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Adult Nontypeable *Haemophilus influenzae* Meningitis: A Case Report in the Post-Hib Era

Vishal Didwania [1,*]

[1] Metro Infectious Disease Consultants, Burr Ridge, IL 60527, USA
* Corresponding author: vdidwania@innovativeventures.com

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Abstract: Adult *Haemophilus influenzae* meningitis is uncommon in the post-Hib vaccine era and frequently occurs in association with an ENT source, cerebrospinal fluid (CSF) leakage, or immune compromise. We describe a 47-year-old woman with several days of progressive headache, fever, and evolving neck pain after repeated evaluations with unrevealing acute neuroimaging. CSF demonstrated neutrophilic pleocytosis, hypoglycorrhachia, and elevated protein. CSF culture and a multiplex meningitis/encephalitis PCR panel both identified *H. influenzae*; the isolate was nontypeable and beta-lactamase negative. She was treated with ceftriaxone 2 g intravenously every 12 h for 14 days and returned to her clinical baseline. This case highlights a less fulminant adult presentation and provides a focused review of modern strain epidemiology, pathogen-directed treatment, public-health implications, and a stepwise evaluation for anatomic or immunologic vulnerability after adult *H. influenzae* meningitis.

Keywords: meningitis; *Haemophilus influenzae*; nontypeable; invasive disease; CSF leak; immunodeficiency; chemoprophylaxis

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Highlights

- In a nationwide Dutch adult cohort, *Haemophilus influenzae* caused approximately 4% of community-acquired bacterial meningitis episodes; nontypeable strains accounted for 79% of typed adult *H. influenzae* meningitis isolates.
- Nontypeable *H. influenzae* is unencapsulated and distinct from non-b encapsulated serotypes. Meningitis represents a smaller proportion of invasive nontypeable disease than of invasive

encapsulated disease, but nontypeable strains predominate among contemporary adult *H. influenzae* meningitis isolates.
- Predisposing conditions are common, particularly ENT infection, cerebrospinal fluid leakage or prior cranial structural disruption, and immunocompromised states.
- WHO guidance recommends 7–10 days of pathogen-directed therapy for *H. influenzae* meningitis; treatment selection should reflect beta-lactamase status and susceptibility, with duration individualized when clinically appropriate.
- After diagnosis, confirm strain type and susceptibility, report invasive disease, and evaluate for an ENT source, occult CSF leak, and immune vulnerability. Routine chemoprophylaxis is not currently recommended for contacts of isolated nontypeable disease.

1. Introduction

The epidemiology of invasive *Haemophilus influenzae* infection changed substantially after widespread use of the *H. influenzae* type b (Hib) conjugate vaccine. Invasive Hib disease declined dramatically, particularly in young children, while nontypeable strains and non-b encapsulated serotypes now account for most remaining invasive disease. Nontypeable *H. influenzae* is unencapsulated and is distinct from the non-b encapsulated serotypes a, c, d, e, and f [1,2].

Adult *H. influenzae* meningitis is now uncommon but remains clinically important. In a contemporary nationwide Dutch cohort, *H. influenzae* caused approximately 4% of adult community-acquired bacterial meningitis episodes, and nontypeable strains accounted for 79% of typed adult *H. influenzae* meningitis isolates. Predisposing conditions were common, particularly ENT infection, CSF leakage, and immunocompromised states [3]. This report describes an adult with nontypeable *H. influenzae* meningitis after a several-day progressive illness and uses the case to review the modern epidemiology, treatment, and evaluation for underlying vulnerability.

2. Case Presentation

A 47-year-old woman with chronic lumbar radiculopathy received an epidural steroid injection 6 days before headache onset. The temporal association was noted, but there was no evidence that the procedure caused the subsequent infection.

She awoke with a severe constant occipital headache accompanied by vomiting, marked fatigue, dizziness, and chills. At the first emergency department evaluation, her WBC count was 15.3×10^9/L. CT and MRI of the brain showed no acute intracranial process. Scattered punctate cerebral calcifications were interpreted as sequelae of remote neurocysticercosis, without active lesions. She was discharged but returned 2 days later with persistent headache, now frontal, and a reported temperature of 102 °F at home. She received symptomatic treatment for possible migraine.

Three days later, she returned with continued fever and headache and new neck pain and stiffness. On arrival, her temperature was 102 °F, heart rate 70 beats/min, respiratory rate 18 breaths/min, blood pressure 139/71 mmHg, and oxygen saturation 100% on room air. She was alert and oriented, could move her neck, and had a nonfocal neurologic examination. No rash was identified.

Admission laboratory studies showed a WBC count of 13.0×10^9/L, hemoglobin 11.7 g/dL, platelets 374×10^9/L, sodium 136 mmol/L, and creatinine 1.06 mg/dL. Lumbar puncture demonstrated CSF glucose 29 mg/dL with paired serum glucose 130 mg/dL (CSF-to-serum ratio 0.22), protein 107 mg/dL, WBC 1159 cells/μL (63% neutrophils, 26% lymphocytes, and 11% monocytes), and RBC 115 cells/μL. Urinalysis, blood cultures, and respiratory PCR testing for SARS-CoV-2, influenza A/B, and respiratory syncytial virus were negative.

Empiric therapy consisted of ceftriaxone 2 g intravenously every 12 h, vancomycin for 24 h, and ampicillin and acyclovir, which were discontinued within 48 h after microbiologic clarification.

CSF culture and the multiplex meningitis/encephalitis PCR panel both identified *H. influenzae*. The isolate was nontypeable and beta-lactamase negative. Ceftriaxone 2 g intravenously every 12 h was continued for a total of 14 days; the longer course was individualized because of the delayed diagnosis and prolonged pre-treatment symptom course.

At follow-up approximately 1 week after discharge, she had returned to her clinical baseline with complete resolution of headache and had tolerated therapy without complication.

3. Discussion

3.1. Diagnostic Course and CSF Inflammatory Profile

The central diagnostic feature of this case was the evolution from severe headache with preserved mental status and unrevealing acute neuroimaging to a febrile meningeal syndrome over several days. The chronic cerebral calcifications were a potential diagnostic distractor but did not explain the progressive illness. Adult *H. influenzae* meningitis may have a less fulminant presentation than pneumococcal meningitis, with relatively preserved consciousness and a lower frequency of the classic triad [4]; earlier adult series likewise described a comparatively benign clinical course [5].

CSF showed neutrophilic pleocytosis, elevated protein, hypoglycorrhachia, and a CSF-to-serum glucose ratio of 0.22. A CSF-to-blood glucose ratio $\leqslant$0.4 is considered abnormally low [6]. Together, these findings supported bacterial meningitis. Pathogen-specific cohort medians demonstrate group-level differences in inflammatory profiles, although substantial overlap prevents identification of the causative organism from CSF indices alone (Table 1).

Table 1: Median CSF Inflammatory Profiles by Pathogen in Adult Community-Acquired Bacterial Meningitis.

Organism	CSF Leukocytes (Cells/mm^3)	Granulocytes (%)	Protein (mg/dL)	Glucose (mg/dL)
Streptococcus pneumoniae	2339	95	426	3.6
Neisseria meningitidis	6112	95	393	10.8
Listeria monocytogenes	812	82	255	36.9
Haemophilus influenzae	3930	89	321	18.0

Note: Values are cohort medians and illustrate group-level differences. Substantial overlap exists, and these findings should not be used to infer the causative pathogen in an individual patient. Source: Drost et al. [4].

3.2. *H. influenzae* in the Post-Hib Vaccine Era

Before Hib vaccination, encapsulated serotype b caused most invasive *H. influenzae* disease in young children. After introduction of Hib polysaccharide and conjugate vaccines, invasive Hib disease declined by 99% among children younger than 5 years [7]. Current invasive disease is driven primarily by nontypeable strains and, to a lesser extent, non-b encapsulated serotypes. In U.S. surveillance from 2009–2015, nontypeable strains had the highest overall incidence of invasive disease, and the highest incidence occurred in infants younger than 1 year and adults 65 years or older [1,2].

It is important to distinguish strain categories precisely. Hib is encapsulated serotype b. Non-b encapsulated strains include serotypes a, c, d, e, and f. Nontypeable *H. influenzae* lacks a capsule and therefore has no capsular serotype [1,7]. There are currently no vaccines for non-b or nontypeable *H. influenzae* disease [1].

3.3. Meningitis as a Subset of Invasive *H. influenzae* Disease

Invasive *H. influenzae* disease includes meningitis, bacteremia or sepsis, bacteremic pneumonia, epiglottitis, septic arthritis, and other infections of normally sterile sites. Among invasive *H. influenzae* cases with a recorded clinical syndrome in U.S. Active Bacterial Core surveillance from 2009–2015, meningitis accounted for approximately 7% overall: 16.4% of Hib, 12.0% of non-b encapsulated, and 5.4% of nontypeable infections [2].

These percentages use cases with a recorded clinical syndrome within each strain category as the denominator. The Dutch adult meningitis cohort used a different denominator: patients who already had *H. influenzae* meningitis. In that cohort, nontypeable strains accounted for 79% of typed adult *H. influenzae* meningitis isolates. Thus, meningitis constituted a smaller share of invasive nontypeable disease than of invasive encapsulated disease, while nontypeable strains nevertheless predominated among contemporary adult *H. influenzae* meningitis isolates because nontypeable strains cause a much larger overall burden of invasive disease [2,3].

3.4. Risk Factors and Clinical Implications

Adult *H. influenzae* meningitis often occurs in the setting of an underlying portal of entry or host vulnerability. In the contemporary Dutch adult cohort, predisposing factors included ENT infection, CSF leakage, and immunocompromised states [3]. Earlier adult series also identified otitis or sinusitis and remote neurosurgery or head trauma as common predisposing conditions [5]. CDC-recognized risk factors for invasive *H. influenzae* disease include asplenia, HIV infection, immunoglobulin or complement deficiencies, sickle cell disease, and malignancy requiring hematopoietic stem-cell transplantation, chemotherapy, or radiation therapy [1].

The exact frequency of individual predisposing conditions varies by cohort and case definition. For practical purposes, identification of adult *H. influenzae* meningitis should prompt a structured search for an ENT source or CSF leak and a stepwise assessment for immune vulnerability, with expanded testing guided by age, recurrence, severity, clinical history, and whether an anatomic source is identified.

3.5. Pathogen-Directed Treatment

WHO guidance recommends 7–10 days of pathogen-directed therapy for *H. influenzae* meningitis and lists ampicillin or amoxicillin for beta-lactamase-negative isolates and ceftriaxone or cefotaxime for beta-lactamase-positive isolates [6]. Third-generation cephalosporins remain effective therapy for susceptible *H. influenzae* [8]. This patients beta-lactamase-negative isolate was treated with ceftriaxone 2 g intravenously every 12 h for an individualized 14-day course because of delayed diagnosis and initiation of effective therapy. Delayed presentation is not itself a guideline-defined indication for prolonged therapy, and treatment duration should be individualized according to clinical response and complications.

3.6. Evaluation After Diagnosis and Public-Health Considerations

The evaluation after adult *H. influenzae* meningitis should be targeted rather than automatically exhaustive. The initial assessment should confirm microbiologic classification and identify common structural or secondary immune risk factors. Expanded anatomic or immunologic testing is most appropriate when the source is unclear, disease is recurrent, or the clinical history suggests a specific defect. Recurrent bacterial meningitis literature supports beta-2 transferrin or beta-trace testing and dedicated skull-base imaging for suspected CSF leakage, and selective assessment of quantitative immunoglobulins, specific antibody responses, IgG subclasses, and complement function [9] (Table 2).

Table 2: Suggested Evaluation After Adult *H. influenzae* Meningitis.

Domain	Initial Assessment	Additional Evaluation When Indicated
Microbiology and public health	Review strain type/serotype, beta-lactamase status when reported, and antimicrobial susceptibility data; report invasive disease to public health [1,6,7].	Forward isolate or specimen to a public-health laboratory if local typing is unavailable; determine contact management according to strain and outbreak context [7].
ENT source	Assess for otitis, sinusitis, mastoiditis, or another ENT source; review existing imaging [3].	Consider ENT consultation and targeted sinus or temporal-bone imaging when symptoms or findings suggest a local source [3,9].
CSF leak	Ask about prior cranial trauma, skull-base fracture, ENT or neurosurgical procedures, unilateral clear rhinorrhea or otorrhea, and prior meningitis [9].	Test drainage for beta-2 transferrin or beta-trace protein when available; obtain high-resolution skull-base/temporal-bone CT or appropriate MR imaging when a leak is suspected [9].
Secondary immune compromise	Test for HIV in adults with bacterial meningitis [10]; review medications and immunosuppression and assess for malignancy, transplantation, anatomic or functional asplenia, and sickle cell disease [1].	Additional testing directed by identified abnormalities or clinical history.
Humoral or complement deficiency	Consider quantitative IgG, IgA, and IgM when no clear source is identified or infection is recurrent or otherwise unusual; evidence is strongest in recurrent meningitis [9].	SPEP/immunofixation, IgG subclasses, specific vaccine-antibody responses, CH50/AH50, and immunology referral selectively [9].

Note: This is a practical, stepwise framework rather than a validated mandatory testing panel. Expanded immunologic testing is extrapolated in part from recurrent bacterial meningitis literature and should be individualized according to age, recurrence, disease severity, clinical history, and whether an anatomic source is identified.

Invasive *H. influenzae* disease is nationally notifiable, although specific reporting requirements vary by state [1,7]. There are currently no vaccines for non-b or nontypeable *H. influenzae* disease [1]. Public-health recommendations differ by strain; routine chemoprophylaxis is not recommended for contacts of isolated nontypeable *H. influenzae* disease [1,7].

4. Conclusion

Adult *H. influenzae* meningitis is uncommon in the post-Hib era but remains clinically relevant, particularly when nontypeable disease occurs in association with an ENT source, CSF leak, or immune vulnerability. The distinction between invasive-disease proportions and the strain distribution among meningitis cases is essential: meningitis represents a smaller proportion of invasive nontypeable disease, yet nontypeable strains account for most contemporary adult *H. influenzae* meningitis isolates. After pathogen-directed therapy, clinicians should confirm strain classification, address public-health requirements, and pursue a stepwise anatomic and immunologic evaluation to reduce the risk of an unrecognized predisposing condition.

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