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(CASE REPORT)

Haemophilus influenzae meningitis: A rare etiology of bacterial meningitis in adults! A case report

Wiam Ait Driss *, Malika Idalene and Noura Tassi

Department of Infectious Diseases, Mohammed VI university hospital, Cadi Ayyad University. Marrakech, Morocco.

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Abstract

Invasive Haemophilus influenzae infections can occur as a sepsis, an invasive pneumonia, an epiglottitis and an acute bacterial meningitis. H. influenzae meningitis accounts for about 4% of community-acquired bacterial meningitis in adults.

We report a case of Haemophilus influenzae serotype b (Hib) meningitis in a 37-year-old patient having an osteomeningeal breach as a predisposing factor. The diagnosis was established by lumbar puncture. The course was satisfactory with antibiotic treatment. The systematic search for an osteomeningeal breach as well as other risk factors is crucial in any adult patient presenting with an episode of confirmed H. influenzae meningitis. The quick performance of bacteriological samples is primordial in order to obtain an early diagnosis and to ensure a good clinical outcome.

Keywords: Haemophilus influenza; Lumbar puncture; Meningitis; Osteomeningeal breach

1. Introduction

Haemophilus influenzae (H. influenzae) is a gram-negative coccobacillus bacteria for which humans are the only known natural host. This bacteria is a familiar colonizer of the human respiratory tract, as part of the normal human flora. The transmission is airborne, and the nasopharyngeal carriage is usually asymptomatic. Invasive H. influenzae infections occur when the bacteria cross the nasopharynx, invade the bloodstream, and then proceed to normally sterile sites. Invasive H. influenzae infections can manifest as a sepsis, an invasive pneumonia, an epiglottitis, and an acute bacterial meningitis [1]. The H. influenzae meningitis represents roughly 4% of community-acquired bacterial meningitis in adults [2]. Here, we report a case of Haemophilus influenzae serotype b (Hib) meningitis; a rare etiology of bacterial meningitis in adults.

2. Case report

A 37-year-old patient with a history of frontal impact cranial trauma 2 years ago, for which he was operated in neurosurgery with epilepsy sequelae, was admitted for a febrile meningeal syndrome. The symptoms started 4 days before his admission by the sudden onset of headache, photophobia and vomiting, along with spinal pain -especially in the cervical region-, complicated by the onset of a generalized tonicoclonic seizure, all occurring in a feverish state. The questioning did not reveal any mention of respiratory or urinary symptoms, nor of antibiotic therapy. On clinical examination, the patient was obnubilated with a Glasgow score of 14/15, polypneic at 29 cycles/min, and had a fever of 38.8 degrees. On neurological examination, we noted a stiff neck with a positive kerning and Brudzinski sign and a shaky walk. The remaining somatic examination was unremarkable.

* Corresponding author: Wiam Ait Driss
Department of Infectious Diseases, Mohammed VI university hospital, Cadi Ayyad University Marrakech, Morocco.

An emergency brain CT scan showed a sequelae-like left frontal hypodensity, a frontal sequelae fracture extending to the anterior and posterior sides of the frontal sinus, and a fracture of the left ethmoid roof with a fill-in of the ethmoidal cells, creating an osteomeningeal breach. The lumbar puncture showed a cloudy fluid with a cellularity of 1280 elements -mainly polymorphonuclear neutrophils PPN (70%)-, a severe hypoglycorrachia of 0.01 g/L with a concomitant glycorrachia/glycemia ratio of 0.007, and a proteinorrachia of 0.87 g/L. Direct examination of the CSF revealed gram-negative bacilli with a ceftriaxone-sensitive group B Haemophilus influenzae on culture. The C-reactive protein (CRP) was high at 193mg/L. The haemogram showed a hyperleukocytosis at 12910 with PPN predominance. On the biochemical side, there was a slight disturbance of the hepatic balance with a cytolysis: ALAT at 66 (1,5N) and ASAT at 94 (2N). A respiratory PCR performed before the onset of polypnea had detected a Haemophilus influenzae. An intravenous antibiotic therapy using meningeal dose of ceftriaxone was prescribed for a 7 days period.

The course was good, with complete recovery of consciousness, apyrexia, disappearance of the meningeal syndrome, and normalization of the biological check-up. The etiological workup in search of risk factors for developing an invasive H. influenzae infection was negative; HIV serology was negative, complement dosage was normal, and an abdominal ultrasound scan in search of asplenia did not reveal any defects.

3. Discussion

First, Haemophilus influenzae (H. influenzae) was isolated in 1883 by Koch from conjunctivitis pus, and then in 1893 by Pfeiffer from the sputum of patients with flu. It has long been considered the etiologic agent of influenza. Humans are the only known reservoir; before the diffusion of Hib vaccination, children under 5 years of age were the primary reservoir of the germ, with a 3-9% colonization rate of the nasopharynx [3]. Since vaccination, this carriage has decreased considerably, currently older children and adults are more likely to harbor the germ (asymptomatic carriers) and probably constitute the primary reservoir. Haemophilus influenzae remains one of the main bacterial agents causing infections that can be invasive and severe, particularly in children under 5 years of age [4]. The great majority of systemic Haemophilus influenzae infections are due to encapsulated type b strains as a result of the major role of the polyribosephosphate (PRP) as a virulence factor (resistance to phagocytosis and complement action). The other serotypes are in fact destroyed during the septicemic phase and represent a lower risk of secondary localization (arthritis, meningitis) [5], thus, they could reveal subjacent comorbidities. The non-encapsulated form is encountered in localized manifestations in adults and children, particularly in the otorhinolaryngological (ENT) and bronchopulmonary areas [5].

Nevertheless, non-typeable strains of H. influenzae can lead to invasive infections [1]. A prospective cohort study of 80 adult cases of H. influenzae meningitis published in 2021 in the Netherlands has shown that in adult patients, the majority of H. influenzae meningitis occurs with non-typeable strains [2]. Another study carried out in Portugal on the emergence of non-encapsulated and encapsulated strains of Haemophilus influenzae isolates of non-b serotype between 1989 and 2001 on 119 isolates has concluded that the rate of non-encapsulated strains increased (from 19 to 80%) when the first invasive Haemophilus influenzae serotype f strain was discovered [6]. In our case, CSF culture had isolated a H. influenzae type b. Influenzae represents 4% of community-acquired bacterial meningitis in adults. CSF leakage from an osteomeningeal breach, ENT infections and immunosuppression have been shown to be the major risk factors for developing H. influenzae meningitis in adults [2]. Symptoms that indicate a CSF leakage include rhinorrhea and otorrhea, but CSF leakage may also go unnoticed [7]. In patients with skull base fracture due to head trauma, CSF leakages resolve spontaneously within 24 hours of onset [8,9], but once persisting, meningitis occurs in 7-30% of patients [9]. Cerebrospinal fluid leakages have been previously reported in 3% to 8% of patients with community-acquired bacterial meningitis and 38% of patients with recurrent meningitis [10]. Our patient had an osteomeningeal breach detected on cerebral CT following an old operated head injury as a risk factor, and clinically he did not present neither rhinorrhea nor otorrhea. Asplenia, agammaglobulinemia and deficiencies of early complement components (e.g. C1-C3) are also related to the risk of invasive infections caused by H. influenzae [1]. In our case we did not detect such abnormalities.

Clinically, the most common symptoms of H. influenzae meningitis in adults are neck stiffness, fever, photophobia, confusion, headache and vomiting. Clinical manifestations that are associated with meningitis include Kernig's sign and Brudzinski's sign. Cutaneous hyperesthesia may be associated with other signs such as tachycardia or bradycardia, respiratory disorders, and vasomotor disorders [1]. Other neurological complications of H. influenzae b meningitis may involve intracranial abscess, hydrocephalus, subdural effusion or empyema and ventriculitis. In our case, the clinical presentation combined a clear febrile meningeal syndrome complicated by a seizure and H. influenzae pneumonia.

According to the literature, H. influenzae b meningitis is associated with a mortality rate of 4-5% and severe neurological sequelae (deafness, epilepsy and intellectual impairment) of up to 8-11% [1]. Poor prognostic factors are the occurrence

of systemic complications such as sepsis, pneumonia, convulsions impairment of consciousness (Glasgow Coma Scale <14) and immunodepression [2]. In our patient, we noticed that seizures along with H. influenzae pneumonia were unfavorable factors.

The timely management of the patient and the administration of an appropriate antibiotic therapy guaranteed a rapid positive outcome. In addition, many studies have reported the efficacy of vaccination in reducing the incidence of pneumococcal, Hib, and meningococcal meningitis. Therefore, it is recommended to use vaccinations against Hib, PPV-23, meningococcal serogroup ACYW and meningococcal serogroup B in patients with bacterial meningitis with osteomeningeal breach to reduce the risk of recurrence [7].

4. Conclusion

H. influenzae meningitis in adults is a rather rare disease that can affect elderly individuals with comorbidities, as well as patients with predisposing factors, particularly immunosuppression, ENT infections and osteomeningeal breach. Studies have shown that the incidence of H. influenzae meningitis is considerably higher in patients with CSF leak. Consequently, it seems to be necessary to systematically search for an osteomeningeal breach in any adult patient with a history of head trauma, even ancient, presenting an episode of confirmed H. influenzae meningitis. Rapid bacteriological sampling is crucial for an early diagnosis and for the guidance of antibiotic therapy in order to ensure a good outcome.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest.

Statement of informed consent

Informed consent was obtained. The patient understands that her name and initials will not be published and has given her consent for clinical information to be reported in a case report.

Author contributions

All authors of the manuscript contributed to this work. They read and approved the final version.

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