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## Case Report

# Two cases of spontaneous rupture of an umbilical hernia, a rare complication of portal hypertension

## Abstract

Portal hypertension is a severe complication of liver cirrhosis frequently leading to formation of ascites. We describe two patients that presented with a spontaneous rupture of an umbilical hernia, a rare complication of liver cirrhosis. Umbilical hernia itself however is a common complication of portal hypertension occurring in about 20% of the patients. In general, umbilical hernias in patients with liver cirrhosis warrant elective surgical repair, in a center of expertise with liver cirrhosis, after optimal management of ascites.

## Introduction

Portal hypertension is a haemodynamic abnormality and one of the most severe complications of liver cirrhosis which include ascites, hepatic encephalopathy and bleeding from gastro esophageal varices [1]. We describe a rare complication of portal hypertension in two patients with alcoholic liver cirrhosis and discuss the treatment strategies to prevent this complication.

## Case Reports

Patient A, a 63-year-old male patient, was diagnosed in 2009 with alcoholic liver cirrhosis and an umbilical hernia. His further medical history included diabetes mellitus with micro- and macrovascular complications and obesity. He presented at our emergency department after he noticed a massive fluid leak from his umbilicus. Prior to presentation he had suffered from abdominal pain and an umbilical mass for three weeks. On physical examination we saw an alert patient, haemodynamically and respiratory stable, without fever or jaundice. The abdomen was distended by an enormous mass of ascites and a very large reducible umbilical hernia with skin necrosis and leaking ascites (Figure 1).

Laboratory values at admission are shown in the Table 1. The Child Pugh classification was grade B (score 8) and the MELD-score 9. Ascites white blood count was  $0.9 \times 10^9/L$ , suggestive of infected ascitic fluid.

Operation followed on the day of admission. The necrotic skin and hernia sac were excised, the hernial sac had no other

content than ascites. The umbilical defect of 5 cm was closed with polydioxanone (PDS) sutures. Mesh was not used because of suggestive infection of the ascites. Abdominal drainage of ascitic fluid was performed in order to decrease the tension on the wound. Subsequently, he was treated with strict bed rest and diuretics. Culture of the ascitic fluid confirmed bacterial peritonitis with several anaerobes that was treated with antibiotics and albumin.

At this time we considered, but decided not to place a transjugular intrahepatic portosystemic shunt (TIPS), because of suggestive infection of the ascites in a patient with active



Figure 1:

Table 1:

	Patient A	Patient B
Hemoglobin (g/dL)	9.0	7.0
MCV (fL)	81	75
Platelet count (x 10 <sup>9</sup> /L)	203	228
White blood count (x 10 <sup>9</sup> /L)	7.9	7.4
Prothrombin time (INR)	1.2	1.1
Albumin (g/L)	30	38
Sodium (mmol/L)	136	141
Potassium (mmol/L)	3.6	3.6
Creatinine (μmol/L)	93	78
CRP (mg/L)	96	2
Alkaline phosphatase (U/L)	141	92
Gamma glutamyl transpeptidase (U/L)	153	106
Aspartate aminotransferase (U/L)	26	36
Alanine aminotransferase (U/L)	19	13
Serum bilirubin (μmol/L)	14	5

alcohol abuse. After antibiotic treatment, there was no refractory ascites.

Following wound healing, after a period of three weeks, the abdominal drain was removed. The next week, the patient had renewed abdominal distention with a tender umbilical hernia. Abdominal ultrasound showed incarcerated bowel. For the second time he underwent emergency surgery, which was four weeks after the first operation he had. Strangulated omentum was removed and an incarcerated but viable loop of the small bowel was reduced and the recurrent hernia defect of 5 cm was closed again and an onlay monofilament polypropylene mesh of 10 x 10 cm reinforced the abdominal wall. Ascites culture was sterile at this time, but the operation was complicated by prosthetic mesh infection with an *Enterococcus faecalis*, successfully treated with antibiotics for one week. Later, a period of recurrent ascites and again bacterial peritonitis was also treated with antibiotics for another two weeks.

After two months of hospitalisation, the patient was discharged for further rehabilitation. One year later the patient was admitted again, now with refractory ascites and encephalopathy, but without signs or symptoms of a recurrent hernia, both on clinical and ultrasound examination.

Patient B, a 52-year old male was diagnosed in 2012 with alcoholic liver cirrhosis. During the past year he developed ascites and an umbilical hernia. He was referred from a regional hospital to our centre because of multiple liters of ascites which had leaked from a spontaneous rupture of his umbilical hernia.

Prior to referral he actively abused alcohol and had no physical complaints.

On physical examination we saw an alert patient, haemodynamically and respiratory stable, without fever or jaundice. The abdomen was not distended and showed an umbilical hernia with a small ulcer of 5 mm, no longer leaking ascitic fluid. The Child Pugh classification was grade A (score

6) and the MELD-score 7. Diuretics and cefotaxim were already started at the regional center and were continued. There were no signs of infected ascites.

A CT-scan of the abdomen was made which showed a cirrhotic liver, ascites and esophageal varices, but no signs of a patent umbilical vein. Several days later operation followed. The hernial sac was excised and abdominal drainage of ascites took place. The defect had a diameter of 1 centimeter. The peritoneum was closed with PDS sutures. The hernial defect was closed with a 6 x 6 cm soft polypropylene sublay mesh.

The patient recovered quickly from surgery. He was discharged from the hospital after a week when a stable situation was reached with diuretic therapy.

## Discussion

Spontaneous rupture of an umbilical hernia with massive ascitic leakage is a rare complication of portal hypertension in liver cirrhosis [2-6]. However, umbilical hernia itself is a common complication of portal hypertension occurring in about 20% of the patients [7]. Any surgical intervention in patients with advanced liver disease has a high risk of complications. Therefore timing of umbilical hernia repair in cirrhotic patients with ascites is difficult. Umbilical hernia repair in patients with a patent umbilical vein is contraindicated. Repair of an umbilical hernia necessitates the complete freeing of the umbilical ring and the ligation of the reopened umbilical vein. If the vein is ligated during umbilical hernia repair, the outflow of the portal circulation is hampered leading to acute portal vein thrombosis and subsequent acute liver failure necessitating emergency liver transplantation [7, 8].

Although bowel incarceration is reported as a rare complication in this specific patient group, mortality rates are reported as up to 30% when emergency surgery for incarceration or strangulation is necessary [9-11]. Therefore, in general, umbilical hernias in patients with ascites warrant elective surgical repair, in a center of expertise with liver cirrhosis, after optimal management of ascites. Even for patients on a waiting list for liver transplantation elective surgery should be considered due to a long waiting time [7,8,12]. In our cirrhotic patients with an umbilical hernia, and without an open umbilical vein, elective hernia repair would have prevented spontaneous rupture of hernias. We presented two cases which confirm the need for correction of an umbilical hernia, preferably in a stable clinical situation.

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