Acute Subdural Abscess: A Case Study and Comprehensive Literature Review

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ABSTRACT

This study presents a case of a 32-year male who developed acute subdural abscess after experiencing influenza A and sinusitis. Initial attempts at culturing pathogens yielded negative results. However, the application of metagenomic next-generation sequencing (mNGS) technology successfully identified *Streptococcus intermedia* as the causative agent. Subsequently, targeted treatment regimens were formulated based on the mNGS findings, which led to a remarkable improvement in the patient's clinical condition. This research underlines the indispensable role of mNGS in the accurate diagnosis and efficacious treatment of subdural abscesses, offering significant implications and potential guidance for future clinical practice in this domain.

Key Words: *Streptococcus pneumoniae*, *Influenza-A virus, Acute meningitis, Subdural abscess, Metagenomic next-generation sequencing*.

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INTRODUCTION

Subdural empyema (SDE) represents a formidable infectious disease within the central nervous system, accounting for approximately 20% of localised intracranial infections.[1] It typically manifests in the subdural space between the dura mater and the arachnoid membrane, with pus frequently accumulating on the dorsal surface of the cerebral convexity, and in rarer instances, between the cerebral falx and the inner surface of the cerebral hemispheres.

The valvular structure of Breschet's venous sinuses facilitates the bidirectional flow of fluid, blood, and pathogens, thereby promoting the dissemination of spinal lesions. Upon the formation of SDE, the intracranial compartment may exhibit mass effects, presenting with a constellation of nonspecific symptoms such as fever, headache, vomiting, and malaise. As the disease progresses, neurological deficits including seizures, meningeal irritation symptoms, and even life-threatening conditions such as coma and death may ensue.[2]

In the present case, the patient's seizure episode following a week of fever and headache was indicative of the advancement of SDE.

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Previous studies have established that focal neurological deficits and seizures are independently associated with adverse clinical outcomes (death or disability), thus underscoring the criticality of timely and accurate diagnosis for effective patient management. Herein, a detailed case of SDE is reported along with an exhaustive review of the literature concerning the presentation, investigations, management strategies, and outcomes of SDE patients.

CASE REPORT

A 32-year male presented with one-week history of progressively worsening headache and fever, accompanied by nausea and vomiting. He had been diagnosed with influenza A one-week earlier and had received oseltamivir treatment, yet his symptoms failed to improve.

In the emergency room, he exhibited convulsions, loss of consciousness, and oral salivation. Clinically, his neck demonstrated slight rigidity. The muscle strength and tone of his limbs were within normal limits, and bilateral Kernig's sign was positive. A brain MRI scan revealed the presence of subdural effusion, along with suspected blood or pus at the top of the left forehead (Figure 1 A-C). Additionally, extensive inflammation was detected in the maxillary, ethmoidal, and frontal sinuses on both sides. Laboratory examination on the same day disclosed a white blood cell count of 11.62×10^9/L, a neutrophil count of 9.36×10^9/L, a neutrophil percentage of 80.50%, and a C-reactive protein level of 162.62 mg/L.

During the initial five days of illness, the patient endured severe headache, fluctuating body temperature, and elevated infection markers (Table I).

Table I: Blood routine changes of the patient.

Days	WBC / ($\times 10^9$/L)	Neutrophils / ($\times 10^9$/L)	Neutrophils / (%)	CRP mg/L	PCT ng/ml
d1	14.68	12.03	81.8	121.39	0.079
d4	11.48	7.96	69.40	63.28	0.075
d9	10.18	7.29	71.60	25.04	-
d19	8.20	3.71	45.20	1.55	0.095

WBC: White blood cell; CRP: C-reactive protein; PCT: Procalcitonin.

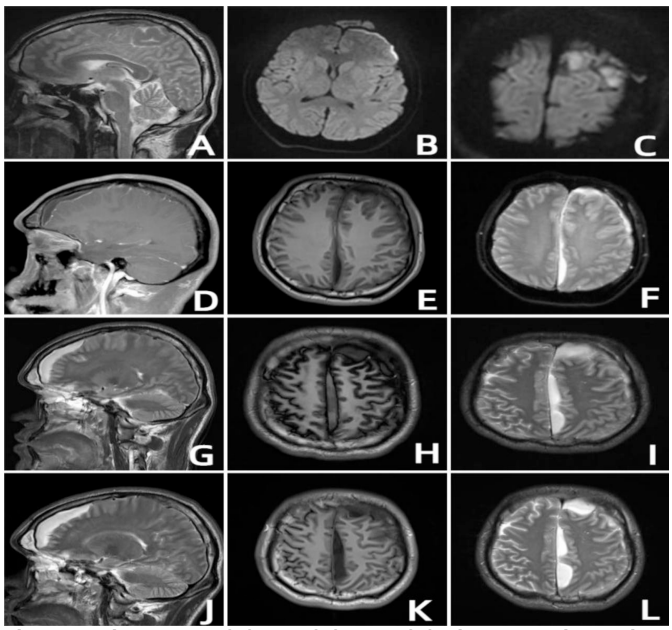

Figure 1: d1 MRI; T2 (A), T 2 (B), DWI (C); d6 MRI; enhanced T1 enhancement (D), T1 (E), T2 (F); d9 MRI; enhanced T2 (G), T1 (H), T2 (I); d19 head MRI; T2 (J), T1 (K), T2 (L).

Tests for hepatitis B, hepatitis C, syphilis, and HIV were all negative. A lumbar puncture (LP) yielded pale yellow, and turbid cerebrospinal fluid (CSF) with a pressure of 360 mmH_2O. Pan's test was weakly positive. CSF cytology revealed with 48% neutrophils and 50% lymphocytes. The protein level was 1500 mg/L, chlorine 122 mmol/L, and glucose 3.76 mmol/L. Notably, no bacteria, fungi, or *cryptococcus* were detected in the CSF, sputum, or blood cultures. Consequently, empirical and symptomatic treatment was initiated.

On the 6th day, the patient's condition deteriorated, characterised by lethargy and delayed reaction time. He experienced over 10 recurrent seizures, accompanied by symptoms such as refractory crying, rightward eye deviation, right-sided mouth deviation, and convulsions of the right limb, each episode lasting from a few seconds to two minutes. An emergency MRI scan demonstrated left frontotemporal and midline fluid signals, with significant enhancement of the adjacent meninges (Figure 1D-F). Meningitis with subdural pus was suspected, along with the inflammation of the bilateral maxillary sinuses, ethmoid sinuses, and left frontal sinus.

A multidisciplinary consultation was promptly convened. The neurosurgery department noted the presence of subdural pus and severe sinusitis. Given the clear annular cistern, a conservative medical management was initially recommended. However, the decompressive craniectomy was suggested if oedema progression and exacerbation of midline displacement occurred. The ENT department diagnosed acute sinusitis based on nasal mucosal congestion and turbinate hypertrophy, although the definitive relationship between meningitis and sinusitis remained undetermined. They proposed the collection and culturing of sinus pus under general anaesthesia. Considering the multidisciplinary perspectives and adhering to the treatment approach for brain abscesses, the patient was administered intensive antibacterial treatment with ceftriaxone and metronidazole while continuing antiviral therapy.

Subsequent LP revealed a CSF pressure of 400 mmH_2O, yet the culture remained negative. In light of this, high-throughput sequencing of pathogens from CSF and blood samples was performed. The metagenomic next-generation sequencing (mNGS) analysis of CSF detected *Streptococcus intermedia* with 445 sequences and a relative abundance of 18.36%, while the mNGS of peripheral blood also identified it with 4 sequences and a relative abundance of 17.36%. Importantly, no bacterial drug resistance gene was detected. This enabled the clear diagnosis of the patient's condition as SDE caused by *Streptococcus intermedia*, effectively ruling out viral encephalitis. Following the appropriate treatment, the patient's clinical symptoms improved. The MRI scan was consistent with the diagnosis of meningitis and subdural abscess, and the midline displacement showed improvement (Figure 1 G-L). The patient was discharged to continue anti-infection treatment and later underwent trepanation and drainage at another hospital, followed by continued anti-infection treatment until full recovery.

DISCUSSION

Given the patient's severe condition, identifying the pathogen was of paramount importance. Considering his history of influenza A infection, the aetiology of SDE was ambiguous. During the treatment process, culturing the pathogen and determining its medicine sensitivity were essential steps. However, the inability to detect the specific micro-organisms in blood and CSF cultures impeded the diagnostic process. As a result, empirical antiviral and antibacterial combination therapy was initially implemented.

The high-risk factors for SDE encompass cranial surgery, open head trauma, respiratory infections, and inadequately

treated ear and sinus infections. It has been reported that 40-80% of patients present with concurrent ear and sinus infections. Dietler *et al*.'s review of SDE cases revealed that 24% of SDE patients were triggered by sinusitis, with frontal and anterior ethmoid sinusitis being particularly prone to developing intracranial infectious complications.[2] In the present case, SDE was confirmed by cranial MRI, and the patient had acute sinusitis, which is commonly caused by *Streptococcus Miller*. This genus, which includes *Streptococcus intermedia*, *Streptococcus angina*, and *Streptococcus constellatus*, typically colonises the upper respiratory, gastrointestinal and reproductive tracts. Among them, *Streptococcus intermedia* is more likely to induce suppurative infections of the central nervous system.[3]

Currently, third-generation antibiotics, such as cephalosporins and vancomycin combined with metronidazole, are commonly employed empirically for comprehensive coverage treatment.[4] Ceftriaxone is frequently utilised in the infection of central nervous system infections due to its favourable penetration of the blood-brain barrier. In the pre-antibiotic era, SDE posed a significant threat to life. With the advent of antibiotics, the mortality rate has substantially decreased, and comprehensive antibiotic coverage remains the first-line treatment for brain abscesses.[5] Nevertheless, surgical intervention must be considered when antibiotics prove ineffective. Trepanation, drainage, and subdural irrigation are the conventional approaches for managing SDE.[6]

Nearly 50% of SDE patients exhibit low sensitivity to blood cultures, primarily because antibiotic treatment is often initiated when specimens are collected. Moreover, CSF culture is rarely sensitive in detecting microorganisms, thereby presenting challenges to diagnosis and treatment.[2] The mNGS offers a revolutionary diagnostic tool by identifying pathogens through high-throughput sequencing.[7] A broad range of pathogens, such as viruses, bacteria, fungi, and/or parasites, can be identified from cultures or directly from clinical samples on the basis of uniquely identifiable DNA and/or RNA sequences.[8] For example, a critically ill 14-year boy with meningoencephalitis diagnosed by mNGS was the first reported case to demonstrate the utility of mNGS in providing clinically actionable information. The successful diagnosis enabled targeted antibiotic treatment, leading to the patient's recovery.[9] Consequent upon the relatively rapid nature of molecular detection techniques, mNGS has emerged as a crucial approach in clinical practice for its capacity to identify a more diverse spectrum of pathogens.[10] In comparison to conventional methods, mNGS has demonstrated 100% diagnostic accuracy, 95% sensitivity, and 96% specificity for CSF samples of hospitalised patients.[11] Fortunately, mNGS successfully identified *Streptococcus intermedia* in CSF and blood samples, as the pathogen responsible for SDE in this case.

In summary, mNGS plays a pivotal role in precisely targeting pathogens, thereby providing crucial information for informed medical decision-making. Appropriate antibiotic therapy continues to be vital in controlling the source of infection. Additionally, interdisciplinary collaboration and multidisciplinary management strategies are essential components in the diagnosis and treatment of subdural abscesses, facilitating improved patient outcomes and advancing the field of neurological infectious diseases.

PATIENT'S CONSENT:

Informed consent was obtained from the patient regarding the publication of this case report.

COMPETING INTEREST:

The author declared no conflict of interest.

AUTHOR'S CONTRIBUTION:

SJ: Drafted, revised, and approved the final version of the manuscript to be published.

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