

A CASE OF INFECTIVE ENDOCARDITIS CAUSED BY BETA-LACTAM RESISTANT *STREPTOCOCCUS ALACTOLYTICUS*

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ABSTRACT

Background: *Streptococcus alactolyticus* is a rare cause of human infections, with limited reports of infective endocarditis (IE).

Case report: We present a case of a 68-year-old male with severe aortic stenosis who developed *S. alactolyticus* associated IE resistant to beta-lactams. Initial treatment with vancomycin and gentamicin led to temporary improvement, but the patient later developed complications, including splenic infarction and an aortic root abscess. Despite intensified antimicrobial therapy, he ultimately succumbed to multiorgan failure.

Conclusion: This case underscores the importance of early identification of resistant pathogens, appropriate antibiotic selection, and vigilant monitoring for complications. The rising incidence of beta-lactam resistance in streptococcal infections highlights the need for ongoing epidemiological surveillance and research to optimize treatment strategies.

KEYWORDS

Infective endocarditis, *Streptococcus alactolyticus*

LEARNING POINTS

- *Streptococcus alactolyticus* is a rare but emerging cause of infective endocarditis that necessitates increased clinical awareness.

INTRODUCTION

Infective endocarditis (IE) is a life-threatening inflammation of the endocardium, with high mortality despite advances in antimicrobial therapy and cardiac surgery. It is primarily caused by bacteria, fungi, or other microorganisms that enter the bloodstream and adhere to damaged cardiac structures. Individuals with prosthetic heart valves,

structurally abnormal heart valves, or congenital heart defects are at increased risk. Gram-positive cocci, including *Staphylococcus*, *Streptococcus*, and *Enterococcus* species, are the leading cause of IE, with *Staphylococcus aureus* being the most frequently isolated pathogen^[1]. Group D streptococci, such as *Streptococcus gallolyticus* and *Streptococcus bovis*, are strongly associated with IE and

underlying colonic neoplasms^[1]. *Streptococcus alactolyticus*, a subspecies within the *Streptococcus bovis/equinus* complex (SBSEC), is commonly found in the gastrointestinal tracts of animals and fermented dairy products^[2]. We present a unique case of *S. alactolyticus* IE, highlighting the pathogen's evolving resistance patterns and the potential for severe complications despite aggressive antibiotic management.

CASE DESCRIPTION

A 68-year-old male with a history of atherosclerotic coronary artery disease and severe aortic valve stenosis presented to the emergency department with a one-month history of fever and exertional dyspnea. His medical history included a non-ST-elevation myocardial infarction (NSTEMI), heart failure, severe aortic stenosis, diabetes mellitus type 2, and dyslipidemia. On admission, he was afebrile but tachycardic (heart rate: 124 bpm) and hypertensive (blood pressure: 160/100 mmHg). Cardiac examination revealed a holosystolic murmur over the aortic valve. Electrocardiography (ECG) showed sinus tachycardia. It is noteworthy that during a thorough clinical examination, Janeway lesions were observed on his palms. Laboratory findings showed leukocytosis (19,130 cells/ μ l, 85% polymorphonuclear cells), hyperglycemia (glucose: 261 mg/dl), elevated C-reactive protein (3.5 mg/dl; normal <0.7), an erythrocyte sedimentation rate of 94 mm/h, significantly elevated troponin (7000 pg/ml; normal <0.017), and microcytic hypochromic anemia.

The patient was admitted for further evaluation. Transthoracic echocardiography confirmed severe aortic valve stenosis and heart failure with preserved ejection fraction. Although transesophageal echocardiography initially did not reveal vegetations, infective endocarditis remained highly suspected. A computed tomography (CT) scan of the abdomen, brain, and chest showed micro ischemic lesions in the left pons and right basal ganglia, splenomegaly (14 × 17 × 7 cm) with an ischemic splenic infarct (Fig. 1A), and a suspected rectal polyp. Fundoscopic examination did not reveal Roth spots.

Within days, *S. alactolyticus* was isolated from multiple blood cultures. The patient met one major and three minor Duke's criteria, confirming the diagnosis of infective endocarditis. Antibiotic susceptibility testing showed beta-lactam resistance (Table 1), prompting treatment with vancomycin and gentamicin, with close monitoring of serum drug levels. Given the severity of aortic stenosis, the patient

Antimicrobial	MIC	Interpretation
Benzylpenicillin	0.5	I
Ampicillin	2	I
Cefotaxime	0.5	S
Ceftriaxone	1	R
Gentamicin	≤ 64	
Erythromycin	≤ 0.12	IE
Clindamycin	≤ 0.25	S
Teicoplanin	≤ 0.12	S
Vancomycin	0.5	S
Tigecycline	≤ 0.06	IE

Abbreviations: S, susceptible; I, intermediate; R, resistant; IE, insufficient evidence that species is a good target for therapy. MIC may be reported without interpretation.

Table 1. Antibiotic susceptibility profile of *S. alactolyticus* isolated from multiple blood cultures.

was scheduled for valve replacement surgery. Despite initial clinical and laboratory improvement, the patient developed recurrent fever on day 14 of targeted therapy, despite maintaining therapeutic antibiotic levels through drug monitoring. A repeat CT scan revealed multiple hypo vascular splenic lesions suggestive of micro abscesses (Fig. 1B). Transesophageal echocardiography (TEE) identified a hypoechoic mass at the non-coronary leaflet of the aortic valve, with a small communication, indicative of an abscess formation. Although follow-up blood cultures remained negative, antimicrobial therapy was escalated to include meropenem, daptomycin, and vancomycin. However, the patient experienced progressive cardiopulmonary decline and succumbed to multi-organ failure two weeks after fever recurrence. Due to the severity of the patient's condition, a colonoscopy was not performed.

DISCUSSION

S. alactolyticus is a rare human pathogen. While the broader is well known for its association with endocarditis and colorectal malignancies, *S. alactolyticus* remains an uncommon but increasingly recognized cause of IE. Reported

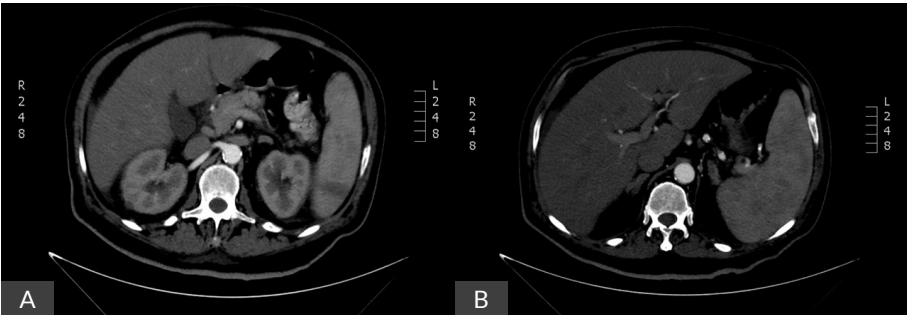


Figure 1. Abdominal computed tomography (CT) scans performed during the patient's hospitalization: A) Initial abdominal CT scan showing an ischemic infarct in the lower pole of the spleen; B) CT scan after clinical deterioration revealing hypo vascular splenic lesions suggestive of micro abscess.

Case reports	Presentation	Therapy and outcome
Almeida et al. ^[2]	<i>S. alactolyticus</i> bacteremia; 65 years/female; Fever, brain renal splenic emboli, MCA mycotic aneurysm; Cardiac history: Hypertrophic obstructive cardiomyopathy, MVP; TEE: Large MV/AoV vegetation, severe MVR/AoVR;	Antibiotics: VAN/GEN/PEN (empiric), CRO (de-escalation); Surgery: Bioprosthetic AoV/MV replacement (after antibiotic treatment); MCA mycotic aneurysm neurosurgical clipping (after cardiac surgery recovery); Outcome: Recovery
Cekmen et al. ^[3]	<i>S. alactolyticus</i> bacteremia; 64 years/male; Fever, acute cardiorespiratory failure, no vascular phenomena; Cardiac history: CABG; TEE: Large AoV vegetation, severe AoVR. Moderate MVR;	Antibiotics: VAN/GEN/PEN (empiric), CRO (de-escalation); Surgery: Bioprosthetic AoV/MV replacement (urgent, during antibiotic treatment); Outcome: Recovery
Mylonas et al. ^[4]	<i>S. alactolyticus</i> bacteremia (GEN resistance); 64 years/male; Low grade fever, acute myocardial ischemia, brain, splenic emboli, spondylodiscitis suspicion; Cardiac history: MVP; TEE: MV vegetation (features of chronicity);	Antibiotics: GEN/CIP/DAP (empiric), CRO (de-escalation); Surgery: MV replacement (after antibiotic treatment in other hospital); Outcome: Recovery
Vinciguerra et al. ^[5]	<i>S. alactolyticus</i> bacteremia; 69 years/male. Fever, cervical spondylodiscitis, spleen infarction; Cardiac history: / TEE: MV vegetations (max 11 mm)	Antibiotics: GEN/CRO; Surgery: Bioprosthetic MV replacement (during antibiotic treatment); Outcome: Recovery
Punama et al. ^[6]	<i>S. alactolyticus</i> and <i>Kocuria kristinae</i> bacteremia; 25 years/female; Chest pain, thrombocytopenia, anemia, septic embolism, SLE suspicion; Cardiac history: / TEE: MV vegetation;	Antibiotics: CTX/GEN (empiric), LVX/GEN (antibiogram-based); Surgery: Scheduled for valve replacement; Outcome: Death
Gherlan et al. ^[7]	<i>S. alactolyticus</i> bacteremia (TIG resistance); 48 years/male; Fever, lumbar pain, no vascular phenomena. Cardiac history: Bicuspid aortic valve; TEE: AoV vegetation (max 13 mm), AoV perforation, severe AoVR;	Antibiotics: CRO; Surgery: Mechanical AoV prosthesis (during antibiotic treatment). Outcome: Recovery
Chakrabarty et al. ^[8]	<i>S. alactolyticus</i> bacteremia 31 years/female; Brain emboli - subarachnoid hemorrhage, MCA mycotic aneurysm; Cardiac history: MVP; TEE: MV vegetation (max 13 mm); severe MVR;	Antibiotics: VAN/FEP/MTZ (empiric), CRO (de-escalation); Surgery: MV replacement deferred due to recent hemorrhagic stroke, microsurgical resection and clipping of MCA pseudoaneurysm; Outcome: Recovery
This case	<i>S. alactolyticus</i> (b-lactam resistance); 68 years/male; Fever, dyspnea; splenic infarct, splenic abscesses; Cardiac history: Severe AoVS; TEE: Severe AoVS, no vegetations (1st), AoV abscess formation (2nd two weeks later).	Antibiotics: VAN/GEN (antibiogram based), MEM/DAP/VAN (escalation); Surgery: Scheduled for valve replacement. Outcome: Death

Abbreviations: TEE, transoesophageal echocardiography; MV, mitral valve; AoV, aortic valve; MVR, mitral valve regurgitation; AoVR, aortic valve regurgitation; MCA, middle cerebral artery; MVP, mitral valve prolapse; CABG, coronary artery bypass grafting; VAN, vancomycin; GEN, gentamicin; PEN, penicillin G; CRO, ceftriaxone; CIP, ciprofloxacin; AMC, amoxicillin-clavulanic; MTZ, metronidazole; FEP, cefepime; DAP, daptomycin; MEM, meropenem; CTX, cefotaxime; LVX, levofloxacin; TIG, tigecycline.

Table 2. Case reports of infective endocarditis caused by *Streptococcus alactolyticus*.

cases exhibit a wide spectrum of clinical presentations, therapeutic responses, and outcomes^[2-8]. Table 2 presents a comparative analysis of all *S. alactolyticus* IE cases reported in the literature so far.

Most cases occur in older adults with predisposing cardiac conditions, though younger patients have also been affected, sometimes with fatal outcomes. Fever and embolic events - such as splenic infarctions, brain emboli, and spondylodiscitis - were common presentations. Structural heart disease, particularly mitral valve prolapses (MVP) and congenital abnormalities like a bicuspid aortic valve, was a frequent predisposing factor. However, in two reported cases, no pre-existing cardiac conditions were identified, suggesting that *S. alactolyticus* can also affect structurally normal hearts^[5,6]. Standard therapy for streptococcal IE typically included beta-lactams, often combined with aminoglycosides for enhanced bactericidal activity. Most patients initially received empiric antibiotic therapy with vancomycin, gentamicin, and penicillin, later adjusted to ceftriaxone. Valve replacement was frequently required, either during antibiotic therapy or after completing antimicrobial treatment.

Our case presents several key differences compared to previously documented instances of *S. alactolyticus* IE, making it a particularly noteworthy contribution to the literature. A major distinguishing feature was confirmed beta-lactam resistance, necessitating a significant shift in antimicrobial therapy. Initial treatment with vancomycin and gentamicin proved ineffective, requiring escalation to meropenem, daptomycin and vancomycin - reflecting an evolving resistance pattern. While resistance to gentamicin and tigecycline has been reported, this is the first documented case of beta-lactam resistance, highlighting an emerging therapeutic challenge^[4,7].

Another challenge was the absence of initial echocardiographic evidence of vegetations. Unlike most cases where vegetations were detected early via TEE, the first TEE in our patient showed no vegetations despite severe aortic valve stenosis. A repeat TEE two weeks later revealed aortic valve abscess formation, emphasizing the importance of serial imaging in diagnostically challenging cases. Regarding the patient's previous cardiac history, he had severe aortic valve stenosis, a relatively uncommon predisposing factor among the reviewed cases.

Our case also aligns with prior reports highlighting the risk of systemic embolization in *S. alactolyticus* IE. However, the combination of splenic infarction and abscess formation distinguished our case from the others. While splenic infarcts were reported in multiple cases^[2,4,5], progression to splenic abscesses was unique. Other cases exhibited systemic embolization, including brain emboli and mycotic aneurysms, but without localized abscess development^[2,4,8].

Unlike most cases, which resulted in recovery following antibiotic therapy and/or surgical intervention, our patient succumbed to the infection. The only other fatality among reviewed cases was reported by Punama et al., involving a young patient with dual bacteremia (*S. alactolyticus* and

Kocuria kristinae) and severe septic embolization^[6]. Delayed diagnosis, beta-lactam resistance, and severe disease progression likely contributed to the poor prognosis in our case.

Given the rarity of *S. alactolyticus* as a human pathogen and its emerging role in IE, heightened clinical vigilance is essential for patients with structural heart disease and persistent bacteremia. The rising incidence of beta-lactam resistance underscores the need for routine antimicrobial susceptibility testing to guide targeted therapy. Despite adherence to guideline-directed treatment, this case highlights the risk of embolic complications and hemodynamic deterioration. Serial imaging, particularly TEE, is crucial for early detection of complications such as abscess formation. Future research should focus on elucidating the pathogenic mechanisms of *S. alactolyticus*, tracking resistance patterns, and optimizing therapeutic strategies to improve outcomes in this rare but potentially lethal infection.

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