Hyperlipidaemia diagnosed at lumbar puncture

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Summary

A patient presenting with subarachnoid haemorrhage and high lipid concentrations in the cerebrospinal fluid (taken at lumbar puncture), who was later shown to have type V hyperlipidaemia is described. This case, so far as can be ascertained by the authors, is the first report of hyperlipidaemia being diagnosed from CSF examination.

Case report

A 47-year-old Indian woman domiciled in England was on holiday when she developed sudden severe occipital headache, vomiting and epigastric pain which radiated to her left loin, back and left leg. She gave a 3-month history of similar but less severe pains. Twenty μg ethinyl oestradiol daily had been prescribed for many months for menopausal symptoms.

On examination she was restless, her abdomen was painful, there was no abdominal distension, bowel sounds were normal and on palpation the abdomen was soft with no hepatosplenomegaly and no other masses palpable. Her optic fundi showed no papilloedema but a peculiar fluorescence of the blood vessels (later identified as lipaemia retinalis). Her pulse rate was 150/min, BP 140/80 mmHg and temperature 38°C. It was suspected that she had had a subarachnoid haemorrhage and at lumbar puncture the CSF was initially white and creamy, but became bloodstained and was under a pressure of 180 mm CSF. A second lumbar puncture 9 days later after a further severe headache showed a uniformly bloodstained fluid which, on standing, showed excess lipid.

Further investigations showed her serum was lipaemic; the serum sodium was 120 mmol/l; potassium, 3.1 mmol/l; chloride, 85 mmol/l; bicarbonate, 19.7 mmol/l; blood urea 3.7 mmol/l (22 mg/100 ml). The serum amylase was 300 u/l (upper limit

FIG. 1. Patient's fasting serum
(a) before
(b) after 500 u. heparin i.v.

of normal); aspartate aminotransferase, 139 mu./ml (normal 10–50); calcium, 1.99 mmol/l (8.0 mg/100 ml); albumin 5 g/l; thyroxine, 211 μ mol/l (normal 58–170). Analysis of her fasting serum lipids showed cholesterol 44.2 mmol/l (1700 mg/100 ml) and triglycerides 69.2 mmol/l (6.12 g/l), electrophoresis showed gross excess of chylomicrons and pre beta lipoproteins. Twenty minutes after an intravenous injection of 500 units of heparin the milky plasma cleared (Fig. 1), the triglycerides increased to 99.2 mmol/l (8.77 g/l) and the electrophoresis showed increased mobility of all lipoprotein fractions.

A diagnosis of subarachnoid haemorrhage and hypertriglyceridaemia with a type V lipoprotein phenotype (Frederickson, Goldstein and Brown, 1978) was made. Treatment with a low fat diet and clofibrate was started and the oestrogen replacement therapy stopped. Four-vessel cerebral arteriography failed to show a source of the subarachnoid bleeding. She was treated conservatively and, when fit, returned to Oxford. In view of the initial symptoms of back pain, a myelogram was performed and showed a block in the lower thoracic cord. At subsequent laminectomy, direct inspection of the spinal cord showed no obvious cause for either the subarachnoid bleeding or the block seen on the myelogram. However, changes due to chronic spinal arachnoiditis were found and assumed to be related to the subarachnoid bleeding as the patient had no history of malfunction or interference with her spinal cord.

Discussion

The finding of a creamy CSF at lumbar puncture in

this patient was disconcerting. The authors have been unable to find any other reports of high lipid concentrations in the CSF. Minute amounts of lipid have been found in the CSF, the concentration is independent of blood levels (Tourtellotte, 1968). The most likely source of the CSF lipid in this case was the subarachnoid haemorrhage, although the site of this bleeding was uncertain. It is interesting to postulate that the high CSF lipid concentration over a long period may have led to the chronic spinal arachnoiditis found in the patient. Her abdominal pain and vomiting, necessitating admission to hospital, could have been due to the hyperlipidaemia (Holt, Aylward and Timbres, 1939) or a mild pancreatitis (Greenberger *et al.*, 1966), but equally the chronic abdominal pains could have been caused by root irritation in the spinal cord.

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