Superior Vena Caval Obstruction Associated With Long-term Peritoneovenous Shunting

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The first described case of superior vena caval obstruction associated with a chronically implanted peritoneovenous shunt occurred in a patient with Budd-Chiari syndrome eight months after placement, and necessitated operative portal decompression and shunt removal.

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PERMANENT PERITONEOVENOUS shunting using a surgically implanted, pressure activated LeVeen valve has become an accepted means of managing patients with medically intractable ascites.[8] Reported complications occurring with frequencies of 5–20% include: wound infection and/or valve sepsis, fluid overload and congestive failure, bleeding diathesis related to intravascular coagulopathy, and shunt occlusion.[18] The previously unreported occurrence of innominate-subclavian and superior vena cava thrombosis after peritoneovenous shunting represents a potentially lethal event, and constitutes the basis for this communication.

Case Report

A 67-year-old white female noted the gradual onset of abdominal swelling and malaise. She had previously taken diethylstilbestrol for 12 months to ameliorate postmenopausal complaints. Laparotomy demonstrated 3.500 ml of clear ascites. Biopsy of a grossly abnormal liver showed congestive changes with "blood pooling" and nonspecific midzonal necrosis consistent with Budd-Chiari syndrome. Neoplastic, infectious, or cirrhotic changes were not evident. Conservative management employing sodium and fluid restriction plus diuretics was unsuccessful. Other causes of hepatic venous occlusion such as polycythemia rubra vera, hypernephroma or mass lesions contiguous with the inferior vena cava were excluded. Angiography demonstrated severe hepatic venous outflow obstruction with patency of both portal and inferior vena caval systems. Placement of a peritoneovenous shunt four months later resulted in prompt resolution of ascites with weight reduction of 14 kg.

Eight months later malaise and abdominal swelling recurred with weight gain of 10 kg. Hematologic studies included a hematocrit of 47%, platelets of 400,000/cm^3, and a prothrombin time of 50%. Liver function testing was normal with the exception of a serum albumin of 1.8 g/dl. Whole blood volume was normal with a markedly decreased plasma volume and a slightly elevated red blood cell volume. Intraperitoneal injection of albumin^{99}Tc yielded scan visualization of the liver, consistent with shunt patency.[3] Hypague injection of the venous shunt tubing demonstrated correct caval positioning of the catheter tip, but complete SVC obstruction with nonvisualization of the right atrium (Fig. 1). Pressure measurements (Fig. 2) demonstrated a right internal jugular vein to right atrial gradient of 9 cm H_2O and a portacaval gradient of 25 cm H_2O. Hepatic vein injection documented pruning and early cutoff of intrahepatic venous radicles (Fig. 3). Both supra and infra hepatic portions of the vena cava, as well as the portal vein, were patent (Fig. 4).

Laparotomy was performed in the hopes of fashioning a side-to-side portacaval shunt but a large caudate lobe precluded partacaval apposition. A side-to-end portarenal shunt was created. The post-shunt portacaval pressure gradient was 8 cm H_2O. Postoperatively, the patient reaccumulated much of the ascities. Repeat angiography five days following operation demonstrated shunt patency with a portacaval gradient of 12 cm H_2O. Intercurrent episodes of Klebsiella sepsis and pneumonia led to transient impairment of both hepatic and renal function. Despite intensive supporting measures, irreversible liver failure and hepatorenal syndrome ensued causing the patient's death. Necropsy demonstrated a patent portarenal anastomosis, left lower lobe pneumonia, and 2.500 ml of sterile intraperitoneal ascitic fluid.

Discussion

The presence of any chronically implanted foreign body within the venous system creates a potentially thrombogenic situation. Catheter-induced intimal injury and impedance of normal flow may induce spontaneous venous thrombosis. Major intrathoracic venous thrombosis has occurred with prolonged superior vena caval catheterization for parenteral feeding. Norlund repeated a 4.7% incidence in 172 patients undergoing chronic superior vena caval catheterization via peripherally-placed and centrally-directed catheters.[4,11] Conversely, percutaneous central placement of venous catheters is less often associated with thrombotic complications, and appears to be the preferred method of catheter placement.[1]

Numerous reports have noted thromboembolic complications of the Spitz-Holter or Pudenz-Heyer ventriculoatrial shunt. Nugent et al. reported a 42% incidence of unrecognized thromboembolic lesions in 11 of 26 children studied with angiocardiography, including two cases of superior vena caval obstruction and two cases of thrombus formation within the right atrium.[13] Emery and Hilton, in a necropsy series, described significant

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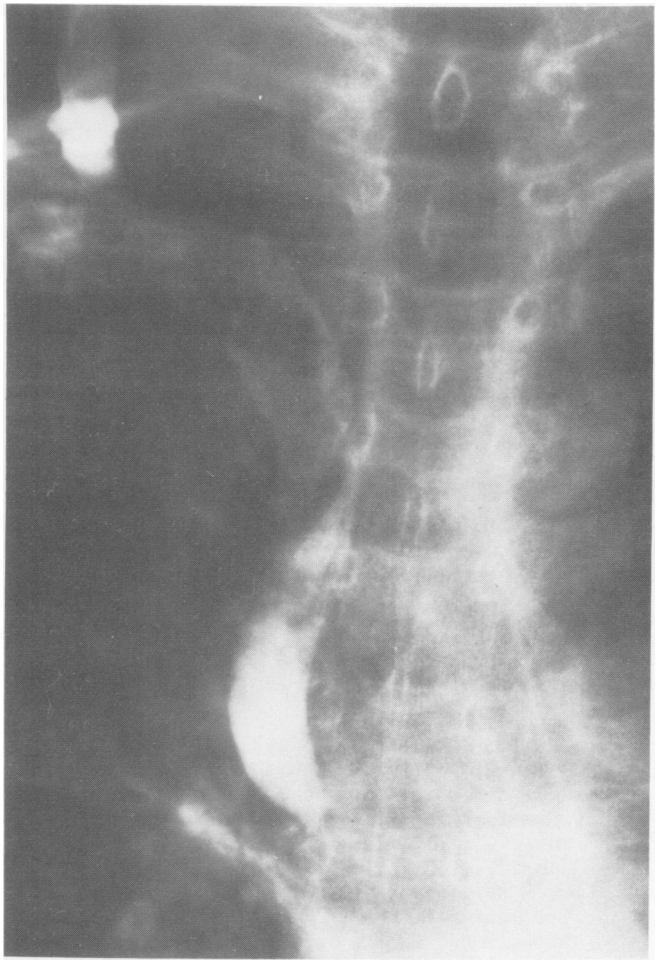

FIG. 1. Right internal jugular venous injection demonstrating superior vena caval obstruction with nonvisualization of the right atrium.

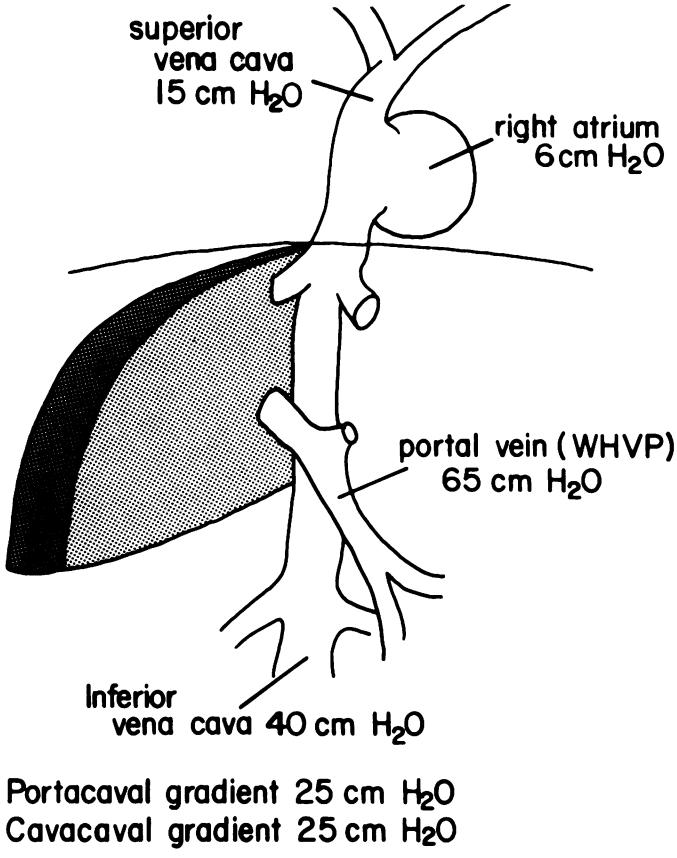

FIG. 2. Portacaval and cava caval relationships in reported patient with idiopathic Budd-Chiari syndrome.

pericatheter thrombosis and/or right atrial thrombus in 73% of 15 patients.[2] McNab confirmed the lethal association of infection and major intrathoracic venous thrombosis among patients with ventriculoatrial shunts.[10] Of 60 patients in his series followed for longer than nine months, ten died as a result of septic clot formation. Sepsis occurred in the complicated ventriculoatrial shunt group with a frequency of 20–67%.[2,10,13] Repeatedly negative blood cultures in the patient presently reported excluded the existance of a septic endophlebitis.

Stoney et al. reviewed the incidence of venous thrombosis following long-term transvenous pacing, and reported a 21% incidence of total venous obstruction in the innominate-subclavian system in 32 patients studied with venography.[20] Superior vena caval obstruction associated with transvenous pacemakers occurs much less frequently, but rarely produces symptoms of superior vena cava syndrome.[5,21]

Kamiya, in reviewing 733 cases of superior vena caval syndrome, found not a single association with chronic vena caval catheterization.[6] The pronounced paucity of symptoms in the presently reported patient is bizarre, since venography did not demonstrate extensive pericaval venous collaterals.

It is surprising that major venous thromboembolic complications have not been described after chronic peritoneovenous shunting. The operative details of the shunt procedure described by LeVeen in 1975 include transmural catheterization of the internal jugular vein

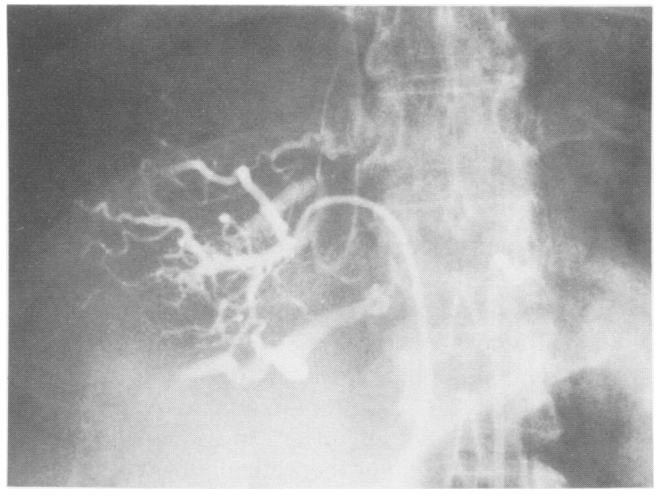

FIG. 3. Hepatic vein injection showing "pruning" and cutoff of intrahepatic venous tributaries.

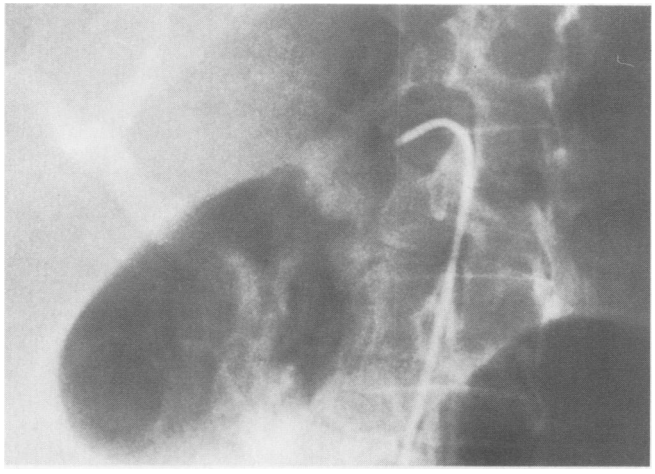

FIG. 4. Venous phase mesenteric arteriogram demonstrating patency of the portal venous system.

with ligation of the cephalad portion of the vein.[9] In contrast to transvenous pacemaker placement, where distal fixation may prevent a "whiplash" effect of the catheter tip, the peritoneovenous catheter tip lies free in the superior vena caval lumen and is subject to positional changes. Malposition of the catheter tip in the superior vena cava was excluded with venography in the current case.

Peritoneovenous shunt occlusion in a patient with venographically demonstrated Budd-Chiari syndrome raises some interesting speculation. Parker reviewed 164 patients with symptomatic hepatic veno-occlusive disease and documented a specific etiology in 29%.[15] Several cases were associated with pregnancy and more recently, cyclic hormonal therapy for contraception has been implicated as an etiologic factor in Budd-Chiari syndrome.[3,19] Permanent peritoneovenous shunting may be relatively contraindicated in this group of patients with potentially abnormal clotting mechanisms. Operative portal decompression, employing a side-to-side or hemodynamically equivalent shunt, affords a viable alternative to both relieve ascites and reduce portal venous hypertension. Excellent palliation without deterioration of liver function in patients with Budd-Chiari syndrome undergoing portosystemic decompression has been reported.[6,17] Orloff reported six patients with Budd-Chiari syndrome treated by operative portal decompression, with a six year survival rate of 83% and permanent relief of ascites.[14] Follow-up liver biopsies in four of his five survivors demonstrated absent hepatic venous congestion and resolution of parenchymal necrosis. Careful patient selection, greater attention to operative detail, and operative intervention prior to advanced fibrous changes within the liver theoretically has the potential to reduce the previously reported shunt mortality of 50%.[6,17]

Management of patients with Budd-Chiari syndrome remains controversial. Operative portal decompression, in addition to correcting the abnormal portacaval pressure relationships, obviates the need to place a foreign body within the venous system that may be a nidus for major thromboembolic complications such as that encountered in our patient. Although effective in reduction of ascites, indiscriminate use of peritoneovenous shunts in patients with idiopathic Budd-Chiari syndrome should be discouraged. Prospective studies comparing the effects of such shunts to operative portal decompression on hepatic pathophysiology and patient survival must be performed in order to provide a more rational basis for treating these patients.

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