

Case Report:**An unusual case of native tricuspid valve endocarditis and sepsis in a child with structurally normal heart mimicking bronchopneumonia****D. Rajasekhar, V. Vanajakshamma, C. Shashanka, M.L. Srinivas Kumar, D. Sarath Babu***Department of Cardiology, Sri Venkateswara Institute of Medical Sciences, Tirupati***ABSTRACT**

Right sided infectious endocarditis (IE) by involvement of tricuspid valve is mainly associated with cardiac malformations or intravenous drug use. Occurrence of right-sided IE in children with structurally normal heart is exceptionally rare. We report the case of 9-year-old child presenting with bronchopneumonia and septicaemia who was diagnosed to have native tricuspid valve staphylococcal endocarditis and was successfully treated.

Key Words: *Tricuspid valve endocarditis, Native valve endocarditis, Endocarditis in children*

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INTRODUCTION

Right-sided infective endocarditis (IE) is common with intravenous drug use (IVDU) and accounts for 35%-60% of endocarditis cases in this population; tricuspid valve is most often involved.¹ Additional risk factors that have been described for tricuspid valve endocarditis include abdominal surgery, incomplete abortion, liver disease and cancer.² *Staphylococcus aureus* is the infecting organism in the majority (80%) of cases with tricuspid valve endocarditis. When *S. aureus* is involved, the course of the disease is commonly acute and rapidly progressive.³ Prior to the increase in the prevalence of IVDU observed in the recent past, right-sided IE was rare and occurred almost exclusively in patients with cardiac malformations. According to current estimates only 5%-10% of right-sided IE occurs in patients without IVDU.

CASE REPORT

A nine-year-old male child with a normal birth and developmental history, presented with a history of fever 2 month ago that lasted for about 15 days and was treated at a local hospital with intravenous injections from which he apparently recovered. No other records were available pertaining to this episode. Subsequently, he had a recurrence of high grade fever associated with cough, purulent expectoration and breathlessness.

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At admission, the child was febrile and toxic. He was haemodynamically stable. There were no skin or mucosal stigmata of endocarditis or venipuncture marks. Respiratory system examination was suggestive of right-sided lower lobe consolidation. On cardiovascular system examination, a systolic murmur suggestive of tricuspid regurgitation was present.

Laboratory examination showed normocytic anaemia (haemoglobin 10.3 g/dL), leukocytosis (total leukocyte count 10,800/mm³) with neutrophilia (polymorphs 85%) and normal renal and liver function tests. Erythrocyte sedimentation rate (ESR) at the end of the first hour was elevated (90 mm). Serological tests for human immunodeficiency virus (HIV) and hepatitis B surface antigen (HBsAg) were negative. Chest radiograph (Figure 1) showed bilateral basal

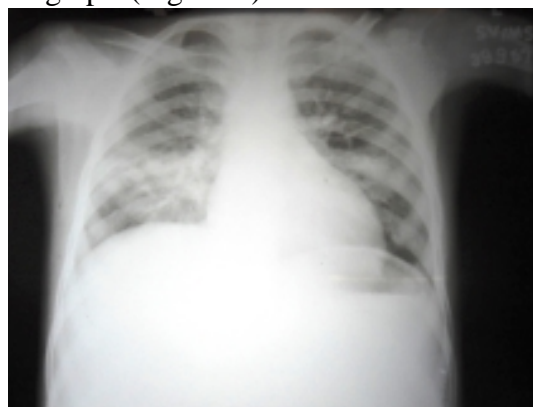


Figure 1 : Chest radiograph (postero-anterior view) showing bilateral parenchymal lesions suggestive of bronchopneumonia

Corresponding Author: Dr D. Rajasekhar, Dean, Professor and Head, Department of Cardiology, Sri Venkateswara Institute of Medical Sciences, Tirupati 517507, India. **e-mail:** sarathpgi@gmail.com

parenchymal lesions, more on the right-side suggestive of bronchopneumonia. Ultrasonography of the abdomen was normal. *Staphylococcus aureus* was grown in all three blood cultures. Sputum culture and urine culture were negative for any growth. Transthoracic echocardiography was normal except for a mobile, 6 x 4 mm, filiform vegetation on the septal leaflet of the tricuspid valve (Figure 2). There were no signs of vegetations in the mitral, aortic or pulmonary valves, and these were all normal. These findings were confirmed on transoesophageal echocardiography.



Figure 2: Echocardiogram (4-chamber view) showing mobile vegetation on the septal leaflet of tricuspid valve

A diagnosis of native tricuspid valve, staphylococcal endocarditis with septic pulmonary embolism was made and the patient was started on intravenous ceftriaxone therapy as per culture and sensitivity, for 28 days. The patient became afebrile, his general condition improved and subsequent chest radiographs showed complete clearance of parenchymal opacities (Figure 3). Repeat blood cultures were negative. Follow-up echocardiography showed a reduction in the size of the vegetation and increase in echogenicity indicating healing (Figure 4).

DISCUSSION

Isolated occurrence of native tricuspid valve endocarditis in subjects without underlying valvular heart diseases, central venous lines or IVDU non-addicted patients is an elusive disease which mimics several other illnesses. In children with normal hearts tricuspid valve endocarditis is an extremely uncommon disease. There are only five such reported cases in published literature in the



Figure 3 : Chest radiograph (posteroanterior view) obtained at the time of discharge showing complete resolution of bronchopneumonia

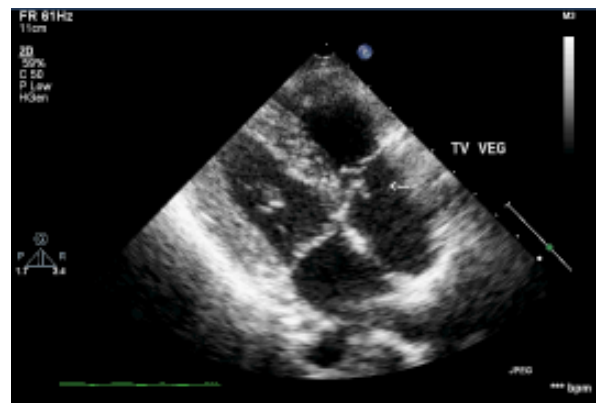


Figure 4 : Echocardiogram (4-chamber view) at the time of discharge showing significant decrease in the size, mobility and increase in the intensity of vegetation suggestive of healing endocarditis

paediatric age group.⁴ Intracardiac and intravenous catheters, abdominal surgery and hyperalimentation are predisposing factors for hospital-acquired right-sided endocarditis, whereas dental and cutaneous infections, alcoholism, liver disease, colon cancer and colonic procedures were identifiable risk factors in community-acquired cases with right-sided endocarditis.

Often, pulmonary rather than cardiac manifestations are usually the predominant clinical features of tricuspid valve endocarditis. Symptoms arising from pneumonia or septic pulmonary emboli from dislodged vegetations are common. The clinical presentation usually consists of persistent fever associated with pulmonary manifestations, anaemia and microscopic

hematuria, signs that constitute the "tricuspid syndrome".² The absence of peripheral stigmata of endocarditis or relevant murmurs in the majority of cases is noteworthy. Right-sided endocarditis involving the tricuspid valve should be included in the differential diagnosis of patients with staphylococcal or streptococcal bacteraemia accompanied by clinical and or radiological signs of pulmonary involvement. Even in the absence of classic predisposing risk factors for right-sided endocarditis, a diagnosis of right-sided endocarditis should be considered and echocardiography should be promptly carried out to document the presence of vegetation.⁴

The microbial etiology of endocarditis depends on the anatomic location of the lesion and the predisposing factors. *Staphylococcus aureus* is the most common microorganism causing tricuspid valve endocarditis; accounting for 50%-80% of all cases.⁵ Other pathogens implicated as aetiological agents include *Streptococcus* spp., Gram-negative bacilli (especially *Pseudomonas aeruginosa*) and *Candida* species.⁶ Although *Streptococcus* species may result in right-sided endocarditis in combination with a left-sided endocarditis, in patients with a history of IVDU or cardiac abnormalities, only a few cases of isolated tricuspid valve endocarditis are described in patients without cardiac abnormalities or IVDU. Similar to the situation in adults, aetiological organisms are also likely to shift towards those with a predilection for prosthetic material (*Staphylococcus epidermidis*) and repeated interventions (*Staphylococcus aureus*).

In a recently published series,⁶ 20% of IE occurred in children with no known predisposing factors; in five of the six children, the organism was *Staphylococcus aureus*. This emphasizes the potential threat of this organism even in patients with structurally normal hearts. In persons without IVDU, endocarditis arising from *Staphylococcus aureus* primarily involves the left-side of the heart and is associated with mortality rates ranging from 25% to 40%. *Staphylococcus aureus* endocarditis in IVDU often involves the tricuspid valve. Cure rates for right-sided *Staphylococcus aureus* endocarditis in IVDUs are high (>85%) and may

be achieved with relatively short courses of treatment (< 4 weeks). Weight of evidence suggests that parenteral β -lactam short-course therapy, with or without aminoglycoside, is adequate for the treatment of uncomplicated oxacillin-sensitive or community acquired *Staphylococcus aureus* right-sided IE.⁷

Interestingly, our patient presented with pleuropulmonary symptoms after a period of hospitalisation for fever. Our patient did not have any other classic risk factors for tricuspid valve endocarditis. The portal of entry of *Staphylococcus aureus* is not clear, though cutaneous route due to intravenous cannulation can be speculated. Superficial skin sepsis due to the multiple intravenous injections the patient had received might have also been the possible source.

As in any case with IE, the clinical picture, positive findings on blood culture and echocardiography are the main diagnostic tools in native tricuspid valve IE. The diagnosis of tricuspid valve endocarditis is often delayed because the cardiac manifestations are subtle and the murmur of tricuspid regurgitation is inconspicuous. It has been suggested that in every child with acute staphylococcal sepsis and no obvious primary focus, early 2D echocardiography should be carried out particularly if the fever does not abate despite antibiotic therapy.⁷⁻⁹

Early surgical management was not considered in our patient because of the good response to antibiotic therapy. In patients with tricuspid valve endocarditis surgical excision of the vegetations with or without prosthetic valve replacement is indicated in the presence of vegetations larger than 1 cm, congestive heart failure or coexistent left-sided endocarditis and is probably needed in 25% of adult patients. Only one of the reported five paediatric patients with tricuspid valve endocarditis was successfully managed by medical treatment alone whereas the remaining four had also undergone surgery (subtotal excision in 2 cases, valve replacement in 2 cases) with three of them surviving.⁴ If appropriate culture and sensitivity data are available, conservative therapy preserving the

native valve should translate into improved late survival, freedom from re-operation and functional outcomes.¹⁰

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