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Wandering hemi-spleen: Laparoscopic management of wandering spleen in a case of polysplenia

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ABSTRACT

INTRODUCTION: Several congenital anomalies of the spleen have been reported. The polysplenia is a rare anomaly in which the normal spleen is replaced with two or more smaller spleens. The wandering spleen is another anomaly resulting from the laxity of the splenic ligaments. The concomitance of both anomalies is very rare.

PRESENTATION OF A CASE: A 22-year old female patient presented with intermittent left hypochondrial pain for more than a year. After a thorough examination of the patient, she only had bilateral accessory nipples. Routine laboratory investigations were all normal. An abdominal ultrasound U/S scan was unremarkable except for a ptotic spleen. with a large splenule 5 cm × 3 cm located near the fundus of the stomach. These findings were confirmed by a CT scan. A decision for a surgical intervention was then made, and the laparoscopic approach was chosen which revealed the condition. Laparoscopic removal of the wandering part was executed. The patient discharged on the first post-operative day.

DISCUSSION: The decision making in cases of wandering spleen is not always the same. The association of a wandering spleen with polysplenia is an asset to the surgical decision, along with the age of the patient.

CONCLUSION: The laparoscopic approach is an important tool in the diagnosis and management of wandering spleen. The diagnosis of polysplenic anomaly could provide a guidance for the surgical strategy in patients with wandering spleen.

1. Background

Numerous congenital anomalies of the spleen have been described, nevertheless; ploysplenia is an infrequently described anomaly.[1,2]

Polysplenia is defined as failure of fusion of the primordial buds, as a result, the splenic mass is usually divided into fairly equally sized masses varying in number from two to six and ranging from 1 cm to 6 cm in diameter, which together are equal to the mass of a normal spleen.[3]

Wandering, ptotic or ectopic spleen, which refers to migration of the spleen from its normally fixed location in the left upper quadrant is a more frequently described condition. Laxity of these ligaments and abnormal length of the splenic vessels can result in excessive splenic mobility and therefore, in the phenomenon of ptotic or wandering spleen.[4–8]

Wandering spleen can cause various manifestations that range from being asymptomatic in some cases,[13] to chronic or acute abdominal pain in others.[9–12]

To the best of our knowledge, there were no previous reports describing both abnormalities in a single case that was managed laparoscopically. We hereby present a case of wandering spleen in a case of polysplenia.

2. Case study

A 22-year old female patient presented with intermittent left hypochondrial pain for more than a year. The patient had bilateral accessory nipples. Routine laboratory investigations were all normal.

Abdominal ultrasound scan (U/S) was unremarkable except for a ptotic spleen, with a large splenule 5 cm × 3 cm located near the fundus of the stomach. These findings were confirmed by a CT scan (Fig. 1). A decision for surgical intervention was then made, and a laparoscopic approach was attempted.

Under general anesthesia, in a 45° right lateral position, three trocars were inserted, one 10 mm umbilical trocar, a 5 mm epigastric trocar and a 5 mm left iliac trocar that was replaced later by 12 mm trocar.

Exploration of the abdomen revealed polysplenic anomaly (Fig. 2). There were 2 spleens; an upper smaller one and a lower larger one. The lower spleen was ptotic and wandering (Figs. 3 and 4). The upper spleen had a well developed vascular

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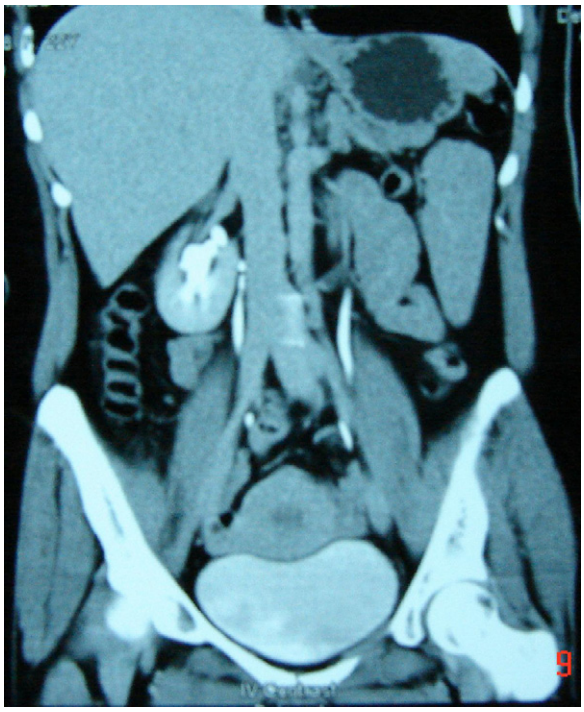

Fig. 1. CT scan showing the two splenic parts.

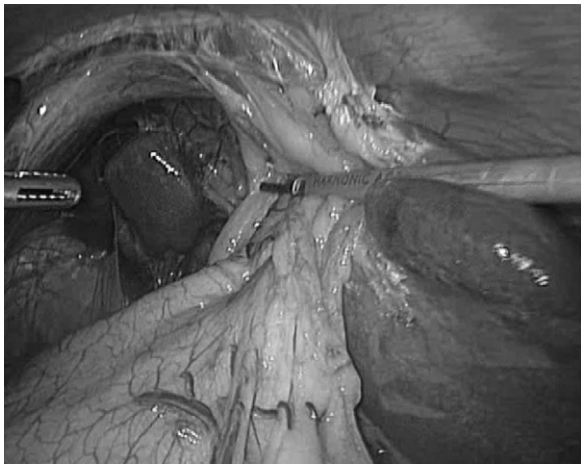

Fig. 2. The upper smaller spleen.

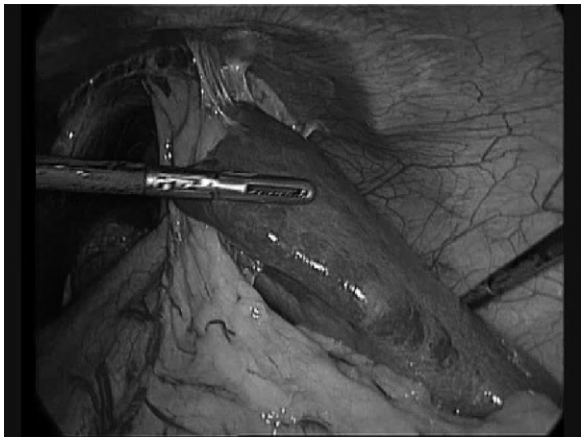

Fig. 3. Adhesions to the upper border of the wandering part of the spleen.

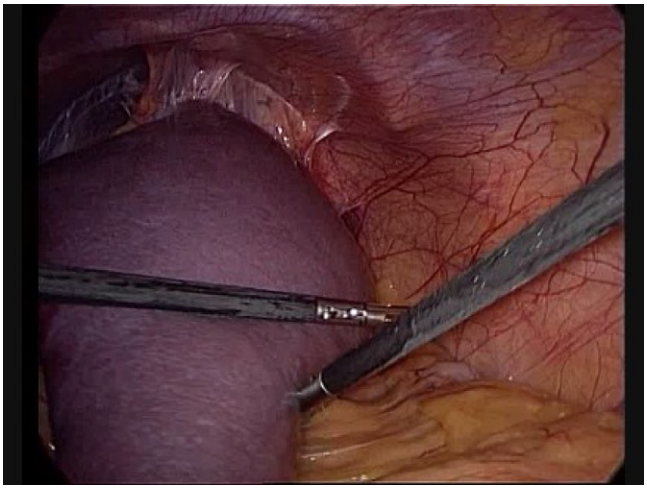

Fig. 4. The lack of attachment with the rest of the ptotic spleen.

supply coming from the short gastric vessels, while the pedicle of the lower part of the spleen was elongated and divided into large central and smaller peripheral branches to the lower pole. Only the lienorenal ligament was attached to the lower spleen and it was lax and longer than usual, with adhesions between the upper border of this lower spleen attached and the anterior abdominal wall and the lower most fibers of the diaphragm. The upper spleen was adherent to the diaphragm by dense fibrous adhesions laterally with a free medial side.

The decision to surgically remove the wandering spleen was made, and this was completed laparoscopically. The dissection was performed with an ultrasonic energy device that also controlled the minor hilar vessels. The main vascular supply was controlled with single firing of 45 mm vascular endostapler. The spleen was extracted through the umbilical port within a modified endo-bag after morcellation. Absolute haemostasis was checked, and no drain was left before port sites closure (Fig. 5).

The total anesthesia time was 73 min, with actual surgical time form skin incision to skin closure being 63 min. The pneumo-peritoneum time was 44 min. The remaining 19 min were consumed in the morcellation–extraction of the spleen, and the post sites closure.

The patient was discharged on the first post-operative day in good condition. On the fifth post-operative day she returned back with abdominal pain and vomiting. U/S diagnosed portal and

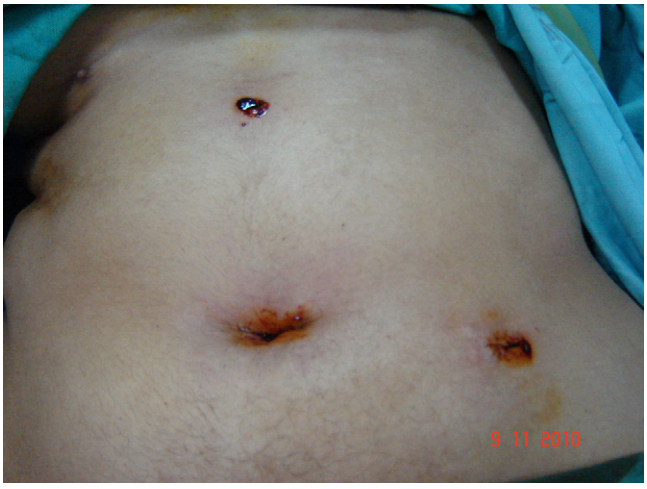

Fig. 5. The trocars sites and post-closure view.

superior mesenteric venous thrombosis. The patient was re-admitted and was started on anticoagulation (full dose low molecular weight heparin substituted later on with oral anticoagulant). The patient was discharged on the fourth day, tolerating oral feeding, with minimal abdominal symptoms.

A repeat mesenteric Doppler U/S was performed 5 months after discharge, which revealed the resurgence of the portal blood flow, with near total re-canalization of the vein.

3. Discussion

The spleen develops from a mesenchymal condensation in the dorsal mesogastrium of the lesser sac in the fourth week of gestation. [4]. During fetal development, rotation of the intestinal tract and growth of the dorsal mesentery carries the spleen to the left side of the abdominal cavity where it is anchored to the left kidney and stomach by the lienorenal and gastrosplenic ligaments, respectively. Failure of fusion of the mesogastrium with the posterior body wall leads to incomplete formation of splenic supporting ligaments and this is considered to be the mechanism by which wandering spleen occurs.[14]

The primordial buds of the spleen are created from isolated clusters of mesenchymal cells, as an elevation on the left dorsal mesogastrium. The clusters of isolated cells rapidly fuse and vascularise. The multifocal origin of the organ is proven by clusters of small spleens found in aborted embryos.[15,16]

Some of the developmental anomalies of the spleen such as lobular or accessory spleens are relatively frequent, while others, such as wandering spleen or presence of more than one spleen (polysplenia), are rare.[3,17]

In our patient laparoscopic removal of the wandering spleen was decided based on the intra-operative finding. The pattern of vascular supply, the main splenic vessels to the lower spleen and short gastric vessels to the upper spleen; supported the diagnosis of polysplenia. The decision to remove the lower wandering spleen was based on the fact that the patient had two independent splenic units, expecting that this would cause minimal adverse effects to the patient; if any. Perisplenic adhesions that were found during the intervention were interpreted as a result of perisplenitis due to the recurrent ischemic events resulting from ptosis. We thought that splenopexy,[18–21] if performed; would be an extensive intervention for the situation. Apart from bilateral accessory nipples, there were no other obvious abnormalities; that are frequently associated with polysplenia.[22]

Portal vein thrombosis (PVT) after splenectomy is a known complication occurring in 0.5–55% of reported cases, depending on definition and patient population studied.[23–27] A postulated mechanism of PVT after splenectomy is progression of a clot from the ligated/stapled splenic vein stump to the portal vein, with or without involvement of the mesenteric veins.[25] Diminished portal flow because of removal of the splenic component also contributes to a low-flow state, which favors clot formation. Third, the absence of valves in the portal venous system and its inflow may allow small limited thrombosis to easily propagate.

There has been increasing concern that laparoscopic surgery may increase the risk of mesenteric and PVT.[26,27] Laparoscopic surgery may increase venous thrombosis through a number of mechanisms. First, insufflation of the abdomen with the rise in intra-abdominal pressure may increase the risk of venous stasis.[28,29] Second, the technique of ligation of the splenic vessels with a stapler or coagulation device may have ramifications not seen with suture ligation, as is typically done with open procedures.[25] Third, the 20–30◦ anti-trendelenburg position that is required during laparoscopic upper abdominal surgery could aggravate the reduction in the portal flow.[30] Despite these hypotheses, a recent review could not confirm that laparoscopic approach for splenectomy would increase the risk of splenic or PVT over the open approach.[31] The fact that a significant proportion of patients pass asymptomatic, and as the mesenteric Doppler U/S examination is not a part of the routine postoperative work-up, it is difficult to accurately estimate the actual incidence of post-splenectomy splenic or PVT.[24,27]

Conflict of interest

None.

Funding

None.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-Chief of this journal on request.

Authors' contribution

Bekheit, the first assistant, diagnosed the case, reviewed the literature and edited the manuscript; Katry, the surgeon, revised the manuscript; Ezzat, assisted in the final revision of the manuscript.

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