

## CASE REPORT

# A case of fatal acute bacterial meningoenkephalitis with extremely high cerebrospinal fluid white blood cell count

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## Abstract

Acute bacterial meningoenkephalitis is still prevalent despite the widespread vaccination and still fatal despite the advances in antimicrobial therapy. Identifying patients at risk, lowering the threshold of clinical diagnosis and early treatment of such a curable disease will save patients' lives.

## KEYWORDS

bacterial meningitis, cerebrospinal fluid, otitis media, pus, pyorrhachia

## 1 | INTRODUCTION

Bacterial meningitis is one of the most life-threatening neurological emergencies. Delayed diagnosis and management results in high morbidity and mortality. Although most cases of bacterial meningitis do not have a precise mechanism, local spread from adjacent structures, such as the ear, nose, and throat is not uncommon. Many bacterial species cause bacterial meningitis; however, *Streptococcus pneumoniae* is the most frequent lethal pathogen. The cerebrospinal fluid (CSF) is often turbid with an elevated white blood cell (WBC) count and proteins. We present the case of a 58-year-old man who had chronic otitis media. He presented to our center with short history of headache, sore throat, cough and fever. He also had history of chronic noncommunicable diseases. He also had

prior history of meningitis likely related to his otomastoiditis that required temporary external ventricular drainage for hydrocephalus. He was initially treated for respiratory tract infection and discharged home. He presented a day later with worsening headache, and altered sensorium. At that time, he was thoroughly evaluated and was found to have fulminant bacterial meningitis due to *S. pneumoniae*. His CSF had appearance of frank pus. The CSF WBC count was 158,000 cells/ $\mu$ L (0–5 cells/ $\mu$ L), with the CSF protein 18.67 mg/mL (0.15–0.6 mg/mL). His neuroimaging showed extensive parenchymal damage with thick exudates in the CSF spaces. There was also radiological evidence of cerebral venous thrombosis. He was treated with standard of care doses of intravenous antibiotics and other supportive care. Unfortunately, he succumbed to his illness. Our case is a rare case of the CSF changing to a

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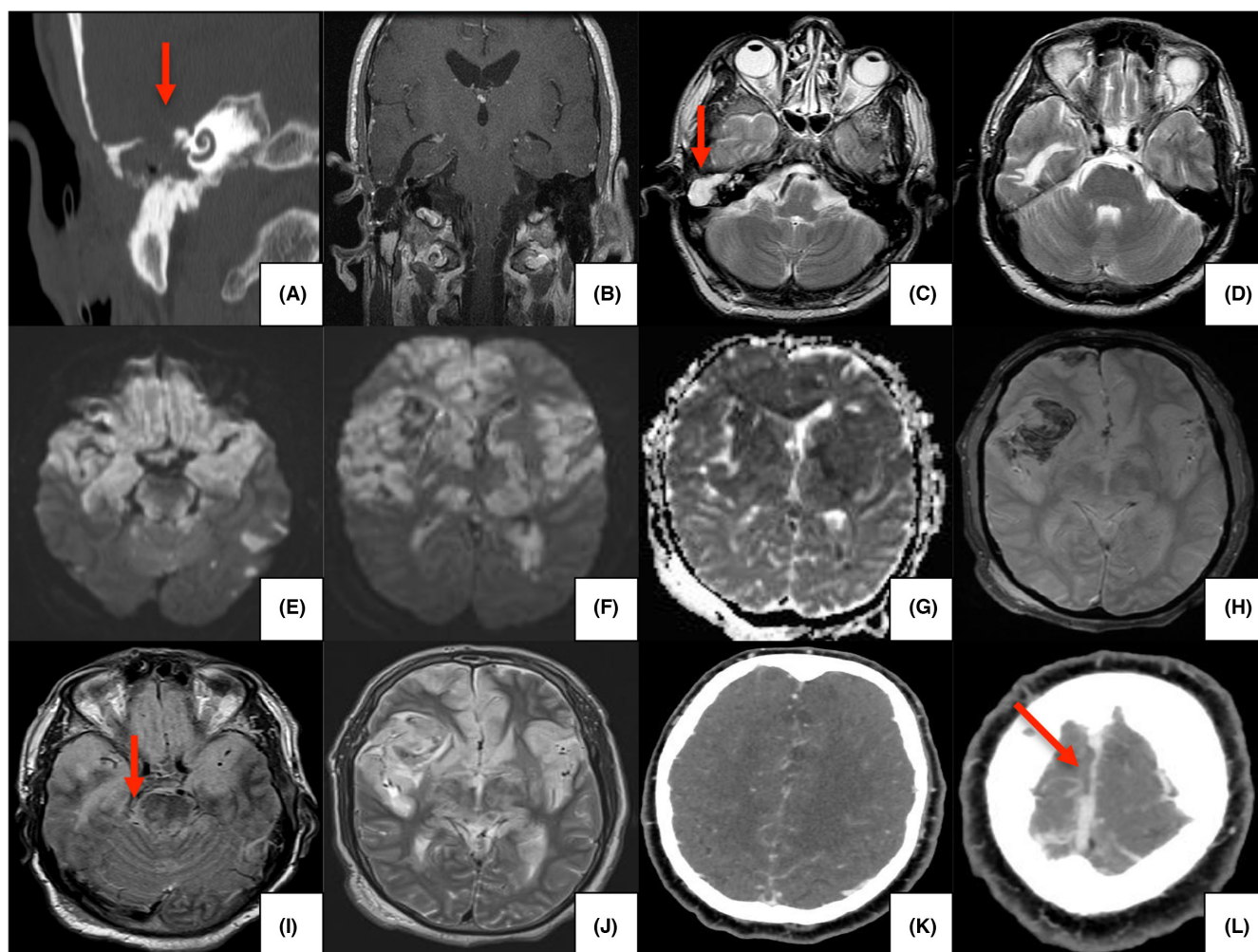
frank pus-like appearance. The CSF WBC count was extremely high that has not been previously reported in the searched literature.

The estimated global incidence of bacterial meningitis is more than 1.2 million cases per year.<sup>1</sup> *S. pneumoniae* is the most frequent pathogen causing bacterial meningitis in adults, with a frequency ranging from 9.6% to 75.2%.<sup>2,3</sup> Recurrent community-acquired bacterial meningitis accounts for approximately 6% of meningitis cases, with *S. pneumoniae* responsible for the majority of the cases.<sup>4</sup> Although a high white blood cell (WBC) count in the cerebrospinal fluid (CSF) is a hallmark of pyogenic meningitis, the frank pus-like appearance of CSF is extremely rare. We report a case of pyogenic meningitis with an exceptionally

high CSF WBC count, with the CSF looking like frank pus. We propose that the pus-like appearance of CSF be termed pyorrhachia. In developing countries, the estimated mortality rate of bacterial meningitis was 22.1%, with 17.1% of the mortality attributed to *S. pneumoniae*.<sup>5</sup> Our patient unfortunately also succumbed to his illness, hence our case was the case of a fatal meningoencephalitis.

## 2 | CASE REPORT

A 58-year-old man presented at the emergency room with a two-day history of headache, sore throat, cough, and fever. He had a past medical history of uncontrolled



**FIGURE 1** Coronal CT bone window image (A). Post contrast T1-weighted image (B). Axial T2-weighted images (C, D, J). Axial diffusion weighted images (E, F). Axial apparent diffusion coefficient image (G). Axial susceptibility weighted image (H). Axial fluid attenuated inversion recovery image (I). Axial CT venogram images (K, L). Pocket of CSF collection in the surgical cavity of the right middle ear and the mastoid bone communicating with the temporal lobe indicating pseudomeningocele (A, B, C, D). Extensive hyperintense signals with corresponding diffusion restriction in both frontal and temporal lobes, insular cortices, hippocampi, basal ganglia and brainstem representing severe meningoencephalitis (E, F, G, I, J). Gyriiform haemorrhagic foci are noted along the peri sylvian, insular cortices, and frontal lobe (H). Multiple foci of exudates along the leptomeninges of brainstem and cerebellar convexities (E, I). Partial filling defects in venous sinus confluence and superior sagittal sinus indicating venous thrombosis (K, L).

diabetes mellitus, hypertension, bronchial asthma, and chronic otitis media. He required an automastoidectomy for the chronic otitis media, complicated by a tegmen tympani defect and secondary pseudomeningocele formation communicating with the left temporal horn (Figure 1). He had bacterial meningitis 6 years ago, complicated by hydrocephalus that required a temporary external ventricular shunt placement. The patient was initially managed as a case of upper respiratory tract infection and discharged on the same day with a course of oral antibiotics. He returned to the emergency room 1 day later because of worsening headache and vomiting associated with altered sensorium. The patient was well built but sick-looking. His systolic blood pressure was 160/75 mm Hg, heart rate 110 beats/min, respiratory rate 27/min, the temperature 38°C, and oxygen saturation 96% at room air. His initial neurological examination revealed no eye-opening to pain, and he produced sounds only as a verbal response. His pupils' size was 4 mm and reactive to light, and he blinked to a threat in both eyes. With the fundus examination, there was bilateral papilledema. He had a normal vestibulo-ocular reflex, positive corneal reflex, no facial asymmetry, the gag reflex was preserved, and in response to painful stimulus on the sternum, he showed withdrawal response bilaterally. He had nuchal rigidity with positive Brudzinski and Kernig signs. His arterial blood gas results showed a pH of 7.512,  $p\text{CO}_2$  of 30.8 mmHg,  $p\text{O}_2$  of 80.2 mmHg, and an  $\text{HCO}_3^-$  of 24.1 mmol/L. The laboratory results were significant with the WBC count of 13,000 cells/ $\mu\text{L}$ , the lactic acid was 2.49 mmol/L (0.5–2.2 mmol/L), and the potassium was 2.7 mmol/L (3.6–5.2 mmol/L).<sup>\*</sup> The other

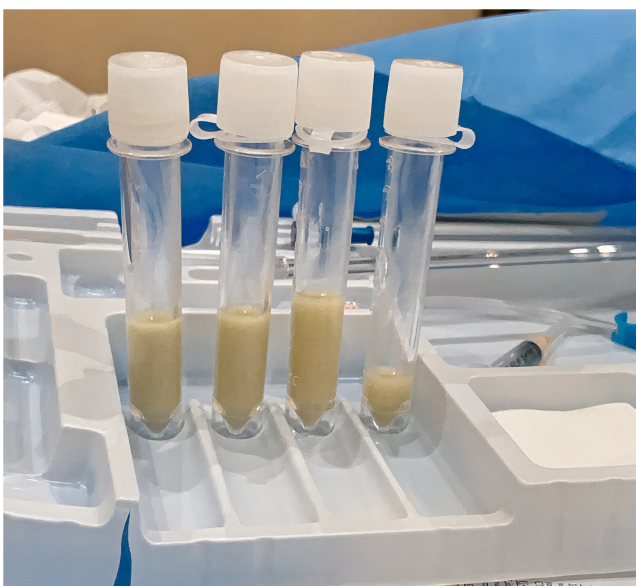


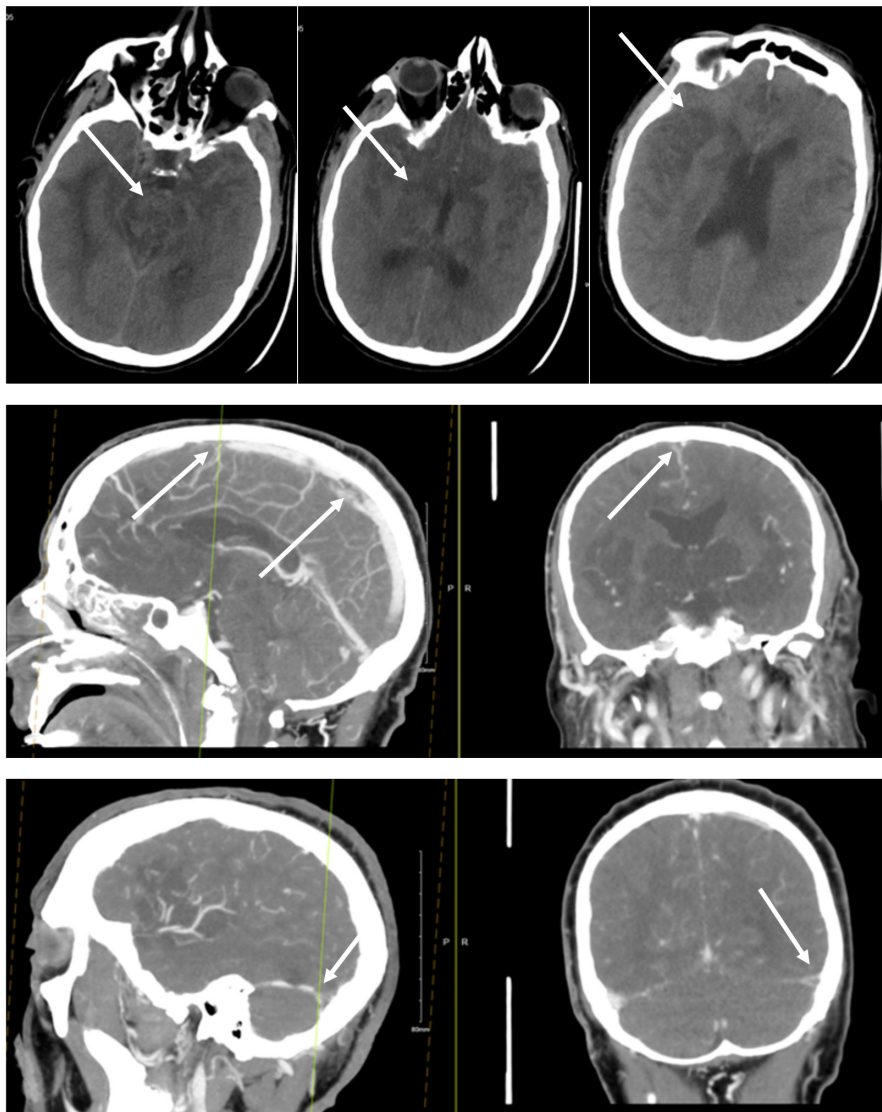
FIGURE 2 Cerebral spinal fluid (CSF) appearance as yellow thick liquid resembling pus.

laboratory parameters including hemoglobin, platelets, renal function, liver function, coagulation profile and cardiac enzymes were within normal limits. Initial brain computerized tomography (CT) scan showed no significant abnormality. Following the brain CT, a lumbar puncture was performed. The appearance of the CSF was a thick yellow liquid resembling pus (Figure 2). The CSF WBC count was 158,000 cells/ $\mu\text{L}$  (0–5 cells/ $\mu\text{L}$ ), the CSF protein 18.67 mg/mL (0.15 to 0.6 mg/mL), the CSF lactic acid 42.37 mmol/L (0–3 mmol/L), the CSF glucose 0.1 mg/100 mL (50–80 mg/100 mL), the CSF RBCs were less than 1 RBC/ $\text{mm}^3$  ( $<1$  RBC/ $\text{mm}^3$ ), and the CSF culture indicated *S. pneumoniae* (see endnote 1). He was started on a regimen with intravenous meropenem 2000 mg every 8 h and intravenous vancomycin 1200 mg every 8 h, and dexamethasone 12 mg every 6 h. He was also given one dose of intravenous metronidazole 1200 mg once. Intravenous ceftriaxone 2000 mg every 12 h was also added to the regimen. Due to respiratory failure, he was intubated with an endotracheal tube and transferred to an intensive care unit. On the second day of admission, his level of consciousness deteriorated, and his Glasgow Coma Scale (GCS) was 3 out of 15. A repeated brain CT scan showed multiple hypodensities and brain venogram revealed multiple filling defects in the superior sagittal sinus and left transverse sinus (Figure 3). The brain magnetic resonance imaging (MRI) showed extensive gyriform diffusion restriction abnormality involving the frontal, anterior temporal lobes, insular cortices, and bilateral deep gray nuclei and brainstem suggestive of meningoencephalitis with gyriform hemorrhagic foci in the insula and frontal lobes (Figure 4). Multiple small collections were also noted along the leptomeningeal surface of the brain stem and cerebellar convexities representing exudates (Figure 4). Unfortunately, on the fifth day of admission, his physical examination continued to show GCS of 3 out of 15, and brainstem reflexes were absent. A brain perfusion scan showed no brain perfusion, and he was declared brain dead.

### 3 | DISCUSSION

Our patient's history of chronic otitis media with mastoiditis was possibly the most important factor of the recurrent severe bacterial meningitis. Otitis media have many serious complications, including mastoiditis, labyrinthitis, seventh cranial nerve palsy, meningoencephalitis, brain abscess, and cerebral venous thrombosis.<sup>6</sup> Acute bacterial meningitis is a devastating complication of otitis media with a reported mortality rate of 41% and chronic otitis media was the cause in 74% of the mortality cases.<sup>7</sup> Our patient had a significantly





**FIGURE 3** CT brain without contrast showing multiple brainstem, subcortical and cortical hypodensities. CT venogram showing filling defects in multiple sinuses.

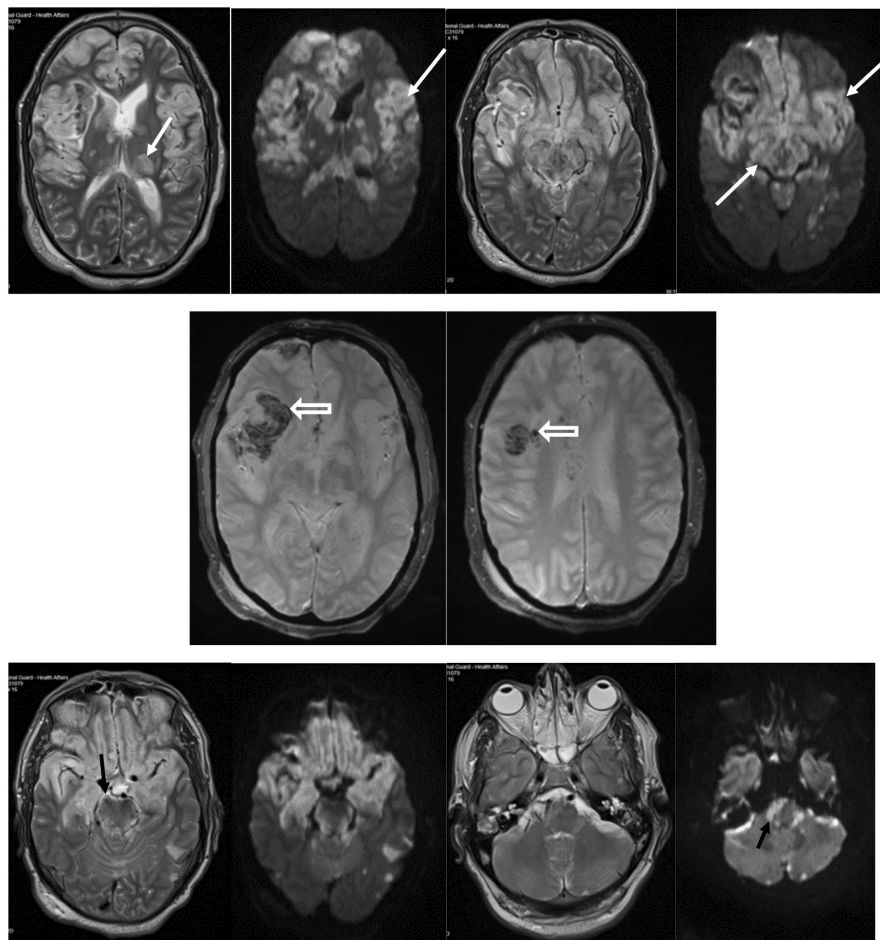
elevated CSF WBC count of 158,000 cells/ $\mu\text{L}$  as a result of severe bacterial meningoencephalitis, with no radiological evidence of abscess formation. However, the meningoencephalitis was severe enough to form brainstem and cerebellar convexity exudates observed on the brain MRI. There is no report in literature with a CSF WBC level as high as in our patient. We found one report of a 55-year-old woman with acute bacterial meningitis who had a high level of white cell count in the CSF of 104,000 cells/ $\text{mm}^3$ , but unlike our patient the CSF appearance was turbid. Similar to our patient, the meningitis was due to *S. pneumoniae* bacteria; with no evidence of an intracranial abscess on brain imaging.<sup>8</sup> Of the many factors contributing to our patient's mortality is his initial symptomatology of cough, sore throat, and headache that misled emergency physicians not to recognize meningoencephalitis early and administer the appropriate antibiotics. A delayed presentation to hospital, decreased level of consciousness on presentation,

*S. pneumoniae* infection, abnormal brain imaging, and the use of intracranial measuring devices are some of the factors that predict a poor prognosis and mortality in an adult with fulminant bacterial meningitis.<sup>9</sup> A history of prior complicated bacterial meningitis and complicated chronic otitis media are essential factors to be considered in any patient presenting with fever and headache, regardless of other symptomatology to rule out central nervous system (CNS) infection.

#### 4 | CONCLUSIONS

Bacterial meningoencephalitis carry devastating outcomes without rapid detection and treatment. Having low threshold for diagnosis can save patients' lives. Our patient's fatal meningoencephalitis was associated with peculiar CSF appearance. Turbid CSF with a high WBC count is often present in bacterial meningitis; however,

**FIGURE 4** The brain magnetic resonance imaging (MRI) showed extensive gyriform T2 hyperintensities and diffusion restriction abnormality involving the frontal lobes, anterior temporal lobes, insular cortices, and bilateral deep gray nuclei and brainstem suggestive of meningoencephalitis (white arrows) with gyriform hemorrhagic foci seen on gradient echo sequences in the insula and frontal lobes (hollow arrows). Multiple small collections were also noted along the leptomeningeal surface of the brain stem and cerebellar convexities (black arrows) representing exudates.



frank pus-like CSF with a WBC level of 158,000 cells/ $\mu$ L is extremely rare. The range of the white cell count at which the CSF turns from a turbid to a pus-like appearance is unknown. The severity of the bacterial meningoencephalitis and the CSF WBC count and its appearance could have direct correlation. We propose that CSF resembling pus should be called pyorrhachia. More reports in future may support our understanding of this extremely rare phenomenon.

#### AUTHOR CONTRIBUTIONS

Supervised by Dr. Ismail A. Khatri, Dr. Salih Bin Sailh, Dr. Makki Almontashri and Dr. Nassir Alotaibi. Patient was under the care of Dr. Khatri. Dr. Alhodaif, Dr. Khatri, Mr. Sultan Alotaibi and Dr. Almontashri wrote the Report.

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None.

#### CONFLICT OF INTEREST STATEMENT

None.

#### DATA AVAILABILITY STATEMENT

Data sharing not applicable—no new data generated. Data sharing is not applicable to this article as no new data were created or analyzed in this study.

#### ETHICS STATEMENT

Ethics approvals were obtained from the Institutional Review Boards of the authors' hospital.

#### CONSENT

Written informed consent was obtained from the patient next of kin to publish this report in accordance with the journal's patient consent policy.

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#### ENDNOTE

\* Our laboratory is College of American Pathologists accredited. CSF WBC count was done manually using a hemocytometer with 1/20 and 1/50 sample dilutions with saline. The differential count

was performed by adding two drops of 5% bovine albumin solution to 1 mL of diluted CSF samples; 1/20 and 1/50 dilution in saline. Samples were centrifuged at 750 RPM for 5 min, and then the slides were stained with Wright's Giemsa stain.

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