Cardiology in the Young

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A rare paediatric case of possible infective endocarditis caused by *Streptococcus anginosus*

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Brief Report

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Abstract

Infective endocarditis in children is a rare but serious condition that requires prompt management. We present a case of an 11-year-old boy with subacute bacterial endocarditis caused by *Streptococcus anginosus,* an unusual causative microorganism for infective endocarditis. The patient presented with a history of malaise, fatigue, and one subjective tactile fever. To the best of our knowledge, this is the first paediatric case of possible infective endocarditis caused by *Streptococcus anginosus*.

Background

Infective endocarditis is a life-threatening condition with relatively low incidence in adult and paediatric populations.[1] In children, infective endocarditis is linked with CHDs and corresponding surgical repair, with an overall mortality risk of 5–10%.[2–4] The presentation of infective endocarditis is greatly variable, commonly involving fever and new-onset cardiac murmurs with gram-positive bacteraemia.[4] In this case report, we present an interesting case of a young boy who was diagnosed with possible infective endocarditis caused by *Streptococcus anginosus*.

Case presentation

An 11-year-old boy presented to the emergency department with a 3-to-4-week history of malaise, diminished appetite, nausea, and new-onset "bad taste." He appeared generally pale, with increased irritability and somnolence. History was obtained from his father, who was predominantly concerned with his son's mood and appetite changes, with vague recollection of one subjective tactile fever 2 days prior to presentation.

The patient had a restrictive moderate perimembranous ventricular septal defect, bifid thumb on his left hand, and mild developmental delay. He was treated for lobar pneumonia earlier in the year. His family had emigrated from Pakistan 8 years ago, with no international travel in the last 4 years. Family medical history was not significant.

The patient was tachycardic on presentation (131 beats/minute), blood pressure was 101/60 mmHg, temperature was 37°C, and respiratory rate was 24 breaths/minute with an oxygen saturation of 100% on room air. His weight was 42.7 kg, with no recent fluctuations. Physical examination revealed moist mucus membranes with conjunctival pallor. A pansystolic ejection murmur was auscultated at the left sternal border, in the fifth intercostal space, graded as 4/6 severity with a palpable thrill, with no signs of heart failure. There were no rashes, Janeway lesions, Osler nodes, or splinter haemorrhages.

Investigations/treatment

The patient's initial workup revealed haemoglobin 84 g/L (115–155), mean corpuscular volume 58.7 fL (77–95), leukocytes 16.9×10^9/L (4.5–13.5) with neutrophils 12.3×10^9/L (1.8–8.0), reticulocyte haemoglobin 20.1 Picogram (33–40.2), and C-reactive protein 68.6 mg/L (<10). Follow-up bloodwork continued to reveal low haemoglobin and mean corpuscular volume. There were low iron levels and decreased transferrin saturation. An abdominal ultrasound showed hepatosplenomegaly with no infarcts. Fundoscopic examination was negative for Roth spots.

The patient was started on intravenous ceftriaxone. His initial blood culture was positive for *Streptococcus anginosus* after a 2.4-day incubation. A transthoracic echocardiogram showed abundant tricuspid tissue with multiple echogenic densities located on aneurysmal and subvalvular tissue involving mobile extensions flailing in the right ventricular cavity and outflow tract (please see Fig 1). These findings were not visible on a transthoracic echocardiogram completed 6 months prior, making it difficult to distinguish their presence as a manifestation

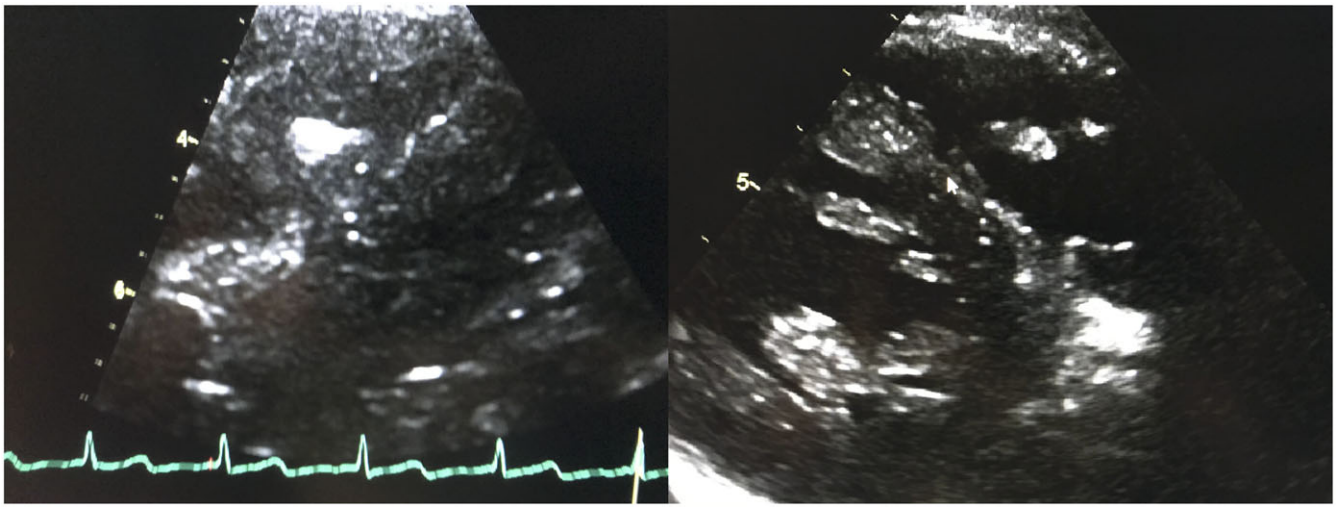

Figure 1. Echogenic densities noted on initial transthoracic echocardiography study completed following the patient's presentation to the institution. Views 1 and 2.

of possible endocarditis or simply accessory tricuspid tissue shrouding the ventricular septal defect.

Infectious disease specialists were consulted due to the confounding presentation. With his vague history, lack of fever, some increased cardiac echogenicities confounded by a long-standing ventricular septal defect, and unusual cultured microorganism, this was not thought to be a true case of infective endocarditis. He did not have any dental caries or abscesses. Antibiotics were stopped, with a plan to obtain a febrile blood culture prior to re-starting antibiotics.

Two days after discontinuing antibiotics, the patient's temperature spiked to 38.3°C, and a repeat blood culture was also positive for *Streptococcus anginosus* after a 3.8-day incubation period. The third and fourth cultures corroborated *Streptococcus anginosus*. The patient was restarted on IV ceftriaxone for 4 weeks as per American Heart Association recommendations.[4] Overall, he remained stable through his treatment course, with persistent tachycardia, and appeared well on follow-up with a negative blood culture, improved appetite, and no echocardiographic changes.

Discussion

Patients with infective endocarditis can present sub-acutely with an indolent course and non-specific symptomatology.[5] A high index of suspicion is key, as classical findings of Janeway lesions, Osler nodes, and splinter haemorrhages are rarely seen.[4] This was exemplified in our case, as the patient presented with a vague history of only one tactile fever and non-specific symptoms that were difficult to clearly delineate from his concurrent anaemia and mild developmental delay.

Blood cultures and echocardiography play a critical role in the detection and management of infective endocarditis.[4] Common causative organisms include *Viridans group streptococci*, *HACEK* group organisms, *Streptococcus gallolyticus*, and *Staphylococcus aureus*, while *Streptococcus anginosus* is highly unusual.[4,6] Four blood cultures were obtained from different sites, each one more than 12 hours apart, and the case was identified as possible infective endocarditis as per the Duke Criteria.[4]

There were no signs of any abscesses, osteomyelitis, or other infections. Prior to this, the patient did not have any recurrent respiratory or systemic infections, and as such an immunodeficiency workup was not completed, although a referral as outpatient was done to be thorough. A genetic panel including microarray was ordered to assess for Holt–Oram syndrome, due to the patient's congenital defects, developmental delay, and possible consanguinity in his parents.

Transthoracic echocardiography typically reveals intracardiac masses suggestive of vegetations,[4] which were not clearly delineated in our case; in an attempt to close the ventricular septal defect, extra tissues often develop, making it difficult to diagnose infective endocarditis until more obvious echogenic findings are detected. Transthoracic echocardiography studies have been noted to be sufficient in analysing manifestations of possible or definitive infective endocarditis, so transesophageal studies were not performed.[4]

Unrepaired cyanotic defects and the use of prosthetic materials in defect repair are associated with the highest risk of adverse outcomes related to endocarditis[7]; in our case, the patient had a small-to-moderate perimembranous ventricular septal defect, almost completely covered by tricuspid tissue with a restrictive left-to-right shunt. Although this is a case of possible infective endocarditis, following American Heart Association guidelines, the patient will likely require antibiotic prophylaxis, and closure of the defect will be re-discussed at an upcoming cardiology conference, as the family had initially declined intervention.[7]

To the best of our knowledge, there are no published paediatric cases of possible infective endocarditis caused by *Streptococcus anginosus*. A comprehensive review of the literature revealed two cases in patients aged 20–26 years, one case in a patient aged 42 years, four cases in patients aged 50–59 years, five cases in patients aged 63–68 years, one case in a patient aged 71 years, and one case in a patient aged 84 years. The youngest case involved an 18-year-old female presenting with malaise, prolonged low-grade fevers, and underlying mitral valve prolapse. The highly varied case population and presentation makes it difficult to confidently describe management and prognosis for infective endocarditis caused by *Streptococcus anginosus*.

Hallmarks of subacute bacterial endocarditis in paediatric populations include the presence of low-grade fevers and weight loss, with potential new-onset cardiac murmurs and progressive signs of heart failure.[8] The absence of these factors, alongside the rarity of *Streptococcus anginosus* as a causative organism for

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infective endocarditis, contributed to significant confusion in determining a definitive diagnosis. We recommend a high degree of suspicion for possible infective endocarditis in children with CHDs presenting with non-specific symptoms, with judicious use of blood cultures and echocardiography to guide diagnosis.

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Conflicts of interest. None.

References

1. Yang E, Frazee BW. Infective endocarditis. Emerg Med Clin North Am 2018; 36: 645–663. DOI 10.1016/j.emc.2018.06.002.
2. Cahill TJ, Prendergast BD. Infective endocarditis. Lancet 2016; 387: 882–893. DOI 10.1016/S0140-6736(15)00067-7.
3. Dixon G, Christov G. Infective endocarditis in children: an update. Curr Opin Infect Dis 2017; 30: 257–267. DOI 10.1097/QCO.0000000000000370.
4. Baltimore RS, Gewitz M, Baddour LM, et al. Infective endocarditis in childhood: 2015 update: a scientific statement from the American Heart Association. Circulation 2015; 132: 1487–1515. DOI 10.1161/CIR.0000000000000298.
5. Chambers HF, Bayer AS. Native-valve infective endocarditis. N Engl J Med 2020; 383: 567–576. DOI 10.1056/NEJMcp2000400.
6. Doern CD, Burnham CA. It's not easy being green: the viridans group streptococci, with a focus on pediatric clinical manifestations. J Clin Microbiol 2010; 48: 3829–3835. DOI 10.1128/JCM.01563-10.
7. Wilson W, Taubert KA, Gewitz M, et al. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation 2007; 116: 1736–1754. DOI 10.1161/CIRCULATIONAHA.106.183095. Epub 2007 Apr 19. Erratum in: Circulation 2007; 116: e376-e377. PMID: 17446442.
8. Cox DA, Tani LY. Pediatric infective endocarditis: a clinical update. Pediatr Clin North Am 2020; 67: 875–888. DOI 10.1016/j.pcl.2020.06.011.

https://doi.org/10.1017/S104795112200227X Published online by Cambridge University Press