Splenic peliosis associated with rupture in a renal transplant patient

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Summary
A patient is described who, 2 years after renal transplantation, presented as an acute abdominal emergency due to a ruptured spleen. Histologically this displayed peliosis.

Introduction
Peliosis is an uncommon entity consisting of blood-filled cavities found most frequently in the liver (peliosis hepatis), although its rare occurrence in the spleen has been described. Originally splenic peliosis was considered a histological curiosity with no clinical significance. Recently, however, 2 cases of fatal haemoperitoneum have been reported following rupture of splenic peliotic cysts (Taxy, 1978; Benjamin and Shunk, 1978).

The authors report an additional case of splenic peliosis associated with non-fatal rupture in a renal transplant patient.

Case report
In 1967, a 21-year-old man presented with chronic renal failure due to chronic glomerulonephritis. After initial peritoneal and haemodialysis in hospital, home dialysis was commenced in 1969. A successful cadaver renal transplant was performed in May 1972. Weekly intramuscular injections of 250 mg testosterone were begun in June 1971 for anaemia (6 g/dl) but were stopped after transplantation. He was maintained on immunosuppressive therapy (azathioprine 100 mg and prednisolone 12.5 mg daily) and remained well until October 1974. He then presented with a sudden onset of pain in the left upper abdominal quadrant with radiation to the shoulder. A mass developed in the left upper quadrant and his haemoglobin fell from 14 to 11 g/dl. The clinical impression was that of a ruptured spleen. There had been no history of trauma, and tests for infectious mononucleosis were
The liver showed case conforming with debilitating artery and vein and the macrophages a large liver function negative.

The spleen weighed 210 g and the distal end showed a large ruptured subcapsular haematoma. The red pulp contained scattered blood-filled cavities (up to 3 mm diameter), adjacent to the haematoma and in subcapsular areas (Fig. 1). The larger cavities appeared continuous with ectatic sinusoids but littoral cells were not always demonstrable. These appearances were considered to be those of splenic peliosis. In addition, plentiful haemosiderin-containing macrophages were present in the red pulp and in places lined the sinusoids and peliotic cavities. No increase in fibrous tissue could be demonstrated, and the white pulp was mildly depleted. The splenic artery and vein were normal.

**Discussion**

Splenic peliosis was initially described in patients with debilitating diseases such as tuberculosis and malignancy. Peliosis hepatis was also invariably present and recent reports confirm such an association (Taxy, 1978; Benjamin and Shunk, 1978; Lacson, Berman and Neiman, 1979). Taxy (1978) has reported 2 cases of splenic peliosis without hepatic involvement and it is likely that the present case conforms to isolated splenic peliosis, although a liver biopsy was not performed.

Suggested aetiological factors in the development of peliosis have included anabolic steroids and azathioprine (Taxy, 1978; Benjamin and Shunk, 1978; Lacson, et al., 1979; Degott et al., 1978) and it is perhaps relevant that the patient had received both these drugs. An interesting finding in this case was the heavy deposition of haemosiderin in the spleen. This feature is frequently seen in renal dialysis and transplant patients, and is currently under investigation. It is possible that the haemosiderin interfered with vascular drainage and gave rise to phlebectatic peliotic cavities as described by Yanoff and Rawson (1964). From the distribution of the lesions it would appear probable that the haemorrhage originated from a ruptured peliotic cavity. This complication should be considered in renal transplant patients.

**References**


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