Infectious endocarditis cause by *Abiotrophia defectiva*

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**ABSTRACT**

We report a case of 61-year-old man presented with progressive dyspnea for 5 month followed by respiratory failure. Transthoracic echocardiogram revealed bacterial vegetation, rheumatic change mitral valve and destruction of the aortic valve. Initial hemoculture reported gram-positive cocci in chain for two specimens and its resistance to penicillin and vancomycin. Daptomycin was administered as a primary drug, but the latter hemoculture was identified as *Abiotrophia defectiva*. Infectious endocarditis caused by *A. defectiva* was diagnosed, he was treated with diuretic and penicillin G. Aortic valve replacement and valvulopathy of mitral valve was done due to congestive heart failure and large vegetation. After operation and total penicillin G 4 weeks, he improved and was discharged from the hospital. Infectious diseases caused by *A. defectiva* are extremely rare, and identification of this pathogen is important, as its bacterial characteristics require proper attention. (*J Infect Dis Antimicrob Agents 2012;29:21-5.*)

**Note:** This case was presented in the Interhospital Case Conference on Infectious Diseases (ICCID), 18 August 2011, Bangkok, Thailand.

**INTRODUCTION**

*Abiotrophia* species, first described as nutritionally variant (deficient) streptococci in 1961, were originally discovered as small satellite colonies around coagulase-negative bacteria or with supplementation of complex media with cysteine or pyridoxal. Taxonomic studies of the *Abiotrophia* spp. concluded that this group of organisms should be reclassified separately into *Abiotrophia defectiva* and *Granulicatella adiacens*, *G. balaenopterae*, and *G. elegans*. Although *Abiotrophia/Granulicatella* are parts of the normal flora of the oral cavity, the genitourinary tract, and the intestinal tract, the pathogenic potential of these organisms has been well established.

They have been estimated to cause approximately 5-6% of microbiologically proven cases of endocarditis.
and have likewise been implicated in the pathogenesis of culture-negative endocarditis.\textsuperscript{5} However, with current laboratory media and techniques, recovery of strains is no longer as significant a problem. Among NVS, \textit{A. defectiva} seems especially suited to cause endovascular infection because of its ability to adhere to fibronectin in the extracellular matrix.

\textit{Abiotrophia defectiva}, a nutritionally variant streptococcus (NVS), represents a rare but clinically important cause of infective endocarditis. Endocarditis caused by NVS carries greater morbidity and mortality than endocarditis caused by other streptococci and it has been reported to result in heart failure by destroying heart valves. Therefore, immediate treatment based on a correct diagnosis and identification of the pathogen have a bearing on the prognosis of patient with endocarditis caused by \textit{Abiotrophia defectiva}.

**CASE REPORT**

A 61-year-old Thai man, from Songkhla province, presented with progressive dyspnea for 5 months. The patient denied underlying disease and alcoholic drinking but he smoked approximately 30 pack-years. Five months prior to admission, he developed progressive dyspnea and follow by respiratory failure. He was admitted at a provincial hospital due to congestive heart failure caused by NSTEMI. He received intubation and was on a respirator for six days. After discharge from provincial hospital, despite good compliance of medication, he was still dyspnea so he came to our hospital for further investigation and treatment.

On examination, the patient was afebrile with a temperature of 37.5°C, regular heart rate of 100 beats/min, blood pressure of 120/60 mmHg, respiratory rate of 30 breaths/min, and room air oxygen saturation of 94%. Physical exam was significant for cardiac auscultation that was regular rate and rhythm with a 3/6 diastolic murmur heard at the right upper sternal border, 2/6 pansystolic murmur heard at apex, engorged neck veins, fine crepitation both lungs and nontender hepatomegaly (liver span 10 cm.). No Janeway’s lesions and Osler’s nodes. The eye examination revealed no Roth’s spots.

His initial laboratory studies were remarkable for a creatinine of 1.82 mg/dl, a normocytic anemia (hemoglobin 7.5 g/dl), white blood cells of 16,000/ mm\(^3\) (neutrophil 83%, lymphocyte 10%, monocyte 7%), and platelet of 154,000/mm\(^3\). No microscopic hematuria. Liver function test was total bilirubin 2.0 mg/dl, direct bilirubin 0.6 mg/dl, aspartate aminotransferase 285 IU/L, alanine aminotransferase 541 IU/L and serum albumin 2.9 g/dL. His chest radiograph showed increased cardiothoracic ratio and bilateral interstitial infiltration. His electrocardiograms showed sinus tachycardia 120/min, incomplete RBBB, left ventricular hypertrophy by voltage, ST depression and T inversion in V5, V6. Transthoracic echocardiograms revealed mobile vegetation at aortic valve 2.41 cm. with severe aortic regurgitation and moderate to severe mitral valve.

Initial hemoculture reported gram-positive cocci in chain two specimen and its resistance to penicillin and vancomycin. Daptomycin was administered as a primary drug. During admission, he developed respiratory failure from congestive heart failure. His clinical features suggested need for surgical intervention due to large vegetation and valvular dysfunction. Aortic valve replacement and valvuloplasty of mitral valve was done. The valve culture was negative. During the early postoperative period, his hemoculture was identified as \textit{Abiotrophia defectiva}. The organism was susceptible to penicillin with the minimum inhibitory concentration (MIC) for penicillin of 0.002 \(\mu\)g/mL. It showed technical error about initial procedure for drug susceptibility. Diagnosis of infective endocarditis was made using modified Dukes criteria.
and antimicrobial was changed from daptomycin to penicillin G sodium (PGS) 24 million units per 24 hr iv, divided q 4 hr total 4 weeks. No plus gentamicin due to acute kidney injury. Two month after surgical and antimicrobial therapy, he had no fever with marked improvement of clinical signs and symptoms without relapse or complication.

**DISCUSSION**

Infectious endocarditis caused by *Abiotrophia defectiva* has been rarely reported. *Abiotrophia* species were first described as nutritionally variant streptococci (NVS) in 1961, having been found to grow as satellite colonies around those of helper bacteria (staphylococci) (Figure 1) and later, in media supplemented by sulhydryl compounds, pyridoxine, or cysteine.1,6,7

More than 100 cases of NVS endocarditis have been reported in the literature.8-16 NVS strains are part of the normal oral, intestinal and genitourinary flora. The portal of entry in most cases of endocarditis is via the mouth, teeth or throat. NVS cause approximately 3% to 5% of cases of streptococcal infective endocarditis. Despite a considerable number of published cases worldwide, this is the first case report from Thailand. Because *Abiotrophia* spp. are fastidious organisms, it is likely that most cases are misdiagnosed as culture-negative endocarditis; therefore, their role in endocarditis may be underestimated. The present case highlights the importance of the correct microbiological diagnosis. *Abiotrophia defectiva* has many bacterial characteristics, including (1) vitamin B6 and L-cysteine are required for growth, thus this bacteria cannot grow in culture plates without these nutrients, but can grow in chocolate agar. (2) *Abiotrophia defectiva* was originally considered coccobacillus, but its appearance differs depending on various culture conditions.17 (3) *Abiotrophia defectiva* grows more slowly than other streptococci18 and this may be one of the reasons for the difficulty in identification. (4) Higher affinity with the endocardium19,20 because of its ability to adhere to fibronectin in the extracellular matrix.

Our patient did not have any underlying cardiac or immunosuppressive illness and did not undergo dental manipulations. The portal of entry

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*Figure 1. Abiotrophia spp. (formerly known as nutritionally deficient streptococci) showing satellite growth with Staphylococcus aureus.*
of bacteria into the endovascular system remains obscure. However, the observed poor dental hygiene might have facilitated the process towards infectious endocarditis.

To our knowledge, for the treatment of endocarditis caused by *Abiotrophia defectiva*, a sufficient dose of effective antimicrobial agent should be administered for an adequate period of time. Published reports suggest penicillin G and gentamicin as effective empiric of choices of antimicrobial agents for patients with endocarditis caused by this pathogen.\textsuperscript{10,12} Antimicrobial treatment is the initial step of therapy but surgical intervention is inevitable in at least half of the cases. In our case, only penicillin G was given because he had acute kidney injury, aortic valve replacement and valvuloplasty of mitral valve was done. Two months after surgery he was doing well without relapse or complication.

In conclusion, we have reported a case of infectious endocarditis caused by *Abiotrophia defectiva*. It is an important agent causing infectious endocarditis with increased morbidity and mortality. Prompt attention to correct identification of this pathogen and surgical treatment of heart valves must be given careful attention by the attending physicians.

References