Spontaneous Splenic Rupture: A Rare Complication of Chronic Pancreatitis

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Abstract

We describe a case of spontaneous splenic rupture in a patient with chronic pancreatitis and discuss the pathogenesis and diagnosis. Splenic rupture is a very rare complication of chronic pancreatitis. Anemia and hypovolemic shock may not be present, making the diagnosis difficult. A high index of suspicion combined with adequate evaluation and imaging studies are critical for correct diagnosis and prompt treatment of this potentially lethal condition.

MeSH Words: Spontaneous splenic rupture, chronic pancreatitis, complications

Introduction

We describe a rare case of life-threatening spontaneous splenic rupture complicating chronic pancreatitis. Our findings highlight the importance of a high index of suspicion in these cases and prompt, adequate evaluation for diagnosis.

Case report

A 45-year-old man presented to the Emergency Department (ED) complaining of severe left upper quadrant abdominal pain radiating to the left lumbar area of 3 days' duration. His past medical history was remarkable for cholelithiasis that had been treated 4 years previously with laparoscopic cholecystectomy, in addition to peptic ulcer and "pancreatic cysts" with elevated pancreatic enzyme levels. The patient had already scheduled an appointment with a gastroenterologist. He admitted occasional alcohol use but denied alcohol abuse. He also denied any kind of trauma.

On physical examination, vital signs were normal. The abdomen was soft and nondistended and tender to palpation in the left upper quadrant and left costovertebral angle, without spinal tenderness. Findings on blood tests (whole blood count), liver function tests and urinalysis were normal or negative. Blood amylase level measured 154 U/L (normal 30-110 U/L). Abdominal plain x-ray film showed...
calcifications projecting onto the pancreatic tail. The spleen shadow was normal. Treatment with intravenous fluids and parenteral diclofenac led to an improvement in the patient's condition. The pain subsided, and he and was discharged home with a referral to a gastroenterologist.

Ten hours later the patient returned to the ED because of recurrent pain. On physical examination, temperature was 36.3°C per os, pulse rate 100 beats/minute, blood pressure 130/83 mmHg, and O₂ saturation 97% in room air.

The abdomen was mildly distended, with generalized tenderness on palpation, especially in the left side. Laboratory values, including amylase levels, were within normal limits. Plain abdominal x-ray film revealed a clear enlargement of the splenic shadow compared with the film made the night before and an elevated left hemidiaphragm (Fig.1). Ultrasonography scan showed an enlarged spleen and hyperechoic multilayered areas within the spleen consistent with hematoma or bleeding, in addition to free fluid in the peritoneal cavity (Fig. 2).

Emergent computed tomography (CT) angiography was performed to confirm the diagnosis of ruptured spleen and to delineate the celiac axis and splenic vessels. The CT scan showed an enlarged spleen with a nonhomogeneous multilayered appearance, a pseudocyst measuring 32 mm in diameter in the pancreatic body, calcifications in the pancreatic tail and head consistent with chronic pancreatitis, a large volume of free fluid in the abdominal cavity, and a small left pleural effusion (Fig. 3).

The splenic artery and vein were detected and appeared normal.

Fig. 1. Plain abdominal x-ray in a patient with splenic rupture complicating chronic pancreatitis. Note enlarged splenic shadow and elevated left hemidiaphragm.

Fig. 2. Ultrasound scan in the same patient showing an enlarged spleen containing hyperechoic multilayered areas consistent with hematoma or bleeding.

Fig. 3. CT scan showing an enlarged spleen with a nonhomogeneous multilayered appearance, a pseudocyst in the pancreatic body, calcifications in the pancreatic tail and head, and a large volume of free fluid in the abdominal cavity.

The patient had mild tachycardia (100/minute) but was otherwise hemodynamically stable. Hemoglobin level was 12.3 gr/dl (13.9 gr/dl the night before).

The patient underwent emergent laparotomy. A ruptured spleen and a large hemoperitoneum were found, and splenectomy was performed. The postoperative course was uneventful although the patient required a blood transfusion.
Pathological study showed a normal spleen size (11.5x7.5x3.8 cm) and linear tears in the spleen tissue up to 2.5 cm long and 1.2 cm deep. Microscopic examination showed linear tears in the splenic tissue with recent hemorrhages. The spleen parenchyma had no abnormalities. As the patient had denied any trauma in the recent past, we assumed the rupture was spontaneous.

**Discussion**

Chronic pancreatitis has been well defined histopathologically, but for clinical purposes, the diagnosis is not generally based on histological findings. Diagnostic CT has a reported sensitivity of 74-90% and a specificity of 85%. It may reveal calcifications and cystic areas not noted on ultrasound scan or plain films [1].

Except for splenomegaly due to splenic vein thrombosis, other splenic complications rarely occur during the course of chronic pancreatitis, with a reported incidence of 2.2% [2]. They include splenic necrosis, intrasplenic pseudocysts, and spontaneous splenic rupture.

Splenic rupture should be considered “spontaneous” only if it occurs in the absence of trauma in a patient free of diseases that could involve the spleen [3]. True spontaneous rupture is extremely rare, but it is associated with high morbidity and mortality [2]. The spontaneous rupture of a diseased spleen has been reported more often, though it is also rare [3]. The diseases that most often induce spontaneous splenic rupture are hematological and other malignancies [4], some infectious diseases (mononucleosis, malaria), and acute or chronic pancreatitis. Other causes are hamartoma, hemangioma, cysts, autoimmune diseases, hemolytic anemias, pregnancy, amyloidosis, and portal hypertension [4]. Some authors suggested that spontaneous splenic rupture associated with disease should be categorized as "pathological" rupture [3]. However, in most reports, splenic rupture is called spontaneous whether or not the rupture is pathologic.

Three hypotheses have been proposed to explain the pathogenesis of spontaneous splenic rupture associated with chronic pancreatitis. The most likely mechanism in most cases is thrombosis of the splenic vein with portal hypertension. An enzymatic mechanism may also be responsible, especially in cases of pseudocysts. Another possible source of bleeding is a splenic artery pseudoaneurysm eroded by the contents of the pseudocysts. The mechanical hypothesis claims that perisplenitis secondary to chronic pancreatitis fixes the spleen, making it more vulnerable to rupture even after minimal trauma [5].

In our case, findings on CT angiography and pathological spleen study were not compatible with any of the proposed mechanisms other than pseudocyst and pancreatic calcifications. Given that the patient denied any trauma, we assumed the rupture had occurred spontaneously.

Because of the rarity of spontaneous splenic rupture, its diagnosis may be missed and treatment delayed, leading to an increase in morbidity and mortality. Splenic complications should be considered in the differential diagnosis of pain in the left abdominal upper quadrant, especially if the patient is hemodynamically unstable. Anemia and hemorrhagic shock may not occur at presentation, as in our case, making the diagnosis even more difficult. Ultrasonography and especially CT may assist clinicians in identifying pathological splenic rupture and in seeking risk factors, such as pseudocysts, splenic vein thrombosis, and intrasplenic collection [6].

Bleeding from a lacerated or ruptured spleen does not cease spontaneously and requires immediate surgical attention. Once the diagnosis of splenic rupture is established, splenectomy with or without distal pancreatectomy seems to be the appropriate mode of treatment. Embolization of the splenic artery, if possible, can stop the bleeding in selected cases [7].

**References**


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