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Culture-Negative Neutrophilic Meningitis Followed by Suboccipital Abscess and Sepsis: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 65-year-old
Final Diagnosis: Sepsis
Symptoms: Pain
Clinical Procedure: —
Specialty: Neurology

Objective: Unknown etiology

Background: Aseptic meningitis is a relatively common disease but is extremely rarely complicated by a suboccipital abscess. This report describes and discusses a rare case of a patient with culture-negative pleocytosis suggestive of bacterial infection, followed by suboccipital abscess and sepsis.

Case Report: The patient was a 65-year-old woman who developed a throbbing, holocranial headache that was initially interpreted as a tension-type headache. After signs of meningismus later developed, she was diagnosed with aseptic bacterial meningitis based on a cerebrospinal fluid (CSF) cell count of 15 127 cells/μL, primarily granulocytes containing bacteria-like structures. However, blood, urine, and CSF cultures were negative. Using empirical antibiotic therapy, the patient achieved near complete recovery before discharge. More than a month after this episode, the patient developed a suboccipital abscess complicated by sepsis, necessitating repeated surgical interventions and adjustments to antibiotic therapy. Observations from this case are applicable to patients with strong CSF and clinical indicators of bacterial infection despite negative routine cultures, especially when subsequent deep soft-tissue infection or sepsis emerges. Recurrent headaches or neck pain after initial improvement should prompt reassessment for a deep extracranial infectious complication.

Conclusions: This case suggests that a presentation initially classified as aseptic meningitis may actually be a culture-negative infection and can be followed by deep soft-tissue abscess formation and sepsis. It also demonstrates that the precise pathogen and source of infection may remain unconfirmed but that empirical antibiotic therapy can be beneficial.

Keywords: Meningitis, Aseptic • Abscess • Headache Disorders, Secondary

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Introduction

Aseptic meningitis is an inflammation of the meninges with the classic symptoms and signs of meningitis, including pleocytosis in the cerebrospinal fluid (CSF), but negative routine bacterial cultures or viral diagnostics [1]. Nevertheless, aseptic meningitis is treated empirically with antibiotics or antivirals, with the risk that the causative agent is resistant to these drugs. Causes of aseptic meningitis include infectious agents such as enteroviruses, herpesviruses, HIV, mumps, measles, West Nile virus, lymphocytic choriomeningitis virus, arboviruses, *Borrelia burgdorferi*, *Treponema pallidum*, leptospires, *Mycoplasma pneumoniae*, *Cryptococcus*, *Coccidioides*, *Toxoplasma gondii*, and *Naegleria fowleri* [1]. It can also occur secondary to sinusitis, mastoiditis, dental infections, or epidural abscesses; to medications such as nonsteroidal anti-inflammatory drugs, trimethoprim-sulfamethoxazole, intravenous immunoglobulins, amoxicillin, and monoclonal antibodies; to autoimmune diseases; or to malignancies that infiltrate the meninges [2]. Aseptic meningitis is often milder than bacterial meningitis.

A suboccipital abscess is a rare, deep neck infection in the occipital region that can sometimes cause severe meningeal irritation and mimic meningitis. Causes of suboccipital abscesses include hematogenous spread, the spread of infections from the middle ear, mastoiditis, sinuses, teeth, or scalp, infections penetrating the neck, spinal surgery, complications following epidural anesthesia or injections, and dermal infections [3]. Cases in which aseptic meningitis initially developed followed by a suboccipital abscess have been rarely described [4]. The following report documents a rare clinical combination and

sequence of culture-negative meningitic pleocytosis with later suboccipital abscess and sepsis, possibly linked to an occult extracranial source. This case has the potential to raise diagnostic awareness, improve source identification, and illustrate boundary conditions.

Case Report

The patient was a 65-year-old woman with known arterial hypertension, hyperlipidemia, obesity, deep vein thrombosis in her right leg, hypothyroidism, hyperuricemia, chronic dental infections, and depression. In December 2016, she developed previously unknown, severe, holocranial, throbbing headaches, which were initially interpreted as tension headaches and treated with tizanidine, analgesics, and injections of local anesthetics into the neck muscles. As the headaches persisted and she also experienced general fatigue, exhaustion, mild dizziness upon sitting, and a persistent fever lasting 3 days, she was admitted to the neurology department 1 month later. The clinical neurological examination revealed neck stiffness and a positive Brudzinski sign, but was otherwise unremarkable. Blood test results showed an elevated C-reactive protein (CRP) level and leukocyte count, reduced serum sodium, and elevated glucose and HbA1c levels (7.8%) (Table 1). Blood and urine cultures remained sterile. The chest X-ray was unremarkable. Computed tomography (CT) of the thorax and abdomen showed only cholecystolithiasis and a cortical cyst of the right kidney. The cranial CT scan with contrast medium did not show meningeal enhancement or a suboccipital lesion. CSF analysis revealed cloudy CSF with a cell count of 15 127 cells/ μ L,

Table 1. Abnormal blood and cerebrospinal fluid values of the index patient during hospitalization days 1 to 25.

Parameter	Reference range	Day 1	Day 2	Day 4	Day 18	Day 25
C-reactive protein	< 5 mg/L	57.3	66.9	70.2	21.4	9.5
Leucocyte count	4-9 G/L	23.1	15.6	13.5	10.8	9.0
Sodium	136-145 mmol/L	126	136	138	144	142
Potassium	3.4-4.5 mmol/L	Hemolysis	3.0	3.4	4.1	3.8
Glomerular filtration rate	> 90 mL/min/1.7 m ²	67	72	83	88	90
Glutamate-oxalate transaminase	10-35 U/L	Hemolysis	44	nd	nd	22
Glutamate-pyruvate transaminase	10-35 U/L	Hemolysis	109	nd	153	65
Gamma-glutamyl transpeptidase	< 40 U/L	Hemolysis	184	nd	153	102
CSF cell count	< 6/ μ L	15127	6127*	nd	nd	nd
CSF protein	150-450 mg/L	1820	1830*	nd	nd	nd
CSF lactate	< 2 mmol/L	9.3	nd	nd	nd	nd

Note: "nd" indicates not done. * Indicates repeated analysis of the cerebrospinal fluid of hospitalization day 1.

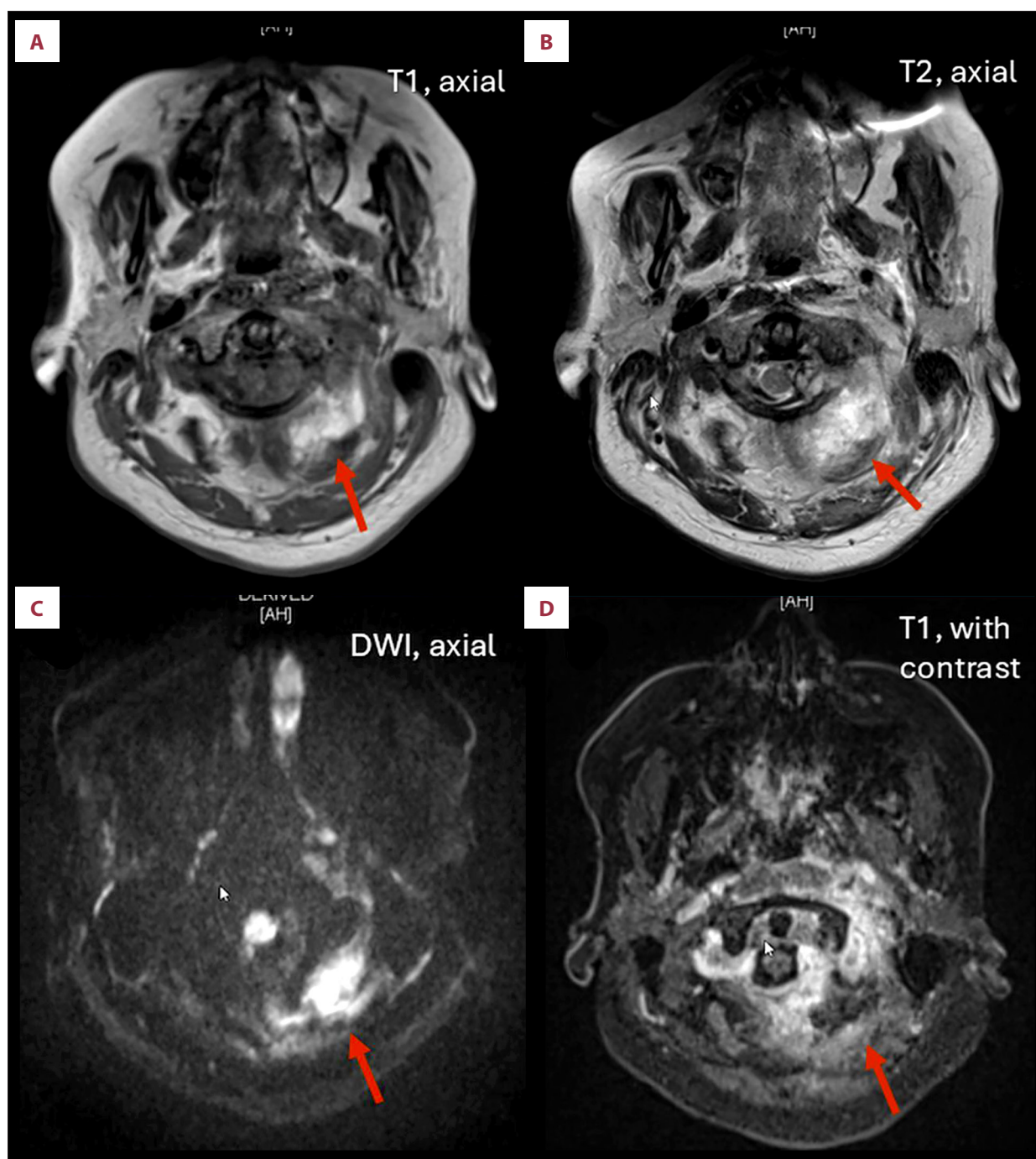


Figure 1. Magnetic resonance imaging of the brain showing a mass lesion in the left nuchal soft tissue that extended to the parapharyngeal area. (A) T1-hypointensity; (B) T2-hyperintensity; (C) diffusion-weighted imaging hyperintensity; and (D) perifocal enhancement.

predominantly neutrophils, bacteria-like structures within the granulocytes, and significantly elevated lactate and protein levels suggesting bacterial meningitis (Table 1). The CSF culture remained sterile. CSF cytology showed predominantly neutrophils with intracellular structures consistent with poorly preserved cocci. The patient received intravenous ampicillin (12

g/d) and ceftriaxone (4 g/d) for 14 days. Piracetam was administered for the headaches. Under this treatment, the patient recovered almost completely.

Thirty-four days after discharge, the patient was readmitted due to exacerbating neck pain, swelling of the cervical muscles,

and headaches. Magnetic resonance imaging (MRI) of the brain revealed an extensive suboccipital soft tissue abscess extending into the parapharyngeal region (Figure 1). Despite initiating therapy with amoxicillin and clavulanic acid (9 g/d), and aspiration of the abscess yielding 16 mL of pus, the mass increased in size, and the patient developed sepsis. The culture prepared from the pus showed no growth. Surgical abscess drainage and irrigation were subsequently performed. Postoperatively, the patient required 10 days of intensive care unit (ICU) care, including 6 days of mechanical ventilation. During her ICU stay, metronidazole (1500 mg/d) was added to her treatment regimen. On day 44 of her hospital stay, the patient was discharged after near-complete remission.

Discussion

The presented patient's case is interesting for several reasons. First, despite repeated cultures, no specific pathogen responsible for the meningitis and subsequent suboccipital abscess could be identified. However, indications of a likely bacterial cause included the presence of cocci in granulocytes in the CSF cytology, the high cell count in the CSF, the predominant proportion of granulocytes in the CSF, the eventual effectiveness of the antibiotic therapy, the sepsis, and the nuchal abscess formation. The reason for the repeatedly negative blood, urine, and CSF cultures remains unclear. However, several explanations are possible: the infection may already have been partially suppressed by the patient's immune system; the bacterial load may have been too low for detection; contamination with coagulase-negative staphylococci may have interfered with pathogen detection; or the culture methods may have been inadequate. It is also conceivable that the chronic dental infections activated the immune system, which in turn suppressed excessive bacterial growth. A temporary weakening of the immune system, for example due to a viral coinfection, may then have allowed it to exacerbate and become symptomatic. It can be ruled out that pre-analytical antibiotics were used. Another explanation could be that meningitis was caused by a pathogen that did not grow on standard media. These are difficult to culture in blood and CSF due to complex nutritional requirements (demanding organisms) or specific environmental conditions such as low oxygen or high CO₂ levels, which are not guaranteed by standard laboratory media. Particularly difficult to cultivate are *Bartonella* sp., *Brucella* sp., *Legionella* sp., *Coxiella burnetii*, *Tropheryma whipplei*, *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, *Kingella*, *Abiotrophia* and *Granulicatella* spp., *Cryptococcus neoformans*, *Mycobacterium tuberculosis*, *Borrelia burgdorferi*, and *Treponema pallidum* [5]. Aseptic pleocytosis has been reported not only with bacterial infections but also with viral, fungal, and parasitic infections; the use of nonsteroidal anti-inflammatory drugs (eg, ibuprofen), sulfonamides, penicillins,

intravenous immunoglobulins, monoclonal antibodies, or anti-seizure medications; and systemic or inflammatory conditions such as lupus erythematosus, Sjögren syndrome, Behçet disease, neurosarcoidosis, Fabry disease, SARS-CoV-2 vaccination, MOG-associated disease, leukemia, lymphoma, or metastases from solid tumors [1,6-8].

The second point is that the origin of the meningitis could not be clearly identified. Since there was no history of subdural abscess, traumatic brain injury, or prior brain or spinal surgery, it is unlikely that the meningitis originated in the central nervous system. It is more probable that the focus was outside the central nervous system and that the spread to the meninges was hematogenous. Causes of spread such as otitis, mastoiditis, respiratory tract, or gastrointestinal tract involvement were ruled out. Because the onset of the suboccipital abscess could not be precisely determined, it is also conceivable that the suboccipital abscess developed before the spread to the meninges. However, arguments against the abscess as the origin of the meningitis are that it was not present during the meningitis and that the headaches were initially holocranial and occurred before the neck pain, which remained unilateral with the abscess. It is also conceivable that the diabetes had been present subclinically for months and thus promoted the bacterial infection, as has been previously reported [9]. Whether chronic dental infections were the starting point for meningitis remains speculative, as information about their type, location, severity or extent, approximate timing, and exact management was unavailable.

Third, the combination of aseptic meningitis with an abscess or sepsis has only rarely been described [10]. The most common abscess following aseptic meningitis is in the pituitary gland [10-12]. In most of these cases, the abscess preceded the meningitis. However, the timing of abscess formation and the causal relationship between the abscess and meningitis often remains unclear. To our knowledge, aseptic pleocytosis with subsequent suboccipital or nuchae abscess has only been described once [4]. This involved a 26-year-old man with osteomyelitis of the occipital bone and a pseudomeningocele following aseptic meningitis [4]. In this patient, the mass was treated by fine-needle aspiration followed by surgical debridement. This revealed a 3-cm erosion in the occipital bone with intact dura mater [4].

Observations from this case are most applicable to patients with strong CSF and clinical indicators of bacterial infection despite negative routine cultures, especially when subsequent deep soft tissue infection or sepsis emerges. In such circumstances, contrast-enhanced CT, brain MRI, and polymerase chain reaction (PCR)-based testing can help reveal the underlying etiology. Furthermore, recurrent headaches or neck symptoms after initial improvement should prompt reassessment

for a deep extracranial infectious complication. Despite the extensive clarification, the present study also has some limitations. The patient did not undergo a second lumbar puncture, no bacterial or viral PCRs were performed, no methods other than standard ones were used for pathogen culture, and the patient did not have a spinal MRI at the onset of headaches to potentially detect the abscess earlier.

Conclusions

In summary, this case suggests that a presentation initially classified as aseptic meningitis may actually be a culture-negative infection and can be followed by deep soft tissue abscess formation and sepsis. It also demonstrates that the precise pathogen and source of infection may remain unconfirmed if the patient is not extensively examined, but that empirical antibiotic therapy and repeated surgical abscess resection can be beneficial. Clinicians should consider occult infectious sources when CSF and clinical presentation strongly suggest bacterial infection despite negative routine cultures. Furthermore, in cases of meningitis of unclear etiology, even if treatment is successful and the patient is discharged smoothly, outpatient follow-up is important, as this can help to detect hidden foci of infection early.

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Ethical Approval

The study was approved by the institutional review board.

Informed Consent

The patient provided consent for participation in this study and for publication of this case report.

Availability of Data and Material

All data are available from the corresponding author.

Institution Where Work Was Done

The study was carried out at the Neurology & Neurophysiology Centre, Vienna, Austria.

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