



ELSEVIER

Contents lists available at ScienceDirect

## International Journal of Surgery Case Reports

journal homepage: [www.casereports.com](http://www.casereports.com)

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



Piriyanakan Ananthavarathan, Kamlesh Patel\*, Catherine Doran, Nigel Suggett

Department of Colorectal Surgery, New Queen Elizabeth Hospital Birmingham, University Hospitals Birmingham NHS Foundation Trust, Mindelsohn Way, Edgbaston, Birmingham B15 2GW, United Kingdom

## ARTICLE INFO

## Article history:

Received 25 January 2016

Received in revised form 20 March 2016

Accepted 8 April 2016

Available online 13 April 2016

## Keywords:

Atraumatic splenic rupture

Acute appendicitis

## 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.

© 2016 Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## 1. Introduction

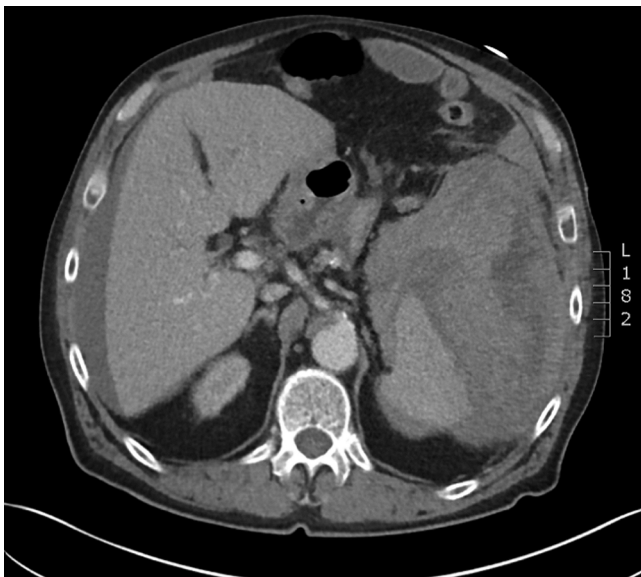
Atraumatic rupture of the spleen is a surgical emergency which poses diagnostic difficulties. Its underlying pathology can primarily be attributed to one of the following categories: primary splenic cause, neoplastic, infectious, haematological, inflammatory, iatrogenic or idiopathic [1]. Neoplastic 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.

\* Corresponding author.

E-mail address: [kamleshpatel@doctors.org.uk](mailto:kamleshpatel@doctors.org.uk) (K. Patel).



**Fig. 1.** Massive perisplenic hematoma with associated perihepatic hematoma.



**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 Focused 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  $20.1 \times 10^9/L$ , 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  $\mu\text{mol/L}$ ). Whilst the patient responded transiently to fluid resuscitation, a non-contrast CT scan of the abdomen revealed an acute perisplenic hematoma 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 hematoma 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 abdomen. 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 excised. 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 × 70 × 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.

### 3. Discussion

Our case demonstrates the dangers attributing shock to a single cause. In our patient the clinical picture of decompensated shock was secondary to haemorrhagic shock due to atraumatic splenic rupture on a background of appendiceal abscess. The patient's history and blood tests pointed to acute sepsis with the patient meeting criteria for severe septic shock which was thus falsely deemed to be the cause of shock. Without clear history of attributing factors, the possible aetiology of splenic rupture in these instances is often challenging to identify. In our case, the lack of a history of physical injury suggests traumatic splenic injury is unlikely. The use of the Orloff and Peskin criteria [12] may also be used to assist diagnosis of atraumatic splenic injury given that all four criteria are met; including no obvious antecedent history of traumatic injury; no clear indication of disease in other organs that may drive splenic rupture; no evidence of previous splenic trauma or rupture such as perisplenic adhesions or scarring; and a normal spleen on histopathological examination other than stripping of the splenic capsule.

This makes the diagnosis of atraumatic splenic injury far more likely, though its association to the presentation of appendicitis remains unclear. A possible explanation could be the development of a splenic abscess leading to secondary appendicitis, though histological examination did not show this. Crate and Payne [13] have suggested the need for full virological studies to be undertaken to confirm the absence of recent viral infection that predispose splenic involvement: however, our patient gave no history of preceding symptoms suggestive of prodrome or infection. Recent literature has also demonstrated cases of atraumatic splenic rupture that occur secondary to Anisakis appendicitis [14]; involving parasitic infiltration of the appendix by nematodes secondary to consumption of raw seafood. However, this was also absent on histological examination.

Alternatively, it can be argued that the superimposed presentation of appendicitis may in fact be a purely incidental finding in a patient presenting with atraumatic spontaneous splenic rupture. Yet the history of five days' right iliac fossa pain which was initially treated as diverticulitis with antibiotics suggests the contrary; wherein in actual fact the patient had an inadequately treated form of acute complicated appendicitis. The identification of a localised appendix-abscess cavity in theatres also supports this notion, wherein worsening inflammation and micro-perforation lead to signs of sepsis, which were interpreted as septic shock due to hypotension and tachycardia. It is plausible that the sepsis itself may drive dynamic changes in the spleen causing its rupture and subsequent hypovolaemic shock.

Several mechanisms for atraumatic splenic rupture have been proposed. These include vascular causes such as splenic thrombosis or infarction which may lead to subcapsular haemorrhage; damage to the fibrous splenic capsule such as through cellular infiltration; mechanical non-traumatic damage, for instance, through stretching of the splenic capsule with diaphragmatic or abdominal muscle contraction; or vasculitic causes, for example through the formation of a splenic vessel aneurysm. Atraumatic splenic rupture may also occur from raised intrasplenic pressure, such as in cases of venous congestion secondary to torsion of an abnormally mobile spleen, splenic vein spasm or portal hypertension [11,15–18]. Changes in intrasplenic pressure may also result from splenic oedema; haemostasis such as through formation of a splenic parenchymal haematoma; or cellular hyperplasia driven by an underlying acute inflammatory response [11]. In our patient, the presence of an underlying acutely decompensated appendicitis and sepsis suggests this to be the most likely mechanism underpinning atraumatic splenic rupture and warrants a need for further

understanding of splenic involvement in the acute inflammatory cascade.

### 4. Conclusion

As the above discussion illustrates, our patient presented with acute haemorrhagic shock secondary to atraumatic splenic rupture which may have occurred independent of, or be coincidental to, acute appendicitis with abscess formation. Patients in the emergency setting typically present with a worsening inflammatory process and often show signs typical of septic shock. However, it is fundamental to consider other causes of acute decompensation, such as haemorrhagic shock secondary to atraumatic splenic rupture in our case, as these other causes of shock are often identified late and can be fatal if missed.

### Conflict of interest

None declared.

### Funding

None.

### Ethical approval

Not required.

### Consent

Informed consent obtained from patient.

### Author contribution

Piriyankan Ananthavarathan—Manuscript compilation.  
Kamlesh Patel—Manuscript Compilation.  
Catherine Doran—Review prior to submission.  
Nigel Suggett—Review prior to submission.

### Guarantor

Kamlesh Patel.  
Nigel Suggett.

### References

- [1] B.H. Hyun, C.F. Varga, R.J. Rubin, Spontaneous and pathologic rupture of the spleen, *Arch. Surg.* 104 (1972) 652–657.
- [2] P. Renzulli, A. Hostettler, A.M. Schoepfer, B. Gloor, D. Candinas, Systematic review of atraumatic splenic rupture, *Br. J. Surg.* 96 (October (10)) (2009) 1114–1121.
- [3] M. Casanova-Roman, J. Casas, A. Sanchez-Porto, B. Nacle, Spontaneous rupture of the spleen associated with Legionella pneumonia, *Can. J. Infect. Dis. Med. Microbiol.* 21 (2010) 107–108.
- [4] B.L. Hernani, Acute pancreatitis complicated with splenic rupture: a case report, *World J. Gastrointest. Surg.* 7 (2015) 219.
- [5] H. Mirfazaelian, A. Oryan, A. Davari, K. Daneshbod, Y. Daneshbod, Spontaneous splenic rupture in melanoma, *Case Rep. Pathol.* (2014) 1–3.
- [6] S.E. O'Brien, Spontaneous rupture of the spleen in pregnancy, *Can. Med. Assoc. J.* 89 (1963) 667–668.
- [7] C. Wang, X. Tu, S. Li, G. Luo, E.R. Norwitz, Spontaneous rupture of the spleen: a rare but serious case of acute abdominal pain in pregnancy, *J. Emerg. Med.* 41 (2011) 503–506.
- [8] H.J. Kim, G.W. Lee, D.J. Park, J.D. Lee, S.H. Chang, Spontaneous splenic rupture in a hemodialysis patient, *Yonsei Med. J.* 46 (3) (2005) 435–438.
- [9] A. Bhangu, K. Søreide, S. Di Saverio, J. Hansson Assarsson, F.T. Drake, Acute appendicitis: modern understanding of pathogenesis, diagnosis, and management, *Lancet* 386 (2015) 1278–1287.
- [10] R. Patel, A. Pai, I. Al-Shoek, C. Evans, A. Gordon, 2012. Splenic abscess rupture postappendectomy, *BMJ Case Rep.* (2012), <http://dx.doi.org/10.1136/bcr.01.2012.5705> (Published 2012).

- [11] A.K. John, Splenic rupture in a patient with pelvic abscess and sepsis, *Singapore Med. J.* 53 (2012) 6–8.
- [12] M.J. Orloff, G.W. Peskin, Spontaneous rupture of the normal spleen; a surgical enigma, *Int. Abstr. Surg.* 106 (1958) 1–11.
- [13] I.D. Crate, M.J. Payne, Is the diagnosis of spontaneous rupture of a normal spleen valid? *J. R. Army Med. Corps* 137 (1991) 50–51.
- [14] J. Valle, E. Lopera, M.E. Sanchez, R. Lerma, J.L. Ruiz, Spontaneous splenic rupture and Anisakis appendicitis presenting as abdominal pain: a case report, *J. Med. Case Rep.* 6 (2012) 114.
- [15] E. Wehbe, S. Raffi, D. Osborne, Spontaneous splenic rupture precipitated by cough: a case report and a review of the literature, *Scand. J. Gastroenterol.* 43 (2008) 634–637.
- [16] N.T. Toubia, M.M. Tawk, R.M. Potts, G.T. Kinasewitz, Cough and spontaneous rupture of a normal spleen, *Chest J.* 128 (2005) 1884–18846.
- [17] W.G. Byerly, Thrombosis of the splenic vein with secondary rupture of the spleen, *N. C. Med. J.* 36 (1975) 352–354.
- [18] J.C. Thijs, A.J. Schneider, J.M. van Kordelaar, Spontaneous rupture of the spleen complicating portal hypertension, *Intensive Care Med.* 9 (1983) 299–300.

#### Open Access

This article is published Open Access at [scimedirect.com](http://scimedirect.com). It is distributed under the [IJSCR Supplemental terms and conditions](#), which permits unrestricted non commercial use, distribution, and reproduction in any medium, provided the original authors and source are credited.