Utility of Transesophageal Echocardiography in the Conservative Management of Prosthetic Valve Endocarditis*

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Prosthetic valve endocarditis is a formidable complication following cardiac valve replacement. Surgical intervention has resulted in a significant reduction in mortality when certain complications prevail. We report two such cases of prosthetic valve endocarditis in which the use of transesophageal echocardiography permitted close surveillance during medical therapy and thus avoided the need for surgical intervention. Therefore, with the improved ability to monitor disease progression with transesophageal echocardiography, nonsurgical management of prosthetic valve endocarditis remains an option. (Chest 1992; 102:1886-88)

Prosthetic valve endocarditis (PVE) is a well-recognized and formidable complication occurring in 2 to 4 percent of patients following cardiac valve replacement.¹ Surgical intervention appears to have resulted in a significant reduction in mortality in mechanical PVE and whereas some investigators suggest urgent surgery for this potentially life-threatening illness,² others suggest surgical intervention only in certain circumstances.

Absolute indications for urgent surgery are as follows: (1) severe heart failure; (2) persistent sepsis despite antibiotic therapy; (3) fungal etiology; (4) valve obstruction; (5) new heart block (including 1st degree AVB); and (6) unstable prosthesis. Relative indications for early surgery include the following: (1) nonstreptococcal organism; (2) emboli; (3) early-onset PVE; (4) vegetations by echocardiography; and (5) paravalvular leak.³

Due to concomitant medical problems, we sought to treat two patients with mechanical PVE with medical therapy alone, but using transesophageal echocardiography (TEE) to closely monitor their progress, and report on their successful outcome. TEE was performed in the standard fashion, the manner of which is well described.⁴ The role of this relatively new imaging modality is expanding and the current indications are listed elsewhere.⁵⁶

**Case Reports**

**Case 1**

A 33-year-old male intravenous drug abuser, with a St. Jude mitral valve replacement, was admitted to the hospital with headache, lethargy, abdominal pain, and fever. Examination revealed a fever of 39.5°C, heart rate of 100/min, blood pressure of 110/70 mm Hg, a crisp valve click, a 1/6 systolic ejection murmur, hepatosplenomegaly, and embolic lesions on the right great toe. The patient was HIV positive with a WBC count of 21.7 × 10⁹/cu mm. Chest roentgenogram was normal and the electrocardiogram (ECG) revealed sinus rhythm, intra-atrial conduction delay, and left ventricular hypertrophy. Blood cultures grew methicillin-sensitive Staphylococcus aureus. No abnormalities were seen on transthoracic echocardiography; however, TEE revealed multiple mobile vegetations on the left atrial surface of the mitral prosthesis (Fig 1). Vancomycin and gentamycin therapy was initiated and changed to nafcillin and rifampin on final organism identification. The fever settled by day 4, followed by low-grade fever for two weeks. The

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**FIGURE 1.** Serial transesophageal echocardiograms of the left atrium at the level of the mitral valve, showing the following: Top: admission: multiple vegetations (hollow arrows) on the atrial side of a normally seated St. Jude mitral prosthetic valve. The sewing ring of the prosthesis is shown by the solid arrows. Center: 6 weeks: reduction in size of the vegetations. Bottom: 1 year: further reduction in size of vegetations.
leukocyte count returned to normal by the 11th day. Blood cultures were negative on day 7. Intravenous antibiotic therapy was continued for a further six weeks followed by oral dicloxacillin for three months. TEE at six weeks (Fig 1) revealed reduction in size of the vegetations. At one year he remained asymptomatic, and TEE demonstrated further reduction in size of the vegetations (Fig 1).

CASE 2

A 78-year-old man with a Bjork-Shiley mitral valve, prior coronary artery bypass graft (CABG) and recurrent rectal abscesses was admitted to the hospital with chills, rigors, and fever. Examination revealed a fever of 39.2°C, heart rate of 72/min, blood pressure of 130/70 mm Hg, and a 3/6 holosystolic murmur along the left sternal border. WBC count was 9.1 × 10⁹/μL, ECG showed sinus rhythm, first-degree AV block and left bundle branch block, and chest roentgenogram revealed a right lower lobe infiltrate. Blood cultures grew Enterococcus. Transthoracic echocardiogram revealed mild left ventricular dysfunction, a normal prosthetic valve, and a tricuspid valve vegetation. TEE showed a moderate-sized vegetation on the left atrial side of the prosthetic valve, paravalvular regurgitation (Fig 2), and confirmed the tricuspid valve vegetation. Vancomycin and gentamicin therapy was initiated and changed to penicillin and gentamicin on final organism identification. The fever resolved rapidly and blood cultures were negative by the fifth day. Intravenous antibiotic therapy was continued for a further six weeks. TEE at six weeks revealed no vegetations on either the tricuspid or the prosthetic valve, and 2+ paravalvular regurgitation (Fig 2). He did well for 17 months, except for hemolysis, associated with worsening of the paravalvular regurgitation by transthoracic echocardiogram. With increasing transfusion requirements for anemia, surgical revision of the valve in this high-risk patient was unavoidable. A Carpentier-Edwards bioprosthesis and two-vessel bypass surgery were performed. The source of the paravalvular leak was located to the superior aspect of the valve, which was detached from the sewing ring of the prosthesis. Significant bleeding was encountered related to underlying thrombocytopenia and coagulopathy. Multiple complications ensued and the patient died on the second postoperative day.

DISCUSSION

Despite the belief by some that patients with mechanical PVE should undergo operation as soon as the diagnosis has been established, we have documented the successful medical management of this condition. The enhanced ability to evaluate and monitor mechanical prostheses and adjacent endocardial structures during and following active infection provides greater latitude in determining the need for, and timing of, surgical intervention.

Echocardiography is the noninvasive imaging diagnostic procedure of choice in patients with suspected endocarditic lesions. Transesophageal echocardiography has increased the accuracy of diagnosing valvular vegetations to >90 percent compared with 78 percent attainable with the transthoracic route. Its value is even greater in the
Tension Pneumopericardium in an Infant*

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Pneumopericardium in newborns is most often a complication of mechanical ventilation and frequently results in fatal cardiac tamponade. We report the case of a mechanically ventilated 33-day-old full-term gestation infant with interstitial pneumonitis who developed tension pneumopericardium. Treatment includes lowering peak inspiratory pressure and decompressing the pericardial space with tube drainage following pericardiocentesis.

(Chest 1992; 102:1888-91)

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Tension pneumopericardium is a rare, but serious complication of mechanical ventilation in infants. Early diagnosis and treatment may be life-saving. We report an infant with interstitial pneumonitis who developed tension pneumopericardium associated with barotrauma.

CASE REPORT

A 33-day-old, 4.0-kg white male infant, product of a full-term gestation, presented to the Emergency Department with a one-week history of both vomiting and seven to eight watery stools per day. In addition, he had exhibited a three-day history of fever, cough productive of clear sputum, and clear rhinorrhea. His medical history was benign with a term delivery at 2,996 g. On physical examination, he was tachypneic (respiratory rate of 102 breaths/min) with grunting. His heart rate was 164 beats/min with a blood pressure of 87/48 mm Hg. His rectal temperature at the time of hospital admission was 38.3°C. His chest examination revealed poor air movement with bilaterally coarse breath sounds and mild intercostal retractions. Results of his examination were otherwise unremarkable. His arterial blood gas on room air was pH 7.24, Pco2 of 30 mm Hg, Po2 of 64 mm Hg, bicarbonate of 12.6 mEq/L, base excess of −13.4 mEq/L. His admission chest roentgenogram revealed mild diffuse pneumonitis and pulmonary hyperinflation consistent with a viral etiology. His white blood cell count was elevated to 20,800 cells per cubic millimeter with 24 percent bands, 20 percent polymorphonuclear leukocytes, and 50 percent lymphocytes. Serum electrolytes were as follows: sodium, 141 mEq/L; potassium, 5.1 mEq/L; chloride 107 mEq/L; CO2, 14 mEq/L. Serum urea nitrogen and serum creatinine were 47 mg/dl and 0.7 mg/dl, respectively.

He was admitted to the Pediatric Intensive Care Unit and treatment was initiated with ampicillin and cefotaxime. Nafcinil was added later subsequent to deterioration. Despite early and aggressive fluid resuscitation (15 ml/kg of normal saline solution, 30 ml/kg of plasma protein fraction [Plasmanate], and maintenance fluid at twice the normal rate), he developed oliguria and hypotension for which dopamine infusion was instituted at 5 μg/kg/min. Both his respiratory status and his hemodynamic status continued

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