Abdom Imaging 26:290–293 (2001)
DOI: 10.1007/s002610000158

Abdominal Imaging

Spontaneous rupture of the spleen: report of two cases

P. Torricelli,[1] **C. Coriani,**[1] **M. Marchetti,**[1] **A. Rossi,**[2] **A. Manenti**[2]

[1]Department of Radiology, University of Modena and Reggio Emilia, Policlinico, Via del Pozzo 71, 41100, Modena, Italy
[2]Department of Surgery, University of Modena and Reggio Emilia, Policlinico, Via del Pozzo 71, 41100, Modena, Italy

Received: 10 August 2000/Accepted: 20 September 2000

Abstract
Spontaneous splenic rupture is rare and in most cases occurs in a spleen affected by hematologic, neoplastic, or infectious disease or as a complication of pancreatitis. We report the imaging findings in two cases of spontaneous splenic rupture due to non-Hodgkin's lymphoma and pancreatic pseudocyst.

Key words: Spleen, rupture—non-Hodgkin's lymphoma—Pancreatitis—Computed tomography.

Spontaneous rupture of the spleen is an uncommon, dramatic, abdominal emergency that requires immediate diagnosis and prompt surgical treatment to ensure the patient's survival. Spontaneous rupture rarely occurs in a histologically proven normal spleen and, in such cases, is called a "true spontaneus rupture." Spontaneous rupture usually occurs in a diseased spleen and is called a "pathologic spontaneous rupture" [1]. Oncologic-hematologic diseases [2], infectious diseases [3, 4], and chronic and acute pancreatitis [5–7] are the most common causes of spontaneous pathologic rupture.

We report two cases of spontaneous rupture in patients with non-Hodgkin's lymphoma and pancreatic pseudocyst.

Case reports

Case 1

A 51-year-old caucasian male was admitted to our Medical Department with mild weight loss, fever, asthenia, and abdominal pain. A mild hypochromic anemia and increased erythrocytes sedimentation ratio were found; coagulation tests were normal. Abdominal ultrasonography (Fig. 1A) showed an enlarged spleen with multiple, solid, hypoechoic, rounded lesions up to 3 cm in diameter. Multiple enlarged lymph nodes, both intraabdominally and retroperitoneally, were also found. The liver, pancreas, kidneys, and adrenal glands were normal.

On the third day after admission, the patient complained of a sudden, severe pain in the left hypochondrium associated with a sudden drop in hemoglobin. Computed tomography (CT) showed a massive hemoperitoneum with tears on the splenic surface (Fig. 1B–C). The splenic parenchyma was completely disrupted by low- and high-density areas of attenuation. Laparotomy showed a massive hemoperitoneum and an enlarged spleen with many surface nodules, one of which, lying on the diaphragmatic surface, was torn and bleeding profusely. Splenectomy was performed and the histologic diagnosis was large B-cell, Burkitt-like non-Hodgkin's lymphoma of the spleen, mostly localized to the white pulp.

Postoperative course was normal. Three weeks afterward, the patient underwent antitumoral chemotherapy, with complete remission of the tumor. Sixteen months later, he is well, with no sign of tumor recurrence.

Case 2

A 67-year-old caucasian male had been admitted many times over the previous 3 years to the Surgical Department for recurrent episodes of ethanol-related pancreatitis. During the last admission, a large pseudocyst involving the body and tail of the pancreas was demonstrated on CT (Fig. 2A). The pseudocyst was close to the surface of the spleen, whose contour was deformed by a deep indentation. A few days after the last discharge, the patient was admitted to Emergency Department with sudden

Correspondence to: P. Torricelli

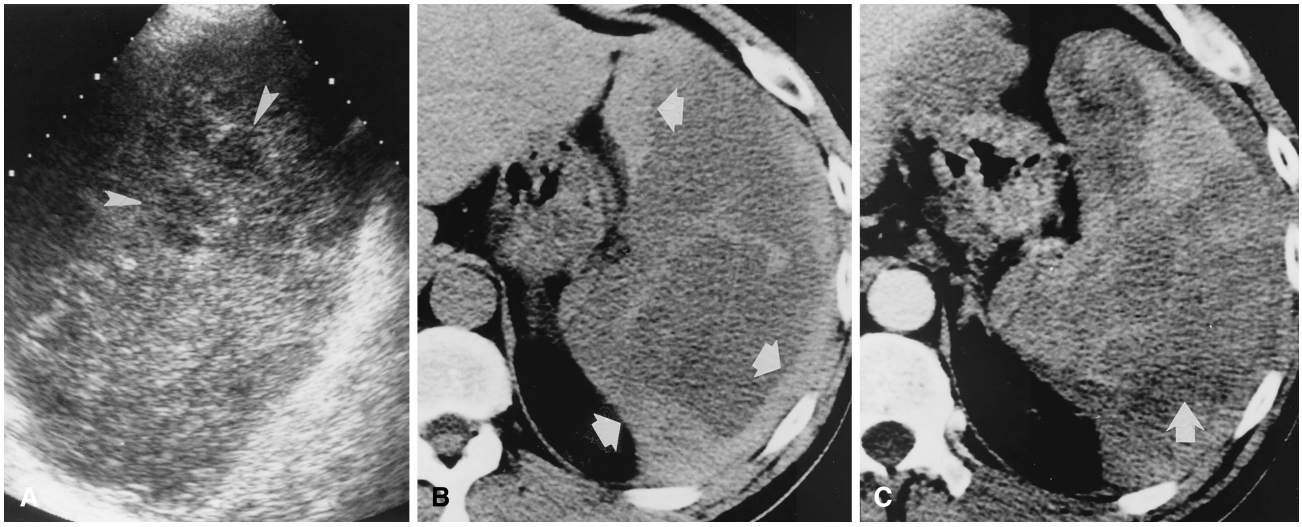

Fig. 1. Case 1. **A** Ultrasonography shows an enlarged spleen with many solid hypoechogenic nodular lesions (*arrowheads*). **B** Precontrast CT shows the spleen surrounded by intraperitoneal hemorrhagic fluid, with high attenuation values (*arrows*). No clear lesions of splenic parenchyma are visible. **C** Postcontrast CT shows most of the splenic parenchyma infiltrated by solid low-density nodular lesions (*arrow*); the posterior surface of the spleen is disrupted.

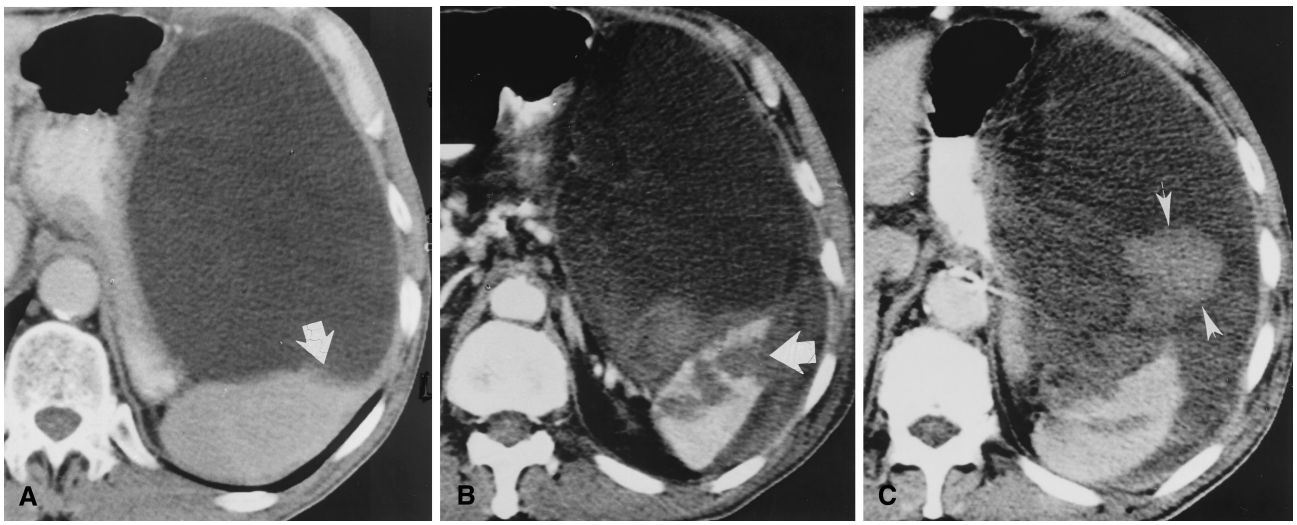

Fig. 2. Case 2. **A** Contrast-enhanced CT before the spleen rupture. A huge cystic mass arising from the pancreatic tail, with homogeneous fluid content, occupies the left hypochondrium. The mass dislocates the stomach and shows a close relation with the splenic surface, whose border is deformed by a deep indentation (*arrow*). **B, C** Contrast-enhanced CT at the time of the rupture. A large tear of the splenic surface is clearly demonstrated (*arrow*). The pseudocyst is now filled with high-density material (*arrowheads*) due to intracystic hemorrhage. Massive hemoperitoneum is also visible.

onset of pain in the left hypocondrium with clinical and laboratory signs of acute blood loss.

Abdominal CT showed massive hemoperitoneum and large tears of the splenic surface (Fig. 2B–C). The pseudocyst contained hyperdense fluid due to intracystic bleeding arising from the ruptured spleen.

Laparotomy showed a massive hemoperitoneum with both fresh and clotted blood. The pancreatic pseudocyst was filled with blood, and the spleen was pushing into its cavity, with a large capsular tear and acute bleeding. Many perisplenic adhesions were also found. Splenectomy was performed, and anatomic evaluation showed the splenic tissue to be normal. The postoperative course was uneventful, and the patient was discharged 2 weeks later. Two months later, CT-guided drainage of the pancreatic pseudocyst was successfully performed.

Discussion

According to the criteria described by Orloff and Peskin in 1958 [1], splenic rupture should be considered "spontaneous" only if it occurs in the absence of trauma in a spleen unaffected by intra- and perisplenic diseases and in patients free of diseases that could involve the spleen. True spontaneous rupture of the spleen is extremely rare. There have been very few cases reported in the literature [1, 4, 8, 9].

The spontaneous rupture of a diseased spleen, even if rare, is more frequent and, according to the criteria of Orloff and Peskin, should be categorized as a pathologic rupture in the same way as a pathologic fracture of the bone. Diseases that more commonly may induce the spontaneous rupture of the spleen are the oncologic and hematologic diseases [2, 12], some infectious diseases such as infectious mononucleosis and malaria [3, 4], and acute or chronic pancreatitis [5–7]. Other rare causes of splenic rupture include some congenital splenic lesions as hamartoma, hemangioma, and cysts and a miscellaneous group of diseases such as autoimmune diseases, hemolytic anemias, pregnancy, amyloidosis, and portal hypertension [1, 4, 8, 10, 11].

However, in most reports in the current literature, splenic rupture is commonly categorized as spontaneous when it occurs without trauma, whether or not the rupture is pathologic. Oncologic and hematologic diseases often cause spontaneous rupture of the spleen; in a recent review of the literature, Giagounidis et al. [2] found 136 cases reported between 1861 and 1999. Non-Hodgkin's lymphoma is the most frequently reported cause of splenic rupture followed by, in order of frequency, acute myeloid leukemia, chronic myeloid leukemia, and lymphoblastic acute leukemia.

There are three main pathogenetic factors that may explain the rupture [2]. First, and most important, the congestion of the splenic parenchyma by blast cells; second, coagulation disorders leading to intrasplenic and subcapsular hemorrage, and third, splenic infarction.

Our first case occurred in a patient affected with non-Hodgkin's lymphoma. Here, the infiltration of the splenic parenchyma by blast cells, previously shown by abdominal ultrasonography, was surely the main cause of the rupture because the coagulation tests of the patient were normal and no clear splenic infarcts had been demonstrated. Moreover, at surgery, rupture and bleeding of a splenic nodule lying on the diaphragmatic surface of the spleen was found. The splenic rupture occurred before the start of antitumoral chemotherapy, which may have released lytic and vasoactive enzymes due to chemotherapy-induced cell necrosis. Giagounidis et al. reported that approximately 18% of the 136 spontaneous ruptures due to oncologic diseases occurred a few hours before beginning antitumoral chemotherapy. In our case, the clinical onset of symptoms was quite typical and the splenic lesions were already known. This made the CT diagnosis easy, based mainly on the hemoperitoneum and the huge infiltration of the splenic parenchyma. However, in some cases, the CT diagnosis of pathologic splenic rupture may be difficult because the infarction of the spleen may obscure the splenic lesions that are then found at surgical or anatomic evaluation.

Splenic involvement in pancreatitis is not common and has been reported in 1–5% of cases of pancreatitis [5–7]. This association may be explained by the close anatomic relation between the spleen and the pancreas and by the anatomic manner a pancreatic disease can spread to the spleen, such as the splenorenal ligament, which contains splenic vessels.

Splenic damage may be produced through three mechanisms: (a) encasement of splenic vessels, causing infarction or pseudoaneurysms; (b) intrasplenic dissection of pancreatic fluid or a pseudocyst; and (c) the damage of small intrasplenic vessels by pancreatic enzymes that cause intrasplenic or subcapsular hematoma [5]. Splenic rupture due to intrasplenic dissection of a pancreatic pseudocyst may be facilitated by even minimal or mild trauma and may be facilitated by perisplenic adhesions due to recurrent pancreatitis. The splenic bleeding may be intracystic or intraperitoneal, and in both cases it can be easily detected by CT, which shows hyperdense intracystic fluid or intracystic fluid–fluid levels [5, 13].

Although the CT signs of splenic rupture have been widely described, less attention has been given in the literature to signs that may help in predicting splenic rupture. In our case, we emphasize the finding of indentation and deformation of the spleen by the pseudocyst that was observed in the CT scans performed some days before the rupture. Even though we do not have a close surgical or anatomic correlation of this finding, we postulate that it may represent the sign of the initial intrasplenic dissection that was subsequently found at surgery at the time of the rupture. At that time, CT clearly showed both the splenic tears and the signs of intracystic and peritoneal bleeding, even though no fluid–fluid levels were found inside the pseudocyst.

Some investigators [6] have stated that splenic lesions during pancreatitis, including subcapsular and perisplenic fluids, can spontaneously heal without surgery. Conversely, we and others [5] believe that surgery is mandatory in the case of splenic rupture.

In conclusion, the reported cases are an additional contribution to the sparse literature on the spontaneous rupture of the spleen due to oncologic and hematologic disorders and pancreatic pseudocyst. We confirm the established CT signs of rupture [13] and emphasize a CT finding that may be useful for predicting rupture, even if additional, more consistent cases are needed to confirm its utility. Moreover, we suggest that patients with oncologic or hematologic diseases and with splenic tumor–related lesions should be monitored carefully, in particular at the

beginning of chemotherapy, for signs of pathologic spontaneous rupture of the spleen.

References

1. Orloff M, Peskin GW. Spontaneous rupture of the normal spleen, a surgical enigma. *Surg Gynecol Obstet* 1958;106:1–11
2. Giagounidis AAN, Burk M, Meckenstock G, et al. Pathologic rupture of the spleen in hematologic malignancies: two additional cases. *Ann Hematol* 1996;73:297–302
3. Barrier JH, Bani-Sadr F, Gaillard F, et al. Spontaneous rupture of the spleen revealing primary human immunodeficiency virus infection. *Clin Infect Dis* 1997;25:336–337
4. Tu AS, Tran MHT, Larsen CR. Spontaneous splenic rupure: report of five cases and a review of the literature. *Emerg Radiol* 1997;4: 415–418
5. Fishman EK, Soyer P, Bliss DF, et al. Splenic involvement in pancreatitis: spectrum of CT findings. *AJR* 1995;164:631–635
6. Rypens F, Devière J, Zalkman M, et al. Splenic parenchymal complications of pancreatitis: CT findings and natural history. *J Comput Assist Tomogr* 1997;21:89–93
7. Toussi HR, Cross KS, Sheean SJ, et al. Spontaneous splenic rupture: a rare complication of acute pancreatitis. *Br J Surg* 1996;83:632
8. Denehy T, Mcgrath EW, Breen JL. Splenic torsion and rupture in pregnancy. *Obstet Gynecol Surg* 1988;43:123–131
9. Klinkert P, Kluit AB, de Vries AC, et al. Spontaneous rupture of the spleen: role of ultrasound in diagnosis, treatment and monitoring. *Eur J Surg* 1999;165:712–713
10. Bhagrath R, Bearn P, Sanusi FA, et al. Postpartum rupture of the spleen. *Br J Obstet Gynecol* 1993;100:954–955
11. Yoshizawa J, Mizuno R, Yoshida T, et al. Spontaneous rupture of splenic hamartoma: a case report. *J Pediatr Surg* 1999;34:498–499
12. Sugahara K, Togashi H, Aoki M, et al. Spontaneous splenic rupture in a patient with large hepatocellular carcinoma. *Am J Gastroenterol* 1999;94:276–278
13. Rabushka LS, Kawashima A, Fishman EK. Imaging of the spleen: CT with supplemental MR examination. *RadioGraphics* 1994;14: 307–332