The following report concerns a case of nonbacterial thrombotic endocarditis that developed in a patient being treated with a LeVeen peritoneovenous shunt for intractable ascites secondary to Laënnec's cirrhosis. To the best of our knowledge, such an association has not been previously described. (Texas Heart Institute Journal 1987; 14:215-218)

Key words: Endocarditis; peritoneovenous shunt; liver cirrhosis, alcoholic; ascites

Nonbacterial Thrombotic Endocarditis (NBTE) is the commonest form of endocarditis. It can occur at any age, but most cases are seen in the elderly. The literature is replete with various terms that refer to this condition—namely, atypical verrucous endocarditis, thromboendocarditis, marantic or malignant endocarditis, and Libman-Sacks disease.1,2 Morphologically, NBTE is characterized by one or more uninflamed valve leaflets and vegetations that consist of necrotic debris, fibrinoid material, and trapped disintegrating fibroelastic and inflammatory cellular tissue. The vegetations are usually located on the valvular margins, which are most exposed to the bloodstream. The mitral and aortic valves are most frequently involved; tricuspid or pulmonary valve involvement is rare. As in any other type of endocarditis, the major complication is embolism and infarction of various involved organs.1,2

The predisposing factors associated with NBTE include prolonged use of intracardiac central venous lines, pulmonary artery catheterization in adults, and malignancies, debilitation, and other wasting diseases.1,2 The following brief report suggests that the use of a peritoneovenous shunt for chronic intractable ascites secondary to Laënnec's cirrhosis may also be a cause of NBTE.

CASE REPORT

A 67-year-old man with cirrhosis, ascites, and esophageal varices was hospitalized because of generalized weakness and hepatic encephalopathy. The patient had drunk a fifth of vodka and a pack of beer per day for many years but had had no recent alcoholic intake. A year before the present admission, a LeVeen shunt had been inserted to treat refractory ascites and bleeding esophageal varices. These lesions were also treated with sclerotherapy. For one month before admission, the patient had been unable to drink fluids. His medications included Aldactone, lactulose, folate, and...
Large, nodular vegetations are seen on the tricuspid valve (TV) and in the right atrial endocardium (RA).

Smaller vegetations are present on the mitral (M) and aortic (A) valves.

multivitamins. Abnormal physical findings consisted of icterus, ascites, multiple ecchymoses, and slight obtundation. Pertinent laboratory results included leukocytosis, prolonged bleeding times, macrocytic anemia, abnormal liver function tests (bilirubin, 5.2 mg%; direct bilirubin, 2.6 mg%; lactic acid dehydrogenase, 590 international units), and abnormal renal function tests (blood urea nitrogen, 69 mg%; creatinine, 4 mg%). The electrocardiogram showed a primary atrioventricular block, but the chest radiograph was within normal limits.
The patient was treated with intravenous fluids, fresh frozen plasma, packed red blood cells and platelets, and vitamin K. His urinary output remained low, and his blood urea nitrogen level rose to 100 mg%. Repeated cultures of blood, urine, and peritoneal fluid were negative. He remained afebrile, and his white cell blood count returned to normal. However, it was decided that dialysis would be pointless. Terminally, he had hematemesis. His blood pressure dropped, his breathing became shallow, and he died shortly thereafter.

The immediate cause of death was a massive hemorrhage resulting from erosive esophageal varices. The tip of the LeVeen shunt was located just above the tricuspid valve and was observed on autopsy to be enveloped by a large thrombotic vegetation (Fig. 1) that was attached to the underlying endocardium. Smaller vegetations involved the mitral and aortic valvular margins (Fig. 2), the right atrium, and the right ventricle. Postmortem cultures of blood and vegetations from different foci failed to grow microorganisms. Evidence of multiple emboli and infarction involving the spleen, left lung, epididymis, and brain was noted. Other pertinent autopsy findings included advanced Laënnec's cirrhosis, massive serous ascites, and fibrocongestive splenomegaly.

DISCUSSION

The peritoneovenous shunt is a recent device, designed to treat intractable ascites resulting from varied causes. Over the last 20 years five shunts have been evaluated—namely, the Smith (1962), Hyde (1966), LeVeen (1974), Holter (1975), and Denver (1979) models. These systems, which are activated by a pressure gradient between the peritoneal cavity and the venous circulation, serve as a one-way valve and autogenously reinfuse ascitic fluid. The catheter is usually embedded in the subcutaneous tissue of the chest and drains into the superior vena cava. Patient response, as recorded in the literature and as experienced by us, has been dramatic. Clinical results are characterized by the striking remission of ascites, increases in renal blood flow, an improved glomerular filtration rate, and measurable difference in salt and water excretion.3,4

The incidence of complications, however, has multiplied since the shunts were introduced. Fever, disseminated intravascular coagulopathy, infection, and leakage are commonly reported. In the 40 autopsies performed at the Hines Veterans' Administration Hospital in patients who had received shunts for intractable ascites resulting from Laënnec's cirrhosis, complications included peritoneal fibromatosis (19 patients), bowel obstruction (five patients), superior vena caval thrombosis (two patients), inferior vena caval thrombosis (one patient), and tricuspid valve lithiasis (one patient).4,5

Nonbacterial thrombotic endocarditis complicating the use of a peritoneovenous shunt has not previously been described. A literature review, however, reveals a few documented cases of right-sided bacterial or fungal endocarditis that developed after shunt placement. Valla and associates5 described two cases caused by Staphylococcus epidermidis and Corynebacterium xerosis, whereas Reyes and colleagues5 had one case associated with Trichosporon beigelii, and Van Wyck and coworkers7 had another case secondary to Staphylococcus aureus. In a recent letter, Valla8 also reported an additional three cases of LeVeen shunt endocarditis. Downward displacement and protracted contact between the catheter tip of the venous line of the shunt and the endocardium of the atrial wall, tricuspid valve, or ventricle were most likely the major factors in the development of neoendocardium and subsequent endocarditis in these patients. Other conditions, aside from valvular diseases, that may predispose to endocarditis are immunodeficiency and a deficiency of magnesium.5

REFERENCES
