Spontaneous Umbilical Rupture in Portal Hypertension with Massive Ascites *

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SPONTANEOUS rupture of the umbilicus due to massive ascites is a rare occurrence and usually fatal. Only nine previous cases have been reported, with a fatal outcome in all but two.[3, 4, 11, 20]

This paper reports one additional case of spontaneous umbilical rupture with survival. The patient suffered from an extreme degree of ascites which was totally unresponsive to medical treatment. Repeated paracenteses had become necessary and were associated with marked nutritional depletion and the development of an umbilical hernia. He was finally admitted to the hospital for surgical treatment, but prior to operation, the umbilical hernia burst, resulting in the sudden escape of about 20 liters of ascitic fluid. No deleterious effects occurred and 3 days later a side-to-side portacaval anastomosis was performed. Recovery from the operation was uncomplicated and he has obtained lasting relief from his ascites, with great improvement in his nutritional state.

Case Report

A 45-year-old man was admitted to the Royal North Shore Hospital on January 24, 1961 with complaints of gross abdominal swelling.

Thirteen months before admission he had noticed increasing swelling of the abdomen and reported to his local doctor (N. A. L.) who discovered the presence of ascites together with nodular enlargement of the liver. The patient had been accustomed to heavy daily alcohol intake but had no previous illness. His weight was 127 Kg.

He was referred to one of us (I. D. T.) for investigation. Physical examination on Feb. 23, 1960 revealed considerable ascites with a hard nodular liver palpable 7 cm. below the costal margin. Veins were prominent on the abdominal wall, but the spleen could not be felt. There was slight ankle edema and palmar erythema but no other significant abnormality. Tests of urine were normal. A barium meal x-ray study showed no esophageal varices and liver function tests showed serum bilirubin 0.4 mg./100 ml., prothrombin index 100% of normal, thymol turbidity 0.6 units, serum protein 7.4 g./100 ml. and albumin/globulin ratio 0.65.

The patient was treated with chlorothiazide (0.5 Gm. daily) with potassium chloride (1 Gm. three times daily), a low sodium diet and supplementary vitamins. He abstained from alcohol and tobacco intake. He showed improvement for several months but then his ascites became progressively worse until paracentesis had to be carried out for relief of discomfort. An umbilical hernia and bilateral inguinal herniae became noticeable with the onset of marked ascites.[2] Spironolactone (100 mg./day) was added to his treatment, but paracentesis became necessary with increasing frequency. Generalized wasting continued and the patient lost 18 Kg., notwithstanding gross ascites and edema of the legs.

Liver function tests remained essentially unchanged apart from slight reduction in serum protein level to 6.0 Gm./100 ml. (albumin/globulin ratio 0.76). In September, 1960, frequent infusions of albumin were commenced (100 Gm. every second day for 6 weeks). The patient looked and felt better on this regimen but paracenteses were still required. At admission to the hospital, aspiration

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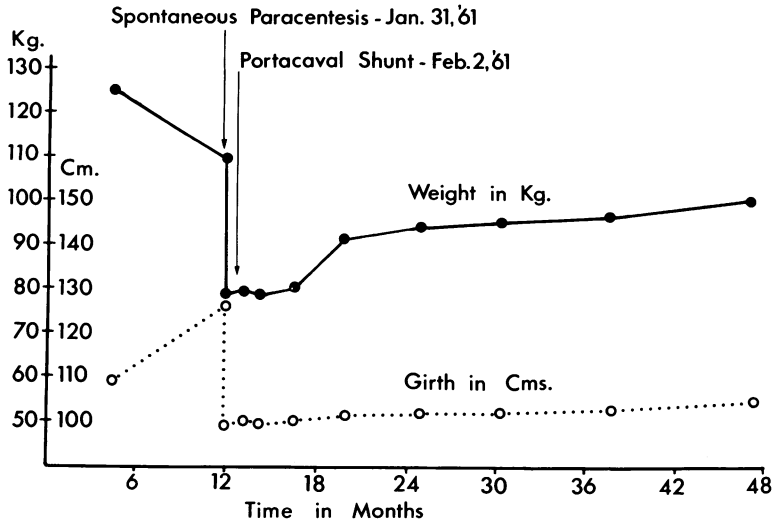

FIG. 1. Girth measurements and weight of patient with spontaneous umbilical rupture. Note weight loss despite increasing ascites up to time of rupture. Also note steady gain in weight with absence of ascites after side-to-side portacaval shunt.

was necessary at intervals of 3 to 4 weeks, with large aspirates of up to 10 L.

The patient was admitted to the hospital Jan. 24, 1961 for operation. Examination now revealed general bodily wasting, but with gross ascites and a protruberant umbilical hernia. Bilateral inguinal herniae were also present and neither liver nor spleen were palpable in the presence of the tense ascites. Blood pressure was 110/80 and pulse rate 80/min. Liver function tests were essentially unchanged and B.S.P. retention was 17%.

On Jan. 29, 1961 the umbilical hernia suddenly ruptured with copious discharge of ascitic fluid. Eighteen liters were collected in various receptacles and several more were lost in the bedding and on the floor. The patient's abdominal girth was reduced by 30 cm. and his weight by more than 27 Kg. (Fig. 1). The umbilicus was dusted with antibiotic powder and a firm abdominal dressing applied. Tetracycline (250 mg. every 6 hours) was begun, and an intravenous infusion of 100 Gm. of albumin and 1 L. of 10% dextrose was given. However the patient showed no sign of distress, maintaining his normal pulse and blood pressure and normal urinary output.

On Feb. 1, 1961 a side-to-side portacaval anastomosis was performed through a right thoracoabdominal incision. The patient made a satisfactory recovery and was discharged from hospital on the 20th postoperative day.

He has remained free of ascites for 3 years since operation. There has been a marked improvement in his nutritional state and he works at his previous occupation as a publican without limitation. He has no clinical symptoms of hepatic encephalopathy while on an unrestricted diet.

Discussion

Spontaneous paracentesis by umbilical rupture has been previously reported in nine cases of massive ascites, with death resulting in seven.[3, 4, 11, 20]

Large therapeutic paracenteses have also been associated with serious complications and have been held directly responsible for the death of patients in several instances.[5, 8, 9, 15]

The apparently benign effect of the spontaneous paracentesis in this patient, with a sudden loss of approximately 20 L. of ascitic fluid, prompts further speculation on the mechanism of the harmful effects described.

Death has been attributed to one or more of the following complications: hypotension, renal insufficiency, hepatic failure and peritonitis.[4, 5, 8, 16] Acute renal failure has been ascribed to a sudden increase in renal interstitial pressure as a result of decrease in intra-abdominal pressure.[4] The significance of purely mechanical effects in the production of renal failure must be questioned from the behavior of this patient who developed no oliguria or other evidence of renal failure.

A more likely explanation for the onset of acute renal failure is that given by Papper [16]

who suggests that large paracentesis may be followed by further decrease in an already precariously lowered volume of extracellular fluid, giving rise to hypotension and renal failure. This concept is supported by the observations of Gabuzda *et al.*[5] who noted that rapid recurrence of ascites after paracentesis was accompanied by decreases in plasma volume, serum sodium level and urine volume.

It is believed that this patient demonstrates the real value of repeated albumin infusions in supporting plasma volume. This patient developed no evidence of hypotension, hyponatremia or hypo-albuminemia despite the sudden loss of 20 L. of ascitic fluid and felt much improved as a result of the repeated infusions.

Peritonitis was associated with the death of four of the reported patients.[4] In this patient, peritoneal infection was probably avoided by the use of antibiotics and by the early definitive operation before ascites had redeveloped. In the reported cases, peritonitis often developed after some elapse of time with re-accumulation of ascitic fluid and repeated leakage from the perforation.

The successful application of a side-to-side portacaval shunt for the control of intractable ascites has been recently established by McDermott,[13, 14] Welch and others.[18] This represents an important change from earlier surgical practice, as the presence of ascites previously had been widely accepted as a contraindication to shunt operation for portal hypertension.[12] This attitude was determined by a high operative mortality in the presence of ascites, together with the fact that hydrostatic factors were often thought to be less significant than metabolic factors such as hyperaldosteronism, sodium retention, hypoalbuminemia and liver failure.[18] However, increasing evidence indicates that ascites, at least in some cases, is directly attributable to hepatic congestion caused by hepatic venous occlusion. Pathologic evidence of such postsinusoidal, hepatic outflow block,[7, 17] and the relatively high protein content of ascitic fluid is in keeping with the concept of an hepatic lymphorrhea resulting from hepatic congestion.[6] This concept is supported by certain clinical observations, *i.e.*, the absence of ascites in extrahepatic portal hypertension and the occasional presence of marked ascites in the presence of normal serum albumin where the plasma osmotic pressure would be normal. Side-to-side or double-barrelled portacaval shunts designed to produce hepatic decompression in addition to portal decompression have now been shown to be effective in many cases of ascites which have proved intractable to medical treatment.[1, 10, 13, 14, 19]

Summary

A case of spontaneous rupture of the umbilicus is reported as a complication of massive ascites due to hepatic cirrhosis. Nine previous cases of this complication have been reported, seven of which proved fatal. In this patient, ascites had progressed to a stage where frequent paracentesis was obligatory, despite medical treatment which included sodium restriction, diuretics and spironolactone. The rupture was accompanied by the sudden loss of 20 liters of ascitic fluid. Large therapeutic paracenteses have been reported to be sometimes lethal, and the various factors to which survival of this patient is attributed have been discussed. Soon after the umbilical rupture a side-to-side portacaval shunt was performed, with recovery and lasting relief of the ascites.

References

1. Barker, H. G. and K. Reemtsma: The Portacaval Shunt Operation in Patients with Cirrhosis and Ascites. Surgery, **48**:142, 1960.
2. Baron, H. C.: Umbilical Hernia Secondary to Cirrhosis of Liver. New Eng. J. Med., **263**: 824, 1960.
3. Eisenberg, M. M. and S. J. Hoye: Spontaneous Perforation of Umbilical Hernia; Need for

Early Herniorrhaphy in Ascites. Arch. Surg., **81**:514, 1960.
4. Flood, F. B.: Spontaneous Perforation of the Umbilicus in Laënnec's Cirrhosis with Massive Ascites. New Eng. J. Med., **264**:72, 1961.
5. Gabuzda, G. J., Jr., H. S. Traeger and C. S. Davidson: Hepatic Cirrhosis: Effects of Sodium Chloride Administration and Restriction, and of Abdominal Paracentesis on Electrolyte and Water Balance. J. Clin. Invest., **33**:780, 1954.
6. Gray, H. K.: Clinical and Experimental Investigations of the Circulation of the Liver. Ann. Roy. Coll. Surg. Eng., **8**:354, 1951.
7. Hales, M. R., J. S. Allan E. M. Hall: Injection Corrosion Studies of Normal and Cirrhotic Livers. Am. J. Physiol., **35**:909, 1959.
8. Hecker, R. and S. Sherlock: Electrolyte and Circulatory Changes in Terminal Liver Failure. Lancet, **2**:1121, 1956.
9. Holley, H. L. and J. S. McLester: Salt Depletion Syndrome Associated with Decompensated Cirrhosis of Liver. JAMA, **145**:392, 1951.
10. Large, A. M. and C. G. Johnston: Evaluation of the Portacaval Shunt. Am. J. Gastroent., **36**:15, 1961.
11. Lerner, S. and M. S. Rost: Spontaneous Abdominal Paracentesis. JAMA, **170**:1310, 1959.
12. Linton, R. R.: The Selection of Patients for Portacaval Shunts. Ann. Surg., **134**:433, 1951.
13. McDermott, W. V., Jr.: The Treatment of Cirrhotic Ascites by Combined Hepatic and Portal Decompression. New Eng. J. Med., **259**:897, 1958.
14. McDermott, W. V., Jr.: The Double Portacaval Shunt in the Treatment of Cirrhotic Ascites. Surg. Gynec. & Obstet., **110**:459, 1960.
15. Nelson, W. P., III, J. D. Rosenbaum and M. B. Strauss: Hyponatremia in Hepatic Cirrhosis following Paracentesis. J. Clin. Invest., **30**:738, 1951.
16. Papper, S.: Role of Kidney in Laënnec's Cirrhosis of Liver. Medicine, **37**:399, 1958.
17. Piper, D. W.: A Radiographic Study of the Portal and Hepatic Venous Systems in Cirrhosis of the Liver. Am. J. Dig. Dis., **6**:499, 1961.
18. Sherlock, S.: Discussion on Ascites and its Management. Proc. Roy. Soc. Med., **52**:247, 1959.
19. Welch, S. C., H. F. Welch and J. H. Carter: The Treatment of Ascites by side-to-side Portacaval Shunt. Ann. Surg., **150**:428, 1959.
20. Yonemoto, R. H. and C. S. Davidson: Herniorrhaphy in Cirrhosis of Liver with Ascites. New Engl. J. Med., **255**:733, 1956.