

CASE REPORT

Lesson learnt: don't prescribe heparin for hemoperitoneum!

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This paper describes a case of spontaneous rupture of the spleen that was misdiagnosed as pulmonary embolism. Because of a lack of history of trauma, rarity of occurrence, and confusing clinical presentations, an unwary clinician may fail to diagnose this condition in time. It may result in erroneous treatment of a condition, which is potentially curable.

A 48 year old computer engineer presented to the accident and emergency department with lower chest and upper abdominal pain of 40 minutes' duration. The pain started an hour after he had eaten his supper, while sitting at home. The pain was sharp and severe in nature; there was no radiation, no associated aggravating or relieving factors. He felt tightness and discomfort around the chest. There was no significant medical history. On arrival his blood pressure was 96/46 mm of Hg; pulse rate 90/min and regular; respiratory rate 26/min. Examination of the chest confirmed good bilateral air entry without any adventitious sounds. Abdominal examination revealed moderate tenderness in epigastrium but no rebound; there was no rigidity and bowel sounds were present. Laboratory investigations revealed a haemoglobin concentration of 128 g/l, leucocyte count of $11.9 \times 10^9/l$, platelet count of $340 \times 10^9/l$, normal values of urea, electrolytes, amylase, and cardiac enzymes. Arterial blood gas analysis showed acute respiratory alkalosis and hypoxia. Erect chest radiography was normal and did not show any free gas under the diaphragm. There was suspicion of slight changes in the electrocardiogram (S₁, Q₃, T₃) but no changes suggestive of acute myocardial infarction.

He was transferred to the surgical ward in view of abdominal pain. However, the clinical features and investigations were not conclusive. A medical opinion was obtained. Pulmonary embolism was thought to be the provisional diagnosis. Therefore, in addition to supportive treatment, he was given intravenous heparin. On review six hours later, his main complaint appeared to be upper abdominal pain. He was no longer complaining of any chest symptoms. Abdominal examination revealed rigidity and rebound tenderness in the upper abdomen. It was becoming clear that the underlying disorder was intra-abdominal rather than intrathoracic.

A perforated hollow viscus was thought to be the most probable diagnosis. At emergency laparotomy a large subcapsular haematoma was found near the splenic hilum, which had ruptured the splenic capsule. No peri-splenic adhesions or scarring of spleen were noted. The total blood loss was 1400 ml and he required three units of packed red cell transfusion. A splenectomy was performed. The spleen was not enlarged and found to be normal, both macroscopically and microscopically. His postoperative recovery was uneventful.

Although he was heparinised initially, heparin was not thought to be the cause of splenic rupture (as the patient already had symptoms before treatment with heparin). However, addition of an anticoagulant might have compounded the situation!

In retrospect, he categorically denied any history of trauma in the preceding 12 months. He remained well on regular follow ups and was subsequently discharged from the clinic after one year.

DISCUSSION

It is recognised in the literature that presentation of spontaneous rupture of spleen can be deceptive and may present as pulmonary embolism.¹ The other conditions that may be mimicked are perforated peptic ulcer,² angina pectoris, myocardial infarction, ectopic pregnancy, acute appendicitis, and acute sigmoid diverticulitis.¹

Acute abdomen had quite aptly been described as "a trap for the unwary".³ Despite a careful approach, there would remain some situations where clinical features would be non-specific in nature and base line investigations would fail to provide any diagnostic information. However, a high index of suspicion and repeated clinical examinations would be helpful to assess such a situation. If the patient failed to make any satisfactory progress and the condition still remained unclear, it would be worth performing special investigations at an early stage. In this case an ultrasound scan of the abdomen would have clinched the diagnosis preoperatively. Ultrasonography of the abdomen should be the investigation of choice in similar circumstances, particularly in the absence of any associated feature of deep venous thrombosis of legs to suggest a possible source of pulmonary embolism.

Spontaneous rupture of spleen was first described by Atkinson⁴ in 1874. However, its existence had always been doubted.⁵ The condition can be fatal, unless diagnosed, and treated promptly.⁶ Absence of any history of trauma makes the diagnosis very difficult and causes delay in treatment.

Orloff and Peskin⁷ performed an extensive review in 1958 and specified four criteria that must be met before a rupture of spleen could be called spontaneous. The criteria are (1) no history of trauma or unusual effort; (2) no evidence of any disease that may affect the spleen adversely; (3) no evidence of peri-splenic adhesions or scarring of the spleen to suggest trauma or rupture; and (4) the spleen should be normal on macroscopical and histological examination. The present case fulfils all these criteria.

It is now well recognised in the literature that spontaneous rupture of spleen does occur, although the actual mechanism remains uncertain.⁸ It is suggested that greater awareness and familiarity with this uncommon condition would increase early diagnosis, resulting in prompt surgical intervention and improved survival.

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