IASR J. Med. Pharm. Sci.
ISSN: 2582-9343
Website: https://ijmps.com/
Open Access Journal
Country: India

IASR Journal of
Medical and
Pharmaceutical Science

CASE REPORT OPEN ACCESS

Suspected Spontaneous Cerebrospinal-Fluid Rhinorrhea Preceding Pneumococcal Meningitis Complicated by Multifocal Cerebral Infarction and Hydrocephalus: A Case Report

Dr Sonia D Pawaskar[1], Dr Parul[2], Dr Rahul D Pawaskar[3]

[1]Associate Consultant, Department of Emergency Medicine, Manipal Hospital Goa
[2] Department of Neurology, Manipal Hospital Goa
[3] Senior Resident, Department of General Surgery, AJ Institute of Medical Science, Mangalore

OPEN ACCESS

Corresponding Author
Dr Sonia D Pawaskar
Associate Consultant, Department of Emergency Medicine, Manipal Hospital Goa

Received: 05 Jul 2026
Revised: 15 Jul 2026
Accepted: 20 Aug 2026
Published: 10 Sept 2026

ABSTRACT

Streptococcus pneumoniae remains the leading cause of community-acquired bacterial meningitis in adults and is frequently associated with severe neurological complications despite appropriate antimicrobial therapy. We report a 48-year-old woman who presented with a two-day history of isolated rhinorrhea treated as a simple upper respiratory tract infection, followed by rapid progression to fever and altered sensorium. Neuroimaging revealed bilateral thalamic infarcts involving the posterior limb of the internal capsule and left superior cerebellum, consistent with extensive inflammatory vasculopathy.
Cerebrospinal fluid culture confirmed pneumococcal meningitis. Her clinical course was further complicated by communicating hydrocephalus requiring endoscopic third ventriculostomy and external ventricular drainage. Despite the severity of vascular and cerebrospinal fluid complications, she demonstrated meaningful neurological recovery at 1.8 years from symptom onset, obeying commands and ambulating with support. This case highlights the fulminant evolution of pneumococcal meningitis from seemingly minor upper respiratory symptoms to devastating central nervous system injury, while emphasizing that substantial long-term functional recovery is achievable with timely, aggressive, and multidisciplinary management.

Key Words: *Pneumococcal meningitis, CSF rhinorrhea, hydrocephalus, vasculopathy*

INTRODUCTION

Acute bacterial meningitis is a life-threatening neurological emergency characterized by high mortality and significant long-term neurological sequelae, with Streptococcus pneumoniae remaining the most common etiological agent in adults worldwide. The disease process is driven by an intense inflammatory cascade within the subarachnoid space, resulting in blood–brain barrier disruption, cerebral edema, endothelial injury, and inflammatory vasculitis. These pathophysiological mechanisms predispose to thrombosis of small perforating vessels, impaired cerebral autoregulation, and secondary ischemic injury. Deep gray matter structures, particularly the thalami, are especially vulnerable due to their end-arterial blood supply from small perforators lacking significant collateral circulation. Concurrently, severe meningeal inflammation may obstruct arachnoid villi function, impairing cerebrospinal fluid (CSF) resorption and leading to communicating hydrocephalus.

We report the case of a 48-year-old woman who initially presented with a two-day history of isolated rhinorrhea, which was managed as a benign upper respiratory tract infection, before rapidly progressing to fever and altered sensorium. There was no history of fall or trauma. Neuroimaging demonstrated bilateral thalamic infarcts involving the posterior limb of the internal capsule and the left superior cerebellum, suggestive of extensive inflammatory vasculopathy affecting the perforating vessels. Cerebrospinal fluid analysis and culture confirmed pneumococcal meningitis.

Within two days, her condition worsened, necessitating hospital admission. During the course of her illness, she developed seizures and a progressive decline in consciousness, eventually becoming mute with quadriplegia. Her clinical course was marked by significant central nervous system injury, multiple hospital admissions, and delayed neurological recovery. Appropriate investigations were carried out as part of her evaluation and management.

During hospitalization, she developed communicating hydrocephalus, requiring endoscopic third ventriculostomy and external ventricular drainage. Despite severe vascular involvement and cerebrospinal fluid flow complications, the patient exhibited delayed but meaningful neurological recovery at 1.8 years from symptom onset, regaining the ability to follow commands and ambulate with support. This case highlights the fulminant progression of pneumococcal meningitis from seemingly trivial upper respiratory symptoms to severe cerebrovascular and CSF-related complications, while emphasizing the potential for significant long-term functional recovery with timely antimicrobial therapy, close neurological monitoring, and multidisciplinary critical care management.

PATHOPHYSIOLOGY

Etiology (Non-Traumatic CSF Rhinorrhea)

In a spontaneous (non-traumatic) CSF leak, the most common cause is Idiopathic Intracranial Hypertension (IIH), where chronically high brain pressure acts as a "slow-motion blowout" that thins and eventually ruptures the skull base (1). This process is frequently associated with obesity and Empty Sella Syndrome[13], where high pressure flattens the pituitary gland (2). Other causes include congenital bone defects, where small "potholes" in the skull develop over time, and connective tissue disorders like Marfan syndrome that weaken the protective dural lining (3). In the spine, leaks are often triggered by bone spurs piercing the dura or abnormal CSF-venous fistulas (3).

The "Erosive Pressure" Mechanism

The primary driver of spontaneous cerebrospinal fluid (CSF) rhinorrhea is chronically elevated intracranial pressure (ICP), typically associated with Idiopathic Intracranial Hypertension (IIH). The skull base, particularly the cribriform plate and the lateral recess of the sphenoid sinus, acts as a point of least resistance. Constant CSF pulsations and elevated hydrostatic pressure lead to the gradual thinning (dehiscence) of these bony partitions. Over time, this pressure causes the dura mater and arachnoid membrane to herniate through these focal defects, forming small meningoceles or meningoencephaloceles that eventually rupture, manifesting as watery rhinorrhea [1, 2].

The Role of Obesity and Venous Pressure

Epidemiological data strongly correlate spontaneous leaks with high Body Mass Index (BMI). In these patients, increased intra-abdominal and intra-thoracic pressure impairs venous return via the jugular system, subsequently elevating intracranial venous pressure and CSF volume. This chronic "over-inflation" of the subarachnoid space necessitates a pressure-release mechanism, which the skull base eventually provides [3].

Pneumococcal meningitis [12]can progress rapidly following minor respiratory symptoms. The inflammatory response results in endothelial dysfunction and vasculitis, predisposing to thrombosis of perforating vessels supplying deep brain structures such as the thalami.

Communicating hydrocephalus develops due to impaired CSF absorption secondary to meningeal inflammation. Early neurosurgical intervention is crucial in preventing further neurological deterioration.

Although bilateral thalamic infarcts are often associated with poor prognosis, this case highlights that meaningful delayed recovery is possible with aggressive multidisciplinary management and rehabilitation.

Vasculopathy in Pneumococcal Meningitis

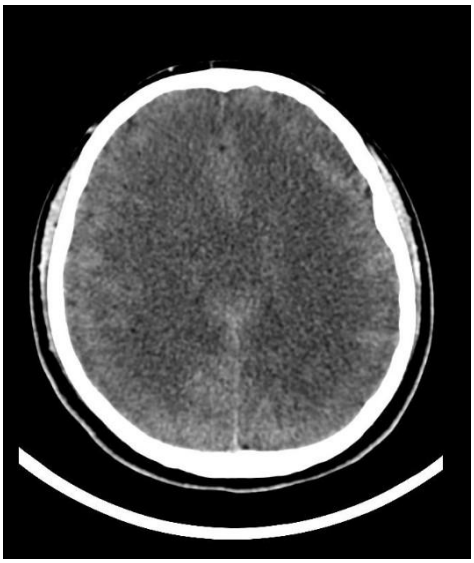
Cerebrovascular complications are a well-recognized manifestation of pneumococcal meningitis, with inflammatory vasculopathy reported in approximately 15–30%[8,9] of cases. The underlying mechanism involves intense meningeal inflammation leading to endothelial injury, vessel wall inflammation, and subsequent thrombosis of small perforating arteries. This process predisposes to ischemic infarctions, particularly in deep

gray matter structures such as the thalami, which are supplied by end-arterial perforators[10] and lack significant collateral circulation.

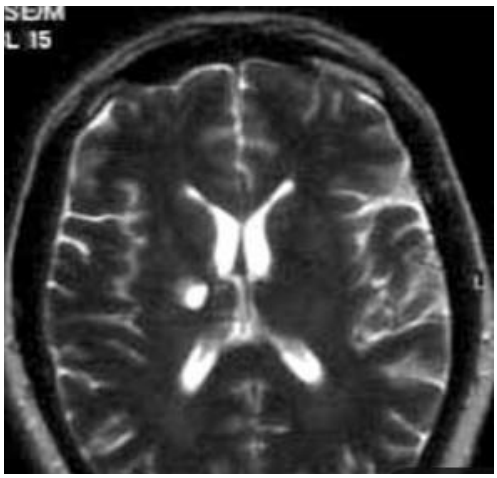
Were other vasculitides ruled out?

In the present case, alternative primary vasculitic etiologies were not extensively pursued, as the diagnosis of pneumococcal meningitis provided a well-established and sufficient explanation for the observed cerebrovascular complications. The pattern of multifocal infarcts involving deep perforator territories, in conjunction with an acute fulminant clinical course, strongly favored infection-associated inflammatory vasculopathy.
Furthermore, in the setting of critical illness, additional evaluation for primary central nervous system vasculitis was unlikely to alter immediate management and was therefore not prioritized.

Investigations



CT Brain (18 September 2023): Diffuse cerebral edema with effacement of cortical sulci and bilateral slyvian fissure ,diffuse cerebral edema.



MRI Brain (22 September 2023): Acute infarction involving the right thalamus with bilateral hemispheric white matter changes.

MRI Stroke Protocol (26 September 2023): Diffusion restriction in bilateral anterior thalami, posterior limb of internal capsule, and left superior cerebellum, consistent with multifocal acute infarcts secondary to inflammatory vasculopathy.

Cerebrospinal Fluid Analysis: Culture positive for Streptococcus pneumoniae. Tuberculosis testing negative.
TREATMENT

Antiepileptic therapy. Patient was started on inj zaxter 2gm iv tds with inj monocef 2gm iv bd. pt was also on inj dexa 4mg tds initially and later tapered.

Serial imaging later demonstrated progressive ventriculomegaly consistent with communicating hydrocephalus. She underwent a left frontal burr hole procedure with endoscopic third ventriculostomy and external ventricular drainage under general anesthesia.

DISCUSSION

Spontaneous cerebrospinal fluid (CSF) rhinorrhoea occurs rarely .Cerebrospinal fluid (CSF) Rhinorrhoea is the leakage of CSF through nasal cavity, due to abnormal communication between the arachnoid membrane and nasal mucosa.

Middle-age (fourth to fifth decade) group, female gender, and obesity (body mass index > 40) are the most commonly reported risk-factors for this rare entity.

CSF rhinorrhea can also be caused by a pre-existing weakness of the meninges combined with rapid violence.
Intracranial pressure changes lead the dura mater to gradually herniate into the bone fissure, causing the dura to thin over time.
A deteriorating dural structure can easily result in diverticula or expansions, as well as an increased chance of dural rupture, which can cause CSF leakage into the epidural area.

The majority of leaks are caused by trauma, both inadvertent and iatrogenic, and CSF rhinorrhea is most usually produced by trauma involving the skull base and face fractures. Nasal leakage is routinely disregarded in people who have no history of trauma.
Cerebrospinal fluid (CSF) rhinorrhea, when left untreated, can lead to meningitis and other serious complications.

Rationale for Not Using Acetazolamide

The use of acetazolamide was not considered in this patient due to the acute and severe nature of intracranial pathology. In the setting of pneumococcal meningitis complicated by diffuse cerebral edema, multifocal infarcts, and evolving hydrocephalus, acetazolamide offers limited therapeutic benefit as the primary pathology involves impaired cerebrospinal fluid absorption and inflammatory obstruction rather than overproduction. Furthermore, its potential to induce metabolic acidosis may exacerbate cerebral edema and adversely affect intracranial dynamics. Given the patient's rapid neurological deterioration and progression to communicating hydrocephalus, definitive neurosurgical intervention with cerebrospinal fluid diversion was deemed more appropriate than medical management.

Why was beta transferrin not done?

On presentation there was no active rhinorrhea hence beta transferrin was not done Radiographic Markers of High Pressure
On imaging, this etiology is supported by indirect signs of intracranial hypertension, most notably Empty Sella Syndrome. This occurs when high-pressure CSF flattens the pituitary gland against the sellar floor. Other markers include perioptic nerve sheath prominence and the presence of arachnoid pits or "scalloping" along the inner table of the sphenoid bone, confirming that the leak is a symptom of a systemic pressure disorder rather than an isolated traumatic event [1, 4].

Secondary Signs (High Pressure): There is evidence of a partially empty sella turcica, with the pituitary gland flattened against the sellar floor. This finding, combined with the spontaneous nature of the leak, is consistent with underlying intracranial hypertension.

CONCLUSION

This case profoundly emphasizes how a seemingly trivial symptom such as isolated watery rhinorrhea can precede a catastrophic neurological event. It highlights the unpredictable and aggressive nature of acute bacterial meningitis, where rapid inflammatory injury can lead to devastating cerebral complications with long-lasting functional consequences. At the

same time, it underscores a powerful and often overlooked message: even after severe neurological insult, meaningful recovery remains possible. The trajectory from critical illness to gradual functional improvement reflects the resilience of the human brain and the importance of sustained, comprehensive care. Ultimately, this case serves as a reminder that early clinical vigilance can prevent morbidity, and that hope for recovery should persist even in the face of profound neurological injury.

OUTCOME AND FOLLOW UP

At discharge, she had significant neurological deficits; however, at 1.8 years of follow-up, she showed substantial neurological improvement, obeying simple commands and ambulating with support.

PREVENTION

Prevention of cerebrovascular complications in pneumococcal meningitis is centered on early recognition and timely initiation of appropriate antimicrobial therapy to attenuate the inflammatory cascade. The adjunctive use of corticosteroids, particularly dexamethasone administered prior to or concomitantly with the first dose of antibiotics, has been shown to mitigate inflammation-mediated endothelial injury[csf] and reduce the risk of neurological sequelae. Vigilant neurological monitoring, coupled with early neuroimaging, facilitates prompt detection of evolving vasculopathy and raised intracranial pressure, allowing timely escalation to intensive care and neurosurgical interventions when required. At a population level, pneumococcal vaccination remains the most effective strategy for reducing disease burden and preventing associated vascular complications.

Clinical Differentiation: CSF Rhinorrhea vs Simple Cold

This case highlights the critical importance of carefully differentiating cerebrospinal fluid (CSF) rhinorrhea from a seemingly benign upper respiratory tract infection at the initial point of contact. Unlike a simple cold, CSF rhinorrhea typically presents as a persistent, unilateral, clear watery nasal discharge that lacks the accompanying features of viral illness such as nasal congestion, sneezing, sore throat, or mucopurulent secretions. A key distinguishing clinical feature is its positional nature, with patients often reporting increased leakage on bending forward, straining, or performing Valsalva maneuvers—the so-called “reservoir sign.” The discharge may also be described as having a salty or metallic taste and tends to be continuous rather than intermittent. Importantly, the absence of systemic symptoms early in the course and the failure to respond to standard symptomatic therapy should raise suspicion. Subtle historical clues, including spontaneous onset without typical infective prodrome or a history of trauma, prior surgery, or conditions predisposing to raised intracranial pressure, further support the diagnosis. Early recognition of these distinguishing features is crucial, as missed or misdiagnosed CSF rhinorrhea can serve as a direct portal for infection, predisposing to serious complications such as pneumococcal meningitis. This underscores the need for heightened clinical vigilance and a low threshold for diagnostic evaluation in patients presenting with atypical rhinorrhea.

MULTIDISCIPLINARY CONTRIBUTION TO RECOVERY

Beyond appropriate antimicrobial therapy, the patient’s favorable neurological recovery was driven by a coordinated multidisciplinary approach. Aggressive neurocritical care, including intracranial pressure management with osmotherapy and corticosteroids, played a pivotal role in limiting secondary brain injury. Timely neurosurgical intervention, in the form of external ventricular drainage and endoscopic third ventriculostomy, was crucial in addressing evolving communicating hydrocephalus. In addition, effective seizure control with antiepileptic agents, meticulous correction of metabolic and electrolyte disturbances, and comprehensive supportive care—including respiratory support, nutritional optimization, and rehabilitation—contributed significantly to recovery. The integrated efforts of emergency medicine, neurology, neurosurgery, and critical care teams were instrumental in achieving a meaningful functional outcome despite the severity of the initial insult.

References

1. van de Beek D, de Gans J, Tunkel AR, Wijdicks EFM. Community-acquired bacterial meningitis in adults. N Engl J Med. 2006;354:44–53.
2. Pfister HW, Borasio GD, Dirnagl U, Bauer M, Einhäupl KM. Cerebrovascular complications of bacterial meningitis in adults. Neurology. 1992;42:1497–1504.

3. Kasanmoentalib ES, Brouwer MC, van de Beek D. Hydrocephalus in bacterial meningitis. Neurology. 2010;75:918–923.
4. de Gans J, van de Beek D. Dexamethasone in adults with bacterial meningitis. N Engl J Med. 2002;347:1549–1556.

5. Thrupthi S, Kumar MJ. Spontaneous cerebrospinal fluid rhinorrhoea: A case report and management. *J Res Med Dent Sci*. 2021;9(11):257–259.6.Mokri B. The Monro-Kellie hypothesis: applications in CSF volume depletion. *Neurology*. 2001;56(12):1746–1748.

6. Schlosser RJ, Wilensky EM, Grady MS, et al. Spontaneous cerebrospinal fluid rhinorrhea: A manifestation of benign intracranial hypertension. *Laryngoscope*. 2003;113(12):2088–2093.

7. Brouwer MC, van de Beek D.Cerebral vasculopathy in bacterial meningitis. *Curr Opin Neurol*. 2010;23(3):292–7.

8. Klein M, Koedel U, Pfister HW.Cerebrovascular complications of bacterial meningitis in adults. *Neurology*. 2011;76(4):e23–5.

9. Carrera E, Bogousslavsky J. The thalamus and its vascular syndromes. *Neurology*. 2006;66(7):962–9.

10. Tunkel AR, van de Beek D, Scheld WM. Acute meningitis. *N Engl J Med*. 2004;351(18):1849–59.

11. Prosser JD, Vender JR, Solares CA. Traumatic cerebrospinal fluid leaks. *Otolaryngol Clin North Am*. 2011;44(4):857–73.

12. Schlosser RJ, Bolger WE. Spontaneous nasal cerebrospinal fluid leaks and empty sella syndrome. *Am J Rhinol*. 2003;17(2):91–6.