Isolated basal ganglia mucormycosis in an immunocompetent man with a history of intravenous drug use: a case report

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ABSTRACT

Background: Isolated cerebral mucormycosis is a rare, life-threatening infection, usually occurring in immunocompromised hosts but also reported in people with intravenous drug use, with a predilection for the basal ganglia.

Case Presentation: A 30-year-old previously healthy man with a history of intravenous drug use presented with new-onset generalized seizures, altered mental status, and left-sided hemiplegia. Magnetic resonance imaging showed a 4.4 × 4.3 × 5.1 cm ring-enhancing mass in the right basal ganglia with extension into the thalamus and temporal lobe and mild midline shift. Stereotactic biopsy with conservative debulking revealed a fungal abscess; Gomori methenamine silver staining demonstrated broad pauci-septate hyphae with irregular right-angle branching, supporting a diagnosis of cerebral mucormycosis. He was treated with liposomal amphotericin B and isavuconazole, followed by prolonged oral isavuconazole. Complete excision was not feasible because of the deep lesion location.

Conclusion: Follow-up magnetic resonance imaging at 16 weeks showed a reduction in lesion size and edema with partial neurologic improvement, although residual left-sided weakness persisted. This case highlights the need to consider isolated cerebral mucormycosis in basal ganglia lesions in patients with intravenous drug use, even without traditional immunosuppressive risk factors. Early tissue diagnosis, prompt antifungal therapy, and multidisciplinary surgical assessment are essential, particularly when complete resection carries a high risk.

Keywords: Mucormycosis, basal ganglia, cerebral infection, intravenous drug use, amphotericin B, isavuconazole, central nervous system fungal infection.

Type of Article: CASE REPORT **Specialty:** Infectious Diseases

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Introduction

Mucormycosis is a life-threatening invasive fungal infection caused by molds of the order Mucorales, most commonly *Rhizopus*, *Mucor*, and *Rhizomucor* species [1]. It usually occurs in patients with impaired host defenses, including those with uncontrolled diabetes mellitus, hematologic malignancy, neutropenia, corticosteroid exposure, transplantation, iron overload, or advanced human immunodeficiency virus infection [1,2]. Intravenous drug use is an additional recognized risk factor, especially in cases with central nervous system involvement [3-5].

The most common clinical forms are rhino-orbito-cerebral and pulmonary disease, with cerebral involvement usually occurring by contiguous extension from the sinuses or orbit [2]. In contrast, isolated cerebral mucormycosis without sinonasal or pulmonary disease is rare and has been reported predominantly in people with a history of intravenous drug use [3-5]. In these cases, the basal ganglia are frequently involved, possibly because of hematogenous seeding to this highly vascular deep gray matter region [3-5].

We report a case of isolated basal ganglia mucormycosis in an immunocompetent young man with a history of intravenous drug use who showed early radiologic improvement and partial neurologic recovery after limited neurosurgical intervention and antifungal therapy.

Case Presentation

A 30-year-old previously healthy man presented with a 2-day history of new-onset generalized seizures, altered mental status, and left-sided hemiplegia. He was intubated on arrival and admitted to the intensive care unit. Because of his altered sensorium and mechanical ventilation, the history was limited. Examination showed 0/5 motor

power in the left upper and lower limbs, increased tone, hyperreflexia, and a positive Babinski sign on the left. No cranial nerve deficits were documented. Track marks were present on both forearms, supporting a history of intravenous drug use. No other cutaneous signs of infection were identified.

Magnetic resonance imaging of the brain demonstrated a 4.4 × 4.3 × 5.1 cm ring-enhancing mass centred in the right basal ganglia, with extension into the right thalamus and temporal lobe and mild leftward midline shift (Figure 1A). There was no radiologic evidence of sinus disease or extracranial extension. The patient underwent stereotactic-guided mini-craniotomy and corticectomy for tissue diagnosis and conservative debulking. Frozen section examination showed necrotic brain tissue with fungal hyphae, raising concern for a fungal abscess. Permanent histopathology demonstrated glial tissue with suppurative necrosis and numerous fungal spores and hyphae. Periodic acid-Schiff and Gomori methenamine silver stains highlighted abundant fungal elements. The formal report described a necroinflammatory process consistent with a fungal abscess and found no evidence of malignancy. Species-level identification was not available. However, the Gomori methenamine silver (GMS)-stained sections were retrospectively reviewed by the treating clinical team, in conjunction with the consultant pathologist, and demonstrated broad pauci-septate hyphae with irregular right-angle branching, supporting a diagnosis of mucormycosis in the appropriate clinico-radiologic context (Figure 2).

Fungal cultures were performed on biopsy material; however, growth was negative, and polymerase chain reaction-based molecular identification was not pursued at our institution at the time of diagnosis. The diagnosis therefore rested on the combination of characteristic histomorphology on GMS stain and the supporting clinico-radiologic context. Serum beta-D-glucan was negative (<31 pg/ml), a finding that does not exclude mucormycosis because Mucorales are typically not detected by this assay. Additional workup, including human immunodeficiency virus serology, fasting glucose, and glycated hemoglobin, were unremarkable. Computed tomography of the sinuses, chest, abdomen, and pelvis did not reveal an extracranial source of infection.

Following biopsy and limited debulking, antifungal therapy was started with liposomal amphotericin B at 5 mg/kg intravenously once daily and isavuconazole at a loading dose of 200 mg intravenously every 8 hours for 6 doses, followed by 200 mg daily. Based on the pathology findings and the clinico-radiologic picture, the lesion was managed as presumed proven invasive cerebral mucormycosis according to EORTC/MSGERC Guidelines [6]**.** Because of the deep basal ganglia location and predicted surgical morbidity, complete excision was not considered feasible, and further immediate aggressive resection was deferred. The patient was later transitioned to prolonged oral isavuconazole, with infectious diseases follow-up documenting a planned total treatment duration of 18–24 months because of residual deep-seated disease and incomplete radiologic resolution.

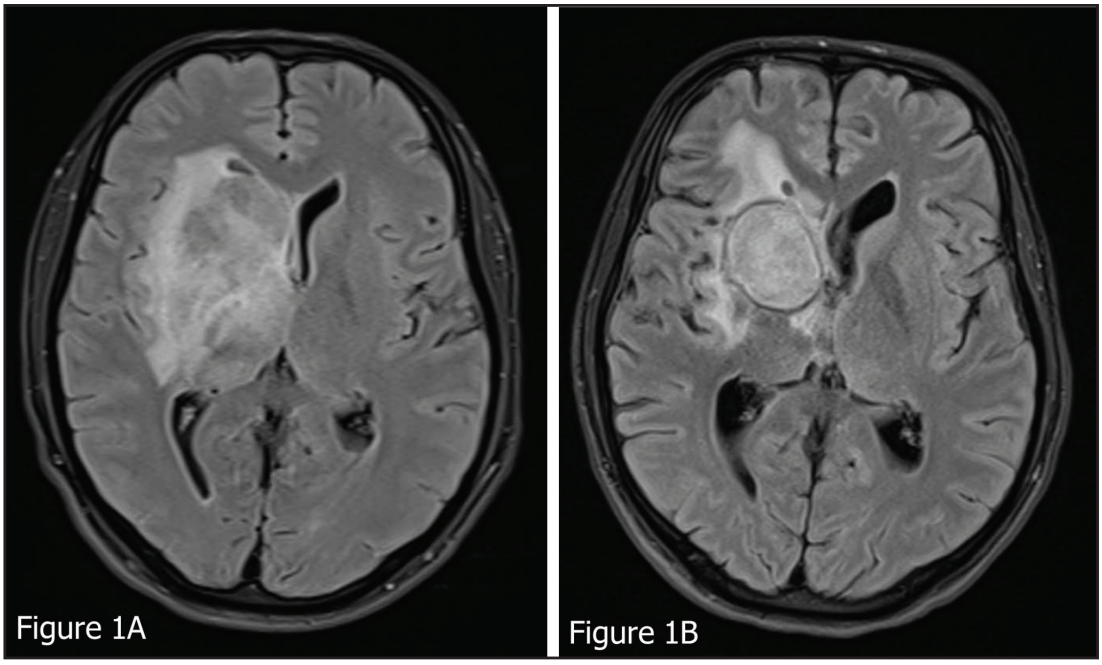

Figure 1. *A: T1-weighted axial view of MRI demonstrating an enhancing right basal ganglia mass measuring 4.4 × 4.3 × 5.1 cm, extending into the right thalamus and right temporal lobe, with mild leftward midline shift. B: T1-weighted axial view of MRI at 16 weeks post-treatment showing reduction in the right basal ganglia mass to (4.0 × 2.9 × 3.0 cm), with decreased peri-lesional edema and improved midline shift.*

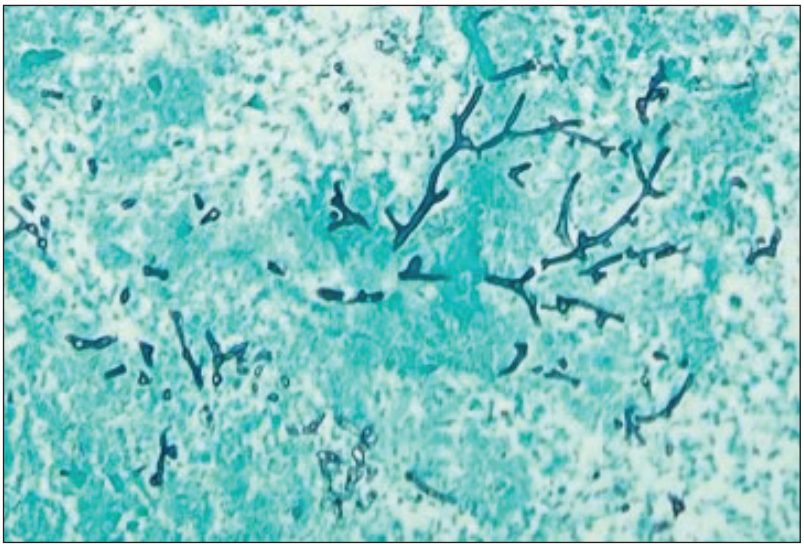

Figure 2. *Gomori methenamine silver (GMS) stain showing broad, aseptate hyphae with irregular, right-angle branching invading brain parenchyma (as shown in black arrows)—features consistent with Mucorales species.*

Serial imaging showed no significant interval change at 6 weeks. At 16 weeks, follow-up magnetic resonance imaging demonstrated a reduction of the lesion to 4.0 × 2.9 × 3.0 cm, with decreased perilesional edema and resolution of the midline shift (Figure 1B). Clinically, the patient showed gradual neurologic improvement but had persistent left-sided weakness, more pronounced in the upper limb, and later developed a seizure disorder requiring levetiracetam. During long-term follow-up, his course was complicated by interruptions in clinic attendance and medication access, with documented periods off isavuconazole. On a subsequent visit following a documented period of isavuconazole, the patient presented with recurrent headache and vomiting. Clinical assessment raised concern for disease relapse, and reinitiation of dual antifungal therapy was recommended. Repeat magnetic resonance imaging was planned at that visit, but could not be obtained before the patient was lost to follow-up. The absence of imaging confirmation means that relapse remains clinically suspected rather than radiologically confirmed, which is acknowledged as a limitation. Amphotericin-related hypokalemia and hypomagnesemia were documented and managed with monitoring, hydration, and electrolyte replacement. Subsequent long-term follow-up was incomplete because the patient stopped attending the infectious diseases clinic, and the final outcome after the planned antifungal course could not be determined.

Discussion

Isolated cerebral mucormycosis is an uncommon but well-recognized presentation of invasive Mucorales infection. Unlike the more typical rhino-orbito-cerebral form, isolated brain involvement has been reported mainly in young, otherwise immunocompetent men with a history of intravenous drug use [3-5,7]. In the patient-level meta-analysis by Kerezoudis et al., intravenous drug use was the dominant risk factor, and basal ganglia involvement was a recurring pattern [3]. Similar observations have been reported in earlier case series and reviews [4,5,7,8]. Our case closely fits this clinic-radiologic profile.

The predilection for the basal ganglia is thought to reflect hematogenous dissemination of fungal elements introduced during injection, followed by seeding of highly vascular deep gray matter structures [3-5,7,8]. Although our patient had no diabetes, human immunodeficiency virus infection, or other conventional immunosuppressive risk factors, intravenous drug use likely represented the major predisposing factor. The absence of sinus, pulmonary, or other extracranial disease further supported the diagnosis of isolated cerebral infection.

Diagnosis remains challenging because clinical presentation and neuroimaging may mimic a tumor, pyogenic abscess, or other fungal infections. Definitive diagnosis depends on tissue examination. In this case, biopsy with histopathology was decisive, demonstrating a fungal abscess with broad pauci-septate hyphae and irregular right-angle branching on Gomori methenamine silver stain, a morphology that strongly supported mucormycosis. Culture and molecular confirmation were unavailable, which is a recognized limitation in many reported cases [3,9]**.** When available, polymerase chain reaction and other molecular methods may help support the diagnosis in culture-negative disease [9]**.**

The initial use of liposomal amphotericin B at 5 mg/kg per day alongside concurrent isavuconazole represented an individualized clinical decision rather than a guideline-mandated combination. The ECMM/MSGERC 2019

global guideline strongly recommends liposomal amphotericin B as first-line therapy for cerebral mucormycosis, with doses of up to 10 mg/kg per day considered for CNS involvement, while isavuconazole is moderately recommended either as monotherapy or as a step-down agent following initial disease control [10]. Upfront combination antifungal therapy is only marginally supported in the same guideline, with no specific clinical data available for the liposomal amphotericin B plus isavuconazole pairing at the time of publication [10]. However, a subsequent murine model by Gebremariam et al. demonstrated synergistic activity of this combination against *Rhizopus delemar* and *Mucor circinelloides*, with an overall survival of 80% in the combination arm compared with 50% for either agent alone [11].

In the specific context of CNS mold infections where the causative pathogen remains uncertain at the time of treatment initiation, the rationale for upfront dual therapy becomes more defensible, though still not guideline recommended. CNS mold infections may be caused by *Aspergillus* species, for which voriconazole or isavuconazole constitutes first-line therapy, or by Mucorales, for which liposomal amphotericin B or isavuconazole are preferred [12,13]. The combination of both agents, therefore, provided broad empiric coverage for both diagnostic possibilities while awaiting further microbiologic results [13]. Furthermore, the ECMM/MSGERC mucormycosis guideline acknowledges that in cases of extensive disease, rapid progression, or poor general condition, the addition of isavuconazole or posaconazole to liposomal amphotericin B can be considered [10]. From a pharmacokinetic standpoint, liposomal amphotericin B achieves therapeutic concentrations in brain parenchyma despite limited cerebrospinal fluid distribution and is recommended at higher doses for CNS mucormycosis [10,14], while isavuconazole has intermediate CNS penetration and is FDA-approved for both aspergillosis and mucormycosis [13]. The combination therefore ensured parenchymal antifungal coverage via liposomal amphotericin B alongside an agent with broader CNS distribution.

CNS aspergillosis and mucormycosis carry extremely high mortality, and the 2016 IDSA aspergillosis guidelines acknowledge that combination therapy for CNS disease is initiated by some practitioners, given the mortality associated with this form of dissemination, while recognizing the absence of controlled data supporting improved outcomes [15]. Given the severity of the disease, the deep lesion location, and the inability to achieve complete surgical clearance, the clinical team chose upfront dual therapy on the basis of this collective evidence and the need to maximize antifungal coverage in a high-risk, surgically inaccessible lesion. Early transition to oral isavuconazole monotherapy for long-term suppression is consistent with guideline recommendations for step-down therapy following initial disease stabilization [10]. The deep basal ganglia location, involvement of eloquent structures, and expected surgical morbidity favored limited debulking rather than aggressive excision. Although surgical debridement is generally recommended when feasible, selected patients with surgically high-risk lesions may still achieve short-term disease control with biopsy or limited debulking combined with antifungal therapy [3,16].

The patient demonstrated interval radiologic improvement at 16 weeks and partial neurologic recovery, although persistent hemiparesis remained. This residual deficit likely reflects irreversible injury to deep motor pathways. His later interruptions in therapy and follow-up complicated long-term assessment and prevented confirmation of the final treatment outcome. Nevertheless, the early clinical and radiologic response observed supports the value of early tissue diagnosis, prompt initiation of antifungal therapy, and close multidisciplinary follow-up, though the inability to confirm the final outcome due to loss to follow-up remains an important limitation of this report.

Conclusion

Isolated basal ganglia mucormycosis is a rare but serious diagnostic consideration in patients with basal ganglia lesions and a history of intravenous drug use, even in the absence of traditional immunosuppressive risk factors. It must be acknowledged that the final treatment outcome in this case could not be established because the patient was lost to long-term follow-up, which is a recognized limitation of this report. Nevertheless, the early clinical and radiologic response observed supports the key educational messages: tissue diagnosis is essential, and early antifungal therapy with multidisciplinary surgical decision making is critical when lesions are deep and surgically high risk.

What is new?

This case adds to the small published literature on isolated cerebral mucormycosis in people who inject drugs by documenting a rare combination of features: an immunocompetent host, isolated basal ganglia involvement without any extracranial source, negative serum beta-D-glucan (highlighting the known insensitivity of this marker for Mucorales), and partial radiologic and neurologic recovery despite incomplete surgical resection. It further illustrates that prolonged oral isavuconazole represents a clinically viable step-down and maintenance strategy when complete resection is not feasible, and that treatment interruption carries a real risk of relapse even after initial radiologic improvement. The case also demonstrates the diagnostic value of careful morphologic review of Gomori methenamine silver-stained sections when culture and molecular confirmation are unavailable.

List of Abbreviations

BDG	Beta-D-glucan
CNS	Central nervous system
CT	Computed tomography
GMS	Gomori methenamine silver

HbA1c	Glycated hemoglobin
HIV	Human immunodeficiency virus
IVDU	Intravenous drug use
MRI	Magnetic resonance imaging
PAS	Periodic acid-Schiff
PCR	Polymerase chain reaction

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent for publication

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

Ethical Approval

Ethical approval is not required at our institution to publish an anonymous case report.

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Summary of the case

No.	Item	Details
1	Patient (gender, age)	30-year-old male
2	Final diagnosis	Isolated basal ganglia cerebral mucormycosis
3	Symptoms	New-onset generalized seizures, altered mental status, and left-sided hemiplegia
4	Medications	Liposomal amphotericin B, isavuconazole, levetiracetam
5	Clinical procedure	Stereotactic-guided mini-craniotomy, biopsy, and conservative debulking