Journal of Communicable Diseases
Volume 58, Issue 1 - 2026, Pg. No. 168-173
Peer Reviewed & Open Access Journal

Case Report

Invasive *Candida* Meningitis in an Immunocompetent Neurosurgical Patient: A Rare Case Report of Post-Traumatic Complication

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DOI: https://doi.org/10.24321/0019.5138.202610

I N F O

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How to cite this article:
Bhaumik N, Easwaran S, Mahadevan L. Invasive *Candida* Meningitis in an Immunocompetent Neurosurgical Patient: A Rare Case Report of Post-Traumatic Complication. J Commun Dis. 2026;58(1):168-173.

Date of Submission: 2025-09-17
Date of Acceptance: 2026-02-19

A B S T R A C T

Candida meningitis is a rare but serious central nervous system infection, primarily affecting neurosurgical patients with indwelling devices. It mimics symptoms similar to bacterial or tuberculous meningitis, complicating early diagnosis. We report the case of a 32-year-old male who developed *Candida albicans* meningitis following ventriculoperitoneal (VP) shunt placement after decompressive craniotomy for traumatic brain injury. Cerebrospinal fluid (CSF) analysis showed pleocytosis, elevated protein, and markedly low glucose levels. Fungal cultures grew *Candida albicans*, confirmed by the VITEK® 2 Compact system, with antifungal susceptibility to amphotericin B, fluconazole, and echinocandins. The patient was started on liposomal amphotericin B and fluconazole, achieving transient clinical improvement; however, he subsequently developed septic shock and succumbed despite intensive management. This case emphasises the rarity of *Candida* meningitis, the diagnostic difficulties it presents, and the importance of early antifungal therapy and device removal in improving outcomes.

Keywords: Candida meningitis, Fungal meningitis, Candida albicans, Post-traumatic infective meningitis, antifungals

Introduction

Candida meningitis remains an uncommon but increasingly recognised clinical infection, primarily affecting immunocompromised individuals, including those with HIV/ AIDS and malignancies, along with organ transplant recipients and premature neonates.[1] In rare instances, immunocompetent individuals may develop *Candida* meningitis following invasive procedures such as neurosurgery or intravenous drug use, suggesting haematogenous or direct inoculation routes as possible mechanisms of infection. Neurosurgical interventions, particularly those involving external ventricular drains (EVDs), ventriculoperitoneal (VP) shunts, or craniotomies, have been identified as significant risk factors for the development of fungal meningitis due to the disruption of the blood–brain barrier and the potential for

direct inoculation of pathogens.[2] The clinical presentation is often non-specific and may include headache, fever, nuchal rigidity, altered mental status, and fatigue, which can easily mimic bacterial or tuberculous meningitis.[3] This diagnostic ambiguity often leads to delays in appropriate treatment.

A major challenge in diagnosing *Candida* meningitis is the limited sensitivity of conventional diagnostic tools, such as cerebrospinal fluid (CSF) and blood cultures. Moreover, imaging findings and CSF profiles are often non-specific, further complicating timely recognition.[4] These limitations may result in patients initially being treated with broad-spectrum antibiotics for presumed bacterial meningitis, only to be correctly diagnosed after their condition fails to improve.

Given these diagnostic and therapeutic challenges, the prognosis of *Candida* meningitis remains poor, particularly when treatment is delayed. Amphotericin B, often in combination with flucytosine or fluconazole, is the mainstay of therapy; however, outcomes can vary depending on the *Candida* species involved, prior antifungal exposure, the patient's underlying health condition, and the resistance profile of non-*albicans Candida*.[5,6]

In this context, we report a case of *Candida* meningitis in a 32-year-old male patient following neurosurgery after sustaining a traumatic brain injury in a road traffic accident. This case highlights the potential for *Candida* species to cause central nervous system (CNS) infections in previously healthy individuals undergoing neurosurgical intervention, and underscores the need for heightened clinical suspicion and timely antifungal therapy in similar scenarios.

Case Description

A 32-year-old male with a traumatic brain injury sustained in a road traffic accident underwent emergency left frontotemporoparietal (FTP) decompressive craniotomy at another hospital for left temporal epidural haematoma (EDH), subdural haematoma (SDH), and temporal contusion with mass effect.

On postoperative day nine, the patient developed high-grade fever and a decline in neurological status. Neuroimaging showed hydrocephalus, for which a VP shunt was inserted. The shunt subsequently became blocked, necessitating conversion to an EVD. Follow-up CT demonstrated collapse of the right frontal ventricle with drainage occurring from the left frontal and right temporal horns. The patient was referred to our tertiary care centre for further management. Given the clinical suspicion of meningitis, a VP shunt was re-inserted, and CSF samples were obtained and sent for analysis. The reports of the analysis have been tabulated in Table 1. Other routine investigations done are tabulated in Table 2 and routine CSF analysis done is tabulated in Table3.

Table 1 legend: PCR – Polymerase chain reaction; HSV – Herpes simplex virus; CMV – Cytomegalovirus; VZV – Varicella zoster virus.

The CSF sample was subjected to bacterial and fungal cultures. The BACT/ALERT 3D system flagged the specimen positive after 8 hours of incubation. Microscopy showed moderate pus cells along with occasional budding yeast cells. A smear prepared from the flagged-positive broth also revealed similar findings (Figure 1). Culture of both the direct specimen and the flagged broth on blood agar, chocolate agar, and MacConkey agar produced creamy, pasty white colonies, which were identified as *Candida albicans* using the VITEK® 2 Compact system. Furthermore, inoculation onto Hicrome agar yielded green, creamy colonies, consistent with the identification of *Candida albicans* by the VITEK® 2 Compact system (Figure 2). Antifungal susceptibility testing showed sensitivity to fluconazole, voriconazole, amphotericin B, caspofungin, anidulafungin, micafungin, and flucytosine. No bacterial growth was observed.

Table 1.Routine Investigations Done

Test	Result
Ziehl–Neelsen stain for Acid Fast Bacilli	Negative for acid-fast bacilli
GeneXpert for Mycobacterium tuberculosis	Negative for MTB
Viral PCR (HSV, enteroviruses, CMV, VZV)	Negative
Wet mount	Budding yeast cells were seen.
India ink preparation	Negative for Cryptococcus species
CSF cytology	Negative for malignant cells

Table 2.Microbiological Investigations Done from the CSF Sample

Test	Result	Interpretation
Haemoglobin (Hb)	8.1 g/dL	Decreased (Low)
Red Blood Cell count	2.93 million/cu mm	Decreased

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202610

Packed Cell Volume	25.3%	Decreased
Platelet count	4.81 lakhs/cu mm	Elevated
Total Leukocyte Count	13,630/cu mm	Elevated
Differential count	Neutrophils 77.7% (Elevated), lymphocytes 15.5% (Decreased), monocytes 4.1%, eosinophils 2.3%, basophils 0.4%	Neutrophilia
C-Reactive Protein	201.1 mg/dL	Elevated (Marked inflammation)
Procalcitonin	3.42 ng/mL	Elevated
Alkaline Phosphatase	243 U/L	Elevated
Aspartate Aminotransferase / Alanine Aminotransferase	35/ 27 U/L	Normal

Table 3.Routine Analysis of CSF Sample

Parameter	Result	Interpretation
Appearance	Reddish, turbid	Abnormal
Cell count	1441 cells/cu mm	Pleocytosis
Differential count	Neutrophils 94%, lymphocytes 6%	Neutrophilic predominance
Red Blood Cell counts	Numerous	Suggests bleeding/ trauma
Protein	167.9 mg/dL	Elevated
Glucose	< 20 mg/dL (serum glucose 110 mg/dL)	Decreased (Very low CSF:serum ratio)

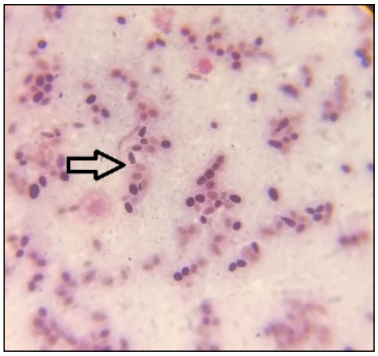

Figure 1.Gram Stain of Budding Yeast Cells in 100X Oil Immersion Field

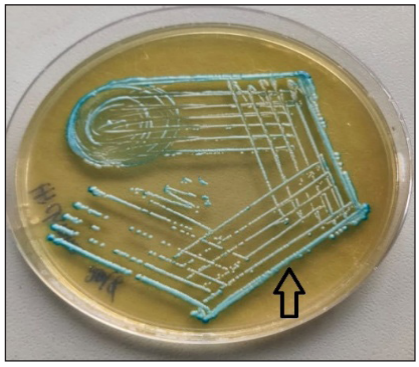

Figure 2.Green Coloured Colonies of *Candida albicans* on Hicrome Agar

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202610

Based on the culture findings, antifungal therapy was initiated with liposomal amphotericin B (5 mg/kg/day) in combination with fluconazole (loading dose 12 mg/kg on day one, followed by 6 mg/kg/day). Nephrology input was obtained, and renal function was monitored regularly. By the sixth day of therapy, the patient showed clinical improvement and was transferred to the ward.

On the twelfth day, the patient developed tachycardia and desaturation. He was found to be semiconscious, disoriented, and had a Glasgow Coma Scale (GCS) of 8/15. He was shifted to the intensive care unit (ICU), where persistent tachycardia and unrecordable blood pressure were noted. Despite fluid resuscitation, he required dual inotropic support. Due to haemodynamic instability, he was intubated and placed on mechanical ventilation. Laboratory investigations revealed leucocytosis, elevated C-reactive protein, and features consistent with septic shock. Broad-spectrum antibiotics (inj. meropenem 2 g IV BD) were initiated. Bedside echocardiography ruled out pericardial effusion and right atrial or ventricular dilatation.

Nephrology consultation was again sought for reduced urinary output, and the patient was managed with diuretics and close fluid balance monitoring. Despite aggressive supportive measures, including diuretics for renal dysfunction, the patient's condition continued to deteriorate. Two days later, he developed sudden bradycardia and cardiac arrest. Cardiopulmonary resuscitation (CPR) was performed as per Advanced cardiac life support (ACLS) protocol, including defibrillation for ventricular fibrillation, but return of spontaneous circulation was not achieved. The patient was declared deceased.

Discussion

Fungal infections of the CNS were once considered rare clinical events; however, in recent decades, the incidence of *Candida* infections, particularly candidaemia, has risen significantly worldwide.[7] *Candida* can disseminate haematogenously from remote foci such as the lungs or central venous catheters, eventually invading the CNS.[8] Clinical manifestations of *Candida* meningitis often mimic bacterial meningitis, with symptoms including fever, headache, altered mental status, and signs of meningeal irritation, making timely diagnosis difficult.[9,10]

Our case highlights the development of *Candida* meningitis in a neurosurgical patient following VP shunt placement. This emphasises the dual impact of neurosurgery and indwelling foreign devices as critical predisposing factors. The blood–brain barrier (BBB) is often compromised during neurosurgical procedures, and the presence of a shunt provides an additional nidus for colonisation and infection. These disruptions facilitate fungal penetration into the CNS, compounding the risk already heightened by perioperative broad-spectrum antibiotics and immunosuppressive states.[11]

The pathogenesis of *Candida* meningitis remains incompletely understood. However, fungal invasion of the CNS likely occurs via penetration of the BBB, facilitated by mechanisms such as transcellular migration, paracellular migration, or the "Trojan horse" method (where fungi are transported within infected immune cells).[12] In neurosurgical patients, direct disruption of the BBB, coupled with postoperative immune suppression, increases susceptibility to CNS fungal infections. Additionally, foreign materials such as shunts or EVDs can serve as niduses for colonisation and subsequent infection.[13] In our patient, the VP shunt likely played a dual role—both as a route of infection and a risk factor due to the immunological impact of neurosurgery.[14] Literature supports this observation: O'Brien et al. documented 11 cases of *Candida* CSF infections following neurosurgical interventions, with common associations being prior antibiotic use and intracranial devices such as VP shunts, lumboperitoneal shunts, and Gliadel wafers.[15] The mortality rate in adult post-neurosurgical *Candida* meningitis is high, ranging from 27% to 33% in reported series, especially in untreated or late-diagnosed cases.[16]

The diagnosis of *Candida* meningitis remains challenging. CSF fungal cultures by conventional and automated methods (BACT/ALERT 3D), though considered the gold standard, suffer from limited sensitivity and prolonged turnaround time. Time-to-positivity for *Candida* species in automated cultures is a parameter that may help predict the prognosis of systemic infection with *Candida*.[17] Direct microscopy often fails to detect fungal elements. Molecular techniques like PCR have shown promise, but they are not yet standardised in most secondary clinical laboratories. In our case, culture was instrumental in confirming the diagnosis, though the delay in detection posed a therapeutic challenge.

Therapeutically, the Infectious Diseases Society of America (IDSA) recommends initiating treatment with liposomal amphotericin B (5 mg/kg daily), with or without flucytosine (25 mg/kg four times daily). Step-down therapy with fluconazole (400–800 mg/day) is appropriate once clinical stability and microbiological clearance are achieved, given its excellent penetration into CSF and brain tissue.[18] The optimal duration of therapy is not well-defined, but antifungal treatment is generally continued until all clinical, CSF, and radiological abnormalities have resolved. In our case, antifungal therapy was initiated promptly following identification of *Candida* in CSF, underscoring the importance of early recognition.

Equally important is the removal of infected foreign devices such as shunts or drains to ensure optimal therapeutic outcomes. In our patient, the VP shunt was removed following confirmation of fungal infection, which contributed

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202610

to clinical recovery. Literature strongly supports device removal as a critical component of management, as retention of colonised foreign material significantly reduces the chances of cure.

Despite antifungal treatment, outcomes remain guarded, particularly in patients with multiple comorbidities or delayed diagnosis.

Antifungal susceptibility patterns remain favourable for *Candida* species, with isolates generally sensitive to liposomal amphotericin B and fluconazole. Resistance, however, is an emerging concern in certain regions, making local susceptibility profiles important in guiding therapy. Our case reinforces the growing recognition of *Candida* as an emerging CNS pathogen, particularly in post-neurosurgical settings. Clinicians should suspect *Candida* meningitis in patients with prior neurosurgery, especially when symptoms persist despite broad-spectrum antibacterial therapy. In such cases, repeat CSF analysis with fungal cultures and, where available, molecular testing should be pursued aggressively.

Conclusion

Candida meningitis is a serious but often underdiagnosed complication in neurosurgical patients, with diagnosis frequently delayed due to non-specific symptoms and inadequate standard tests. Our case underscores the importance of early suspicion, especially when certain risk factors, like prior neurosurgery and placement of VP shunts, are present. Successful treatment relies on prompt antifungal therapy, removal of infected devices, and careful monitoring. Future research should aim to develop rapid diagnostic tools and establish evidence-based guidelines for treating central nervous system fungal infections.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: The authors declare that no generative artificial intelligence (AI) or AI-assisted technologies were used in the preparation of this manuscript.

Sources of Funding: The authors declare that no funding was received for this study.

Conflict of Interest: The authors also declare that there are no conflicts of interest.

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ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202610