

Tricuspid Valve Endocarditis

H. Thomas Milhorn, Jr., M.D., Ph.D.

Tricuspid valve endocarditis is a disease primarily found in intravenous drug abusers. It occurs much less often than left-sided endocarditis and has a better prognosis. The triad of intravenous drug abuse, *Staphylococcus aureus* septicemia, and pulmonary embolism with or without a tricuspid regurgitation murmur can be pathognomonic for tricuspid valve endocarditis. Pulmonary signs and symptoms predominate.

Case Report

A 32-year-old woman came to the emergency department of a major hospital complaining of a cough productive of yellow sputum and fever, chills, backache, and increasing dyspnea for about 1 week. She stated that her appetite had been poor and that she had lost 20 pounds over the previous 4 weeks. She gave a history of intravenous cocaine use.

On admission her temperature was 39.4°C (103°F), her respiratory rate was 22/min, and her blood pressure was 98/60 mmHg. She had a grade 2/6 systolic murmur without radiation heard best along the left sternal border. She had no knowledge of a previous murmur. Pulmonary examination found a few scattered wheezes in the left lower base with moderately decreased breath sounds over the right lower lung field. Percussion was bilaterally equal, and vocal fremitus was normal. Needle tracts were numerous on both arms. Roth spots, Osler nodes, splinter hemorrhages, and Janeway lesions were not present. The remainder of her physical examination was normal.

Admission laboratory work showed her white cell count was $16 \times 10^6/L$ (16,000/mm³); hemoglobin, 57 g/L (5.7 g/dL); and hematocrit, 0.18. A urine analysis was normal. A chest radiograph showed diffuse bilateral parenchymal densities suggestive of cavitating pneumonia.

A tentative diagnosis of tricuspid valve endocarditis with septic emboli was made, and after obtaining blood cultures, intravenous nafcillin and gentamicin therapy was started. Two units of packed cells were transfused. An echocardiogram was obtained, which showed vegetations on the tricuspid valve.

During the patient's hospitalization she was given an additional 2 units of packed cells. The blood cultures grew *S. aureus*, which was sensitive to nafcillin. The gentamicin was discontinued. After 5 days of intravenous antibiotics, she continued to have temperatures up to 40.6°C (105°F). Because of her slow response to treatment and the development of an elevated aspartate aminotransferase (AST) (1.03 $\mu\text{kat/L}$, [62 U/L]), an infectious disease specialist was consulted. As it was possible that nafcillin was causing the allergic hepatitis with fever, the drug was discontinued; she was given intravenous vancomycin, and her fever resolved by the end of her first week of hospitalization. The patient received intravenous antibiotics for a total of 6 weeks. Signs and symptoms of failure of the right side of the heart did not develop. A follow-up chest radiograph showed clearing of the pulmonary infiltrates, and a follow-up echocardiogram showed resolving tricuspid valve vegetations. Blood cultures were negative. At the time of discharge her white cell count was $7.5 \times 10^6/L$ (7500/mm³), hemoglobin and hematocrit were 114 g/L (11.4 g/dL) and 0.34, respectively, and her AST was within normal limits. After 44 days in the hospital, she was referred to a local chemical dependency center for treatment of cocaine dependence.

Pathogenesis

Infective endocarditis involving right-sided valvular structures is a disease that mainly occurs in intravenous drug abusers and almost always involves otherwise anatomically normal valves. It has been hypothesized that damage by persistent bombardment of the endothelial surface with particulate matter from injected material can damage the tricuspid valve, thus predisposing the person to the development of endocarditis. The exact

Submitted, revised, 29 July 1991.

From the Department of Family Medicine, University of Mississippi Medical Center, Jackson. Address reprint requests to H. Thomas Milhorn, Jr., M.D., Ph.D., Department of Family Medicine, University of Mississippi Medical Center, 2500 N. State Street, Jackson, MS 39216.

Table 1. Comparison of Right-Sided and Left-Sided Endocarditis.*

Right Side	Left Side
Occurs mainly in intravenous drug abusers	Is more commonly associated with congenital heart disease
<i>Staphylococcus aureus</i> is the most common organism	<i>Streptococcus viridans</i> is the most common organism
Polymicrobial involvement occurs frequently	Polymicrobial involvement is less likely
Represents 5% to 10% of all infective endocarditis	Represents 90% to 95% of all infective endocarditis
Pulmonary symptoms are the usual presenting complaints	Systemic distal embolization is common
Congestive heart failure is unusual	Congestive heart failure is common
The condition is well tolerated hemodynamically	Is poorly tolerated hemodynamically
Medical therapy is highly successful	Medical therapy has a poorer success rate
Prognosis is relatively good	Prognosis is poor
Surgery usually does not require immediate valve replacement	Surgery usually requires immediate valve replacement

*Based on Chan P, Ogilby JD, Segal B. Tricuspid valve endocarditis. *Am Heart J* 1989; 117:1140-6.

pathogenesis of infective endocarditis in intravenous drug abusers, however, is still unclear.^{1,2}

Other predisposing factors for the development of infective endocarditis include alcoholism, dermal infections, extensive burns, generalized sepsis, immunodeficiency states such as lymphoma, congenital cardiac lesions associated with left-to-right shunts (ventricular septal defect, patent ductus arteriosus), and catheterization. Rheumatic tricuspid valve disease is rarely complicated by infective endocarditis.^{1,2}

Infecting pathogens in tricuspid valve endocarditis include *S. aureus* (50 to 80 percent), *Pseudomonas aeruginosa* (10 to 40 percent), α -hemolytic streptococcus (10 to 20 percent), *Candida* (3 to 5 percent), and others (10 to 20 percent). The infection is polymicrobial in 10 to 20 percent of cases.¹

Tricuspid valve involvement occurs in only 5 to 10 percent of patients with infective endocarditis. The lower rate of right-sided endocarditis is due to (1) the lower rate of congenital and rheumatic heart disease affecting the right-sided valves; (2) the lower right-sided heart pressures, which produce less stress on the right-sided valves; and (3) possibly the reduced right-sided heart oxygen content.

Tricuspid valve endocarditis differs in a number of ways from left-sided infectious endocarditis, as shown in Table 1.^{1,2}

Clinical Picture

In tricuspid valve endocarditis, pulmonary symptoms (cough, sputum production, hemoptysis, pleuritic chest pain, dyspnea) rather than those of congestive heart failure are the usual presenting complaints. Pneumonia or septic pulmonary emboli resulting from dislodgement of vegetative material are common. Chest radiographs usually

show multiple segmental infiltrates with a predilection for the lower lobes. Cavitation of the infiltrative lung lesions, pleural effusions, and empyema are common when *S. aureus* is the pathogen.²

Systemic embolization rarely occurs with tricuspid valve endocarditis. When it does, usually it is due to a patent foramen ovale or septic thrombi formed in the pulmonary veins.²

Cardiac manifestations in tricuspid valve endocarditis are less prominent than in left-sided endocarditis. Tricuspid regurgitation, however, is present in most patients, although it is often missed. Deep inspiration augments the murmur. Cardiomegaly and right ventricular failure, when present, are associated with a poor prognosis. Rupture of tricuspid valve chordae tendineae can occur but produces no clinical evidence. Death usually results from pulmonary regurgitation and respiratory distress syndrome. Uncontrolled sepsis, severe right ventricular failure, and involvement of the left-sided valves are less common causes of death.²

Diagnosis

The presence of fever, pulmonary signs and symptoms, and a systolic murmur heard best along the left sternal border in an intravenous drug abuser should arouse one's suspicion of the possibility of tricuspid valve endocarditis. The murmur of tricuspid regurgitation, however, can be absent on admission. The triad of intravenous drug abuse, *S. aureus* septicemia, and pulmonary embolism with or without a tricuspid regurgitation murmur is said to be pathognomonic for tricuspid valve endocarditis.³

Two-dimensional echocardiography plays an important role in identifying vegetations, which appear as irregular echo masses attached to the tricuspid valve. It also detects right ventricular

dilation, paradoxical septal motion, and dilated inferior vena cava, which can be associated with tricuspid regurgitation. Because of its inability to display intracardiac spatial relation, as well as imaging all three leaflets of the tricuspid valve, two-dimensional echocardiography is superior to M-mode echocardiography, detecting vegetations in more than 80 percent of patients with tricuspid valve endocarditis.^{2,4-6}

Tricuspid valve vegetations must be differentiated from a right atrial myxoma and a myxomatous, fibrotic, or calcified tricuspid valve, which have similar echocardiographic appearances.²

Treatment

Medical

Medical therapy results in a favorable outcome in most patients with tricuspid valve endocarditis. While awaiting the results of blood cultures, patients should receive antibiotic therapy that gives good coverage for *S. aureus*, α -hemolytic streptococcus, and *P. aeruginosa*. Six to eight weeks of parenteral therapy might be required. The vegetations tend to resolve with bacteriologic cure. Medical therapy is less effective for *P. aeruginosa* and fungal infections than for gram-positive organisms.²

Surgical

The greatest management problem in patients with tricuspid valve endocarditis is to decide whether surgical intervention is indicated and, if so, when. The only two definite indications for surgery are persistent sepsis and congestive heart failure. Surgery may be required for involvement of left-sided heart valves or for infection with gram-negative organisms or *Candida*. Persistent fever without evidence of sepsis is not an indication for surgery. It warrants an extensive work-up to rule out other causes of fever.^{7,8}

When surgical intervention is indicated, three options exist: (1) valve-sparing débridement, (2) valvectomy without prosthetic replacement, and (3) valvectomy with prosthetic replace-

ment. Valve-sparing débridement is done only occasionally and is limited to those cases with a discrete vegetation and little or no valve damage.^{7,8}

Because most patients with infective endocarditis involving the tricuspid valve are intravenous drug abusers who may continue to use drugs after treatment, it is reasonable to perform tricuspid valve excision without prosthetic replacement to prevent the introduction of a "foreign body" that could serve as a nidus for future infection. About 20 to 30 percent of these patients, however, will require subsequent tricuspid valve replacement because of intractable right-sided heart failure.^{7,8}

References

1. Chan P, Ogilby JD, Segal B. Tricuspid valve endocarditis. *Am Heart J* 1989; 117:1140-6.
2. Panidis IP, Kotlen MN, Mintz GS, Segal BL, Ross JR. Right heart endocarditis. Clinical and echocardiographic features. *Am Heart J* 1984; 107:759-64.
3. Julander I, Arneborn P, Back E, Hoglund C, Svanbom M. Intravenous drug addiction—staphylococcal septicemia—pulmonary embolism: a triad pathognomonic for tricuspid valve endocarditis. *Scand J Infect Dis* 1983; 15:257-65.
4. Ginzton LE, Siegel RJ, Criley JM. Natural history of tricuspid valve endocarditis: a two dimensional echocardiographic study. *Am J Cardiol* 1982; 49:1853-9.
5. Bayer AS, Blomquist IK, Bello E, Chiu C, Ward JJ, Ginzton LE. Tricuspid valve endocarditis due to *Staphylococcus aureus*: correlation of two-dimensional echocardiography with clinical outcome. *Chest* 1988; 93:247-52.
6. Manolis AS, Melita H. Echocardiographic and clinical correlates in drug addicts with infective endocarditis: implication of vegetation size. *Arch Intern Med* 1988; 148:2461-5.
7. Yee ES, Khonsari S. Right-sided infective endocarditis: valvuloplasty, valvectomy or replacement. *J Cardiovasc Surg* 1989; 30:744-8.
8. Barbour DJ, Roberts WC. Valve excision only versus valve excision plus replacement for active infective endocarditis involving the tricuspid valve. *Am J Cardiol* 1986; 57:475-8.