Case Report

Infective Endocarditis Due to *Abiotrophia defectiva* and Its Feared Complications in an Immunocompetent Person: Rare, But Real

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Abstract

*Abiotrophia defectiva* is nutritional deficient streptococci that cannot be cultured on routine culture medias. Even though fastidious in growth requirement, it is a virulent bacterium preferentially affecting endovascular structures and is implicated in many culture-negative endocarditis cases. Unlike other organisms, it is known for heart valve destruction leading to heart failure and excessive embolization rates. It’s inherent resistance to routinely used antibiotics also contributed to increased mortality and morbidity in affected individuals and warrants timely diagnosis and prompt treatment. Our patient, a previous healthy individual, acquired this rare bacterium from intravenous drug abuse and developed infective endocarditis with valve destruction, heart failure, and distal embolization to multiple organs. He underwent multiple surgeries including mitral valve replacement and embolectomy with clinical improvement. Our case reiterates the possibility of rare cause of common diseases and raises awareness of infective endocarditis caused by *A. defectiva* among medical professionals.

Keywords: *Abiotrophia defectiva*, acute limb ischemia, infective endocarditis, nutritional deficient streptococci

INTRODUCTION

Advances in medical technology have brought new insight into the pathophysiology of rare diseases. *Abiotrophia defectiva*, a variant of streptococci, was first described around five decades ago; however, it has been classified and reclassified many times in light of new information. In the recent past, it has been increasingly implicated in human diseases, particularly endocarditis, with grave complications. We present a similar case of infective endocarditis caused by *A. defectiva* in a healthy individual with native valves along with its feared complications.

CASE REPORT

A 31-year-old white male presented with dyspnea associated with generalized weakness and lethargy for the past 4 months. In the past month, his breathing had further worsened with episodes of syncope with exertion. He also reported an intermittent fever and had lost 40 pounds in 2 months. He smokes cigarettes and drinks alcohol regularly along with intravenous (IV) methamphetamine once or twice per week.

On presentation, he was febrile, tachycardic, and hypotensive and was admitted to the hospital for the management of septic shock. The examination showed a Grade 2 systolic murmur in the mitral area and tenderness of the upper abdomen. He was treated with IV fluids and broad-spectrum antibiotics as per protocol. Computed tomography (CT) of the abdomen showed geographical enhancement of the spleen suspicious of infarction, along with multiple small renal infarction. On the basis of these findings, infective endocarditis with embolization was suspected. Echocardiogram confirmed 2.5 cm × 1.5 cm vegetation in both mitral leaflets with evidence of severe mitral regurgitation [Figure 1].

While being worked up for mitral valve replacement, he suddenly developed numbness of his left arm. On examination, the left arm was pale with an absent radial pulse. Bedside, arterial Doppler revealed a possible...
obstruction of the proximal brachial artery with maintained blood flow distally. Meanwhile, his blood cultures grew *A. defectiva* (nutritionally deficient *Streptococcus* [NVS]) from both of the bottles [Figure 2]. Vascular surgery was consulted, and CT angiogram of the left upper extremity was performed, revealing an occlusion of the left brachial artery distal to the deep brachial branch point [Figure 3]. There was a reconstitution of flow distally within the ulcer artery, which was patent to the level of the wrist, and the radial artery did not become opacified until just proximal to the wrist joint, suggesting extension of the thrombus into the radial artery. The patient was initiated on anticoagulation therapy and underwent left brachial artery balloon embolectomy with extraction of two clots/vegetation, one approximately the size of a cashew nut and another the size of a peanut. The patient later underwent mitral valve replacement under pump. Both mitral valve leaflets appeared mushy and destroyed and the vegetation extended into the chordae tendineae closest to the papillary muscle and also into the left atrial wall [Figure 4]. The native valve was successfully replaced with an On-X33-mm prosthetic valve. *A. defectiva* was pan-sensitive to ceftriaxone, gentamycin, penicillin, and vancomycin; hence, treatment was tailored to IV penicillin G at 4 million units every 4 h and IV gentamycin at 1 mg/kg every 8 h for a total of 6 weeks. He improved symptomatically and was discharged home along with home antibiotics and anticoagulation therapy. At the follow-up appointment, he reported well-being and had gained 10 pounds of weight.

**Discussion**

*A. defectiva* is a NVS first described in the mid-19th century when colonies were observed only along *Staphylococcus* cross streak on blood agar, a phenomenon described as satellitism. These bacteria do not synthesize pyridoxine, L-cysteine, or other essential nutrients required for

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**Figure 1:** Transthoracic echocardiogram. Long axis parasternal view (a), magnified parasternal view (b), short axis parasternal view (c), apical four chamber view (d) shows vegetation in both mitral leaflets. Apical color flow image (e) and continuous wave Doppler (f) show severe mitral regurgitation

**Figure 2:** Growth of colonies of *Abiotrophia defectiva* in the chocolate agar plate (right side) compared to sparse grown in blood agar (left side) due to lack of nutrients such as pyridoxal phosphate

**Figure 3:** Computed tomography angiogram of left upper extremity. The proximal part of the left brachial artery is patent, but the distal part of brachial artery shows occlusion with possible thrombus (blue arrow in panel a-e and f). The sagittal reconstruction images show infarct in the spleen (yellow arrow in panel e)

**Figure 4:** Postoperative mitral valve specimen shows large vegetation in both mitral leaflets with almost complete destruction of native valve
growth and depend on other bacteria or enriched media to proliferate. Initially, they were classified with other NVS such as *Granulicatella*; however, with the advent of the use of 16s ribosomal RNA sequencing technology, they are classified into a separate genus.[1] *A. defectiva* is pleomorphic organisms appearing as Gram-positive cocci, coccobacilli, and bacilli forms depending on the culture media. They are nonsporulating, nonmotile, and facultative anaerobes.[1]

They can be detected by automated blood culture systems but do not grow in subcultures, unless the chocolate agar is enriched with pyridoxine or cysteine. They appear as gray-white nonhemolytic colonies. The rapid and accurate method of identification is 16s RNA gene amplification followed by polymerase chain reaction-restriction fragment length polymorphism.

*A. defectiva* is observed in the oral cavity or intestines of 11% of healthy individuals and implicated in 6% of all cases of infective endocarditis.[1] As of 2012, nearly 125 cases of infective endocarditis had been described in the literature.[4] They secrete exopolysaccharide and fibronecin and are able to bind to endovascular structures, leading to various complications, such as the destruction of valves and congestive heart failure, and can also cause septic embolization. Immunosuppression, pregnancy, and prosthetic valves are the common predisposing factors for this rare infection although infection in healthy individuals has been reported in the literature. Unlike infections caused by other streptococci, infective endocarditis by *A. defectiva* produces small vegetation, as well as embolisms in one-third of the cases.[4,5] The aortic valve is the most common valve affected followed by the mitral valve. The virulent infection can lead to destruction of the valve, warranting surgery in 30% of cases.[4,5] Despite being a rare infection, it can cause significant morbidity with mortality that can exceed 17%.[4,5] *A. defectiva* can also cause osteomyelitis, cerebral brain abscess, implantable cardioverter defibrillator lead infection, mycotic aneurysm, endophthalmitis, and septic arthritis.

*A. defectiva* is very sensitive to vancomycin, cephalaxine, and aminoglycosides; however, 90% of the isolates are resistant to penicillin.[6] The American Heart Association guidelines recommend treatment with penicillin and gentamycin for 4–6 weeks.[7]

Being a case report, this paper is limited by publication bias and overstressing of a rare phenomenon. Notwithstanding if the limitations, our case reiterates the possibility of rare causes of common diseases and increases the awareness of *A. defectiva*-related human diseases among medical professionals.

**Conclusion**

*A. defectiva* is a rare but important cause of infective endocarditis. It can rapidly lead to valve destruction causing heart failure and can also result in embolism in distal organs. With early diagnosis and treatment, grave complications can be prevented.

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**Conflicts of interest**

There are no conflicts of interest.

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