Meningitis Due to *Haemophilus influenzae* Type e

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*Haemophilus influenzae* type e was isolated from the cerebrospinal fluid and blood of an 8-month-old child with meningitis.

*Haemophilus influenzae* type b has been reported to cause over 95% of serious *H. influenzae* infections in young children (7). To our knowledge there have been only three previous references to serious infection caused by *H. influenzae* type e. The type e strains were isolated from joint fluid from a case of septic arthritis (2), from cerebrospinal fluid (CSF; 5), and from the tracheae of two children who died from pneumonia (8).

We recently isolated a strain of *H. influenzae* type e from CSF and blood of an 8-month-old female. She had a history of an upper respiratory infection 3 days before admission. Upon hospital admission the patient had a temperature of 104°F (40°C) and was semicomatose. The CSF contained 278 polymorphonuclear leukocytes, 31 monocytes, and 368 erythrocytes per mm³. The CSF glucose was 83 mg/dl and protein was 48 mg/dl. Her admission leukocyte count was 19,400 cells/mm³. The differential showed 26 bands, 52 segmented neutrophils, 15 lymphocytes, and 7 monocytes. The blood glucose level was 207 mg/dl. A Gram stain of CSF revealed a moderate amount of pus cells and a few gram-negative cocobacilli. She was started on ampicillin and chloramphenicol. When susceptibility studies were furnished, the chloramphenicol was terminated. The ampicillin was continued intravenously until the 5th hospital day, when a purpuric rash developed. A repeat lumbar puncture was negative for cellular constituents, and no organisms were isolated. The ampicillin was discontinued and oral chloramphenicol was started. The patient remained afebrile and was released on the 14th hospital day.

The organism was isolated from blood inoculated to brucella broth with 0.05% sodium polyanethol sulfate, with vacuum and CO₂ (Pfizer). The *H. influenzae* from CSF was isolated on chocolate agar (BBL). The organism exhibited typical characteristics of *H. influenzae*, i.e., gram-negative pleomorphic rod, fastidious growth requirements, and requirement for X and V factors (4). Serological typing by Quellung reaction and slide agglutination showed both isolates to be type e. Antiserum was produced by the Massachusetts Department of Public Health, Division of Biologic Laboratories, Boston. The minimum inhibitory concentration of ampicillin was 1.0 µg/ml, as determined by the method of Jorgensen and Lee (3).

A report by Dawson and Zinnemann showed a high incidence of *H. influenzae* type e nasopharyngeal carriers, but, at the same time, no meningitis cases caused by type e were reported (1). We have no studies to demonstrate the frequency of the six *H. influenzae* serotypes in our community. Whereas efforts are presently directed toward development of an effective *H. influenzae* type b vaccine (6), this case would indicate the possibility of other serotypes of *H. influenzae* causing serious infections. Our patient did not exhibit any obvious defects that would have predisposed her to infection with this strain. As there has been only one other reported case of type e meningitis, it probably is a rarely encountered pathogen.

The serotyping was performed by the Massachusetts Department of Public Health Bacteriology Laboratory.

LITERATURE CITED