Delayed diagnosis of splenic rupture following minor trauma: Beware of comorbid conditions

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ABSTRACT

Although rare, spontaneous splenic rupture has been reported in cases of chronic lymphocytic leukemia (CLL). In contrast, there are no published reports of splenic injury resulting from minor trauma in CLL patients. We report the case of an 81-year-old woman with a history of CLL, who presented with minor trauma to her coccygeal region, and was found to have a splenic rupture. We briefly describe the incidence, pathogenesis, presentation, diagnosis, management and prognosis of splenic injury, especially in cases with a history of splenomegaly.

Key words: splenic rupture; chronic lymphocytic leukemia

RÉSUMÉ

Bien que rares, des ruptures spontanées de la rate ont été signalées dans des cas de leucémie lymphocytaire chronique (LLC). Par contre, il n'existe aucun rapport publié de blessure à la rate résultant d'un traumatisme mineur chez les patients atteints de LLC. Nous décrivons le cas d'une femme âgée de 81 ans ayant des antécédents de LLC qui s'est présentée pour un traumatisme mineur à la région coccygienne et chez qui l'on constata une rupture de la rate. Nous exposons brièvement l'incidence, la pathogenèse, la présentation, le diagnostic, la prise en charge et le pronostic de la blessure à la rate, en particulier dans les cas présentant des antécédents de splénomégalie.

Introduction

Splenic rupture is generally associated with direct traumatic injury to the abdomen. However, if no source of trauma is apparent then the splenic rupture is deemed to be spontaneous in nature. Spontaneous splenic rupture is a rare but potentially lethal event.^{1,2} Therefore, non-traumatic rupture should lead one to consider some pathology that may be contributing to enlargement or fragility of the spleen.

Spontaneous rupture has been reported in several conditions, including infectious mononucleosis, HIV, hepatitis A and cytomegalovirus.² Traumatic splenic rupture has also been associated with metabolic and hematological conditional conditions.

tions such as amyloidosis, Wilson's disease, idiopathic thrombocytopenic purpura, leukemia, lymphoma, and multiple myeloma.³ Globally, malaria is the most common infectious cause of spontaneous splenic rupture.^{2,4} Drugs associated with atraumatic splenic rupture include heparin, warfarin and streptokinase.³ Pregnancy, chronic pancreatitis and extra-corporeal shockwave lithotripsy have also been associated with atraumatic splenic rupture.³

Spontaneous rupture of the spleen associated with CLL has also been reported in the literature. 5.6 However, there are no reports of splenic injury secondary to minor trauma in CLL patients. This report describes one case of a seemingly trivial injury that lead to splenic laceration in a CLL patient.

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Case report

An 81-year-old woman was brought to the emergency department (ED) after slipping off her bed from a sitting position with legs dangling, and falling onto her buttocks. She reported discomfort in the gluteal region and the midscapular region after the fall, but was able to walk normally. The patient denied any head trauma and there was no report of loss of consciousness or dizziness.

Her past medical and surgical history included CLL, appendectomy, choledocholithiasis, dyspepsia, cataract surgery and hyperthyroidism. Her CLL was of indolent type and had been managed for the past 12 years primarily with steroids. The medical history was obtained directly from the patient and from her previous medical records. She denied any allergies, and her regular medications consisted of omeprazole, prednisone, methimazole, disodium etidronate, lubricating eye drops and calcium supplements.

Initial vital signs revealed an alert and oriented lady, with a blood pressure of 142/79 mm Hg, pulse of 79 beats/min, respiratory rate of 16 breaths/min, oxygen saturation of 94% on room air and a normal body temperature. Cardiovascular and pulmonary exams were unremarkable. The abdominal exam revealed an obese person with no evidence of organomegaly or localized tenderness, and was otherwise unremarkable. The remainder of the examination was normal.

The patient's complete blood count revealed a hemoglobin level of 131 g/L, platelet count of 182×10^9 /L, and a leukocyte count of 145.6×10^9 /L. The lymphocyte count was 132.5×10^9 /L, while the monocyte count was 4.4×10^9 /L. Her last known hemoglobin level, taken 18 months earlier, was 149 g/L. Additional investigations included urinalysis, electrolyte, glucose and coagulation profiles, troponins, amylase, and liver, renal and thyroid function tests, all reported as normal. Radiographs of the lumbrosacral spine revealed only degenerative changes with no new fractures. The patient was considered to have a musculoskeletal contusion. She was managed with acetaminophen and discharged from the ED with a prescription for acetaminophen and codeine.

Several hours later, she returned to the ED after experiencing a brief episode of dizziness and abdominal discomfort, which occurred shortly after arising from her chair. The physical exam was unchanged except for mild epigastric tenderness. Investigations included a normal chest x-ray and CT of the head.

The patient was observed in the ED for an additional 12 hours overnight but she continued to complain of epigastric pain and distension despite receiving analgesia and

therapy for suspected dyspepsia. Physical examination revealed decreased bowel sounds and increasing abdominal distension. Cullen's sign, a bluish tinge around the umbilicus, was noted as well as significant pain and rebound tenderness. The clinical impression was hemoperitoneum, and the patient's hemoglobin had fallen to 96 g/L. An urgent surgical evaluation was requested, and a CT of the abdomen was ordered.

The abdominal CT revealed intrasplenic hematoma with splenic laceration and surface splenic disruption associated with mild to moderate amounts of hemoperitoneum and splenic enlargement. Shortly thereafter, the patient underwent emergency splenectomy, during which 2L of blood was suctioned from the peritoneal cavity. At this time, 2 tears in the splenic pulp on the posterior aspect near the hilum were noted. Formal pathology assessment revealed a grossly enlarged (650 g) beefy red spleen with multiple areas of hematoma. Histology revealed diffuse infiltration of the red pulp by B cells — a picture consistent with splenic CLL.

The patient experienced postoperative difficulties with atelectasis-related hypoxia and episodes of supraventricular tachycardia, both of which were successfully managed. She was vaccinated against meningococcus and hemophilus influenza type B, and started on long-term prophylactic amoxicillin (250 mg po bid) as well as weekly erythropoetin injections. The patient was discharged home on post-operative day 8 in stable condition.

Discussion

Spontaneous splenic rupture is a rare, life-threatening, but well recognized complication of hematological malignancies. Acute leukemias account for 40% of all cases of splenic rupture associated with hematologic malignancies.⁶ Spontaneous splenic rupture is also more common in males, however the reasons are not yet known.⁶ Spontaneous splenic rupture is more common in patients with acute leukemia than in those with chronic leukemia.⁷

Normal spleen weight decreases with age and is usually less than 250 g.8 As defined above, spontaneous splenic rupture is not a result of trauma. Because our patient presented with a history of CLL with splenomegaly, she may have suffered a spontaneous splenic rupture. However, with a history of fall, we believe that this presentation was not spontaneous but traumatic in nature and that the underlying pathology contributed to it.

The rib cage offers protection to the spleen against trauma. In settings of splenic pathology, the spleen is more prone to rupture not only due to the altered consistency but also due to the splenomegaly that extends the spleen below the rib cage.⁵

Abdominal pain is the most common symptom of splenic rupture.⁶ The character of the pain can be variable, from localized left upper-quadrant pain to left-sided chest pain to generalized abdominal pain. Patients with splenic injuries may also develop nausea and vomiting, syncope, abdominal distension, hypotension, tachycardia, peritoneal irritation, fever, anemia and Kehr's sign (pain at the left shoulder due to diaphragmatic irritation).^{6,7}

A diagnosis of splenic injury must be considered in all patients with hematologic malignancies, even in the absence of trauma. Presenting signs of splenic injury in patients with hematologic malignancies may differ from those with normal spleens. Bauer and colleagues report the presence of Kehr's sign in 17% of patients with hematologic malignancies compared with 65% of patients with a rupture of a previously normal spleen.⁶

Splenic rupture may be managed medically or surgically. In settings where the rupture is related to leukemic infiltration, chemotherapy or radiotherapy may preclude the need for surgery; however, Bauer and colleagues described 53 cases of malignancy-related splenic rupture, noting a 78% survival rate in patients who underwent surgery compared with 38% overall. These authors recommend emergent splenectomy for patients with malignancy-related splenic rupture.

Conclusion

In patients with illnesses known to cause splenomegaly, splenic injury can occur even after trivial trauma. A low threshold for imaging and a more extensive work-up for intra-abdominal injuries is warranted in such patients. If hemoperitoneum or other intra-abdominal trauma is suspected, then imaging is necessary unless immediate laparotomy is planned.

Competing interests: None declared.

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