Tricuspid Endocarditis With Pulmonary Emboli

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Introduction

Right-sided endocarditis accounts for 5–10% of all cases of infective endocarditis.[1] Its epidemiology, diagnosis, and treatment differ from that of left-sided endocarditis, making it a distinct entity. This article presents a case of tricuspid valve endocarditis in an intravenous drug user, and discusses the presentation, diagnosis, and therapeutic considerations unique to right-sided endocarditis.

Case Report

A 35-year-old man presented to the urgent care clinic with a painful, draining wound on his left shin, which had started 7 days prior.

His medical history was notable for intravenous drug abuse, chronic hepatitis C, and a history of recurrent tricuspid valve endocarditis with methicillin-resistant *Staphylococcus aureus*. Five years prior he received a porcine tricuspid valve replacement secondary to a large vegetation on his tricuspid valve and poor response to conservative medical management. His postoperative course was complicated by a run of torsades, which developed into third-degree atrioventricular block, necessitating placement of a dual-chamber pacing, dual-chamber sensing, and dual-action (DDD) pacemaker. From that time until this admission, he had recurrent abscesses that required incision and drainage, but no history of recurrent endocarditis.

His physical examination on admission revealed a temperature of 37.6°C, pulse 108 beats/min, blood pressure 99/59 mm Hg, and oxygen saturation 84% on room air. He was alert and oriented to person, place, and time, yet tearful. He had a grade 3/6 systolic murmur, heard at both the right and left sternal border, and his lungs had crackles bilaterally throughout. He had a large ulcer on his left shin and multiple smaller ulcers on both lower extremities, all with surrounding erythema.

His laboratory values included elevated creatinine (2.9 mg/dL). Urinalysis showed an elevated specific gravity of 1.025 and hyaline casts. His white-blood-cell count was 16,600 cells/uL, and his platelet count was 40,000/uL. All of those values are consistent with septic physiology. The chest radiograph showed diffuse patchy bilateral infiltrates, consistent with septic emboli (Fig. 1). His electrocardiogram showed a paced rhythm. Lower-extremity radiographs did not show osteomyelitis. With the input of the cardiology and infectious disease services, he was started on vancomycin, gentamicin, and rifampin for suspected recurrent endocarditis. His blood cultures grew methicillin-resistant *S. aureus*, and a transthoracic echocardiogram confirmed a 1.5 cm × 1.0 cm vegetation on his tricuspid valve, with tricuspid regurgitation (Fig. 2).

On the next day the patient required increasing amounts of oxygen and was transferred to the medical intensive care unit. A chest computed tomogram showed bilateral pulmonary emboli and peripheral abscesses (Fig. 3). He developed signs of right-sided heart failure, including jugular venous distention and peripheral edema. Given these new signs of heart failure, he was evaluated by the cardiothoracic surgical team for a second valve replacement. They recommended lower-extremity debridement prior to valve replacement, to reduce the risk of persistent bacteremia. Shortly after the debridement, the patient developed profound sepsis, at which time the family decided to initiate comfort care measures. The patient died 4 days after admission.

Discussion

Epidemiology

Right-sided infective endocarditis occurs predominantly in intravenous drug users. Also at risk are patients with pacemakers, central venous catheters, and congenital heart diseases.[1] The tricuspid valve is af-

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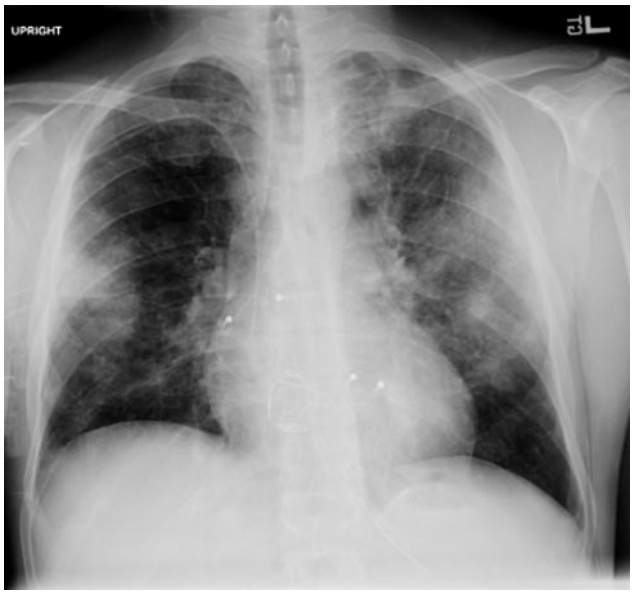

Fig. 1. Initial anteroposterior chest radiograph showing bilateral patchy opacities in the middle and upper lobes.

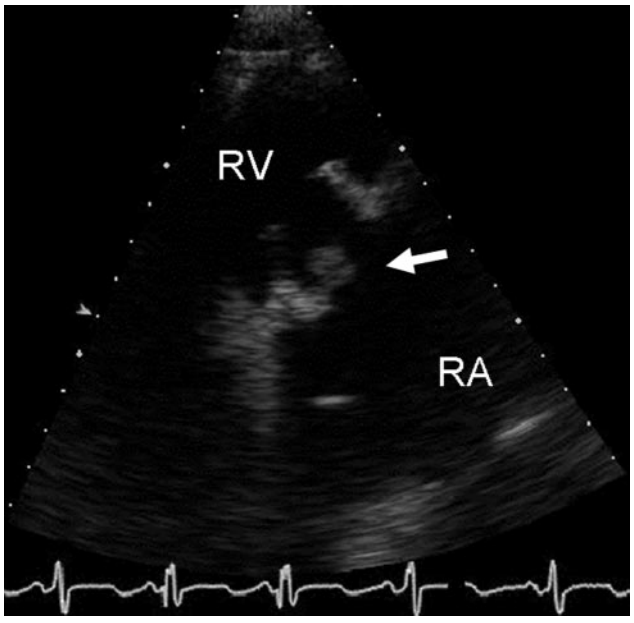

Fig. 2. Transthoracic echocardiogram showing a bioprosthetic tricuspid valve with a 1.0 cm × 1.5 cm vegetation (arrow). RA = right atrium. RV = right ventricle.

fected more frequently, and among intravenous drug users, methicillin-sensitive *S. aureus* accounts for the majority of cases. In the United States, the incidence among intravenous drug users is 0.15–2% per year, and is currently decreasing, possibly reflecting an increased awareness of the risk of transmitting human immunodeficiency virus,[2] which is associated with an increased risk of infective endocarditis in intravenous drug users.[2]

Fig. 3. Chest computed tomogram showing diffuse hazy ground-glass opacification and multiple bilateral cavitary lesions.

In contrast to the mortality of left-sided infective endocarditis (20–30%), the prognosis of right-sided staphylococcal infective endocarditis is more favorable, with a mortality of less than 10%. Gram-negative bacterial and fungal causes of endocarditis portend a poorer prognosis than cases secondary to staphylococcal causes.[2] A concurrent diagnosis of acquired immune deficiency syndrome, in which the cluster-of-differentiation (CD4) cell count is less than 200 cells/μL, predicts a higher mortality rate.[3]

Presentation

The most common presentation in right-sided infective endocarditis is persistent fever, bacteremia, and pulmonary emboli. Fifty-five percent of chest radiographs at presentation show evidence of pulmonary infiltrates compatible with emboli.[4]

Many presenting symptoms, such as chest pain, dyspnea, cough, and hemoptysis, result from septic emboli to the pulmonary vasculature. Complications such as pulmonary infarction, pulmonary abscess, pneumothorax, pleural effusion, and empyema can result from emboli. Paravalvular abscess occurs infrequently, as reported by Moss and Munt.[4] Multiple pulmonary emboli and/or destruction of the tricuspid valve may result in cardiac complications such as right-sided chamber dilation, right heart failure, and worsening of tricuspid regurgitation. Chronic right-atrial dilation may lead to supraventricular arrhythmias, including atrial fibrillation and flutter.[4]

Diagnosis

In 1986, Robbins et al[5] presented major and minor diagnostic criteria for active right-sided endocarditis. The major criteria were fever and an echocardiogram showing vegetation. The minor criteria were 2 tempo-

Table 1. Modified Duke Criteria for the Diagnosis of Infective Endocarditis

Major Criteria	
Blood cultures	Typical microorganisms (Viridans streptococci, *Streptococcus bovis*, HACEK group, *Staphylococcus aureus*, enterococci) in 2 separate cultures, in the absence of a primary focus
	Persistently positive blood cultures drawn more than 12 hours apart, or majority of ≥4 separate blood cultures, with the first and last drawn more than 1 hours apart
	Single positive blood culture for *Coxiella burnetii* or antiphase 1 immunoglobin G antibody titer >1:800
Endocardial involvement	Positive echocardiongram (transesophageal echocardiogram recommended in patients with prosthetic valves) and one or more of:
	Oscillating intracardiac mass on a valve or support structure, or in the path of regurgitant jets, or on implanted material, in the absence of an alternative explanation
	Abscess
	New partial dehiscence of prosthetic valve
Minor Criteria	
Predisposing heart condition or intravenous drug user	
Fever > 38.0ºC	
Vascular phenomena: arterial emboli, septic pulmonary infarct, intracranial hemorrhage, conjunctival hemorrhage, Janeway lesions	
Immunologic phenomena: Osler's nodes, Roth spots, glomerulonephritis, rheumatoid factor	
Microbiologic evidence: positive blood culture with plausible organism but not meeting a major criterion	
To Establish Diagnosis	
Definite infective endocarditis	Pathology: vegetations or intracardiac abscess, confirmed by histology
	2 major criteria
	1 major and 3 minor criteria
	5 minor criteria
Possible infective endocarditis	1 major and 1 minor criteria
	3 minor criteria
Rejected infective endocarditis	Alternative diagnosis
	Resolution of manifestations of infective endocarditis with ≤4 days of antibiotic therapy
	No pathology evidence of infective endocarditis at surgery or autopsy after ≤4 days of antibiotic therapy
	Does not meet above criteria for possible infective endocarditis

HACEK group = *Haemophilus* species, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species.
(Data from Reference 6.)

rally spaced positive blood cultures, evidence of septic pulmonary emboli, absence of systemic emboli, and a murmur consistent with tricuspid or pulmonic valve regurgitation. In this diagnosis system, 2 major criteria or 1 major plus 3 minor criteria were required for diagnosis.[5] Subsequently, the Duke criteria, first published in 1994 and modified in 2000, became the standard for diagnosing all cases of infective endocarditis, both right and left-sided (Table 1).[6] The sensitivity and specificity of the Duke criteria for right-sided infective endocarditis have not been thoroughly studied. One limitation of the Duke criteria is that a right-sided murmur may be more difficult to detect on auscultation. Right-sided murmurs can, however, be augmented with deep inspiration.[5]

As with left-sided infective endocarditis, transthoracic echocardiogram with combined M-mode and 2-dimensional imaging is an appropriate first-line study; it can detect vegetations in roughly 80–100% of patients with tricuspid valve infective endocarditis.[7] A chest radiograph may reveal infiltrates consistent with emboli and is therefore a critical component in the work-up of infective endocarditis. The presence of peripheral embolic manifestations or neurologic symptoms should prompt a work-up for paradoxical embolus through a patent foramen ovale or for concurrent left-sided infective endocarditis. Transesophageal echocardiogram is generally recommended for patients with prosthetic valves, suspicion or existence of a paravalvular abscess,

and for a definitive study in patients with a negative transthoracic echocardiogram in whom the clinical suspicion of infective endocarditis is still high.[7]

Treatment Options

Uncomplicated methicillin-sensitive *S. aureus* right-sided endocarditis can be treated with an intravenous course of nafcillin plus an aminoglycoside. The standard duration of treatment is 4–6 weeks. A 2-week course is acceptable if (1) the infection is confined to the tricuspid valve, (2) the vegetation is <2 cm, and (3) there are no complications, such as evidence of right-sided heart failure or systemic emboli.[3] In our institution we prescribe a full 4–6-week course for all patients with human immunodeficiency virus whose CD4 count is less than 200 cells/μL, and for patients who do not respond to antibiotics, based on either clinical findings or on a failure to clear blood cultures within 2–4 days of initiating antibiotics. The addition of gentamicin for 3–5 days for native valve endocarditis results in earlier defervescence, reduces leukocytosis, and sterilizes blood cultures, but there is no proven mortality benefit. For methicillin-resistant *S. aureus* infections, a 4-week course of vancomycin with 1–2 weeks of an aminoglycoside is generally recommended.[3]

There is some evidence to suggest a role for oral therapy in adult intravenous drug users with native right-sided valve endocarditis and no evidence of complicating factors such as respiratory compromise or hypotension. In one study, a 28-day course of oral treatment with ciprofloxacin and rifampin was found to be an acceptable alternative to intravenous therapy.[8] In this population, rifampin may decrease methadone's effectiveness and precipitate symptoms of opiate withdrawal.[3] At this time, there is insufficient evidence to support the use of linezolid as the sole treatment for staphylococcal endocarditis. It is used in clinical practice as salvage therapy.

Most patients can be treated with antibiotics alone, making surgical intervention infrequent. Indications for surgery are not as well defined for right-sided as for left-sided infective endocarditis. Persistent sepsis and intractable right heart failure despite appropriate medical management are relative indications for surgical intervention.[4] Paravalvular abscess, large vegetation size, or endocarditis from a fungal or Gram-negative source may prompt surgical consultation.[4,7]

In this patient population, surgery is approached with caution, because a large proportion of patients resume intravenous drug use following their hospital discharge. Some experts recommend avoiding mechanical valves in the setting of persistent intravenous drug use, because of the likelihood of noncompliance with anticoagulation therapy.[3] Additionally, prosthetic material is also typically avoided, given the high the risk of reinfection in this population.[3] Some authors advocate a cryopreserved mitral homograft valve rather than mechanical valve.[4]

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