Case Report

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Azole-Resistant *Candida albicans* Meningitis in an Unvaccinated Infant with Fatal Outcome: A Case Report from Yemen

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Abstract

Background:
Fungal meningitis caused by Candida species is a rare but life-threatening condition in infants, frequently associated with delayed diagnosis and high mortality. Central nervous system involvement by *Candida albicans* remains uncommon outside neonatal intensive care settings, posing significant diagnostic and therapeutic challenges, particularly in resource-limited countries.

Case Presentation:

We report a fatal case of azole-resistant Candida albicans meningitis in an unvaccinated male infant aged less than one year who presented with fever and recurrent seizures of one-week duration. Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis, marked hypoglycorrhachia, and elevated protein levels despite a clear macroscopic appearance. Direct microscopy and fungal culture confirmed *Candida albicans*, while routine bacterial cultures were negative. Antifungal susceptibility testing demonstrated resistance to multiple azole agents, including fluconazole and voriconazole, with preserved susceptibility to amphotericin B. Hematological evaluation showed leukocytosis with relative lymphocytosis, and serum electrolyte analysis revealed significant hyponatremia. Despite clinical management, the patient's condition deteriorated, resulting in death.

Conclusion:
This case highlights the diagnostic complexity and poor prognosis of *Candida albicans* meningitis in infancy, particularly in the presence of azole resistance. Early inclusion of fungal investigations in suspected meningitis, prompt species identification, and antifungal susceptibility testing are essential for guiding effective therapy. Increased awareness of invasive and drug-resistant Candida infections is critical to improving outcomes in pediatric patients in resource-limited settings.

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Introduction

Fungal meningitis is a rare but life-threatening condition in pediatric populations, particularly among neonates and infants. Among fungal pathogens, Candida albicans is an uncommon cause of central nervous system (CNS) infections; however, when present, it is associated with high morbidity and mortality, especially in cases of delayed diagnosis and treatment. Candida meningitis often presents with nonspecific clinical manifestations, making early recognition particularly challenging in low-resource settings (Yuan et al., 2021; Daniel et al., 2023).

In infants, the clinical presentation of *Candida* meningitis may include fever, seizures, lethargy, and feeding difficulties, which substantially overlap with the manifestations of bacterial and viral meningitis. Cerebrospinal fluid (CSF) findings may be misleading, as CSF can appear macroscopically clear despite underlying invasive fungal infection. Consequently, microbiological culture and antifungal susceptibility testing remain essential for establishing a definitive diagnosis (Shen et al., 2023; Shatrit et al., 2025).

The emergence of antifungal resistance, particularly resistance to azole agents such as fluconazole and voriconazole, has further complicated the management of invasive candidiasis. Azole-resistant *Candida* species have been increasingly reported in both adult and pediatric populations, limiting therapeutic options and contributing to unfavorable clinical outcomes (Mirhendi et al., 2022; Vitale, 2021). In cases involving the CNS, amphotericin B remains the cornerstone of treatment due to its reliable penetration into the CSF and its broad antifungal activity (Cornely et al., 2025).

In Yemen, the available literature on *Candida* infections has primarily focused on adult populations and specific clinical contexts, with very limited attention to pediatric patients. Several local studies have investigated the distribution of *Candida* species and their antifungal susceptibility patterns among women and heterogeneous clinical samples. Studies conducted in Aden and other Yemeni regions have documented variable antifungal resistance profiles, particularly against azole compounds, reflecting the emergence of antifungal resistance in the local setting (Salem et al., 2023; Gubran et al., 2026). Similarly, investigations among pregnant women have highlighted the predominance of *Candida albicans* and non-*albicans Candida* species, along with reduced susceptibility to commonly used azole antifungals (Ali et al., 2024). However, these studies were restricted to non-pediatric populations and superficial or mucocutaneous infections and did not address invasive candidiasis or central nervous system (CNS) involvement. Consequently, there is a conspicuous lack of published data on *Candida* meningitis or other invasive CNS *Candida* infections among infants and young children in Yemen. This absence of evidence underscores the rarity and underreporting of such severe pediatric cases and highlights the critical need for case-based documentation to enhance clinical awareness and inform diagnostic and therapeutic decision-making in resource-limited settings.

In developing countries, including Yemen, the burden of severe pediatric infections is further exacerbated by delayed healthcare seeking, limited access to advanced diagnostic facilities,

suboptimal vaccination coverage, and constrained availability of antifungal agents. Published data on fungal meningitis in infants from such settings remain scarce, and case reports play a critical role in improving clinical recognition and guiding management strategies. Herein, we report a fatal case of azole-resistant *Candida albicans* meningitis in an unvaccinated infant, highlighting the diagnostic challenges, antifungal resistance profile, and adverse clinical outcome.

Materials and Methods

Study Design and Setting

This study is a descriptive case report documenting a laboratory-confirmed case of fungal meningitis caused by *Candida albicans*. The case was identified in 2021 during routine clinical care at Al-Thawra General Hospital, a major tertiary referral hospital in Sana'a City, Yemen.

Patient and Clinical Assessment

The patient was a male infant aged less than one year who presented with fever and seizures. According to the caregiver, the symptoms had been present for approximately one week prior to hospital admission and showed progressive worsening. The patient had received antipyretic medication before presentation, with no history of antibiotic use. Upon admission, a comprehensive clinical evaluation was performed, including neurological assessment and monitoring of vital signs. Due to strong clinical suspicion of meningitis, a lumbar puncture was performed as part of routine diagnostic management.

Cerebrospinal Fluid Collection and Macroscopic Examination

CSF was obtained by lumbar puncture under sterile conditions as part of routine diagnostic evaluation. The CSF sample was subjected to immediate macroscopic examination for color and clarity, followed by laboratory analysis.

White Blood Cell Enumeration in Cerebrospinal Fluid

White blood cell (WBC) enumeration in the CSF was performed manually using a Neubauer hemocytometer counting chamber. An aliquot of well-mixed CSF was loaded into the chamber, and leukocytes were counted under light microscopy according to standard manual counting procedures. The total WBC count was calculated and expressed as cells per cubic millimeter (cells/mm^3). Differential leukocyte count was performed manually using stained CSF smears examined microscopically.

Biochemical Analysis of Cerebrospinal Fluid

CSF glucose and protein concentrations were measured using standard biochemical methods. Serum glucose was assessed concurrently to allow appropriate interpretation of CSF glucose levels. Biochemical analyses were performed using the cobas® c 311 analyzer (Roche Diagnostics, Germany).

Hematological and Serum Electrolyte Analysis

Peripheral venous blood samples were collected under aseptic conditions for hematological and biochemical investigations. Complete blood count, including total and differential white blood cell count, hemoglobin concentration, packed cell volume, and platelet count, was performed using an automated hematology analyzer (Mindray, Shenzhen, China).

Serum electrolyte analysis, including sodium measurement, was performed using an automated electrolyte analyzer (GENRUI GE 200 Electrolyte Analyzer, Genrui Biotech Inc., China), in accordance with the manufacturer's instructions.

Microbiological Examination and Fungal Identification

CSF samples were cultured on Sabouraud Chloramphenicol Agar and incubated at 35–37 °C. Fungal growth was assessed based on colony morphology, microscopic characteristics, and germ tube test for preliminary identification of *Candida albicans*, as previously described (Westblade et al., 2023).

Routine bacterial cultures were also performed by inoculating CSF samples onto blood agar, MacConkey agar, and chocolate agar plates, followed by incubation under appropriate conditions.

Antifungal Susceptibility Testing

Antifungal susceptibility testing of the *Candida albicans* isolate was performed using the disk diffusion method in accordance with the Clinical and Laboratory Standards Institute (CLSI) guidelines (CLSI, 2018; CLSI, 2020). A standardized yeast inoculum was prepared by suspending freshly grown colonies in sterile saline and adjusting the turbidity to match a 0.5 McFarland standard, as recommended for antifungal disk diffusion testing of yeasts (CLSI, 2018). The suspension was uniformly inoculated onto Mueller–Hinton agar supplemented with 2% glucose and 0.5 µg/mL methylene blue to enhance fungal growth and improve inhibition zone edge definition (CLSI, 2018; Pfaller & Diekema, 2012).

Antifungal disks containing fluconazole (25 µg), voriconazole (1 µg), itraconazole (10 µg), ketoconazole (10 µg), miconazole (10 µg), nystatin (100 units), and amphotericin B (20 µg) were aseptically placed on the inoculated agar surface. The plates were incubated at 35 ± 2 °C for 24 hours under aerobic conditions (CLSI, 2018).

After incubation, the diameters of inhibition zones were measured in millimeters using a calibrated ruler. Results were interpreted as susceptible or resistant according to CLSI interpretive criteria where available (CLSI, 2020). For antifungal agents lacking established CLSI disk diffusion breakpoints, inhibition zone diameters were reported descriptively and interpreted in relation to available reference data and published literature (Pfaller & Diekema, 2012).

Data Collection and Management

Clinical, laboratory, and microbiological data were extracted from the patient's medical and laboratory records. All data were anonymized prior to analysis, and only information relevant to diagnosis, management, and outcome was included in this report.

Results

Clinical Presentation

A male infant aged less than one year was admitted with fever and recurrent seizures. The duration of illness prior to CSF sampling was approximately one week, with progressive worsening of symptoms. The patient had received antipyretic therapy before admission, with no documented prior antibiotic use. The child was unvaccinated.

On admission, the patient was lethargic and exhibited neurological manifestations in the form of seizures, accompanied by documented fever.

Cerebrospinal Fluid Findings

Macroscopic examination of the CSF revealed a clear and colorless appearance (Table 1). Cytological analysis showed an elevated total white blood cell (WBC) count with lymphocytic predominance. Biochemical analysis demonstrated marked hypoglycorrhachia and elevated protein concentration, while CSF lactate dehydrogenase (LDH) levels were within the normal reference range.

Table 1. Cerebrospinal fluid laboratory findings

Parameter	Result	Reference range
Appearance	Clear	Clear
Color	Colorless	Colorless
Total WBC count (cells/mm³)	12	≤5
Neutrophils (%)	0	0–6
Lymphocytes (%)	68	40–80
Monocytes (%)	32	15–45
CSF glucose (mg/dL)	29	60–80
CSF protein (mg/dL)	159	20–72
CSF LDH (U/L)	22	≤250

Microbiological Results

Direct microscopic examination of the cerebrospinal fluid by Gram staining demonstrated the presence of yeast cells consistent with fungal elements. Subsequent fungal culture of the CSF sample yielded visible growth on Sabouraud Chloramphenicol Agar. Further identification was achieved through evaluation of colony morphology, microscopic examination of yeast cells, and a positive germ tube test, which collectively confirmed the isolate as *Candida albicans*.

In parallel, routine bacterial cultures were performed by inoculating the CSF specimen onto blood agar, MacConkey agar, and chocolate agar. Following incubation under appropriate conditions, no bacterial growth was observed on any of the media, thereby excluding a concurrent bacterial etiology.

Antifungal Susceptibility Profile

Antifungal susceptibility testing performed using the disk diffusion method demonstrated a clear susceptibility of the *Candida albicans* isolate to polyene antifungal agents, including amphotericin B and nystatin. In contrast, the isolate exhibited resistance to multiple azole compounds, including fluconazole, itraconazole, voriconazole, ketoconazole, and miconazole. The complete antifungal susceptibility profile is summarized in Table 2.

Table 2. Antifungal susceptibility pattern of the isolated *Candida albicans*

Antifungal agent	Susceptibility result
Amphotericin B	Sensitive
Nystatin	Sensitive
Fluconazole	Resistant
Itraconazole	Resistant
Voriconazole	Resistant
Ketoconazole	Resistant
Miconazole	Resistant

Hematological and Serum Electrolyte Findings

As shown in Table 3, peripheral blood analysis revealed leukocytosis, with a total white blood cell count of 14.0 × 10^9/L. Differential leukocyte count showed relative lymphocytosis with a reduced neutrophil percentage. Hemoglobin concentration and packed cell volume were mildly below the reference range for age.

The platelet count was 514 × 10^9/L, which falls within the normal reference range for infants of this age group according to age-adjusted pediatric reference values.

Serum electrolyte analysis demonstrated hyponatremia, with a sodium level of 125 mmol/L.

Table 3. Hematological and serum electrolyte findings

Parameter	Result	Reference range
Hemoglobin (g/dL)	12.0	13.0–18.0
Packed Cell Volume (%)	38.0	40–54
Total WBC count (×10^9/L)	14.0	4–11
Neutrophils (%)	33.2	40–70
Lymphocytes (%)	57.3	20–45
Monocytes (%)	8.4	2–10
Eosinophils (%)	1.1	0–6
Platelet count (×10^9/L)	514	190–660 (infants)
Serum sodium (mmol/L)	125	135–145

Clinical Outcome

Despite medical management, the patient's clinical condition deteriorated, and the case resulted in death.

Discussion

Fungal meningitis caused by *Candida* species represents a rare but severe form of invasive candidiasis in infants and young children, often associated with high morbidity and mortality. In the present case, the combination of subacute clinical progression, neurological manifestations, and characteristic cerebrospinal fluid abnormalities is consistent with previously described presentations of fungal meningitis, which frequently overlap with bacterial and viral etiologies and may delay diagnosis (Faix & Chapman, 2003; Pappas et al., 2016).

The cerebrospinal fluid profile observed in this patient—characterized by lymphocytic pleocytosis, marked hypoglycorrhachia, and elevated protein concentration—aligns with patterns reported in fungal central nervous system infections. Unlike acute bacterial meningitis, which typically demonstrates neutrophil predominance, fungal meningitis often induces a lymphocyte-dominant inflammatory response and a more indolent course, contributing to diagnostic uncertainty. Importantly, the clear and colorless appearance of the CSF in this case reinforces the well-established observation that grossly normal CSF does not exclude serious fungal infection and highlights the necessity of microbiological culture in suspected cases (Faix & Chapman, 2003).

Isolation of *Candida albicans* from cerebrospinal fluid is clinically significant, as CSF is a sterile site. When correlated with compatible clinical and biochemical findings, such isolation should be interpreted as evidence of invasive infection rather than laboratory contamination. Culture-based identification and direct microscopic examination remain essential diagnostic tools, particularly in resource-limited settings where molecular assays may not be routinely available (Lilien et al., 1978).

A prominent and clinically relevant finding in this case was the antifungal susceptibility pattern, which demonstrated resistance to multiple azole agents, including fluconazole and voriconazole, with preserved susceptibility to amphotericin B. Azole antifungals are frequently used in the management of candidal infections due to favorable pharmacokinetics and cerebrospinal fluid penetration; however, increasing azole resistance among *Candida* species has been increasingly reported and is associated with therapeutic failure in invasive disease. Current international guidelines continue to recommend amphotericin B–based regimens as first-line therapy for severe invasive candidiasis and central nervous system involvement, with azoles reserved for step-down therapy only when susceptibility is confirmed (Pappas et al., 2016; Cornely et al., 2025).

In addition to cerebrospinal fluid and microbiological findings, this case demonstrated notable hematological and biochemical abnormalities, including leukocytosis with significant relative lymphocytosis. Lymphocytosis is a common inflammatory response in fungal central nervous system infections. Hyponatremia, in particular, is a well-recognized complication of meningitis in

children and is frequently attributed to the syndrome of inappropriate antidiuretic hormone secretion (SIADH), which is triggered by central nervous system inflammation (Singhi, 2004; Ellison & Berl, 2007). Previous pediatric studies have shown that hyponatremia in the context of meningitis is associated with increased disease severity and may contribute to neurological deterioration if not promptly recognized and managed (Moritz & Ayus, 2010).

The clinical course in this patient was rapidly progressive and resulted in a fatal outcome. Several factors may have contributed to this unfavorable prognosis, including delayed presentation, progression of neurological symptoms prior to lumbar puncture, electrolyte imbalance, and limited effective antifungal options due to azole resistance. Delayed diagnosis and delayed initiation of appropriate antifungal therapy have consistently been identified as major predictors of mortality in neonatal and infant candidiasis, particularly when central nervous system involvement is present (Faix & Chapman, 2003; Pappas et al., 2016).

Most previously reported cases of *Candida* meningitis in infants have involved premature neonates, prolonged hospitalization, indwelling catheters, or prior exposure to broad-spectrum antibiotics. In contrast, the present case occurred in an infant without documented immunodeficiency or prior antibiotic exposure, suggesting that invasive candidal meningitis can also occur in community settings and in the absence of classic risk factors. This observation underscores the importance of maintaining a high index of suspicion for fungal etiologies in infants presenting with meningitis and atypical cerebrospinal fluid profiles, regardless of apparent risk status.

Overall, this case highlights the diagnostic and therapeutic challenges associated with *Candida albicans* meningitis in infancy and emphasizes the need for early inclusion of fungal cultures, routine antifungal susceptibility testing, and careful monitoring of hematological and electrolyte abnormalities. In resource-limited settings, strengthening laboratory capacity and adherence to guideline-based management are critical steps toward improving outcomes in this rare but devastating condition.

Conclusion

This case highlights the rarity and severity of *Candida albicans* meningitis in infancy and underscores the substantial diagnostic and therapeutic challenges associated with this condition. The presence of atypical cerebrospinal fluid findings, coupled with delayed clinical presentation, emphasizes the need for early consideration of fungal etiologies in infants presenting with meningitis and progressive neurological symptoms. The identification of an azole-resistant *Candida albicans* isolate further illustrates the critical importance of routine fungal culture and antifungal susceptibility testing in guiding effective therapy. Strengthening clinical awareness and laboratory capacity, particularly in resource-limited settings, is essential to improving early diagnosis and outcomes in this rare but life-threatening infection.

Abbreviation

CARE – CAse REport guidelines

CLSI – Clinical and Laboratory Standards Institute

CNS – Central Nervous System

CSF – Cerebrospinal Fluid

LDH – Lactate Dehydrogenase

SIADH – Syndrome of Inappropriate Antidiuretic Hormone Secretion

WBC – White Blood Cell

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This research received no external funding.

Conflicts of Interest

The authors declare no conflicts of interest.

Ethical Approval

Ethical approval for this study was obtained from the Faculty of Science, Sana'a University. Permission to collect clinical samples was granted by the administration of Al-Thawra General Hospital. This study was conducted in accordance with the Declaration of Helsinki and prepared following the CARE guidelines for case reporting. Written informed consent was waived as no identifiable patient data were included.

Author Contributions

Conceptualization, A.A.A.-Q. and Q.Y.M.A.; methodology, A.A.A.-Q. and S.A.; investigation, A.A.A.-Q.; data curation, W. H. E. and S.A.; writing-original draft preparation, A.A.A.-Q. and S.A.; writing-review and editing, Q.Y.M.A. and S.A.; supervision, Q.Y.M.A. and A.A. H. All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this published article.

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