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BEYOND PNEUMONIA AND SEPSIS : MRSA TRICUSPID VALVE INFECTIVE ENDOCARDITIS PRESENTING WITH SEPTIC PULMONARY EMBOLI : A CASE REPORT

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ABSTRACT Background: Right-sided infective endocarditis (IE) is an uncommon but potentially life-threatening condition that often presents with nonspecific respiratory and systemic manifestations, leading to delayed diagnosis. Methicillin-resistant Staphylococcus aureus (MRSA) is a highly virulent pathogen associated with persistent bacteremia, septic embolization, and significant morbidity. Early recognition of infective endocarditis remains crucial for preventing severe complications and improving clinical outcomes. Case Summary: A 21-year-old male presented with fever, respiratory distress, generalized weakness, decreased urine output, and burning micturition. Initial evaluation suggested lower respiratory tract infection, urosepsis, and acute kidney injury. Blood and urine cultures grew methicillin-resistant Staphylococcus aureus (MRSA). Transthoracic echocardiography revealed an 18 × 12 mm tricuspid valve vegetation, while computed tomography pulmonary angiography demonstrated septic pulmonary emboli. The findings fulfilled the Modified Duke Criteria for definite MRSA tricuspid valve infective endocarditis. The patient was managed with targeted antimicrobial therapy and multidisciplinary supportive care. Conclusion: This case highlights the importance of considering infective endocarditis in patients presenting with unexplained sepsis, persistent bacteremia, respiratory manifestations, and multiorgan dysfunction. Early echocardiographic evaluation and microbiological investigation are essential for the timely diagnosis of right-sided MRSA infective endocarditis. Prompt recognition and multidisciplinary management can help prevent severe complications and improve patient outcomes.

KEYWORDS : Infective endocarditis; Methicillin-resistant Staphylococcus aureus (MRSA) bacteremia; Tricuspid valve vegetation; Septic pulmonary emboli; Sepsis; Acute kidney injury; Lower Respiratory Tract Infection.

INTRODUCTION

Infective endocarditis is a microbial infection of the endocardial surface of the heart and remains associated with significant morbidity and mortality despite advances in antimicrobial therapy and diagnostic techniques.[1,2] Right-sided infective endocarditis accounts for approximately 5–10% of all cases and frequently presents with respiratory manifestations secondary to septic pulmonary embolization.[2,3] This atypical presentation often delays diagnosis.[3] We report a case of definite MRSA tricuspid valve infective endocarditis presenting as sepsis with respiratory and renal involvement.

CASE PRESENTATION

A 21-year-old male presented to the Emergency Department of tertiary care hospital of Vadodara, Gujarat with a 10–15 days history of intermittent high-grade fever associated with productive cough, progressive respiratory distress, generalized weakness, decreased urine output, and burning micturition. On presentation, he was febrile (101.4°F), tachycardic (156 beats/min), tachypneic (42 breaths/min), and hypoxemic, with an oxygen saturation of 84% on room air. His blood pressure was 124/76 mmHg. In view of severe hypoxemic respiratory failure and increased work of breathing, non-invasive ventilatory support in form of BiPAP was initiated promptly. On examination, the patient was conscious, alert, and oriented with time, place and person, along with pallor present. No peripheral stigmata of infective endocarditis including Osler nodes, Janeway lesions, splinter hemorrhages, petechiae, and clubbing noted. On respiratory examination, bilateral crepitations were present, with decreased air entry over the left lower lung field. Cardiovascular examination showed normal heart sounds without an audible murmur. Abdominal examination was unremarkable. Neurological examination revealed a Glasgow Coma Scale score of 15/15 with no focal neurological deficits. Based on the presenting complaints of fever, cough, respiratory distress, decreased urine output, and burning micturition, along with clinical examination findings, the initial provisional diagnosis was lower respiratory tract infection, acute kidney injury, and urosepsis.

INVESTIGATIONS

Initial laboratory investigations demonstrated severe anemia, marked leukocytosis, elevated inflammatory markers, thrombocytopenia, acute kidney injury, and significantly raised D-dimer levels. Serial hematological and biochemical parameters obtained during hospitalization are summarized in Table 1. Microbiological evaluation revealed growth of methicillin-resistant Staphylococcus aureus (MRSA) in both blood and urine cultures. Transthoracic echocardiography demonstrated a mobile vegetation measuring 18 × 12 mm attached to the tricuspid valve. Owing to severe hypoxemia, tachypnea, and markedly elevated D-dimer levels (>100,000 ng/mL), Computed tomography pulmonary angiography (CTPA) was performed and demonstrated findings suggestive of septic pulmonary emboli.

Table 1: Serial laboratory investigations during hospitalization.

Parameter	Day 1	Day 2	Day 3	Day 4	Day 5
Hemoglobin (g/dL)	5.5	6.5	7.8	8.6	8.7
Total Leukocyte Count (/mm³)	40,710	25,190	15,100	12,810	12,630
Platelet Count (/mm³)	140,000	92,000	132,000	196,000	216,000
Serum Creatinine (mg/dL)	1.8	2.3	2.0	1.9	0.8
CRP (mg/L)	201.29	248.65	181.61	64.12	20.3
Blood Urea (mg/dL)	106.81	141.23	—	—	—
D-Dimer (ng/mL)	>100,000	—	—	—	—

The final diagnosis of infective endocarditis was established based on the Modified Duke Criteria. The patient fulfilled two major criteria, namely positive blood cultures for methicillin-resistant Staphylococcus aureus (MRSA), a typical causative organism of infective endocarditis, and echocardiographic evidence of a mobile tricuspid valve vegetation measuring 18 × 12 mm. Additionally, two minor criteria were present, including fever (101.4°F) and septic pulmonary emboli representing a vascular phenomenon. The presence of two major criteria was sufficient to classify the case as definite MRSA tricuspid valve infective endocarditis according to the Modified Duke Criteria.

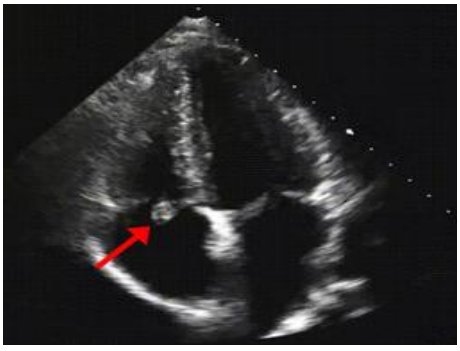

Figure 1: Apical 4-chamber view showing a large tricuspid valve vegetation

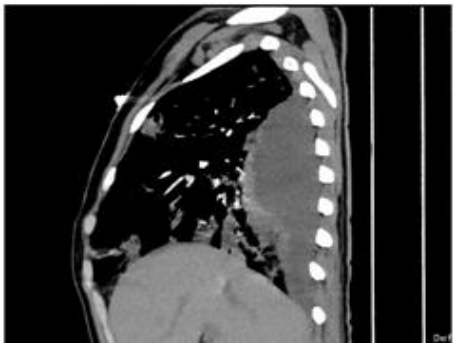

Figure 2: Blood culture growth of MRSA on Blood Agar.

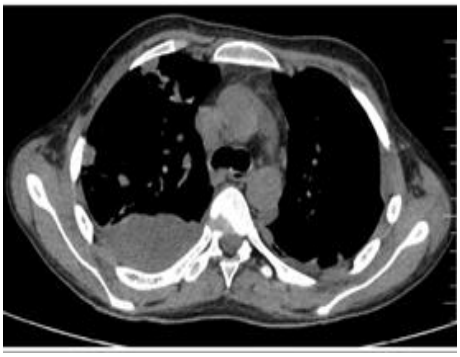

Figure 3: CTPA demonstrating septic pulmonary emboli. (Axial chest slice)

Figure 4: CTPA demonstrating septic pulmonary emboli.(Sagittal slice)

Table 2: Modified Duke Criteria

Category	Criterion
Major Criterion	Positive blood cultures for infective endocarditis: Typical microorganisms consistent with infective endocarditis from two separate blood cultures; or persistently positive blood cultures; or a single positive blood culture for Coxiella burnetii or anti-phase I IgG antibody titer ≥1:800.
	Evidence of endocardial involvement: Positive echocardiogram showing vegetation, abscess, or new partial dehiscence of a prosthetic valve; or new valvular regurgitation.
Minor Criterion	Predisposition: predisposing heart condition or intravenous drug use.
	Fever ≥38.0°C (100.4°F).
	Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, or Janeway lesions.
	Immunologic phenomena: glomerulonephritis, Osler nodes, Roth spots, or rheumatoid factor.
	Microbiological evidence not meeting a major criterion, including positive blood culture results or serological evidence of active infection with an organism consistent with infective endocarditis.

TREATMENT AND OUTCOME

The patient was managed with broad-spectrum antimicrobial therapy comprising meropenem (2 g), doxycycline (100 mg), and teicoplanin (800 mg), along with blood transfusion, respiratory support. Owing to severe hypoxemia at presentation, respiratory support was initiated with non-invasive ventilation (NIV), followed by stepwise de-escalation to oxygen via face mask, nasal prongs, and subsequently room air. Serial clinical and laboratory monitoring demonstrated resolution of fever, a decline in leukocyte count and inflammatory markers, improvement in renal function parameters, and stabilization of hemoglobin levels. In view of the tricuspid valve vegetation measuring 18 × 12 mm with septic pulmonary emboli, a CTVS opinion was sought. As there was no evidence of severe tricuspid regurgitation, refractory heart failure, persistent bacteremia despite appropriate antimicrobial therapy, recurrent embolic events, or other indications for urgent surgical intervention, the patient was advised continued conservative management with targeted antimicrobial therapy and close follow-up. The patient showed satisfactory clinical improvement and was discharged in a hemodynamically stable condition.

DISCUSSION

Right-sided infective endocarditis (RSIE) accounts for approximately 5–10% of all cases of infective endocarditis, with the tricuspid valve being the most commonly affected

structure. Unlike left-sided disease, RSIE frequently presents with pulmonary manifestations resulting from septic embolization, often mimicking pneumonia and contributing to diagnostic delays.[2,3]

In the present case, prolonged fever, cough, respiratory distress, oliguria, and burning micturition initially suggested lower respiratory tract infection, urosepsis, and acute kidney injury. However, persistent methicillin-resistant *Staphylococcus aureus* (MRSA) bacteremia, concomitant MRSA isolation from urine culture, and echocardiographic evidence of a large tricuspid valve vegetation (18 × 12 mm) raised suspicion for infective endocarditis. The diagnosis was further supported by CTPA findings of septic pulmonary emboli, fulfilling the Modified Duke Criteria for definite infective endocarditis.[4]

MRSA is a highly virulent pathogen associated with persistent bacteremia, large vegetations, and septic embolic complications. Septic pulmonary embolization, resulting from fragmentation of infected tricuspid vegetations, is a characteristic feature of RSIE and may manifest as peripheral pulmonary nodules, cavitary lesions, pulmonary infarction, or pleural effusions.[2 3] In our patient, severe hypoxemic respiratory failure requiring non-invasive ventilatory support was likely attributable to extensive septic pulmonary embolization.

Echocardiography remains the cornerstone of diagnosis, while CTPA is valuable for identifying septic pulmonary emboli and defining the extent of pulmonary involvement. Early recognition and targeted antimicrobial therapy are crucial to prevent recurrent embolization, persistent infection, and multiorgan dysfunction. Although surgery may be required in selected cases with refractory infection or recurrent embolic events, our patient responded favorably to medical management alone, with resolution of both systemic and respiratory manifestations.

This case underscores the importance of considering RSIE in patients with persistent fever, *S. aureus* bacteremia, and unexplained pulmonary infiltrates, as early diagnosis and appropriate treatment can significantly improve outcomes.

CONCLUSION

This case underscores the diagnostic challenge of right-sided MRSA infective endocarditis presenting as sepsis with respiratory manifestations and multiorgan dysfunction. The presence of persistent MRSA bacteremia, septic pulmonary emboli, and tricuspid valve vegetation fulfilled the Modified Duke Criteria for definite infective endocarditis. Early suspicion, timely echocardiographic evaluation, and targeted antimicrobial therapy are crucial for reducing morbidity and improving patient outcomes.

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