



Infective Endocarditis Caused by *Abiotrophia defectiva* in a Pediatric Patient

Bir Çocuk Olguda *Abiotrophia defectiva*'nın Etken Olduğu Enfektif Endokardit

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Abstract

Abiotrophia defectiva is a nutritional defective streptococcus that is difficult to grow in routine culture media, is found in the oral mucosa, gastrointestinal and urogenital system flora and is a rare cause of infective endocarditis. An 11-year-old boy with underlying operated congenital heart defect presented with prolonged fever. *Abiotrophia defectiva* is yielded in the blood cultures taken properly with a pre-diagnosis of infective endocarditis. Although there was no vegetation or other finding of endocarditis on echocardiography, surgery decision was made because bacteria continued to grow in the blood culture under appropriate antibiotic treatment. During surgery, it was observed that the conotruncal valved conduit was severely degenerated and calcified. A new conotruncal valve conduit replacement was performed.

Serious complications occur more frequently in infective endocarditis caused by *Abiotrophia defectiva* compared to other etiologic agents of infective endocarditis. With this case, we wanted to emphasize that in *Abiotrophia defectiva* infective endocarditis, the possibility of antibiotic treatment failure and surgical procedure requirement is quite high.

Keywords: *Abiotrophia defectiva*, infective endocarditis, child, conduit

Öz

Abiotrophia defectiva rutin kültür ortamında üretilmesi zor bir nutrisyonel defektif streptokoktur; oral mukoza, gastrointestinal ve ürogenital sistem florasında bulunan bir bakteridir ve enfektif endokarditin nadir bir etkenidir. Altta yatan opere konjenital kalp defekti olan 11 yaşında erkek çocuk uzamış ateş şikayeti ile başvurdu. Enfektif endokardit ön tanısı ile uygun şekilde alınan kan kültürlerinde tekrarlayan *Abiotrophia defectiva* üremeleri oldu. Ekokardiyografide vejetasyon ya da endokarditin başka bir bulgusu olmamasına karşın, uygun antibiyotik tedavisi altında kan kültüründe bakteri üreme devam etmesi sebebiyle cerrahi kararı alındı. Cerrahi sırasında hastada mevcut pulmoner kapaklı konduitin ileri derecede dejenere ve kalsifiye durumda olduğu görüldü. Mevcut kapak çıkarılarak, yeni bir pulmoner kapaklı konduit replasmanı yapıldı. *Abiotrophia defectiva*'nın yol açtığı enfektif endokarditte ciddi komplikasyonlar diğer etkenlere oranla daha sık ortaya çıkmaktadır. Bu olgu ile *Abiotrophia defectiva* enfektif endokarditinin antibiyotik tedavisi ile düzelmeme ve hastanın cerrahiye gitme ihtimalinin oldukça yüksek olduğunu vurgulanmak istenmiştir.

Anahtar Kelimeler: *Abiotrophia defectiva*, enfektif endokardit, çocuk, konduit

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Introduction

Infective endocarditis (IE) is one of the most feared complications of congenital or acquired heart diseases. Along with the typical involvement of natural or prosthetic valves, it may also be seen in cardiac defects, intracardiac patches or artificial vessels used as shunt (1). The agents vary depending on the development level of the country and underlying valve pathologies of the patient, but the most common ones are streptococci, staphylococci, and enterococci (2,3). *Abiotrophia defectiva* has been rarely reported among IE agents. *A. defectiva* is a nutritional defective streptococcus that is difficult to grow in routine culture. Therefore, it is ranked among culture-negative IE agents. It is known that it causes more severe infections compared to other streptococci. *A. defectiva* is found in the oral mucosa and in gastrointestinal and urogenital system flora (3,4). Here, it was aimed to report a case of prosthetic valve endocarditis caused by *A. defectiva* in a child with an underlying operated congenital heart defect.

Case Report

An 11-year-and-4-month-old male patient presented to our hospital with fever that had started 19 days ago. In a health center the patient applied to with fever 9 days prior, the patient had been started on amoxicillin-clavulanate treatment with a preliminary diagnosis of tonsillitis, but the fever had persisted. Patient history revealed that he had undergone surgical repair with pulmonary valved conduit due to truncus arteriosus when he was 34-days old, and at age 2 years, the patient had also undergone conduit replacement surgery. Physical examination revealed an incision scar secondary to the operation on his anterior thoracic wall and 3/6 pansystolic murmur heard from all foci. It was learned that the cardiac murmur was the same in his former clinical examinations. Laboratory results of the patient was as follows: leucocyte count: 8.000/mm³ (4.000-10.000/mm³), hemoglobin 10.0 gr/dL (11-18 gr/dL), mean erythrocyte volume (MCV): 79 fL (80-100 fL), red blood cell count: 3.75 x 10³/µL (3.5-6.1 x 10³/µL), platelet count: 238.000/mm³ (142.000-424.000/mm³), lactate dehydrogenase (LDH): 647 IU/ml (125-248 IU/ml), C reactive protein (CRP): 73 mg/L (0-5 mg/L), and erythrocyte sedimentation rate (ESR): 26 mm/h (0-20 mm/h). Complete urinalysis was normal. Transthoracic echocardiography reported truncus arteriosus corrected with conduit, conduit narrowness, conduit deficiency (first to second degree), aortic regurgitation (first to second degree), tricuspid regurgitation (first degree), and no change in valve leakages compared to the echocardiography taken 1 month ago. Without any other focus for fever, the patient was hospitalized with a preliminary diagnosis of infective endocarditis (IE). Blood culture was taken from 3 different areas on two extremities. The patient, who was followed without antibiotics, had fever every day, and blood culture was

sent on each day. Upon detecting a signal on all blood cultures on the fourth day of his hospitalization, vancomycin (60 mg/kg/day), gentamycin (5 mg/kg/day), and ampicillin/sulbactam (200 mg/kg/day) treatments were started considering IE. The fever dropped on the second day of antibiotic treatments. All growths in the blood culture were reported as *A. defectiva* on the sixth day of hospitalization. Antibiogram showed that it was erythromycin-resistant but sensitive to penicillin, levofloxacin, rifampin, amoxicillin, teicoplanin, vancomycin, linezolid, and ceftriaxone. Vancomycin treatment was stopped as a result of this antibiogram. The same microorganisms with the same antibiogram continued to grow in blood cultures taken tridaily; however, the fever of the patient did not occur again. Vegetation was not detected on transthoracic echocardiography which was repeated due to persisting blood culture growths. Conduit replacement was decided on since growth continued on the 18th day of hospitalization. During the operation, it was seen that the pulmonary valved conduit (Contegra[®]) used in previous surgery was severely degenerated and calcified, valve opening was 3-4 mm, and vegetation was not present on the valve. The conduit was resected, and right ventricle pulmonary artery continuity was provided with pulmonary valved conduit. Patient's complaints did not continue in outpatient follow-up visits.

Discussion

Bacteria of the *Abiotrophia* family were first defined by Frenkel and Hirsch as nutritional variant streptococci in 1961. In 1989, they were divided into two groups as *Streptococcus defectivus* and *Streptococcus adiacens* performing DN-ADNA hybridization by Bouvet and colleagues. In 1995, two species including *Abiotrophia defectiva* and *Abiotrophia adiacens* were defined by Kawamura as a result of 16S rRNA sequencing comparison (3, 4). They are in the shape of Gram-positive pleomorphic cocci or coccobacilli, and may be stained like gram-negative bacilli, gram-negative cocci and even fungi in suboptimal conditions (5,6). Together with leading to endocarditis in individuals with underlying valve diseases (61-90%), *A. defectiva* may also cause endocarditis in individuals with no prior valve disease (7-9).

A. defectiva is responsible for 4.3-6% of all streptococcal endocarditis (6). In endocarditis cases in which *A. defectiva* is the agent, treatment failure and mortality have been reported as 41% and 17%, respectively. Generally, its mortality is higher compared to endocarditis caused by viridans streptococci and other agents (0-12%), and clinical course is more insignificant. The reason why mortality is higher is because serious complications such as acute heart failure, severe valve damage, cardiogenic shock, and multiorgan failure related to septic embolism and systemic emboli develop more frequently in IE caused by viridans streptococci (3,5,10,11).

It has been reported in many published studies that despite appropriate antibiotic treatment, treatment failure rate is high (41%), relapse can be seen, and serious valve damage develops while under sensitive antibiotic treatment in endocarditis caused by *A. defectiva* (1-7,10,12). It has been reported that prosthetic valve replacement is required in 48% of the cases (7). Along with patients needing surgery since the infection could not be taken under control, there are also cases in the literature requiring surgical intervention due to severe valve damage in the follow-up period despite yielding negative culture results while on antibiotic treatment and terminating the necessary duration of antibiotic treatment (13). The reason for *A. defectiva* to more frequently cause complications and lead to surgical intervention compared to other agents is considered to be due to its capability to adhere to endothelia cells and immobilized fibronectin with high affinity (9).

In the European and American Heart Association guidelines, the endurance of culture growths despite appropriate antibiotic treatment is an indication for surgery (14,15). Although vegetation or conduit destruction and calcification were not seen on echocardiography in our patient, bacterial growth in blood cultures led us to surgical treatment. Despite having had similar results with previous echocardiographies, it was seen during surgery that the conduit was severely damaged and calcified. Just as having caused the severe damage seen in the conduit in our patient, severe damage and calcification of the conduit may have caused the bacteremia to endure by preventing sufficient penetration of the antibiotics.

To conclude, severe complications in IE caused by *A. defectiva* occur more frequently compared to other IE microorganisms. The possibility of obtaining no recovery despite appropriate and timely antibiotic treatment in IE caused by *A. defectiva* and the necessity of surgical intervention are rather high. In this study, it was aimed to emphasize that clinicians should keep surgical intervention mind in cases where *A. defectiva* is detected as the IE agent.

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