

Short report

Pathology of *Yersinia enterocolitica* meningitis

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SUMMARY *Yersinia enterocolitica*, a gram negative aerobic non-haemolytic bacillus, has been identified as a cause of meningitis only once before and the neuropathological features of *Y. enterocolitica* meningitis have not been reported in the literature. We describe the pathological features of a case of acute fatal meningitis caused by *Y. enterocolitica*, serotype 0:18, in a 47-year-old male alcoholic with cirrhosis. The organism presumably reached the meninges via the blood stream from the abdominal cavity. Intravascular fibrin thrombi, both recent and organising, were associated with multiple foci of necrosis in the brain. In spite of vigorous antibiotic therapy for three weeks the organism was cultured from the brain at autopsy. Among the various infections caused by *Y. enterocolitica*, meningitis appears to be the most difficult to eradicate.

Yersinia enterocolitica, an aerobic, non-haemolytic, gram negative bacillus, has been associated with intestinal illness in school children.¹ An appendicitis-like syndrome of terminal ileitis,² and septicemia has occurred in debilitated adults with cirrhosis and blood dyscrasias.^{2,3} Only one previous case of meningitis due to this organism has been found in the literature.^{4,5} We describe the first case of *Y. enterocolitica* meningitis with pathological studies.

Case history

A 47-year-old white male with a long history of alcohol abuse was admitted to hospital with a two day history of fever, vomiting, jaundice, abdominal distention, and confusion progressing to coma. He had a distended abdomen containing fluid, spider angiomas over the trunk, neck rigidity and bilateral extreme plantar responses. The peritoneal fluid contained 2,050 WBC/mm³ and an amylase of 115 units. There was a leucocytosis of 25 000/mm³ with 90% neutrophils and pronounced toxic granulation. The cerebrospinal fluid was cloudy with an opening pressure of 465 mm water, and contained 10 700 WBC/mm³,

74 RBC/mm³, protein 0.720 g/l, glucose nil. Computerised tomography showed hydrocephalus and periventricular oedema, but no focal lesions. He was treated with systemic and intrathecal gentamicin (120 mg every 8 hours and 5 mg once a day, respectively), systemic penicillin G (2 million units every 2 hours), and chloramphenicol (1 g every 6 hours). The peritoneal fluid and cerebrospinal fluid cultures grew *Y. enterocolitica*, serotype 0:18. Later chloramphenicol was replaced by sulfamethoxazole-trimethoprim due to the persistence of organisms in the cerebrospinal fluid. Subsequent cultures then were negative. Although the patient developed some spontaneous movements towards the end of the second week, his clinical course was complicated by hepatic encephalopathy, thrombocytopenia and gastrointestinal bleeding in the third week, and he died on the 22nd day of his illness.

Pathologic findings

At necropsy there was healed peritonitis, hepatic portal cirrhosis with Mallory bodies in the hepatocytes, esophageal varices, renal focal tubular necrosis, myeloid hyperplasia of bone marrow and bilateral testicular atrophy.

The brain weighed 1320 grams and was moderately swollen without uncal or tonsillar herniation. There was yellowish exudate in the meninges, most prominently over the base of

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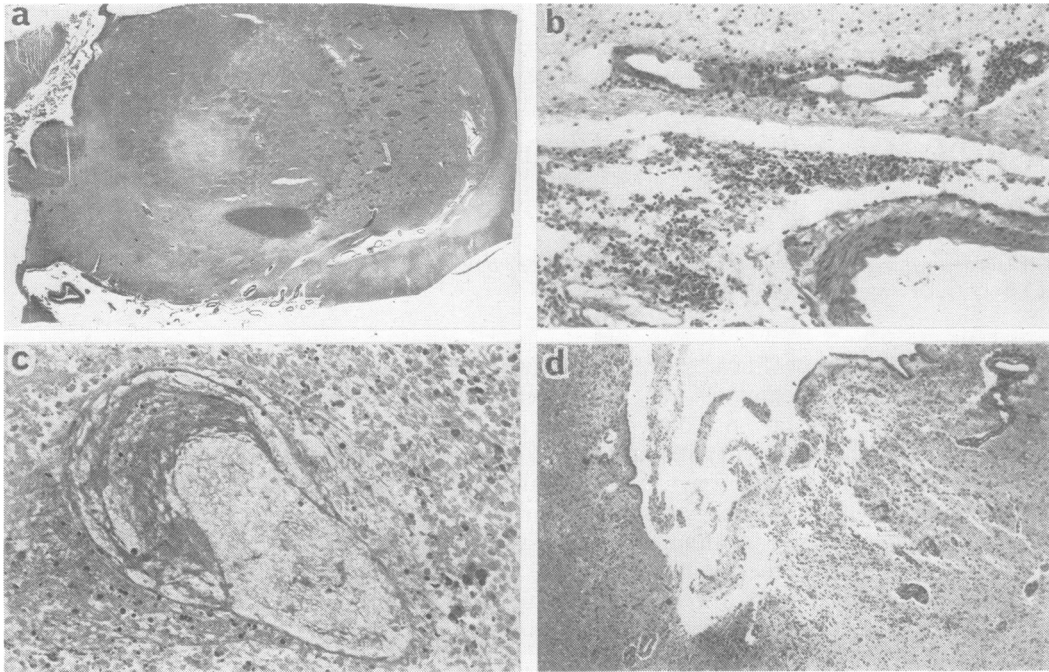


Fig 1 Microscopic appearances in the brain. (a) A focus of necrosis in globus pallidus. (Iron Hematoxylin; Mag $\times 3$). (b) Meningeal lymphoplasmocytic infiltrate and perivascular cuffing. (H & E; Mag $\times 120$). (c) Fibrin thrombus in a blood vessel. (H & E; Mag $\times 180$). (d) Ependymal necrosis, intraventricular exudate and periventricular inflammation. (H & E; Mag $\times 80$).

the brain and the superior and inferior surfaces of the cerebellum. The ventricles were confirmed and contained thick yellowish exudate and adhesions. The occipital horns were filled with soft greenish yellow material. There were multiple 0.5 to 1 cm diameter foci of softening and grey-brown discoloration in the genu of corpus callosum, both caudate nuclei and globus pallidus (fig 1a), left putamen, left internal capsule and basis pontis. Microscopic examination revealed predominantly lymphoplasmocytic exudate in meninges (fig 1b) with a suggestion of increased fibrous tissue. Occasionally small penetrating vessels showed either fibrin thrombi in the lumen (fig 1c), or organising eccentric fibrous intimal lesions. There were large areas of denudation of the ventricular lining with periventricular oedema, gliosis and lymphoplasmocytic perivascular cuffs (fig 1d). The choroid plexus showed necrosis, oedema, acute and subacute inflammation and flattened lining cells. The areas of necrosis in the central nuclei and pons were approximately ten days old. They showed intense macrophage infiltration and peripheral capillary proliferation.

Although the organisms were not demonstrated in the microscopic sections, brain tissue cultures grew *Y. enterocolitica*. The peritoneal fluid was sterile.

Discussion

We believe this patient is the first case of meningitis caused by *Y. enterocolitica* to be examined post mortem. *Y. enterocolitica* can thus cause acute and subacute opportunistic meningitis in debilitated adults. The primary route of entry was presumably from the gastrointestinal tract, the organism reaching the meninges from the blood stream. It is possible that the associated cirrhosis caused failure of hepatic reticuloendothelial cells to sequester the bacilli. In other bacterial meningitides parenchymal necrosis is uncommon, apart from meningitis due to *H. influenzae*.⁶

The difficulty in eradicating the organism from the brain during three weeks of antibiotic therapy may be due to persistence of the organism in necrotic brain foci together with inadequate local antibiotic levels. The findings show that a

negative cerebrospinal fluid culture should not be taken as an indication to stop antibiotic therapy in *Y. enterocolitica* meningitis, which we suggest, should be continued for at least four to six weeks. This prolonged treatment may not be indicated for acute peritonitis by the same organism; this patient's peritonitis was considered cured at necropsy.

References

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