Umbilical Hernia Incarceration: A Complication of Medical Therapy of Ascites

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Umbilical hernias occur frequently among patients with cirrhosis and ascites. Common complications include incarceration, leakage, and rupture. Two patients experienced umbilical hernia incarceration while undergoing medical therapy of their massive ascites—a complication not previously described. Decompression of ascites apparently causes decreased tension on the umbilical hernia ring with subsequent trapping of the hernia sac contents. Mortality of incarcerated umbilical hernias is significant (3-14%). Patients receiving medical therapy for ascites should be examined carefully for the presence of umbilical hernias and, if present, the hernias should be observed closely during the course of treatment.

INTRODUCTION

Umbilical hernias are common among patients with hepatic cirrhosis and ascites. Hernia incarceration has been described in ascitic patients after needle paracentesis and after peritoneovenous shunting, presumably as a result of decompression of the abdomen (1, 2). We recently observed the incarceration of umbilical hernias in two cirrhotic patients who were undergoing successful diuresis of ascites during medical management of their condition. Both patients required emergency surgery and strangulated bowel was encountered in one. This unusual but potentially fatal complication of the medical treatment of ascites has not been previously described.

CASE REPORTS

Patient 1

A 34-year-old man with a history of severe alcoholism of several years duration presented with the chief complaint of abdominal swelling. The patient was noted to be mildly jaundiced with cutaneous angiomata and obvious gynecomastia. Massive ascites was present and his abdominal wall possessed multiple dilated superficial veins. Treatment with fluid and salt restriction (1500 ml fluid and 1.0 g sodium per day) and diuretics (spironolactone 200 mg/day and furosemide 40–120 mg/day) was instituted. A liver scan revealed hepatocellular disease. After 3 wk the patient had lost 30 lb. Routine exam found his ascites to be diminished and he was noted to have a nontender, easily reducible umbilical hernia. Medical treatment of his ascites continued and after 6 wk the patient's weight loss totalled 50 lb. One evening he complained of abdominal pain and vomited. Examination found him to have high-pitched bowel sounds and a tender irreducible umbilical hernia with reddening of the overlying skin. Abdominal x-ray revealed small bowel obstruction. Emergent surgical exploration was performed through a transverse subumbilical incision. A strangulated Richter's-type hernia was found necessitating resection of 10 cm of ileum with primary anastomosis in addition to Mayo-type umbilical herniorrhaphy. Postoperatively the patient had no complications. Follow-up examination 4 months later found him to be continuing his abstinence from alcohol. His hernia repair site was well healed without evidence of recurrence and he was free of ascites.

Patient 2

The patient was a 55-year-old man with biopsy-proven hepatic cirrhosis thought secondary to severe alcohol abuse of 15-20 years duration. He was known to have ascites for at least 4 years and an umbilical hernia had previously been noted. He had two prior episodes of variceal hemorrhage. Admission was prompted on this occasion by a fluid leak from the umbilical hernia which had occurred several days before. Examination found the patient to have persistent ascites and an easily reducible umbilical hernia which possessed a small ulceration. There was no evidence of peritonitis and no fluid was leaking.

A program of fluid and salt restriction (1000 ml fluid per day and 1.0 g sodium per day) and diuretic administration (150 mg spironolactone per day) was begun. By the 10th hospital day the patient had lost 12 lb. On that day his umbilical hernia became painful and irreducible necessitating emergency operation. Exploration found incarcerated, but viable, small bowel within the hernia sac. A Mayo-type repair was performed and the patient had an uncomplicated postoperative course. Two months later he underwent elective mesocaval shunt without problems. Follow-up examination 1 year later found him to be free of ascites and without evidence of recurrent umbilical hernia.

COMMENT

Umbilical hernias occur in more than 20% of patients with hepatic cirrhosis and ascites (3). They are generally recognized as being the result of increased intraabdominal pressure causing herniation of abdominal contents through the congenital patent ring. Complications are principally incarceration (with or without strangulation), leakage, rupture, and pain (4). Operative repair, elective or emergency, is thought to be of increased risk in the cirrhotic patient (1, 4, 5). But elective herniorrhaphy is generally advised to prevent future complications and the need for emergency procedures (6–8).

Incarceration of a hernia occurs when the hernia contents (usually small bowel) can no longer be replaced to their normal locale. Strangulation results when the contents of an irreducible hernia have sufficient compromise of their blood supply to cause ischemia. If the obstruction is severe, grangrene of the hernia contents will occur.

Umbilical hernia incarceration is associated with significant mortality. A recent report by Hjaltason (9) described 90 umbilical hernias, 16 of which (18%) presented in the incarcerated state. In 12 of these cases small bowel was present within the sac and in two of 16 gangrenous bowel was present requiring resection. Mortality of incarcerated umbilical hernia was 14%. Morgan *et al.* (10) describe 101 cases of incarcerated umbilical hernias. Operative mortality was 3%; nonoperative treatment resulted in 33% mortality (four of 13 patients). O'Hara *et al.* (4) describe 35 patients with cirrhosis, ascites, and umbilical hernias. Thirteen (37%) of these patients were operated on for incarceration; three patients had strangulated bowel. Mortality of incarcerated umbilical hernia in these patients with cirrhosis and ascites was 8.6%.

Decompression of ascitic fluid can result in umbilical hernia incarceration. Baron (1) described two cases of incarceration after paracentesis. Decompression of ascites by peritoneovenous shunting has been reported to cause incarceration in two patients (2). In each of these cases emergent herniorrhaphy was required and resection of bowel was necessary in one instance. In the cases presented herein, decompression of ascites achieved medically by fluid and salt restriction and diuretic administration also resulted in umbilical hernia incarceration. While patient 2 had a hernia of long duration, patient 1 developed a clinically apparent hernia during the course of his therapy. Both patients underwent emergency operation and had uncomplicated postoperative courses. In all of these circumstances, removal of the ascites (by needle, shunt, or medical therapy) apparently leads to decreased tension on the umbilical hernia ring, with subsequent decrease in diameter, and trapping of the hernia sac contents.

Common complications of the medical management of ascites are electrolyte acid base disturbances, hepatic encephalopathy, and hepatorenal syndrome (11). Umbilical hernia incarceration, as demonstrated by these two patients, is another potential serious complication of this form of therapy. It is recommended that before beginning therapy for ascites, patients be examined closely for the presence of umbilical hernias. If a hernia is present, the patient should be made aware of the possibility of incarceration with therapy and the hernia should be observed closely. Elective surgical repair should be performed when the cirrhotic patient's condition permits. If incarceration occurs, prompt surgical therapy is mandatory.

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