Splenosis: a management conundrum in acute abdominal pain

Introduction

Splenic remnants may produce surgical complications many years after the initial trauma. This may present as acute abdominal pain, requiring laparotomy. Investigations such as computed tomography (CT) scans may be normal or inconclusive.

Discussion

Splenic trauma requiring emergency splenectomy may lead to seeding of splenic tissue throughout the peritoneal cavity. Extraperitoneal deposition of splenic tissue following trauma has also been described (Khosravi et al, 2004). Splenosis is usually detected incidentally, many years following the initial trauma.

Post-splenectomy complications are well documented. Symptomatic enlargement of accessory spleens (which occur in 25–40% of the population) following elective splenectomy for haematological conditions such as thrombocytopaenia (Mishin and Ghidirim, 2004) is one of the most frequent complications in the literature.

Abdominal pain may be caused by accessory spleens resulting from cysts, haemorrhage, rupture or torsion of the vascular pedicle (Padilla et al, 1999; Perez Fontan et al, 2001; Wacha et al, 2002). This article presents a case of abdominal pain in a young patient with presumed post-traumatic splenosis. Laparotomy was performed on the basis of clinical signs and acidosis to rule out devitalized tissue, this confirmed the splenosis but failed to explain the cause of the pain. Some authors

Case Report

A 38-year-old woman presented with a 4-day history of left upper quadrant pain and nausea, on the background of occasional mild left upper quadrant pain over the previous year. She gave a history of emergency splenectomy 18 years previously following a jet skiing accident, and was otherwise fit and well. On admission she was in pain, lying in the right lateral position with normal vital signs, a blood pressure of 126/60 mmHg, pulse rate of 72 beats per minute and respiratory rate of 16 breaths per minute. Examination confirmed tenderness in the left upper quadrant and left flank, with no signs of peritonism.

Microscopic haematuria was noted; however, intravenous urogram was normal. The patient continued to complain of pain and developed peritonism in the left subcostal region. All haematological and biochemical indices remained normal. An abdominal computed tomography (CT) was performed to rule out any unexpected pathology. This revealed extensive intra-abdominal nodules thought to represent splenosis, based on the history of traumatic splenic rupture and subsequent splenectomy. Splenosis is defined as autotransplantation of splenic tissue to unusual sites after splenic trauma. Two large nodules were located close to the splenic vein (*Figure 1*). There were no features to suggest either haemorrhage in or infarction of these presumed splenunculi, nor other radiological abnormalities to account for her pain.

In light of normal blood indices and equivocal CT result, an active observation strategy of repeated examination, haematological and biochemical testing was adopted. On day three post-admission her left upper quadrant pain became refractory to morphine patient-controlled analgesia, and arterial blood gases on this occasion confirmed a metabolic acidosis with a base deficit of 8.4.

The decision was taken to proceed to laparotomy looking for devitalized or ischaemic tissue. Except for adhesions and extensive splenic implantation as per CT findings, this was unremarkable. Multiple spenunculi were noted throughout the abdomen, none appeared to have bled or infarcted.

Postoperatively the pain did not settle, requiring increasingly large doses of morphine and necessitating high dependency unit admission. A repeat CT scan, however, showed no change from the previous scan. As the pain failed to resolve or be alleviated by any analgesia, the patient was taken for re-laparotomy 48 hours later. A decision was made to resect the largest splenunculi, which coincided with the anatomical site of the pain. One large splenunculus, lying against the diaphragm, was resected from the splenic vein, another from the omentum and two smaller splenunculi from the region of the pancreatic tail (*Figure 2*).

Postoperatively the patient improved dramatically, was mobilizing well by day 5 and was discharged home pain free 8 days after the second laparotomy. Although the large splenunculus was dusky at resection, histology revealed congestion of the red pulp with no other significant abnormalities.

Figure 1. Computed tomography scan. a. Small arrow shows small splenunculi within omentum. b. Large arrow shows large splenunculus in proximity to splenic vein.

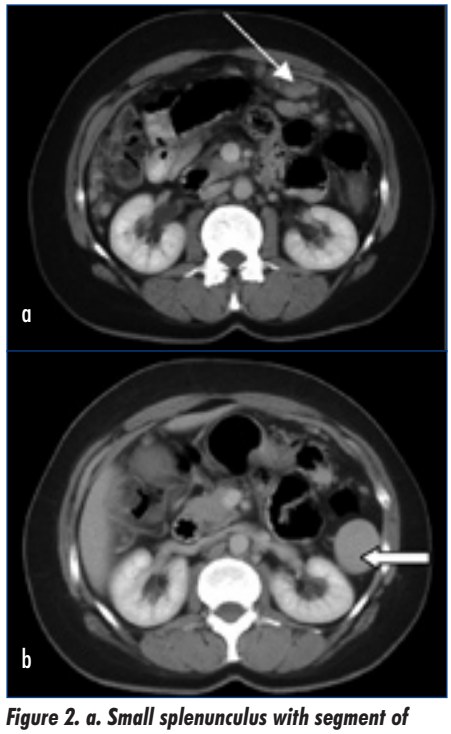
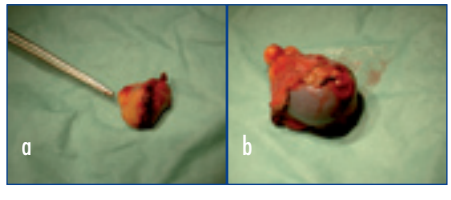


Figure 2. a. Small splenunculus with segment of pancreatic tail. b. Larger spenunculus resected from splenic vein.



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advocate diagnostic laparoscopy, but in this case laparoscopy was deemed unsafe in view of the possibility of extensive adhesions and the deep anatomical location of the nodules. As the splenunculi looked macroscopically normal, laparoscopy may not have aided diagnosis.

The management of presumed splenosis should be individualized based on presentation, and the imaging features of splenosis interpreted according to the patient's clinical condition. Incidental ectopic soft tissue masses in asplenic patients must be differentiated from malignant deposits (Greschus et al, 2003). While technetium-99m labelled red blood cell scans may be of help in confirming the nature of soft tissue masses in an elective setting, this will not aid management in an acute setting. In this case, although the CT findings suggested splenosis, there was no radiological evidence of infarction or haemorrhage, which may have accounted for the patient's symptoms.

While CT aided the diagnostic dilemma, it caused a management conundrum. However, failure of resolution of symptoms with conservative treatment and high dose opioid analgesia forced the authors to resect the incidental spenunculi within the left upper quadrant. This resolved the symptoms, but fails to explain the mechanism of pain as there is no evidence of infarction. The authors speculate that the congestion within the splenunculus may have resulted in a degree of inflammatory response with direct local diaphragmatic irritation.

It is the authors' view that in similar acute presentations, if no other cause can be found, splenunculi in the anatomical site of pain should be resected regardless of CT and macroscopic appearances. The authors believe the acidosis in this case may have been iatrogenic secondary to resuscitation of the patient. Arterial blood gases on day 3 following admission had revealed a base deficit of 8.4, which together with continued pain lead to the decision for laparotomy. However, the patient had received several litres of 'normal' saline maintenance fluid over this time which may have accounted for most of her metabolic derangement. Using the simplified Stewart-Fencl equation to estimate the relative contributions of hyperchloraemia and hypoalbuminaemia to acid-base status the base deficit corrects to virtually zero using formal biochemistry taken at the same time as the blood gas (Na 136, Cl 107, assuming normal preoperative albumin) (Story et al, 2004). This does not mean that laparotomy was not indicated, merely that her base deficit could have been accounted for by hydration with large amounts of 'normal' saline. **BJHM**

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Purple urine bag syndrome in an octogenarian male

An 83-year-old frail man with vascular dementia was admitted to the authors' geriatric ward with general deterioration. Within a few days of admission he had an indwelling urinary catheter inserted for recurrent urinary retention.

On the fifth day post-catheterization the urinary catheter bag had developed an intense purple discolouration (*Figure 1*). Urinalyses showed an alkaline urinary pH of 9 and triple and amorphous phosphate crystals. The chloroform-extracted centrifuged urinary sediment was purple and had an absorption spectrum corresponding to the wavelength of indigo. Catheter urine culture showed a mixed colonization with coliform and staphylococcal species. The purple discoloration spontaneously resolved after 3 days and did not seem to affect the clinical course of the patient.

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Figure 1. Purple discolouration of urinary catheter bag.

Purple urine bag syndrome is a benign phenomenon. Described predominantly in catheterized women with constipation, it may rarely occur in elderly males. Chemical cascades originating from bacterial metabolism of tryptophan in the gut to production of indoxyl in an alkaline urine by phosphatase- or sulphatase-producing bacteria may lead to purple urine bag syndrome, the colour possibly resulting from indigo (blue) and indirubin (red) (Dealler et al, 1988). Awareness of this self-limiting condition is important to avoid anxiety and unnecessary investigations on such urine samples. **BJHM**

Dealler SF, Hawkey PM, Millar MR (1988) Enzymatic degradation of urinary indoxyl sulphate by *Providencia stuartii* and *Klebsiella pneumoniae* causes the purple urine bag syndrome. *J Clin Microbiol* **26**(10): 2152–6