Spontaneous Splenic Rupture As A Complication Of Acute Pancreatitis

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Citation

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Abstract

A 56 yr old man was admitted with a diagnosis of acute pancreatitis. However during in-patient stay he became severely hypotensive with abdominal rigidity. At the proceeding laparotomy the spleen was noted to be disintegrated. Histology of remnant spleen showed no underlying pathology. The patient made an eventual recovery developing a pseudocyst, which was treated with conservative management. Our conclusion was spontaneous splenic rupture secondary to an episode of acute pancreatitis. This is a rare event with catastrophic sequelae if not considered early in the course of the patients presentation. A literature search showed that splenic complications of chronic pancreatitis were not uncommon but were a rare event in an acute episode, with surgical intervention being the optimal management.

INTRODUCTION

Spontaneous splenic rupture is a rare condition that is initially very difficult to diagnose. As such, it is associated with high morbidity and mortality. We present a case of spontaneous splenic rupture associated with acute pancreatitis.

CASE HISTORY

A 56-year-old man was admitted with a 24 hr history of left subcostal and loin pain. He denied any recent history of trauma. On further questioning, with the exception of an above average alcohol consumption, past medical history was unremarkable. On examination, the patient was obese and in obvious distress with marked pyrexia and tachycardia. Chest was clear and the abdomen was noted to be soft but tender only at the left loin. Blood tests revealed an increased white cell count of 16 x 109/l. with biochemistry significantly showing hyperamylasaemia and deranged liver function tests. The patient was therefore diagnosed and treated as acute pancreatitis. A CT scan performed the next day demonstrated an inflamed pancreas with multiple peripancreatic collections. However, sudden respiratory distress with marked hypoxia developed within 12 hours of the CT scan examination. A pulmonary embolism was suspected but was later excluded on the basis of a normal ventilation/perfusion scan. The patient progressed to develop a rigid, tender, distended abdomen with marked cardiovascular instability. An emergency laparotomy was

performed revealing large volume of free blood with clots in the peritoneum. The spleen was noted to be disintegrated and surrounded by a large haematoma. No other abnormality was found at laparotomy. Histology of the remnant spleen was unremarkable and did not reveal a haematological cause for the splenic rupture. Further scans subsequently demonstrated the development of a pseudocyst, which was treated conservatively. The patient has since recovered and has been discharged. We are currently reviewing him regularly as an outpatient.

COMMENT

To our knowledge, spontaneous splenic rupture, as a complication of acute pancreatitis, is a rare condition. As such only seven cases have been reported since 19451.

Splenic rupture with no or minimal force is not uncommon as it is noted in several conditions. Haematological malignancies₂, infectious mononucleosis₃ and malaria₄ are some of the most significant underlying cause of a traumatic splenic rupture. However it may also complicate some rheumatological disorders including systemic lupus erythematosus that can present with an acute abdomen₅. These patients will generally possess a moderate to severely enlarged spleen prior to rupture with histology confirming the pathological nature of the underlying disease. Very rare causes of spontaneous splenic rupture include viral infections such as Hepatitis A₆ and Rubella₇ as well as bacterial pneumonia₈. In these cases, the patients present

with symptoms reflecting the underlying disease, which is confirmed by a positive serology. Similar to our patient, surgical intervention was deemed necessary on these extremely rare cases.

In our patient, the diagnosis of spontaneous splenic rupture, secondary to acute pancreatitis, was ascertained by a raised amylase and a CT scan showing significant pancreatic swelling. The mechanism of splenic rupture secondary to acute pancreatitis has several proposals including splenic vein thrombosis or ectopic pancreatic tissue in spleen, but however largely remains undetermined. Histology of the spleen unfortunately on this occasion did not reveal any of the proposed mechanisms. Splenic complications of chronic pancreatitis are well recognised with an incidence of 2.2%₉. However, as a complication of an acute episode it is very rare and can easily be missed. This can lead to a significant delay in the appropriate treatment of an acutely ill patient. This, in turn, may result in increased morbidity and even mortality. Therefore, spontaneous splenic rupture should be considered in the differential diagnosis in any patient presenting with a raised amylase and is haemadynamically unstable.

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