Medical Memoranda

Liver Abscess and Polyarteritis Nodosa

A case presenting with a large liver abscess, and multiple infarcts due to polyarteritis nodosa, is worth recording.

CASE REPORT

A man aged 71 presented in September, 1950, with a four-months history of gnawing pain in the right hypochondrium, lassitude, loss of weight (2 st.—12.7 kg.), pallor, anorexia, nausea, and vomiting. He had had dysentery in India in 1918, and subsequent irregularly repeated attacks of right-sided abdominal pain until 1940.

The patient was a sallow thin man, afebrile, weight 8 st. (50.8 kg.). A firm tender liver extended into the right iliac fossa. There were signs of right basal pleurisy. B.P. 175/95. Heart and C.N.S., N.A.D. Hb, 9.5 g.; R.B.C., 3,200,000. P.C.V., 30%; W.B.C., 12,000 (polys. 78%); E.S.R. (Westergren), 70 mm. first hour. Trace of proteinuria. Blood urea, 31 mg. per 100 ml.; cholesterol, 131 mg. per 100 ml.; total proteins, 8.2 g. % (albumin 2.8 g., globulin 5.4 g.); bilirubin, 0.4 mg. per 100 ml.; thymol turbidity, 4 units; colloidal gold, negative; alkaline phosphatase, 20 units. Casoni test, negative. Hydatid C.F.T., negative. Faeces: no ova, cysts, amoeba, or occult blood. Chest x-ray examination showed considerable elevation of the right hemidiaphragm.

PROGRESS

Diagnostic liver puncture on December 22 revealed turbid brown fluid. After a course of emetine hydrochloride, 10 gr. (0.65 g.) i.m. in 10 days, 1,500 ml. of similar fluid was aspirated from the right lobe of the liver. The fluid was sterile on culture (aerobic and anaerobic) and contained fat globules, macrophages, and amorphous debris only. Subsequent x-ray examination of the abdomen showed a large abscess cavity with a fluid level. His general condition improved after a course of chloroquine sulphate (13.2 g. in divided dosage). Recurrence of pain, weakness, anorexia, and cough necessitated readmission in April, 1951. The liver remained enlarged and tender three fingerbreadths below the subcostal margin. Hb, 85%; E.S.R. (Westergren), 69 mm. first hour; blood urea, 56 mg. per 100 ml.; B.P. 180/120. Initial symptomatic improvement followed a further course of chloroquine (6.4 g. in 14 days), but he relapsed in May with severe prostrating attacks of pain, vomiting, and rigors. A cerebral episode developed in June with stupor, periodic respiration, papilloedema, headaches, and vomiting. B.P. varied between 225/150 and 190/120. E.E.G. was abnormal. C.S.F.: protein, 100 mg. per 100 ml.; cells, 24 lymphocytes per cubic mm. Presumptive diagnosis was cerebral amoebiasis; courses of emetine (9 gr.—0.58 g.—in nine days) and chloroquine (6.4 g. in 14 days) were followed by improvement clinically and in the C.S.F. findings.

Further enlargement of the liver, however, soon occurred. Prolonged attacks of pain, dyspnoea, pallor, and nausea developed. Attacks increased in severity and duration till pain became constant, with paroxysmal exacerbations. A further course of emetine, 6 gr. (0.4 g.), was given. Further attempts to locate pus were unsuccessful, and the patient died on June 30, 1951.

PATHOLOGICAL FINDINGS

Heart.—Fibrinous pericarditis; weight, 430 g.; left ventricular hypertrophy.

Lungs.—Right lung extensively adherent to right hemidiaphragm.

Liver.—Weight 1,400 g. Two amoebic abscesses were present in the right lobe: one, 20 cm. in diameter, at the

upper extremity, underlying and adherent to the diaphragm; the other, 5 cm. in diameter, at the inferior margin in the midline. The wall of the larger cavity was composed of dense collagen with a ragged lining of necrotic material. No amoebae or organisms were demonstrable. "Iron pigment" was absent, suggesting that an origin from a recently necrotic area of liver was unlikely. The liver appeared fatty. There were large haemorrhagic areas, mainly in the right lobe, sometimes surrounded by paler more solid areas. Other totally pale areas resembling infarcts were also present. The smaller haemorrhagic areas were circular, and appeared to be aneurysmal sacs.

Kidneys.—Weights, 120 and 138 g. Elastosis in the larger and fibrinoid necrosis in the smaller vessels.

Lungs.—No vascular change.

Spleen.—Weight, 130 g.; normal.

Epididymis.—Aneurysmal vessels seen in the right epididymis similar to those in the liver.

Suprarenals.—Vessels of the capsule showed the identical haemorrhagic change observed in the smaller hepatic vessels and in the epididymis.

Hepatic Arteries.—In the portal tracts throughout the liver the small hepatic arteries all had thick walls and occasionally showed hyaline or fibrinoid change. The laminae were reduced, but there was little periarterial inflammatory reaction. In the haemorrhagic areas the arteries were often entirely blocked by a concomitant-intrinsic hyperplasia and had given rise to large areas of hepatic necrosis. In other cases the necrotic arterial vessels had given way with aneurysmal dilatation.

Mesenteric Vessels.—These showed destruction of the medial coat of the arteries with aneurysm formation.

COMMENT

It is probable that this patient had a long-standing amoebic abscess and developed polyarteritis during treatment. Atypical cases of amoebic abscesses have been diagnosed by liver aspiration (Van Raalte, 1949). The presence of polyarteritis nodosa was not diagnosed during life. There were several indications to which we should have paid more attention. The presence of proteinuria and hypertension was not readily explained, and the liver-function tests were persistently abnormal, which, as Sodeman (1950) pointed out, is very unusual in amoebiasis of the liver.

Pass (1935) showed that infarcts of the liver occur in polyarteritis nodosa, frequently becoming converted into abscesses which are difficult to distinguish from amoebic or other abscesses. Some cases of periarteritis nodosa with hepatic infarcts run a course similar to this case, including pain in the liver and even collapse of the right lower lobe of the lung (Davson *et al.*, 1948, Cases 10 and 11).

Our patient's attacks of pain, with circulatory collapse and dyspnoea appearing during the final illness, had been believed to be due to the tracking of an amoebic abscess. They were due, however, to multiple infarction. The episode of drowsiness and headache was presumably hypertensive encephalopathy. There was no post-mortem evidence of amoebic encephalitis.

The polyarteritis may have been caused by hypersensitivity to one of the many drugs given. These included chloroquine, emetine, penicillin, chloramphenicol, "amytal," and aminophylline.

IAN C. GILLILAND, M.D., M.R.C.P.,
Temporary Lecturer and Physician, Dept. of Medicine,
Postgraduate Medical School of London.

GEOFFREY C. MANNING, M.B., B.Ch.,
Medical Registrar, Middlesex Hospital, London.

REFERENCES

- Davson, J., Ball, J., and Platt, R. (1948). *Quart. J. Med.*, 17, 175.
Pass, I. J. (1935). *Amer. J. Path.*, 11, 503.
Sodeman, W. A. (1950). *Amer. J. trop. Med.*, 30, 141.
Van Raalte, H. G. S. (1949). *Ibid.*, 29, 881.