Haemophilus influenzae Type E Meningitis and Bacteremia in a Healthy Adult

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Abstract

Haemophilus influenzae type b causes more than 95% of serious H. influenzae meningitis. H. influenzae type e (Hie) has been implicated in a few cases of meningitis. Here, we present an adult Saudi patient with Hie meningitis and review the literature. The patient, a 19-year-old Saudi male with no significant past medical history, was noted by his family to have some changes in his mentation, confusion and refusal to eat; subsequently, he became unresponsive. Cerebrospinal fluid and blood culture grew Hie. The patient was treated with intravenous ceftriaxone with full recovery.

Key words: Haemophilus influenzae, meningitis, invasive Haemophilus influenzae type e

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Introduction

Most upper respiratory tract strains of Haemophilus influenzae are non-encapsulated and therefore non-typable. Most severe infections are caused by H. influenzae strains belonging to the capsular serotype b (Hib). H. influenzae type b causes >95% of serious H. influenzae meningitis (1). Meningitis caused by H. influenzae other than type b has been infrequently reported (2). H. influenzae type e (Hie) had been implicated in a few cases of meningitis in children (3) and in a few adults (4, 5). This paper describes a case of meningitis caused by H. influenzae type e (Hie) in an adult Saudi patient. A review of the literature is also included.

Case Presentation

The patient was a 19-year-old Saudi male with no significant past medical history. His family noted the following: the patient had changes in mentation, confusion, refused to eat, and soiled toilet seats with fecal material, which was unusual for the patient. The patient remained asleep for many hours and was unresponsive to verbal commands. The patient was taken to the Emergency Room. On examination, he was febrile with a temperature of 40.5°C, blood pressure 110/70, and the pulse was regular at 100/minute. Pupils were reactive and equal in size. His neck was severely rigid and he had positive meningeal signs including Kernig and Brudzinski signs. Ocular movement to doll’s eye maneuver, gag reflex and corneal reflexes were present. Motor examination revealed no lateralizing findings. He did not obey commands or follow verbal questioning but he was awake with his eyes open. Deep tendon reflexes were symmetrical and he had bilateral upward pointed toes. He was initially started on ceftriaxone (2 gm IV q12hours) and acyclovir (10 mg/kg IV q8hours).

Laboratory data revealed a white blood cell (WBC) count of 19,700/mm$^3$ with 35% bands. Renal panel and electrolytes were normal. Serum glucose was 158 mg/dl. An urgent computerized axial tomography (CAT) scan of the brain revealed subtle diffuse abnormalities of the parenchyma suggesting minimal changes of the gray/white matter differentiation indicative of meningitis. Cerebrospinal fluid (CSF) was cloudy with 944/mm$^3$ RBCs, 5500/mm$^3$ WBCs, 86% polymorphonuclear cells, glucose 36 mg/dl, protein 181 mg/dl. CSF Phadebact (agglutination) test was negative for Streptococcus group B, Haemophilus influenzae type b, Streptococcus pneumoniae and Neisseria meningitides anti-
Figure 1. A graphic presentation of the patient’s clinical course from admission to discharge showing the temperature in degrees centigrade (---) and WBC count (△—WBC). CSF, cerebrospinal fluid; Hie, Haemophilus influenzae type e; and GCS, Glasgow coma scale.

gens. Gram stain of the CSF showed Gram negative bacilli. Later, blood and CSF cultures grew Haemophilus influenzae non-type b, β-lactamase negative, and were susceptible to ceftriaxone, ampicillin, chloramphenicol, ciprofloxacin and imipenem.

Acyclovir was discontinued and the patient improved dramatically on ceftriaxone. On the following day, he was awake, alert and oriented. The patient continued to have fever for four days, after which he was afebrile (Fig. 1). The patient completed treatment with intravenous ceftriaxone and was discharged with no neurological deficits. The H. influenzae isolate was sent to the Focus Diagnostics, Inc. (formerly Focus Technologies, Inc.) reference laboratory at Cypress, California for serotyping. The organism was identified as H. influenzae type e.

Discussion

H. influenzae can be classified into non-typable and typable organisms. Typable H. influenzae is further classified according to capsular antigen composition into six capsular serotypes (a to f). The emergence of diseases caused by H. influenzae serotypes other than type b theoretically may follow the decline in the rate of H. influenzae type b (Hib) infection (6). This theory has been disputed by some investigators (7). In one study, Hie was shown to account for around 0.6% of nasopharyngeal carriage in children (8). The data on Hie infection relies mainly on clinical cases of meningitis, pneumonia, and blood stream infection. In a study of 113 cases of invasive diseases due to H. influenzae, 3.3% of the total cases were caused by Hie (9). Hie is thought to be an opportunistic pathogen that causes infections in elderly patients, those with respiratory and liver disease or those with impaired immunity (4, 7). In a study of 26 patients with invasive Hie, 12 patients (46.1%) had underlying conditions (7).

Meningitis due to Hie is also very rare. In a surveillance study from Spain, only one meningitis case was found from a total of 26 cases of H. influenzae serotype e infection (7). In addition to the present case, a total of 13 cases of Hie meningitis were identified from the available medical literature (3-5, 7, 10-16) (Table 1). Of the total Hie meningitis cases, six (43%) were infants, four (28.5%) were children and four (28.5%) were adults. Three patients were described to have an underlying disease. Two patients had CSF leak (5, 15) and one patient had head trauma (4). Similar to the present case, severe meningitis may occur in healthy individuals (7). The clinical features and therapy of Hie meningitis are the same as those for type b H. influenzae disease (11).

The prevalence of antibiotic resistance in Hie varies from one report to another. Similar to a report from Italy (9), the organism in the present case was susceptible to ceftriaxone, ampicillin, chloramphenicol and ciprofloxacin. The resistance rate of Hie isolates in Spain was 61.5% to ampicillin and 11.5% to chloramphenicol (7). Of the reported meningitis cases, ampicillin resistance was described in one case (Table 1) (15). Thus, third generation cephalosporin seems to be adequate treatment for this condition. The initial empiric antimicrobial treatment of meningitis is a combination...
of a third generation cephalosporin (e.g. ceftriaxone or cefotaxime) and vancomycin due to the increasing rates of penicillin resistant *Streptococcus pneumoniae* (17). In our hospital, the rate of penicillin non-susceptible *S. pneumoniae* is about 48% and high-level penicillin resistance is 19.8% of all isolates and 9% of blood isolates (18). However, the CSF gram stain was suggestive of *H. influenzae* (showing gram negative bacilli/coccobacilli) and thus vancomycin was not added empirically in this patient.

In conclusion, we describe a Saudi adult patient with Hie meningitis. The patient had no underlying diseases and had severe meningitis. He subsequently recovered without any neurological deficits. There is no available data on *H. influenzae* serotypes causing disease in Saudi Arabia. The reporting of invasive diseases caused by Hie is important, especially in countries where Hib vaccine coverage has reached high levels (9).

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### References


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**Table 1. Summary of Reported Cases of *Haemophilus influenzae* type e Meningitis**

<table>
<thead>
<tr>
<th>Case Number</th>
<th>Year of Report</th>
<th>Age/Gender</th>
<th>Underlying Condition</th>
<th>Ampicillin Susceptibility</th>
<th>Outcome</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1976</td>
<td>8 mo/F</td>
<td>NR</td>
<td>Y</td>
<td>No complication</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>1977</td>
<td>4 mo/M</td>
<td>NR</td>
<td>Y</td>
<td>Good</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>1979</td>
<td>16 mo/F</td>
<td>NR</td>
<td>Y</td>
<td>No complication</td>
<td>14</td>
</tr>
<tr>
<td>4</td>
<td>1982</td>
<td>8 y/M</td>
<td>NR</td>
<td>Y</td>
<td>Good</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>1985</td>
<td>22 y/M</td>
<td>CSF leak</td>
<td>NR</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>1987</td>
<td>6 y/F</td>
<td>CSF leak</td>
<td>N (beta lactamase +)</td>
<td>Survived</td>
<td>15</td>
</tr>
<tr>
<td>7</td>
<td>1989</td>
<td>3 mo/M</td>
<td>NR</td>
<td>Y</td>
<td>Good</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>1995</td>
<td>6 y/M</td>
<td>NR</td>
<td>NR</td>
<td>No complication</td>
<td>11</td>
</tr>
<tr>
<td>9</td>
<td>1995</td>
<td>8 mo/M</td>
<td>NR</td>
<td>NR</td>
<td>No complication</td>
<td>11</td>
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<tr>
<td>10</td>
<td>2003</td>
<td>7 mo/M</td>
<td>None</td>
<td>Y</td>
<td>Survived</td>
<td>7</td>
</tr>
<tr>
<td>11</td>
<td>2003</td>
<td>35 y/M</td>
<td>Head trauma</td>
<td>Y</td>
<td>Survived</td>
<td>4</td>
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<tr>
<td>12</td>
<td>2003</td>
<td>33 y/M</td>
<td>None</td>
<td>Y</td>
<td>Survived</td>
<td>4</td>
</tr>
<tr>
<td>13</td>
<td>2004</td>
<td>5 mo/F</td>
<td>None</td>
<td>N</td>
<td>No complication</td>
<td>12</td>
</tr>
<tr>
<td>14</td>
<td>2006</td>
<td>19 y/M</td>
<td>No</td>
<td>Y</td>
<td>No complication</td>
<td>Current case</td>
</tr>
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</table>

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