SPLENOSIS: RUPTURE OF SPLEEN, WITH SPLENIC IMPLANTS

REVIEW OF LITERATURE AND REPORT OF CASE

BY

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Autotransplantation of splenic tissue following traumatic rupture of the spleen is a rare finding, and at the time of preparation of this article I was unable to find a case report in British journals.

Storsteen and Remine (1953) collected two cases and reported one of their own. Since then Wise (1953) and later Garamella and Hay (1954) have published cases, bringing the number to 25. The present case is the twenty-sixth, and, apart from drawing attention to this definite clinical and pathological entity, it stresses the importance of its recognition when one is confronted with the condition at laparotomy. In 1896 Albrecht reported a necropsy in which multiple splenic nodules were found throughout the peritoneal cavity (approximately 400). No history of trauma was given in the report, but the widespread distribution (including both domes of the diaphragm) and the number of nodules was consistent with splenic seeding. Albrecht reported the case as one of accessory spleens (spleniculi).

In 1910 von Küttner at necropsy found 80–100 splenic nodules, and in his case there was a definite history of trauma. Shortly after Küttner's case Faltin (1911) noted at operation for appendicitis the existence of numerous splenic nodules where there had been a splenic rupture some six years previously. He suggested the possibility of splenic implantation rather than accessory spleens.

Von Stubenrauch (1912), in publishing a similar case, postulated his splenoid theory. He stated that the loss of splenic tissue could be compensated for in several ways: (1) cellular changes in bone marrow and the lymph nodes as well as in pre-existing haemolymph nodes; (2) by regeneration of incompletely removed splenic tissue or hypertrophy of accessory spleens; or (3) the formation within the peritoneal cavity of organs having similar structure to the spleen—these he named splenoids. However, Kreuter (1920) showed experimentally on monkeys that complete splenectomy did not show any such compensating mechanism. Roettig *et al.* (1943) have successfully carried out peritoneal implantation of splenic tissue in rabbits, and it has been done experimentally on other animals, including rats and dogs.

Splenic tissue in the reported cases has been noted throughout the peritoneal cavity, including the right parietal peritoneum, lesser omentum, right diaphragm, and even subcutaneously in the left thoracic wall (in scar). It would be inconsistent with our knowledge of the embryological development of the spleen from the left side of the dorsal mesogastrium to accept these nodules as congenital accessory spleens.

In 1939, Buchbinder and Lipkoff published their case noted at laparotomy some 19 years after the traumatic rupture of the spleen. The seedlings were numerous throughout the parietal peritoneum, pelvic cavity, omentum, serous coats of small and large intestine, and the diaphragm. They deemed this clinical and pathological entity worthy of a name and called it "splenosis."

In the practice of splenectomy for medical conditions such as congenital haemolytic jaundice and idiopathic thrombocytopenic purpura the removal of splenic tissue must be complete. Not only does this mean that accessory spleens (present in 11% of all age groups) must be sought and removed, but also that the splenic capsule must not be ruptured, with subsequent spill of splenic cells into the peritoneal cavity. If this should occur the peritoneal cavity should be carefully lavaged with saline. The importance of preserving an intact splenic capsule is confirmed by Stobie (1947). He reported a case of congenital haemolytic jaundice in which the capsule was inadvertently ruptured during splenectomy. The patient was re-explored four years later for a recurrence of the jaundice, and multiple splenic seedlings were found.

Diagnosis

At laparotomy one may confuse the condition with endometriosis (Hartman, 1953) or carcinomatosis (Daniel, 1951) or multiple angiomata. With the previous history of a ruptured spleen there should be no difficulty in diagnosis if the condition is borne in mind. The splenic implants resemble normal splenic tissue in colour and are generally sessile but may be pedunculated. They are commonly seen in the greater omentum and on the small and large intestines and parietal peritoneum, but may be present anywhere in the peritoneal cavity.

The splenic implants have no true hilum and the blood vessels enter anywhere at the periphery. Microscopically, they consist of splenic pulp with the trabeculae usually less well developed than in a normal spleen. The capsule is less distinct and the nodule is covered with a mesothelial layer.

Case Report

A 45-year-old man was admitted to hospital on September 10, 1954, with a severe haematemesis. He had been at work one hour previously, when he suddenly felt faint, vomited a considerable quantity of fresh blood, and collapsed. There was no previous digestive history, but he had been taking aspirin for a "cold." His general health had been good. In 1924 splenectomy had been performed for a ruptured spleen following a motor-cycle accident.

On examination he was pale and shocked. His temperature was 97.2° F. (36.2° C.), pulse 100, respirations 22, and B.P. 105/70. He had a left upper paramedian scar on the abdomen. Rectal examination revealed melaena stool. No dilated veins were present, nor were any telangiectases noted. The liver was not palpable. Haemoglobin was 53%, and the coagulation and bleeding times were normal. The blood urea was 37%, and the blood group B, Rh-positive.

During the next 48 hours 6 pints (3.4 litres) of blood was transfused, but, as the bleeding showed no signs of abating, surgical intervention was decided upon. Laparotomy under general anaesthesia was performed and the intestines were found to be full of dark blood. No obvious gastric or duodenal ulcer could be found and the liver was normal. The spleen was not present.

There were numerous small plum-coloured nodules on the small intestine, the mesentery and greater omentum, and the caecum and ascending colon. They were also present on the lesser omentum, near the foramen of Winslow, and the first part of the duodenum. The largest was about 1.5 cm. in diameter, but most were very small. As no obvious site for the bleeding presented, the possibility arose of the nodules being angiomata. Further palpation of the gastroduodenal tract revealed a small nodule on the posterior wall of the first part of the duodenum. On pylorotomy the nodule proved to be a small acute duodenal ulcer—the site of the haemorrhage.

A partial gastrectomy (Polya-Hofmeister) was performed and it was thought probable that the nodules were splenic

seedlings. A nodule was removed for histopathological examination from the region of the free margin of the lesser omentum.

In the post-operative period the patient developed a thrombosis of the right calf. This responded to anticoagulant therapy, and he left hospital for convalescence four weeks after admission.

Pathological Report (Dr. A. G. Stansfield).—"Nodule from free edge of lesser omentum. Small lobulated mass of adipose tissue containing two soft red vascular nodules, the larger 6 mm. in diameter. Microscopically both nodules show the characteristic of splenic tissue, displaying typical Malpighian bodies and venous sinuses separated by Billroth cords. Confirmation of the nature of the tissue is provided by the reticulin pattern in a silver impregnation preparation. The latter shows quite clearly the ladder-like structure of the reticulin fibres in the walls of the venous sinuses. Polymorphonuclear neutrophil leucocytes are present in considerable numbers both in the sinuses and in the pulp cords.

FIG. 1.—Note fibrous capsule, Malpighian bodies, separated by Billroth cords, and venous sinuses—characteristic structure of splenic tissue. (Haematoxylin and eosin. $\times 100$.)

FIG. 2.—Note the ladder-like structure of the reticulin fibres on the walls of the venous sinuses. (Silver impregnation preparation. $\times 450$.)

The nodules are not enclosed by a normal splenic capsule, but there is a more or less distinct fibrous capsule at the periphery which incorporates focal aggregations of lymphoid tissue and in places joins up with surviving remnants of splenic trabeculae. The free surface of the nodule is covered by a continuous layer of mesothelium. The appearance of these nodules suggests that they represent fragments of spleen implanted in the peritoneum."

Summary and Conclusions

Splenosis, or autotransplantation of the splenic tissue, may follow rupture of the spleen. This is a rare condition and fewer than 20 cases have been described as a laparotomy finding.

If one is not familiar with the condition it may be mistaken for endometriosis, multiple angiomas, or carcinomatosis. Although usually symptomless in itself, it has produced small-bowel obstruction through adherence of loops of jejunum.

Elective splenectomy should be performed as carefully as possible to prevent accidental rupture, with later recurrence of the disease—for example, congenital haemolytic jaundice—due to autotransplantation of splenic tissue.

A case of splenosis is reported in which the condition was found at laparotomy for haematemesis. In such a case the possibility of confusion with angiomatosis of intestine is mentioned.

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SYSTEMIC LUPUS ERYTHEMATOSUS PRESENTING WITH SULPHONAMIDE HYPERSENSITIVITY REACTION

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The clinical picture of systemic lupus erythematosus (S.L.E.) is now well known and there have been numerous descriptions in the literature (Baehr *et al.*, 1935; Klemperer *et al.*, 1941; Montgomery and McCreight, 1949; Gold, 1951a; Cohen and Cadman, 1953; Dubois, 1953; Gold and Gowing, 1953; Jessar *et al.*, 1953; Harvey *et al.*, 1954; Baehr, 1955); the pathological changes too have been often described (Klemperer *et al.*, 1941; Harvey *et al.*, 1954). Treatment with cortisone or corticotrophin (A.C.T.H.) is the first measure which has been shown to exert a significant effect on the course of the disease, though it is less certain how it affects the ultimate prognosis (Carey *et al.*, 1950; Soffer *et al.*, 1950, 1952, 1954; Haserick *et al.*, 1951; Dubois *et al.*, 1952; Johnson and Meyer, 1952; Shearn and Pirofsky, 1952; Haserick, 1953; Harvey *et al.*, 1954).

On the other hand, the aetiology of S.L.E. remains obscure. Three factors seem to be concerned: constitutional factors, including sex and age, are