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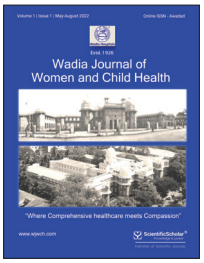

Case Report

Abiotrophia defectiva: A rare and lethal but an easy miss!

S. Prakruthi[1], Jayashree Mishra[2], Disha Sharma[3], Zahabiya Nalwalla[4]

Departments of [1]Pediatrics, [2]Pediatric Cardiology, [3]Microbiology, [4]Pediatric Infectious Diseases, Bai Jerbai Wadia Hospital for Children, Mumbai, Maharashtra, India.

***Corresponding author:**
S. Prakruthi,
Department of Pediatrics,
Bai Jerbai Wadia Hospital
for Children, Mumbai,
Maharashtra, India.

prakruthipraksu.amu@gmail.com

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ABSTRACT

Abiotrophia defectiva is a nutritionally deficient streptococci, a rare pathogen of infective endocarditis (IE) in children but is frequently known to cause valvular dysfunction and embolic complications. Only nine cases of *A. defectiva* endocarditis have been reported so far in world literature. We report a case of *A. defectiva* endocarditis in a 7-year old girl, with a small peri membranous ventricular septal defect with congenital bilateral sensorineural hearing loss status post left-sided cochlear implantation who presented with prolonged fever. *A. defectiva* should be considered in children with endocarditis with a subacute course and atypical presentations. Although fastidious in growth it is a virulent bacterium preferentially affecting endovascular structures and is implicated in many culture negative endocarditis. Our case reiterates the possibility of a rare cause of this common disease and the challenges encountered while isolating this nutritionally deficient organism and its treatment.

Keywords: *Abiotrophia defectiva*, Endocarditis, Ventricular septal defect, Congenital *cytomegalovirus* infection

INTRODUCTION

Abiotrophica defectiva is a Gram-positive coccus which is a normal flora of oropharynx, genitourinary and intestinal mucosa.[1] The organism grows as satellite colonies around other bacteria and requires a complex medium supplemented with pyridoxine or cysteine to grow. The taxonomy of this organism has been repeatedly revised, and with 16S ribosomal (rRNA) gene sequencing in 1995, the organism was placed in the new genus *Abiotrophia*.[2] Endocarditis due to *A. defectiva* is quite rare, accounts for around 6% of streptococcal endocarditis, and some cases of culture negative endocarditis.[3] Only nine cases of pediatric *A. defectiva* infective endocarditis (IE) cases have been reported till date.[4] *A. defectiva* should be considered in children with endocarditis with a subacute course and atypical presentations. Even though fastidious in growth requirement, it is a virulent bacterium preferentially affecting endovascular structures, and is implicated in many culture negative endocarditis, and is frequently known to cause valvular dysfunction and embolic complications.

CASE REPORT

A 7-year-old girl, with small peri membranous ventricular septal defect, presented with prolonged fever of 4 months duration. The fever was continuous, moderate to high grade and

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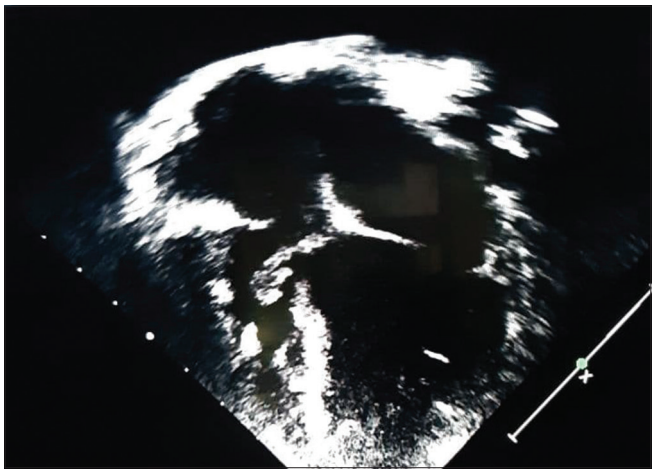

Figure 1: Echocardiography showing large vegetation 16*7 mm attached to tricuspid leaflet.

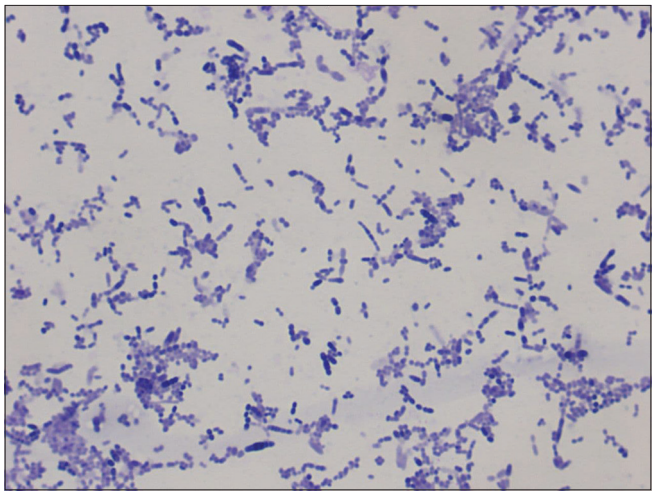

Figure 2: Gram staining -gram positive cocci and coccobacilli in chains.

Figure 3: Chocolate agar with abundant growth after 24 h of incubation.

not associated with chills/rigors or rash. She had congenital bilateral sensorineural hearing loss and had undergone left cochlear implantation at 5 years of age. She had good oral hygiene and there was no previous history of sore throat, dental procedures, or any history suggestive of congestive cardiac failure. Prior to presentation, she had visited multiple physicians and received multiple systemic antibiotics. Blood investigations suggested marginally raised leukocyte count, increased erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), and blood cultures done at three different times were sterile. Kochs workup was negative.

At admission, she was hemodynamically stable and had pallor. There were no peripheral signs of infective endocarditis (IE). Cardiovascular system examination revealed a precordial bulge, apex beat in left 5th intercostal space, 1 cm lateral to mid clavicular line, grade V pansystolic murmur best bheard in the left lower sternal border. The abdomen examination was normal. Central nervous system examination was normal with no focal neurological deficits.

She had a normal complete blood count, with raised CRP of 29 mg/dl and ESR of 100 mm at 1 hr, normal electrolytes, and renal function test. A transthoracic echocardiogram revealed a large vegetation with multiple strands attached to the anterior and posterior leaflets of tricuspid valve, measuring 16*7 mm [Figure 1]. A small peri membranous ventricular septal defect with the left to right shunt and mild-to-moderate tricuspid regurgitation was present with dilated left atrium and left ventricle. The biventricular function was normal.

Three sets of blood cultures were sent from different sites at intervals of 1 h, which were incubated in an automated blood culture system and the child was empirically started on injectable ampicloxacillin and amikacin. Since amikacin affects the outer hair cells, which are already non-functional in congenital profound hearing loss, and our child already had cochlear implant *in situ*, amikacin was continued for gram negative cover. On day 6 of admission, all blood cultures came positive with a time to positivity of 14 hours approximately. Gram stain smear was prepared from the bottles, which showed gram positive coccobacilli, which were pleomorphic [Figure 2]. Streaking was done on 5% sheep blood agar, chocolate agar and MacConkey agar plates. Blood and chocolate agar plates incubated in 5–10% carbon dioxide for 24 hours showed growth. The growth on chocolate agar [Figure 3] was abundant as compared to that of blood agar; hence, the blood agar plate was further incubated for 24 h. A smear made from the plate confirmed Gram-positive coccobacilli showing pleomorphism. The colonies were put for identification on Vitek 2 compact. All cultures grew *A. defectiva* which showed the same sensitivity pattern i.e., sensitive to penicillin, vancomycin, ceftriaxone, clindamycin, chloramphenicol, and resistant to cefotaxime and levofloxacin.

She was started on combination therapy of vancomycin and ceftriaxone according to the sensitivity pattern on day 6 of admission. The ophthalmic examination was normal. Repeat blood culture after 14 days of intravenous antibiotics showed no growth. Repeat 2D Echo after 21 days of treatment with sensitive antibiotics showed a significant reduction in the size of vegetation, 2D Echo on day 42 of antibiotic was normal with no evidence of vegetation. She did not develop any complications during the hospital stay. She was referred to the cardiothoracic team for VSD closure, after completion of 6 weeks of intravenous antibiotics.

DISCUSSION

A. defectiva is a Gram-positive, non-motile, and facultative aerobe. It is a normal flora of the oropharyngeal, genitourinary tract, and intestinal mucosa. It grows as satellite colonies around other bacteria and requires a complex medium supplemented with pyridoxine or cysteine to grow. Taxonomy of this organism has been repeatedly revised, and with 16S rRNA gene sequencing in 1995, it has been placed in the new genus *Abiotrophia*. The definition of *Abiotrophia* is "life nutrition deficiency" meaning the species needs supplemented media for growth and survival.[5] In our case, all blood cultures grew the same organism with the same generation time of 14 hours and had the same antibiotic sensitivity pattern. There was sparse growth on blood agar at the end of 24 hours, while chocolate agar showed good growth at the end of 24 hours.

IE due to *A. defectiva* has been rarely reported in children. A total of nine cases of *A. defectiva* IE have been reported in medical literature since its change in nomenclature in 1995. Three of the nine cases (33.3%) had underlying congenital heart disease. Most of the patients present with subacute course, with prolonged fever or atypical symptoms, such as abdominal pain, chest pain, calf pain, and arthralgia. The left-sided valves are more affected (55.5%) than right-sided valves. Most children have complications (88.9%) such as septic pneumonia, cerebral mycotic aneurysm, splenic infarction, congestive heart failure, valve destruction, and embolization. Rate of complications are more in children than in adults. More than half of the patients (66.7%) require surgery for recurrent embolic events, persistent large vegetation, or valvular dysfunction.[4]

A. defectiva is highly infective and has high binding capacity to extracellular matrix proteins. It produces large amounts of exopolysaccharides which have a major role in biofilm formation.[6] Delayed diagnosis due to subacute nature and difficulty to culture organism, adds to the challenging clinical course. *A. defectiva* endocarditis is generally treated with beta lactam or vancomycin plus gentamycin due to the difficulty in eradicating the organism. Penicillin and gentamicin have been historically used in the treatment, yet according to a recent study only about 10% are susceptible to penicillin, while 95–100% are susceptible to third generation cephalosporins. The American Heart Association guidelines recommend treatment for 4–6 weeks.[7]

CONCLUSION

A. defectiva is a rare but an important cause of IE. It should be considered in children with subacute clinical course and atypical presentation. It can rapidly lead to large vegetations, valve destruction causing heart failure, and embolization. Therefore, it is prudent to have an early diagnosis – additional testing with supplemented media, and treatment with appropriate antibiotics, so that surgery and grave complications can be averted.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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