CASE REPORT

Incarcerated inguinal hernia as a complication of new-onset ascites

Matthew R Zeitler,¹ Noah Wouk²

¹Department of Family Medicine, UNC Health Care System, Chapel Hill, North Carolina, USA
²Department of Family Medicine, UNC Health Care System, Chapel Hill, North Carolina, USA

Correspondence to

Dr Matthew R Zeitler,
 matthew.zeitler@unchealth.unc.edu

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SUMMARY

This case report presents a patient who developed an acute incarcerated inguinal hernia as a result of newly acquired ascites, a complication of ascites that is not well documented in the medical literature. His story highlights an important and potentially morbid complication of new-onset ascites.

BACKGROUND

The following case is presented because it challenges the conventional wisdom regarding the natural history of ascites-associated inguinal hernias and warns of a potential morbid outcome. The majority of the existing data regarding abdominal hernias and ascites are derived from observational studies involving patients with liver cirrhosis, where we know ascites is a risk factor for the development of abdominal wall hernias and their attendant complications (bowel incarceration, bowel strangulation and abdominal wall perforation).¹

At the same time, the incidence and natural history of groin hernias is less well known, particularly among patients without cirrhosis.^{2,3}

CASE PRESENTATION

The patient is a 45-year-old African-American man with a medical history significant for obesity (body mass index 41.9), diet-controlled type 2 diabetes mellitus, hypertension and tobacco abuse (7.5 pack years), who was hospitalised for worsening epigastric pain, nausea and vomiting in the setting of 3 months of dyspepsia, bloating, anorexia and unintentional weight loss. He denied alcohol use or pertinent family history of GI malignancy. On presentation, the day of his hospitalisation, physical examination revealed normal vital signs and a mildly distended, soft abdomen with epigastric tenderness, normoactive bowel sounds, no appreciable free fluid wave, no rebound or guarding and no palpable masses, hernias or hepatosplenomegaly. Of note, the patient had presented to an emergency department 1 month prior with similar symptoms, where X-ray CT of the abdomen revealed duodenitis and right hepatic steatosis without appreciable ascites.

INVESTIGATIONS

Initial laboratory analysis was significant for elevated lipase (3945 U/L (Ref 44–232 U/L)) and elevated liver function tests (total bilirubin 2.8 mg/dL (Ref 0.0–1.2 mg/dL), aspartate transaminase

519 U/L (Ref 19–55 U/L), alanine transaminase 557 U/L (Ref 19–72 U/L) and alkaline phosphatase 276 U/L (Ref 38–126 U/L)). Abdominal ultrasound demonstrated evidence of gallbladder wall thickening, biliary sludge and biliary ductal dilation. The patient was initially diagnosed with acute pancreatitis thought to be secondary to microlithiasis or biliary sludge.

Endoscopic retrograde cholangiopancreatography with endoscopic ultrasound was then performed, revealing inflamed gastric mucosa (which was biopsied), one non-bleeding gastric ulcer, diffuse severe inflammation and oedema in the duodenal bulb, biliary sludge in the gallbladder with gallbladder wall thickening, dilation in the common hepatic duct, pancreatic head parenchymal abnormalities and a moderate amount of peritoneal fluid.

These findings triggered concern for disease outside the gastrointestinal tract, and so repeat X-ray CT of the abdomen was obtained, which confirmed the presence of new moderate-large volume ascites that was not seen on the CT scan performed 1 month prior (figure 1).

Diagnostic paracentesis was then performed, with the following results: serum ascites albumin gradient (SAAG) 0.6 g/dL, amylase 283 U/L (Ref not established) and polymorphonuclear neutrophils 333/mm³. These findings are suggestive of pancreatic ascites, secondary bacterial peritonitis or peritoneal carcinomatosis. Fluid culture grew MSSA thought to be a skin contaminant, as the patient exhibited no other signs or symptoms of infection. The diagnosis of peritoneal carcinomatosis was confirmed when cytological examination showed reactive mesothelial cells. Pathology of the gastric mucosa later returned as invasive poorly differentiated adenocarcinoma with focal signet ring cell differentiation.

On getting up and out of bed on hospital day 5, the patient experienced acute right groin pain radiating to his right testicle. Examination of the external inguinal ring through the scrotal skin revealed a tender palpable mass, and immediate scrotal ultrasound confirmed the presence of incarcerated right inguinal hernia. The patient denied previous history or symptoms of inguinal hernia, chronic cough or recent history of heavy lifting.

DIFFERENTIAL DIAGNOSIS

The most common causes of acute scrotal pain in adults are testicular torsion and epididymitis. Other causes include inguinal herniation, Fournier's

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Figure 1 Serial CT scans obtained during hospitalisation (right) and 1 month prior (left), demonstrating recent accumulation of new ascites.

gangrene and torsion of the appendix testis. Physical examination was not suggestive of testicular torsion, as the patient's cremasteric reflex was intact bilaterally, and he did not exhibit a high-riding, transversely oriented testicle (also known as bell clapper deformity). Because examination did not reveal epididymal tenderness or pain relieved by elevation of the testicle (negative Prehn sign), acute epididymitis was thought to be unlikely. There were no findings of erythema, oedema, induration, bullae or crepitus to suggest Fournier's gangrene. The testis itself was non-tender and there was no blue dot sign to suggest appendiceal torsion. Palpation of a tender bulge through the right external inguinal ring correlated most with acute inguinal herniation, which was confirmed with ultrasound.

TREATMENT

General surgery was notified, and their team was able to successfully reduce the patient's inguinal hernia at bedside.

OUTCOME AND FOLLOW-UP

Since discharge from the hospital, the patient experienced no recurrence of inguinal hernia symptoms.

After receiving the diagnosis of gastric adenocarcinoma, the patient elected to pursue diagnostic laparoscopy with a peritoneal biopsy for surgical staging, which revealed peritoneal carcinomatosis. In light of the diagnosis of metastatic gastric adenocarcinoma—an incurable malignancy—the patient decided to pursue palliative chemotherapy.

DISCUSSION

Cirrhosis is the most frequent cause of ascites; other causes include malignancy, infection, pancreatitis, heart failure, lymphatic leakage and the nephrotic syndrome.¹

Abdominal wall herniation (umbilical, inguinal and incisional) is a potential complication of ascites and carries with it the risk of bowel incarceration, bowel strangulation and abdominal wall perforation.¹ Increased intra-abdominal pressure is the proposed mechanism by which ascites leads to abdominal wall herniation, although comorbid malnutrition may contribute by weakening muscle and fascia.⁴ It is not known whether the incidence of abdominal wall herniation varies with the pathogenesis of ascites (ie, whether malignant ascites is more or less likely to cause herniation when compared with cirrhotic ascites), as the preponderance of available literature addresses only cirrhosis-induced ascites.^{2 4 5} Inguinal herniation is a known complication of pseudomyxoma peritonei, a rare appendiceal neoplasm that produces mucinous ascites.^{6 7}

Umbilical hernias are the most common abdominal wall hernia associated with ascites. Approximately 20% of cirrhotic patients with ascites will develop an abdominal hernia, and the incidence of umbilical herniation increases with the duration and volume of ascites.^{2 5}

Ascites-associated groin hernias are thought to differ from ascites-associated umbilical hernias in important ways, although less is known about their frequency and natural history.^{2 3} For example, in contrast with umbilical hernias, inguinal hernias are described as not influenced by the duration or quantity of ascites, and morbid outcomes have been found to be less frequent.^{2 3} There exist no documented previous descriptions of incarcerated inguinal hernia as a complication of new-onset ascites.

This case suggests that new-onset ascites can precipitate potentially morbid inguinal hernia complications.

Surgical management—including elective surgery for symptomatic hernias—is an option for ascites-associated abdominal hernias, although the underlying comorbid cause of the ascites may increase operative risks.⁴