Case Report

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A Case Report and Current Literature Review of Pneumococcal Meningitis Complicated by Cortical Infarction Secondary to Infectious Cerebral Vasculitis in A Young Male

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Abstract

Bacterial meningitis rarely causes cerebral vasculitis and cerebral infarction. A 23-year-old male presenting with fever, headache, and neck stiffness was diagnosed with pneumococcal meningitis based on cerebrospinal fluid analysis and magnetic resonance imaging (MRI) of the brain. The patient's clinical course was complicated by cerebral vasculitis, as evidenced by a right parietal infarction on brain MRI, resulting in a prolonged intensive care unit stay. He was treated with intravenous antibiotics (ceftriaxone and vancomycin) and corticosteroids. Targeted antibiotic therapy and timely adjunctive initiation of corticosteroids to address inflammation are critical for improving long-term outcomes.

Keywords: *Streptococcus pneumoniae*, cerebral vasculitis, infarction, bacterial meningitis

Introduction

Bacterial meningitis is a critical infection involving the central nervous system, characterized by inflammation of the leptomeninges and the adjacent brain and spinal cord tissues. The most common symptoms include fever, headache, vomiting, neck stiffness, and photophobia. The estimated incidence ranges from 0.6 to 4 cases per 100,000 adults annually, with higher rates reported among elderly populations, particularly in low- and middle-income countries. In adults, the most common causative organisms are *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* (1,2). Among immunocompromised patients, *Listeria monocytogenes* and Group B *Streptococcus* are also common pathogens. If left untreated, bacterial meningitis carries a mortality rate of up to 50%. Complications include seizures, hearing loss, cognitive dysfunction, and long-term neurological deficits. Cerebral vasculitis is a severe complication that can result in ischemic stroke and permanent neurological impairment and has been reported in up to 25% of cases of bacterial meningitis caused by *Streptococcus pneumoniae*. We report a case of a young patient with pneumococcal meningitis confirmed by cerebrospinal fluid (CSF) culture, complicated by cerebral vasculitis.

Case Report

Written informed consent was obtained prior to publication, and all identifying patient details were anonymized in the manuscript. A 23-year-old male with no significant past medical history presented to the emergency department (ED) with a severe occipital headache of two days' duration, accompanied by fever and vomiting for one day. The patient denied any history of diarrhea, dysuria, respiratory symptoms, joint pain, rash, or other

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constitutional symptoms. He reported a remote history of head trauma six years earlier that required suturing, with no subsequent history suggestive of CSF rhinorrhea.

On presentation, the patient was conscious, oriented, and febrile, with a blood pressure of 120/70 mmHg and a pulse rate of 104 beats per minute. Neurological examination revealed neck stiffness and a positive Kernig's sign, while the remainder of the systemic examination was unremarkable.

On the first day of hospitalization in the ED, the patient experienced a generalized tonic-clonic seizure followed by an altered level of consciousness. Intravenous (IV) antiepileptic therapy and supplemental oxygen via nasal cannula were initiated immediately. Baseline laboratory investigations demonstrated neutrophilic leukocytosis (white blood cell count: 17,140/mm³; neutrophils: 87.5%). Renal and liver function tests were within normal limits, as were blood glucose levels (123 mg/dL) and glycated (hemoglobin A1c: 5.6%). Chest radiography was unremarkable. Viral serology, including hepatitis B, human immunodeficiency virus, and hepatitis C, was negative.

Cerebrospinal fluid analysis revealed marked pleocytosis (white blood cell count: 1,067/mm³), predominantly polymorphonuclear leukocytes, elevated protein levels (836 mg/dL in a 1:5 dilution), and markedly reduced glucose levels (<2 mg/dL), findings consistent with bacterial meningitis. A non-contrast computed tomography scan of the brain showed no evidence of acute infarction or hemorrhage. Magnetic resonance imaging (MRI) of the brain demonstrated acute ischemic and inflammatory changes. Diffusion-weighted imaging (Figure 1A) revealed a focal area of hyperintensity in the left parietal cortex, consistent with an acute infarction (red arrow). The corresponding apparent diffusion coefficient map (Figure 1B) showed a concordant hypointense signal, confirming true restricted diffusion (red arrow). Axial T1-weighted post-contrast images (Figure 1C) demonstrated diffuse leptomeningeal enhancement (red arrow), a characteristic radiological feature of infectious meningitis.

Empirical treatment was initiated with IV vancomycin 1 g every 8 hours, ceftriaxone 2 g every 12 hours, dexamethasone 8 mg every 8 hours, and acyclovir 750 mg every 8 hours. Clindamycin 600 mg IV every 12 hours was also administered because aspiration was suspected. The patient was transferred to the intensive care unit for close monitoring. He required supplemental oxygen, which was gradually tapered as his condition improved. Because of persistent fever spikes, a tropical fever panel was ordered; results were negative for dengue, leptospirosis, and scrub typhus. Blood and urine cultures remained sterile; however, CSF culture grew *Streptococcus pneumoniae*. Acyclovir was discontinued, and antibiotic therapy was narrowed to ceftriaxone and vancomycin based on antimicrobial susceptibility testing.

Gradually, the patient's sensorium improved, and he was transferred to the general ward. Antibiotics were continued for 14 days, and dexamethasone was administered for 4 days. At the one-week follow-up, the patient reported residual neck pain and stiffness. Repeat CSF analysis demonstrated a decrease in white blood cell count (111/mm³) and protein level (59 mg/dL), indicating resolving meningitis. Repeat brain MRI showed decreased

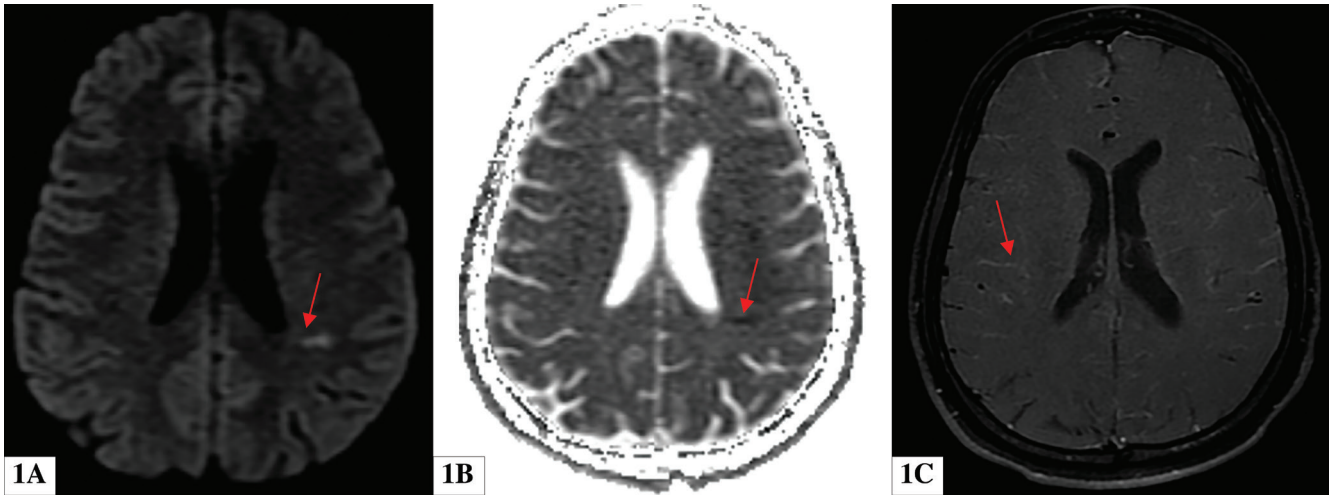

Figure 1. (1A) Brain MRI—axial diffusion-weighted imaging shows a hyperintense signal in the left parietal cortex, consistent with an acute infarct (red arrow). (1B) Brain MRI—corresponding apparent diffusion coefficient map shows a hypointense signal, confirming restricted diffusion (red arrow). (1C) Brain MRI—axial T1-weighted post-contrast image reveals diffuse leptomeningeal enhancement, typical of infectious meningitis (red arrow)
MRI: Magnetic resonance imaging

leptomeningeal enhancement compared with earlier imaging. The patient showed symptomatic improvement and was discharged on aspirin 75 mg once daily and a statin.

Discussion

The most common pathogens causing bacterial meningitis, a condition associated with high morbidity and mortality, include *Streptococcus pneumoniae* (36.5%), *Neisseria meningitidis* (28.8%), and *Streptococcus agalactiae* (15.4%). Cerebrovascular complications—including ischemic and hemorrhagic stroke, intracerebral hemorrhage, and cerebral venous sinus thrombosis—are well-recognized complications of bacterial meningitis and are often associated with poor outcomes. Ischemic stroke, particularly in pneumococcal meningitis, is thought to result from cerebral vasculitis and typically occurs early in the disease course. Up to 10% of bacterial meningitis cases develop ischemic stroke, while neurological complications occur in approximately 15% of cases. Vasculitic complications may present early, as in our case, or may manifest weeks to months later (3,4). Nomura et al. (5) reported progressive vascular involvement in both the anterior and posterior circulations by the second week of illness, as detected on magnetic resonance angiography. Similarly, Dargazanli et al. (4) described a patient who developed aphasia three months after discharge due to severe stenosis of the left carotid and middle cerebral arteries, highlighting delayed-onset vasculopathy.

Cerebrovascular disease in bacterial meningitis can present with variable patterns, affecting cortical regions, deep gray-matter structures such as the basal ganglia, or isolated vascular territories (6). Intracranial and subarachnoid hemorrhages have also been reported, although infrequently, and are typically associated with poor prognosis. The underlying mechanisms remain incompletely understood but likely involve a combination of inflammation-induced endothelial injury, vasospasm, and thrombosis. A review of published case reports (Table 1) highlights the heterogeneity in clinical presentation and imaging findings in pneumococcal meningitis complicated by cerebral vasculitis.

Management of bacterial meningitis complicated by cerebral vasculitis requires a multidisciplinary approach involving neurologists, infectious disease specialists, and intensivists. A high index of suspicion for vasculitis should be maintained in any patient with persistent fever or new neurological deficits despite appropriate antimicrobial therapy. Initial management includes empirical broad-spectrum antibiotics such as ceftriaxone, vancomycin, and ampicillin until culture results are available. Once the causative organism is identified, antibiotic therapy should be tailored accordingly. The blood-brain barrier (BBB) normally prevents pathogens from entering the central nervous system; however, during meningitis, inflammation disrupts the BBB, allowing bacterial invasion and subsequent complications.

Adjunctive corticosteroid therapy, particularly dexamethasone, has been shown to reduce inflammation, cerebral edema, and certain neurological complications in bacterial meningitis, especially in cases caused by *Streptococcus pneumoniae* (7-9). Although the duration and choice of corticosteroid therapy (e.g., dexamethasone versus methylprednisolone) vary across case reports, early administration of dexamethasone has been associated with improved outcomes. In our case, the patient received dexamethasone for four days during the acute phase, in accordance with current recommendations.

Conclusion

Pneumococcal meningitis may be complicated by cerebral vasculitis and infarction early in the disease course, as illustrated in this case. Early neuroimaging, CSF analysis, and timely initiation of appropriate antibiotics and corticosteroids are critical in reducing morbidity. Continued clinical vigilance is essential for early detection

Table 1. Shows a literature review of case reports of pneumococcal meningitis with neurological complications

Serial number	Author	Age/sex	The occurrence of neurological worsening since admission	Treatment given
1	Corchia et al. (3)	48/female	Day 1 of admission with confusion and MRA showing stenosis of the anterior and middle cerebral artery	Dexamethasone for 21 days
2	Dargazanli et al. (4) (2021)	34/male	Ninety days since discharge, presenting with acute aphasia	High-dose steroids, along with antibiotics and antiplatelets
3	Nomura et al. (5)	60/female	Altered mental status with imaging showing vasoconstriction in the anterior and posterior circulation	Intravenous methylprednisolone followed by oral steroids
4	Poil et al. (9) (2018)	48/male	Only persistent fever spikes since four days of admission, with imaging showing thalamic and basal ganglia lacunar infarction	Steroids restarted

MRA: Magnetic resonance angiography

of complications and prompt escalation of therapy when required.

The medical team managing bacterial meningitis should maintain a high index of suspicion for cerebral vasculitis in patients with pneumococcal meningitis who exhibit persistent fever spikes or any neurological deterioration despite appropriate antibiotic therapy. Early identification and timely initiation of corticosteroid therapy may reduce long-term neurological sequelae and improve hospitalization outcomes.

Ethics

Informed Consent: Written informed consent was obtained prior to publication, and all identifying patient details were anonymized in the manuscript.

Footnotes

Authorship Contributions

Surgical and Medical Practices: I.L., V.P., A.C., D.S., Concept: I.L., V.P., A.C., D.S., Design: I.L., V.P., A.C., D.S., Data Collection or Processing: I.L., V.P., A.C., D.S., Analysis or Interpretation: I.L., V.P., A.C., D.S., Literature Search: I.L., V.P., A.C., D.S., Writing: I.L., V.P., A.C., D.S.

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