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Peritoneovenous shunting in intractable ascites

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SUMMARY

Fourteen patients in whom peritoneovenous shunts were inserted for intractable ascites or malignancy were reviewed.

Reduction in ascites was obtained in all patients by the time of discharge with significant diuresis and weight loss. Significant decrease in haemoglobin, packed cell volume, platelet count and prothrombin time also occurred. Coagulation studies were abnormal in 60 per cent of patients in whom they were performed with bruising or detectable bleeding occurring in 28.5 per cent of all patients. Late blockage of the shunt occurred in five patients and was less frequent in Denver than in Le Veen type shunts.

Cumulative mortality one month after shunt insertion was 28.5 per cent and at one year was 78.5 per cent reflecting the severity of the underlying disease.

Peritoneovenous shunting should be reserved for palliation in patients resistant to full conventional medical therapy.

INTRODUCTION

In peritoneovenous shunting, ascitic fluid is returned to the venous circulation using a device consisting of silicone tubing and a one-way valve. A short general anaesthetic is required to insert one end of the shunt into the peritoneal cavity, tunnel the shunt subcutaneously over the rib cage and insert the venous end into the internal jugular vein. Peritoneovenous shunting is successful in the management of resistant ascites in cases of portal hypertension or malignancy. Several complications of the technique have, however, been reported.[1]

We present the limited experience of one unit with peritoneovenous shunting in patients with medically resistant ascites. Medical therapy included bed rest, salt restriction, water restriction and Frusemide and Spironolactone in doses up to 120 mg and 400 mg respectively daily.

PATIENTS

Fourteen consecutive patients in whom peritoneovenous shunts were inserted were reviewed. There were 12 males and two females. The mean age was 55.2 years (range 23-78 years). Ascites was secondary to alcoholic cirrhosis in seven

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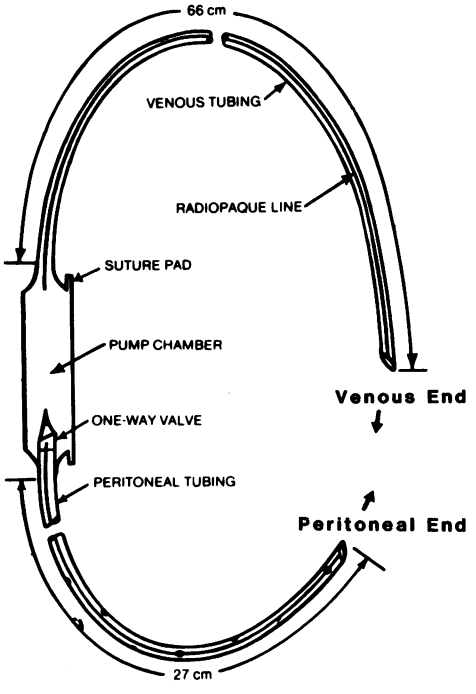

Fig 1. Diagram of Denver type of peritoneo-venous shunt (With permission of Downs Surgical Limited).

patients, to cryptogenic cirrhosis in four, to malignancy in two (one primary hepatocellular carcinoma and one adenocarcinoma of the stomach) and a further patient had severe Budd-Chiari syndrome. Two of the patients with alcoholic cirrhosis were subsequently shown to have hepatocellular carcinoma after insertion of the shunt. In the cirrhotic group there were seven patients in Child's grade C category and five in Child's grade B.

Six LeVeen and eight Denver shunts were inserted (Fig 1). Less than half a litre of ascitic fluid was removed at the time of inserting the shunt. Reduction of ascites was assessed using girth measurements, weight loss and urine output. In eight patients Frusemide was used to promote diuresis postoperatively. In some patients the rate of flow through the shunt was increased by using the technique of intermittent sucking through a straw to increase the negative intrathoracic pressure. Values of sodium, potassium, urea, creatinine, bilirubin, alanine transaminase, total protein, albumin, haemoglobin, packed cell volume, platelet count and prothrombin time before and after shunting were compared using the Wilcoxan Signed Rank Test for non-parametric paired data.

RESULTS

All but one patient had lost weight by the end of the first postoperative week; the mean weight loss was 4.6 kilograms ($p < 0.01$). Figure 2 shows the increased mean urinary output on the third postoperative day ($p < 0.01$) but by the end of the first week this had tailed off. The haemoglobin values fell from 13.6 to 11.2 gm/dl ($p < 0.05$), and packed cell volume from 0.396 to 0.339 ($p < 0.01$). The platelet count fell postoperatively from 187 x 10^3/L to 102 x 10^3/L ($p < 0.05$). Prothrombin time decreased in all 10 patients in whom it was measured (mean values 54 to 30 per cent ($p < 0.01$). No statistically significant changes occurred in the other blood tests.

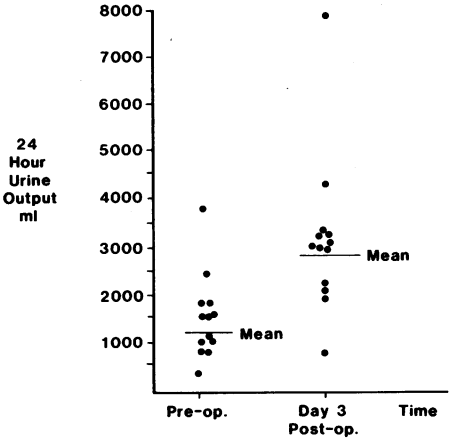

Fig 2. The 24-hour urine output preoperatively and on the third postoperative day. The changes were significant using the Wilcoxan Signed Rank Test ($p < 0.01$).

MORBIDITY

Abnormalities of coagulation were common after shunt insertion. Full coagulation studies were performed in 10 of the 14 patients and were abnormal in six. Clinically detectable bleeding from the wound, gastrointestinal or genitourinary systems was noted in four patients. Excessive bruising occurred in a further two cases and one patient died on the third postoperative day from uncontrollable disseminated intravascular coagulation.

Two patients developed ascitic leaks postoperatively and one of these subsequently developed staphylococcal septicaemia. Although this was initially controlled by antibiotics, one month later further septicaemia and staphylococcal ascitic infection proved fatal. One other patient died of staphylococcal septicaemia 20 days after insertion of the shunt. Two patients developed pulmonary oedema to some degree on the first and second postoperative days respectively, and both responded to conventional medical therapy. One patient bled from oesophageal varices on the second postoperative day but this settled with conservative management. Late blockage of the shunt occurred in five patients after an average of 10 months (range one to 30 months). Of the six episodes of blockage, four occurred at the abdominal catheter and only two at the venous catheter.

MORTALITY

Three patients died during the hospital admission in which the shunt was inserted. One died on the third postoperative day from disseminated intravascular coagulation, the other two from liver failure on the thirteenth and fourteenth postoperative days respectively. Of the 11 patients who survived to leave hospital, nine subsequently died after a mean survival of nine months (range three weeks to 40 months). Death was due to liver failure in four patients and, in two of these, hepatocellular carcinoma was documented prior to death. Other causes of mortality were infected ascites in two, carcinoma in two and variceal haemorrhage in one. The cumulative mortality was 28.5 per cent one month after operation; 42.8 per cent at two months; 57 per cent at six months and 78.6 per cent at one year. Only two patients still survive, one five years, the other seven years after insertion of their shunts.

DISCUSSION

The vast majority of patients with ascites of cirrhotic origin can be successfully managed with low sodium intake, fluid restriction and diuretics. Diuretics, however, contract the extracellular fluid compartment and this may induce renal failure refractory to further therapy. Failure to respond to medical management has been reported in 4.5 per cent of ascitic patients.[2] Intermittent intravenous reinfusion of ascitic fluid was seen to bring transient relief of ascites. In attempts to produce sustained relief a variety of the neurosurgical pressure activated shunts were tried.[3,4] In 1974 LeVeen introduced a peritoneovenous shunt specifically designed for management of resistant ascites.[5] The Denver shunt modification allows manual pumping of the valve.[6] These devices allow the peritoneal cavity to be drained through a one-way valve into a central vein. During inspiration the intraperitoneal pressure rises while the intrathoracic pressure falls; pressure differences of greater than four centimetres of water can then propel ascitic fluid into the central vein.

Peritoneovenous shunting is effective in reducing ascites. The weight loss and increased urine output in our patients cannot be attributed to the fluid volume removed on inserting the shunt, or to paracentesis alone. Indeed the degree of

weight loss and diuresis in our patients was similar to that reported in other series.[7, 8, 11] A fall in packed cell volume has been used as a measure of the effective transfer of fluid into the venous circulation.[8, 9] Although a statistically significant fall in packed cell volume occurred in our series, it was accompanied by a concomitant fall in haemoglobin which may, in part, account for the change. Bernhoft reported 84 per cent control of ascites at two months, 65 per cent at six months and 50 per cent at one and two years, which is slightly better than our findings.[9] In the hepato-renal syndrome, however, the results appear more variable. There are several reports of improvement[1, 7, 10, 11] although, in some of these, strict criteria for the syndrome may not have been applied.[12] The absence of statistically significant changes in total protein and albumin in our series is surprising, for it is assumed that the transfer of protein into the vascular compartment contributes to the reduction in ascites. Greenlee obtained similar results but noted that significant changes in protein concentration occurred six months after shunting.[2]

Coagulation abnormalities after shunt insertion are well recognised, the reported incidence varying from 20 to 91 per cent.[7-16] The incidence of coagulation abnormalities and detectable bleeding in our series is comparable with the observations of most authors.[7, 11, 13, 15] The triggering factor in the fluid has been ascribed to endotoxin, thromboplastin and activated clotting factors. Schwartz[14] and Ragni[15] believe that coagulopathy is related to release into the systemic circulation of ascitic fluid rich in fibrin degradation products. Treatment of the coagulopathy has consisted of heparin, epsilon-amino-caproic acid and infusion of blood products.[7] Infusion of antithrombin III appears ineffective.[16] Removal of an amount of ascitic fluid at the time of operation may reduce the risk of disseminated intravascular coagulation but results appear variable.[1, 8, 13] Spontaneous resolution of the abnormal coagulation tends to occur towards the end of the first postoperative week,[14] but, in severe cases, ligation of the shunt has been advocated to stop progression of the coagulopathy.[7]

Bleeding from oesophageal varices after shunting is related to expansion of the blood volume with resultant increase in portal pressure, and removal of a large amount of ascitic fluid at operation may reduce this possibility. LeVeen suggests that shunting should be performed only after decompression of the portal system.[7] Leakage of ascitic fluid is a commonly recognised problem after shunting.[7-9, 12] If the ascitic fluid becomes infected, mortality is high. Greenlee found infection a major factor in his early postoperative deaths, stating that preoperative ascitic fluid cell counts and culture should be performed to lessen the risk of shunting infected ascites. Foreign bodies such as catheters or intravenous cannulas should be removed early to minimise sources of potential infection. The low incidence of blocked Denver shunts compared with LeVeen in our survey confirms previous reports;[17, 19] it is related to the percutaneous pumping chamber of the Denver shunt which allows dislodgement of fibrin clots.

Significant symptomatic relief of ascites was obtained in our patients, tallying with the findings of other authors.[19-22] A major concern of shunting in malignant disease is the systemic dissemination of tumour cells, but this is usually not important in the short-term survival expected. Maat reported such a case[23] and Berger has noted subcutaneous tumour growth along the shunt.[24] Unfortunately blockage of the shunt is not uncommon in patients with malignant ascites.[22] The high cumulative mortality reflects the severity of the underlying disease process in the patients who were considered for shunting. In liver disease, Child's grading

correlates with survival after shunting, the poorest prognosis occurring in those patients with functional renal failure.[25, 26, 27]

Our study confirms that peritoneovenous shunting is effective in treating intractable ascites. However, morbidity and mortality are such that the technique should be reserved for cases resistant to full dose medical therapy.

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