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 × 10^6/L (16,000/mm^3); 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 μkat/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 × 10^6/L (7500/mm^3), 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
material are common. Chest radiographs usually
show multiple segmental infiltrates with a predi­
lection for the lower lobes. Cavitation of the infil­
trative lung lesions, pleural effusions, and empy­
ema are common when S. aureus is the pathogen.2

Systemic embolization rarely occurs with tri­
cuspid 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 endo­
carditis are less prominent than in left-sided en­
carditis. Tricuspid regurgitation, however, is
present in most patients, although it is often missed.
Deep inspiration augments the murmur.
Rupture of tricuspid valve chordae tendineae can
occur but produces no clinical evidence. Death
usually results from pulmonary regurgitation and respiratory distress syndrome. Uncontrolled sep­
sis, severe right ventricular failure, and involve­
ment 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 regurgita­
tion 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

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

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


Pathogenesis of infective endocarditis in intra­
venous drug abusers, however, is still unclear.1,2

Other predisposing factors for the develop­
ment of infective endocarditis include alcoholism,
dermal infections, extensive burns, generalized
sepsis, immunodeficiency states such as lym­
phoma, congenital cardiac lesions associated with
left-to-right shunts (ventricular septal defect,
palent ductus arteriosus), and catheterization.
Rheumatic tricuspid valve disease is rarely com­
plicated by infective endocarditis.1,2

Infected pathogens in tricuspid valve endo­
carditis include S. aureus (50 to 80 percent),
Pseudomonas aeruginosa (10 to 40 percent),
alpha-hemolytic streptococcus (10 to 20 percent),
Candida (3 to 5 percent), and others (10 to 20 per­
cent). 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 symp­
toms (cough, sputum production, hemoptysis,
pleuritic chest pain, dyspnea) rather than those of
congestive heart failure are the usual presenting
complaints. Pneumonia or septic pulmonary em­
boli resulting from dislodgement of vegetative
material are common. Chest radiographs usually

82 JABFP Jan.–Feb. 1992 Vol. 5 No. 1
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-echocardiography, detecting vegetations in more than 80 percent of patients with tricuspid valve endocarditis. 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-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) valvulectomy without prosthetic replacement, and (3) valvulectomy with prosthetic replacement. 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