the Regional Health Agency Indian Ocean (ARS Océan Indien, Délégation de l’île de Mayotte). She has been working on infectious and tropical diseases for many years.

References


Address for correspondence: Emmanuel Belchior, Sante Publique France, 12 Rue du Val d’Osne, Saint-Maurice CEDEX 94 415, France; email: emmanuel.belchior@santepubliquefrance.fr

Haemophilus influenzae
Type a Meningitis in Immunocompetent Child, Oman, 2015

Kiran P. Sawardekar

Author affiliation: Nizwa Hospital, Nizwa, Sultanate of Oman

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Meningitis caused by Haemophilus influenzae type b (Hib) was eliminated in Oman after the introduction of Hib vaccine in 2001. However, a case of H. influenzae type a meningitis was diagnosed in a child from Oman in 2015, which highlights the need to monitor the incidence of invasive non-Hib H. influenzae disease.

Haemophilus influenzae can be encapsulated (serotypes a–f) or unencapsulated, nontypeable (NTHi) (1). By the end of 2014, all countries in the Eastern Mediterranean Region had introduced H. influenzae type b (Hib) vaccine into their immunization programs; in Oman, where it was introduced in 2001, it led to an elimination of Hib meningitis (2,3). However, Hib vaccine does not cross-protect against other serotypes.

A previously healthy 17-month-old girl with G6PD deficiency was admitted to Nizwa Hospital, Nizwa, Oman, in August 2015 with a 1-day history of fever and lethargy and frequent vomiting and refusal of food for 6–8 hours before admission. She had no history of rash, head trauma, drug ingestion, travel abroad, or contact with animals. Her vaccination record was up to date. Her 3 older siblings were healthy. On examination, she was irritable and febrile (temperature 39°C), with tachypnea, tachycardia, and photophobia. On lung auscultation, a few crackles were heard on the right side. The rest of her physical examination, including a bedside undilated fundoscopic examination, was unremarkable. Blood tests, cerebrospinal fluid examination, and neuroimaging studies were conducted (Table). Results of renal and liver function and metabolic screening tests and serum calcium, troponin T, immunoglobulins, and total complement levels were within reference limits. Diagnostic test results were negative for respiratory viruses including influenza A(H1N1) and Middle East respiratory syndrome coronavirus and for herpes simplex virus types 1 and 2. A chest radiograph showed right middle lobe haziness suggestive of pneumonitis.

The patient was treated with intravenous ceftriaxone. Blood culture revealed H. influenzae type a (Hia), which was serotyped by slide agglutination and determined to
Several case studies have documented prolonged clinical courses of Hia meningitis, with sequelae reported in some children (4,5; online Technical Appendix Table, https://wwwnc.cdc.gov/eid/article/23/7/17-0311-Techapp1.pdf). Hia meningitis is strikingly reminiscent of Hib meningitis, manifesting as a serious illness mostly in otherwise healthy children 6–24 months of age (1,4,5). Hia has been reported to be the most virulent among encapsulated H. influenzae after Hib; the genetic structure of virulent Hia strains closely resembles that of virulent Hib strains with respect to the duplicated arrangement of the capsule locus and, in some cases, partial deletion of the IS1016-bexA gene locus (5–7; online Technical Appendix Table). An active hospital-based surveillance study for meningitis during 1996–2007 in Salvador, Brazil, reported that Hia and Hib meningitis occurred mainly among children <5 years of age; case-fatality rates were higher than those for meningitis caused by types e and f and NTHi strains, which occurred in older age groups and tended to have a better prognosis (6). The study observed an association between IS1016-bexA deletion and poor clinical outcome of Hia meningitis.

Since Hib vaccine implementation, concerns have arisen about serotype replacement and emergence of virulent non-b H. influenzae (5,6; online Technical Appendix Table). With documentation of 3 cases (including the case reported here) of Hia meningitis in the Eastern Mediterranean Region within <2 years (8), more than a decade after Hib vaccine implementation, it is crucial to monitor meningitis in children within the region, complemented by laboratory characterization of incoming specimens by molecular methods for rapid, accurate information on all

### Table. Results of sequential laboratory tests and CT scans of the head during the clinical course of Hia meningitis in a child admitted to Nizwa Hospital, Oman, August 2015*

<table>
<thead>
<tr>
<th>Test type</th>
<th>Hospitalization day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td></td>
</tr>
<tr>
<td>Leukocytes, x 10^9 cells/μL†</td>
<td>Adm 21 8.71 10.52</td>
</tr>
<tr>
<td>Neutrophils, x 10^9 cells/μL†</td>
<td>Adm 21 6.04 7.66</td>
</tr>
<tr>
<td>Lymphocytes, x 10^9 cells/μL‡</td>
<td>Adm 21 2.02 2.38</td>
</tr>
<tr>
<td>Platelets, x 10^9/μL¶</td>
<td>Adm + 12 158.30 41.13</td>
</tr>
<tr>
<td>Hemoglobin, g/dL#</td>
<td>10.18 9.30 9.13</td>
</tr>
<tr>
<td>CRP, mg/L**</td>
<td>Adm + 12 79.30 483.40</td>
</tr>
<tr>
<td>Blood culture</td>
<td>Hia NG NG</td>
</tr>
<tr>
<td>CSF</td>
<td></td>
</tr>
<tr>
<td>Leukocytes, cells/mm³</td>
<td>Adm + 12 2,970 390</td>
</tr>
<tr>
<td>Neutrophils, %</td>
<td>Adm + 12 91 10</td>
</tr>
<tr>
<td>Lymphocytes, %</td>
<td>Adm + 12 90 88</td>
</tr>
<tr>
<td>Protein, mg/dL††</td>
<td>Adm + 12 190.79 100.34</td>
</tr>
<tr>
<td>Glucose, mmol/L††</td>
<td>Adm + 12 1.13 2.24</td>
</tr>
<tr>
<td>Glucose CSF:blood</td>
<td>Adm + 12 0.26 0.56</td>
</tr>
<tr>
<td>Gram stain</td>
<td>Adm + 12 NM NM</td>
</tr>
<tr>
<td>Culture</td>
<td>Adm + 12 NG NG</td>
</tr>
<tr>
<td>CT scan of the head</td>
<td></td>
</tr>
<tr>
<td>Unremarkable</td>
<td></td>
</tr>
<tr>
<td>Mild ventricular dilation with bilateral subdural effusion</td>
<td></td>
</tr>
</tbody>
</table>

*Adm, at admission; Adm + 12, 12 h after admission; CRP, C-reactive protein; CSF, cerebrospinal fluid; CT, computed tomography; Hia, Haemophilus influenzae type a; NA, not applicable; NG, no growth; NM, no microorganisms.
†Refer to range 4.5–14.5 x 10^9 cells/μL.
‡Reference range 1.4–9.0 x 10^9 cells/μL.
§Reference range 1.9–9.8 x 10^9 cells/μL.
¶Reference range 150–450 x 10^9/μL.
#Reference range 11.5–15.5 g/dL.
**Reference range 0–5 mg/L.
††Reference range 15–45 mg/dL.
†Reference range 2.2–3.0 mmol/L.
H. influenzae serotypes and NTHi (I; https://www.cdc.gov/meningitis/lab-manual/full-manual.pdf; online Technical Appendix Table). In Oman, it is mandatory to report cases of Hib meningitis within 24 hours of laboratory diagnosis, and those caused by other serotypes and NTHi within 1 week, to the Department of Communicable Disease Surveillance and Control, Ministry of Health. Evidence of capsule-deficient variants of Hia that cannot be differentiated from NTHi by conventional methods (7) and recurrent invasive diseases (9,10) and outbreaks caused by Hia (9; online Technical Appendix Table) emphasize the necessity for continued surveillance, strong laboratory support, and local epidemiologic studies on non-b H. influenzae disease.

Hia meningitis has been reported mainly in the indigenous peoples of Canada, Alaska (USA), and Australia; in the Navajo and White Mountain Apache tribes in the southwestern United States; and in Utah (USA), Brazil, the Gambia, East Africa, and Papua New Guinea. Sporadic cases have been reported in the rest of the world (I, 10; online Technical Appendix Table). The reasons behind the high rates of invasive Hia disease among indigenous children remain unclear (I). In Canada, where invasive non-b H. influenzae disease has been included in the list of nationally reportable diseases (http://diseases.canada.ca/notifiable/diseases-list) since 2001, a public health–driven initiative has been established to provide a better characterization of the epidemiology of invasive Hia disease and develop a candidate vaccine against Hia (online Technical Appendix Table).


References

Importation of Zika Virus from Vietnam to Japan, November 2016

Takehiro Hashimoto, Satoshi Kutsuna, Shigeru Tajima, Eri Nakayama, Takahiro Maeki, Satoshi Taniguchi, Chang-Kweng Lim, Yuichi Katanami, Nozomi Takeshita, Kayoko Hayakawa, Yasuyuki Kato, Norio Ohmagari

Author affiliations: National Center for Global Health and Medicine, Tokyo, Japan (T. Hashimoto, S. Kutsuna, Y. Katanami, N. Takeshita, K. Hayakawa, Y. Kato, N. Ohmagari); National Institute of Infectious Diseases, Tokyo (S. Tajima, E. Nakayama, T. Maeki, S. Taniguchi, C.-K. Lim)

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We report a case of Zika virus infection that was imported to Japan by a traveler returning from Vietnam. We detected...