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(CASE REPORT)

Pneumococcal meningitis complicated by cerebral venous thrombosis, intraparenchymal hematoma, and delayed cerebral vasculopathy: A case report

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Abstract

Case presentation: A 59-year-old woman with Parkinson disease developed pneumococcal meningoencephalitis complicated by thrombosis of both sigmoid sinuses and the right transverse (lateral) sinus, followed 10 days later by a right frontal intraparenchymal hematoma with intraventricular extension and acute tetraventricular hydrocephalus requiring surgical evacuation and external ventricular drainage.

Discussion: Subsequent neuroimaging showed persistent hydrocephalus, multiple hemorrhagic lesions, diffuse cerebral edema, brain herniation, bilateral cerebellar ischemic lesions, and focal arterial stenoses suggestive of associated inflammatory cerebral vasculopathy. This case highlights the severe venous–hemorrhagic–arterial continuum that may complicate pneumococcal meningitis and underscores the therapeutic uncertainty surrounding anticoagulation in septic cerebral venous thrombosis.

Conclusion: Repeated multimodal neuroimaging, early neurosurgical assessment, and aggressive neurocritical care are crucial, although prognosis remains poor once intraparenchymal hematoma, hydrocephalus, and herniation syndromes have developed.

Keywords: Bacterial meningitis; *Streptococcus pneumoniae*; Cerebral venous thrombosis; Intraparenchymal hematoma; Hydrocephalus; intensive care

1. Introduction

Community-acquired bacterial meningitis in adults remains a diagnostic and therapeutic emergency with substantial mortality. *Streptococcus pneumoniae* remains one of the most frequently implicated pathogens and one of those most strongly associated with severe neurological complications [5-8].

Cerebrovascular complications occur in approximately one quarter of patients with bacterial meningitis and are dominated by arterial ischemic stroke, whereas cerebral venous thrombosis (CVT) and intracerebral hemorrhage are less common but particularly devastating [1,2,5,11].

We report the case of pneumococcal meningitis successively complicated by extensive CVT, intraparenchymal hematoma with intraventricular extension, acute hydrocephalus, malignant intracranial hypertension, and probable secondary arterial vasculopathy. The originality of this case lies in the documented sequence of venous, hemorrhagic, and arterial events during the same intensive care stay.

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1.1. Pathophysiology of neurovascular complications

Neurovascular complications of pneumococcal meningitis result from a complex interaction between meningeal inflammation, endothelial injury, local and systemic activation of coagulation, altered cerebral autoregulation, and intracranial hypertension [5,6,11].

CVT may be promoted by inflammatory extension to the dural sinuses, venous stasis, sepsis-related prothrombotic activation, and endothelial wall injury. The lateral and sigmoid sinuses are among the most frequently involved sites in published series [1].

Intraparenchymal hematoma may result from several non-mutually exclusive mechanisms: hemorrhagic venous infarction, microvascular rupture due to venous congestion, inflammatory vascular fragility, hemorrhagic transformation of ischemia, or aggravation of bleeding risk under anticoagulation in already injured brain tissue [1-4,14,15].

A delayed cerebral arterial vasculopathy may also occur after the initial infectious phase and is responsible for focal stenoses and secondary infarctions. This complication is increasingly recognized in severe pneumococcal meningitis [9,12,13].

2. Case presentation

A 59-year-old woman with Parkinson disease was admitted to a private hospital for impaired consciousness (day 1 of admission), with an initial Glasgow Coma Scale score of 8. Cerebrospinal fluid (CSF) analysis and culture established the diagnosis of *Streptococcus pneumoniae* meningoencephalitis.

Initial CSF analysis showed 800 leukocytes/mm^3 with a mixed differential including 56% lymphocytes, CSF glucose 0.01 g/L with simultaneous blood glucose 2.7 g/L, protein 5 g/L, and chloride 118 mmol/L. Brain MRI on the day of admission showed thrombosis of both sigmoid sinuses and the right transverse (lateral) sinus, diffuse supra- and infratentorial leptomeningitis, and signs of intracranial hypertension.

Table 1 Clinical, radiological, and therapeutic chronology

Timeline	Key event	Diagnostic/therapeutic consequence
Day 1 (admission)	Admission for altered consciousness (GCS 8).	CSF-confirmed pneumococcal meningoencephalitis; initial MRI showed CVT of both sigmoid sinuses and the right transverse (lateral) sinus, diffuse leptomeningitis, and signs of intracranial hypertension; high-dose ceftriaxone, gentamicin, corticosteroids, and therapeutic anticoagulation were initiated.
Day 10	Anisocoria and abnormal transcranial Doppler findings.	CT scan showed a right frontal hematoma with intraventricular flooding and tetraventricular hydrocephalus.
Day 10	Neurosurgical worsening.	Surgical evacuation of the hematoma and placement of an external ventricular drain (EVD), followed by transfer to the university hospital ICU.
Day 11	Postoperative reassessment.	CT scan showed partial hematoma regression, persistent hydrocephalus, and a 20-mm epidural hematoma; CSF obtained from the EVD showed partial biological improvement.
At transfer to the university hospital	No awakening despite sedation discontinuation for 3 days; GCS 3.	EVD in place, reactive semi mydriasis, increased transcranial Doppler velocities; continuation of neurocritical care.
Day 14	Worsening follow-up CT scan.	Multiple right frontoparietal hematomas with ventricular rupture, cerebral edema, herniation, bilateral cerebellar infarcts, and stenoses of both anterior cerebral arteries; no further neurosurgical indication.
Day 21	Prolonged respiratory support.	Tracheostomy; pressure-support ventilation with PEEP under FiO_2 50%.
After 1 month in ICU	No neurological recovery.	Death in the setting of ventilator-associated pneumonia.

The patient initially received meningitis-dose ceftriaxone, gentamicin, corticosteroids, and therapeutic anticoagulation. Ten days after admission, anisocoria associated with abnormal transcranial Doppler findings prompted emergency brain CT, which revealed a right frontal intraparenchymal hematoma with intraventricular extension and acute tetraventricular hydrocephalus.

Surgical evacuation of the hematoma and placement of an external ventricular drain (EVD) were performed before transfer to the university hospital for further management. Follow-up CT on postoperative day 1 showed partial regression of the hematoma, persistent hydrocephalus, and a 20-mm epidural hematoma. CSF sampled from the EVD contained 8 leukocytes/mm^3, 2,340 red blood cells/mm^3, glucose 1.06 g/L, protein 3 g/L, and chloride 80 mmol/L.

On admission to the university hospital ICU, sedation had been discontinued for 3 days without any sign of awakening. Neurological examination showed a Glasgow Coma Scale score of 3, reactive semi mydriasis, an abnormal transcranial Doppler with increased velocities, and an EVD in place. CSF analysis showed 35 leukocytes/mm^3, 5,000 red blood cells/mm^3, glucose 1 g/L, protein 2.8 g/L, and chloride 84 mmol/L.

Follow-up brain CT performed on day 14 demonstrated multiple right frontoparietal intraparenchymal hematomas, some confluent, with ventricular rupture and tetraventricular flooding. Additional findings included subfalcine, uncal, and diencephalic herniation, diffuse cerebral edema, bilateral subacute cerebellar ischemic lesions in the posterior inferior cerebellar artery territories, diffuse reduction in caliber of the arteries of the circle of Willis with focal stenoses of both anterior cerebral arteries, heterogeneous appearance of the right sigmoid sinus, and persistent postoperative changes with a 14-mm epidural hematoma.

After neurosurgical reassessment, no further operative intervention was indicated. Hemodynamically, the patient remained stable under low-dose norepinephrine for blood pressure targets. Respiratory support required tracheostomy on day 21; the patient remained on pressure-support ventilation with PEEP and FiO_2 50%. Treatment included ceftriaxone 4 g every 12 h, enoxaparin 0.6 mL every 12 h, sodium valproate, methylprednisolone, and enteral plus parenteral nutrition. No neurological improvement occurred, and the patient died after one month in the ICU from ventilator-associated pneumonia.

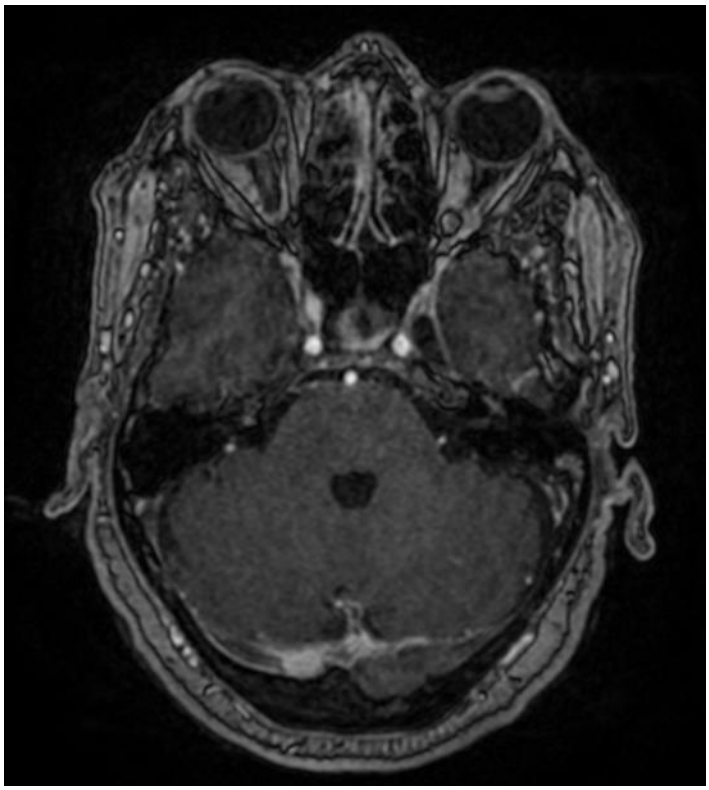

Figure 1 Brain MRI, axial 3D T1-weighted gadolinium-enhanced sequence: filling defect of the right transverse/lateral sinus consistent with cerebral venous thrombosis

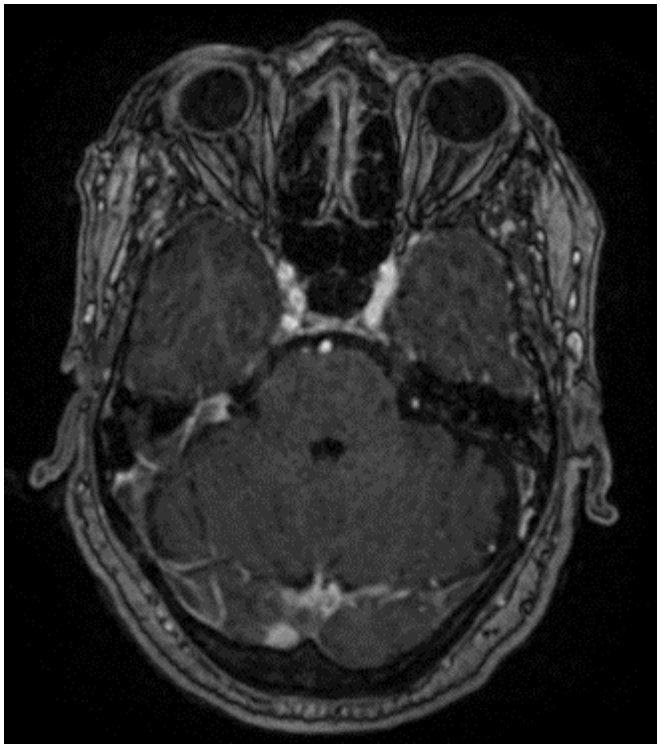

Figure 2 Brain MRI, axial post-contrast T1-weighted sequence: abnormal enhancement in the right latero-sigmoid region consistent with extension of the venous thrombosis

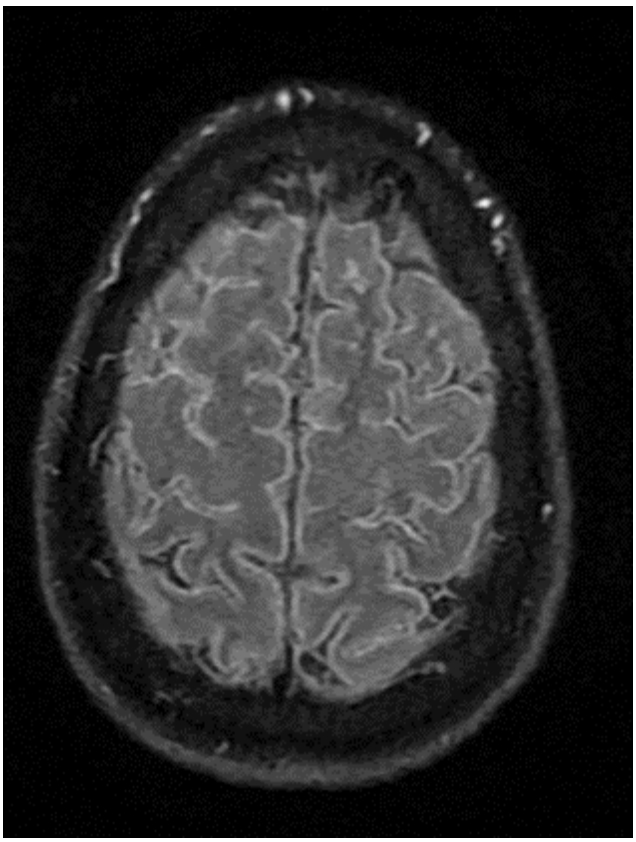

Figure 3 Brain MRI, axial T2-FLAIR sequence: bilateral gyriform hyperintensity of the frontal cortical sulci, more marked on the right, consistent with leptomeningitis

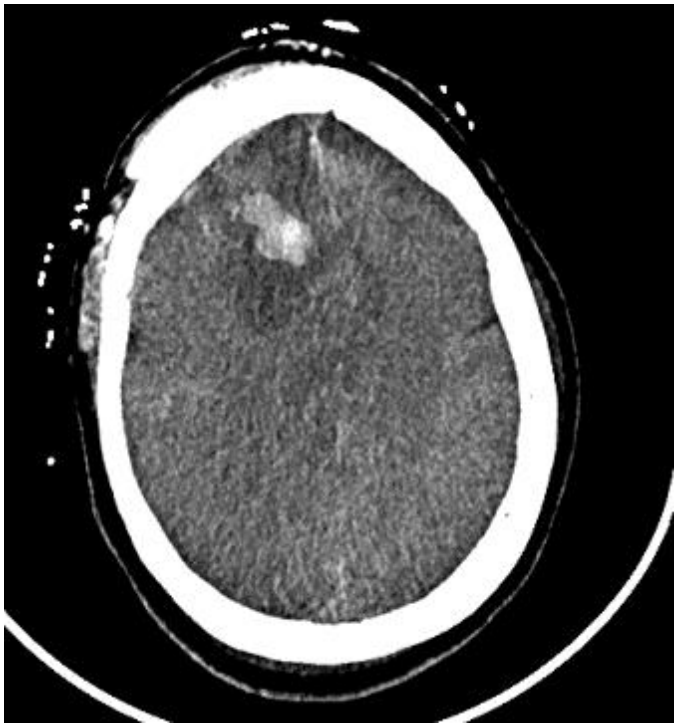

Figure 4 Brain CT, axial non-contrast slice showing a right frontal intraparenchymal hematoma surrounded by hypodense perilesional edema, with mass effect and ventricular extension

3. Discussion

The scientific value of this case lies less in the isolated rarity of each complication than in their documented sequence during a single intensive care stay: extensive septic CVT, intraparenchymal hematoma with ventricular flooding, acute hydrocephalus, malignant intracranial hypertension, diffuse cerebral edema, and subsequent arterial narrowing with ischemic lesions [1,2,5,7,9,12,15].

In the historical series by Kastenbauer and Pfister, intracranial complications occurred in 74.7% of patients, including 28.7% diffuse cerebral edema, 16.1% hydrocephalus, 21.8% arterial complications, 10.3% venous complications, and 9.2% spontaneous intracranial hemorrhage, emphasizing that our observation belongs to the most aggressive cerebrovascular spectrum of pneumococcal meningitis [7].

The sigmoid–lateral location of CVT in our patient is consistent with published series [1,7]. CVT should be suspected in any patient with bacterial meningitis who develops unexplained neurological worsening, anisocoria, intracranial hypertension, focal deficits, or deterioration despite appropriate antimicrobial therapy [1,5]. In the recent ICU cohort reported by Legouy et al., CVT accounted for 9% of intracranial complications and intracranial complications overall were independently associated with poor 90-day neurological outcome [9].

3.1. Anticoagulation: the central therapeutic dilemma

Anticoagulation is the main management issue raised by this case. In non-septic CVT, international guidelines support acute-phase parenteral anticoagulation and reserve decompressive surgery for selected patients with impending herniation [3,4].

By contrast, in CVT associated with bacterial meningitis, the benefit–risk balance must be individualized because intracranial bleeding risk may be amplified by cerebral inflammation, venous congestion, and associated parenchymal lesions [1-4]. The data reported by Mook-Kanamori et al. warrant caution: in a nationwide cohort of 860 episodes of bacterial meningitis, intracranial hemorrhage occurred in 2.8% of cases, with 63% mortality and 96% unfavorable outcome; anticoagulant use at admission was associated with a significantly increased hemorrhagic risk (odds ratio 5.84, 95% confidence interval 2.17-15.76) [14].

In our patient, the delayed intraparenchymal hematoma may have resulted from hemorrhagic venous infarction, severe venous congestion, or inflammatory vascular injury. A facilitating role of anticoagulation cannot be confirmed, but it cannot be excluded either. This case therefore illustrates the therapeutic gray zone represented by septic CVT complicating bacterial meningitis, where radiological dynamics and multidisciplinary reassessment are essential.

3.2. Venous-hemorrhagic-arterial pathophysiological continuum

The demonstrative nature of this observation stems from the coexistence of three intertwined mechanisms: venous thrombosis, parenchymal bleeding, and secondary arterial vasculopathy. Rather than independent complications, these events may represent different expressions of a shared thrombo-inflammatory cascade.

The neuropathological study by Vergouwen et al. provides an important mechanistic perspective: among 16 patients who died from pneumococcal meningitis, large-vessel vasculitis was not found, whereas arterial thrombosis, venous thrombosis, and diffuse intravascular coagulation with vessel clogging were frequent [15]. This observation supports the concept that hemorrhagic, ischemic, and thrombotic lesions may coexist in the same patient because of diffuse endothelial and microvascular dysfunction rather than isolated vasculitis alone.

In addition, the diffuse reduction in caliber of the arteries of the circle of Willis and the bilateral anterior cerebral artery stenoses observed on follow-up CT are compatible with delayed cerebral vasculopathy, a severe complication increasingly reported in pneumococcal meningitis [9,12,13]. The bilateral cerebellar ischemic lesions in the posterior inferior cerebellar artery territories strengthen this interpretation.

3.3. Implications for neurocritical care

Beyond pathophysiological discussion, this case is fundamentally a neurocritical care case. It emphasizes the need for repeated neurological reassessment despite sedation, the value of transcranial Doppler in detecting hemodynamic deterioration, and the importance of repeated neurovascular imaging when the clinical course deviates from the expected trajectory.

It also highlights the practical challenges of balancing cerebral perfusion pressure, blood pressure targets, ventilation, hydrocephalus management, and timing of neurosurgical procedures. In this setting, external ventricular drainage was justified by acute hydrocephalus with intraventricular hemorrhage, whereas the absence of a second neurosurgical indication reflected the diffuse and advanced nature of the lesions.

The initially low CSF leukocyte count in our patient (<1000/mm^3) is also noteworthy. Historical and contemporary series have suggested that a blunted early CSF inflammatory response may be associated with more severe intracranial complications and worse prognosis [7,9,14,15].

Limitations

This report has several limitations. It does not allow precise attribution of the relative causal contribution of CVT, anticoagulation, and inflammatory vasculopathy to the occurrence of intracerebral bleeding. It also lacks advanced multimodal neuromonitoring and histopathological confirmation of the underlying vascular process.

Nevertheless, the clinical and radiological chronology, the consistency of the imaging findings, and the concordance with published series give this observation substantial illustrative value. Prognosis appears especially poor when intracranial hemorrhage, hydrocephalus, herniation signs, and prolonged absence of neurological recovery coexist.

4. Conclusions

Streptococcus pneumoniae meningitis may evolve into a fulminant neurovascular syndrome associating CVT, intraparenchymal hematoma, hydrocephalus, malignant cerebral edema, and secondary arterial vasculopathy [1-5,9,12,14,15].

This case reminds us that management rapidly extends beyond infection control alone and fully enters the field of neurocritical care, requiring repeated imaging, close coordination between intensivists, neuroradiologists, and neurosurgeons, and individualized therapeutic decisions—particularly regarding anticoagulation.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare they have no competing interests.

Statement of informed consent

Written informed consent for publication of anonymized clinical data and imaging was obtained

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Author contributions

All authors contributed to the conception of the manuscript, acquisition of clinical data, drafting or critical revision of the text, and approval of the final version.

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