Splenic rupture following idiopathic rupture of the urinary bladder presenting as acute abdomen

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ABSTRACT

Idiopathic rupture of the urinary bladder is an uncommon condition and represents less than 1% of all bladder rupture cases. In most of the cases the main etiological factor was heavy alcohol ingestion. A combined injury of the spleen and bladder is a very rare condition that is almost often associated with trauma and foreign bodies. In this paper we present the extremely rare clinical course of acute abdomen caused by a combined spontaneous intraperitoneal injury; spontaneous rupture of the urinary bladder and spleen. According to our opinion, spontaneous bladder rupture caused by bladder distension due to alcohol ingestion led to urinary ascites and abdominal distension. Finally, repeated minor abdominal blunt trauma during everyday life, to a moderately distended abdomen caused a spontaneous splenic rupture in the patient with abnormal coagulation studies.

KEY WORDS: Acute abdomen, splenic rupture, urinary ascites

Intraperitoneal injuries of the urinary bladder are most often associated with blunt abdominal injuries while extraperitoneal injuries usually occur with pelvic fractures. Less frequently, bladder rupture occurs spontaneously.[1,2] The incidence of spontaneous bladder rupture has been reported to be 1:126000; with an overall mortality of 47%.[3] Spontaneous rupture of the bladder includes pathologic ruptures and idiopathic ruptures which are without an obvious underlying pathology.

A combined injury of the spleen and bladder is a very rare condition that is usually associated with trauma and foreign bodies.[4,5] To our knowledge, splenic injury following idiopathic rupture of the bladder has not been reported to date.

In this paper we present an unusual acute abdominal clinical course in a male patient that was caused by spontaneous intraperitoneal rupture of the urinary bladder followed by splenic rupture.

Case History

A previously healthy 82-year-old man was admitted to our hospital with five days history of abdominal pain and bloating. He had nausea, vomiting and diarrhea only on the first day after onset of abdominal pain. The patient had no respiratory, cardiovascular or urinary symptoms and he gave no history dysuria and hematuria. He was not taking any medication and he denied any history of fall or abdominal trauma. He estimated his alcohol intake at approximately three liters of beer per day. Cardiothoracic examination showed bronchitic and basal wet pulmonary sounds. Abdominal examination revealed an adipose and significantly distended abdomen with generalized abdominal tenderness and signs of peritonitis. The most painful part of the abdomen was right paraumbilical and there were no external signs of trauma. Bowel sounds were absent. Digital rectal examination revealed a normal sized prostate and empty ampulla recti. The abdominal X-ray did not reveal any pathological signs except distended bowels, but X-ray of the chest showed free fluid in both phrenico-costal sinuses. An abdominal ultrasound showed a large volume of free peritoneal fluid, which seemed to be ascitic fluid because of the hypoechogenic liver suspected on cirrhosis. The kidneys appeared normal and without hydronephrosis. Abdominal CT scan was not carried out because of technical reasons. Laboratory analysis revealed a serum creatinine level of 7 mg/dl, (normal range, 0.8-1.2 mg/dl); bicarbonates 18.9 mmol/L, (normal range, 22-27.7 mmol/L), serum C-reactive protein was 158.9 mg/L, (normal range, <9 mg/L). Serum potassium, sodium levels and liver function tests were normal. White blood

cell count was 16.5×10^9/L, (normal range, $<10\times10^9$/L); while platelet and red blood cell count were normal. Coagulation studies demonstrated PT 58%, (normal range, 70-130%) and APTT 38 %, (normal range, 26-36%). Nasogastric tube yielded 1500 mL of fluid containing stool and bladder catheterization drained one liter of blood-stained urine. Because of laboratory findings and blood-stained urine, a renal physician was called to examine the patient. A diagnosis of prerenal failure probably caused by expansive process in urinary bladder was established. An emergency surgery operation was performed for peritonitis and acute abdomen. At laparatomy, several liters of blood-stained fluid were present in the abdominal cavity, with laceration of the distal part of the spleen about 6 cm long and covered with an old hematoma. Further exploration revealed a 3 cm large perforation at the intraperitoneal portion of the urinary bladder.

Splenectomy, two-layers suturing of the bladder perforation, biopsy of the bladder wall and insertion of bilateral uretheral and bladder catheters was performed. No pathology was found in the bladder wall on histological section. The procedure was well tolerated and postoperative recovery passed without complications, and he was discharged from hospital on the tenth postoperative day in good condition. Uretheral catheters were removed on the 14th day and bladder catheter was removed on the 21st postoperative day. Repeated cystography did not reveal any leakage of urine from the bladder.

Discussion

Spontaneous intraperitoneal rupture of the normal bladder usually presents as an acute abdominal emergency.[2,3,6] In a minority of cases first symptoms of bladder rupture could be painless, slowly increased abdominal distension due to urinary ascites. Because of peritoneal self-dialysis, serum levels of creatinine, urea and potassium will accumulate to toxic levels which, in combination with metabolic acidosis and hyponatremia, may lead to a wrong tentative diagnosis of acute renal failure.[7] The majority of patients with intraperitoneal bladder rupture need laparatomy to confirm the diagnosis and to repair the site of perforation, because constant leakage of the urine into the peritoneal cavity does not allow spontaneous healing of the perforation.[1-3,6,8]

In a majority of cases of intraperitoneal idiopathic ruptures, history of minor trauma of the abdomen following heavy alcohol intake were presented.[6,7,9] Alcohol intoxication increases the risk of bladder rupture by several mechanisms, including the large volume of urine produced by the diuretic effects of alcohol, and overdistention of the bladder with an impaired sensorium. Moreover, alcohol may aggravate preexistent prostatic hypertrophy directly, resulting in the exacerbation of bladder neck obstruction. Additionally, nausea or vomiting associated with alcohol abuse can increase intraabdominal pressure and minor trauma may be forgotten or may go unnoticed by the intoxicated patient. All these facts are associated with delay in diagnosis and management.

As suggested in literature, coagulopathy in uremic patients may be an important risk factor related to spleen rupture and subcapsular hematomas.[10]

Our patient came five days after onset of abdominal pain and bloating and he probably failed to recall a possible minor abdominal trauma after alcohol intake. Location of bladder perforation was in the typically weakest, intraperitoneal portion. We believe that additional splenic rupture was probably caused by repeated minor abdominal blunt trauma during everyday life (including coughing) to the severe distended abdomen due to urinary ascites in patients with coagulopathy.

This case report shows the possibility of spontaneous spleen injury after intraperitoneal bladder rupture, especially in patients with significantly distended abdomen and abnormal coagulation studies.

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