Combined Caval and Portal Hypertension with Cirrhosis of the Liver:

A Problem in Management

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DESPITE the postoperative metabolic problems so frequently encountered and the increased risk of major operative procedures in patients with liver disease, a well constructed portal-systemic shunt offers the best available protection against recurrent bleeding from esophageal varices.[7,10,11,12]

Most patients who bleed from esophageal varices usually have portal pressures in excess of 300 mm. of water.[9,14] Normal inferior vena caval pressures range between 30 and 50 mm. of water varying with respiration, position and cardiac function. The success of the portal-systemic shunt in the control of subsequent bleeding from esophageal varices depends on the large pressure differential between the congested portal circulation and the normal systemic circulation. All of these operations shunt blood from the splanchnic circulation to the inferior vena cava below the liver.

This report discusses two cases in which the construction of a portacaval shunt in patients with cirrhosis of the liver and bleeding esophageal varices was unsuccessful because of striking elevation in inferior vena caval pressure. Retrohepatic compression from a hypertrophied and nodular caudate lobe caused the caval hypertension in both instances.

Case Reports

Case 1. W. C. G. was a 50-year-old man with a history of heavy ingestion of alcohol over many years. He entered the Manchester Veterans Administration Hospital with massive hematemesis, ascites and physical findings and laboratory studies consistent with Laennec's cirrhosis of the liver. The initial bleeding episode was managed with blood replacement and intravenous vasopressin. During the subsequent month he was prepared for elective portal-systemic shunt with a diuretic program and restoration of nutrition.

At laparotomy the free portal pressure was 325 mm. of water and the occluded hepatic portal pressure 300 mm. of water. All pressure measurements were recorded with #19 gauge needle and glass tube manometer connected by intravenous tubing. An end-to-side portacaval shunt was selected rather than a side-to-side shunt due to an hypertrophied caudate lobe. At the conclusion of the shunt the portal pressure had dropped only 40 mm. of water. Neither technical stricture nor angulation of the portal vein could account for this unsatisfactory drop in pressure. Inferior vena cava pressure was recorded at 285 mm. of water and concurrently a superior vena cava pressure was 100 mm. of water. No technical maneuver to rectify this situation was immediately apparent. The patient had a massive gastrointestinal hemorrhage on the tenth postoperative day and died.

At post-mortem examination regenerating nodules in the caudate lobe were found narrowing the lumen of the inferior cava to 5 mm. Retrohepatic obstruction of the vena cava was apparently responsible for the caval hypertension.

Case 2. N. E. was a 40-year-old man with a long history of alcohol ingestion. He was admitted to the Manchester Veterans Administration Hospital with massive upper gastrointestinal bleeding and physical findings and laboratory studies consistent with Laennec's cirrhosis. Because of persistent massive bleeding, a Sengstaken-Blakemore tube was inflated and he was transfused to restore normal blood volume.

At laparotomy on the following day, the free portal pressure was 380 mm. and an hepatic occluded portal pressure was 350 mm. of water. A pressure was recorded in the inferior vena cava as 260 mm. of water. It was not felt to be technically feasible to

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resect the entire caudate lobe. No other solution to avoid the problem of anastomosing two high pressure systems was apparent. Therefore, an end-to-side portacaval shunt was performed. Upper gastrointestinal hemorrhage recurred on the first postoperative day resulting in profound hepatic coma. The patient died on the third postoperative day.

At post-mortem examination the diameter of the retrohepatic vena cava was narrowed to 7 mm. by large regenerating nodules in a cirrhotic caudate lobe.

Discussion

The construction of a satisfactory portacaval shunt between the splanchnic and caval systems below the liver usually results in a marked drop in portal pressure. Most reported series[3,6,7,10,12] indicate that this type of decompression has resulted in a low incidence of recurrent bleeding from esophageal varices. In the two cases described, obstruction in the retrohepatic vena cava resulted in caval hypertension with pressure similar to those found in the splanchnic circulation. The resulting decompression through portacaval shunting was minimal. Recurrent variceal hemorrhage caused death in both patients indicating inadequate portal decompression.

Figure 1 illustrates the anatomical location as seen in both autopsies and the mechanism by which this type of retrohepatic caval compression causes hypertension in the inferior vena cava. A typical Budd-Chiari syndrome was not present in these two cases because the compression was below the entrance of the right and left hepatic veins.

These cases call attention to vena caval hypertension which appears to preclude the chance of a successful portal decompression through standard shunting procedures. The incidence of caval hypertension is not known since, to our knowledge, there are no previous similar case reports. The existence of this syndrome, however, might indicate that caval pressures should be measured prior to operation. If the pressure is significantly elevated in the inferior vena cava (greater than 250 mm. of water), other procedures such as portal-azygous disconnection as described by Tanner,[13] transthoracic ligation of esophageal varices,[1,4] or the transthoracic transposition of the spleen[15] might prove to be a more satisfactory solution to this complicated hemodynamic problem.

Summary

The incidence of rebleeding from esophageal varices is lowest following satisfactory portal-systemic shunts. Retrohepatic vena cava obstruction and subsequent infrahepatic caval hypertension precludes a successful portal-systemic shunt.

This report discusses two cases in which regenerating nodules in a cirrhotic caudate lobe caused significant retrohepatic vena cava obstruction. Preoperative pressure measurements of the infrahepatic and suprahepatic vena cava could document this obstruction and if found another surgical approach to this problem would be indicated.

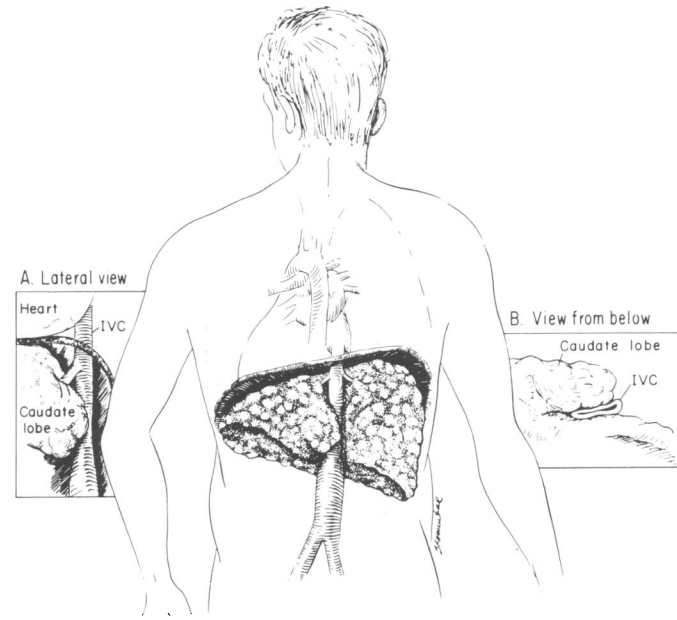

FIG. 1. Illustrates the anatomical location of the retrohepatic inferior vena cava compression in a lateral, inferior and posterior projection.

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Book Review

Renal Transplantation Theory and Practice, by J. HAMBURGER, J. CROSNIER, J. DORMONT, and J-F BACH. Williams and Wilkins Company, Baltimore, 1972, 326 pp.

THIS book reviews the spectrum of problems in renal transplantation from basic transplantation immunology, through numerous clinical aspects of transplantation, to important secondary considerations such as psychological, moral, and legal problems. The text is well organized and every important aspect of transplantation has been covered. Translation from French into the English version has been expertly accomplished by A. Walsh in collaboration with the authors. Every one working in the field should gain from reading the smoothly flowing text, but it will be particularly valuable to residents and fellows still in training because of its concise overview of the subject. Coming from the Hôspital Necker in Paris, it is not surprising that the book is more oriented toward the European literature and accomplishments of European investigators than are most reviews published in this country. It should also be noted that while the authors usually present opposing views in areas of controversy, a certain amount of dogmatic bias remains in the text. As an example, the authors state without qualification that a catheter must be left in place for at least 8 days if the bladder has been opened. Our unit would certainly take exception to such a statement since we have not seen complications with routine removal of the catheter between 24 and 36 hours. Numerous other points in protocol differ from the experience in other transplant centers, but represent honest opinion based on personal experience in a rapidly moving field where guidelines are still unstable. In all, the book is superior, if not outstanding, and will be a welcome text to newcomers entering the field.

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