Case Reports

Tricuspid Valve Endocarditis in a Child with Structurally Normal Heart

G. Karthikeyan
P. Nalini
K.R. Sethuraman

We report a case of isolated tricuspid valve endocarditis (endocarditis afflicting a structurally normal heart) due to *Staphylococcus aureus* seen in a 3 years old male child. We consider that this may be the first instance of an isolated right sided endocarditis in a child being reported in the Indian literature.

From the Departments of Pediatrics and Medicine,Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry.

Reprint requests: Dr. G. Karthikeyan, 4, Singamthoppu 4th Street, Aruppukottai-626 101.

Received for publication: July 19,1995;
Accepted: February 22,1996

Case Report

The patient, a 3 years old male child presented with fever with chills and rigors of one month duration and breathlessness and swelling of face and feet of 7 days duration. Before being admitted in our hospital, he had received intermittent intravenous injections (IV) of ampicillin for the last 10 days and antimalarial drugs. There was no history of peripheral IV cannulation but there was a history of thrombophlebitis following IV injections and needle track scars were also present. He was conscious, febrile, dyspneic, pale, and had a puffy face with pedal edema. His temperature was 103° F; pulse was 160 per min, regular, BP was 90/60 mm Hg and respirations were 48 per min with chest retractions. JVP was elevated and pulsatile. Examination of cardiovascular system revealed marked tachycardia with an S3 gallop. Lung fields were clear. He had a 6 cm soft, tender hepatomegaly and a 4 cm firm splenomegaly. Neurological examination including fundus evaluation was normal. A diagnosis of prolonged fever with anemia with congestive heart failure was made and the possibilities of typhoid

fever and malaria were entertained. Blood culture was taken and intravenous ampicillin was administered along with diuretics.

Investigations revealed a Hb of 8.9 g/dl, total leucocyte count of 8800 with a differential count of N_{88} L_{12}, and ESR of 38 mm fall in 1st h. Peripheral smear showed normocytic, hypochromic RBCs with moderate anisopoikilocytosis, neutrophilic preponderance with toxic changes and a mild shift to left pattern and adequate clumps of platelets. Malarial parasites were not seen in smear. Blood urea was 34 mg/dl, sodium 134 meq/L, potassium 4.2 meq/L, chloride 96 meq/L, SGPT 30 IU, and alkaline phosphatase 100 IU. Urine microscopy was normal and urine cultures were sterile. ECG was within normal limits and X-ray taken at admission showed clear lung fields and mild cardiomegaly.

Spikes of 102° F continued and blood culture yielded growth of *Staphylococcus aureus* resistant to penicillin and ampicillin and sensitive to cefotaxime, cloxacillin, gentamicin, erythromycin and ciprofloxacin. A systolic murmur suggestive of tricuspid valve regurgitation was noted for the first time on the 3rd hospital day. Echocardiogram done on the 3rd hospital day showed vegetations on the tricuspid valve, thus establishing the diagnosis of tricuspid valve endocarditis (TVE). Antibiotics were changed to IV cloxacillin and gentamicin. Blood cultures sent on the 3rd and 4th hospital days also grew *Staphylococcus aureus.* Serum bactericidal titers were 1:8 on 6th hospital day. Blood cultures became sterile from the 6th hospital day. He became afebrile on the 10th hospital day but suffered a sudden death on the 11th hospital day while straining at stools. Request for an autopsy was declined.

Discussion

Tricuspid valve endocarditis without any pre-existing heart disease is primarily a disease of intravenous drug abusers. Non-drug abusers who suffer from TVE are patients with congenitally abnormal tricuspid valves (*e.g.,* Ebstein's anomaly), those with tricuspid valve prosthesis or those with mucosal endocarditis of right ventricle (*e.g.,* complicating left-right ventricular septal defect shunts with jet lesions or at the site of trans venous pacemaker wires(l,2). Septic abortion is an important etiological antecendent for TVE in Indian Experience 3). TVE affecting a structurally normal heart has been described in five neonates with indwelling central venous catheters (4).

TVE in children with normal hearts and without central venous catheters is an extremely uncommon disease. There are only 5 reported cases, to the best of our knowledge (5-7). Apart from fever, respiratory symptoms and a flue like illness were the presenting features in some cases(5,6) whereas infective diarrhea in a 7 year old girl and calcaneal osteomyelitis following heel puncture in a neonate had been the presenting features in another report(7). *Staphylococcus aureus* was the causative organism in all these cases described, with persistent bacteremia being a distinctive clinical feature.

Persistent *S. aureus* bacteremia was a dominant clinical feature in our patient but there was no obvious primary focus. Superficial skin sepsis due to the multiple intravenous injections the patient had received might have been the possible source. Superficial skin sepsis (boils) has been described to be the risk factor for TVE in three adult patients with structurally normal hearts and no evidence of narcotic abuse(8).

The diagnosis of TVE is often delayed because the cardiac manifestations are subtle and the murmur of tricuspid regurgitation is inconspicuous(l,6,7). In our patient also TVE was diagnosed after 2D echocardiography and the congestive failure which the patient had was initially attributed to severe anemia. 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(2,7,9).

Surgical excision of the vegetations with or without prosthetic valve replacement for TVE 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 adults patients(l,8). Only one of the reported five pediatric patients was successfully managed by medical treatment alone whereas the remaining four had also undergone surgery for TVE (subtotal excision 2 cases, valve replacement 2 cases) with three of them surviving. An early surgical management was not considered in our patient because of the response to antibiotic therapy.

Since an autopsy could not be performed upon our patient, we could only speculate that detachment of the vegetations and massive pulmonary embolism might have been the cause of sudden death. Appropriate antibiotic therapy early during his illness might have helped to achieve a better outcome. Nevertheless, it is known that emboli may continue to occur despite successful eradication of the infection(l,2).

REFERENCES

1. Chan P, Ogilby D, Segal B. Tricuspid valve endocarditis. Am Heart J 1989, 117: 1140-1146.
2. Bayer AS. Staphylococcal bacteremia and endocarditis: State of the art. Arch Intern Med 1982,142:1169-1177.
3. Grover A, Anand IS, Varma J, *et al.* Profile of right sided endocarditis: An Indian experience. Int J Cardiol 1991, 33: 83-88.
4. Noel GJ, O'Loughlin JE, Edelson PJ. Neonatal *Staphylococcus epidermidis* right sided endocarditis: Description of five catheterized infants. Pediatrics 1988, 82: 232-234.
5. Goessler MC, Riggs TW, De Leon S, Paul MH. Echocardiographic diagnosis of tricuspid valve endocarditis in a child with a normal heart. Pediatr Cardiol 1982, 2: 141-143.
6. Musewe NN, Hecht BM, Hesslein PS, Rose V, Williams WG. Tricuspid valve endocarditis in two children with normal hearts: Diagnosis and therapy of an unusual entity. J Pediatr 1987,110: 735-738.
7. Tornos MP, Castro A, Toran N, Girona J. Tricuspid valve endocarditis in children with normal hearts. Am Heart J 1989,118: 624-625.
8. Clifford CP, Eykyn SJ, Oakley CM. Staphylococcal tricuspid valve endocarditis in patients with structurally normal hearts and no evidence of narcotic abuse. OJ Med 1994, 87: 755-757.
9. Sheagren JN. *Staphylococcus aureus:* The persistent pathogen. N Engl J Med 1984, 310:1368-1370.