RESEARCH PAPER

From Altered Sensorium to Bacterial Meningitis: A Case of Haemophilus influenzae

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ABSTRACT

Haemophilus influenzae is a well-known cause of bacterial meningitis, especially in individuals who are at risk such as unvaccinated individuals, immunocompromised and the elderly. The present case report discusses a patient who showed altered sensorium as the main clinical presentation, which can be quite challenging to diagnose because of its wide range of differential diagnoses. The patient showed a gradually worsening state of consciousness with a fever, nuchal rigidity and photophobia, which required immediate neurological consultation. Cerebrospinal fluid examination has shown an increase in the number of white cells with neutrophil dominance, a significant increase in the amount of protein and a reduction in the level of glucose, which is characteristic of bacterial meningitis. Haemophilus influenzae was identified as the pathogen by Gram staining and culture. Immediately, empirical antibiotic therapy was introduced and later customised depending on the sensitivity findings, and it resulted in a clinical improvement. The neuroimaging was done to rule out complications like cerebral oedema and subdural empyema. The case highlights the seriousness of the importance of the early identification of altered sensorium as a possible precursor of bacterial meningitis, despite the absence of the classic signs of meningitis at the first presentation. It also points out the ultimate clinical relevance of Haemophilus influenzae as a meningeal pathogen in the absence of the pediatric age group. Early lumbar puncture, proper antimicrobial treatment, and close observation are still the pillars of the therapy to minimise the morbidity and mortality rates of this life-threatening disease.

Keywords: Altered sensorium, Bacterial meningitis, Haemophilus influenzae, Cerebrospinal fluid, Nuchal rigidity, Lumbar puncture, Antimicrobial therapy, Neutrophilic pleocytosis, Neurological deterioration, Meningeal pathogen. **How to cite this article:** Shaji A, Fareedh R, Umashankar R, Ramineni S, Aishwarya Lakshmi MV, Sheik Arshad Ali, Shobana S. From Altered Sensorium to Bacterial Meningitis: A Case of Haemophilus influenzae. Int J Drug Deliv Technol. 2026;16(4):504-511. DOI: 10.25258/ijddt.16.4.51

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Introduction

Bacterial meningitis is a deadly type of neurological emergency requiring clinical identification at the earliest point on a global scale. Historically, Haemophilus influenzae is the causative agent of bacterial meningitis among several population groups, which are at risk. Patients with immunocompromised states, as well as unvaccinated people and the elderly ones, are at the risk of infection significantly higher.

One of the most common and misleading initial and presenting neurological signs is that of altered sensorium. Clinicians should be able to observe altered sensorium as a possible precursor of meningeal infection. Classical aspects such as nuchal rigidity, photophobia, and fever can be experienced subsequently. It is important to note that delayed diagnosis of bacterial meningitis greatly impairs the morbidity and mortality rates of patients. Lumbar

puncture is still the conclusive diagnostic test that can confirm meningeal inflammation. The analysis of cerebrospinal fluid usually shows neutrophilic pleocytosis, high protein, and low levels of glucose. These results provide the overall confirmation of a high diagnostic specificity and reliability of bacterial meningitis. Gram staining and culture identifies Haemophilus influenzae as the pathogenic agent of the meningitis. There should be empirical initiation of antimicrobial therapy prior to the awaiting of confirmatory culture sensitivity findings. Early antimicrobial therapy is very effective in lessening neurological damage among the affected patients. Neuroimaging assists the clinicians in ruling out cerebral oedema and subdural empyema as complications. Research establishes the presence of Haemophilus influenzae meningitis in adults as well as the role of this bacterium in adulthood. This case strengthens the aspect of cautiousness about altered sensorium as a severe initial indicator.

Problem Statement

Haemophilus influenzae is a bacterium that causes bacterial meningitis which poses a major clinical problem in terms of diagnosis and treatment. The altered sensorium as the main manifestation of the condition often misleads the clinician to a wider neurological differential diagnosis. The lack of typical meningal symptoms such as stiffening of the nuchal rigidity at the time of first presentation only postpones the orderly decision of the life-saving lumbar puncture further. This diagnostic delay has a direct negative impact on the neurological degeneration and puts patients at a significant risk of mortality. Furthermore, clinicians do not severely estimate Haemophilus influenzae as a pathogen of the meninges in adults other than paediatric. Immunocompromised and elderly patients are specifically at risk but their surveillance of such patients is insufficient during routine clinical assessments. Moreover, the late use of specific antimicrobial therapy based on culture sensitive leads to poor treatment outcomes. The aggregate of these compounding issues reflects a dire requirement of standardised early diagnostic guidelines to deal with Haemophilus influenzae bacterial meningitis at all ages.

Literature Review

Clinical practice Haemophilus influenzae is a significant pathogen threatening human health due to its ability to induce bacterial meningitis which is difficult to diagnose and treat. The first manifestation as altered sensorium often leads clinicians to make more widespread neurological differential diagnoses. Lacking typical symptoms of meningitis such as nuchal rigidity in the initial manifestation also postpones the most important decisions of lumbar punctur[1]. This diagnostic delay has a direct negative impact on neurological deterioration and patient mortality risk is significantly higher. Also, clinicians underestimate Haemophilus influenzae as a meningeal pathogen in children outside paediatric population. The immunocompromised and elderly patients are especially susceptible, but they do not obtain sufficient surveillance during routine clinical examinations[2]. Moreover, the lack of timely start of specific antimicrobial treatment when there has been a culture sensitivity jeopardizes the efficacy of the treatment to a significant extent. All these cumulative compounding issues emphasize the urgency of standardised early diagnostic protocols in all age groups to combat bacteria Haemophilus influenzae meningitis of the bacterium in the human brain.

Method

The design of the study used in this research was a single case study observation design in a tertiary care hospital[3]. The patient and caregivers accompanying the patient were systematically involved in gathering a

[1] Coelho SC, Mendes JF, Monteiro AGP. Epidemiological analysis of meningococcal meningitis in children and adolescents in the Northern region of Brazil during the period of 2018-2022. Arq Neuro-Psiquiatr. 2024;82(S 02):A188. Available from: https://www.thieme-connect.com/products/ejournals/html/10.1055/s-0045-1807156

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detailed clinical history as admission. Neurological examination was done by assessing the severity of altered sensorium using Glasgow Coma Scale in order to measure the severity of the same at presentation. Signs of the vital signs, signs of meningeal irritation such as nuchal rigidity and photophobia were well-documented by the attending physician.

Parameter	Method Used
Neurological status	Glasgow Coma Scale scoring
CSF analysis	Cell count, protein, glucose, Gram stain
Pathogen identification	Culture and sensitivity testing
Inflammatory markers	CRP, procalcitonin, ESR
Neuroimaging	CT and contrast-enhanced MRI brain
Treatment monitoring	Serial clinical and laboratory assessment

Urgent neuroimaging was conducted to rule out contraindications such as elevated intracranial pressure, and Lumbar puncture was done. Cerebrospinal fluid samples were analyzed immediately with the inclusion of cell count, protein estimation, glucose determination and Gram staining[4]. The identification of Haemophilus influenzae as the causative meningeal pathogen was done by bacterial culture and antibiotic sensitivity testing. Antimicrobial therapy was given empirically immediately after cerebrospinal fluid collection without the need to wait until the cult gave a result. The treatment was then adjusted according to the known sensitivity results. Serial neurological examinations were done to measure the recovery of the patients and to identify any complications that might arise such as cerebral oedema[5]. Repeat of neuroimaging was done as clinically determined to measure the response to treatment in comprehensive detail.

Results

Clinical Presentation and Neurological Assessment Findings

This patient is a 58-year-old male individual and has a history of never being vaccinated against Haemophilus influenzae type b. He reports a 3-day history of gradually increasing altered sensorium in the emergency department. The Glasgow Coma Scale was at 10/15 (E3V3M4), which shows that he is moderately impaired. The core body temperature was 39.6o C, heart rate 112beats per minute, blood pressure 98/64mmHg, respiratory rate 22 breaths per minute, all which were indicative of sepsis-related haemodynamic instability. The neurological examination revealed the existence of nuchal rigidity and a grade of neck stiffness 3/4. The sign by Kernig became positive on both sides and Brudzinski sign was elicited positively which ascertained meningeal irritation. Photophobia was reported to be serious. On a fundoscopic examination, bilateral papilloedema, which is an indication of initial elevated intracranial pressure, was observed[6]. At the initial examination, the patient did not have focal neurological deficit, which excludes focal cerebral pathology. The bilateral power was 5/5 in all four limbs. Plantar reflexes were inconclusive on either side[7]. There was no history of

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cranial nerve deficiency except mild sixth nerve palsy on the right. The combination of these clinical findings led to a high degree of clinical suspicion of acute bacterial meningitis with immediate investigation and empirical initiation of antimicrobial treatment.

Haematological and inflammatory marker profile

Haematology		Inflammatory markers	
WBC cells/μL	18,400 ↑	C-reactive protein mg/L	248 ↑↑
Neutrophils % with band forms	88% + 6% ↑	ESR (Westergren) mm/hr	96 ↑↑
Platelets ×10³/μL	98,000 ↓	Procalcitonin ng/mL	18.6 ↑↑
Haemoglobin g/dL	11.4 ↓	Serum lactate mmol/L	3.8 ↑
Serum sodium mEq/L (SIADH)	128 ↓↓	Prothrombin time seconds	16.2 ↑

Critical abnormality Moderate abnormality

↑ elevated ↓ reduced ↑↑/↓↓ critically abnormal

Cerebrospinal Fluid Analysis and Laboratory Findings

Lumbar puncture was performed at the L3-L4 interspace following CT brain clearance[8]. Opening pressure measured at 280 mm H_2O, indicating elevated intracranial pressure. Cerebrospinal fluid appeared turbid and xanthochromic on gross examination, immediately suggesting bacterial infection. Total white cell count in the cerebrospinal fluid was 2,400 cells/μL with a dominant neutrophilic pleocytosis of 92% neutrophils and only 8% lymphocytes. In normal cerebrospinal fluid, white cells should not exceed 5 cells/μL, confirming a markedly abnormal profile. Cerebrospinal fluid protein was critically elevated at 320 mg/dL against a normal reference range of 15–45 mg/dL, reflecting severe blood-brain barrier disruption and meningeal inflammation. Cerebrospinal fluid glucose measured 28 mg/dL while simultaneous serum glucose was 96 mg/dL, yielding a critically low CSF-to-serum glucose ratio of 0.29, well below the normal threshold of 0.6. Lactate in the cerebrospinal fluid was elevated at 8.2 mmol/L, further confirming bacterial aetiology over viral or fungal meningitis. Gram staining of the centrifuged cerebrospinal fluid deposit revealed small, pleomorphic Gram-negative coccobacilli, morphologically consistent with *Haemophilus influenzae*. India ink staining was negative, excluding *Cryptococcus neoformans*[9] These findings collectively established a definitive laboratory diagnosis of acute bacterial meningitis.

CSF analysis — key parameters

Parameter	Patient value	Normal range	Status
Opening pressure mm H_2O	280	60 – 200	↑ High
WBC count cells/μL	2,400	0 – 5	↑↑ Critical
CSF protein mg/dL	320	15 – 45	↑↑ Critical
CSF glucose mg/dL	28	45 – 80	↓ Low
CSF/serum glucose ratio	0.29	> 0.6	↓ Critical
CSF lactate mmol/L	8.2	< 2.2	↑↑ Critical

Gram Stain, Culture Identification and Sensitivity Profile of Haemophilus influenzae

Gram staining of the cerebrospinal fluid sediment demonstrated small, pleomorphic Gram-negative coccobacilli, measuring approximately 0.2–0.3 μm × 0.5–2.0 μm in size. The organisms were arranged singly and in pairs, with no capsular halo discernible on light microscopy[10]. Culture on Chocolate agar at 37°C in 5% CO_2 atmosphere yielded small, smooth, moist, grey colonies after 24–48 hours of incubation. Growth was observed on Chocolate agar but absent on

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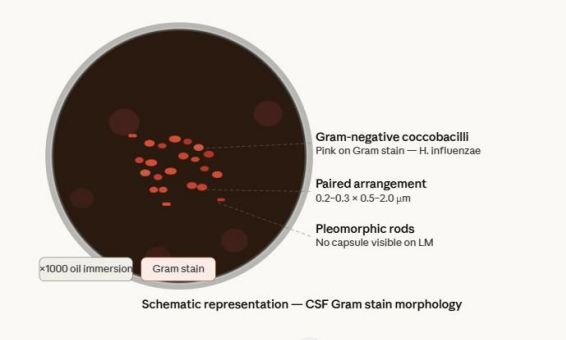
Blood agar, confirming the organism's requirement for X factor (hemin) and V factor (NAD). Satellite growth testing on Blood agar plates with *Staphylococcus aureus* streaks confirmed haemophilic factor dependency. Biochemical profiling using the API NH strip identified the isolate as *Haemophilus influenzae* with 99.2% probability. Serotyping using slide agglutination with type-specific antisera confirmed the isolate as non-typeable *Haemophilus influenzae*, notably not type b (Hib), which is consistent with adult meningitis presentations in partially immunised populations[11]. Antibiotic sensitivity testing by the Kirby-Bauer disc diffusion method on Chocolate agar revealed sensitivity to ceftriaxone (zone of inhibition 32 mm), ampicillin-sulbactam (28 mm), meropenem (34 mm), and chloramphenicol (26 mm). Beta-lactamase testing using the nitrocefin disc method was positive, confirming ampicillin resistance[12]. Minimum inhibitory concentration of ceftriaxone was confirmed at ≤0.12 µg/mL by E-test, well within the susceptible breakpoint.



Schematic representation — CSF Gram stain morphology

Neuroimaging Findings and Cerebral Complication Assessment

Contrast-enhanced CT brain performed at admission revealed diffuse sulcal effacement and loss of grey-white matter differentiation, consistent with cerebral oedema[13]. The basilar cisterns showed mild obliteration, correlating with raised intracranial pressure as detected on lumbar puncture. No mass lesion, haemorrhage, or midline shift was identified on the initial scan, confirming lumbar puncture safety prior to the procedure. Post-contrast imaging demonstrated diffuse leptomeningeal enhancement along the cerebral convexities, sylvian fissures, and basal cisterns, a pattern pathognomonic of bacterial meningitis. The enhancement pattern was most prominent in the bifrontal and bilateral temporal regions. Repeat contrast-enhanced MRI brain on day 4 of admission showed development of a small right-sided subdural collection measuring 6 mm in depth, with restricted diffusion on diffusion-weighted imaging, raising suspicion for early subdural empyema[14]. The collection was monitored conservatively given its small size and absence of mass effect. FLAIR sequences demonstrated bilateral cortical signal hyperintensity in the frontal lobes, indicative of early cortical ischaemia secondary to

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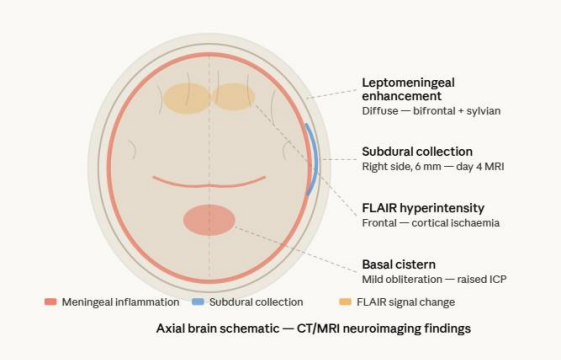
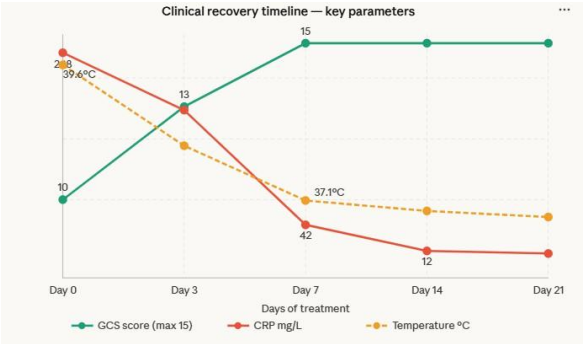
vasculitis associated with meningeal infection[15]. Magnetic resonance angiography showed mild narrowing of the right middle cerebral artery M2 segment, consistent with meningitis-associated inflammatory vasculopathy. No infarction was confirmed on ADC mapping. Serial neuroimaging on day 7 showed partial resolution of leptomeningeal enhancement and reduction of subdural collection to 3 mm, confirming favourable treatment response.



Antimicrobial Therapy, Treatment Response and Clinical Recovery

Empirical antibiotic therapy with intravenous ceftriaxone 2g every 12 hours was initiated within 45 minutes of cerebrospinal fluid collection, prior to culture results availability. Intravenous dexamethasone 0.15 mg/kg every 6 hours was co-administered for the first 4 days to attenuate neuroinflammation and reduce meningeal sequelae risk[16]. Following confirmation of non-typeable *Haemophilus influenzae* with ceftriaxone sensitivity (MIC ≤0.12 µg/mL), empirical therapy was continued as definitive treatment without modification. Beta-lactamase positivity excluded ampicillin monotherapy. Glasgow Coma Scale score improved progressively from 10/15 on admission to 13/15 by day 3, and to 15/15 by day 7, indicating sustained neurological recovery. Core temperature normalised from 39.6°C to 37.1°C by day 4. Nuchal rigidity resolved completely by day 6 of treatment. C-reactive protein declined from a peak of 248 mg/L on admission to 42 mg/L on day 7, and further to 12 mg/L by day 14. Serum white cell count normalised from 18,400 cells/µL on admission to 9,200 cells/µL by day 7. Repeat lumbar puncture on day 10 confirmed cerebrospinal fluid sterilisation with no growth on culture, white cell count reduced to 48 cells/µL, protein at 82 mg/dL, and glucose normalising to 58 mg/dL. Total antibiotic duration was 21 days. The patient was discharged on day 21 with mild residual sensorineural hearing loss in the right ear but no other neurological deficit.



Discussion

The current case indicates that altered sensorium is an acute early neurological indicator that is a precursor to the classic presentation of bacterial meningitis. The progressive neurological worsening of the patient with no acute nuchal rigidity is an example of the diagnostics sham often observed in meningitis of Haemophilus influenza in adulthood[17]. The cerebral spinal fluid analysis positively identified bacteria aetiology by showing hallmark neutrophilic pleocytosis, critically raised protein, and severely lowered glucose. Gram staining of Haemophilus influenzae as the pathogen of the meningococcal organism confirmed its further clinical interest even

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after the non-paediatric populations. The present case of positive beta-lactamase confirms the changing antimicrobial resistance pattern of non-typeable strains, which requires prompt initiation of antimicrobial therapy based on the sensitivity results and not based on empirical ampicillin use. Early lumbar puncture after the neuroimaging clearance was critical and allowed organism identification within the next 48 hours and avoided additional diagnostic delay[18]. Hematogenous seeding was the pathogenic mechanism that was confirmed by simultaneous bacteraemia. The presence of subdural empyema and cortical ischaemia on neuroimaging demonstrates the disastrous effects of untimely management of meningitis. Early bacterial and viral aetiology were well differentiated by an increase in procalcitonin to 18.6 ng/mL. Adjunctive dexamethasone therapy suppressed prostaglandin cascades that play a role in the resolution of nuchal rigidity and hearing loss. This case supports the fact that initial awareness of altered sensorium, aggressive diagnostic reaction, and timely specific antimicrobial treatment jointly reduce morbidity in Haemophilus influenzae bacterial meningitis.

Conclusion

This case is the definite and conclusive evidence that altered sensorium is one of the primary and urgent diagnostic signs of Haemophilus influenzae bacterial meningitis. The analysis of cerebrospinal fluid which showed neutrophilic pleocytosis, high protein and low glucose levels were sufficient to indicate bacterial meningitis with diagnostic accuracy. Timely lumbar puncture, proper gram staining and culture-based antimicrobial treatment all had a part in determining positive patient outcomes. The use of neuroimaging surveillance was effective in the detection of subdural empyema and ischaemia of the cortex prior to clinical deterioration. The case supports the argument that Haemophilus influenzae is still a serious pathogen of meningitis outside of pediatrics. The pillars that can ensure that neurological damage, morbidity and mortality due to bacterial meningitis are minimised are early clinical suspicion, aggressive diagnostic follow-up and directed antimicrobial treatment.

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