Serratia endocarditis, uncommon organism, with significant complications

Anu Anna George,[1] Tejveer Singh,[1] Pradnya Brijmohan Bhattad,[2] Akil A. Sherif,[2] Ajay Kumar Mishra[2]

[1]Department of Internal Medicine; [2]Department of Clinical Cardiology, Saint Vincent Hospital, Worcester, MA, USA

Abstract

Serratia marcescens is an aerobic, Gram-negative bacillus predominantly seen in patients with intravenous drug use, immunosuppression, previous antibiotic exposure, and indwelling catheterization. Gram-negative organism causing infective endocarditis (IE) is rare. *Serratia marcescens* IE is uncommon and is reported to be seen in 0.14% of all cases. In this report, we discuss in detail about a 38-year-old man with a history of intravenous drug abuse presenting with *S. marcescens* related prosthetic valve IE.

Introduction

Serratia marcescens was first identified in 1819, and *S. marcescens* related infective endocarditis was first reported in 1951 [1]. S. *marcescens* is a facultative aerobic, motile, oxidase-negative, non-lactose-fermenting, Gram-negative bacillus of the family Enterobacteriaceae. It is not present in human commensal flora, and infections are mostly acquired in hospitals. It has fimbria-like adhesions, allowing for surface attachment and biofilm formation causing higher host invasion and infection [1,2]. Commonly patients with S. *marcescens*, present with urinary tract infection, pneumonia, wound infections, surgical site infections, skin and soft tissue infections, and bacteremia. Patients with intravenous drug use, immunosuppression, previous antibiotic exposure, and indwelling catheterization are at higher risk of acquiring this infection [2,3]. Infective endocarditis secondary to *S. marcescens* is rare and has been reported in 0.14% of all cases.

Correspondence: Dr. Anu Anna George, MD, Department of Internal Medicine, Saint Vincent Hospital, 123 Summer St, Worcester, MA 01608, USA.
Tel. +1.508.363-5000.
E-mail: Anu.George@stvincenthospital.com

Key words: infection; endocarditis; Serratia, complications.

Contributions: all authors played a significant role in the paper, have seen the manuscript and agree to the content and data.

Conflict of interest: the authors have no conflicts of interest to declare.

Ethics approval: the article does not contain the participation of any human being and animal.

Patient consent: obtained.

Received: 11 October 2022.
Accepted: 13 January 2023.
Early view: 2 February 2023.

Licensee PAGEPress, Italy
Monaldi Archives for Chest Disease 2023; 93:2453
doi: 10.4081/monaldi.2023.2453

Case Report

A 38-year-old gentleman with a history of intravenous drug abuse, prior mitral valve endocarditis (initial methicillin susceptible *Staphylococcus aureus* infection 2 years prior to presentation) requiring mitral valve replacement with bioprosthetic Saint Jude mitral valve, hepatitis C, cirrhosis with portal hypertension, anxiety, depression, and polysubstance abuse presented with a history of high-grade fevers and chills for 5 days. At the time of presentation, he also complained of insidious onset, persistent, mild shortness of breath. On examination, he was febrile with a temperature of 38.9°C, tachycardic with a heart rate of 118/min with the remaining vitals being normal. Cardiovascular examination revealed a pan systolic murmur over the mitral area. Respiratory examination revealed decreased breath sounds predominantly over the infrascapular regions bilaterally. On investigation, he was found to have leukocytosis (white blood cell count of 13,700) and thrombocytopenia with (platelets of 39,000). He was noted to have lactic acidosis with (lactate of 2.7). On his echocardiogram, he was noted to have large vegetation on the bioprosthetic mitral valve, and the anterior lateral annulus (Figure 1 a,b). He was also noted to have dehiscence of the prosthetic mitral valve without severe paravalvular mitral regurgitation. Additional vegetation was also noted on the tricuspid valve and pulmonic valve. Chest tomography showed bilateral pleural effusion and right-sided airspace disease (Figure 1c). Blood culture was positive for *S. marcescens* (Figure 1 d,e).

He was treated with ceftazidime as per the sensitivity pattern, following the recommendation of infectious disease specialists. The patient was discharged to rehab in a stable condition with advice to complete a four-weeks course of ceftazidime following the first negative blood culture.

The patient continued to be stable until his relapse into intravenous drug use. Three weeks after his discharge he presented to the hospital with a recurrence of fever. At this point his repeat blood cultures were negative, however, repeat TEE showed new vegetation involving the tricuspid valve and a reduction in left ventricular ejection fraction to 35% (Figure 2). In the echo patient was noted to have persistent dehiscence of the prosthetic mitral

valve with severe paravalvular mitral regurgitation and worsening tricuspid regurgitation. In view of the recent history of *Serratia* endocarditis, which was sensitive to ceftazidime, antibiotics were continued as per infectious disease recommendations. The patient continued to deteriorate clinically and developed severe acidosis, worsening acute decompensation of heart failure with reduced

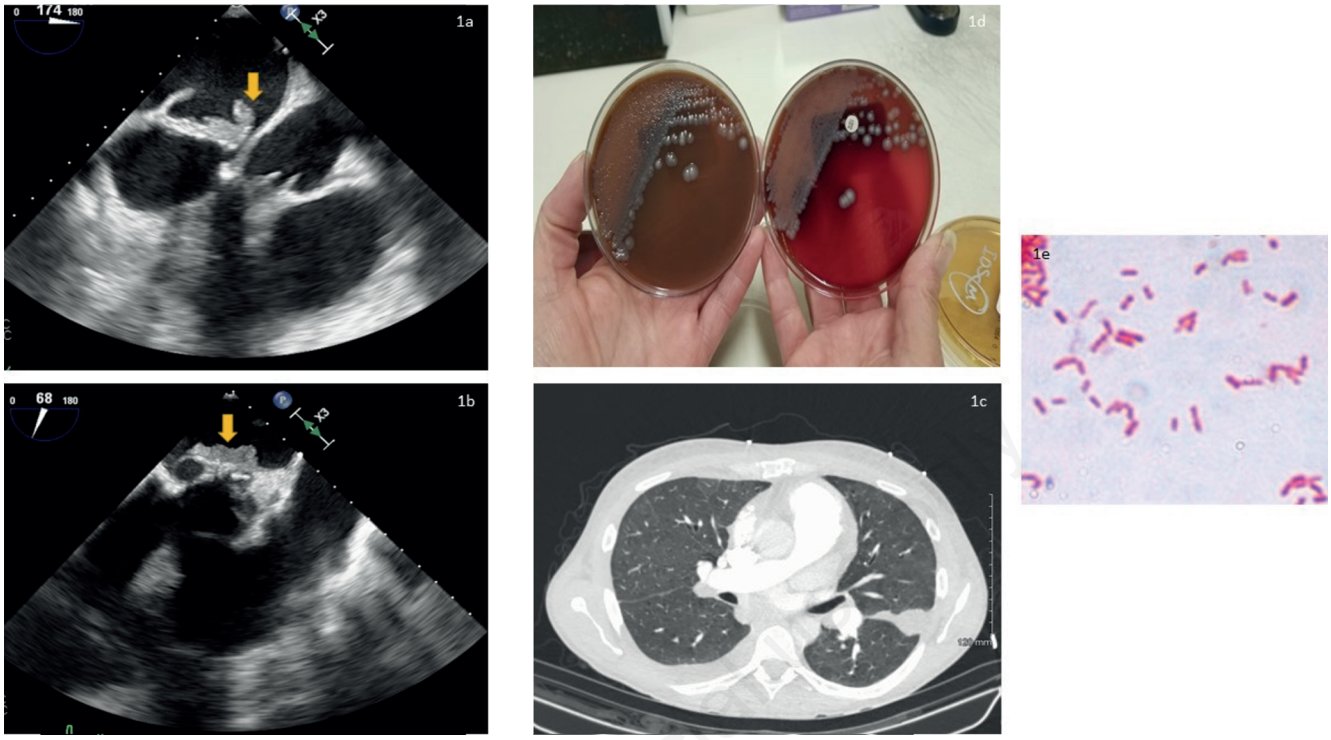

Figure 1. a,b) Echocardiogram showing vegetation on the bioprosthetic mitral valve, and the anterior lateral annulus (yellow arrow). c) Chest tomography showing pleural effusion and right-sided airspace disease. d,e) Blood agar and chocolate agar showing colonies of *Serratia marcescens* and gram stain showing gram-negative bacilli.

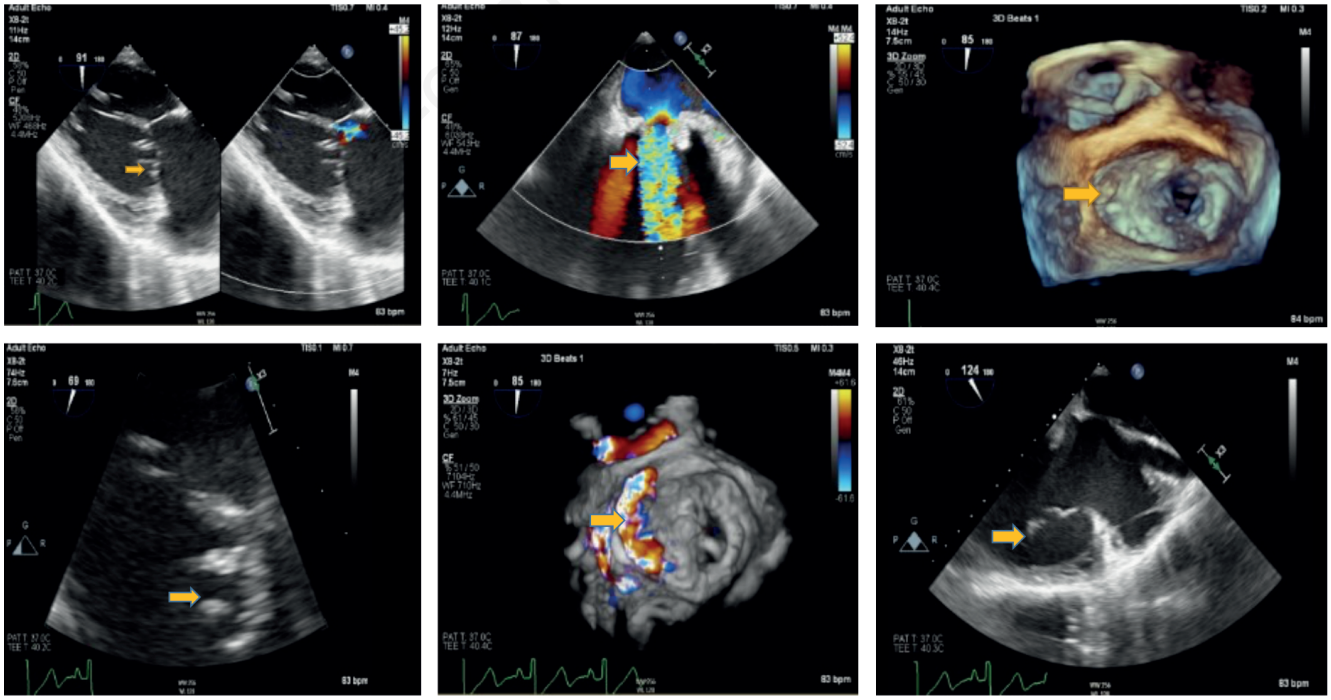

Figure 2. a,b,f) TEE showing tricuspid valve vegetation (yellow arrow). c) Color Doppler showing eccentric mitral regurgitation. d,e) 3D echo showing prosthetic mitral valve paravalvular regurgitation.

Figure 3. Course of the patient's disease.

ejection fraction, renal failure, and bicytopenia requiring endotracheal intubation, mechanical ventilation, and inotropic agent (Figure 3). Cardiothoracic surgery was consulted, and the patient was deemed medically unfit for cardiothoracic surgery. Despite optimal medical management, the patient succumbed to his illness.

Discussion

IE secondary to *Serratia marcescens* is rare. Patients present with similar clinical presentation as that of typical organisms causing IE [2,4]. Most patients with *S. marcescens* related IE have a history of intravenous drug use (IVDU) and indwelling catheterization. Even though patients with IVDU are predisposed to right-sided infective endocarditis, Serratia marcescens related IE among IVDU users can involve both right and left-sided valves like our patient [1,5]. In patients with IE this organism is usually resistant to penicillin and first and second-generation cephalosporins due to the production of AmpC beta-lactamase. Typically, most patients respond well to the third and fourth ingestion of cephalosporins, similar to our patient. *S. marcescens* related IE has been reported to cause higher morbidity and mortality [1,5,6]. There have been reports of valve destruction, paravalvular abscess, and distant embolic events in the past. Multiple, bilateral, valvular involvement is common alike our patient. Our patient also had severe mitral valve regurgitation and paravalvular leak [5,7]. In patients with left-sided IE development of conduction block, complete AV block should warrant transesophageal echocardiogram to rule out aortic root abscess [1,5,8]. In our patient, EKG did not have any evidence of conduction abnormality, and TEE was negative for any aortic root abscess. In patients with *S. marcescens* related left-sided IE, surgical management has been recommended to improve outcomes within 7 to 10 days [1,6,9,10]. Despite receiving appropriate antibiotics, as our patient was deemed unfit for surgery he unfortunately succumbed to his illness (Figure 1) [6,8,11,12].

In a recent review of 72 patients with *S. endocarditis* 18% of patients were noted to have prosthetic valve-related endocarditis. The most common clinical presentations included embolic phenomenon and heart failure. Transthoracic echocardiogram was diagnostic of infective endocarditis and only 35% of patients with the most commonly involved valve was mitral valve. Recent studies have reported that in patients with gram-negative bacteremia and cardiovascular infection transesophageal echocardiogram and FDG PET CT scan was superior in diagnosing endocarditis similar to our patient who was noted to have newer vegetations at each TEE. The meta-analysis including *Serratia* endocarditis patients reported a mortality of around 47%. Early surgery and appropriate antibiotic treatment were associated with reduced mortality. Despite appropriate antibiotic treatment in our patients, recurrence of infection, and worsening of clinical syndrome and subsequent mortality occurred due to reuse of IVDU. A recent study reported that IVDU patients with infective endocarditis tend to have multiple psychiatric comorbidities, prior episodes of infective endocarditis and multiple socioeconomic issues, which contribute towards development of distal embolus, requirement of recurrent admissions, recurrent bacteremia, and overall mobility alike our patient who developed clinical deterioration following drug use.

Prevention continues to be significant in reducing *S. marcescens* related infections as it is frequently acquired from contaminated fluids, medical devices, and medications in the hospital. Infection control, and hygiene, heightened vigilance continues to be crucial in these patients [13,14].

Patients with infective endocarditis secondary to virulent organisms like Serratia would benefit from timely follow-up, optimization of management of psychiatric comorbidities, and medically facilitated deaddiction.

Conclusions

Infective endocarditis secondary to *S. marcescens* is mostly reported in intravenous drug users and healthcare contacts. These patients tend to present with left-sided valvular involvement. They also tend to have higher local and systemic complications. Despite appropriate antibiotics and surgical management, mortality tends to be higher.

References

1. Richardson A, Martinez A, Ghetiya S, et al. Serratia marcescens endocarditis with perivalvular abscess presenting as atrioventricular block. Case Rep Infect Dis 2020;2020:7463719.
2. Yeung H-M, Chavarria B, Shahsavari D. A complicated case of Serratia marcescens infective endocarditis in the era of the current opioid epidemic. Case Rep Infect Dis 2018;2018:5903589.
3. Mishra AK, Sahu KK, Lal A, Menon V. Aortic valve abscess: Staphylococcus epidermidis and infective endocarditis. QJM 2020;113:211-2.
4. Sahu KK, Mishra AK, Sherif AA et al. An interesting case of pacemaker endocarditis. Neth Heart J 2019;27:585-6.
5. Mishra AK, Sahu KK, Lal A. Significance of prolonged PR interval in infections. QJM 2020;113:150-1.
6. Hadano Y, Kamiya T, Uenishi N. A fatal case of infective endocarditis caused by an unusual suspect: Serratia marcescens. Intern Med 2012;51:1425-8.
7. Mishra AK, Sahu KK, Lal A, Sujata M. Systemic embolization following fungal infective endocarditis. QJM 2020;113:233-5.
8. Sahu KK, Mishra AK, Lal A, Kranis M. An interesting case of expressive aphasia: Enterococcus faecalis-related infective

endocarditis complicating as septic emboli. QJM 2020; 113:146-7.
9. Mishra A, Sahu K, Nagabandi S, Benotti J. Infective endocarditis with mitral leaflet perforation and multiple embolic infarcts. QJM 2020;113:757-9.
10. Thomas VV, Mishra AK, Jasmine S, Sathyendra S. Gram-negative infective endocarditis: a retrospective analysis of 10 years data on clinical spectrum, risk factor and outcome. Monaldi Arch Chest Dis 2020;90:1359.
11. Mishra A, Sahu KK, Abraham BM, et al. Predictors, patterns and outcomes following Infective endocarditis and stroke. Acta Biomed 2022;93:e2022203.
12. Bakhit A, Mishra AK, Choudhary K, Khaled Soufi M. Aortic root fistula complicating Austrian syndrome. Monaldi Arch Chest Dis 2021;91:1834.
13. Mishra AK, Sahu KK, George AA, Lal A. Safety and efficacy of thrombolysis and mechanical thrombectomy in infective endocarditis. J Stroke Cerebrovasc Dis 2020;29:104784.
14. Phadke VK, Jacob JT. Marvelous but morbid: infective endocarditis due to Serratia marcescens. Infect Dis Clin Pract (Baltim Md) 2016;24:143-50.
15. Mishra AK, Sahu KK, Baddam V, Sargent J. Stroke and infective endocarditis. QJM 2020;113:515-6.
16. Mishra AK, Lal A, George AA. Letter by Mishra et al Regarding Article, "Infection as a stroke trigger: Associations between different organ system infection admissions and stroke subtypes". Stroke 2019;50:e328.
17. Dahl A, Hernandez-Meneses M, Perissinotti A, et al. Echocardiography and FDG-PET/CT scan in Gram-negative bacteremia and cardiovascular infections. Curr Opin Infect Dis 2021;34:728-36.
18. Mishra AK, Abraham BM, Sahu KK, et al. Harms and contributors of leaving against medical advice in patients with infective endocarditis. J Patient Saf 2022;18:756-9.
19. George A, Alampoondi Venkataramanan SV, John KJ, Mishra AK. Infective endocarditis and COVID -19 coinfection: An updated review. Acta Biomed 2022;93:e2022030.
20. Ioannou P, Alexakis K, Spentzouri D, Kofteridis DP. Infective endocarditis by Serratia species: a systematic review. J Chemother 2022;34:347-59.