Complete Atrioventricular Block Complicating Mitral Infective Endocarditis Caused by *Streptococcus Agalactiae*

Authors' Contribution:
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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient:	**Male, 74**
Final Diagnosis:	**Infective endocarditis**
Symptoms:	**Apetite loss • fever**
Medication:	**—**
Clinical Procedure:	**Transesophageal echocardiography**
Specialty:	**Cardiology**

Objective: Rare co-existance of disease or pathology

Background: Infective endocarditis (IE) involving the mitral valve can but rarely lead to complete atrioventricular block (CAVB).

Case Report: A 74-year-old man with a history of infective endocarditis caused by *Streptococcus gordonii* (*S. gordonii*) presented to our emergency room with fever and loss of appetite, which had lasted for 5 days. On admission, results of serologic tests pointed to severe infection. Electrocardiography showed normal sinus rhythm with first-degree atrioventricular block and incomplete right bundle branch block, and transthoracic echocardiography and transesophageal echocardiography revealed severe mitral regurgitation caused by posterior leaflet perforation and 2 vegetations (5 mm and 6 mm) on the tricuspid valve. The patient was initially treated with ceftriaxone and gentamycin because blood and cutaneous ulcer cultures yielded *S. agalactiae*. On hospital day 2, however, sudden CAVB requiring transvenous pacing occurred, and the patient's heart failure and infection worsened. Although an emergent surgery is strongly recommended, even in patients with uncontrolled heart failure or infection, surgery was not performed because of the Child-Pugh class B liver cirrhosis. Despite intensive therapy, the patient's condition further deteriorated, and he died on hospital day 16. On postmortem examination, a 2×1-cm vegetation was seen on the perforated posterior mitral leaflet, and the infection had extended to the interventricular septum. Histologic examination revealed extensive necrosis of the AV node.

Conclusions: This rare case of CAVB resulting from *S. agalactiae* IE points to the fact that in monitoring patients with IE involving the mitral valve, clinicians should be aware of the potential for perivalvular extension of the infection, which can lead to fatal heart block.

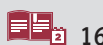**MeSH Keywords:** Atrioventricular Block • Endocarditis, Bacterial • *Streptococcus agalactiae*

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/898142

Background

Infective endocarditis (IE) involving the mitral valve often leads to supraventricular arrhythmia [1]; rarely a life-threatening conduction abnormality such as atrioventricular block (AVB) arises [2,3]. Here, we describe a case of *Streptococcus agalactiae* (*S. agalactiae*) IE involving the mitral valve and resulting in complete AVB (CAVB).

Case Report

A 74-year-old man presented to our emergency room with fever and loss of appetite, both of which had lasted for 5 days. His medical history included coronary artery bypass grafting, alcoholic liver cirrhosis, hypertension, and chronic diabetic kidney disease. He also had a prior (1 year) episode of IE due to *S. gordonii* detected by MALDI-TOF. Vegetation (7 mm) on the posterior mitral leaflet without mitral regurgitation or perforation was observed by transthoracic echocardiography and abolished eventually with penicillin and gentamycin. On presentation, he appeared pale and unwell. His body temperature was 38.8°C, blood pressure was 118/70 mmHg, heart rate was 65 beats/min, respiratory rate was 22 breaths/min, and oxygen saturation (obtained by pulse oximetry) was 96% on room air. On cardiopulmonary auscultation, there was no gallop, but a grade II/VI pansystolic murmur at the apex and minimal bibasilar crackles were heard. A lower limb diabetic cutaneous ulcer and edema were noted.

The electrocardiogram showed no specific changes compared with that during prior hospitalization; normal sinus rhythm, first-degree AVB, incomplete right bundle branch block, and high voltage with ST-T abnormalities in the precordial leads, suggestive of left ventricular hypertrophy (Figure 1). Chest X-ray showed mild cardiomegaly with mild pulmonary edema. Laboratory tests revealed a leukocyte count of 10,300/mm³ (normal range, 4000–8000/mm³) a markedly elevated C-reactive protein level of 25.5 mg/dL (normal range, <0.2 mg/dL), and an elevated N-terminal pro-brain natriuretic peptide (NT-pro-BNP) concentration of 27,498 pg/mL (normal range, <125 pg/mL). Transthoracic echocardiography and transesophageal echocardiography revealed a perimitral abscess, severe mitral regurgitation caused by posterior mitral leaflet perforation, and 2 vegetations (5 mm and 6 mm) on the tricuspid valve (Figure 2A–2C). The patient was initially treated with ceftriaxone and gentamycin. Cultures of the blood and the cutaneous ulcer yielded *S. agalactiae*.

On hospital day 2, however, sudden CAVB requiring transvenous pacing occurred (Figure 3), and the heart failure and infection worsened. Although emergency surgery was considered to control the infection and heart failure and to prevent thromboembolic events, surgery was not performed because of the Child-Pugh class B liver cirrhosis. Despite intensive therapy, the patient's condition continued to deteriorate, and he died on hospital day 16.

On postmortem examination, a 2×1-cm vegetation was found on the perforated posterior mitral leaflet (Figure 4A) and 2 vegetations of 8 mm on the septal leaflet of the tricuspid valve (Figure 4B). Histologic examination of the AV node and His bundle revealed extensive necrosis with fibrosis and calcification (Figure 4C–4E).

Discussion

This case of fatal CAVB was characterized by bilateral IE with mitral and tricuspid valves involvement caused by *S. agalactiae,* by mitral abscess affecting the AV node, and by late Streptococcal reinfection by a different species.

Although IE due to *S. agalactiae* occurs infrequently, its incidence has increased to 3% over recent years [4]. When *S. agalactiae* IE occurs, the patient is likely to be of advanced age or to have a chronic debilitating disease, such as diabetes. Mitral valve involvement is frequently observed, and multiple valve
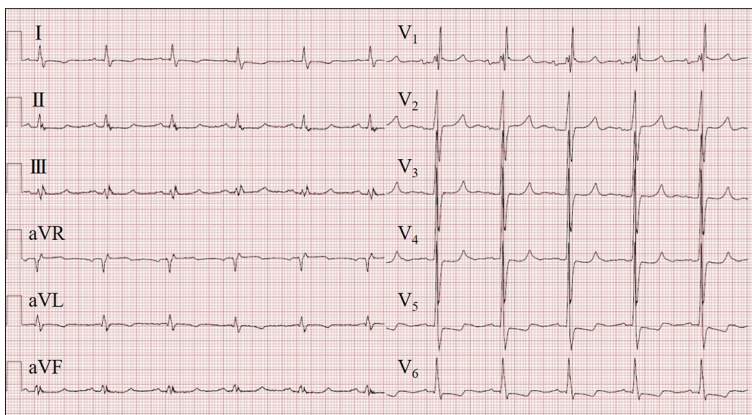
**Figure 1.** Twelve-lead electrocardiogram obtained on admission.
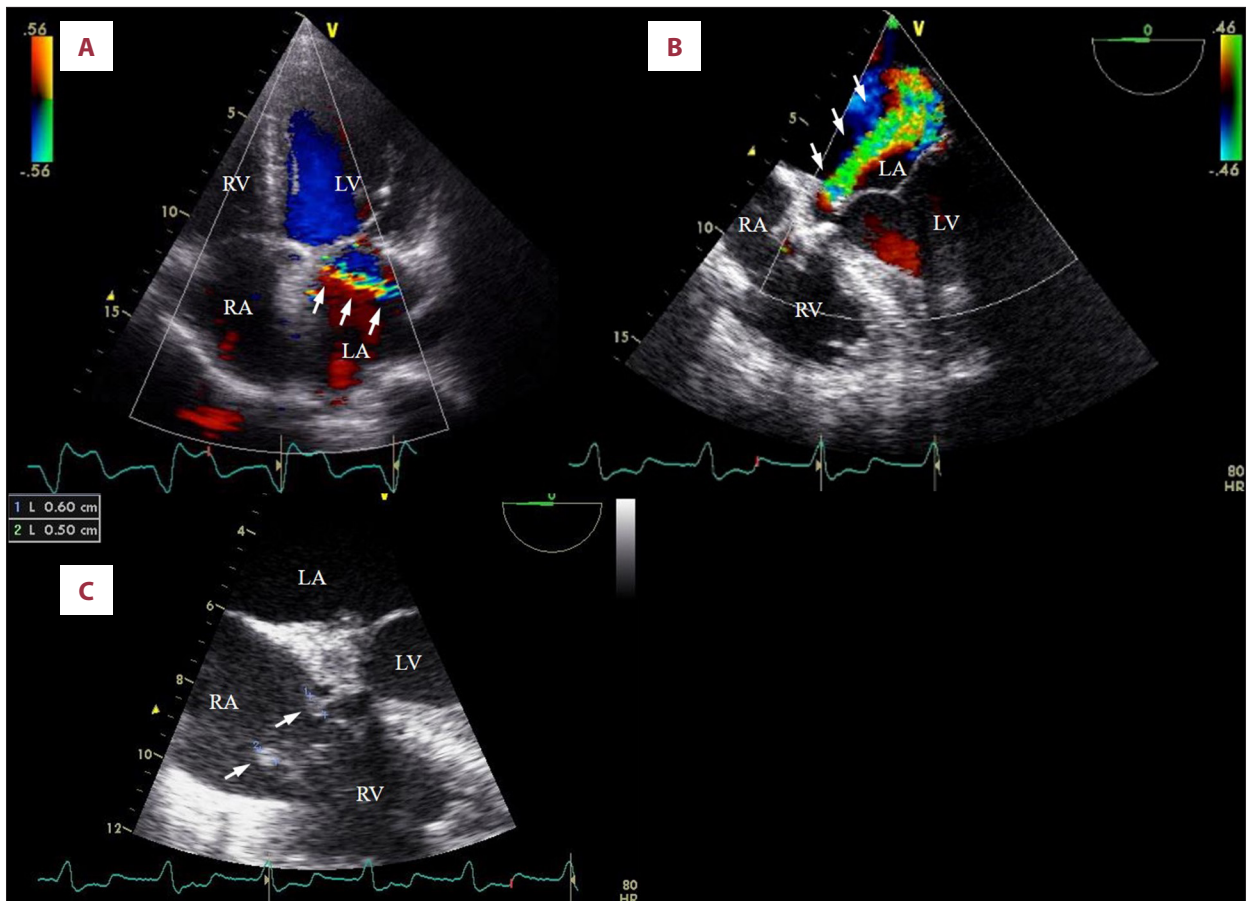
Figure 2. Transthoracic echocardiographic image (**A**) and transesophageal echocardiographic images (**B, C**) obtained on the day of admission (apical 4-chamber view). (**A**) and (**B**) A perimitral abscess and severe mitral regurgitation caused by posterior mitral leaflet perforation are apparent (arrows). (**C**) Two vegetations, one measuring 5 mm and the other measuring 6 mm, are evident on the tricuspid valve on the tricuspid valve.
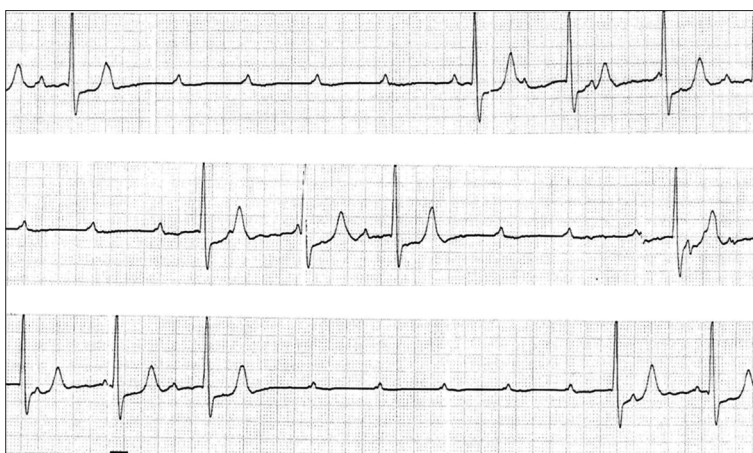
Figure 3. Electrocardiogram showing complete atrioventricular block obtained on hospital day 2.

involvement occurs in 22% of cases (*vs.* 15% of all cases of endocarditis overall) [4,5]. Typically, the endocarditis progresses rapidly and leads to acutely decompensated heart failure resulting from severe valve destruction. Although surgery is frequently performed, mortality remains high at 41–47% [5,6].

IE in the bilateral location is rare and reported to be 15–25% of multivalvular IE [7]. Bilateral IE usually occurs in patients with intracardiac devices [8] or congenital heart disease [9], or intravenous drug users [10]. However, the patient did not have these backgrounds. Furthermore, postmortem examination
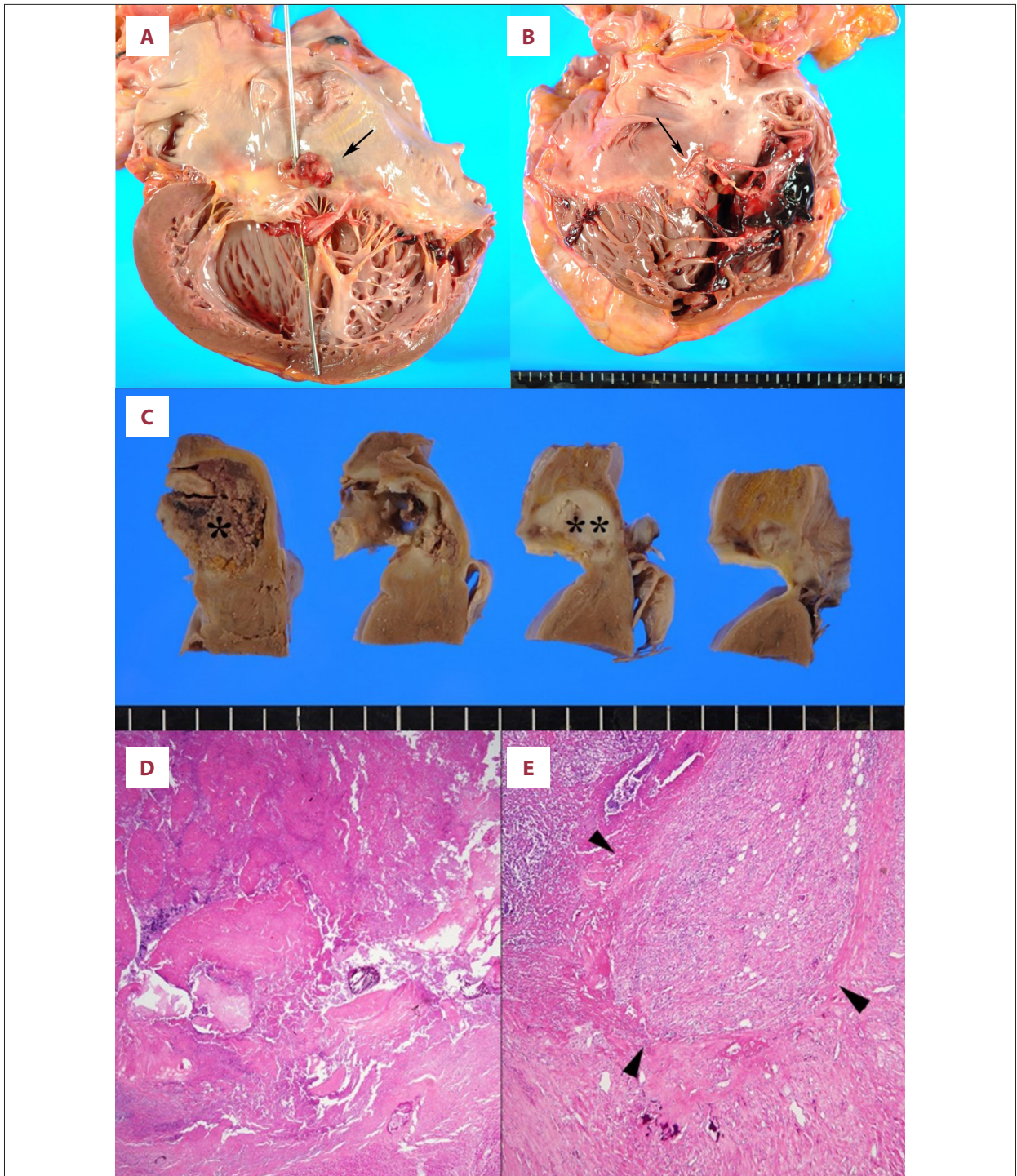
Figure 4. Macroscopic appearance of the left ventricle (**A**) and pathological features of atrioventricular (AV) node and His bundle (**B–D**) on postmortem examination. (**A**) A vegetation, 2×1 cm in size, is present at the perforated posterior mitral leaflet (arrow). The perforation site is indicated by a probe. (**B**) Two vegetations of 8 mm in size are present at the septal leaflet of the tricuspid valve (arrow; another vegetation is hidden behind the septal leaflet in this view.) Serial cross sections including the AV node and His bundle. The area encompassing the AV node and His bundle is replaced by a whitish area with the hyperemic border. (**C**) Histologic examination of the (*) area revealed extensive necrosis with fibrosis and calcification of the AV node. No nodal structure is seen. (**E**) Histologic examination of the (**) area revealed only a vague His bundle structure (arrowhead) that was surrounded by fibrosis with calcification and necrosis.

revealed no atrial or ventricular septal perforation and histologic examination revealed mitral abscess showed no tricuspid involvement. Given that, bilateral cardiac involvement in this case might be caused by hematogenous metastasis.

Conduction abnormalities develop in 4–10% of IE patients. Such abnormalities are most likely in patients with aortic valve IE and occur only rarely in patients with mitral valve infection; only a few such cases have been reported [2,3,11,12]. This is because although the AV node lies adjacent to the mitral valve, the His-Purkinje system is anatomically closer to the aortic valve [12]. In cases of conduction abnormality associated with infective endocarditis, anatomical destruction by an abscess or perivalvular extension of the infection should be considered [3]. Surgery is strongly recommended, even in patients with uncontrolled heart failure or infection [13], but a conduction abnormality that persists even after antibiotic therapy portends a poor prognosis [14]. Furthermore, 41% post-cardiac surgery mortality has been reported for patients with class B cirrhosis, and a Child-Pugh score >7 points has been shown to predict postoperative mortality with 86% sensitivity and 92% specificity [15]. Thus, although involvement of the interventricular septum in the perimitral abscess caused AV node necrosis in our case and led to AVB, the patient was not considered a candidate for surgery because of the Child-Pugh class B cirrhosis (Child-Pugh score 8).

The risk of reinfection defined as a repeat episode of IE has been reported to be 6% [16]. Repeat infective endocarditis due to a different species occurs less frequently. Further studies are warranted for risk stratification and management of repeat endocardial infection.

Conclusions

We encountered a rare case of fatal CAVB resulting from *S. agalactiae* IE. In monitoring IE, clinicians should be aware of the possibility of perivalvular extension of the infection and that such extension can lead to fatal heart block. In such case, surgical treatment is strongly indicated because medical treatment alone is insufficient.

Statement

References:

1. Eisinger AJ: Atrial fibrillation in bacterial endocarditis. Br Heart J, 1971; 33: 739–41

2. Wang K, Gobel F, Gleason DF, Edwards JE: Complete heart block complicating bacterial endocarditis. Circulation, 1972; 46: 939–47

3. Chang M, Cunha AB: *Streptococcus agalactiae* (Group B *Streptococcus*) infective endocarditis complicated by myocardial abscess and heart block: Case report and review. Infectious Disease in Clinical Practice, 2004; 12(2): 107–9

4. Ivanova Georgieva R, Garcia Lopez MV, Ruiz-Morales J et al., Andalusian Group for the Study of Cardiovascular Infections of the Andalusian Society of Infectious Diseases SAEI: *Streptococcus agalactiae* left-sided infective endocarditis. Analysis of 27 cases from a multicentric cohort. J Infect, 2010; 61: 54–59

5. Selton-Suty C, Doco-Lecompte T, Bernard Y et al: Clinical and microbiologic features of multivalvular endocarditis. Curr Infect Dis Rep, 2010; 12: 237–43

6. Sambola A, Miro JM, Tornos MP et al: *Streptococcus agalactiae* infective endocarditis: analysis of 30 cases and review of the literature, 1962–1998. Clin Infect Dis, 2002; 34: 1576–84

7. Selton-Suty C, Doco-Lecompte T, Bernard Y et al: Clinical and microbiologic features of multivalvular endocarditis. Curr Infect Dis Rep, 2010; 12: 237–43

8. Duval X, Selton-Suty C, Alla F et al: Endocarditis in patients with a permanent pacemaker: A 1-year epidemiological survey on infective endocarditis due to valvular and/or pacemaker infection. Clin Infect Dis, 2004; 39: 68–74

9. Yao F, Han L, Xu ZY et al: Surgical treatment of multivalvular endocarditis: twenty-one-year single center experience. J Thorac Cardiovasc Surg, 2009; 137: 1475–80

10. Miro JM, del Rio A, Mestres CA: Infective endocarditis in intravenous drug abusers and HIV-1 infected patients. Infect Dis Clin North Am, 2002;16: 273–95, vii–viii

11. DiNubile MJ, Calderwood SB, Steinhaus DM, Karchmer AW: Cardiac conduction abnormalities complicating native valve active infective endocarditis. Am J Cardiol, 1986; 58: 1213–17

12. Kitkungvan D, Suri J, Spodick D: Complete atrioventricular block: A rare presentation of mitral valve endocarditis. J Invasive Cardiol, 2010; 22: E1–2

13. Dinubile MJ: Heart block during bacterial endocarditis: A review of the literature and guidelines for surgical intervention. Am J Med Sci, 1984; 287: 30–32

14. Javaid M, Awasthi A, Fink G: Complete heart block associated with mitral annular abscess. Mayo Clin Proc, 2005; 80: 1531–32

15. Suman A, Barnes DS, Zein NN et al: Predicting outcome after cardiac surgery in patients with cirrhosis: A comparison of Child-Pugh and MELD scores. Clin Gastroenterol Hepatol, 2004; 2: 719–23

16. Levison ME, Kaye D, Mandell GL, Hook EW: Characteristics of patients with multiple episodes of bacterial endocarditis. JAMA, 1970; 211: 1355–57