Case Report

Unusual Case Report of Infective Endocarditis Due to Streptococcus Agalactiae in Lebanon

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Highlights

- Right-sided infective endocarditis caused by Streptococcus agalactiae is a very rare case. It may be complicated by right-sided heart dilatation leading to right-sided heart failure.
- Treatment guidelines on infective endocarditis caused by this agent remain limited, prompting further study to establish definitive treatment guidance. Surgical approach and antibiotic treatments remain the empirical treatment choice.

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ABSTRACT

Background: Right-sided infective endocarditis (IE) is uncommon. Furthermore, Streptococcus agalactiae (S agalactiae) rarely causes IE, with only 2% to 9% of bacteremia cases manifesting as endocarditis.

Case Presentation: A 30-year-old Lebanese woman presented to the emergency department with high-grade fever and chills. Blood tests were obtained, urinalysis showed leukocytes, and treatment for urinary tract infection was initiated. The patient was hospitalized 3 days later and transferred to the ICU. Echocardiography revealed severe right-sided cardiomegaly, flail tricuspid leaflets with ruptured chordae, and a large coaptation defect. She was diagnosed with right-sided IE due to S agalactiae and underwent tricuspid valve replacement. Unfortunately, the patient died from acute decompensated heart failure.

Conclusion: This case represents an unusual instance of right-sided IE due to S agalactiae in Lebanon, for which definitive treatment guidance remains limited.

Keywords: Streptococcus Agalactiae; Infective Endocarditis; Case Report

RHYTHM
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Background

Over the past decades, infective endocarditis (IE) has remained a serious health problem, with annual mortality consistently at 30%.[1] A recent study at the American University of Beirut Medical Center, Lebanon, reported 80 cases of IE between 2001 and 2014, with streptococci as the most frequently isolated organisms.[2] Compared with left-sided IE, right-sided IE is less common, accounting for only 5% to 10% of cases.[3] IE due to Streptococcus agalactiae (S agalactiae) is particularly uncommon. Only 2% to 9% of bacteremia cases caused by S agalactiae manifest as IE.[4]

We report the case of a 30-year-old woman with S agalactiae IE complicated by right-sided heart dilatation leading to right-sided heart failure on previously normal native valves, who subsequently underwent tricuspid valve replacement surgery.

Case Presentation

A 30-year-old Lebanese woman presented to the emergency department with high-grade fever and chills. She was previously healthy. She was from a lower-middle socioeconomic background and had no significant past medical history or family history of cardiovascular disease. She had married recently and reported that her first sexual intercourse occurred a few days earlier. On physical examination, there was a minor vaginal laceration, possibly related to sexual intercourse.

Blood tests (reference ranges in Table 1) showed a mildly elevated neutrophil percentage (80.2%), a low platelet count (37×10³/µL), elevated C-reactive protein (25.9 mg/dL), and γ-glutamyl transferase (54 U/L). Procalcitonin was 5.59 ng/mL, consistent with ongoing bacterial infection. Amylase was low at 7 U/L. Moreover, β-hCG was below 1.2 mIU/mL, indicating the patient was not pregnant. Interleukin-6 (IL-6) was measured to evaluate multisystem inflammatory syndrome in adults (MIS-A); although the patient reported no viral infection during the past 2 weeks, the level was markedly elevated at 1112 pg/mL. Serum creatinine was 1.4 mg/dL.

Urinalysis revealed a turbid appearance; protein, epithelial cells, granular casts, and amorphous urate were present. White blood cells were 18 to 20/hpf. Urine culture was positive, and gram-positive cocci in chains were observed. Gram stain and culture of urine and blood grew *S agalactiae*, suggesting that the urinary tract infection (UTI) may have been the predisposing factor for IE.

Abdominal ultrasonography showed a gassy abdomen and slight gallbladder contraction with an echogenic thick line, suggesting a cholecystic process with biliary ascariasis. The urinary bladder was not optimally distended, with regular wall thickness; other findings were within normal limits. Empiric treatment for UTI with a β-lactam (meropenem) and a glycopeptide (vancomycin), dose-adjusted for clearance, was initiated for an acutely complicated UTI with fever in the setting of critical illness.

Three days later, she returned to the emergency department with chief complaints of myalgia, high-grade fever, and generalized weakness. She also reported gradual hearing loss; during examination, she was unable to hear the examiner, and communication occurred via typed text. The pattern was sensorineural hearing loss and was initially attributed to possible nerve damage in the context of suspected COVID-19 infection (MIS-A had been ruled out). On auscultation, a systolic murmur that increased with inspiration was heard. Review of systems revealed no prior history of the condition.

Blood tests were obtained again and showed a platelet count of 105 × 10³/µL, an absolute neutrophil count of 12.8 × 10³/µL, a blood urea nitrogen level of 49 mg/dL, and an elevated but down-trending creatinine level of 1.45 mg/dL. Creatine phosphokinase was 37 IU/L, lactate dehydrogenase was elevated at 324 IU/L, and C-reactive protein was 4.81 mg/dL. High-sensitivity troponin T was 0.191 ng/mL. ECG showed sinus tachycardia, right atrial abnormality, right bundle branch block, and a probable anteroseptal infarct (Figure 1).

She was admitted to the hospital. Medications included a proton pump inhibitor (omeprazole) for ulcer prophylaxis in the setting of multiple medications, ondansetron for nausea, ceftriaxone for UTI, and a corticosteroid for suspected MIS-A before laboratory results became available. Albendazole was administered for 4 days as antiparasitic treatment for biliary ascariasis observed on ultrasonography 3 days earlier.

On the first day of hospitalization, the patient developed dyspnea, desaturation, and chest discomfort; an urgent chest computed tomography (CT) scan was ordered. The chest CT showed mild effusion with cardiomegaly. Laboratory results revealed a platelet count of $121\times10^3/\mu L$, and urinalysis showed 16 to 18/hpf white blood cells. High-sensitivity troponin T was elevated at 0.230 ng/mL. The patient was then transferred to the ICU for dyspnea, desaturation, and hypotension.

Five days after the initial hospital admission, brain magnetic resonance imaging (MRI) was performed and showed no intracranial hemorrhage, acute cerebral, brainstem, or cerebellar infarction, no evidence of mass effect, and no midline shift, thereby ruling out septic emboli. Transesophageal echocardiography demonstrated severe right-sided cardiomegaly, with marked dilation of the right atrium and ventricle (Figure 2). The tricuspid leaflets were flail with ruptured chordae and a large coaptation defect, but no vegetation or abscess was detected.

Based on Duke criteria, the case fulfilled two major requirements (positive blood cultures for streptococci, a common IE-causing microorganism, and positive echocardiographic findings consistent with tricuspid valve endocarditis) and one minor requirement (high-grade fever). The patient was diagnosed with right-sided IE due to S agalactiae, classified as a definite case according to the 2023 European Society of Cardiology (ESC) endocarditis guidelines.[5]

Up until the time of surgery, she was treated with phenylpropanolamine for nausea, bisoprolol for heart failure, furosemide for edema secondary to heart failure, antimicrobial therapy for infection, and esomeprazole (Nexium) for peptic ulcer prophylaxis in the setting of polypharmacy.

Approximately 2 weeks before surgery, Doppler echocardiography (Figure 3) was performed and revealed findings similar to those of the initial study. The right atrium remained severely dilated, with an area of 43 cm^2. The right ventricle showed a tricuspid annular plane systolic excursion of 32 mm and a lateral tricuspid annulus peak systolic velocity (S′) of 15 cm/s, indicating severe dilation despite preserved systolic function. The left atrium and ventricle were normal in size, with paradoxical septal motion consistent with right ventricular volume overload. Left ventricular systolic function was normal, with good contractility across all segments (end-diastolic diameter: 33 mm; interventricular septal thickness: 9 mm; posterior wall thickness: 8 mm; and ejection fraction: 55%–60%).

The mitral leaflets appeared myxomatous with mild bileaflet prolapse, without mitral regurgitation (deceleration time:201 ms). A large coaptation defect of the tricuspid leaflets with ruptured chordae was present, unchanged compared with the previous examination. Torrential tricuspid regurgitation was observed, and the inferior vena cava was severely dilated.

One month after the initial hospitalization, the patient underwent valve surgery for massive tricuspid insufficiency. Intraoperative inspection revealed that the anterior leaflet was multiperforated with “rope” rupture and sequelae of vegetation. Intraoperative transesophageal echocardiography also demonstrated a large coaptation defect of the tricuspid leaflets with severe regurgitation and a tricuspid annulus diameter of 49 mm. The valve was destroyed and deemed irreparable.

The anterior and posterior leaflets were resected, the septal leaflet was preserved, and tricuspid valve replacement was performed with an EPIC St Jude 33-mm bioprosthesis, secured with 17 U-shaped TiCron sutures on a Teflon splint.

One week after surgery, Doppler echocardiography showed an ejection fraction of 60%. The right atrium was dilated, and the right ventricle had severely impaired systolic function. The tricuspid prosthetic valve showed no stenosis or regurgitation. The inferior vena cava was mildly dilated with poor inspiratory collapse. Minimal pericardial effusion and bilateral pleural effusions were present (Figure 4).

Postoperative treatments included anticoagulants, as IE increases the risk of vascular complications such as cerebral embolism; a β1-agonist (dobutamine) for decompensated heart failure; and diuretics (furosemide and spironolactone) for heart failure with edema. The patient also received a β-lactam (meropenem), vancomycin, and a fluoroquinolone (levofloxacin) as antibiotics to treat IE.

After surgery, the patient’s condition remained stable, and she was discharged. She returned to the

emergency department 3 weeks after surgery. She was hospitalized for arrhythmia due to acute decompensated heart failure, diagnosed clinically with dyspnea and edema. Atrial flutter was treated with amiodarone. Unfortunately, she died on hospital day 3.

Table 1. Lab results and normal lab value references (Only mentioned tests are included.)

Test	Result	Normal Range
Neutrophils	80.2%	37%–75%
Neutrophil count (absolute number)	12.8 × 10^3/µL	1.5–6.6 × 10^3/µL
Platelet count	First ED visit: 37 × 10^3/µL Second ED visit: 105 × 10^3/µL First hospital day: 121 × 10^3/µL	140–440 × 10^3/µL
C-reactive protein	25.9 mg/dL	0–1 mg/dL
γ-glutamyl transferase (GGT)	54 U/L	5–36 U/L (female)
Procalcitonin	5.59 ng/mL	<0.05: absence of bacterial infection <0.5: bacterial infection unlikely ≥0.5 to <2: possible infection ≥2 to <10: suggestive of bacterial infection ≥10: severe sepsis or septic shock
Amylase	7 U/L	25–125 U/L
β-hCG	<1.2 mIU/mL	<4: not pregnant Pregnancy ranges: 1500–23 000 (4–5 wk) 3400–135 300 (5–6 wk) 10 500–161 000 (6–7 wk) 18 000–209 000 (7–8 wk) 37 500–219 000 (8–9 wk) 42 800–218 000 (9–10 wk)
Interleukin-6	1112 pg/mL	0–7 pg/mL
Creatinine	First ED visit: 1.4 mg/dL Second ED visit: 1.45 mg/dL	0.5–1.3 mg/dL
Blood urea nitrogen (BUN)	49 mg/dL	7–25 mg/dL
Creatine phosphokinase (CPK)	37 IU/L	195–700 IU/L
Lactate dehydrogenase (LDH)	324 IU/L	9–220 IU/L
High-sensitivity troponin T	Second ED visit: 0.191 ng/mL First hospital day: 0.230 ng/mL	0–0.014 ng/mL
WBCs in urine	First ED visit: 18–20/hpf First hospital day: 16–18/hpf	<5/hpf

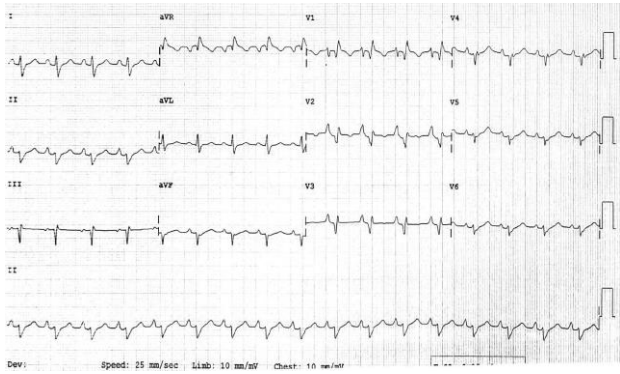

Figure 1. The ECG shows sinus tachycardia, right atrial abnormality, right bundle branch block, and probable anteroseptal infarct.

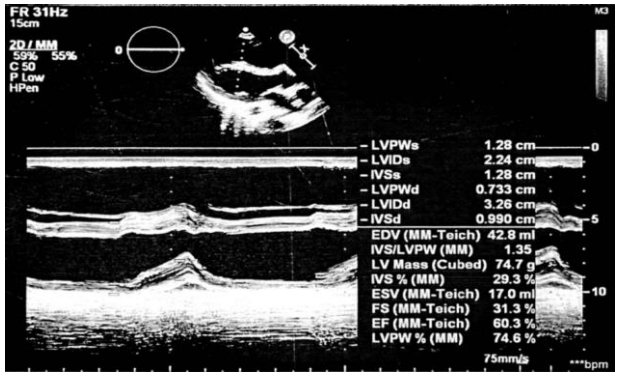

Figure 2. The image shows the patient's transesophageal echocardiogram on the sixth day of hospitalization.

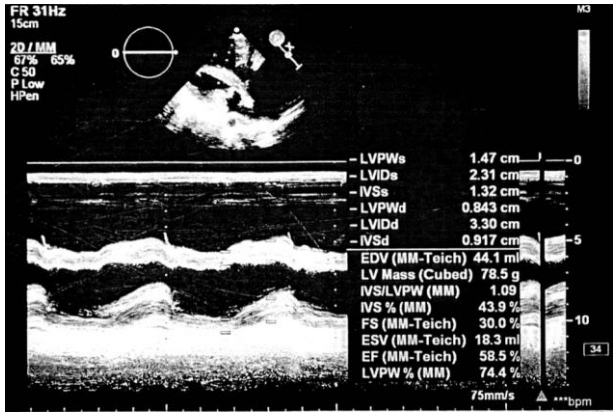

Figure 3. The image presents the patient's Doppler echocardiography 2 weeks before surgery.

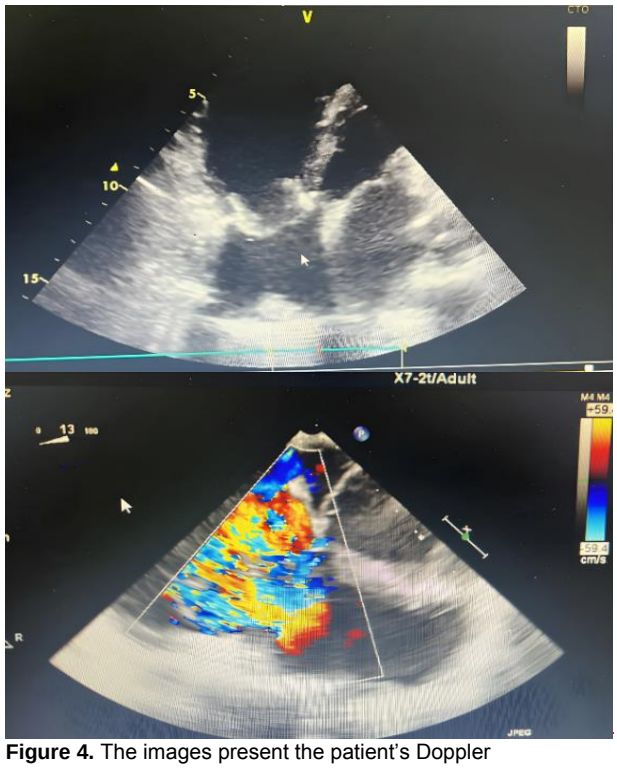

Figure 4. The images present the patient's Doppler echocardiogram post-surgery.

Discussion

IE is an infection involving the cardiac endocardium, typically caused by bacterial bloodstream infection. The 2009 European Society of Cardiology (ESC) guidelines on IE proposed classification based on location, distinguishing right-sided native valve IE from left-sided IE.[6]

In this case, IE was caused by S agalactiae, a group B streptococcus (GBS) that is a β-hemolytic gram-positive bacterium. The presence of S agalactiae is closely associated with genitourinary disorders and infections.[7] The patient in this report was substantially younger (30 y) than those in previously documented cases of S agalactiae IE and had no significant past medical history or cardiac disease. GBS infections more commonly occur in older adults, especially those with comorbidities such as diabetes mellitus, structural heart disease, or chronic kidney disease.[8,9]

Most cases of S agalactiae IE lack a clearly identified source of infection. Nonetheless, at admission, this patient presented with symptoms of UTI, a common clinical manifestation of GBS infection.[10]

Brain MRI in our patient showed no acute cerebral event or infarction. In contrast, cases reported by Ya'qoub et al[11] and D'Angelo et al [12] demonstrated embolic infarctions on brain MRI. Aside from heart failure, embolism is a common complication of IE, with up to 65% of embolic events involving the central nervous system. The size of vegetation is a key factor that increases the risk of embolism, which explains the absence of cerebral infarction in our case, as no vegetation was observed on the initial echocardiography.

Echocardiographic findings confirmed right-sided IE in this patient, a rare presentation, particularly because it was caused by S agalactiae.[13] Right-sided IE accounts for only 5% to 10% of all cases.[14] IE due to S agalactiae is typically characterized by an acute onset, large vegetations, and rapid valve destruction, as was observed during intraoperative exploration of the tricuspid valve. These features underscore the aggressive nature of this microorganism. In this case, however, the initial echocardiography showed no vegetation, although sequelae of vegetation were noted during surgery. Rollán et al[15] reported that the clinical course of S agalactiae IE more closely resembles that of Staphylococcus aureus than that of other streptococci, such as S viridans.

In this case, surgery was performed to replace the tricuspid valve, which was destroyed and demonstrated insufficiency with severe regurgitation. The 2016 and 2019 American Association for Thoracic Surgery (AATS) guidelines on surgical treatment of IE recommend surgery for patients with severe valve dysfunction, signs of heart failure, cardiac fistulas or paravalvular abscesses, prosthetic valve endocarditis, large mobile vegetations, recurrent systemic embolization, or sepsis persisting beyond 5 to 7 days despite adequate antibiotic therapy.[16]

For right-sided IE, surgery is indicated when

infection cannot be controlled, septic pulmonary embolism cannot be prevented, or, less commonly, when there is substantial tricuspid valve regurgitation.[16] The European Society of Cardiology (ESC) further recommends surgery when tricuspid vegetation size is 20 mm or greater.[17]

Intraoperative transesophageal echocardiography in our patient showed a tricuspid annulus diameter of 49 mm, consistent with dilation. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines for valvular heart disease (VHD) state that tricuspid annular dilation (end-diastolic diameter >40 mm) or antecedent clinical manifestations of right-sided heart failure can both warrant consideration for tricuspid valve surgery.[18] The primary objectives of surgery in IE are the removal of infected tissue and the reconstruction of cardiac morphology. Valve repair is preferred over replacement when feasible.[19]

Unfortunately, in this case, the patient died due to acute decompensated heart failure. Endocarditis may precipitate acute deterioration of moderate VHD. A superimposed precipitant of varying severity can impose additive hemodynamic stress, often manifesting clinically or causing abrupt worsening of valve defects. Additionally, functional VHD—particularly mitral regurgitation and tricuspid regurgitation—may result from changes to the cardiovascular substrate associated with progression of heart failure, and their concurrence may complicate the clinical picture. Secondary mitral regurgitation and tricuspid regurgitation arise from structural changes in ventricular or atrial geometry, in contrast to primary valve regurgitation, which occurs when the valve apparatus is anatomically normal.[20]

Conclusion

This report describes an unusual case of right-sided IE due to S agalactiae in Lebanon. Although S agalactiae rarely causes IE, its clinical manifestations are similar to those of IE in general. Still, definitive treatment guidelines for IE caused by this pathogen remain limited.

Declarations:
Ethical Approval

Informed consent was obtained from the patient's family, and they gave their written consent to use the patient's personal data for the publication of this case report and any accompanying images.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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