meal. Blood was present in one specimen. The sugar tolerance curve for hepatic function showed a fasting level of 80 mg.% and a rise of blood sugar after 50 g. of dextrose to 200 mg.%, followed by a lagging fall. There was no glycosuria. The E.S.R. was 31 mm./hour (Cutler technique).

Ten days after admission the patient passed a stool consisting of a large amount of red clotted blood. Proctoscopic examination revealed no abnormality. After this haemorrhage the blood count was: r.b.c. 2,400,000, w.b.c. 7,000, Hb 20%, colour index 1.1. The patient was free of abdominal pain, vomiting, and diarrhoea, and felt better, but showed some oedema of the feet. She was given 5 ml. of liver extract intramuscularly daily, and received two blood transfusions of 250 ml. each on successive days. There was no reticulocyte response to the liver therapy after nine days. She became somnolent and died five weeks after admission.

In view of the long history, the macronormoblastic bone marrow, the lower bowel haemorrhage, the oedema, and the macrocytic anaemia which failed to respond to liver, she was considered a case of post-necrotic fibrosis of the liver.

At necropsy a large pedunculated neoplasm, fungating and foul-smelling, was found arising from the lesser curvature and posterior wall of the stomach and infiltrating the left lobe of the liver, which was in contact with it. There were no other deposits in any part of the body. The liver itself was large and fatty. Microscopic examination of the mass revealed it to be a leiomyosarcoma.

I am indebted to Dr. Dormer, medical superintendent of King George V and Springfield Hospitals, for permission to publish this case.

ELIZABETH GREATHEAD, M.B., Ch.B.,
Medical Officer, Springfield Hospital, Durban.

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Hydatid Cyst and Cirrhosis

The following is a case of hydatid cyst associated with gross cirrhosis of the liver, an association which has rarely been reported.

CASE REPORT

A woman aged 68 was admitted to Edgware General Hospital on Sept. 27, 1947. She had been well and on holiday until that morning, when she vomited a small quantity of blood; during the day she vomited blood several times, in all about half a pint (284 ml.). She was seen by her doctor about 7.45 p.m. and had difficulty in giving her history, as she seemed dazed. She received an injection of morphine and was transferred to hospital. On arrival at 9 p.m. she was semi-conscious and unable to give any history. Her relatives could add little to the above account except to say that she had had two abdominal operations in early life, the first of these at 13 years, and a third in 1931. The only notes available concerning the last stated: "Laparotomy—hydatid cyst." In 1944 she had a haematemesis from which she entirely recovered. She had lived all her life in Lancashire except for pilgrimages to Lourdes, and she was not an alcoholic.

Examination showed a semi-conscious woman, moderately pale and sweating slightly, who was vomiting blood clot. A few purpuric spots were present on the limbs, and on the abdominal wall there were enlarged veins and two upper abdominal laparotomy scars. The liver edge was 2 in. (5 cm.) below the costal margin; it was smooth and hard, with one large rounded eminence. A firm spleen was palpable 1 in. (2.5 cm.) below the costal margin. She also showed bilateral corneal opacities, depressed tendon reflexes, and a right extensor plantar response. The pulse rate was 110 and blood pressure 100/60. She was afebrile and free from jaundice. There were no other significant findings.

On admission her urine contained protein but was otherwise normal. Blood count showed: haemoglobin, 68%; red blood cells, 3,360,000 per c.mm.; white blood cells, 14,400 per c.mm. Plasma proteins: Total, 5.75 g.%; albumin 3.4 g.%, globulin 2.35 g.%. The cerebrospinal fluid was normal in all respects. She was transfused with one litre of blood and 800 ml. of 5% glucose, but failed to improve, and died 36 hours after admission.

Post-mortem Examination (Dr. H. C. Moore).—The body was generally obese. There were large oesophageal varices, and the stomach and intestines contained fresh blood. There was no ascites. The liver weighed 1.95 kg. (normal, 1.2–1.4 kg.) and the right lobe was replaced by a cavity, 18 cm. in diameter, which contained turbid fluid. The walls were composed of a 4-mm. layer of fibrous tissue and compressed liver tissue with a shaggy fibrinous inner surface (see photograph). There was no calcification. The remainder of the liver showed a multilobular cirrhosis with nodules of hyperplastic liver tissue; there were no remnants of other cysts elsewhere in the liver. Gall-bladder and ducts were normal. The spleen was four times its normal size, and firm. Apart from oedema of the lungs there were no other macroscopic abnormalities. Microscopically the liver showed a multilobular cirrhosis. The wall of the cyst consisted of vascular collagenous tissue in which normal bile ducts could still be seen, but no liver parenchyma was present.

The posterior wall of the cyst has been opened; the liver is cirrhotic.

COMMENT

The cause of death appeared to be liver failure due to multilobular cirrhosis, precipitated by a haematemesis from oesophageal varices. Morphine may have slightly hastened death. The large thick-walled cyst which was found at necropsy had existed for many years, and was almost certainly hydatid in origin. Instead of becoming shrunken and calcified after the death of the parasite, as is usual, it had remained distended. Its relationship to the cirrhosis is uncertain.

All authors are agreed on the frequency with which compensatory hyperplasia of the liver cells occurs when part of the liver is destroyed by hydatid disease, but most consider that cirrhosis and hydatid disease occur together merely by coincidence. Dew (1928), for example, states that in view of the great difficulty of excluding other causes it seems that the existence of a true hydatid cirrhosis remains to be proved. A biliary cirrhosis due to obstruction of the bile ducts by hydatid material has been described. It is hard to believe, however, that in the above case multilobular cirrhosis and a thick-walled cyst were present together only by chance. Stevens (1901) described a similar case—that of a man aged 63—in which two cysts were present. Both in his and in my case the cysts had probably existed since childhood, and their great age and survival after the death of the parasite may have had some bearing on the development of cirrhosis.

I am indebted to Dr. G. H. Jennings for his advice and to Miss M. H. Shaw for the photograph.

C. P. SILVER, B.M., M.R.C.P.,
Lately Senior House-Physician,
Edgware General Hospital, Edgware.

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