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Acute appendiceal abscess and atraumatic splenic rupture: A case of dual pathology

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ABSTRACT

INTRODUCTION: Atraumatic splenic rupture is a rare surgical emergency that is often attributed to neoplastic or infectious causes. Rarely, it has been identified to also occur in the setting of an acute severe sepsis and in cases of pelvic or splenic abscess formation post-appendicectomy. However, to our knowledge, the co-presentation of acute appendiceal abscess and splenic rupture has not been previously described.

PRESENTATION OF CASE: We present the case of a 67-year old male with decompensating haemorrhagic shock secondary to atraumatic splenic rupture on a background of an inadequately treated complicated appendicitis originally managed as diverticulitis with antibiotics in the community. Intra-operatively, in addition to a de-gloved, ruptured spleen; an acutely inflamed appendiceal abscess was also identified. A concomitant splenectomy, washout and appendicectomy and was therefore performed.

Histopathological examination revealed a normal spleen with a stripped capsular layer. Mucosal ulceration, transmural inflammation and serositis of the appendix appeared to be consistent with acute appendicitis.

DISCUSSION: Our case demonstrates how inadequately treated sepsis may predispose to an acute presentation of splenic rupture with associated haemorrhagic shock; which may initially be interpreted as septic shock. However, we demonstrate how insults such as sepsis and haemorrhagic shock may co-exist warranting careful consideration of possible dual pathologies in complex presentations which may be life-threatening.

CONCLUSION: While the causal relationship between acute appendicitis and atraumatic spontaneous splenic rupture remains unclear, our case considers and highlights the importance of considering dual pathology in patients presenting in the acute setting.

1. Introduction

Atraumatic rupture of the spleen is a surgical emergency which poses diagnostic difficulties. Its underlying pathology can primarily be attributed to one of the following categories: primary splenic cause, neoplastic, infectious, haematological, inflammatory, iatrogenic or idiopathic [1]. Neoplastic and infectious causes are thought to be the most prevalent cause of atraumatic cases [2]. The existence of idiopathic rupture is widely debated. Splenic injury can be categorised as either traumatic or atraumatic in nature, with the former being the most common aetiology and often associated with a clear history of abdominal injury. Diagnosis of a splenic rupture can be confirmed through imaging modalities such as ultrasound or computerised tomography (CT) although haemodynamic instability hastens the need for emergency resuscitation and operative management. Atraumatic splenic rupture is well recognised in the context of haematological disorders such as sickle cell and leukaemia; and secondary to infections including infectious mononucleosis and malaria. However, cases of splenic rupture have also been described among patients with severe pneumonia [3], pancreatitis [4], malignant melanoma [5], pregnancy [6,7] and among those undergoing haemodialysis [8]; though such associations are not very well understood.

Appendicitis is a very common general surgical emergency. Clinical presentations with an acute abdomen may manifest with a spectrum of presentation including focal peritonism and severe sepsis. Though the risk of mortality is low from acute appendicitis, the rate of complications rises exponentially among cases that perforate or become gangrenous [9]. Splenic rupture has been identified post-appendicectomy secondary to splenic abscess [10] or pelvic abscess [11] yet, to our knowledge, the simultaneous presentation of appendicitis and splenic rupture has not been described.

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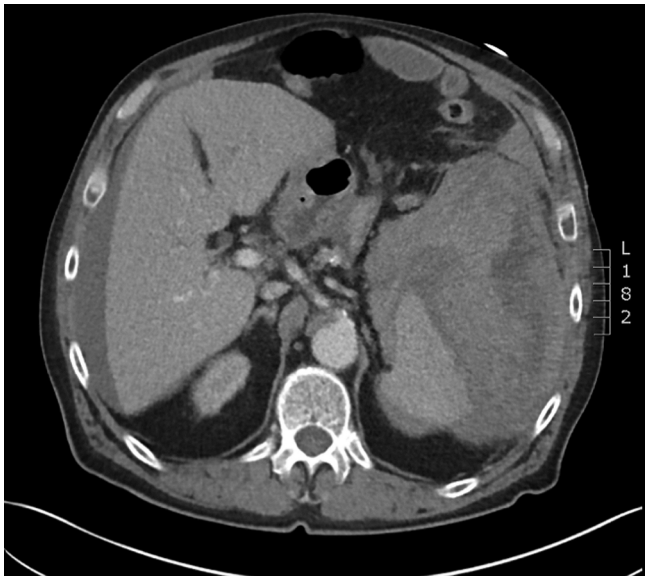

Fig. 1. Massive perisplenic haematoma with associated perihepatic haematoma.

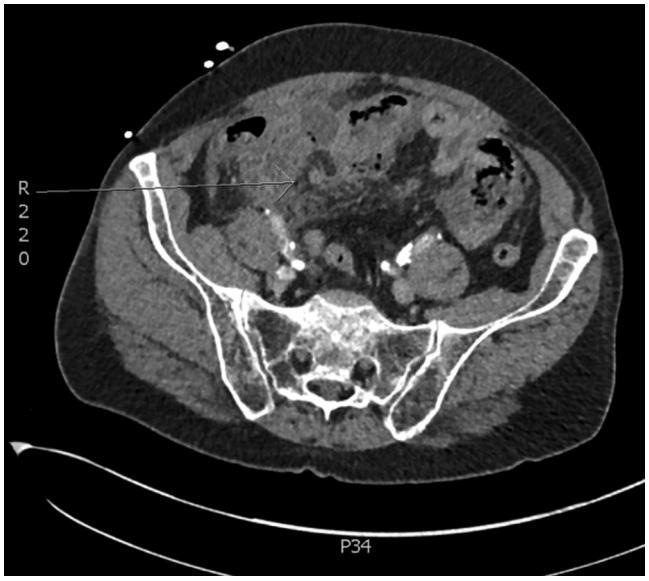

Fig. 2. Walled off pelvic abscess with adjacent locule of free air.

We present a case of an acute appendiceal abscess with concurrent atraumatic splenic rupture.

2. Presentation of case

We present the case of a 67-year-old Caucasian gentleman who presented as an emergency with a 5-day history of constant, progressive lower abdominal pain localising to the right iliac fossa in addition to a 4-h history of left upper quadrant pain. The left upper quadrant pain was sharp in nature, constant and not associated with any trauma or preceding pain in the area. He complained of associated nausea and non-bilious vomiting. The constant lower abdominal pain had been managed in the community as diverticulitis with oral antibiotics. The patient's medical history of note included peripheral neuropathy, hypertension and hypercholesterolaemia. He had a good performance status although mobilised with a walking stick and smoked twenty cigarettes a day. On examination the patient appeared pale and had cold clammy extremities, but was fully conscious. He was initially tachycardic (110 bpm) and hypotensive (74/60 mmHg), showing only transient response to fluid resuscitation. Abdominal examination revealed focal tenderness in the left upper quadrant with no overlying bruising suggesting preceding trauma.

A Focussed Assessment with Sonography for Trauma (FAST) scan was performed which excluded aortic aneurysm, but suggested fluid in both Morrison's and the rectovesical pouch. A venous blood gas showed a haemoglobin level of 72.3 g/L and a lactate of 3.4 mmol/L. The presence of abdominal pain, a low haemoglobin and stage II/III shock in the absence of haematemesis or rectal bleeding raised the suspicion of an intra-abdominal haemorrhage. Blood tests revealed a leukocyte count of 20.1×10^9/L, predominantly a neutrophilia, with an elevated C-Reactive Protein (80 mg/L) and normal platelet count. He had an elevated blood urea (10.4 mmol/L) and Creatinine (154 μmol/L). Whilst the patient responded transiently to fluid resuscitation, a non-contrast CT scan of the abdomen revealed an acute perisplenic haematoma with evidence of blood surrounding the liver (Fig. 1). Furthermore, CT imaging also demonstrated a walled-off pelvic collection with a locule of free air adjacent to it suggestive of appendicitis with localised perforation (Fig. 2). At this point exploratory laparotomy was indicated. Splenic rupture in association with acute intra-abdominal sepsis secondary to complicated appendicitis or diverticulitis was suspected.

The patient was resuscitated with intravenous fluid boluses and blood transfusion, and he was commenced on intravenous antibiotics. Whilst his blood pressure improved, the patient remained tachycardic. He was catheterised, which revealed an inadequate urine output of 20–30 ml/hour preceding laparotomy. After rapid sequence induction, emergency laparotomy revealed a massive perisplenic haematoma in the left upper quadrant of the abdomen with rupture of the splenic capsule and an actively bleeding spleen; and thus a splenectomy was promptly performed. Three litres of blood were evacuated from all 4 quadrants of the abdoemen. Examination of pelvic viscera further revealed an inflammatory mass involving an inflamed appendix central to a walled off abscess cavity. After washout of the abscess cavity, the appendix was xcised. The base of the appendix was not involved in the abscess cavity thus appendicectomy was felt to be the most appropriate management. The abdomen was left open postoperatively with application of a temporary dressing in view of a dusky transverse colon appearance, thought to be due to hypoperfusion secondary to haemodynamic instability, which we felt warranted a second look. The patient was later transferred to the intensive care unit, and his abdominal wound was closed 48 later after a second look laparotomy which revealed healthy, viable small and large bowel. After an initial period of haemodynamic instability in the post-operative period requiring inotropic support, our patient was extubated and transferred to the ward. He was discharged 13 days post-operatively, with long-term prophylactic antibiotics and post-splenectomy vaccinations.

Histopathological examination of the spleen revealed a relatively normal size and weight ($130 \times 70 \times 35$ mm, 156 g) macroscopically. The capsular layer was entirely stripped, and there was evidence of focal exudate on the surface but no evidence of a focal lesion. Microscopic examination of the spleen demonstrated evidence of reactive changes (red pulp expansion and T-cell infiltration) with no evidence of lymphomatous, leukaemic or parasitic infiltration, or any neoplastic or viral pathology. Histopathology of the appendix was consistent with an acute appendicitis, demonstrating ulceration of the mucosal layer with transmural inflammation and extensive serositis, without convincing evidence of parasitic or malignant changes.