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Flavobacterium meningosepticum as an opportunist

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SUMMARY *Flavobacterium meningosepticum* was isolated from the cerebrospinal fluid of an adult immunodeficient female. In spite of prompt therapy the patient succumbed to the infection. The opportunistic role of the organism is discussed.

The organism *Flavobacterium meningosepticum* owes its name to the yellow colour of its colonies and its initial isolation from the cerebrospinal fluid (CSF) (King, 1959). Several reports of neonatal meningitis have been reported thereafter from various countries, including India. In an addendum to her paper, King (1959) noted meningitis due to this organism in a 69-year-old woman with polycythaemia vera. Subsequently, Lapage and Owen (1973) reported a fatal case from Botswana of a 19-year-old man with meningitis, who was suffering from aplastic anaemia. In general, adults whose immune status was normal before this infection seem to make a good recovery with appropriate antibiotics (Werthamer and Weiner, 1972; Bagley *et al.*, 1976).

The following account reports isolation of this organism from an immunodeficient patient, in whom it was responsible for death.

Case report

A 26-year-old Parsee woman developed chronic renal failure following rapidly progressive glomerulonephritis. She was on maintenance haemodialysis from June 1975 and bilateral nephrectomy was performed on 3 January 1976. She received a kidney transplant but the kidney was rejected and transplant nephrectomy was performed after 10 days. She was maintained on haemodialysis.

Immunosuppressive drugs given during the period of transplant were: prednisolone, 2·5 mg/kg day initially, reduced to 1 mg/kg, and tapered to 5 mg/day after removal of the kidney. The dose of 5 mg/day was continued thereafter. Cyclophosphamide, 2 mg/kg, was also given during the transplant period.

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After two months she developed an intermittent pyrexia (37·6-40°C) together with hepatosplenomegaly. Biopsy of a cervical lymph node showed evidence of tuberculosis, and the following therapy was begun: isoniazid, 300 mg/day; ethambutol, 0·6 g/day; pyrazinamide, 0·5/day; together with pyridoxine, 10 mg/day.

However, the pyrexia continued and she became drowsy. There was no neck stiffness and examination of the CSF obtained by lumbar puncture showed: glucose 3·5 mmol/l, chloride 126 mmol/l with a cell count of 6/mm^3. Culture of CSF was sterile. She became apyrexic and her mental acuity improved, but after a week fever recurred and persisted thereafter, ranging from 37·6° to 40·2°C. She again became stuporous, her level of consciousness worsened, and she did not respond to questioning. Neck rigidity was noted and a repeat lumbar puncture done two weeks after the first showed turbid CSF with glucose 1·5 mmol/l, protein 1·56 g/l, chloride 123 mmol/l, and 175 cells/mm^3 (predominantly polymorphs). Gram-negative bacilli were seen on direct smear of the centrifuged deposit. The WBC count at this time was 7200/mm^3, neutrophils 84%, lymphocytes 10%, eosinophils 2%, and monocytes 4%. Gentamicin, 60 mg iv, and chloramphenicol, 250 mg 6-hourly iv, were started immediately. The CSF culture grew *Flavobacterium meningosepticum*, and erythromycin, 2 g/day, was started after a sensitivity report. She steadily deteriorated and died. Necropsy was refused.

Bacteriological studies

The centrifuged deposit of the turbid CSF received was inoculated on blood-agar, MacConkey agar, nutrient agar, and Sabouraud's agar. The antibiotic

sensitivity was performed by the standardised disc method of Bauer *et al.* (1966). The organisms were identified by conventional techniques (Sonnenwirth, 1970) and the culture was sent to the Centre for Disease Control, Atlanta, Georgia, USA, for serotyping.

Results

The organisms were aerobic, non-motile, Gram-negative rods. The colonies on blood agar were 0·5 to 1 mm in diameter, entire, convex, opaque, and smooth. A bluish sheen appeared in the medium around the colonies. On nutrient agar and Sabouraud's agar the colonies showed distinct yellow coloration, which was enhanced when incubated beyond 48 hours at room temperature. No growth was noticed on MacConkey agar. Biochemical characteristics are summarised in the Table. The organism was found to belong to serotype C and was sensitive to erythromycin (15 μg/l), nalidixic acid (30 μg/l), lincomycin (2 μg/l), and novobiocin (5 μg/l).

Table *Biochemical characteristics of the isolated organism*

Oxidase	+	Triple sugar iron slant/butt. Alk/–	
Catalase	+	Glucose	+
Indole	+ (weak)	Lactose	–
Urea	–	Maltose	–
H_2S	+	Sucrose	–
Aesculin hydrolysis	+	Mannitol	–
Citrate	–	Adonitol	–
Gelatin liquefaction	–	Dulcitol	–

Discussion

In newborns, *Flavobacterium meningosepticum* causes meningitis and septicaemia with a high fatality rate (Cabrera and Davis, 1961; Sugathadasa and Arseculeratne, 1963; Agarwal and Ray, 1971).

In healthy adults without prior debilitating illness, infections by this organism have responded promptly to treatment with appropriate antibiotics (Werthamer and Weiner, 1972; Bagley *et al.*, 1976). In patients with chronic illness or low immune status, such as aplastic anaemia, opportunistic infection with this organism has been fatal (Lapage and Owen, 1973).

This patient was on high doses of immunosuppressives and subsequently she developed tuberculous lymphadenopathy. Although the immunosuppressive drugs were withdrawn early following rejection of the kidney, her condition continued to be poor. The CSF drawn at the first lumbar puncture was sterile, but a fortnight later she developed meningitis due to *Flavobacterium meningosepticum*. The organism might have reached the meninges through the blood stream or it might have been introduced directly into the CSF during the first lumbar puncture. Blood cultures were not done during the terminal illness but it is thought that direct entry was more likely.

Flavobacterium meningosepticum is ubiquitous in the hospital environment (Olsen, 1967) and has been found even in antiseptic solutions such as chlorhexidine (Coyle-Gilchrist *et al.*, 1976). In this case an intense search for the source of infection was not made. Opportunistic infections are common in immunosuppressed patients, and one would expect this organism to be a frequent cause of terminal disease in hospitals with transplant programmes. Curiously, we have not found any published reports of infection with this organism in immunosuppressed patients.

It cannot be adequately stressed that every possible precaution should be taken to deny these opportunist organisms entry into immunosuppressed patients. Any procedure which offers them access into the tissues, even such simple procedures as intravenous infusions, should be done with the utmost attention to asepsis.

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