

Aseptic Meningoencephalitis and False-Positive Result for *Cryptococcus* Spp. due to a Leukocyte Exclusion Artifact: A Case Report

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ABSTRACT

Introduction: Aseptic meningoencephalitis presents as a critical diagnostic emergency in immunocompetent adults. While cerebrospinal fluid (CSF) wet India ink mounting is a classic, cost-effective tool for rapid detection of *Cryptococcus* spp., it carries a well-documented risk of false-positive results due to leukocyte exclusion artifacts in samples with marked pleocytosis, often leading to inappropriate and potentially nephrotoxic antifungal overtreatment.

Objective: To describe the diagnostic dilemma, clinical features, and molecularly guided antifungal de-escalation in an immunocompetent adult with aseptic meningoencephalitis presenting with a false-positive India ink stain on CSF.

Case Presentation: A 45-year-old immunocompetent male presented with high-grade fever (40 °C), expressional aphasia, paraparesis, and positive meningeal signs following recent NSAID self-medication. Initial CSF analysis revealed marked pleocytosis (up to 20,000 cells/μL, 100% PMNs) and elevated protein levels (1,590 mg/L). A preliminary direct microscopic report indicated positive India ink staining caused by a leukocyte exclusion halo artifact, prompting the immediate empiric initiation of liposomal amphotericin B (300 mg/day) and fluconazole (1,200 mg/day). However, multiplex real-time PCR (FilmArray ME panel) returned negative for *Cryptococcus neoformans/gattii* and other neurotropic agents, and 7-day fungal cultures and contrast-enhanced brain MRI showed no evidence of invasive infection. Antifungals were promptly discontinued, and the patient achieved complete neurological recovery and hospital discharge under supportive anti-inflammatory therapy. **Conclusions:** Leukocyte exclusion artifacts in pleocytic CSF can closely mimic encapsulated *Cryptococcus* yeasts (pseudocryptococcosis). Rapid confirmation via multiplex PCR is essential to prevent diagnostic error, avoid unnecessary antifungal-induced nephrotoxicity, and optimize antimicrobial stewardship in acute aseptic meningoencephalitis.

Keywords: Aseptic meningitis; Meningoencephalitis; *Cryptococcus*; India ink; False positive; FilmArray; Polymerase chain reaction; Antimicrobial stewardship.

INTRODUCTION

Acute central nervous system infections represent critical medical emergencies associated with high morbidity, severe neurological complications, and significant mortality rates worldwide. Prompt identification of the causative agent or early recognition of a non-infectious inflammatory etiology is essential to establish targeted therapy and avoid adverse clinical outcomes. Clinically, affected patients typically present with non-specific features such as progressive headache, high-grade fever, acute altered mental status, neck stiffness, and focal neurological deficits. Consequently, immediate lumbar puncture for

cytochemical and microbiological cerebrospinal fluid analysis remains the cornerstone of initial evaluation. ¹

Aseptic meningoencephalitis encompasses a heterogeneous spectrum of inflammatory meningeal and parenchymal disorders characterized by sterile routine bacterial and fungal cultures. While viral pathogens are the most commonly identified infectious causes, a substantial proportion of episodes arise from parainfectious processes, systemic autoimmune diseases, or drug-induced aseptic inflammation. In particular, nonsteroidal anti-inflammatory drugs are well-known triggers that can induce acute meningeal irritation, fever, and marked cerebrospinal fluid

pleocytosis with elevated protein concentrations, closely mimicking life-threatening invasive infections.²

Conversely, cryptococcosis is an invasive fungal disease caused by encapsulated yeasts of the *Cryptococcus neoformans* and *Cryptococcus gattii* species complexes, both exhibiting strong neurotropism. While *Cryptococcus*

neoformans primarily affects severely immunocompromised individuals such as patients with advanced HIV infection, *Cryptococcus gattii* has a documented ability to cause invasive disease and cryptococcomas in immunocompetent hosts. Central nervous system cryptococcosis represents an absolute medical emergency requiring rapid intervention, as untreated infection leads to progressive neurological deterioration, intracranial hypertension, and near-universal mortality.³

Historically, direct wet India ink microscopy has served as the standard, low-cost point-of-care test for identifying encapsulated fungal cells in cerebrospinal fluid. This negative staining technique highlights the thick glucuronoxylomannan polysaccharide capsule as a clear halo excluding carbon particles on a dark background. However, the diagnostic sensitivity of India ink is highly variable, ranging between 42% and 86%, and is notably diminished in early stages of infection or when fungal burdens fall below 1,000 colony-forming units per milliliter.⁴

In addition to variable sensitivity, wet India ink preparations are susceptible to significant operator-dependent interpretive errors and microscopic artifacts. In cerebrospinal fluid specimens with marked inflammatory pleocytosis or high cellularity, leukocytes can displace colloidal carbon particles and generate clear pericellular halos. This pseudocryptococcal phenomenon has been experimentally demonstrated to occur when centrifugal force or inflammatory disruption causes leukocyte membranes to leak viscous nuclear DNA, creating a morphological pseudocapsule that mimics encapsulated yeasts.⁵

The implementation of automated molecular testing, such as the multiplex real-time polymerase chain reaction FilmArray Meningitis/Encephalitis panel, has substantially improved the diagnostic evaluation of central nervous system infections. This platform allows simultaneous, highly sensitive, and specific detection of major bacterial, viral, and fungal targets, including *Cryptococcus neoformans* and *Cryptococcus gattii*, within two hours. High-precision molecular assays and lateral flow antigen tests are critical for resolving discrepancies arising from direct staining and preventing diagnostic misclassification.⁶

Avoiding a false diagnosis of cryptococcal meningoencephalitis is critical to prevent unnecessary exposure to standard induction antifungal regimens combining liposomal amphotericin B with flucytosine or high-dose fluconazole. Prolonged and unindicated

administration of polyenes carries substantial risks of severe acute kidney injury, tubular necrosis, electrolyte wasting, and hematologic toxicity. We report a case of an immunocompetent adult presenting with aseptic meningoencephalitis and an initial false-positive India ink result due to a leukocyte exclusion artifact, highlighting the essential role of multiplex PCR for prompt antifungal de-escalation and diagnostic stewardship.⁷

CASE PRESENTATION

Patient Information and Baseline History

A 45-year-old male teacher presented to the emergency department with acute neurological deterioration. His medical history was notable for systemic arterial hypertension and grade II obesity (body mass index: 36.7 kg/m²). He had no history of immunosuppression, tuberculosis, or human immunodeficiency virus (HIV) infection, and denied toxic habits or animal exposure. Forty-eight hours prior to admission, he developed generalized myalgias and took self-prescribed oral naproxen and ibuprofen. During the early morning hours preceding admission, he developed documented high-grade fevers reaching 40 °C, followed by acute cognitive impairment, expressional aphasia, and progressive motor weakness in both lower extremities.

Clinical Findings and Physical Examination

Upon initial evaluation in the emergency department, vital signs showed a blood pressure of 139/83 mmHg, heart rate of 100 beats/min, respiratory rate of 22 breaths/min, and axillary temperature of 36.0 °C (following antipyretic administration). On neurological examination, the patient was awake but agitated, uncooperative, and disoriented in time, space, and person. He presented with motor aphasia, generalized muscular rigidity, and bilateral asterixis. Cranial nerve assessment revealed horizontal nystagmus on lateral gaze and sluggish pupillary light reflexes. Meningeal signs were markedly positive, with prominent neck stiffness and bilateral positive Kernig and Brudzinski signs.

Diagnostic Assessment

Non-contrast head computed tomography showed no acute intracranial hemorrhage, midline shift, or mass effect. A lumbar puncture was performed, yielding clear cerebrospinal fluid (CSF) with normal opening pressure. Initial CSF cytochemical analysis demonstrated severe hyperproteinorrachia (1,590 mg/L), preserved glucose (66.1 mg/dL, with a concurrent serum glucose of 108 mg/dL), and pleocytosis of 8,000 cells/μL. Direct microscopic wet examination with India ink was reported as "positive," as clear halos surrounding cellular structures were visualized, which were preliminarily interpreted as encapsulated blastoconidia of *Cryptococcus spp.*

Blood testing revealed mild leukocytosis ($12.19 \times 10^3/\mu\text{L}$ with 77.3% neutrophils) and normal serum procalcitonin (0.0299 ng/mL). Serological tests for HIV-1/2, hepatitis B and C viruses, and *Brucella abortus* were non-reactive. GeneXpert MTB/RIF in CSF was negative. Subsequent control CSF analysis on hospital day 3 confirmed persistent marked pleocytosis (20,000 cells/ μL , 100% polymorphonuclear leukocytes) and elevated protein (596 mg/L). A multiplex real-time polymerase chain reaction (BioFire FilmArray Meningitis/Encephalitis Panel) performed on CSF was completely negative for all bacterial, viral, and fungal targets, specifically confirming *Cryptococcus neoformans/gattii* as "Not Detected." Automated 7-day fungal and bacterial cultures of CSF yielded no growth. Brain magnetic resonance imaging with intravenous gadolinium contrast showed no leptomeningeal enhancement, parenchymal lesions, cryptococcomas, or diffusion restriction.

Therapeutic Intervention and Clinical Outcome

Empiric antimicrobial coverage was initiated upon admission with intravenous ceftriaxone (2 g every 8 hours), vancomycin (1,500 mg every 12 hours), ampicillin (2 g every 4 hours for *Listeria monocytogenes* coverage), and acyclovir (15 mg/kg every 8 hours). Following the preliminary laboratory notification of positive India ink staining, liposomal amphotericin B (3 mg/kg/day, 300 mg daily) and intravenous fluconazole (1,200 mg/day) were added to the regimen under presumptive cryptococcal meningoencephalitis. Adjunctive intravenous dexamethasone (0.075 mg/kg every 12 hours) was co-administered.

Once the negative FilmArray ME result and sterile fungal cultures were confirmed, all antifungal agents and antibacterial coverage were immediately discontinued to avoid polyene-induced nephrotoxicity. Retrospective analysis of the India ink smear confirmed that the perceived capsules were pseudo-cryptococcal artifacts caused by the marked mass of polymorphonuclear leukocytes displacing the colloidal carbon particles. The patient exhibited rapid clinical improvement, achieving complete restoration of cognitive functions, fluent speech, and full motor recovery. He was discharged in stable neurological condition without residual deficits under a definitive diagnosis of resolved aseptic meningoencephalitis.

METHODS

Study Design and Ethical Considerations

This report was prepared following the CAse REport (CARE) guidelines for consensus-based clinical case reporting. Clinical, laboratory, imaging, and therapeutic data were retrieved retrospectively from electronic medical records. All identifiable personal data, exact dates, and geographical references were strictly anonymized to protect patient privacy in compliance with institutional guidelines and

international ethical standards for biomedical publications.

Cerebrospinal Fluid Analysis and Direct Microscopy

Lumbar puncture was performed under strict aseptic conditions to collect cerebrospinal fluid (CSF) for cytochemical, microbiological, and molecular evaluations. Cytochemical parameters, including glucose, total protein, and cell counts with differential analysis, were determined using standard automated hospital laboratory analyzers. Direct negative staining was conducted using a wet mount preparation with commercial India ink to evaluate for the presence of encapsulated yeasts. Smears were examined under light microscopy with standard low- and high-power objectives.

Microbiological Cultures and Antimicrobial Susceptibility Testing

CSF specimens were inoculated onto routine media, including blood agar, chocolate agar, and Sabouraud dextrose agar, and incubated at 30 °C to 37 °C to monitor for bacterial and fungal growth over an automated 7-day incubation period. Serial blood cultures were collected in paired aerobic and anaerobic bottles and processed using continuous-monitoring blood culture systems. Automated antimicrobial susceptibility testing was conducted following standard clinical laboratory guidelines.

Molecular Assays

Multiplex real-time polymerase chain reaction (PCR) was performed on uncentrifuged CSF using the BioFire FilmArray Meningitis/Encephalitis (ME) Panel (BioFire Diagnostics, Salt Lake City, UT). This fully automated nested multiplex PCR assay simultaneously screens for 14 neurotropic pathogens: six bacteria (*Escherichia coli* K1, *Haemophilus influenzae*, *Listeria monocytogenes*, *Neisseria meningitidis*, *Streptococcus agalactiae*, *Streptococcus pneumoniae*), seven viruses (cytomegalovirus, enterovirus, herpes simplex virus 1, herpes simplex virus 2, human herpesvirus 6, human parechovirus, varicella-zoster virus), and one fungal target (*Cryptococcus neoformans/gattii*). Nucleic acid amplification for *Mycobacterium tuberculosis* complex in CSF was performed using the automated GeneXpert MTB/RIF real-time PCR assay.

Neuroimaging Protocols

Initial brain computed tomography was acquired as a non-contrast axial study to rule out mass effect and acute hemorrhage. Follow-up brain magnetic resonance imaging was conducted on a high-field scanner including T1-weighted, T2-weighted, fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), susceptibility-weighted imaging (SWAN), and 3D time-of-flight (TOF)

magnetic resonance angiography sequences, with and without intravenous gadolinium contrast administration, to detect parenchymal lesions, meningeal enhancement, or vascular pathology.

RESULTS

Clinical and Laboratory Findings in Cerebrospinal Fluid

Initial lumbar puncture revealed clear cerebrospinal fluid with a marked inflammatory response characterized by severe hyperproteinorrachia (1,590 mg/L), normal glucose concentration (66.1 mg/dL; concurrent serum glucose: 108 mg/dL), and a pleocytosis of 8,000 cells/ μ L. Direct wet-mount examination with India ink was preliminarily reported as "positive" due to the visualization of clear halos surrounding cellular elements (Figure 1). Repeat CSF cytochemical analysis on hospital day 3 confirmed persistent neutrophilic pleocytosis (20,000 cells/ μ L, 100% polymorphonuclear leukocytes), elevated protein levels (596 mg/L), and normal glucose (69.4 mg/dL; concurrent serum glucose: 120 mg/dL).

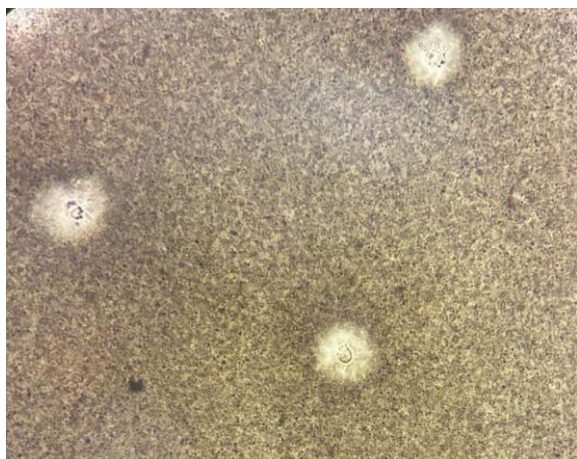


Figure 1. Wet India ink preparation of cerebrospinal fluid demonstrating a pseudo-cryptococcal artifact.

Light microscopy reveals clear halos surrounding cellular elements against a colloidal carbon background, mimicking the polysaccharide capsule of *Cryptococcus spp.*. These structures correspond to an exclusion artifact generated by marked leukocyte pleocytosis rather than true encapsulated yeasts with refractile cell walls or narrow-based budding.

Molecular and Microbiological Diagnostics

Multiplex real-time PCR testing of the CSF via the BioFire FilmArray ME Panel showed no detectable nucleic acids for any of the 14 targets, specifically resulting in "Not Detected" for *Cryptococcus neoformans/gattii*, *Listeria monocytogenes*, *Streptococcus pneumoniae*, *Neisseria meningitidis*, herpes simplex viruses 1 and 2, and enteroviruses. GeneXpert MTB/RIF in CSF was negative for *Mycobacterium tuberculosis* complex. Automated bacterial and fungal cultures of CSF yielded no

microbial growth after 7 days of incubation. Paired peripheral blood cultures showed no pathogen isolation. Blood serological testing was non-reactive for HIV-1/2 antibodies, hepatitis B surface antigen, anti-hepatitis C antibodies, and *Brucella abortus*. Serum procalcitonin levels remained within normal limits across serial evaluations (0.0299 ng/mL to 0.0489 ng/mL).

Neuroimaging and Therapeutic Outcomes

Initial unenhanced head computed tomography and subsequent brain magnetic resonance imaging with intravenous gadolinium contrast showed no leptomeningeal enhancement, parenchymal lesions, cryptococcomas, cerebral edema, or acute diffusion restriction on DWI sequences. Following molecular exclusion of *Cryptococcus spp.*, empirical induction therapy with liposomal amphotericin B (300 mg daily) and fluconazole (1,200 mg daily) was discontinued after 48 hours without clinical deterioration. Under supportive therapy and corticosteroids, the patient experienced complete resolution of fever, expressive aphasia, cognitive disorientation, and lower-extremity paraparesis, achieving full functional recovery prior to discharge.

DISCUSSION

This case highlights two critical diagnostic and therapeutic challenges in neuroinfectious disease management: establishing the diagnosis of aseptic meningoencephalitis in an immunocompetent host and recognizing microscopic pseudo-cryptococcal artifacts on cerebrospinal fluid (CSF) examination. The initial diagnostic confusion prompted aggressive empiric antifungal induction therapy, which was subsequently rationalized through the integration of multiplex molecular testing, sterile fungal cultures, and negative neuroimaging findings.¹

The clinical presentation of fever, expressive aphasia, cognitive disorientation, and marked nuchal rigidity with positive Kernig and Brudzinski signs strongly pointed to an acute meningoencephalic process. In acute aseptic meningoencephalitis, routine bacterial and fungal cultures of the CSF remain sterile, while cytochemical parameters typically reveal marked pleocytosis and hyperproteinorrachia with preserved or mildly reduced glucose levels. Although viral pathogens represent the leading infectious cause of aseptic meningitis, up to two-thirds of presumed viral central nervous system infections yield negative results on routine molecular panels due to early viral clearance, low viral loads, or agents not covered by standard assays. Furthermore, non-infectious etiologies must be considered, including drug-induced aseptic meningitis associated with nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen and naproxen, which classically induce sudden neutrophilic pleocytosis and rapid clinical resolution upon drug withdrawal.²

True central nervous system cryptococcosis caused by *Cryptococcus neoformans* or *Cryptococcus gattii* carries severe morbidity and high case fatality rates if untreated or inappropriately managed. While *Cryptococcus neoformans* is predominantly recognized in patients with advanced HIV infection and profound CD4 lymphopenia, invasive disease and large cerebral cryptococcomas caused by *Cryptococcus gattii* have been increasingly characterized in immunocompetent hosts. In genuine fungal meningoencephalitis, clinical deterioration is usually accompanied by elevated intracranial opening pressure, low CSF glucose levels, positive fungal cultures, and distinct leptomeningeal or parenchymal enhancement on contrast-enhanced neuroimaging.³

The initial misclassification in our patient arose from a false-positive reading on direct wet India ink microscopy of the CSF. Although wet India ink mounting remains a standard, inexpensive test for detecting encapsulated yeasts, its sensitivity and specificity are variable and heavily technician dependent. In specimens with marked leukocyte pleocytosis, intact and disintegrating polymorphonuclear leukocytes can displace the colloidal carbon particles, creating a clear pericellular halo that mimics the mucopolysaccharide capsule of *Cryptococcus spp.* As demonstrated experimentally by Thiruchelvan et al., high-speed centrifugation and cellular membrane damage can lead to the extravasation of viscous nuclear DNA around leukocytes, generating a morphological pseudocapsule that lacks true fungal features such as double-contoured refractile walls or narrow-based budding.^{4,5} The rapid introduction of the BioFire FilmArray ME Panel in this case was decisive for diagnostic reconciliation. Automated multiplex real-time PCR platforms provide simultaneous detection of major bacterial, viral, and fungal neurotropic agents within two hours, demonstrating superior diagnostic accuracy compared to conventional direct microscopy. Coupled with negative fungal cultures at 7 days, normal procalcitonin levels, and the absence of cryptococcal lesions on brain MRI, the negative FilmArray result provided definitive evidence to exclude true cryptococcal invasion and avoid unwarranted, prolonged antifungal courses.⁶

The prompt discontinuation of liposomal amphotericin B and high-dose fluconazole prevented avoidable iatrogenic complications. Polyene-based induction regimens are notoriously associated with severe nephrotoxicity, acute tubular necrosis, electrolyte wasting, and anemia, underscoring the necessity of diagnostic stewardship before committing patients to weeks of toxic therapy. This case illustrates that in pleocytic CSF specimens with discordant microscopic findings, clinicians must rely on rapid molecular assays, antigen testing, and cultural correlation to rule out pseudo-cryptococcal artifacts and safely de-escalate therapy in aseptic meningoencephalitis.⁷

CONCLUSION

Marked leukocyte pleocytosis in cerebrospinal fluid represents a significant diagnostic pitfall during routine direct wet India ink microscopy examinations, as cellular crowding and mechanical trauma can generate distinct pericellular exclusion halos that closely mimic encapsulated *Cryptococcus* species. These morphologic artifacts, frequently arising after high-speed centrifugation and membrane disruption, lack the defining microbiological features of true blastoconidia, such as refractile double-contoured cell walls, internal cytoplasmic structures, and characteristic narrow-based budding. Relying solely on preliminary or operator-dependent microscopic findings in heavily pleocytic samples can precipitate substantial clinical misinterpretation and prompt immediate, unwarranted therapeutic escalations in patients who do not harbor true invasive fungal disease.

To resolve discordant direct microscopic evaluations, clinicians must systematically correlate cytochemical patterns with high-precision diagnostic modalities, including automated multiplex real-time polymerase chain reaction panels, lateral flow cryptococcal antigen testing, and automated fungal cultures. The implementation of the BioFire FilmArray ME Panel provides rapid, highly sensitive, and specific simultaneous detection across multiple bacterial, viral, and fungal targets within two hours, offering definitive diagnostic clarity and effectively ruling out opportunistic pathogens. Integrating advanced molecular diagnostics into routine emergency neuroinfectious workups minimizes reliance on ambiguous microscopic stains, accelerates accurate syndromic identification, and strengthens overall diagnostic stewardship in tertiary care hospital settings.

Ultimately, the timely exclusion of central nervous system cryptococcosis through combined molecular and microbiological confirmation enables early, safe de-escalation of aggressive empiric antifungal regimens. This rapid discontinuation directly prevents prolonged exposure to polyene-based therapies, substantially mitigating the known risks of severe nephrotoxicity, acute tubular necrosis, profound electrolyte wasting, and treatment-related anemia. Furthermore, establishing a definitive diagnosis of aseptic meningoencephalitis redirects the clinical strategy toward appropriate supportive care and targeted anti-inflammatory management, ensuring optimal functional recovery and preventing avoidable iatrogenic morbidity.

DECLARATIONS

Ethics Approval and Consent to Participate

This case report was conducted in accordance with the ethical principles of the Declaration of Helsinki and applicable institutional guidelines. Institutional ethics

committee approval was waived because this report involved a single anonymized case, in accordance with institutional policy.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical images. All potentially identifiable personal information was removed or anonymized.

Availability of Data and Materials

The clinical data supporting the findings of this case report are available from the corresponding author upon reasonable request, subject to institutional policies and protection of patient confidentiality.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

MRN, GRG, EMRR, KFON, LFHQ, FJVL, VVR, and MRMP contributed to the clinical management, data collection, interpretation of clinical and laboratory findings, and preparation of the manuscript. All authors reviewed the manuscript critically, approved the final version, and agree to be accountable for all aspects of the work.

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