CASE REPORTS

Delayed rupture of the spleen in rheumatoid arthritis

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Abstract
When tripping and falling patients with rheumatoid arthritis may adopt the ‘fetal tuck’ position to protect their painful deformed hands. There is then a risk of splenic injury by the left elbow, which may not be immediately apparent.

Rupture of the spleen associated with splenomegaly of rheumatoid arthritis is well known and may occur after trauma or spontaneously.1 2 Splenomegaly in rheumatoid arthritis occasionally requires elective splenectomy but with growing regard for splenic conservation there may now be fewer indications to do so. We report a case where a common mishap led to a dangerous sequence of events in a patient with rheumatoid arthritis and splenomegaly. The phenomenon of delayed rupture of the spleen in such cases has not been previously reported.

Case report
A 56 year old man presented to casualty with a two hour history of severe epigastric pain that radiated to the left shoulder. An electrocardiogram was normal. Two weeks previously, while at work as a factory health and safety officer, he had tripped on a piece of loose board and fallen forwards. He had instinctively adopted a ‘fetal tuck’ position to protect rheumatoid hands (figure). He remembered striking his left brow and shoulder on a bench before landing on his left side with the left elbow tucked against the lateral chest wall.

He was admitted to hospital because he had been knocked out. Head injury observations were stable and he went home after 24 hours. Six days later he was still feeling shaken, lethargic, and dyspeptic. A haemoglobin concentration taken at that time was 77 g/l. By the time this result was available the patient had had the acute events that led to this presentation.

On admission the patient was conscious but in pain, very pale, and shocked. He was taken for operation immediately and a diagnosis of delayed rupture of the spleen was confirmed. Blood and clot (6 litres) were evacuated from the peritoneal cavity. There was a large ruptured subcapsular haematoma round an enlarged spleen (14 cm×14 cm×6 cm; 417 g). Splenectomy was performed followed by autologous transplant of minced splenic tissue into an omental pouch. After the operation the patient was treated with prophylactic antibiotics and was subsequently vaccinated against pneumococcal infection. There were no clinical or radiological signs of rib fracture.

Discussion
The incidence of palpable splenomegaly in patients with rheumatoid arthritis is 5–10%, but splenomegaly shown by volume measurement from isotope scans occurred in 60–70% of cases with active disease.4 The pathological characteristics of Felty’s syndrome5 were believed to stem from splenomegaly with exaggerated splenic function, and splenectomy has often been advocated in such cases.6 Many factors commonly combine to produce the decrease in blood elements, however, including the presence of antibodies to white cells in the serum of patients with Felty’s syndrome.7 This explains in part why the results of elective splenectomy for hypersplenism may be disappointing. Neutropenia was not present after the operation in this case and splenomegaly had not been apparent.

Delayed rupture of the spleen is well described8 but is a rarity in patients with rheumatoid arthritis. It is presumed that a subcapsular haematoma developed and self tamponade occurred; then, two weeks later, liquefaction and absorption of haematoma led to massive secondary haemorrhage. Rib fractures may occur more commonly in osteopenic rheumatoid patients and thereby damage the spleen, but this did not happen in this case. It illustrates how splenic rupture may arise as a result of a direct blow over the spleen, however, because the operative findings showed a split in the organ rather than a penetrating injury or tearing of the pedicles by shearing forces.
Alternatives to emergency splenectomy were discussed by Cooper and Williamson,3 but in this case the indications for emergency total splenectomy were clear. Reports of the smaller risk of post-splenectomy sepsis after surgery for rupture rather than thalassaemia indicated that the presence of functioning splenic cells, splenosis, may confer some protection to the patient, and therefore autologous transplantation has a place in the operative management of these cases. The protection is presumed to be due to the antibody activity of the autotransplanted splenic tissue as pneumococcal clearance rates are dependent on a main arterial supply to the splenic tissue.3 Further studies are required to evaluate precisely autologous splenic transplantation, but a combination of splenosis, regular immunisation, and antibiotic prophylaxis offers the best protection for the patients.

In patients with rheumatoid arthritis early diagnosis of splenomegaly with splenic conservation is preferable, but if splenomegaly is increasing or symptomatic then elective splenectomy should be considered. Indications for splenectomy remain clinically and haematologically based but, if patients are considered to be at risk of splenic rupture, monitoring of splenic volume by serial ultrasound scanning is recommended. Progressive enlargement may require surgery on purely mechanical grounds and partial splenectomy3 may be useful to reduce the volume of splenic tissue while maintaining main arterial supply and full splenic function.

Rheumatoid patients need to be aware of the risks of splenic rupture, particularly if they have developed new reflexes to protect painful hands. Formal counselling and advice in joint protection may be useful, especially for patients who are prone to falling. Instruction on how best to cope with falling, such as teaching rolling techniques, may help the patients to avoid damaging vital organs and joints.

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