

# Enterococcal Native Tricuspid Valve Endocarditis in Acquired Immunodeficiency Syndrome: A Case Report and Short Review of Literature

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**Abstract:** *Tricuspid valve endocarditis (TVE) is usually reported in individuals with history of intravenous drug abuse or in those with preexisting structural heart disease. The clinical features of TVE range from acute febrile illness and pleuropulmonary symptoms to refractory right heart failure. We report a case of a 43-year-old acquired immunodeficiency syndrome (AIDS) patient with structurally normal cardiac valves and without any history of parenteral drug abuse or intravascular catheter use, presenting with prolonged fever and progressive dyspnoea, who was subsequently diagnosed with enterococcal tricuspid valve endocarditis. The case report highlights that AIDS should be considered as a differential diagnosis in TVE patients without any etiologies for TVE such as intravenous-drug use (IVDU), intracardiac device, or structural heart disease including congenital heart disease.*

**Keywords:** Tricuspid valve Endocarditis (TVE), Enterococcus, Intravenous drug use (IVDU), Acquired immunodeficiency syndrome (AIDS)

## 1. Introduction

Infective endocarditis (IE) usually involves the left side of the heart. Only in 5-10% of IE cases, the valves in the right side (e.g. tricuspid valve, pulmonary valve) are involved. When present, tricuspid valve endocarditis (TVE) is almost invariably associated with either a history of intravenous drug use (IVDU), or procedures like placement of pacemaker or other intracardiac devices or pre-existing structural heart disease.<sup>[1-4]</sup> The clinical features, management and the course of disease in right-sided IE differ from those in left-sided IE, with the former carrying a more favorable prognosis.<sup>[1-4]</sup> Thus, early diagnosis and management of right-sided IE is desirable. The frequency of involvement of different cardiac valves among IE patients with a positive history of IVDU is as follows: tricuspid valve alone or in combination - 73%; aortic valve - 7%; mitral valve - 6%, and aortic and mitral valves in combination - 1.5%.<sup>[3]</sup> Here, we report a case of TVE in a patient with morphologically normal valve and without history of IVDU, who was co-infected with human immunodeficiency virus (HIV). Further, we describe the complications that arose during the course of treatment, which were managed conservatively, with the patient eventually discharged in a clinically stable condition.

## 2. Case Report

A 43-years-male presented with 3 months history of recurrent fever with temperature spikes up to 103<sup>0</sup>F (39.4<sup>0</sup>C), dyspnea on exertion and cough. He had difficulty in breathing especially on climbing stairs and when attempting to sleep in supine position. He had no chest pain, sore throat, rash, edema of legs, dysuria or gastrointestinal symptoms. There was no past history of any cardiac disease, tuberculosis, diabetes mellitus or intravenous drug abuse. The patient had history of unprotected sex with multiple sexual partners and was found positive for HIV 5 years ago. Although he was then started on antiretroviral therapy (ART) (Zidovudine and Lamivudine), he discontinued medications two years after initiation of therapy. On physical examination, the patient looked ill and fatigued but there was no obvious respiratory distress. The patient was dehydrated, had raised body temperature (101<sup>0</sup>F), tachycardia (pulse rate of 110/minute) and tachypnea (respiratory rate 28 per minute); but was normotensive with normal peripheral oxygen saturation. On abdominal examination there was hepatosplenomegaly. Cardiovascular examination showed grade 4/6 pansystolic murmur over the left parasternal area with accentuation of murmur during inspiration. Other systemic examinations were unremarkable. Except for a raised ESR and positive CRP, his initial blood parameters were within normal limits. Enzyme Immunoassay (EIA) test for human

immunodeficiency virus (HIV 1) by was positive which was later confirmed by Western blot test. Serological studies for hepatitis B, hepatitis C and syphilis were negative. His CD4 lymphocyte count was 8.5% (absolute count of 36 cells/ $\mu$ L). Chest radiograph was normal and sonography of abdomen confirmed hepatosplenomegaly. The electrocardiogram was normal. Transthoracic echocardiography showed large vegetations attached to the anterior and septal cusps of tricuspid valve and severe tricuspid regurgitation with right ventricular systolic pressure of 50 mmHg; the mitral and aortic valves were normal. There was no regional wall motion abnormality and systolic function was normal with ejection fraction of 65% (Figures 1 and 2). Empirical antibiotic therapy was started with intravenous ceftriaxone (2 gm/day in divided doses twice daily) with other supportive medications. Blood culture and sensitivity report after 48 hours revealed growth of *Enterococcus* species sensitive to vancomycin, cephalosporin and imipenem. As the fever was persistent, antibiotic was switched to intravenous vancomycin (1.5 g/day in divided doses). The patient responded initially with disappearance of fever. However, after seven days of therapy, there was mild renal dysfunction which deteriorated to acute renal failure over the next 72 hours. Therefore, injection vancomycin was discontinued and hemodialysis was initiated. Alternatively, intravenous linezolid (1.2 g/day in divided doses) and imipenem (1.5 g/day in divided doses) were started. Gradually the renal function improved, but after five days there was severe pancytopenia. Thus, linezolid injection was also stopped, whereas imipenem injection was continued. Further, 4 units of platelets, and 2 units of packed cells were transfused. Gradually the patient improved, and repeat blood culture after 14 days was sterile. The patient was counseled for continuation of ART with zidovudine and lamivudine, and oral cotrimoxazole was advised as prophylaxis against opportunistic infections. He was discharged uneventfully and was doing well at six monthly follow up.

### 3. Discussion

Right-sided IE is a well defined entity, with risk factors, clinical presentation, echocardiography findings, and bacteriological profile distinct from that in left-sided IE. IVDU and intracardiac devices like pacemakers are the leading predisposing factors.<sup>[2-5]</sup> Thus, right-sided IE involving tricuspid valves in the absence of IVDU history or pacemaker implantation is a very rare phenomenon, supposedly present only in 5%-10% of right-sided IE cases. From previous studies, it is evident that right-sided IE in non-IVDU subjects who did not have a pacemaker, whenever present, was usually associated with other risk factors like intravascular catheter use, uncorrected congenital heart disease, puerperal sepsis, alcoholism or immunodeficiency.<sup>[4-6]</sup> In fact, immunodeficient HIV-positive patients with history of IVDU had 2.31 - 8.31 times greater odds of developing right-sided IE as compared to HIV-negative individuals.<sup>[4,7]</sup> Endocarditis in HIV-positive patients almost exclusively occurs in IVDU, which can be explained by the fact that IVDU is a major risk factor for HIV, and that endocarditis is one of the classical complications of IVDU [7-9]. Such patients in the presence of severe immunosuppression (CD4 count < 200 cells/ $\mu$ L) also have a very high risk of mortality.<sup>[7]</sup> However, in HIV-

patients without history of IVDU or other known predisposing factors for the matter, information on the clinical presentation and outcome of right sided endocarditis is very limited.<sup>[8]</sup>

Thus, establishing the clinical diagnosis of right-sided IE in such cases, as in our case is very challenging. Fever is often the predominant manifestation. But due to the absence of other known risk factors, endocarditis is usually not thought of initially. Often, the fever may be associated with pulmonary manifestations mimicking a respiratory tract infection.<sup>[2,4-5,9]</sup> Or sometimes, the respiratory symptoms may be accompanied by anaemia and microscopic haematuria - a condition referred to as 'tricuspid syndrome'.<sup>[10]</sup>

*Staphylococcus aureus* is the most common organism responsible for right-sided IE (including TVE) in both IVDU and non-IVDU subjects.<sup>[2,4-5]</sup> Other frequent pathogens are *coagulase-negative staphylococcus*, and *viridans group streptococci*.<sup>[4]</sup> *Enterococci* is rather rare in right-sided IE. In a large review of 121 cases of right sided infective endocarditis by Ortiz C et al of, *enterococcus* has been reported only in 2% of cases.<sup>[11]</sup> Due to a dearth of reliable bactericidal agents, the management of enterococcal IE has long been recognized as an important clinical challenge. Enterococci are relatively more resistant to  $\beta$ -lactam antibiotics, and hence penicillin monotherapy which often has good performance in staphylococcal or streptococcal IE is unlikely to yield good results in case of enterococcal IE. For effective action, anti-enterococcal therapy requires synergistic action of penicillin, ampicillin or vancomycin in combination with aminoglycosides (either gentamicin or streptomycin).<sup>[3,4]</sup> However, the toxicity incurred when using these recommended antibiotic combinations, as exemplified by our case, may compound the treatment difficulties. Besides, the emergence of multi-drug resistant strains may pose additional challenges.<sup>[3, 4]</sup>

### 4. Conclusion

To conclude, AIDS should be considered as a differential diagnosis in TVE patients without any etiologies for TVE such as intravenous-drug user (IVDU), intracardiac device, or structural heart disease including congenital heart disease. Antibiotic therapy should empirically cover for likely pathogens till the culture reports are available to avoid treatment failure.

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**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

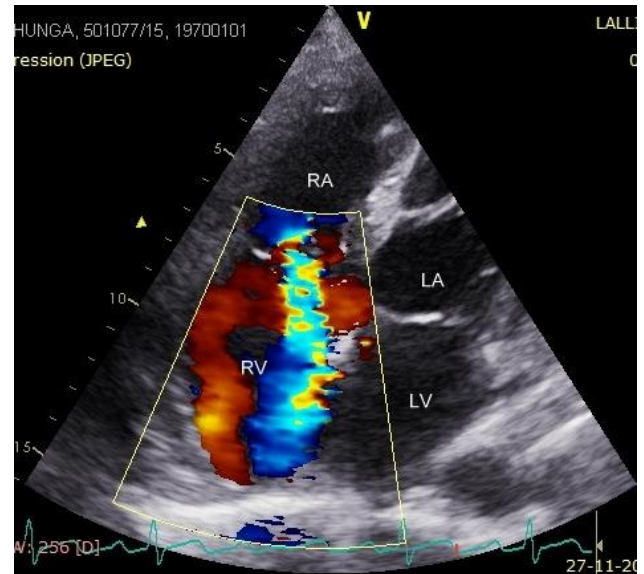
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**Data availability statement:** data underlying this article are available in the article and in its online supplementary material.

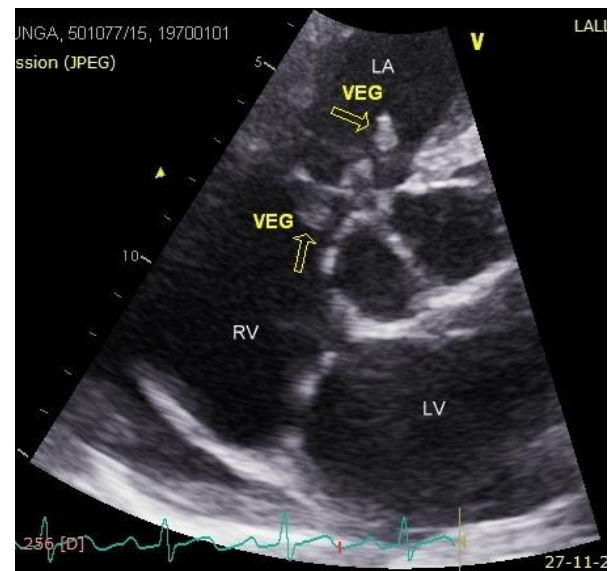
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## Figure Legends



**Figure 1:** Two dimensional echocardiography apical four chamber view showing moderate tricuspid regurgitation



**Figure 2:** Apical four chamber view showing large vegetation prolapsing through the tricuspid valve orifice