A Challenging Case of *Streptococcus pneumoniae* Meningitis in a 64-Year-Old Woman Who Presented with Symptoms of Cerebellar Hemorrhage

**Patient:** Female, 64-year-old

**Final Diagnosis:** Bacterial meningitis

**Symptoms:** Altered mental status • headache

**Medication:** —

**Clinical Procedure:** —

**Specialty:** Infectious Diseases

**Objective:** Rare disease

**Background:** There is a recognized association between bacterial meningitis and intracranial hemorrhage. However, acute neurological symptoms at presentation, with confirmation of hemorrhage on imaging, may delay further investigations, including blood culture for diagnosing an infection. This report presents a challenging case of *Streptococcus pneumoniae* meningitis in a 64-year-old woman who presented with symptoms of cerebellar hemorrhage.

**Case Report:** This report describes a 64-year-old woman who had a medical history of untreated diabetes mellitus. She was brought to our hospital with headache and impaired consciousness, complicated with fever. Based on the hemorrhage in the left cerebellar hemisphere detected in the head CT findings, the patient was initially diagnosed with cerebellar hemorrhage. However, a positive blood culture after 12 hours of admission made the physician consider a central nervous system infection as the cause of the hemorrhage and perform a lumbar puncture. Therefore, the patient was diagnosed with acute bacterial meningitis caused by *Streptococcus pneumoniae*, and antibiotic treatment was started immediately. Although her general condition improved after antibiotic treatment, her mental status did not improve completely.

**Conclusions:** This report highlights that the clinicians should be aware that bacterial meningitis may result in intracranial hemorrhage. Patients with symptoms of a hemorrhagic stroke should be thoroughly investigated to avoid a delay in the treatment of infection.

**Keywords:** Intracranial Hemorrhage, Hypertensive • Meningitis, Bacterial • Brain Hemorrhage, Traumatic • *Streptococcus pneumoniae*

**Abbreviations:** GCS – Glasgow Coma Scale; CRP – C-reactive protein; CT – computed tomography; MRI – magnetic resonance imaging; MIC – minimum inhibitory concentration

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Background

Bacterial meningitis is a life-threatening disease with an incidence of 2 cases per 100,000 adults worldwide [1]. Approximately 400 adults suffer yearly from bacterial meningitis in Japan [2]. The mortality rate of acute untreated bacterial meningitis is 100% [3]. Additionally, despite the availability of effective antibiotics and intensive neurological care, the overall mortality remains high, with 17-34% of survivors displaying persistent unfavorable neurological outcomes [4-6].

The clinical symptoms of bacterial meningitis include fever, nuchal rigidity, and an altered mental status [4]. Bacterial meningitis can also be complicated by cerebrovascular disorders, including ischemic strokes, intracerebral hemorrhage, and cerebral sinus thrombosis, which are major contributing factors to high disability and mortality among patients [7]. Cerebrovascular complications and ischemic strokes are common in patients with bacterial meningitis, occurring in up to 15% and 10% of cases, respectively [7,8]. Although a study reported that only 2.3% of patients display cerebellar hemorrhage as a complication of bacterial meningitis, the prognosis is poor, requires timely diagnosis, and necessitates prompt treatment [7]. Previous studies suggested that infectious intracranial aneurysms and intracerebral vasculitis can cause cerebrovascular complications in bacterial meningitis [9]. In addition, several case reports documented complications of intracranial hemorrhage in patients with bacterial meningitis [10-12].

It is crucial to immediately identify the causative bacterial pathogens and initiate appropriate treatment for patients with bacterial meningitis. Previous studies have shown that a delay in starting antimicrobial treatment is associated with an increase in mortality and residual neurologic deficits [13,14]. Herein, this report presents a challenging case of Streptococcus pneumoniae meningitis in a 64-year-old woman who presented with symptoms of cerebellar hemorrhage. Written informed consent was obtained from the patient’s family for publication of this case report and accompanying images.

Case Report

A 64-year-old woman presented with headache 2 days prior to admission. On the day of admission, she was found by family members to have an altered mental status, characterized by incoherent states and newly developed lethargy. The patient was brought to our hospital by ambulance. She had a medical history of untreated diabetes mellitus. On physical examination, she was febrile at 38.6°C, her pulse was 128 beats/min, and her blood pressure was 180/88 mmHg. She had cognitive impairment, with a Glasgow Coma Scale (GCS) score of 10/15 (E3V2M5). Laboratory findings showed a white blood cell count of 21,600/mm³ (neutrophil, 92%) and a C-reactive protein (CRP) level of 0.09 mg/dL. Serum glucose and hemoglobin A1c levels were elevated to 468 mg/dL and 16.2%, respectively. Liver function, renal function, and serum electrolyte levels were within normal limits (Table 1).

Computed tomography (CT) of the head was performed immediately to examine the cause of the patient’s impaired consciousness. Although physical and neurological findings were...
examined in the Emergency Department, the progression of the case revealed that this assessment may have been insufficient. Head CT revealed a high-density focus in the left cerebellar hemisphere and fluid collection in the right maxillary paranasal sinus (Figure 1A, 1B). She was diagnosed with cerebellar hemorrhage, which was initially considered the cause of her altered mental status. The patient was admitted to the neurosurgical unit. At this point, bacterial meningitis was not suspected.

She was started on intravenous nicardipine, and the dose was adjusted to maintain systolic blood pressure lower than 140 mmHg. In addition, 2 sets of blood cultures were obtained upon admission to the hospital because the patient was febrile. Both blood cultures grew gram-positive cocci in chains 12 hours after admission. The Department of Internal Medicine was also consulted. On physical examination, the patient’s vital signs were as follows: temperature, 37.8°C; pulse, 102 beats/min, and blood pressure, 142/74 mmHg. The patient’s impaired consciousness continued to worsen (GCS score, 8/15; E2V2M4). A stiff neck was also recognized on clinical examination. The CRP level was elevated to 13.8 mg/dL. She was also noted to have sinusitis on CT on admission (Figure 1B). Considering these findings, we suspected bacterial meningitis and immediately performed a lumbar puncture. Cerebrospinal fluid analysis revealed pleocytosis of 217 cells/mm³ (70% polymorphonuclear), elevated protein level (647 mg/dL), and low glucose level (4 mg/dL). Acute meningitis was strongly suspected to be the underlying cause of her altered mental status, cerebellar hemorrhage, and fever. She was administered 9.9 mg of dexamethasone intravenously 4 times daily, 2 g of ceftriaxone twice daily, and 2 g of vancomycin as a loading dose followed by 1 g twice daily, initiated 12 hours after admission [15].

On day 3 of admission (2 days after initiation of antibiotic treatment), her impaired consciousness persisted; therefore, we decided to perform brain magnetic resonance imaging (MRI). MRI revealed bright sulci with features corresponding to subarachnoid hemorrhage, and no aneurysm was noted on magnetic resonance angiography on day 4 of admission (Figure 2). Septic embolism and infectious endocarditis were suspected to be the cause of the subarachnoid hemorrhage; however, transthoracic echocardiography detected no vegetation or valvular disease.

On day 5, her body temperature normalized. *Streptococcus pneumoniae* was isolated from both blood and CSF cultures, confirming the diagnosis of bacterial meningitis. The isolate was resistant to penicillin (minimum inhibitory concentration [MIC] ≥0.12 µg/mL) and susceptible to ceftriaxone (MIC <1.0 µg/mL); therefore, we discontinued vancomycin [16]. We completed the 14-day course of intravenous antimicrobial therapy. CRP was normalized. Her mental status improved; however, her impaired consciousness persisted (GCS 11/15; E3V3M5), and she had dysphagia. The patient was transferred to a rehabilitation center on day 35.

**Discussion**

To the best of our knowledge, we present an extremely rare case of acute bacterial meningitis complicated by intracerebral hemorrhage due to *Streptococcus pneumoniae*. The patient’s initial presentation with altered mental status raised concern for an intracerebral hemorrhage, which was confirmed by head CT. However, further investigation revealed bacterial meningitis as the underlying cause of the patient’s symptoms, as evidenced by pleocytosis and elevated protein levels in the cerebrospinal fluid. The initial misdiagnosis of cerebellar hemorrhage highlights the importance of a thorough diagnostic workup in cases of altered mental status.

By treating the bacterial meningitis promptly, the patient’s condition began to improve, with her body temperature normalizing and CRP levels returning to normal. Despite the improvement, her mental status remained impaired, requiring further rehabilitation. The case underscores the importance of a multidisciplinary approach in managing these complex cases, ensuring prompt and accurate diagnosis to guide appropriate treatment. The successful outcome in this case, despite the rare complication of a concomitant cerebellar hemorrhage, emphasizes the importance of continuous monitoring and rapid response in the management of such cases.
and subarachnoid hemorrhages. This case of bacterial meningitis was complicated by both cerebellar and subarachnoid hemorrhage. In addition, this was a didactic case because her altered mental status was initially attributed only to cerebellar hemorrhage, and the delayed diagnosis of bacterial meningitis might have led to her persistently altered mental status. Thus, the case highlights the need for clinicians to be aware that bacterial meningitis can result in intracranial hemorrhage.

Acute bacterial meningitis is a medical emergency that results in substantial morbidity and mortality despite the availability of effective antibiotic therapy [17]. In particular, pneumococcal meningitis, which is sometimes complicated by neurological (eg, cerebral edema, infarction, intracranial hemorrhage) and systemic (eg, septic shock, acute respiratory distress syndrome, and disseminated intravascular coagulation) complications, is associated with poor prognosis [18]. To improve prognosis, the most important initial issues are timely diagnosis and prompt administration of appropriate antimicrobial therapy. A head CT scan is not always necessary in patients with bacterial meningitis. However, head CT findings should always be taken into consideration before performing a lumbar puncture in patients with suspected bacterial meningitis and an altered mental status [19]. In such cases, blood cultures should be collected immediately, and antibiotic therapy should be started prior to CT.

Ischemic stroke is the most common cerebrovascular complication of bacterial meningitis, and subarachnoid and intracranial hemorrhages are very rare [20]. In our extensive literature search, 4 articles investigating the incidence rate of stroke and intracranial hemorrhage related to bacterial meningitis were identified [18,21-23] (Table 2). The review revealed that stroke occurred as a complication in more than 10% of bacterial meningitis cases. Meanwhile, the incidence of both subarachnoid and cerebral hemorrhage as a complication of bacterial meningitis is only 0.2%, as shown in Table 2. Furthermore, cerebellar hemorrhage is a rare complication of bacterial meningitis, and only 1 case has been reported [24]. Because of this rarity, the diagnosis of meningitis can be delayed, as in our case, among patients with intracerebral hemorrhage.

The pathogenetic mechanisms involved in the development of intracerebral hemorrhage likely include a destructive process against the blood vessels and an inflammatory response generated in the subarachnoid space due to the infection. This process may eventually lead to the formation of microaneurysms with fatal rupture, resulting in a cerebral hematoma or subarachnoid hemorrhage [25]. Cerebral vasculopathy, including microaneurysms in bacterial meningitis, can occur due to infection by various organisms such as Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenzae, and Staphylococcus aureus [8,12,26,27]. However, pre-existing risk factors of atherosclerosis, such as prior stroke, diabetes,
mellitus, cardiovascular disease, and arterial hypertension, were not significantly associated with intracranial complications in patients with bacterial meningitis [21]. Although MRI did not show any aneurysms in our case, there is a possibility that the microaneurysms were formed from the infection.

Stroke, either ischemic or hemorrhagic, associated with bacterial meningitis can occur at an early phase, such as on admission and within 1 week after admission, or at a late phase, such as 1 week or even after successful treatment [8,21,28]. In previous case reports, all the reported cases were complicated with hemorrhagic stroke after the diagnosis of bacterial meningitis [10-12]. On the other hand, when a hemorrhagic stroke is diagnosed on admission, the diagnosis of bacterial meningitis can be delayed, as in our case, through the diagnostic delay mechanism of anchoring and early closure [29]. To prevent this, physicians should be aware that hemorrhagic stroke can be the initial manifestation of bacterial meningitis. Additionally, the emergency physician performed a head CT scan before sufficiently examining the physical findings. If we had taken a more detailed medical history and performed the physical examination more comprehensively, we could have diagnosed and treated the cause immediately. A previous study shows that the incidence of diagnostic delay in emergency departments is higher than in outpatient departments [30]. These diagnostic delays may be caused by insufficient assessment, inappropriate response to diagnostic imaging, and failure to recommend appropriate diagnostic imaging.

In general, patients with cerebellar hemorrhage usually have headaches, nausea, vomiting, vertigo, and ataxia. Unless the hemorrhage is large or complicated by compression of the brainstem, other etiologies should be considered. Additionally, our patient was febrile during the first medical examination. Although fever can occur in cases of ventricular hemorrhage, it usually does not occur in cases of cerebellar hemorrhage [32]. In particular, bacterial meningitis must be considered as a differential diagnosis in patients with cerebellar hemorrhage presenting with altered mental status and fever. In this case, although the patient had a clinical presentation and medical history typical for bacterial meningitis, she was initially misdiagnosed because cerebellar hemorrhage was discovered on head CT examination.

Conclusions

This report highlights that clinicians should be aware that bacterial meningitis can result in intracranial hemorrhage. Patients with symptoms of a hemorrhagic stroke should be thoroughly investigated to avoid a delay in the treatment of infection.

Disclosures

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

Declaration of Figures’ Authenticity

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