

## CASE SERIES

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# Cryptococcus in the absence of immunosuppression: Diagnostic and therapeutic challenges

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## ABSTRACT

Invasive cryptococcal infections, particularly those caused by *Cryptococcus neoformans*, are significant contributors to morbidity and mortality among immunocompromised patients, especially individuals living with human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS). Recognized as a critical fungal pathogen by the WHO in 2022, the increasing prevalence of such infections raises concerns, particularly as case reports emerge detailing instances in seemingly immunocompetent individuals. Here we report two cases, one in which the host had no known immunocompromising condition at time of diagnosis, and the other in which invasive infection was found without clear evidence of pulmonary or neurological involvement. We discuss the evolving epidemiology and diverse manifestations of invasive cryptococcal infections, and emerging associations with conditions that may predispose to infection in the seemingly immunocompetent host. These cases highlight disseminated cryptococcosis in patients without traditional severe immunosuppression, emphasizing the need for high clinical suspicion.

**Keywords:** *Cryptococcus neoformans*, Diffuse large B-cell lymphoma, Disseminated cryptococcosis, NASH cirrhosis, Non-HIV immunosuppression

## How to cite this article

Gayed S, Schworer S, Yalin E, Stromberg J, Herce M. Cryptococcus in the absence of immunosuppression: Diagnostic and therapeutic challenges. J Case Rep Images Infect Dis 2026;9(1):13–18.

Article ID: 100021Z16SG2026

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doi: 10.5348/100021Z16SG2026CS

## INTRODUCTION

Invasive cryptococcal infections in immunocompetent individuals have been thought to be a rare occurrence, and most published case studies highlight possible risk factors, including undiagnosed immunodeficiencies, malignancies, chronic lung disease, and host genetics [1]. However, the prevalence of cryptococcus in individuals without known causes of immunosuppression is higher than conventionally thought, representing 20% of reported cases in one multicenter cohort study of 306 patients from 1990 to 1996 [2]. Additionally, common conditions such as diabetes, NASH cirrhosis, or alcohol use may represent subtle ongoing immunosuppression that predisposes to infection. Traditionally, pulmonary and neurological involvement were felt to be the hallmarks of invasive infection. But clinical presentations can vary widely, and the underlying pathophysiology is not always well understood, which can create diagnostic and therapeutic challenges.

## CASE SERIES

### Case 1: Disseminated Cryptococcal Infection

A male patient in his seventies presented to the emergency department with altered mental status for a

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Received: 22 November 2026

Accepted: 26 February 2026

Published: 16 May 2026

week prior to admission, weakness, and recurrent falls without loss of consciousness. Although the patient did not have classical immunosuppression, he did have a history of uncontrolled diabetes, NASH cirrhosis (complicated by esophageal varices and a history of trans jugular intrahepatic portosystemic shunt in 2016), and a history of diffuse large B cell lymphoma, diagnosed 2018 and in remission (with a history of receiving chemotherapy several years prior, but details not known at time of admission), which likely contributed to altered host defenses. He also had coronary artery disease, atrial fibrillation, asthma, gout, and hypertension. He lived with his family and was a former smoker with a 6-pack year history but quit 26 years ago.

He initially presented to an outside hospital for worsening altered mental status and, in the emergency department (ED), was noted to have a fixed upper gaze and seizure-like activity. A code stroke was deployed, and a head computed tomography (CT) was notable for new left-sided subdural hematoma, thought due to falls at home. He was transferred to our tertiary center for intensive care. He became febrile one week into his hospitalization and remained persistently febrile, prompting the collection of peripheral and central line blood cultures and infectious disease evaluation. His hospital course was further complicated by progressively worsening mental status and generalized jerking movements, prompting anti-epileptic treatment. A repeat CT head revealed bilateral subdural hygromas and an increase in size of the left-sided subdural hematoma. He had an electroencephalogram which showed features of moderate encephalopathy, but no seizures or epileptiform activity. He eventually required intubation for airway protection.

## Case 2: Cryptococcal Meningitis

A male patient in his sixties with a history of lumbar disc herniation and spinal stimulator placement in 2021, hypothyroidism, and norm calcemic hyperparathyroidism presented after a fall while gardening and was noticed to have anisocoria. Over the month preceding presentation, he reported progressive lower extremity weakness, headaches, retro-orbital pain, light-headedness, and blurry vision. He was previously seen in the emergency department for these symptoms, along with chest pain, and was discharged with plans for outpatient cardiac workup. Subsequently, he started having cognitive issues, including new difficulties with basic daily activities. He lived with his wife and had no history of exposure to birds or caves, although he vacationed in Kentucky, Ohio, and Pennsylvania over the prior year. In terms of surgical history, he had multiple lumbar microdiscectomies at L4-5 with eventual L4-5 posterior lumbar interbody fusion. This was complicated by persistent dysesthesias in his left foot, prompting dorsal column stimulator placement at T8 in 2021. He also had carpal tunnel release surgery one month prior to presentation.

His neurological exam was notable for significantly impaired attention and concentration. He was noted to have increased reflexes, graded as 2 plus in both upper and lower extremities symmetrically. He was also noted to have anisocoria and the absence of a pupillary constriction to light in both eyes, prompting an Ophthalmology evaluation. He underwent a lumbar puncture, and Infectious Disease was consulted based on his cerebrospinal fluid culture findings, below.

## Investigations

Case 1: Initial workup was notable for a negative HIV test, a leukocytosis with neutrophilic predominance (Table 1), and negative blood cultures from peripheral draws and a central line. However, repeat blood peripheral cultures yielded *Cryptococcus neoformans*. The serum cryptococcal antigen was also positive, with a titer of 1:640. A lumbar puncture was not clinically feasible due to the presence of a rightward midline shift and the associated risk of uncal herniation, limiting central nervous system (CNS) assessment. The image of the chest did not indicate infection, and echocardiography showed no valvular vegetations. An ophthalmology examination revealed multiple white creamy lesions deep to the retina in the left eye, consistent with fungal endophthalmitis (Figure 1).

Table 1: Complete blood count for patient 1

Lab name	Result	Reference range
WBC	<b>17.3 H</b>	3.6–11.2 × 10 <sup>9</sup> /L
RBC	2.82	4.26–5.60 × 10 <sup>12</sup> /L
Hgb	<b>8.3 L</b>	12.9–16.5 g/dL
Hct	<b>25.2</b>	39.0–48.0%
MCV	89.3	77.6–95.7 fL
MCH	29.6	25.9–32.4 pg
MCHC	33.1	32.0–36.0 g/dL
RDW	<b>18.5 H</b>	12.2–15.2%
MPV	9.8	6.8–10.7 fL
Platelet	<b>147 L</b>	150–450 × 10 <sup>9</sup> /L
Absolute neutrophils	<b>13.9 H</b>	1.8–7.8 × 10 <sup>9</sup> /L
Absolute lymphocytes	1.4	1.1–3.6 × 10 <sup>9</sup> /L
Absolute monocytes	1.5	0.3–0.8 × 10 <sup>9</sup> /L
Absolute eosinophils	0.3	0.0–0.5 × 10 <sup>9</sup> /L
Absolute basophils	0.1	0.0–0.1 × 10 <sup>9</sup> /L
Anisocytosis	Slight	Not present
Polychromasia	Slight	Not present

H: Abnormal high; L: Abnormal low.

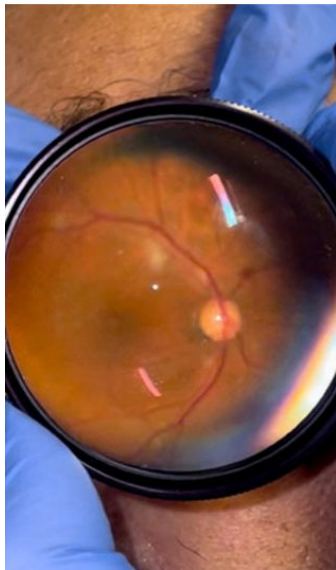


Figure 1: Ophthalmic examination of patient 1, showing multiple white creamy lesions deep to the retina in the left eye.

Case 2: Initial laboratory tests revealed a normal total white blood cell count, normal kidney and liver function, and negative HIV and rapid plasma reagin (RPR) tests. A lumbar puncture was obtained with an opening pressure of 16 cm H<sub>2</sub>O, a leukocytic pleocytosis (52 cells/ $\mu$ L, reference  $\leq 5$   $\mu$ L), elevated protein (89 mg/dL, reference 19–65 mg/dL), and hypoglycorrhachia (31 mg/dL, reference 48–79 mg/dL). A cerebrospinal fluid (CSF) culture was positive for *Cryptococcus neoformans*. A CSF cryptococcal antigen was positive with a titer of 1:1280 and a serum cryptococcal antigen was also positive with a titer of 1:5120. A complete blood count (CBC) with differential was within normal limits except for persistent lymphopenia throughout admission (range of 0.4–0.9, reference  $1.1\text{--}3.6 \times 10^9/\text{L}$ ). His absolute CD4, CD8, CD3, and CD19 cell counts were below normal range, while immunoglobulin levels were within normal to low normal ranges (Table 2). Tetanus and diphtheria antibodies returned at protective levels. Per chart review, the patient was lymphopenic as early as 2019, with absolute lymphocyte count  $0.8 \times 10^9/\text{L}$  (reference  $1.5\text{--}5.0 \times 10^9/\text{L}$ ).

## Differential Diagnosis

Case 1—differential diagnosis for persistent fevers in intensive care unit (ICU)

- Invasive fungal infection, as patient was febrile on broad spectrum antibiotics
- Central nervous system infection, secondary to bilateral hygromas and subdural hematoma as nidus
- Syphilis vasculitis
- Silent aspiration events
- Hospital acquired pathogen via tracheostomy tube

- Acalculous cholecystitis versus Pancreatitis
- Pressure ulcer infection
- Possible drug related fever reaction

Case 2—differential:

- Cryptococcal meningoencephalitis
- Bacterial meningoencephalitis
- CNS Syphilis infection
- CNS viral infection: arbovirus, varicella zoster, toxoplasmosis, Epstein–Barr virus (EBV), cytomegalovirus (CMV), adenovirus, enterovirus
- CNS malignancy

Table 2: Immunology test results for patient 2

Lab name	Result	Reference range
CD3% (T cells)	<b>55 L</b>	61–86% {of lymphs}
CD4% (T helper)	40	34–58% {of lymphs}
CD8% T suppressor	15	12–38% {of lymphs}
CD19% (B cells)	8	7–23% {of Lymphs}
CD16/56% NK cell	34	1–27% {of lymphs}
Absolute CD3 count	<b>330 L</b>	915–3400 {cells}/ $\mu$ L
Absolute CD4 count	<b>240 L</b>	510–2320 {cells}/ $\mu$ L
Absolute CD8 count	<b>90 L</b>	180–1520 {cells}/ $\mu$ L
Absolute CD19 count	<b>48 L</b>	105–920 {cells}/ $\mu$ L
Absolute CD16/56	204	15–1080 {Cells}/ $\mu$ L
CD4:CD8 ratio	2.7	0.9–4.8
IgM	<b>33 L</b>	40–230 mg/dL
Total IgG	754	650–1600 mg/dL
IgA	150.5	70.0–400.0 mg/dL
Total IgE	48.3	2–214 IU/mL
Diphtheria IgG Ab	Positive, >1.00	Vaccinated: Positive ( $\geq 0.01$ IU/mL)
Tetanus IgG Ab	Positive, 0.49	Vaccinated: Positive ( $\geq 0.01$ IU/mL)

H: Abnormal High; L: Abnormal low; Ab: Antibody

## Treatment

Case 1: The patient was initiated on liposomal intravenous (IV) Amphotericin B 4 mg/kg daily and flucytosine 25 mg/kg four times a day for disseminated cryptococcal infection.

Case 2: The patient was started on induction treatment for cryptococcal meningitis with liposomal IV amphotericin B 4 mg/kg daily and flucytosine 25 mg/kg four times a day.

## Outcome and Follow-up

Case 1: Due to the progression of the hematoma on imaging and worsening mental status attributed to worsening intracranial hemorrhage, he was transitioned to comfort care. The patient is currently at home with hospice.

Case 2: The patient had steady improvement in cognition with cryptococcal-directed therapy. A repeat lumbar puncture showed an opening pressure of 7 cm H<sub>2</sub>O, persistent leukocytic pleocytosis (63 cells/ $\mu$ L, reference  $\leq$ 5/ $\mu$ L), elevated protein (67 mg/dL, reference 19–65 mg/dL), and normal glucose level (48 mg/dL, reference 48–79 mg/dL), all improved compared to initial lumbar puncture. Cerebrospinal fluid culture finalized without growth. The patient was transitioned to maintenance therapy of fluconazole 800 mg daily, discharged, and has since continued to improve at home, approaching his baseline.

Immunology was consulted for assistance in working up underlying immunological factors. His pneumococcal antibody levels were protective ( $>$ 1.3 mcg/mL) for 15/23 serotypes. However, the patient received the pneumococcal 20-valent conjugate vaccine two months prior to admission and thus interpretation of these values is limited. Lymphocyte proliferation in response to mitogens found that on day 0 lymphocyte viability at 74%, just barely below the threshold of 75%, but T and B cell proliferation responses to mitogens phytohemagglutinin (PHA) and pokeweed mitogen (PWM) were normal. A mutation panel was sent to evaluate for genetic causes of primary immunodeficiency, but was unrevealing. Following this workup, a repeat lymphocyte subsets count was repeated 6 weeks after his initial presentation, and showed that his CD4 count was 462, while his CD3 count was 682—both still lower than normal range, but improved from prior. His transient lymphopenia suggests temporary immune dysregulation, predisposing to opportunistic infection despite the lack of an overt immunodeficiency.

## DISCUSSION

*Cryptococcus* spp. are environmental basidiomycetous fungi [3] first identified by Busse and Buschke in 1894 [4]. A notable increase in invasive cryptococcal infections has been observed since the HIV pandemic [5], with *Cryptococcus neoformans* and *Cryptococcus gattii* being the primary species involved in cryptococcal disease. *C. neoformans* primarily comprises serotypes A and D, while *C. gattii* includes serotypes B and C [5]. Due to widespread vaccination against *Streptococcus* spp. and *H. flu* and increased use of immunosuppressive therapy, *Cryptococcus* spp. have become an increasingly common cause of CNS infection worldwide, including in patients without a known immunocompromising condition at time of diagnosis. These patients have lower rates of positive CSF cultures and are more likely to clear cultures after two weeks of therapy.

*Cryptococcus* spp. possess several virulence factors, with the most significant being a thick polysaccharide capsule that aids in evading phagocytosis by the host immune system [5]. *Cryptococcus neoformans* exhibits unique pathogenicity, notably its ability to survive at 37°C

and secrete diphenol oxidase, which facilitates melanin production. Melanin not only enhances survival in the environment but also renders melanized *C. neoformans* more resistant to degradation by host defenses, including cell wall-degrading enzymes [6].

Diphenol oxidase functions as an antioxidant, enabling extended survival within host cells by consuming superoxide ions, although it primarily protects against extracellular enzymes rather than intracellular ones. This capability is a major virulence factor, contributing to the organism's pathogenicity [7]. Both *C. neoformans* and *C. gattii* have a propensity to infect the central nervous system, especially in patients with impaired cell-mediated immunity. In addition to neurological infections, *Cryptococcus* can affect the lungs, skin, bones, joints, and eyes [8]. The antiphagocytic capabilities of *Cryptococcus* allow it to evade phagocytosis by macrophages, employing a survival strategy akin to that seen in environmental amoebas [9]. This phenomenon, often referred to as the “Trojan horse” mechanism, facilitates dissemination through the bloodstream and the crossing of the blood-brain barrier, leading to invasion in microcapillaries [10]. *Cryptococcus gattii*, primarily found in tropical regions, has been associated with infections in immunocompetent hosts, whereas *C. neoformans* predominantly causes disease in those with impaired immune systems, such as people with HIV [11].

As *Cryptococcus* infections become more prevalent overall, diverse clinical presentations must be kept in mind when evaluating for the pathogen. For the patient in case 1 with NASH cirrhosis, there is a known association of susceptibility to *cryptococcus* in patients with cirrhosis [12]. Research suggests immune dysfunction in the setting of cirrhosis—specifically impaired cell-mediated immunity and surveillance—may be important in driving susceptibility to *Cryptococcus*, especially spread to less common organ systems [13]. Cirrhosis is associated with a 5.3 fold higher rate of extrapulmonary dissemination [14] and was likely a risk factor for our patient. While the cause of the patient's subdural hematoma was thought to be due to recent falls, we do note the inability to definitively rule out a CNS infection with a lumbar puncture.

Meanwhile, there have been increasing numbers of invasive *C. neoformans* infections found in seemingly immunocompetent individuals, prompting further investigation into these atypical presentations. The patient from case 2 was initially suspected to have idiopathic CD4 lymphopenia, due to the history of persistent lymphopenia in the absence of exposure to immunomodulating medications, viral infections, or malignancy. Other case reports have been published documenting the connection between idiopathic CD4 lymphopenia and disseminated *cryptococcus* infections [15]. However, as noted above, repeat lab work six weeks later showed improvement in all lymphocyte lines, and his absolute CD4 count of 462 cells/ $\mu$ L put him above the threshold (less than 300) required for diagnosis of idiopathic CD4 lymphopenia [16]. The underlying etiology

of his years-long lymphopenia, as well as the cause of its resolution, remains unclear, but his case emphasizes the importance of considering Cryptococcal infections in patients without overt immunocompromising conditions.

## CONCLUSION

These cases underscore that disseminated cryptococcosis can occur in patients without classical immunosuppression, particularly those with subtle immune compromise. Early recognition and prompt antifungal therapy are critical for favorable outcomes.

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## Author Contributions

Salma Gayed – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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**Guarantor of Submission**

The corresponding author is the guarantor of submission.

**Source of Support**

None.

**Consent Statement**

Written informed consent was obtained from the patient for publication of this article.

**Conflict of Interest**

Authors declare no conflict of interest.

**Data Availability**

All relevant data are within the paper and its Supporting Information files.

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