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Torsion of a wandering spleen: a case report with emphasis on image findings

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Torsion of a wandering spleen: a case report with emphasis on image findings

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INTRODUCTION

The spleen measures roughly 11 cm (4.3 inches) in length in healthy adult people. It typically weighs between 150 and 200 g (5.3–7.1 oz) and is located in front of the 9th to 12th thoracic ribs [1].

The peritoneum surrounds the spleen, which is suspended by several ligaments, as follows [2]:

1. The gastroplenic ligament: this ligament connects the splenic hilum to the stomach’s greater curvature; it contains small gastric vessels as well as related lymphatics and sympathetic nerves.
2. The lienorenal ligament: this ligament connects the spleen hilum to the anterior surface of the left kidney; it contains the pancreas tail and splenic arteries.
3. The phrenicocolic ligament: this ligament is a horizontal peritoneal fold that runs from the colon’s splenic flexure to the diaphragm at the mid-axillary line; it forms the upper end of the left Para-colic gutter.

Laxity of the splenic ligaments may cause excessive mobility of the spleen, allowing it access to the lower quadrant of the abdomen (wandering spleen) (Fig. 1) [3].

Wandering spleen predisposes the spleen to torsion, impaired blood flow, and ischemia, and can manifest itself in a variety of ways, ranging from mild intermittent abdominal pain to an abrupt abdominal crisis. The lack of distinct signs and symptoms, combined with the condition’s rarity, precludes clinical diagnosis [4].

Case presentation

We present a case of splenic torsion in a 26-year-old female patient. She was admitted to our hospital with sudden onset generalized abdominal pain of 5 days, localized mainly in the left upper quadrant with a previous history of a similar attack two months ago.

On examination, she was febrile (37°C) with a normal heart rate (85 beats per minute) and blood pressure (110/70 mm Hg). Physical examination revealed generalized abdominal tenderness with palpable tender spleen reaching the lower abdomen.

Laboratory studies showed leukocytosis (17 × 10³/µl), normocytic normochromic anemia (8.2 g/dl), and platelets count (144 × 10³/µl). Also, blood film was done and showed toxic granulation of neutrophils, absolute lymphopenia, and red blood cells (RBCs) showed anisocytosis and poikilocytosis with reticulocyte count 1%.

Pelvi-abdominal ultrasound (U/S) showed an enlarged spleen till the pelvis, homogenous with scattered calcification and mild pelvic collection. Doppler Sonography on portal circulation and splenic vessels demonstrated patency of portal vein with normal velocity 15 cm/s with no signs of obstruction in it or its branches, and splenic vessels also showed preserved blood flow with velocity in splenic vein 32 cm/s.

Enhanced computed tomography demonstrated markedly enlarged spleen and atrophied right kidney with compensatory hypertrophied left kidney (Fig. 2a, b).

The patient’s past history included previous attacks of abdominal pain responded to medical treatment in the form of analgesics and many laboratory studies exclude medical causes of splenomegaly (lactate dehydrogenase (LDH), titer for brucella, and hepatitis markers).

Because of the large size of the spleen and liability to trauma with recurrent attacks of abdominal pain, the patient was referred to splenectomy.

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Exploratory laparotomy through the left Kocher’s incision was performed.

The spleen was enlarged with no definite peritoneal attachments except for adhesive bands around the spleen, and the vascular pedicle was twisted 360° anticlockwise, causing venous outflow obstruction and thrombosis; however, the splenic artery was patent and pulsatile. The tail of the pancreas was not involved. Splenectomy was performed with ligatures placed on the splenic vein and artery separately (Fig. 3a, b).

During the postoperative course, blood tests revealed only one abnormality related to gradual elevation of platelets count up to $1350 \times 10^3$ on day 3.

Precautions were taken early postoperative in the form of good intravenous hydration, a prophylactic dose of enoxaparin sodium 40 mg (4000 IU) once daily, oral salicylate therapy 75 mg per day, and compression stocking thrombo embolus deterrent (TEDS).

Unfortunately, on day five postoperative, the patient developed extensive deep venous thrombosis, reducing the platelet count to $670 \times 10^3$. Thrombosis involved the inferior vena cava (IVC) up to the level of renal veins and we had to use an IVC filter to avoid attacks of pulmonary embolism with anticoagulant therapy.

Histopathology evaluations showed severe congestion of the spleen with extensive hemorrhagic infarction (Fig. 4a), congested hilar lymph nodes with focal hemorrhage (Fig. 4b), and hilar vessels filled with blood clots (Fig. 4c).

**DISCUSSION**

The spleen is typically held in a relatively fixed position by the suspensory ligaments and by the pressure exerted by surrounding organs and musculature. The suspensory ligaments derive from the dorsal mesogastrium. Incomplete fusion of mesogastrium or improper fixation results in an abnormally mobile spleen with a long vascular pedicle and the development of a wandering spleen [5].

Because of its rarity, wandering spleen is rarely diagnosed clinically (<0.2%). It has a female predominance [6].

The clinical presentation varies considerably, ranging from asymptomatic intermittent pain and discomfort to an abrupt abdominal crisis. Splenomegaly as a result of venous stasis and congestion, and splenic vein thrombosis as a result of reduced arterial supply resulting in splenic infarction and necrosis are the major complications of splenic torsion. Although laboratory tests are frequently nonspecific, they may indicate elevated inflammatory markers and signs of hypersplenism [7].

Reactive thrombocytosis after splenectomy occurs at a rate of ~75%–82%. Thrombosis associated with an elevated platelet
count after splenectomy is a well-known complication, with a frequency of roughly 5% [8].

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Conflicts of interest
There are no conflicts of interest.

References