Case report

A case of infective endocarditis along with a ruptured valve caused by *Streptococcus agalactiae* in an immunocompetent man

Kiyozumi Suzuki¹, Yuji Hirai², Fujiko Morita³, Yuki Uehara³, Hiroko Oshima³, Kazunori Mitsuhashi³, Atsushi Amano⁴, Toshio Naito⁵

¹ Department of General Medicine, Juntendo University Faculty of Medicine, Japan
² Department of Cardiovascular Surgery, Juntendo University Faculty of Medicine, Japan

**ABSTRACT**

*Streptococcus agalactiae* (*S. agalactiae*) is a major cause of invasive disease in neonates and pregnant women, but has also recently been observed among non-pregnant adults, especially elderly persons or persons with underlying chronic disease. *S. agalactiae* is also a rare cause of infective endocarditis, and most cases require early surgery. We report the case of a 43-year-old previously healthy man who experienced rapid progressive culture-negative infective endocarditis with aortic valve vegetation and severe aortic regurgitation, which was complicated by lumbar spondyloïdoliscitis. Emergency aortic valve replacement was performed on the day of his admission, which revealed a congenital bicuspid aortic valve was ruptured by the vegetation. The resected aortic valve specimen was submitted for 16S ribosomal RNA gene sequencing, which revealed that the pathogen was *S. agalactiae*. Therefore, *S. agalactiae* should be considered a potentially causative pathogen in cases of rapid progressive infective endocarditis, even if it occurs in a non-pregnant immunocompetent adult.

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**Introduction**

*Streptococcus agalactiae* (*S. agalactiae*) is also known as group B streptococcus (GBS), and causes invasive disease in neonates and pregnant women. However, the incidence of invasive GBS disease among non-pregnant adults has recently increased, especially among elderly persons. Nevertheless, invasive GBS disease is rare among non-pregnant immunocompetent adults, and most patients have a chronic underlying disease, such as diabetes mellitus, neurological disease, renal failure, malignancy, or liver disease [1–4]. *S. agalactiae* is also a rare cause of infective endocarditis (IE), and most cases require early surgery because of serious complications, such as major emboli or heart failure with rapid heart valve destruction [5–8]. Identification of the causative pathogen is essential for the diagnosis and appropriate treatment of IE, although blood culture-negative endocarditis accounts for 2.5–31% of all cases of endocarditis [9]. One of the most common causes of blood culture-negative endocarditis is the administration of antimicrobial agents before performing the blood cultures. In this report, we describe our experience with a case of rapid progressive culture-negative IE in an immunocompetent man, which was caused by *S. agalactiae*, based on the results from 16S ribosomal RNA (16S rRNA) gene sequencing using the resected valve.

**Case report**

A 43-year-old previously healthy Japanese man presented with a 3-week history of a high-grade fever (>39 °C), rigor, and lumbago. He was treated for a common cold using cefditoren pivoxil, although his family physician did not perform blood cultures. The patient was subsequently referred to our hospital because his symptoms did not resolve. On the first day, chest computed tomography revealed bilateral lower lobe consolidation with pleural effusion. Therefore, we performed two sets of blood cultures and started treatment using ceftriaxone (2 g once daily), based on a preliminary diagnosis of community-acquired pneumonia. One week after his first visit, he was admitted to our hospital after he developed dyspnea with or without effort (body temperature: 36.5 °C, heart rate: 117 beats/min, blood pressure: 98/55 mmHg, respiratory rate: 20 breaths/min, and oxygen saturation: 95% in room air). Chest radiography revealed a butterfly shadow with cardiomegaly and bilateral pleural effusion, which was consistent with congestive heart failure. Transthoracic
echocardiography revealed a 12 × 10-mm vegetation on the aortic valve and severe aortic regurgitation (Fig. 1). A detailed medical interview revealed that he had undergone dental care without tooth extraction at 1 month before his presentation to our hospital.

At the admission, we started treatment using ampicillin/sulbactam (12 g daily in four doses) and gentamicin (180 mg daily in three doses) instead of ceftriaxone for native valve endocarditis, and the patient immediately underwent emergency aortic valve replacement. The resected aortic valve was bicuspid (fusion of the right and left coronary cusps), and both leaflets of the bicuspid aortic valve (BAV) were ruptured by the vegetation (Fig. 2a). No organisms were detected from a total of four sets of blood cultures that were performed before the surgery. Gram staining of the resected valve revealed Gram-positive cocci (Fig. 2b), although no organisms were found in the tissue culture. Therefore, we performed 16S rRNA gene sequencing using the resected valve, which revealed bacterial DNA that was 99% identical to the sequence of S. agalactiae. Magnetic resonance imaging on day 8 also revealed spondylodiscitis in the L5 and S1 vertebrae and in the L5–S1 disc space (Fig. 3). Therefore, we diagnosed the patient as having IE that was caused by S. agalactiae and complicated by lumbar spondylodiscitis. On day 29, the antimicrobial treatment was changed to ampicillin monotherapy (12 g daily in six doses).

On day 43 after the surgery, the intravenous ampicillin was changed to oral amoxicillin (1500 mg daily in three doses) to treat the spondylodiscitis. The patient was discharged from our hospital on day 51, and subsequently completed the 106-day regimen of antimicrobial treatment.

**Discussion**

The course of this case highlights two important clinical issues. First, S. agalactiae should be considered a potentially causative pathogen in cases of rapid progressive IE, even among non-pregnant immunocompetent adults. Although GBS is a rare cause of IE, GBS endocarditis is associated with a poor prognosis (a mortality rate of 35–56% because of major emboli or heart failure), compared to IE that is caused by other streptococci (e.g., the viridans group); therefore, early surgery is recommended for GBS endocarditis [5–8]. In the present case, the man did not exhibit the typical risk factors for GBS disease, although we discovered that he had undiagnosed congenital BAV. Similarly, most cases of BAV are not detected until the onset of infection or calcification, despite BAV being the most common congenital cardiac abnormality (found in 1–2% of the population) [10].

![Fig. 1. Transthoracic echocardiography reveals a 12 × 10-mm vegetation on the aortic valve (arrow).](image1)

![Fig. 2. A. The resected aortic valve revealed a bicuspid valve (fusion of the right and left coronary cusps), which was ruptured by the vegetation. B. Gram staining of the resected specimen reveals Gram-positive cocci (×1000).](image2)
individuals’ respiratory tissue specimens [15]. In the present case, the patient was successfully treated using antimicrobial therapy (ampicillin/sulbactam and gentamicin) and surgery. Therefore, although we could not perform antimicrobial susceptibility testing, we selected ampicillin to replace the ampicillin/sulbactam as definitive therapy for the IE, after we had identified the S. agalactiae using 16S rRNA gene sequencing.

**Conclusion**

*S. agalactiae* can causes rapid progressive IE, even among non-pregnant immunocompetent adults. Furthermore, antimicrobial stewardship is important, as the administration of antimicrobial agents before performing blood cultures is a major cause of culture-negative IE. Therefore, although *S. agalactiae* is a rare cause of rapid progressive IE, it should be considered as a potentially causative pathogen, even among non-pregnant immunocompetent adults. The increasing incidence of PRGBS with elevated β-lactam MICs may make it increasingly difficult to select an appropriate antimicrobial treatment for GBS endocarditis.

**Conflicts of interest**

The authors declare no conflicts of interest.

**Acknowledgement**

None.

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