Nefropatik Sendromlu Bir Hastada Karın Ağrısı

Abdominal Pain in a Patient With Nephrotic Syndrome

Kübra Kaynar[1], Özlen Bektaş[2], Semih Gül[1], Serdar Türkyılmaz[3], Murat Buran[2], Şükrü Ulusoy[1]

[1]Karadeniz Teknik Üniversitesi Tıp Fakültesi, Nefroloji BD, Trabzon

[2]Karadeniz Teknik Üniversitesi Tıp Fakültesi, İç Hastalıkları AD, Trabzon

[3]Karadeniz Teknik Üniversitesi Tıp Fakültesi, Genel Cerrahi AD, Trabzon

ÖZET

Amaç: Bu vaka ile, tedaviye dirençli fokal segmental glomerüloskleroz öyküsü bulunan ve ana şikâyeti, şiddetli karın ağrısı olan bir hasta tartışılmıştır. Nefrotik sendroma ikincil asiti olan 36 yaşındaki erkek hasta, asit dekompresyonu için sıvı ve tuz kısıtlaması ve diüretik kullanmakta iken, şiddetli karın ağrısı ile acil servise başvurdu. Hastada karın ağrısının inkarsere umbilikal herniye ikincil geliştiği saptanıp, herni içeriği redükte edildi. Bu vaka ile klinisyene, nefrotik sendroma ikincil gelişen asitin tıbbi tedavisi sırasında, umbilikal herni varlığının araştırılması ve nefrotik sendromu olan hastalarda karın ağrısı etiyolojisinde, umbilikal herni inkarserasyonunun hatırlatılması amaçlanmıştır.

Anahtar sözcükler: nefrotik sendrom, asit, umbilikal herni inkarserasyonu

ABSTRACT

Objectives: We aimed to report a case with a history of therapy-resistant focal and segmental glomerulosclerosis presenting with the main complaint of severe umbilical pain. A 36-year-old male patient with ascites secondary to nephrotic syndrome applied to emergency service with severe abdominal pain. Decompression of ascites achieved medically by fluid and salt restriction and diuretic administration had led to umbilical hernia incarceration in this patient. The content of the hernia was reducted. With this case, we alarm the clinician for searching presence of umbilical hernia in the medical management of ascites and thinking of incarcerated umbilical hernia for abdominal pain in patients with nephrotic syndrome.

Keywords: nephrotic syndrome, ascites, umbilical hernia incarceration

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Introduction

Umbilical hernias occur frequently among patients with cirrhosis and ascites. Although in most pediatric patients, ascites formation is probably a common manifestation of general fluid retention, in most adult patients with nephrotic syndrome, ascites can be probably attributed to both hypoalbuminemia and the presence of liver disease or congestive heart failure (1). There are also reports about ascites formation in 18% of the patients with biopsy proven focal segmental glomerulosclerosis (2).

Yazışma Adresi: Dr. Kübra Kaynar
Karadeniz Teknik Üniversitesi Tıp Fakültesi
Nefroloji BD, 61080, Trabzon
Tel : 0 (462) 377 54 83
Faks : 0 (462) 325 05 18
E-posta : kkaynar@yahoo.com

Here, we have presented a case with nephrotic syndrome secondary to therapy-resistant focal segmental glomerulosclerosis and incarcerated umbilical hernia due to ascites decompression. As far as we know, there is no reported case with incarcerated umbilical hernia secondary to nephrotic syndrome in the literature.

Case Report

A 36-year-old man (1.78 m in height and, 98 kg in weight) with a history of therapy-resistant focal and segmental glomerulosclerosis of a year duration presented with the main complaint of severe umbilical pain to the emergency service. Fourteen months ago he had swelling on his legs and face. Renal biopsy revealed focal segmental glomerulosclerosis in some other center outside the city. It was learned that the patient had taken three cour-

ses of cyclophosphamide pulses and methyl prednisolone therapy for 1 year. He also had taken the medications of furosemide, verapamil, essential amino acids, atorvastatin, lansaprazole, iron sucrose. There was no history of remission. His rate of renal function loss was 10 mL/min/month. While he applied to our department he was taking diuretics (furosemide 500 mg/day, spironolactone 100 mg/day) for medical treatment of his edamatous condition. The patient was noted to be mildly anemic with massive ascites. Physical examination revealed high-pitched bowel sounds and a tender umbilical hernia with reddening of the overlying skin (Figure 1). The secondary causes of focal segmental sclerosis were absent in the patient (no history of heroin abuse, morbid obesity or HIV positivity). Spot urinary total protein to creatinine ratio was 14. Biochemical analysis revealed total protein: 3.4 gr/dL, albumin: 1.4 gr/dL, total cholesterol: 512 mg/dL, HDL cholesterol: 69 mg/dL, LDL cholesterol: 362 mg/dL, triglyceride: 181 mg/dL. Complete blood count revealed Hb: 8.4gr/dL, WBC: $5.9X10^9$/L, plt was $361X10^9$/L. Glomerular filtration rate was 15 mL/min. The liver function tests of the patient were totally normal. ALT was 18 u/L, AST was 22 u/L. Doppler analysis revealed hepatic veins were normal and portal vein diameter was 12 mm, flow pattern was normal. Liver and spleen were normal ultrasonographically. TSH levels were 3.21 mu/L (Normal: 0.27-4.2) free T4 was 11.9 pmol/L and they were in normal range. Results of renal sonography revealed right kidney was 114 mm, with increased paranchymal echogenicity. Left kidney was 116 mm and paranchymal echogenicity was increased. The echocardiography was normal.

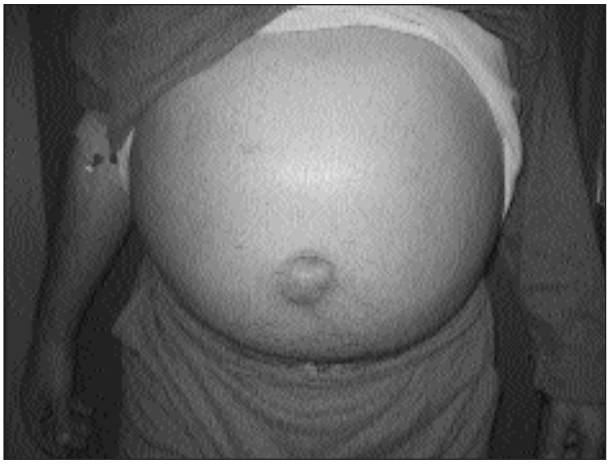

Figure 1. Incarcerated umbilical hernia.

Emergent reduction of the incarcerated hernia was done in this patient because physical examination revealed high pitched bowel sounds and there was no air fluid levels in the abdominal plain film. There was no bowel content in the umbilical sac ultrasonographically. The reduction of the hernia was successfull. In the follow up, he had pulmonary edema, he was overvolemic and resistant to maximum dose of diuretic therapy. After regular hemodialysis programme was started for him and dry weight was achieved, ascites had completely disappeared and the patient is planned to go for surgical repair of umbilical hernia.

Discussion

In most adult patients with nephrotic syndrome, ascites can be attributed to both hypoalbuminemia and the presence of liver disease or congestive heart failure, with increased hepatic sinusoidal pressure (1). So liver diseases and congestive heart failure were searched in the patient. There was no abnormality in echocardiography and liver function tests were also normal. Spleen and liver were in normal shape and size. Hepatitis markers were negative. Thyroid function tests were all normal. So the ascites of the patient was attributed solely to hypoalbuminemia secondary to nephrotic syndrome. It is known that ascites is one of the rare complications of the nephrotic syndrome. Agarwal SK et al reported that ascites were observed in 12 patients of a total of 65 adult cases with biopsy-proven focal segmental glomerulosclerosis (2).

Ascites formation is associated with an increase in intraabdominal pressure. As a result of elevated intraabdominal pressure, the peritoneum is thrust forward through the umbilical ring, leading to herniation of the skin envelope. Diuretics preventing renal sodium retention remain the cornerstone of the treatment of nephrotic edema. Accordingly, the association of amloride and furosemide provides a powerful treatment allowing progressive removal of edema from patients with nephrotic syndrome (3). However, decompression of ascites achieved medically by fluid and salt restriction and diuretic administration may lead to umbilical hernia incarceration treatment of which is often more complex (4). Incarceration of umbilical hernia was also reported in cirrhotic patients following diuresis, paracentesis, and peritoneovenous shunting (5).

Umbilical hernia is a consequence of incomplete closure or weakness at the umbilical ring, where protrusion of intraabdominal contents may occur. Umbilical hernias 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 (6). Umbilical hernia represents 6% of all abdominal wall hernias in the adult. Umbilical hernias are usually suggested by the patient's clinical history and confirmed by findings from a physical examination. The risk factors other then cirrhosis and nephrotic syndrome for umbilical hernia generation are maternal obesity, chromosomal abnormalities, Ewing's sarcoma family of tumors, CAPD and Cantrell's syndrome (7-12). No clues for these diseases were present for our patient.

Emergent reduction of the incarcerated hernia was done in this patient because physical examination revealed high pitched bowel sounds and there was no air fluid levels in the abdominal plain film. There was no bowel content in the umbilical sac ultrasonographically. The reduction of the hernia was successfull. This maneuver could have resulted in a disaster but emergent surgical repair of this clinical problem might have also led to a disaster because of massive ascites and severe hypoalbuminemia. Umbilical herniorrhaphy in the good risk patient is a relatively simple operation with low morbidity and mortality. Umbilical herniorrhaphy in patients with ascites presents an operative risk of much greater magnitude and may pose a considerable technical problem when complicated by leakage of ascitic fluid, necrosis of the abdominal wall with ulceration, infection, rupture or strangulation. The most important is to choose the right time for surgery before complications occur: strangulation or umbilical ruptures. In these cases (umbilical hernia with ascites), the rate of morbidity and mortality is high, and recurrences as well. That was the reason why we planned to repair the umbilical hernia of our patient surgically after ascites management.

In our case, a patient with therapy-resistant focal and segmental glomerulosclerosis presented with acute abdominal pain was discussed. Due to nephrotic syndrome, he had massive ascites. Consequently umbilical hernia developed secondary to massive ascites. As in this case, decompression of ascites and edema achieved medically by fluid and salt restriction and diuretic administration resulted in umbilical hernia incarceration. Decompression of ascitic fluid can result in umbilical hernia incarceration (4). So if a hernia is present in a patient with ascites, the patient should be made aware of the possibility of incarceration with medical therapy of the ascites and the hernia should be observed closely.

Discussion

With this case, we wanted to report a case of umbilical hernia incarceration secondary to ascites which is not a common complication of nephrotic syndrome. We wanted the clinician to be alarmed for incarcerated umbilical hernia for searching etiology of abdominal pain in patients with nephrotic syndrome and ascites besides other diseases like peritonitis, intussusception, amyloidosis.

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