. The most common site for an arterial aneurysm is the abdominal aorta, and a diameter of more than 3 cm is generally considered as aneurysmal [1]. The incidence of AAA has been increasing [2]. This has been attributed in part to increased use of ultrasonography and other cross-sectional imaging (computed tomography and magnetic resonance imaging of abdomen), resulting in increased incidental case finding. AAA greater than 4 cm in diameter is considered clinically important, and its prevalence was found to be 1.4 percent be-

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†Abbreviations: GBS, Group B Streptococcus; AAA, abdominal aortic aneurysm; DVT, deep venous thrombosis; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; CT, computed tomography; MIC, minimum inhibitory concentration.

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tween the ages of 50 to 79 in a study performed in the United States [3]. Another study performed in Europe estimated the prevalence of AAA greater than 4 cm to be about 1 percent in men between the ages of 55 and 64 [4]. The incidence of ruptured AAA is also reported to be increasing [5]. The reasons behind this trend have not been studied in a scientific way. The authors believe this could be a result of increased life expectancy and increased prevalence of other risk factors described below.

The exact underlying cause of AAA is uncertain. The key event is destruction of elastin in the aortic wall that creates abnormal stress as a result of the blood pressure within the lumen [6].The strongest risk factor associated with AAA is cigarette smoking [3,7]. Age is also considered a significant risk factor with a dramatic increase in prevalence in patients older than 60 [1,4]. Other significant risk factors include male sex, hypertension, and Caucasian race [4,7]. Atherosclerosis has been considered the etiology of an overwhelming majority of AAAs [8]. It is interesting to note that several risk factors associated with AAA also predict atherosclerosis. Some examples are cigarette smoking, hypertension, and high serum cholesterol. Hence, AAA and atherosclerotic disease usually coexist. It is unclear whether atherosclerotic disease causes AAA or vice-versa [9]. Genetic causes for AAA have also been suggested [10]. Infectious agents, cystic medial necrosis, and Marfans syndrome are other rare causes of AAA.

A mycotic aneurysm is destruction of the vessel wall leading to dilatation of an artery caused by infection due to a microorganism. Mycotic aneurysms are uncommon. In one study, only 0.9 percent of all aneurysms were found to be mycotic [11]. The infectious agent is generally a bacterium, although other microorganisms are also implicated in rare cases [12]. GBS causing a mycotic aneurysm is extremely rare, and so far, only five cases have been reported [13-17]. Among these cases, three were associated with AAA [13], and one each was associated with ascending aorta [15] and common femoral artery [14]. It is interesting to note that the incidence of mycotic AAA has increased in the post antibiotic era [18], while the incidence of peripheral mycotic aneurysms has decreased [19]. The reasons behind this shift are not clear.

GBS is a pathogen that colonizes the gastrointestinal and genital tracts in humans [20]. It is a gram positive coccus. In pregnant and postpartum women, GBS is known to cause urinary and genital tract infections, postpartum endometritis, puerperal sepsis, and bacteremia [21,22]. GBS is also an important causative agent of illnesses in infants (respiratory tract infections and septicemia) as a result of vertical transmission [23]. A significant proportion of GBS cases have been reported in elderly populations such as non-pregnant women older than 65 and nursing home residents [17]. However, it is exceedingly rare for GBS to cause an AAA.

CASE REPORT

A 75-year-old Caucasian male presented with 2-day history of left leg swelling and discomfort. Review of systems was positive for mild back pain, fever, night sweats, and weight loss. Past medical history was significant for ischemic heart disease, hypertension, hyperlipidemia, and chronic obstructive lung disease. He also had a 60-packs-a-year cigarette smoking history. There was no family history of AAA. One month ago prior to presentation, he was treated for suspected bronchitis/exacerbation of chronic obstructive lung disease with 1 week of intravenous moxifloxacin (400 mg daily) as an inpatient and 1 more week of oral moxifloxacin (400 mg daily) as an outpatient. Physical examination was significant for 4+ pitting edema and tenderness in the left lower extremity. The patient's laboratory workup was remarkable for elevated C-reactive protein (CRP) 23.630 mg/dl (normal value <0.8 mg/dl); elevated erythrocyte sedimentation rate (ESR) of >100mm in the first hour (normal range 0-20 mm/h); leucocytosis, white blood cell count 14,000/cu mm (normal range, 3,000-10,000/cu mm);

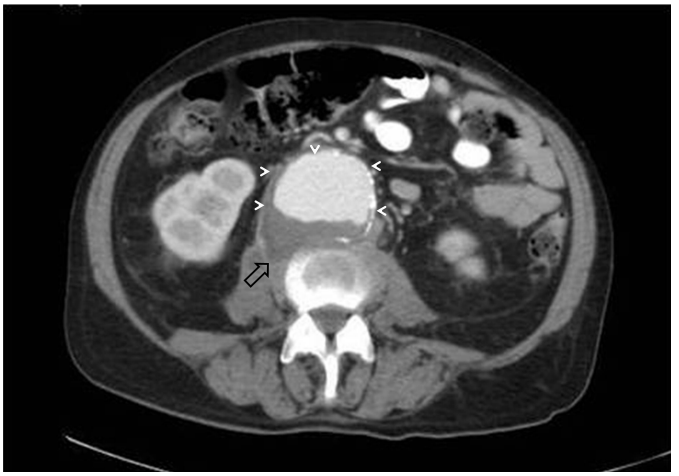

Figure 1. Axial contrast enhanced CT of the abdomen demonstrating an infrarenal abdominal aortic aneurysm (arrowheads). The large black arrow shows a small area of low attenuation in the anterior right psoas muscle that was confirmed to be a contained rupture during surgery.

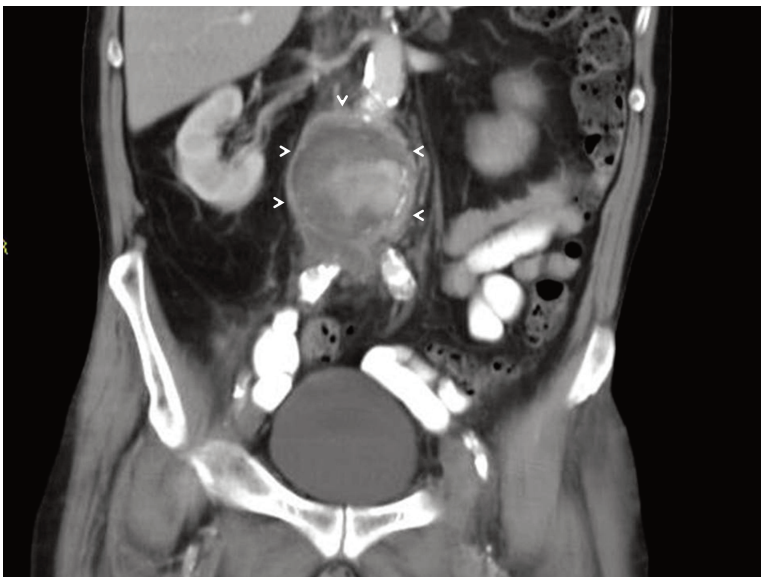

Figure 2. Reconstructed CT image in the coronal plane shows an irregular infrarenal abdominal aortic aneurysm (arrowheads).

and neutrophilia 81 percent (normal range, 45-70 percent). Doppler ultrasound of the lower extremities demonstrated extensive deep venous thrombosis (DVT) involving the left popliteal vein, left common femoral vein, and extending into the left external iliac vein. Anticoagulation was started with low-molecular weight heparin, and further imaging was done at this time as extensive DVT raised the suspicion of malignancy. Contrast enhanced chest, abdomen, and pelvis computed tomography (CT) showed an infrarenal irregular abdominal aortic aneurysm (5.7 cm x 4.0 cm x 5.1 cm) with a contained rupture (Figures 1 and 2). A mycotic aneurysm was suspected with clinical picture of fever, low back pain, leucocytosis, and the irregular CT appearance of the aneurysm. Intravenous Vancomycin 1gm every 12 hours and intravenous Piperacillin-Tazobactam 3.375gm every 6 hours was started empirically.

Emergency laparotomy was performed and confirmed the presence of a contained aneurysmal leak. The aneurysm was excised, and circulation was re-established by collagen-impregnated Dacron graft presoaked in Rifampin.

Transesophageal echocardiogram did not show any valvular vegetations. Histological examination of excised wall showed heavy infiltration with Gram-positive cocci within the arterial wall (Figures 3 and 4). The culture of the aneurysmal contents was

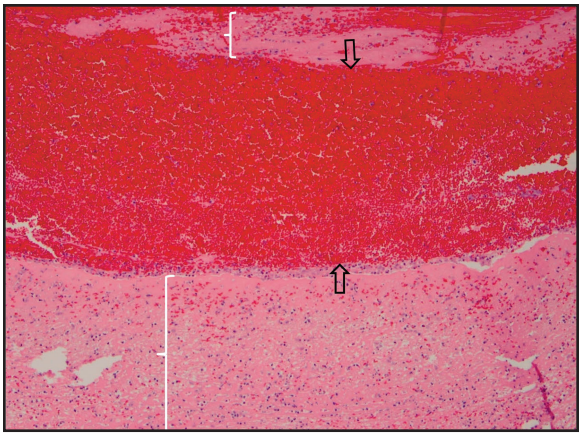

Figure 3. Abdominal aortic aneurysm — thrombus, microscopically (10x) shows mural thrombus consisting layers of red blood cells (between the block arrows) alternated with fibrin admixed with platelets (shown by brackets).

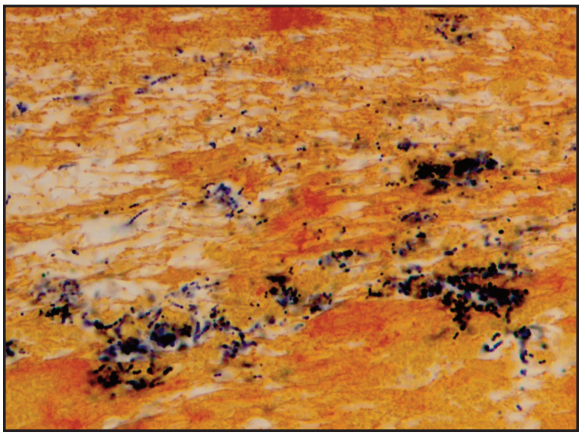

Figure 4. Abdominal aortic aneurysm — bacteria, tissue gram stain of thrombus (100x, oil immersion) shows gram positive cocci in pairs or chains consistent with Group B Streptococci.

positive for GBS, consistent with the diagnosis of mycotic AAA. Further laboratory evaluation showed that the causative GBS in this case was sensitive to penicillin G: minimum inhibitory concentration (MIC) <0.03mg/L. After culture results, patient's antibiotics were replaced with intravenous gentamicin (60 mg every 12 hours for 2 weeks) and intravenous ceftriaxone (2 g once daily for 2 weeks). He developed a granulocytopenic reaction that was attributed to ceftriaxone, and antibiotic treatment was changed to intravenous moxifloxacin (400 mg once daily, MIC: <1mg/L). After 6 weeks of parenteral antibiotics, suppressive long-term oral antibiotic therapy with moxifloacin (400 mg orally daily) was started. Blood cultures taken at the time of admission and during hospitalization were all negative. Magnetic resonance imaging of the lumbar spine did not show any evidence of diskitis and/or osteomyelitis.

Our patient has remained asymptomatic for more than 1 year. There are no signs of relapse of infection, and inflammatory markers are within normal limits (CRP: 0.3 mg/dl, ESR 16mm/hr).

DISCUSSION

Sir William Osler first described the term mycotic aneurysm in a landmark lecture to the Royal College of Physicians in 1885 [24]. He described the vegetations in the internal surface of the aorta as "fresh fungus" and hence coined the term "mycotic." Sir Osler postulated that embolization of organisms from bacterial endocarditis led to septic degeneration of the vessel wall, leading to formation of an aneurysm. Even though bacteria are causative agents in the majority of cases, eventually the term "mycotic aneurysm" became synonymous to describe all types of infected aneurysm. As it is a misnomer, it has been suggested that "infected aneurysm" is a better term [25].The mechanism is believed to be microbial arteritis as result of invasion of bacteria in a previously normal or damaged arterial wall [26]. Mycotic aneurysms can occur almost anywhere in the body. Abdominal aorta was found be the second most common site (31 percent), after femoral artery (38 percent) in one study [26].

There are several mechanisms that may describe pathogenesis of a mycotic aneurysm. In the pre-antibiotic era, septic emboli origi-

Table 1. Review of previously published case reports of GBS-associated AAA.

	Blackett et al. 1989	Andreasen et al. 2001	Chandrikakumari et al. 2007	Present Case
Age/Gender	61/Male	40/Male	69/Male	74/Male
Presenting complaints	Severe low back pain	Acute abdominal pain	Abdominal pain radiating to flanks	Back pain, low grade fevers, leg swelling
Suspected source of infection	Osteomyelitis of L2/L3	Unknown	Infected epidermal inclusion cyst	Unknown
Pyrexia (+/−)	+	+	-	+
Blood cultures	Negative	Negative	Not done	Negative
Vegetations on Echocardiography	Not seen	Not seen	Not seen	Not seen
Surgical treatment	Excision and grafting	Excision and Iliaco-femoral bypass graft	Excision and grafting	In situ reconstruction with rifampin-soaked graft
Graft material	Knitted Dacron	Unknown	Allograft	Dacron
Antibiotics and duration	Benzylpenicillin (2 wk) and erythromycin (8 wk)	Cefuroxime (4 wk)	Benzylpenicillin (2 wk), ceftriaxone (4 wk), and moxifloxacin (18 wk)	Gentamycin (2 wk), ceftriaxone* (2 wk) followed by moxifloaxin (4 weeks); Life-long suppression (Oral moxifloxacin)
Outcome	Cured	Cured	Cured	Cured

*Ceftriaxone was replaced with Moxifloxacin due to drug reaction.

nating from bacterial endocarditis was most common [27]. In our case, bacterial endocarditis was ruled out with repeated negative blood cultures at the time of admission and during hospitalization. No valvular vegetations were seen on transesophageal echocardiogram. Other causes of bacteremia such as recent dental work were also not present. Skin and soft tissue infections due to GBS could lead to bacteremia and remote hematogeneous seeding. No such infections were noted in the index case. Our patient does have a history of suspected bronchitis/exacerbation of chronic obstructive lung disease about a month prior to presentation. However, blood cultures were not taken during that admission. Any kind of arterial trauma, iatrogenic or self induced, may predispose the arterial wall to infection [28]. However, in our case, there is no history of injection drug use or an intervention such as percutaneous arterial puncture. Direct contiguous spread of local infection also may cause microbial invasion of the vessel wall [26,29]. In the index case, cross-sectional imaging with CT did not demonstrate any abdominal focus of infec-

tion. There was a concern for diskitis and/or osteomyelitis that could have spread locally to involve the aorta. A magnetic resonance scan of the lumbar spine did not show any evidence of diskitis and/or osteomyelitis, ruling this out as a cause of mycotic AAA. Diabetes, alcoholism, and malignancy that cause impaired immunity also have been identified as risk factors [30]. In our case, none of these risk factors were present. The source of infection is not found in most cases of mycotic aneurysm [18,25], and the same is true in our case.

The most common organisms causing mycotic aneurysms were *Streptococcus pyogenes*, *S. pneumoniae*, and staphylococci in the pre-antibiotic era [27]. With the wide availability of antibiotics, a shift has been noted in the microbiology of these infections. In the post antibiotic era, the most common causative organisms are the Salmonella species (up to 50 percent) and the *Staphylococcus aureus* [19]. Untreated Salmonella gastroenteritis could lead to invasive infection and involvement of the arterial wall [31] and has a predisposition for early rupture [32]. Other organisms such as *Listeria monocytogenes*, *Bacteroides fragilis*, and *Campylobacter fetus* have also been reported [12].

Antibiotics

For treatment purposes, we followed the recommendations for antimicrobial management of infective endocarditis, as mycotic AAA essentially is bacterial infection of the abdominal aortic wall [33]. It is recommended that patients receive 6 weeks of antimicrobial therapy with penicillin, cefazolin, or ceftriaxone. Some studies have recommended the addition of gentamicin to penicillin or a cephalosporin for at least the first 2 weeks of the 6-week course of antimicrobial therapy [34]. As per current recommendations, our patient received a total of 6 weeks of intravenous antibiotic treatment, detailed above in the case report section [33]. There are no definitive recommendations for the duration of the postoperative antibiotic therapy in published literature. In the previously published case reports of infected abdominal aortic aneurysms, the maximum duration of treatment was 18 weeks and all cases were cured [13,16,17] (Table 1). It has been suggested that it may be desirable to use long-term oral antibiotic suppression treatment in patients at high risk of recurrence of infection, such as those with interposed vascular grafts in infected areas [33]. A small animal experimental study also supported long-term antibiotic suppression and showed that total "graft sterility" cannot be expected in the case of an overwhelming bacterial challenge [35].

The fact that the patient received moxifloxacin prior to this admission for suspected bronchitis could have led to a better outcome. Multiple recent studies have shown that GBS is still highly sensitive to moxifloxacin [36]. Good outcome was also reported with moxifloxacin in another published case report [17].

Surgery

Surgical repair is the first-line treatment for an infected aortic aneurysm and may be done by a synthetic extra anatomic bypass or in situ reconstruction with a graft. There is limited evidence-based information that favors any one particular surgical approach over the other [37]. Earlier studies showed lower risk of infection with autologous vein grafts [38,39]; however, in situ reconstruction, which was employed in our case, has been the favored technique in recent studies [40]. Experimental studies in small animals have shown decreased adherence of bacteria to the graft and decreased incidence of graft infection if the grafts were soaked in rifampin solution prior to implantation [41,42]. The collagen-impregnated Dacron graft used for in situ reconstruction in this case was pre-soaked in rifampin solution.

CONCLUSION

Although mycotic AAA is rare, it should be considered in patients with fever of unknown origin, vague abdominal and back pain, and constitutional symptoms. Mycotic AAA with GBS is extremely rare in

the post-antibiotic era. There is a paucity of evidence-based human studies regarding optimal surgical and antibiotic management.

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