Medical Memoranda

Ruptured Splenic Arterial Aneurysm during Parturition

Berger, Forsee, and Furst (1953) state that only 152 cases of aneurysm of the splenic artery have been reported in the literature since Crisp's description in 1847. They also note that prior to rupture the surgical mortality rate is approximately 15%, whereas after rupture the mortality rate approximates 100%.

Sheehan and Falkiner (1948) state that of the cases of splenic arterial aneurysm so far recorded in patients below the age of 45, 41 were in women. Furthermore, 23 of these 41 women were pregnant, and rupture nearly always occurred at seven to nine months' gestation. These authors analysed 163 routine obstetric necropsies and found that in the three following clinical conditions the spleen was commonly enlarged in the second half of pregnancy (weighing more than 200 g.): (1) severe anaemia of pregnancy, (2) accidental haemorrhage of the abruptio type, and (3) puerperal thrombophlebitis or gross septic endometritis. They considered that this splenic enlargement, with the presumptive changes in splenic blood supply, might be a predisposing factor in rupture of splenic arterial aneurysms in pregnancy. It is interesting to note that eclampsia and other toxaemias of pregnancy did not appear to be associated with pathological increase in the weight of the spleen.

In view of the rarity of ruptured splenic arterial aneurysm the following case is recorded.

CASE REPORT

The patient was a primigravida aged 22. Her expected date of delivery was October 11, 1953. Early in pregnancy the haemoglobin was 93%, and the Kahn reaction was negative. When she was 14 weeks pregnant the blood pressure was 140/70 mm. Hg and there was no albuminuria. No signs of pre-eclamptic toxaemia were present until she was 37 weeks pregnant, when the blood pressure rose to 160/110; there was no albuminuria at this time. At the 39th week she developed slight oedema; the foetus presented by the vertex in the left dorso-anterior position, and the foetal heart was heard. The head was not engaged, but could be pushed through the pelvic brim. The pelvic shape and measurements were normal. Her weight had increased by 23 lb. (10.4 kg.) in 16 weeks, and a trace of albumin was found in a catheter specimen of urine.

On admission to hospital on October 6 she looked well and had no complaints; the physical signs were as already noted. She was given amylobarbitone, ¼ gr. (50 mg.) t.d.s. and 3 gr. (200 mg.) at night. At 6 p.m. surgical induction was performed (hindwaters). At 7.30 p.m. uterine contractions were regular and strong, and the cervix was dilated two fingerbreadths, with the head in mid-cavity. Next day, at 1 a.m., the cervix was fully dilated. At 1.30 a.m. the mother's pulse was 88, the foetal head was advancing, and the foetal heart rate was 130 beats a minute. Uterine contractions were strong and occurred at two-minute intervals. At 1.40 a.m. a stillborn male infant was delivered. The placenta and membranes were expelled complete at 2 a.m.

At 6.30 a.m. the patient suddenly collapsed, the pulse became imperceptible at the wrist, and her blood pressure fell to 80/50; she was pale, sweating, and cyanosed. Restlessness and air hunger were pronounced, and there were no abnormal signs in the thorax or abdomen. Resuscitative measures were of no avail, and she died at 7.20 a.m.

Post-mortem examination of the baby revealed complete atelectasis of both lungs, with petechial haemorrhages in the serous membranes. The cause of death was intrauterine asphyxia.

Post-mortem examination of the mother showed a well-nourished very pale young woman, with no obvious oedema. The heart showed slight hypertrophy of the left ventricle, and the myocardium appeared normal, but there were a few subendocardial petechial haemorrhages. In the greater sac of the peritoneal cavity 2 pints (1,140 ml.) of fluid blood was found, and in the lesser sac there was a similar amount of non-laminated clotted blood. On dissecting out the splenic artery an aneurysm was found, ½ in. (1.3 cm.) in diameter, 1 in. (2.5 cm.) from the splenic hilum. There was considerable extraperitoneal extravasation of blood in the region of the pancreas. The haematoma had finally ruptured into the lesser sac. The spleen was collapsed but healthy; it was not weighed. The kidneys showed marked cloudy swelling of the cortex with partial obliteration of the cortical pattern. The anterior lobe of the pituitary gland was enlarged. Histological examination of the aneurysm revealed marked degeneration and fragmentation of both internal and external elastic laminae and thickening of the tunica media due to oedema and mucoid degeneration.

Probably the raised blood pressure produced by the pre-eclamptic toxaemia and the physical efforts in the second stage of labour precipitated the rupture. If this was the case, then, as in other reported cases, there was a latent interval between the rupture of the aneurysm and the final collapse, when the haematoma burst into the peritoneal cavity.

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Agranulocytosis Due to "Iodolysin"

"Iodolysin" is a drug used, mainly in general practice, in the treatment of minor rheumatic diseases. It consists of allylthiourea ethyl iodide, containing 43% allylthiourea and 47% of iodine. The purpose of this report is to draw attention to the occurrence of agranulocytosis as a side-effect of iodolysin therapy.

CASE REPORT

A 57-year-old woman was admitted to hospital on July 28, 1954, complaining of drowsiness, lack of strength, and frontal headaches. Her daughter stated that she had deteriorated mentally in the past year; her memory for recent events was poor, and she had become incontinent of urine. She had suffered from "rheumatism" in her legs for the past few months, for which her doctor had given her, on July 8, 48 iodolysin tablets, one to be taken twice a day. These she had taken as instructed. Three days before admission she had become febrile. On the day of admission she was unable to walk and travelled to the hospital by taxi.

On examination she was drowsy, delirious, and uncooperative. Her tongue was dry and she had several large boils on her chest with considerable surrounding cellulitis. Her temperature was 102.4° F. (39.1° C.), pulse rate 140, blood pressure 160/100. She had auricular fibrillation and slight ankle oedema. Her respiratory rate was 24 a minute.

Within four hours her temperature rose to 105° F. (40.6° C.). Her blood pressure fell to 140/90. Respirations were now 40, with crepitations at both lung bases. A provisional diagnosis of staphylococcal septicaemia was made, and she was given 500,000 units of crystalline penicillin intramuscularly three-hourly, 1 g. of streptomycin at once and 0.5 g. six-hourly; 1 mg. of digoxin was also given intravenously. A white-cell count at this time showed only 1,000 leucocytes per c.mm. The haemoglobin was 58%; blood urea, 68 mg. per 100 ml. Blood culture taken five hours after starting antibiotic treatment was sterile.