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Case Report

Internal Medicine Section

Haemophilus Influenzae Meningoencephalitis with Multifocal Haemorrhagic Cerebral Infarction and Lobar Pneumonia in an Immunocompetent Young Adult: A Diagnostic and Therapeutic Challenge

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ABSTRACT

Haemophilus influenzae meningoencephalitis complicated by multifocal haemorrhagic cerebral infarction is exceedingly rare in immunocompetent adults and presents a formidable diagnostic challenge. This case report is of a 20-year-old unvaccinated male who presented with high-grade fever, altered sensorium, generalised tonic-clonic seizures, and dysarthria of seven days' duration. Neurological examination disclosed meningeal signs and a Glasgow Coma Scale (GCS) of 12/15. Magnetic Resonance Imaging (MRI) brain with contrast revealed multiple acute haemorrhagic infarcts involving the left fronto-temporo-parietal cortex, basal ganglia, insular cortex, and periventricular white matter, with extension to the right cerebellum on Day 3 imaging. Concurrent left lower lobe consolidation was confirmed on High-Resolution Computed Tomography (HRCT) thorax, and echocardiography demonstrated mildly reduced ejection fraction (45%) with inferoseptal hypokinesia without vegetations. CSF analysis revealed a normal cell count (2 cells/µL, lymphocyte predominant), with normal protein and glucose levels; conventional culture was negative. Pathogen identification was achieved exclusively via the CSF BioFire FilmArray Meningitis/Encephalitis (ME) panel, which returned positive for *H. influenzae*. Following clinical deterioration and rising inflammatory markers on ceftriaxone-based empirical therapy, treatment was escalated to meropenem (2 g every 8 hours) with adjunctive dexamethasone, resulting in progressive clinical improvement by Day 7. This case illustrates the diagnostic utility of multiplex molecular panels in culture-negative bacterial meningitis and underscores the potential for *H. influenzae* to cause extensive cerebrovascular injury through septic arteritis and vasculitis. Timely carbapenem escalation may be life-saving when cephalosporin therapy fails.

Keywords: Bacterial, Carbapenems, Central nervous system, Meningitis, Polymerase chain reaction, Vasculitis

CASE REPORT

A 20-year-old male resident of rural Gujarat, with no documented co-morbidities and no prior *Haemophilus influenzae* type b (Hib) vaccine, presented to the emergency department with a seven-day history of high-grade continuous fever, occasional cough with whitish expectoration, progressive alteration of sensorium over two days, and two episodes of generalised tonic-clonic seizures in the preceding 24 hours. Slurring of speech was also noted by the family. A history of cattle exposure raised the possibility of zoonotic aetiology.

On admission, the patient was febrile (temperature 100°F), tachycardic (pulse 112/min), normotensive (BP 120/70 mmHg), and hypoxic (SpO_2 94% on room air). GCS was E4M6V2 (total 12/15). Pupils were equal and reactive bilaterally. Meningeal signs were present: neck rigidity was mild, and Kernig's and Brudzinski's signs were positive [1]. Motor power was intact in all four limbs; lower limb reflexes were hyperreflexic with flexor plantar responses bilaterally. Speech was dysarthric with a recurring verbal stereotypy ("ma-ma-ma"), consistent with Broca's aphasia and insular apraxia of speech secondary to left fronto-insular infarction. Chest auscultation revealed decreased air entry with coarse crepitations at the left lung base. There were no skin rashes, petechiae, or lymphadenopathy. Initial differential diagnosis was bacterial meningitis, viral meningoencephalitis, autoimmune encephalitis or zoonotic infection with cerebral involvement with left lower pneumonia.

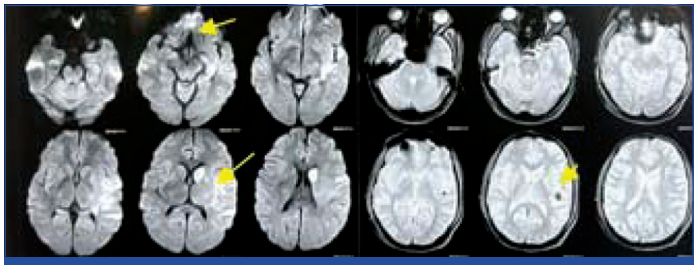
Initial haematological investigations showed haemoglobin 14.6 g/dL, total leucocyte count (TLC) 10,990/cumm, and platelets 3.5 lac/cumm. Inflammatory markers were elevated: C-Reactive Protein (CRP) 97 mg/L, erythrocyte sedimentation rate 75 mm/hr, Procalcitonin (PCT) 0.15 ng/mL. Serial ammonia levels remained largely within the reference range (31-123 µg/dL), with a transient mild elevation on day 3 (140 µg/dL) that resolved spontaneously on follow-up (40 µg/dL). Liver transaminases were raised (SGPT 141 IU/L, SGOT 77 IU/L) and serum Lactate Dehydrogenase (LDH) was markedly elevated at 1,558 IU/L, consistent with multiorgan involvement [Table/Fig-1]. Renal function and electrolytes were within normal limits. Blood cultures and sputum cultures yielded no growth.

The CSF analysis on admission showed a total cell count of 2 with 100% lymphocytes, protein 20 mg/dL, glucose 118 mg/dL (concurrent random blood glucose 140 mg/dL), and adenosine deaminase 6 IU/L, findings not initially consistent with bacterial meningitis. CSF Herpes Simplex Virus (HSV) Polymerase Chain Reaction (PCR) was negative. Serological screening for malaria, dengue, leptospirosis, brucellosis, and widal test were all negative. Autoimmune panel (ANA, ANCA) was negative. MRI brain with contrast on admission demonstrated multiple acute haemorrhagic infarcts involving the left fronto-temporo-parietal cortex, basal ganglia, and insular cortex [Table/Fig-2,3]. Chest X-ray and HRCT thorax confirmed dense consolidation of the entire left lower lobe [Table/Fig-4]. Echocardiography revealed ejection fraction of 45% with inferoseptal hypokinesia; no vegetations or valvular pathology were identified.

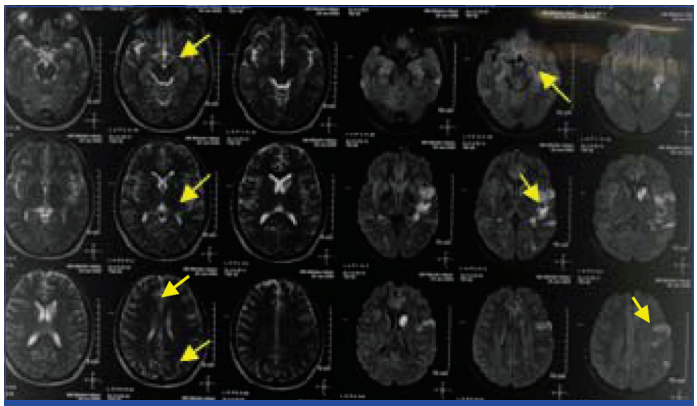
So as the clinical features were consistent with meningoencephalitis and community-acquired lobar pneumonia patient was started on empirical therapy was initiated with intravenous ceftriaxone (2 g

Parameter (Normal Range)	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Hb (g/dL) (13.5-17.5)	14.6	13.8	13.1	12.7	12.9	12.4	12.0
TLC (/cumm) (4,000–11,000)	10,990	11,000	14,000	18,500	20,300	11,000	9,800
Platelets (lac/cumm) (1.5-4.5)	3.5	2.75	2.75	2.4	2.42	2.5	2.6
PT/INR (11-13.5 sec / <1.1)	15.1/1.07	21/1.5	18.9/1.35	—	16/1.14	—	15/1.07
APTT (sec) (25-35)	28.9	30	37.7	—	34	—	44
Creatinine (mg/dL) (0.7-1.2)	0.7	0.8	0.8	0.9	1.2	1.0	1.3S
Bilirubin (mg/dL) (<1.2)	1.0	0.9	5.5	3.6	2.9	2.6	2.8
SGOT (IU/L) (<40)	77	72	68	69	59	50	—
SGPT (IU/L) (<41)	141	129	110	115	109	98	60
LDH (IU/L) (140-280)	1,558	—	865	—	—	—	—
CRP (mg/L) (<5)	97	—	98	—	220	—	—
PCT (ng/mL) (<0.5)	0.15	—	—	—	20	—	—
Na (mEq/L) (136-145)	137	140	142	146	138	139	142
K (mEq/L) (3.5-5.0)	3.9	4.0	4.5	3.4	4.0	5.0	4.4
Ammonia (µg/dL) (31-123)	115	—	140	—	—	—	40

[Table/Fig-1]: Serial laboratory investigations during hospital stay.
Hb: Haemoglobin; TLC: Total leucocyte count; PT: Prothrombin time; INR: International normalised ratio; APTT: Activated partial thromboplastin time SGOT: Serum glutamic oxaloacetic transaminase;SGPT: Serum glutamic pyruvic transaminase LDH: Lactate dehydrogenase; CRP: C-Reactive protein; PCT: procalcitonin; Na: Sodium; K: Potassium. — denotes not tested on that day.

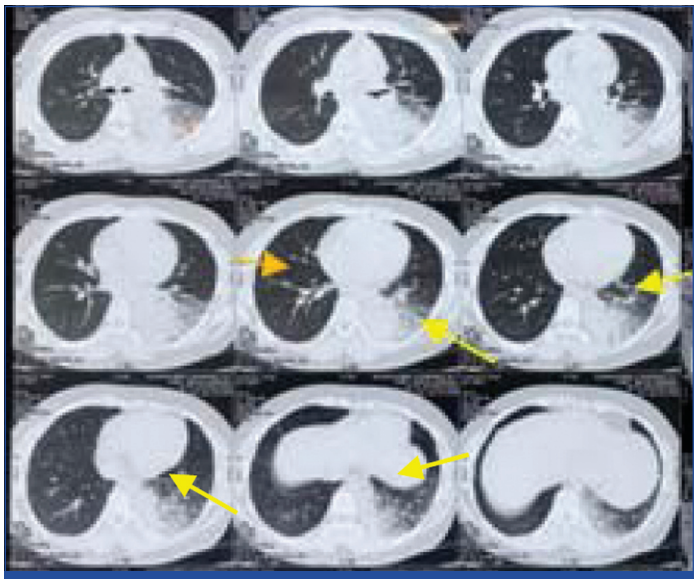


[Table/Fig-2]: Composite axial MRI (left column: DWI, T2; right column: T2/FLAIR, post-contrast T1) demonstrating restricted diffusion, confluent T2/FLAIR hyperintensity with vasogenic oedema, and leptomeningeal enhancement involving the left fronto-temporo-parietal cortex, basal ganglia, insular cortex, and periventricular white matter, consistent with acute haemorrhagic infarction and bacterial meningitis.



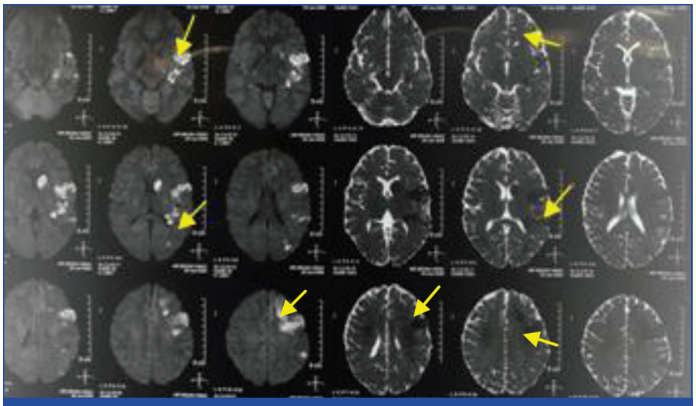
[Table/Fig-3]: Axial T2 (right) and FLAIR (left) sequences demonstrating multifocal hyperintensity involving the left fronto-parietal region, caudate nucleus, putamen, insular cortex, fronto-temporo-parieto-occipital cortical grey matter, and periventricular white matter, with an additional focus in the left inferior frontal gyrus and sulcal FLAIR hyperintensity reflecting leptomeningeal inflammatory exudate; remaining parenchyma, posterior fossa, and cervico-medullary junction are unremarkable.

every 12 hours), vancomycin (1g every 12 hours), and aciclovir (10 mg/kg every 8 hours), the latter given in view of the initial lymphocytic pleocytosis raising the possibility of viral encephalitis. Empirical doxycycline was added initially to broaden coverage against zoonotic differentials including brucellosis and rickettsiosis, in view of cattle exposure history and was subsequently discontinued once the molecular diagnosis was confirmed. Adjunctive dexamethasone (0.15 mg/kg every 6 hours) was commenced to mitigate neuroinflammation and cerebral oedema, alongside levetiracetam for seizure prophylaxis in the context of extensive cortical involvement, and low-dose aspirin for cerebrovascular protection.



[Table/Fig-4]: Plain HRCT thorax demonstrating left lower lobe consolidation collapse with air bronchograms and ground-glass opacities consistent with acute 1infective lobar pneumonia secondary to *H. influenzae*, focal right upper lobe ground glass opacity indicating early bilateral involvement, reactive mediastinal lymphadenopathy, and no pleural effusion, pericardial effusion, or bony lesions.

On day 2, transient coagulopathy (PT/INR 21/1.5 on Day 2, APTT prolongation) was consistent with early Disseminated Intravascular Coagulation (DIC). Despite three days of this regimen, the patient demonstrated progressive clinical deterioration with worsening sensorium. Notably, TLC rose from 10,990/cumm on Day 1 to 14,000/cumm on Day 3, CRP increased to 98 mg/L indicating progressive sepsis despite initial antibiotic therapy. Repeat MRI brain with MR venogram on Day 3 revealed extension of haemorrhagic transformation to the periventricular white matter adjacent to the left occipital horn and a new infarct in the right cerebellum. Cerebral venous thrombosis was excluded on MR venography [Table/Fig-5]. A CSF BioFire FilmArray ME panel was dispatched and returned positive for *H. influenzae*, allowing the diagnosis to be formally established. It is acknowledged that the panel detects *H. influenzae* at species level without serotyping capability, rare false-positive results have been reported and clinical correlation with neuroimaging, inflammatory markers, and therapeutic response therefore remains essential in interpreting this result. Antibiotic susceptibility testing was not performed, as no organism was isolated on conventional culture [2,3]. The patient was accordingly diagnosed with bacterial meningoencephalitis secondary to *H. influenzae*, complicated by



[Table/Fig-5]: DWI (right) and ADC (left) confirming multifocal cytotoxic oedema involving the left fronto-parietal cortex, caudate nucleus, putamen, insular cortex, fronto-temporo-parieto-occipital grey matter, and periventricular white matter, with haemorrhagic transformation at the left inferior frontal gyrus on GRE, an acute right cerebellar infarct confirming multivascular territory involvement, and patent cerebral venous sinuses excluding venous thrombosis, attributing haemorrhagic infarction to arterial septic vasculitis.

multifocal haemorrhagic cerebral infarction in the territory of the left middle cerebral artery involving the left fronto-temporo-parietal cortex, basal ganglia, insular cortex, and periventricular white matter with extension to the right cerebellum, secondary to septic arteritis and vasculitis. Associated complications included community-acquired lobar pneumonia, Broca's aphasia with insular apraxia of speech, sepsis-induced myocardial dysfunction, and hepatitis with early DIC.

In light of this confirmed diagnosis and ongoing clinical deterioration, antibiotic therapy was escalated on Day 3 to intravenous meropenem (2 g every 8 hours), which was continued for a total of 10 days. Vancomycin was maintained throughout the admission, doxycycline was continued for seven days in total, and dexamethasone was sustained as adjunctive therapy. Levetiracetam and low-dose aspirin were continued concurrently.

Concurrent speech and language therapy was initiated from Day 3 alongside the escalated antimicrobial regimen. Recovery followed a clinically meaningful trajectory over the course of admission, consistent with the MRI findings demonstrating relative sparing of Wernicke's area while Broca's area and the insular cortex bore the brunt of haemorrhagic infarction - a pattern that predicted comprehension recovering ahead of expressive output. On presentation, the patient's sole verbal output was a recurring stereotypy of "ma-ma-ma" produced in response to every stimulus, with no apparent comprehension of spoken commands or orientation to his name.

By Day 4, inflammatory markers rose significantly, with total leucocyte count reaching 18,500/cumm, On Day 5 CRP 220 mg/L, and PCT 20 ng/mL. TLC further increased to 20,300/cumm; however, early neurological improvement became evident. He was following simple one-step commands reliably and communicating intent through eye contact and purposeful head gestures. By Days 6 to 7, there was resolution in sepsis and gradual decrease in total count to 9800/cumm. He began producing isolated meaningful words contextually and could follow two-step commands.

At discharge, the patient was communicating in short phrases with substantially improved comprehension. He was prescribed aspirin, atorvastatin, and levetiracetam with multivitamin supplementation, and was referred for ongoing outpatient speech and language therapy and audiological assessment.

At outpatient review 15 days after discharge, the patient presented in a clinically stable condition. Vital signs were within normal limits: temperature 98.6°F, pulse 82/min, blood pressure 118/68 mmHg, and SpO_2 98% on room air. He was afebrile, alert, and oriented to person, place, and time. Significant further improvement in speech was noted: the patient was conversing in complete sentences, initiating speech spontaneously, and responding to complex questions with appropriate content - a remarkable contrast to the "ma-ma-ma" verbal stereotypy observed at admission. Residual mild word-finding difficulty and occasional articulatory imprecision were noted on formal speech assessment, consistent with resolving Broca's aphasia and insular apraxia, but these did not impair functional communication. He was independently ambulatory, had resumed oral feeding without difficulty, and reported no recurrence of fever, headache, or seizures. Patient was advised to continue levetiracetam, aspirin, atorvastatin and outpatient speech therapy sessions. Audiological assessment was arranged to screen for sensorineural hearing loss. The overall clinical trajectory at 15 days was strongly positive, affirming the benefit of early microbiological diagnosis, timely carbapenem escalation, and structured speech rehabilitation in achieving meaningful functional recovery in this young patient.

At one-month follow-up, the patient was afebrile, fully oriented, and haemodynamically stable with no infective relapse. Speech had normalised to near-baseline with ability to do daily routine activities. Repeat echocardiography demonstrated normalisation of left ventricular ejection fraction to 55% with resolution of inferoseptal hypokinesia. Audiological assessment revealed no sensorineural hearing loss. Antiepileptic levetiracetam with antiplatelet aspirin and atorvastatin was continued with speech therapy. The clinical trajectory at one month was excellent with meaningful functional recovery.

DISCUSSION

Bacterial meningoencephalitis is a life-threatening infection characterised by simultaneous inflammation of the meninges and cerebral parenchyma, representing a more severe and complex entity manifesting clinically as altered sensorium, seizures, focal neurological deficits, and behavioural disturbances superimposed upon the classical meningitic syndrome of fever, headache, and meningism [4]. The present case illustrates several clinically important challenges encountered in the diagnostic and therapeutic management of a young immunocompetent adult with culture-negative, molecularly confirmed *H. influenzae* meningoencephalitis complicated by multifocal haemorrhagic cerebral infarction.

Despite considerable advances in the prevention and management of community-acquired bacterial meningitis over the past three decades, the global burden of disease remains substantial. The introduction of conjugate vaccines targeting the three predominant causative organisms - *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* has contributed meaningfully to reducing disease incidence; however, serotype replacement by non vaccine pneumococcal strains and the emergence of antimicrobial-resistant bacterial isolates continue to undermine these gains, ensuring that bacterial meningitis remains a significant public health challenge worldwide [4]. The neuroimaging pattern in this case - multifocal haemorrhagic infarction spanning the left fronto-temporo-parietal cortex, basal ganglia, insular cortex, periventricular white matter, and contralateral cerebellum - is highly atypical for straightforward bacterial meningitis and requires a broad differential including cerebral venous thrombosis, vasculitis, septic embolism, and autoimmune encephalitis. MR venography was critical in excluding cerebral venous thrombosis, as anticoagulation would be indicated for thrombosis but potentially harmful in the setting of active haemorrhagic infarction [5]. The pathogenesis of cerebral infarction in bacterial meningitis involves cortical arteritis, vasospasm, and septic arteritis within the intense subarachnoid inflammatory milieu, resulting in progressive thrombosis and haemorrhagic transformation [6]. The extension of infarction to the right cerebellum on Day 3 imaging underscored the urgency of microbiological identification and escalation.

The deceptively bland CSF findings in this case illustrate the well-recognised phenomenon of non purulent CSF in bacterial meningitis, which may occur in early disease, partially treated infection, or when organisms have been cleared from the CSF compartment whilst parenchymal and vascular damage persists [4]. Relying solely on CSF cytochemistry to exclude bacterial aetiology is therefore potentially dangerous, particularly in cases presenting with neurological catastrophe.

The BioFire FilmArray ME panel proved pivotal, providing a definitive diagnosis within hours in a culture-negative presentation. Studies have reported sensitivity exceeding 90% for *H. influenzae* detection compared with under 60% for culture in antibiotic-pre-treated samples [7]. Such multiplex molecular diagnostics are increasingly indispensable in atypical or culture-negative central nervous system infections. Failure to respond to ceftriaxone combined with rapidly rising inflammatory markers prompted escalation to meropenem; beta-lactamase-producing *H. influenzae* strains constitute 30-40% of clinical isolates in some series, and carbapenems offer superior CSF penetration and reliable activity against such strains [8]. Adjunctive dexamethasone was maintained throughout, supported by Level-I evidence for reduction in sensorineural hearing loss and neurological sequelae [9,10].

Duong MT et al., provided a comprehensive review of neuroimaging patterns in intracranial infections, describing the spectrum of meningitis complications including cerebral infarction, cerebritis, and haemorrhagic transformation as recognised sequelae of bacterial meningitis [5]. That review shares with the present case the recognition of vascular complications - particularly ischaemic infarction - as an established consequence of bacterial meningitis-associated vasculitis, and underscores the role of advanced neuroimaging in characterising disease severity and guiding management. However, the present case goes beyond what was documented in that review in several critical respects: haemorrhagic transformation occurring across multiple bilateral vascular territories, involvement of the contralateral cerebellum, and a culture-negative presentation in which the causative pathogen *H. influenzae* was identified through the BioFire FilmArray ME molecular panel. This constellation of bilateral multiterritorial haemorrhagic transformation in the setting of culture-negative *H. influenzae* meningitis represents a substantially more complex and severe vascular phenotype than the imaging patterns conventionally described in the literature.

Chekrouni N et al., analysed 82 adults with community-acquired *H. influenzae* meningitis in the Netherlands over a 12-year period [11]. That cohort had a median age of 61 years and 79% of isolates were non typeable strains; importantly, predisposing factors - otitis or sinusitis in 49%, immunocompromising conditions in 25%, and CSF leak in 17% - were present in the overwhelming majority of patients. Pneumonia as a co-morbidity was identified as a significant predictor of unfavourable outcome (odds ratio 5.8). The present case diverges sharply from this epidemiological profile: index patient was a 20-year-old immunocompetent male with no identifiable predisposing condition, no ENT source, and no CSF leak, placing him in a distinctly uncommon demographic for adult *H. influenzae* meningitis. Furthermore, whilst the Dutch cohort had an overall unfavourable outcome rate of 17%, present patient achieved functional recovery - underscoring that young age and absence of co-morbidities may confer a degree of resilience even in severe presentations, provided escalation is timely.

Abou-Hanna J et al., described a 12-month-old immunocompetent infant with *H. influenzae* type f meningitis complicated by bilateral subdural empyemas, central venous thrombosis requiring anticoagulation, and bilateral sensorineural hearing loss requiring cochlear implants [12]. That case, like the present one, involved a fully immunocompetent host without classical predisposing risk factors, supporting the emerging recognition that non type b *H. influenzae* strains can cause severe invasive disease independent of immune status. Notably, initial microbiological identification in that case was achieved by conventional CSF culture, and the organism was ceftriaxone-sensitive, permitting successful completion of a four-week ceftriaxone course. This contrasts with index case, where culture was negative and clinical ceftriaxone failure necessitated carbapenem escalation - highlighting that non type b *H. influenzae* strains may differ substantially in antibiotic susceptibility profiles and that culture-negative presentations demand parallel molecular diagnostic strategies from the outset. Additionally, central venous thrombosis - a major therapeutic dilemma in that infant - was actively excluded in index patient by MR venography, allowing haemorrhagic infarction management without the competing risk of anticoagulation.

The speech recovery in this patient is explained by the MRI anatomy. Haemorrhagic infarction of the left inferior frontal gyrus (Broca's area) and insular cortex, with relative sparing of the posterior superior temporal gyrus (Wernicke's area), produced a classic Broca's aphasia with superimposed insular apraxia of speech - accounting for the initial recurring verbal stereotypy as the sole output. The earlier recovery of comprehension over expression, facilitated by targeted antimicrobial therapy and structured speech therapy, reflects peri-infarct plasticity and language network reorganisation expected in young adults [13].

CONCLUSION(S)

H. influenzae meningitis with multifocal haemorrhagic cerebral infarction is a rare but life-threatening presentation in unvaccinated immunocompetent young adults. Non purulent CSF findings must not be used to exclude bacterial aetiology, and multiplex molecular diagnostics such as the BioFire FilmArray ME panel are invaluable in culture-negative cases. Cerebral venous thrombosis must be actively excluded via MR venography before therapeutic decisions are made regarding anticoagulation. When cephalosporin therapy fails, escalation to meropenem with adjunctive dexamethasone may be life-saving. The stepwise recovery of speech in this patient - from a stereotyped "ma-ma-ma" through emerging comprehension and guided speech therapy to functional communication at discharge - illustrates both the severity of *H. influenzae*-mediated cerebrovascular injury and the remarkable rehabilitative potential of young patients when infection is controlled promptly. Hib vaccination of unvaccinated adults in endemic regions remains an important preventive strategy.

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REFERENCES

[1] Mehndiratta M, Nayak R, Garg H, Kumar M, Pandey S. Appraisal of Kernig's and Brudzinski's sign in meningitis. Ann Indian Acad Neurol. 2012;15(4):287-88.
[2] Leber AL, Everhart K, Balada-Llasat JM, Cullison J, Daly J, Holt S, et al. Multicenter evaluation of biofire film array Meningitis/Encephalitis panel for detection of bacteria, viruses, and yeast in cerebrospinal fluid specimens. J Clin Microbiol. 2016;54(9):2251-61.
[3] Ramachandran PS, Ramesh A, Bhatt DL, Gelfand JM, Sample HA, Zorn KC, et al. Integrating central nervous system metagenomics and host response for diagnosis of tuberculous meningitis and its mimics. Nat Commun. 2022;13(1):1-14.
[4] van de Beek D, Brouwer M, Hasbun R, Koedel U, Whitney CG, Wijdicks E. Community-acquired bacterial meningitis. Nat Rev Dis Primers. 2016;2:16074.
[5] Duong MT, Rudie JD, Mohan S. Neuroimaging patterns of intracranial infections: Meningitis, cerebritis, and their complications. Neuroimaging Clin N Am. 2023;33(1):11-41.
[6] Kasanmoentalib ES, Brouwer MC, van der Ende A, van de Beek D. Hydrocephalus in adults with community-acquired bacterial meningitis. Neurology. 2010;75(10):918-23.

[7] Messacar K, Breazeale G, Robinson CC, Dominguez SR. Potential clinical impact of the film array meningitis encephalitis panel in children with suspected central nervous system infections. Diagn Microbiol Infect Dis. 2016;86(1):118-20.
[8] Tristram S, Jacobs MR, Appelbaum PC. Antimicrobial resistance in *Haemophilus influenzae*. Clin Microbiol Rev. 2007;20(2):368-89.
[9] de Gans J, van de Beek D. Dexamethasone in adults with bacterial meningitis. N Engl J Med. 2002;347(20):1549-56.
[10] Bijlsma MW, Brouwer MC, Kasanmoentalib ES, Kloek AT, Lucas MJ, Tanck MW, et al. Community-acquired bacterial meningitis in adults in the Netherlands, 2006–14: A prospective cohort study. Lancet Infect Dis. 2016;16(3):339-47.
[11] Chekrouni N, Brouwer MC, van der Ende A, van de Beek D. Community-acquired *Haemophilus influenzae* meningitis in adults. J Infect. 2021;82(5):145-50.
[12] Abou-Hanna J, Panning K, Mehta H. *Haemophilus influenzae* type f meningitis complicated by bilateral subdural empyema, central venous thrombosis, and bilateral sensorineural hearing loss in an immunocompetent 12-month-old. Cureus. 2019;11(6):e4850.
[13] Saur D, Lange R, Baumgaertner A, Schraknepper V, Willmes K, Rijntjes M, et al. Dynamics of language reorganization after stroke. Brain. 2006;129(Pt 6):1371-84.

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