Infective Endocarditis of the Tricuspid Valve in a Non-Drug User

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A case of infective endocarditis of the tricuspid valve is described in a young female patient with no history of intravenous drug abuse. The patient suffered from symptoms of septic emboli of the lung and right heart failure. She was ultimately treated by tricuspid valve replacement. There was no recurrence of symptoms following surgery.

Key words: Infective endocarditis, Tricuspid regurgitation, Tricuspid valve replacement

CASE REPORT

A 26-year-old Japanese housewife was transferred from another hospital with persistent hemoptysis and high fever in May 1989.

She developed a fever of up to 38°C and a productive cough in October 1988, one month before the delivery of her first baby. Chest X-ray showed well demarcated infiltrates in the right middle and lower lung fields (Fig. 1). Antibiotics were given intravenously, and the fever subsided and her condition improved within two wk. She had a healthy baby by natural delivery. The fever and cough recurred in November 1988. She was re-admitted to another hospital. Chest X-ray showed the same pattern of infiltrates, but in a different site in the right lung. Several antibiotics were used with limited success. One blood sample yielded Staphylococcus aureus. She developed hemoptysis while in hospital. She remained in hospital for three months. The same symptoms recurred in May 1989, and she was admitted to a third hospital, where she stayed a wk, and then was transferred to our hospital.

On admission, she looked chronically ill. Her body temperature was 39°C, pulse 110/min, respira-
Infective Endocarditis in a Non-Drug User

Fig. 2. Doppler echocardiogram. Doppler echocardiogram shows severe tricuspid regurgitation (mosaic pattern in RA). Ao, aorta; LA, left atrium; RA, right atrium; RV, right ventricle

Fig. 3. Perfusion lung scan. The perfusion lung scan shows multiple cold areas (arrows).

Fig. 4. Tricuspid area at surgery. An almost completely destroyed tricuspid valve is viewed from the right auricle.

Fig. 5. Tricuspid area at surgery. An almost completely destroyed tricuspid valve is viewed from the right auricle.

Infective Endocarditis in a Non-Drug User

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tion rate 20/min, and blood pressure 120/70 mmHg. On examination, the external jugular vein was distended in a sitting position, and prominent v waves were observed. A Levine 3/6 pansystolic murmur was heard at the right sternal border. The lungs were clear. The liver was palpated 4 cm below the right costal margin. The extremities were normal. Urinalysis was normal. The white cell count was 25,500/mm³, hemoglobin was 9.4 g/dl. C-reactive protein was 40.8 mg/dl (N < 0.2 mg/dl). Blood samples taken after 48 h without the use of antibiotics revealed no bacterial growth. Doppler echocardiogram revealed marked tricuspid regurgitation. No vegetations were observed. The other valves were intact. There was no ventricular or atrial septal defect (Fig. 2). The perfusion lung scan showed multiple cold areas (Fig. 3). The diagnosis of infective endocarditis of the tricuspid valve, and pulmonary infarction due to septic emboli was made. The patient’s medical history was reconfirmed, and she strongly denied the use of intravenous drugs. There was no history of recent surgery or invasive procedures.

Tricuspid valve replacement was performed in July 1989. At surgery, the anterior and posterior leaflets were totally destroyed, and only the septal leaflet looked normal. There were no vegetations (Fig. 4). A porcine replacement valve was inserted. Her recovery was excellent, and she was discharged three wk after surgery with no medication. She was well and active twelve months after surgery.

DISCUSSION

In North America and Western European countries, infective endocarditis of the tricuspid valve is typically caused by intravenous narcotics abuse (1–4). The present patient revealed no history of narcotic drug use, and showed no signs of recurrence twelve months after discharge. Therefore, intravenous drug abuse was practically ruled out as the source of infection. The patient had had no recent surgery or invasive procedures. The first episode of pulmonary septic embolism characterized by typical chest X-ray findings (Fig. 1) occurred in October 1988. Although the first episode developed during pregnancy, it developed before delivery and the delivery was normal. The exact source of infection is therefore unknown in this case. However, the etiologic agent was most likely Staphylococcus aureus since it had been cultured from blood during the second hospital admission, and destruction of the valve seen during
the surgery was severe. The relatively long course of the present case of Staphylococcus infective endocarditis may be explained by its partial treatment with vigorous antibiotic administration at various hospitals. In Japan there have been only four reported cases of infective endocarditis of the tricuspid valve during the last decade, and none were caused by drug addiction. No source of infection was detected in three. Osteomyelitis was the source of infection in an 8-month-old boy (5). In Western countries there have also been cases reported in which the source of infection was unknown in pediatric patients with normal hearts (6, 7). In two pediatric cases reported by Musewe et al, tricuspid endocarditis was caused by Staphylococcus aureus septicemia following upper respiratory infection (7). This may also occur in adult cases. Further investigations should be carried out in the future to elucidate the source of infection.

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REFERENCES