Management of Spontaneous Umbilical Hernia Disruption in the Cirrhotic Patient

JOHN H. LEMMER, M.D., WILLIAM E. STRODEL, M.D., JAMES A. KNOL, M.D., FREDERIC E. ECKHAUSER, M.D.

Umbilical hernia is a common finding in cirrhotic patients with ascites. Spontaneous disruption of the hernia and attendant discharge of ascitic fluid is an unusual and rarely reported complication in these patients and is associated with an overall mortality rate of nearly 30%. During the 5-year period 1977–1982, nine patients with hepatic cirrhosis and ascites were treated for spontaneous rupture of an umbilical hernia. Ascites was attributed to alcoholic cirrhosis in all cases and was present for an average of 21 months prior to rupture. In two cases, failed peritoneovenous shunts resulted in reaccumulation of massive ascites. Initial management included sterile occlusive dressings, fluid repletion, and intravenous antibiotic administration. Hernia repair was performed an average of 4.2 days after rupture. General anesthesia was used in eight cases and local anesthesia in one case. In one instance, the hernia became incarcerated and required urgent repair. Postoperative complications, including wound infection and colonic dilatation, occurred separately in two patients (22%). One patient died of hepatic failure 28 days after operation, for an overall mortality rate of 11%. Surviving patients have been followed for an average of 8 months, and most have done well. Spontaneous rupture of umbilical hernia in patients with ascites occurs uncommonly. Operative management is indicated uniformly and can be conducted safely when the patient's condition has stabilized. The prognosis is favorable for patients with good hepatic reserve.

SPONTANEOUS RUPTURE is a well recognized but uncommon and rarely reported complication of umbilical hernia in patients with hepatic cirrhosis and ascites. Since the first description in 1901 by Mixter, only 30 cases of umbilical hernia rupture and leakage of ascites have been described.[1] The serious nature of this complication is evident from the fact that ten of the 30 patients reported (30%) with this condition died. Nonoperative treatment has been almost uniformly fatal. Despite the fact that surgical intervention appears to be the treatment of choice, the timing of operation remains controversial.

Reprint requests: William E. Strodel, M.D., D2220 SACB, University Hospital, Ann Arbor, MI 48109.

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From the Department of Surgery, University of Michigan Medical School, and The Surgical Service, Veterans Administration Medical Center, Ann Arbor, Michigan

University of Michigan Experience

From 1977 to 1982, nine patients (8 men and 1 woman) with cirrhosis and umbilical hernia rupture associated with leakage of ascitic fluid have been treated at the University of Michigan. The mean age of all patients was 49 years (range, 27 to 65 years). Eight patients experienced acute rupture with the spontaneous discharge of large amounts of fluid ranging from 1 to 6 L, while one patient had gradual leakage of small amounts of fluid. One patient had undergone placement of a peritoneovenous (LeVeen) shunt that was nonfunctional at the time of hernia rupture, and another patient had previously had a peritoneovenous shunt inserted but later removed because of the development of a coagulopathy.

No precipitating factors were identified prior to rupture or fluid leakage. In all cases, the sudden loss of ascitic fluid was tolerated well, and in no case did cardiovascular instability develop. In six patients, rupture of the hernia occurred less than 24 hours prior to admission. In-hospital rupture occurred in two other patients who had been hospitalized for control of their ascites. One patient had first noted perforation and discharge of several liters of fluid 10 days prior to admission.

Physical stigmata of hepatic cirrhosis and portal hypertension were observed commonly and ascites was easily detectable in all patients. In addition, all patients had evidence of skin ulceration overlying the umbilical hernia. Average serum albumin and bilirubin levels were 3.5 gm/dl (range, 2.0 to 4.6 gm/dl) and 1.5 mg/dl (range, 0.7 to 3.5 mg/dl), respectively. The diagnosis of hepatic cirrhosis was confirmed by liver biopsy or gross inspection in all cases. Clinical details are summarized in Table 1.

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Urgent (within 24 hours of rupture) umbilical herniorrhaphy was performed in four cases. In the remaining five cases, hernia repair was performed 2 to 11 days (mean, 4.2 days) following rupture. The patients in the latter group were treated initially with sterile dressings applied to the hernia site, intravenous antibiotics, and fluid and electrolyte repletion. One patient developed an incarceration of his hernia 10 days following rupture and required emergency herniorrhaphy. Hernia repair was performed using general anesthesia in eight cases and local anesthesia in one case. The operative technique included debridement of necrotic skin overlying the hernia, running closure of the fascia with continuous non-absorbable suture (2.0 polypropylene), and primary closure of the skin. Intraoperative cultures of the ascitic fluid obtained in all cases were negative.

One patient died following operation (11%); postoperative complications occurred in two cases (22%). One patient, who had undergone urgent repair, developed massive non-obstructive colonic dilatation. Tube cecostomy was effective in decompressing the colon, but the patient died of liver failure 28 days following rupture. On admission, his levels of bilirubin and albumin had been 3.5 mg/dl and 2.6 gm/dl, respectively. Another patient developed a wound infection that later healed by secondary intention. The eight patients who survived operation (89%) have been followed for an average of 8 months. There has been no evidence of hernia recurrence, and good control of ascites has been achieved with medical means in all cases.

TABLE 1. *Clinical Details of Nine Patients with Ruptured Umbilical Hernia*

Age	Sex	Encephalopathy	Duration of Ascites (yrs)	Albumin (gm/dl)	Bilirubin (mg/dl)
55	M	No	5	2.6	3.5
65	M	No	.6	2.0	1.0
55	M	No	4	4.6	1.5
27	F	No	1	4.4	1.0
49	M	No	2	2.8	2.4
57	M	No	1	3.3	1.1
37	M	No	.6	3.4	0.8
50	M	No	1	4.4	0.7
48	M	No	2	3.9	1.6
		Mean	1.9	3.5	1.5

Discussion

In 1901, Mixter described the case of a 30-year-old woman with an umbilical hernia that ruptured spontaneously and discharged a large quantity of peritoneal fluid.[1] At operation, herniated omentum was resected and the hernia was repaired. The patient recovered completely. Since this first description, several single case reports and small scattered series have been reported, establishing that umbilical hernia disruption is an unusual but potentially serious complication of hepatic cirrhosis.

Abdominal wall hernias are known to occur more frequently in patients with hepatic cirrhosis, especially those with ascites, than in patients without liver disease. Chapman[2] reported an incidence of 10% in patients with liver disease not associated with ascites and one of 24% in similar patients with ascites. Likewise, Henrikson[3] noted that the overall incidence of hernias among patients with cirrhosis was 16%. In Chapman's series, umbilical hernia was the most common type of hernia (24 of 47 hernias), followed by inguinal, ventral, and epigastric hernias. Six patients had multiple hernias. Elevated intraabdominal pressure and a congenital, but previously unapparent, defect of the abdominal wall were presumed to be important factors in the formation of these hernias. Complications of umbilical hernia in the cirrhotic patient include acute rupture with discharge of copious amounts of ascites, gradual leakage of small amounts of ascites, incarceration, and rarely evisceration.

In a collected review of the literature, 30 patients (22 men and eight women) with ruptured umbilical hernia and leakage of ascites have been reported.[4–19] The average patient age was 51 years, with a range of 29 to 76 years. All patients had demonstrable ascites for an average of 21 months before hernia disruption. Nearly 30% of patients had a previous history of gastrointestinal blood loss. The etiology of cirrhosis included alcohol abuse in 17 patients (57%) and biliary tract disease in one patient (3%); in the remaining 12 cases (40%), no etiology was identified. Signs of cirrhosis and portal hypertension, including gynecomastia, angiomata, and muscle wasting were commonly observed. Ulceration of the skin overlying the hernia was described in 22 of the 30 cases (73%) prior to disruption. Disruption occurred spontaneously in 26 cases; in four other cases, rupture was apparently precipitated by local trauma, coughing, vomiting or esophagoscopy. In 17 cases, a large volume of ascitic fluid escaped following rupture (mean, 7.7 L; range, 1 to 20 L). In seven cases, leakage of fluid was slow and intermittent, and in six other cases, the data was insufficient to determine the rate of fluid loss. In three instances, evisceration of omentum and small bowel occurred; small bowel resection was required in one case.

Despite the large amounts of ascitic fluid that may be discharged at the time of acute rupture, spontaneous

paracentesis is usually tolerated well. In all reported cases except one, acute hernia rupture has been associated with a stable blood pressure and pulse. Data on the hemodynamic effects of paracentesis are somewhat discordant. In separate studies, Halpan[20] and Cruikshank[21] observed no important circulatory changes after rapid removal of large volumes of ascitic fluid in patients with pelvic tumors. Kowalski[22] investigated a group of cirrhotic patients and found that cardiac output was unaffected by paracentesis. Knauer[23] noted a significant increase in cardiac output after removal of small amounts of ascites fluid (250 to 1000 ml) and a slight decrease to below-control levels after removal of larger volumes (1250 to 1500 ml). Guazzi[24] removed 500 to 5000 ml of ascitic fluid over 50 to 90 minutes in 21 patients with cirrhosis and observed that cardiac output and stroke volume increased significantly. This phenomenon appeared to be related to augmented venous return. The fall in intraabdominal pressure following paracentesis was believed to decrease functional obstruction of the inferior vena cava and, therefore, resulted in increased venous return. Hypotension was observed in one patient following rupture. It is possible that this response was caused by a neural mechanism rather than by acute fluid shifts.

The timing of surgical repair following umbilical hernia disruption has been variable. Four patients underwent operation within 48 hours of disruption and two patients died. Seven patients underwent operation more than 48 hours after rupture and one patient died. Three patients underwent delayed repair at 11, 21, and 28 days following rupture and all of them survived. In 16 cases, the time between hernia disruption and repair was not known. In general, if incarceration of the hernia, evisceration, or peritonitis were not present, a longer period of preoperative hospitalization would allow time to improve nutritional status in addition to control of the patient's ascites.

Emergency operation did not appear to enhance survival. Initial stabilization with nonoperative measures, including intravenous antibiotics, sterile occlusive dressings, correction of coagulation abnormalities, and administration of intravenous fluids may be requisite prior to operation. However, delayed repair may predispose patients to an increased risk of postoperative wound infection; the two wound infections in the collected series occurred in patients who underwent delayed repair at 3 and 4 days, respectively, following disruption.

Twenty-two patients underwent operation and eight patients refused repair. Local anesthesia was used in eight patients, general anesthesia in five, spinal anesthesia in one, and no anesthesia in one. The type of anesthesia was not specified in seven instances. Two patients also underwent portasystemic shunting procedures at the time of herniorrhapy and both of them survived. Death following herniorrhaphy was attributed to variceal bleeding, renal failure, and sudden death in each of three cases (14%). Major postoperative complications, including variceal hemorrhage and wound infection, occurred in two cases and were treated successfully. Five other patients eventually died 2 months to three years following hernia repair.

Of the eight patients treated nonoperatively (compression bandages, paracentesis, and antibiotics), seven died (88% mortality rate). Death following nonoperative management was frequently attributed to peritonitis (five cases), most commonly due to *Staphylococcus aureus* (three cases). Other causes of death included liver failure and renal insufficiency.

In 1960, Baron[10] described the various indications for umbilical hernia repair in 16 cirrhotic patients with ascites. Six patients died after operation, five from advanced hemorrhage and one from liver failure. He postulated that interruption of portasystemic collateral channels in the umbilical region at the time of herniorrhaphy resulted in increased volume and pressure in the coronary-esophageal veins and thus increased the propensity for variceal rupture. In the collected series, variceal hemorrhage occurred after hernia repair in two patients, an incidence of 9%. One of these patients died. Of the patients who did not undergo operation, two developed variceal bleeding following rupture (25%). O'Hara[17] reported a similar incidence of postoperative hematemesis (6%) in 35 cirrhotic patients who underwent umbilical hernia repair for various emergent and elective indications. These figures are consistent with the reported rates of 11% to 34% for hemorrhage from esophageal varices in cirrhotic patients without umbilical hernia. A more plausible explanation for variceal hemorrhage would seem to be a sudden increase in portal pressure, precipitated by the intravenous administration of volume expanders such as albumin and crystalloid solution during the acute resuscitative phase.

Fisher and Calkins[19] proposed that the level of serum albumin was of prognostic value in patients with umbilical hernia disruption. They observed that all patients with serum albumin levels above 2.4 mg/dl survived. Since the albumin level is presumably a good measure of hepatic reserve, such a relationship is not unexpected. Measurements of serum albumin were available from 18 patients in the collected series. The mean albumin level was 2.8 gm/dl. Of 11 patients with a serum albumin level greater than or equal to 3.0 gm/dl, three died (mortality rate of 27%); in contrast, of the seven patients

with albumin levels of less than 3.0 gm/dl, three died (mortality rate of 43%). In our experience, three patients had serum levels of albumin of less than 3.0 gm/dl and one died (mortality rate of 33%); none of the patients with levels of albumin greater than or equal to 3.0 gm/dl died.

The advisability of elective repair of the uncomplicated umbilical hernia in cirrhotic patients has long been a subject of debate. Elective herniorrhaphy obviates emergent procedures for complications such as perforation or incarceration. Based largely on Baron's discouraging results,[10] some surgeons have taken the view that elective herniorrhaphy should not be performed on patients with ascites. However, Baron's 38% mortality rate for patients with ascites undergoing umbilical hernia repair is based on both elective and emergent cases. Deaths occurred in cases that required operation for acute perforation or incarceration. Yonemoto and Davidson[6] successfully performed umbilical herniorrhaphy on nine cirrhotic patients without complication, except for one instance of wound infection. They stressed the importance of preoperative nutritional support and control of ascites to achieve these results. More recently, O'Hara et al.[17] reported umbilical herniorrhaphy in 35 patients with cirrhosis. Sixteen of these procedures were performed for urgent or emergent indications, with an overall mortality rate of 16%. No deaths occurred in patients who underwent elective herniorrhaphy. The majority of the patients in this series had been hospitalized for less than 1 month prior to operation, and three of the six deaths occurred in patients who had been hospitalized for less than 2 weeks. O'Hara concluded that a shorter period of preoperative preparation increased the mortality and morbidity rates. It is apparent that elective umbilical herniorrhaphy can be performed safely in the cirrhotic patient with ascites. Certainly, the best results will be obtained with preoperative efforts to correct fluid and electrolyte abnormalities, to improve nutrition, and to control ascites.

Peritoneovenous shunts may be indicated to control ascites in a small percentage of patients with medical intractability. Eisenstadt[25] reported five such patients with umbilical hernias who underwent peritoneovenous shunt. Three of these patients developed an incarcerated hernia following reduction in ascites volume and a decrease in the diameter of the fascial defect. Perhaps hernia repair in this setting, should precede or be performed coincident with shunt insertion.

The development of an umbilical hernia in a patient with ascites should alert the physician to a potentially serious condition. The presence of discoloration, ulceration, or a rapid increase in the size of the hernia signal impending rupture. O'Hara described herniorrhaphy in seven such patients, two of whom died of hepatorenal failure. Optimal preparation of the patient should include antibiotics, ulcer care, careful diuresis of ascites, and nutritional support. If rupture occurs, these measures can be continued for several days with close observation of the patient prior to umbilical hernia repair.

Summary

A total of 39 cases of umbilical hernia disruption (30 cases from a review of the literature and nine cases from our institution) have been reported in cirrhotic patients with ascites. Umbilical hernia disruption is an unusual but potentially fatal complication of ascites. Operative management of the ruptured, leaking, or ulcerated hernia is clearly indicated—but not necessarily on an emergent basis. Thirty-one patients have undergone hernia repair with an operative mortality rate of 13%. Seven of the eight patients treated without operation died. The timing of operation depends on the patient's general condition and the degree of hepatic reserve. In the absence of other indications such as incarceration, evisceration, or peritonitis, the patient's general condition may be improved prior to herniorrhaphy. Repair may be performed urgently or may be delayed in order to correct coagulation defects and fluid and electrolyte abnormalities. In either case, sterile occlusive dressings and antibiotics should be used from the time of admission until operation. Decompression of ascites and correction of fluid and electrolyte abnormalities should contribute to a successful surgical result. Impending rupture of an umbilical hernia should be handled in a similar manner. Elective repair of the uncomplicated umbilical hernia in the cirrhotic patient is recommended, but only following in-hospital optimization of the patient's general state of health.

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