

# *Abiotrophia defectiva* endocarditis in a child – a case report

## *Abiotrophia defectiva* kao uzročnik endokarditisa u djeteta – prikaz bolesnika

Lorna Stemberger Marić<sup>1,2</sup>

Nina Krajcar<sup>1</sup>

Goran Tešović<sup>1,3</sup>

<sup>1</sup>University Hospital for Infectious Diseases  
„Dr. Fran Mihaljević“ Zagreb

<sup>2</sup>School of Dental Medicine,  
University of Zagreb

<sup>3</sup>School of Medicine, University of Zagreb

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### Abstract

We present a case of endocarditis due to *Abiotrophia defectiva* in a child, developed as a complication of a previously unrecognized patent ductus arteriosus (PDA). The patient was successfully treated with antimicrobial therapy and later underwent transcatheter closure of PDA. As the organism may not be isolated in routine culture media, and may present with atypical clinical symptoms, a high index of suspicion should be maintained in children with subacute symptoms even with no underlying heart disease. Even patients with early appropriate antibiotic therapy and rapid achievement of bacteriological clearance remain at high risk for embolic and immune-mediated complications and irreversible valvular damage. Our case highlights the importance of careful interpretation of serologic tests and the value of good physical examination (auscultation!) in order not to miss the diagnosis of congenital heart defects.

### Sažetak

U radu prikazujemo slučaj djeteta s endokarditisom čiji je uzročnik *Abiotrophia defectiva*, a koji je nastao kao komplikacija prethodno neprepoznatog otvorenog Botallovog duktusa. Dijete je uspješno liječeno antimikrobnim lijekovima nakon čega je provedeno i transkatetersko zatvaranje srčane greške. S obzirom na to da se ovaj uzročnik teško izolira na rutinskim bakterijskim podlogama i može uzrokovati atipične simptome, potreban je poseban oprez čak i u one djece koja nemaju podležeću srčanu bolest, ali se klinički prezentiraju sa subakutnim simptomima. Imunološke komplikacije, embolizacije i trajno oštećenje zalistaka često se javljaju čak i u onih bolesnika s pravovremeno započetim ispravnim antibiotskim liječenjem i kod kojih je brzo postignut terapijski učinak antibiotika. Također ističemo važnost pažljive interpretacije seroloških testova kao i kvalitetnog fizičkog pregleda (auskultacije!) zbog mogućnosti propuštanja dijagnoze prirodene, dotad neprepoznate, srčane greške.

## Introduction

The spectrum of childhood infective endocarditis (IE) has been changed during the past several decades, largely because of the declining incidence of rheumatic heart disease and the increasing success in the treatment of patients with congenital heart disease (CHD). Among children with underlying CHD, *Streptococcus* and *Staphylococcus spp* are equally and the most frequently isolated pathogens, while in patients with structurally normal heart, *Staphylococcus aureus* causes approximately 50% of cases [1]. *Abiotrophia defectiva* is a form of nutritionally variant Streptococci (NVS) and causes about 4% to 6% of all streptococcal endocarditis [2].

We present a case of endocarditis due to *Abiotrophia defectiva* in a child, developed as a complication of a previously unrecognized patent ductus arteriosus (PDA).

## Case report

A 5.5-year-old, previously healthy girl, presented to her primary care pediatrician with several days history of high grade fever and fatigue. Due to suspected acute otitis media, empiric antibiotic therapy with cefixime was initiated and fever resolved promptly after one day of treatment. Soon after discontinuation of therapy she became subfebrile again and as her symptoms persisted for the next 2 weeks, she was examined in a regional hospital.

Laboratory check-up was done and due to positive CMV IgM (1.82) and negative IgG (0.32) antibody accompanied with hepatosplenomegaly on physical examination, acute CMV infection was diagnosed. Since fever persisted and her fatigue was worsening after almost 5 weeks of illness, she was diagnosed with fever of unknown origin and transferred to our institution for further diagnostic procedure. Laboratory results at that time revealed slightly elevated ESR of 30 mm/h, CRP of 15 mg/L and normal leukocyte count with mild neutrophilia (70%, ANC 4900/mm<sup>3</sup>). Repeated CMV serology came completely negative. On physical examination at admission she was subfebrile (T<sub>ax</sub> 37.5°C) and pale, but other vital signs were normal. A pansystolic murmur grade two of six (II/VI) was heard along the left sternal border and her liver and spleen were both palpable for 3 cm below the costal margin. Laboratory results showed normal ESR (10 mm/h), slightly elevated CRP (14.2 mg/L), normal leukocyte count, slightly decreased haemoglobin level (103 g/L) and prothrombin time (0.65) accompanied with hypergammaglobulinemia (15.0 g/L). Autoimmunity markers (ANA, ANCA, dsDNA, RF, CCP) were all negative, as well as serology to *B.henselae* and *B.quintana*. Chest X-ray was normal, except mildly enlarged heart shadow with prominent left heart contour. Abdominal ultrasound confirmed mild hepatosplenomegaly (liver 8.5 cm, spleen 12 cm). Transthoracic echocardiogram was performed and showed opened PDA, 4 mm wide in diameter, with left to right flow (4 mm/s) restrictive on pulmonary end. Regurgitation was mild and there were no signs of pulmonary hypertension; right heart chambers were only mildly enlarged. No intracardiac vegetations were seen. Three blood cultures were drawn during the first 48 hours of hospitalization and all yielded *Abiotrophia defectiva*. The isolate was found to be sensitive to penicillin, ampicillin, ceftriaxone, vancomycin and linezolid. Dual parenteral antimicrobial therapy consisting of ampicillin (300 mg/kg/day divided q6h) and gentamicin (7 mg/kg/day divided q8h) was initiated. After five days ampicillin was switched to ceftriaxon (100 mg/kg/day q12h) and gentamicin was continued. After 18 days, the parenteral therapy was switched to oral cefpodoxime-proxetil (10 mg/kg/day q12h) and the patient was discharged home. The cefpodoxime-proxetil therapy lasted for 4 weeks. The patient's later clinical course was favourable. She became afebrile within two days from antimicrobial therapy initiation, with gradual complete regression of hepatosplenomegaly and normalization of laboratory findings. All blood cultures taken under antimicrobial therapy remained sterile. After hospital discharge, sanitation of two caries lesions was done. Cardiologist indicated transcatheter closure of PDA which was done after treatment completion.

## Discussion

Since the prevalence of rheumatic heart disease significantly declined within the past two decades, CHD has

become the predominant underlying condition in children diagnosed with IE living in developed nations. In fact, an increase in the number of IE cases associated with CHD has been observed due to the fact that most patients with CHD survive much longer than several decades ago [1].

The oral mucosa and tooth surfaces of children who are beyond infancy are populated by a variety of pathogenic and nonpathogenic bacteria, which are representative of hundreds of strains of aerobic and anaerobic species. *A. defectiva* is found under normal conditions in the oral cavity, gastrointestinal tract, and genitourinary system. It has been involved in ocular infections—endophthalmitis or keratitis, otitis and paranasal cavities infections, osteo-articular infections, infections following arthroplasty, cerebral and pancreatic abscesses and iatrogenic meningitis [2,3]. IE caused by this organism usually is subacute in presentation, as it was in our patient.

A PDA that persists beyond one month of age is estimated to occur in 0.3–4 per 1000 live births, and accounts for approximately 10% of all CHD. It has been estimated that one-third of adults with unrepaired, hemodynamically significant PDAs will die of heart failure, pulmonary hypertension, or endocarditis by the age of 40, and this figure rises to two-third by age 60. In the preantibiotic era, IE was the single most common cause of death in PDA patients (42–45% of deaths) [4]. Between 1939 and 1971, five case series were published characterizing the association between IE and PDA. The majority of authors concluded that IE is rare in patients with PDA, but that when it does occur, surgical PDA ligation should be performed [2–5].

*Abiotrophia defectiva* endocarditis has been rarely reported in children. *Chang et al* reviewed 13 cases of IE caused by NVS in children reported in English literature since 1971. Nine of these patients (69%) had underlying heart disease [2].

In comparison to beta-hemolytic streptococci (BHS), NVS demonstrate different antimicrobial susceptibility. NVS susceptibility to penicillin (8% according to some studies), carbapenems, cephalosporins, daptomycin, or macrolides is not certain [5]. Nevertheless, the isolate from our patient had good antimicrobial susceptibility.

Several studies in adults have reported high rates of complications in *Abiotrophia* endocarditis including bacteriological failure rate up to 40%, fatal congestive cardiac failure in 17–20% of them, and embolization occurred in up to 27% of cases. This has been attributed to difficulty in isolating this organism and high drug resistance rates [2,3,5]. In contrast, our patient achieved relatively rapid bacteriological clearance, good clinical outcome and no relapse in the 12 months follow-up period (to date). However, it must be noted that even in patients with early appropriate antibiotic therapy and rapid achievement of

bacteriological clearance, these patients remained at high risk for embolic and immune-mediated complications and irreversible valvular damage.

As the organism may not be isolated in routine culture media, and may present with atypical clinical symptoms, a high index of suspicion should be maintained in children with subacute symptoms even with no underlying heart disease. Our case highlights the importance of careful interpretation of serologic tests, always in conjunction with clinical findings, in order not to miss proper diagnosis due to cross reactivity of serology. Also, even in the 21st century, we point out the value of good physical examination (auscultation!) not to miss the diagnosis of congenital heart defects.

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