pericarditis cured, all treated with amphotericin B, with or without fluocytosine, combined with pericardietomy. Two other patients treated with amphotericin B but without surgery died, and they were considered microbiologic failures. The remaining patients were untreated.

Pericardial drainage is considered essential for successful therapy of bacterial pericarditis. One or more pericardio-centeses may suffice, but open surgical drainage, either by a pericardial window or pericardietomy, is often needed.6,7 The literature regarding Candida pericarditis is too sparse for definitive recommendations with respect to need and technique of pericardial drainage. The single patient whose case is reported herein demonstrates that a single pericardiocentesis, which did not completely evacuate the pericardial space and was associated with technical difficulties possibly due to loculations, in combination with anti-Candida chemotherapy can result in cure, and that thoracotomy is not necessary in all cases.

The patient's immunocompetence and overall good health combined with adequate anti-Candida chemotherapy likely contributed to the salutary outcome. Apparently purulent pericarditis caused by bacteria such as Staphylococcus aureus usually requires effective drainage whereas that caused by Mycobacterium tuberculosis and possibly Candida frequently does not require drainage.6 Reasons for differing requirements for surgical drainage are not well-defined. Speculatively, pericarditis caused by mycobacteria and fungi is often subacute; the relatively slow clinical course may allow sufficient time for antimicrobials and the immune system to effect cure without courting tamponade or persistent sepsis.

The pharmacokinetics of amphotericin B are poorly understood, especially with respect to pericardial penetration.3 The amphotericin B level in pericardial fluid was 50 percent of serum in a single patient with Candida pericarditis.4 Serum levels in the range of 1.0 to 2.4 mg/L have been measured after commonly used doses of amphotericin B.5 Since the MIC of our isolate to amphotericin B was less than 0.5 mg/L, it is likely that inhibitory concentrations were present in pericardial fluid for at least part of the dosing interval. The use of fluocytosine likely increased the potency of the anti-Candida chemotherapy.

Although problems with antifungal susceptibility testing are recognized, it is of interest and possibly of therapeutic import that the Candida isolate in this report was highly resistant to several azoles, including ketoconazole and fluconazole, and suggests need for caution in use of azoles for treatment of deep-seated Candida infections.

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Nonbacterial Thrombotic Endocarditis*

Assessment by Transesophageal Echocardiography

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Nonbacterial thrombotic endocarditis (NBTE) is difficult to detect antemortem and is often not suspected until embolic events occur. Transthoracic echocardiography is useful in diagnosing NBTE, but it may be limited by suboptimal imaging and resolution. Herein we describe the first reported case of NBTE diagnosed by transesophageal echocardiography. As early detection and treatment may avert significant embolic complications, transesophageal echocardiography should be strongly considered if other techniques are nondiagnostic and clinical suspicion of NBTE remains high.

(Chest 1992; 102:954-56)

| DIC = disseminated intravascular coagulation; NBTE = nonbacterial thrombotic endocarditis |

Nonbacterial thrombotic endocarditis (NBTE) is difficult to detect antemortem, and it is usually discovered at autopsy. Because new cardiac murmurs are an infrequent finding,1 NBTE may not be suspected until embolic events occur. Unfortunately, arterial embolization is common in NBTE and may cause significant morbidity. As early diagnosis and treatment may prevent or reduce embolic complications,2 accurate antemortem detection of thrombotic valvular vegetations is necessary. Herein, we describe an unusual example of NBTE, the first reported case (to our knowledge) diagnosed by transesophageal echocardiography.

CASE REPORT

A 64-year-old woman initially presented with right ophthalmoplegia and right cranial nerve palsies. Computed tomographic (CT) scans and cerebral angiography revealed bilateral internal carotid artery aneurysms at the junctions of the cavernous and precommunicating segments. She was referred for neurosurgical clipping and repair. Her physical examination revealed right cranial nerve III, IV, V, and

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Nonbacterial Thrombotic Endocarditis (Blanchard, Ross, Dittrich)
VI palpsies and was otherwise normal. Laboratory studies were notable for normal coagulation test results, white blood cell count, bleeding time, and electrocardiogram. As the planned procedure involved cardiopulmonary bypass, a transthoracic echocardiogram was performed and demonstrated normal cardiac size, function, and valves.

Craniotomy with aneurysm clipping was performed, and involved cardiopulmonary bypass and hypothermia for 7 h and total circulatory arrest for 2 h. Postoperatively, progressive thrombocytopenia and prolongation of the prothrombin time occurred, despite platelet transfusion and vitamin K administration. Fibrinogen levels dropped and fibrin degradation products were present in the patient's serum, consistent with disseminated intravascular coagulation (DIC). Subsequently, adult respiratory distress syndrome developed, and urine output dropped markedly. Empiric broad-spectrum antibiotic therapy was started, despite multiple negative blood cultures, and intravenous pressors were used to maintain adequate blood pressure. One week after surgery, acute right heart ischemia developed. A transthoracic echocardiogram revealed new mitral regurgitation and a possible mitral vegetation. To better assess cardiac and valvular function, biplane transesophageal echocardiography (Hewlett-Packard Sonos 1000, Andover, Mass) was performed. Large mitral vegetations (Fig 1) and severe mitral regurgitation were present. In addition, leaflet thickening and multiple discrete vegetations were noted on the aortic, tricuspid, and pulmonic valves in several different echocardiographic views. Mild tricuspid insufficiency was present, and ventricular function was otherwise normal. A repeated head CT scan showed new areas of low attenuation consistent with ischemic injury as well as a midline shift toward the right. Intravenous heparin therapy was begun, but the patient remained neurologically unresponsive and died ten days after surgery.

At autopsy, fibrinous, thrombotic vegetations were noted on all cardiac valves and were largest on the mitral valve (Fig 2). The vegetations were loosely adherent, and the underlying valvular structures showed no evidence of destruction or erosion. No bacteria were seen on microscopy and all cultures were negative. Thrombotic material and emboli were present in the renal and cerebral vasculature.

**DISCUSSION**

Originally described more than a century ago, NBTE has long been associated with malignant neoplasms (especially adenocarcinoma) and hypercoagulable states, and occurs in 10 to 15 percent of patients with DIC. Definitive antemortem diagnosis has been difficult, as the thrombotic vegetations are generally small and nondestructive. The first reported identification of NBTE by M-mode echocardiography was in 1976, and since then, several cases of diagnosis by two-dimensional echocardiography have been reported.

Earlier in this century, NBTE was thought to be an incidental autopsy finding of limited importance. Over time, however, it has become clear that significant complications are associated with thrombotic valvular vegetations: systemic embolization, particularly to the cerebral vasculature, is common, and may be avoided in some cases by prompt and aggressive anticoagulation. Transthoracic echocardiography is an excellent noninvasive diagnostic technique, but it may be limited by suboptimal image quality and resolution, especially when vegetations are under 2 to 3 mm in size or the patient cannot be properly positioned. Transesophageal echocardiography overcomes these limitations and provides superior images and detail of cardiac chambers and valves; in recent studies involving infective endocarditis, this technique was more effective than transthoracic imaging in detecting bacterial vegetations and abscesses.

In the present case, fulminant DIC occurred in the perioperative period, probably secondary to release of brain tissue factors into the systemic circulation and prolonged cardiopulmonary bypass. Transthoracic echocardiography was adequate before surgery, but because of suboptimal positioning, complete visualization of the cardiac valves could not be performed postoperatively. Excellent images of the myocardium and all valves were obtained by transe-
HIV-Associated Pericardial Effusions*

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Following a case of cardiac tamponade in a patient with the acquired immunodeficiency syndrome (AIDS), we examined the frequency and clinical spectrum of pericardial effusions associated with human immunodeficiency virus infection (HIV) at our institution. Of 187 hospitalized patients documented to have pericardial effusions over a one-year period, 14 (7 percent) were known to be HIV-positive at the time of their echocardiograms. One patient presented with a large effusion and cardiac tamponade, three had moderate effusions, and ten had small effusions. The probable effusion etiology was established in four cases and included endocarditis (2), lymphoma (1), and myocardial infarction (1). In hospital mortality was 29 percent (4 of 14). From our study, as well as a growing number of reports in the literature, we conclude that HIV-associated pericardial effusions are frequently seen and that their clinical spectrum is broad. (Chest 1992; 102:956-58)

Infection with the type 1 human immunodeficiency virus (HIV) has been associated with a variety of cardiac complications, including myocarditis, endocarditis, and dilated cardiomyopathy. In addition to these complications, a number of reports have described pericardial effusions associated with HIV infection.4,14 Following a recent case of cardiac tamponade in an AIDS patient, we examined the frequency and clinical spectrum of HIV-associated pericardial effusions at our institution.

CASE REPORT

A 36-year-old man with AIDS presented with four days of fevers, chills, night sweats, and progressive dyspnea. He had been treated for pulmonary tuberculosis during the previous six months but had discontinued his medications one month before admission. On physical examination, the patient was in moderate distress with a heart rate of 112 bpm, a blood pressure of 94/76 mm Hg, a respiratory rate of 29/min, and a temperature of 38.1°C. Examination was also remarkable for oral thrush, bilateral basilar rales, elevated jugular venous pressure, and distant heart sounds.

Initial laboratory test results were unremarkable. A chest roentgenogram, however, revealed an enlarged cardiac silhouette, bilateral pleural effusions, and apical scarring. Subsequently, an echocardiogram showed a large pericardial effusion associated with right ventricular collapse, right atrial collapse, inferior vena cava plethora with blunted response to respiration, and respiratory variation of mitral inflow velocities.

Because of the patient's hemodynamic instability, a pericardiocentesis was performed. More than 1,000 ml of serosanguineous fluid was drained, and blood pressure rose to 115/65 mm Hg. The next day, a pericardial window was created but subsequent laboratory examination of pericardial tissue and fluid showed only...