Right-sided native-valve infective endocarditis caused by Streptococcus acidominimus: A case report

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Abstract

Streptococcus acidominimus is rarely pathogenic in humans. In the literature, there are only few cases related to S. acidominimus, including pneumonia, meningitis, brain abscess, pericarditis, sepsis, and endocarditis. Up to now, only a few cases have been published on infective endocarditis due to S. acidominimus, commonly causing left-sided disease. Increasingly, it was noted that it is resistant to beta-lactams. There are no published cases of infective endocarditis due to S. acidominimus in Sri Lanka. Here, we report a case of right-sided native-valve infective endocarditis caused by S. acidominimus which was sensitive to beta-lactams in a 52-year-old man with congenital heart disease.

Keywords

Case report, native-valve infective endocarditis, Streptococcus acidominimus, beta-lactam

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Introduction

Right-sided infective endocarditis (IE) is uncommon, representing 5%-10% of all IE cases, and it is more commonly associated with intravenous drug use, intracardiac devices, and central venous catheters.¹ Although Staphylococcus aureus and viridans group streptococci are the most common microorganisms that account for IE, there has been an increase in the frequency of uncommon microorganisms in recent years.² Streptococcus acidominimus, a gram-positive, spherical, short-chained, catalase-negative, and weak facultative anaerobic cocci, belongs to the viridans streptococci group and is usually considered as a bacterial pathogen in veterinary medicine. It is commonly isolated from vaginal mucosa, on the skin of calves and raw milk. Although viridans group streptococci are a commonly encountered pathogen in IE, S. acidominimus is known to cause human infection rarely.³ There are only few cases of S. acidominimus infection reported in humans.

Here, we present a patient diagnosed with right-sided subacute IE caused by *S. acidominimus*.

Case presentation

A 52-year-old man presented with complaints of intermittent fever associated with loss of appetite, loss of weight, and

generalized malaise for a duration of 2 weeks. He also complained of shortness of breath on exertion without any bleeding manifestations or history of recurrent infections. There was no chronic cough, hemoptysis, joint pain or swelling, and rashes. The patient did not reveal urinary symptoms or history of chronic diarrhea. There were no features suggestive of auto-immune diseases and no recent use of medication before the onset of fever. There was no contact history or past history of tuberculosis. He is a general clerk in occupation and there was no close contact with animals. There were no recent dental procedures he had undergone and he maintained good oral hygiene. There was no significant travel history and history of intravenous drug use. He was a known patient with ventricular septal defect (VSD) and true dextrocardia and was not on regular follow-up due to refusal of surgical interventions. The patient was not on any long-term

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Table 1. Laboratory investigations.

| Investigation | Value | Reference range |
|---|---|-----------------|
| Hemoglobin (g/dL) | 8.0 | 13.5–16.5 |
| Total white blood cells (10 ⁹ /L) | 6.68 | 7–11 |
| Platelets (10 ⁹ /L) | $181 	imes 10^3$ | 150-450 |
| Erythrocyte sedimentation rate | 118mm/1st hour | |
| C-reactive protein (mg/dL) | 73.6 | <5 |
| Creatinine (µmol/L) | 107.6 | 74–110 |
| Creatinine xlearance(mL/min/1.73 m ²) | 68 | |
| Sodium (µmol/L) | 137 | 136–146 |
| Potassium (µmol/L) | 4.3 | 3.5–5.1 |
| Aspartate aminotransferase (U/L) | 24.4 | <50 |
| Alanine aminotransferase (U/L) | 8 | <50 |
| Gamma glutamyl transferase (U/L) | 34.4 | <55 |
| Alkaline Pphosphatase (U/L) | 49.1 | 30–120 |
| Total bilirubin (μmol/L) | 15.15 | 5–21 |
| Urine full report | Normal | |
| Urine protein to creatinine ratio (mg/mmol) | 15.6 | <20 |
| Lactate dehydrogenase (U/L) | 204 | 125–243 |
| Blood picture | Evidence of anemia of chronic disease with features of infective and inflammatory process | |
| Iron studies | Normal | |
| Reticulocyte count | Normal | |
| Ultrasound abdomen | Situs inversus and mild splenomegaly | |

medications. At presentation, he was alert and not dyspneic, and mild pallor was present. His oral hygiene was good. His blood pressure was 110/56 mmHg, pulse rate 88 beats per minute, regular respiratory rate 12/min, SPO₂ 98%, and temperature 37 C. A pansystolic murmur was heard with evidence of dextrocardia. Mild splenomegaly was found on abdominal examination without hepatomegaly. Respiratory and neurological examination was unremarkable.

Laboratory investigations of the patient are listed in Table 1.

Electrocardiogram showed right axis deviation, inversion of all complexes (inverted P and T waves, negative QRS complexes), "global negativity," positive QRS in aVR, and absent R-wave progression (Figure 1). Two-dimensional echocardiogram revealed tricuspid valve large vegetation attached to septal leaflets of the tricuspid valve, hypermobile with a high risk of embolization, and other vegetation attached to the lateral leaflet of the tricuspid valve and also dextrocardia and restrictive VSD with a left-to-right shunt.

All three blood cultures yielded pure growth of *S. acidominimus*, sensitive to ampicillin, penicillin, clindamycin, and erythromycin. The organism was isolated using BACTECTM FX aerobic blood culture system by BD, and antimicrobial sensitivity tests were determined by disk diffusion using CLSI initially but ID by VITEK 2TM.

The patient was diagnosed with right-sided IE and started on intravenous penicillin 4 million units every 4 hourly and he was given oral clindamycin 300 mg every 6 hourly for 7 days due to cannula site infection. Resolution of fever was observed after 48 h of initiation of antibiotic therapy. Follow-up blood culture at 72 h was negative. Significant biochemical response was observed in C-reactive protein and erythrocyte sedimentation rate after 1-week and 2-week course of antibiotic therapy, respectively. He was successfully treated for 6 weeks with a course of intravenous penicillin, and there were no complications of IE reported during the treatment. Follow-up two-dimensional echocardiogram after completion of antibiotics showed complete resolution of the vegetations.

Discussion

Native-valve IE is uncommon, with an incidence of approximately 2–10 cases per 100,000 person-years.⁴ It usually occurs as a consequence of two main factors, which include the presence of microorganisms in the bloodstream and abnormal cardiac endothelium that facilitates adherence of organism and growth.

The diagnosis of IE is made when a microorganism is demonstrated by the culture of a specimen from vegetation; when an embolism or an intracardiac abscess or active endocarditis is confirmed by histological examination of the vegetation or intracardiac abscess; and when two major clinical criteria, one major and three minor criteria, or five minor criteria of Modified Duke criteria are met.⁵ The sensitivity of the modified Duke criteria for IE is approximately 80% for definite cases and higher if possible cases are included.^{4,6}

There are less published data on right-sided IE. Rightsided IE accounts for 5%–10% of all IE cases,¹ which is



Figure 1. Electrocardiogram of the patient.

associated with intravenous drug use; the presence of a cardiac implantable device; an intravenous line such as a central line, intra-aortic balloon pump, or ventricular assist device; and an underlined right-sided cardiac anomaly. *S. aureus* is the most common cause of right-sided IE, accounting for 70% of cases.⁷ The next most common pathogens are Streptococci and Enterococci accounting for 5%–30% and 2%–5%, respectively. Among Streptococci groups, viridans streptococci are the commonest, which account for approximately 20%, and *Streptococcus gallolyticus* (formerly *Streptococcus bovis*) and other streptococci account for approximately 15%.⁴

S. acidominimus belongs to the viridans group streptococci and is rarely pathogenic in humans.⁸ It is commonly isolated from the bovine vaginal mucosa, skin of the calves, and raw milk of cows. Here, in our patient, we were unable to find the source of infection. Since it is rarely associated with infections in humans, the clinical and epidemiological significance of this organism is obscure. In the literature, there are approximately 20 case reports that S. acidominimus is a human pathogen that causes pneumonia, pericarditis, IE, sepsis, brain abscess, and Gradenigo's syndrome.² Only a few case reports were published with regard to IE caused by S. acidominimus, and most of them were leftsided IE.^{2,9,10} Two are in the adult group, particularly among the elderly, and others are in the pediatric age group. The first of these patients was a 80-year-old woman with aortic valve replacement admitted for acute prosthetic valve endocarditis caused by S. acidominimus, which was sensitive to penicillin, clindamycin, erythromycin, ceftriaxone, doxycycline, chloramphenicol, levofloxacin, and vancomycin.11 The other case was an 81-year-old man with elective transcatheter aortic valve implantation admitted with acute mitral valve endocarditis caused by *S. acidominimus*. The strain was sensitive to ampicillin, cephalosporin, tetracycline, and vancomycin and was resistant to penicillin, macrolides, trimethoprim–sulfamethoxazole, and fosfomycin. He was successfully treated with a 6-week course of vancomycin.¹⁰ In our case presentation, subacute and constitutional symptoms were more prominent unlike in other cases. Like in other cases, VSD is an important risk factor for endocarditis in this patient. This patient did not have any animal contact just like in the other cases.

Although the first cases of *S. acidominimus* were betalactam-susceptible, recently cases resistant to beta-lactam group antibiotics have been reported.⁸ In our case, penicillin was found to be susceptible. Due to lack of data, there is no consensus of optimal treatment. Here, we believe that the description of further cases may lead to more formal therapeutic strategies.

Here, in our case, we present a patient with right-sided IE associated with congenital heart disease with the bacteriological diagnosis of *S. acidominimus*. Although recent studies found more beta-lactam-resistant *S. acidominimus*, in our case it was sensitive to antibiotics including penicillin, ampicillin, clindamycin, gentamicin, and erythromycin. He was treated successfully with a 6-week course of penicillin.

Due to the current economic downturn in Sri Lanka, the patient's printed ultrasound images were not available for inclusion in the case report.

Conclusion

A 52-year-old man with right-sided native-valve endocarditis caused by *S. acidominimus* was successfully treated with intravenous penicillin.

Author contributions

All authors contributed to the initial assessment, investigation, analysis, and final diagnosis for the better interest of the patient. All authors have read and approved the manuscript.

Consent for publication

Written informed consent for publication of the patient's clinical details and clinical images was obtained from the patient. A copy of the consent form is available for review by the editor of this journal.

Declaration of conflicting interests

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Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient for their anonymized information including patient's clinical details and clinical images to be published in this article.

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