Medical Memoranda

Spontaneous Rupture of the Spleen

A case of spontaneous rupture of the spleen complicating localized perforation of a gastric ulcer is here reported. No previous case has been found in the literature.

CASE REPORT

A man aged 74 was admitted on November 18, 1952. Eleven hours previously he had been seized with sudden severe pain in the left upper abdomen. This quickly spread all over the abdomen and was accompanied by pain in the tip of the left shoulder, weakness, and a "giddy feeling." There was no vomiting. He completely denied any injury, fall, or unusual occurrence. He had had indigestion, rather suggestive of ulcer, for many years, and also bronchitis.

On examination he resented movement and was obviously in pain. His pulse was 80, of good volume; blood pressure 170/90; no appreciable cardiac disease. Moist sounds were heard at base of left lung. The abdomen was generally tender with rigidity but more especially in the upper half. Bowel sounds were present. There was no reduction of liver dullness. He had slight tenderness per rectum.

He was regarded as a case of perforated ulcer, but if the history had given reasonable excuse in the way of trauma he would have fitted the picture of ruptured spleen.

Operation.—The abdomen was found to be full of blood and a mass of clot was present under the left diaphragm. On removal of the clots, a thickening was felt in the greater curvature of the stomach, opposite the spleen, which was large, soft, and ruptured. It was found that a large subcapsular haematoma had completely separated the spleen from its capsule, and both were ruptured. On splenectomy, clot was noticed in the veins only. The gastric lesion was an ulcer with thickening just over 1 in. (2.5 cm.) diameter, and a perforation was seen in a wad of omentum adherent to it. The whole area was inflamed and friable, and the appearances suggested that the perforation had been walled off by the omentum and the area of the splenic hilum. The perforation was confirmed by forceps passing direct into the stomach through it. Simple closure was performed. There were no glands, the liver was clear, and the ulcer was not obviously malignant. The abdomen was closed with drainage.

He received 3 pints (1.7 litres) of blood, and progressed very satisfactorily apart from a resuture of the wound on November 29. A blood count on November 19 showed: W.B.C., 35,000 (86% polymorphs)—simple hypochromic anaemia. Later counts still showed no evidence of specific dyscrasia in the peripheral blood. A barium meal on December 31 showed an ulcer crater high up on the greater curvature of the stomach.

The pathologist reported that no specific splenic disease was present, but there was general congestion and two veins were partially occluded by thrombus. The patient was discharged home on January 12, 1953, symptom-free, and has remained so on an ulcer diet. A barium meal on June 5 showed no evidence of the ulcer.

COMMENT

There is insufficient pathological evidence from the specimen to assess the time interval between the perforation and the splenic rupture. The sequence of events would appear to have been: (1) perforation of the ulcer with immediate walling off by omentum in the region of the hilum of the spleen; (2) local inflammation and thrombosis of the splenic veins with swelling and congestion, leading to a subcapsular haematoma; (3) rupture of the subcapsular haematoma, probably by twisting or some other trivial injury, marking the onset of the clinical picture. On further inquiry after recovery the patient stated that shortly before the onset he had assisted his invalid wife to move and

change her position, as he had previously had to do on many occasions, but he did not attach any significance to this himself.

It is probable that the onset of the majority of spontaneous splenic ruptures is due to slight or unnoticed injury, trivial, yet effective enough on a spleen which is enlarged, soft, or otherwise rendered susceptible by disease. Coughing and vomiting (Moore and Chapman, 1951), sneezing, twisting, lifting, diarrhoea, defaecation, etc., as quoted by Hershey and Lubitz (1948), have been regarded as sufficient trauma. In some reported cases of spontaneous rupture of the normal spleen, ruptured subcapsular haematoma were found. It seems likely here that the liability to very easy ("spontaneous") rupture has been produced by the presence of a silent subcapsular haematoma from some previous cause. In malarial cases of spontaneous rupture, very trivial injury acting on a silent subcapsular haematoma has been regarded as not uncommon (Hershey and Lubitz, 1948).

Spontaneous rupture of the spleen is difficult to diagnose, and even the broader diagnosis of internal haemorrhage is by no means usually made. The analysis by Hershey and Lubitz (1948) of 65 published cases with a malarial basis shows that the diagnosis was made only eight times and was suggested in two others. The evidence of internal haemorrhage in the clinical picture may vary from nil to gross. Pain in the tip of the shoulder in an acute case should arouse strong suspicion of blood in the peritoneal cavity. This sign was present in all of three recent cases of ruptured spleen that I have encountered, but unfortunately it is not at all a constant sign. Zabinski and Harkins (1943) reported it occurring in only 19 out of 68 cases of ruptured spleen, and Hershey and Lubitz (1948) as present in only 6 of 65 cases. Ballance's sign, allegedly specific for rupture of the spleen, was reported in only one of these 65 cases.

I wish to thank Mr. John Talbot for the opportunity of dealing with this case.

BLAIR A. THOMPSON, M.B., F.R.C.S.Ed.,
Senior Surgical Registrar, Harold Wood Hospital, Essex.

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Rupture of the Splenic Artery Complicating Pregnancy

Aneurysm of the splenic artery is not unduly rare. In 1942 Sherlock and Learmonth collected 125 cases, and in 1947 Cosgrove, Watts, and Kaump stated that approximately 144 cases had been described. They pointed out that in the younger age groups females outnumbered males. It is not surprising, therefore, that the condition should occasionally be associated with pregnancy. Cases in which such an association occurred have been recorded fairly often, and Chalmers (1949) gives an excellent review of the subject. He points out that only two cases are recorded as having survived—namely, those of MacLeod and Maurice (1940) and of Gillam (1942). Chalmers states that the prognosis is poor because unawareness of the condition leads to difficulties in diagnosis and delay in treatment.

In some of the cases mentioned above no aneurysm was demonstrated although the artery had ruptured. Gillam (1948) states: "It seems probable that the splenic artery is liable to have weak spots, perhaps congenital in origin, where rupture with or without previous aneurysm formation is prone to occur."

The case presented below, in which no aneurysm was present, is reported in response to Chalmers's (1949) appeal that all cases should be recorded, to bring the condition to the notice of the profession.

CASE HISTORY

The patient, a healthy young woman aged 32, was 20 weeks pregnant and had attended her doctor for routine antenatal examinations, at which nothing abnormal was found.

On Christmas Day, 1949, when dusting a picture, she was literally struck down with sudden intense pain in the left hypochondrium. She managed to telephone a neighbour, who helped her to bed. She had another severe attack of pain, vomited once, had a large bowel movement, and lost consciousness for a few minutes. Her pulse was not palpable, blood pressure was not recordable, and the heart rate was extremely slow. She was tender in the left hypochondrium, the uterus was soft and not tender, and there was no sign of external bleeding. Nikethamide was given, and she improved very considerably, the systolic pressure rising to 70 mm. Hg, but the heart rate remained slow at 80 a minute. No abnormality was detected in the heart, lungs, or nervous system.

The pulse rate rose to 130 a minute and a definite fullness became palpable below the left costal margin, the uterus remaining soft and painless. A definite diagnosis of bleeding from the spleen or its pedicle was made.

She was removed to hospital, and as soon as a blood transfusion was running satisfactorily operation was begun although the blood pressure was not recordable.

The abdomen was opened through an upper left rectus-splitting incision. The general peritoneal cavity contained a large quantity of liquid and clotted blood. The spleen was elevated and the lienorenal ligament was felt to tear as the spleen was delivered into the wound. The ligament and the tail of the pancreas were deeply blood-stained, and about one inch (2.5 cm.) from the hilum the splenic artery was bleeding briskly from a lateral defect (see illustration).

Schematic cross-section showing site of rupture into the general peritoneal cavity.

The vessel was clamped proximally, and as bleeding continued, but with diminished vigour, it was also clamped distal to the bleeding-point. There was now an interval to allow the transfusion to overtake blood loss. Splenectomy was rapidly performed, the lesser sac was opened to confirm the absence of blood, and the abdomen was closed with stab drainage to the loin. At the end of the operation the systolic pressure was 70 mm. Hg.

On the 11th day she was spontaneously delivered of a macerated male infant. She was discharged on the 17th day, after an uninterrupted convalescence.

Post-operative blood examination revealed no abnormality in red or white cells, and the Paul-Bunnell reaction was negative. The spleen, which was about twice normal size, was histologically examined, and the only abnormality found was venous congestion.

The patient has remained well, and in 1952 had a normal pregnancy.

DISCUSSION

The initial bradycardia in association with low blood pressure made diagnosis difficult at the outset, but the ensuing tachycardia and the abdominal findings made the diagnosis of haemorrhage from the spleen or its pedicle reasonably certain. Before the onset of tachycardia the

help of a medical colleague, Dr. R. A. Pyper, was obtained in order to exclude coronary thrombosis.

No aneurysm was present, and the site of rupture was not examined histologically. To have removed it would have prolonged the operation and might have damaged the pancreas. The presence or absence of an aneurysm would seem to depend on the degree of weakness in the wall of the artery, and no useful purpose would be served by differentiating between cases with and without an aneurysm.

Primary rupture into the general peritoneal cavity is very unusual and has not previously been reported. The usual sequence of events, according to Sherlock and Learmonth (1942), who studied 125 cases (14 in pregnancy) in which rupture had occurred in 56, is for a retroperitoneal false aneurysm to form. This later ruptures into the lesser sac. The fact that primary rupture, in this case, was into the general peritoneum probably made the diagnosis easier than in previously recorded cases.

Rupture has hitherto been reported only as a complication of the later months of pregnancy, and concealed accidental haemorrhage has frequently been diagnosed and the rupture discovered at operation or necropsy. The severe shock, the death of the foetus, and the tender and possibly rigid abdomen must make such a diagnosis almost inevitable.

Chalmers (1949) states that when the condition occurs "in a pregnancy of over four months' duration the uterus must be emptied first, to facilitate the approach to the bleeding vessel." In the present case a 20-weeks pregnancy did not interfere in the slightest with the operative manipulations.

Ligation of the artery on both sides of the rupture would almost certainly have sufficed in the present case, but splenectomy presented no difficulty and ligation was not considered. Chalmers (1949) and Lennie and Sheehan (1942) state that after the vessel is ligated the spleen may be left to undergo aseptic atrophy. The occurrence of subsequent atrophy would depend on the site of the ligatures. In the present case the spleen would have received a good blood supply through the gastric branches. Aird (1946) describes a case of severe bleeding from a splenic artery eroded by a peptic ulcer. The splenic artery was ligated, and even after a subsequent gastrectomy there were no signs of splenic infarction.

SUMMARY

A case of spontaneous rupture of the splenic artery occurring in pregnancy is described.

The primary rupture was into the general peritoneal cavity. This contributed to the patient's recovery because diagnosis was not delayed and treatment was instituted immediately.

Although the uterus was enlarged to 20 weeks' pregnancy there was no necessity to empty it before attacking the bleeding-point.

Ligation of the splenic artery proximal to its gastric branches will not result in infarction of the spleen.

We gladly acknowledge our indebtedness to Dr. W. Bingham for the anaesthesia and for resuscitation of the patient; Dr. R. A. Pyper, who saw the patient soon after the onset of symptoms and confirmed the absence of coronary disease; and to Dr. W. Vint for the histological examination of the spleen. One of us (H. W. G.) is indebted to Mr. W. W. Bassett for the opportunity of treating this patient in Lurgan and Portadown Hospital.

H. W. GALLAGHER, M.B., F.R.C.S.Ed.,
Consultant Surgeon, Banbridge Hospital.

K. HUDSON, M.B., M.R.C.O.G.,
Lately Consultant Obstetrician and Gynaecologist,
Banbridge and Lurgan and Portadown Hospitals.

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