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A case of Rhizopus infection in an immunocompetent IVDA host

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A case of *Rhizopus* infection in an immunocompetent IVDA host

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Abstract

Introduction: Mucormycosis is a rare fungal infection most often seen in patients with neutropenia, diabetic ketoacidosis, or prolonged use of corticosteroids. Here we present an unusual case of isolated cerebral mucormycosis in a young, immunocompetent patient with a history of intravenous drug abuse (IVDA).

Discussion: Though isolated cerebral mucormycosis is rare, it has an exceedingly poor prognosis with high morbidity and mortality. The most significant risk factor for isolated cerebral infection is IVDA, likely secondary to injected illicit drugs contaminated with *Rhizopus* spores. These patients typically present with brain abscess in the basal ganglia. Management includes source control (abscess debridement) and aggressive amphotericin B therapy.

Conclusions: High clinical suspicion for isolated cerebral mucormycosis in IVDA patients with focal neurologic deficits can greatly improve patient outcomes. Early intervention with stereotactic brain biopsy, amphotericin B, and abscess debridement can increase the likelihood of survival and can minimize permanent neurologic sequelae.

Central Nervous System Mucormycosis

Risk Factors

Diabetes Mellitus
Malignancy
Stem cell transplantation
IVDA[6]

Organism

Most common: ***Rhizopus*** species
Other: ***Mucor***, ***Lichtheimia*** species
Rhizopus, *Mucor* and *Lichtheimia* constitute ~ 80% of mucormycosis cases[6]

Therapy and Management

Surgical intervention[6]
- **Sinus debridement** (if rhino cerebral)
-**Neurosurgery** if there is evidence of increased intracranial pressure or hydrocephalus

Antifungal therapy[6]
- Liposomal **amphotericin B** 5-10 mg/kg/day IV for initial 28 days
-Alternative: **Isavuconazole** 300mg TID for 2 days followed by 300mg QD, IV or PO
- Step-down: **Isavuconazole** 300mg TID for 2 days followed by 300mg QD PO
-Duration: at least **6 months**

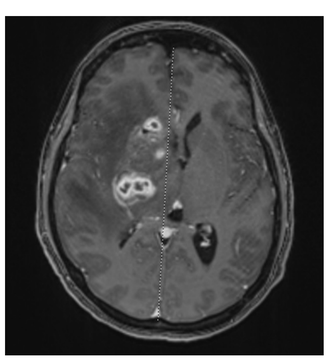
Case Presentation

A 36-year-old female with chronic Hepatitis C and IVDA presented with dense hemiparesis of the left upper and lower extremities upon wakening. Her Glasgow Coma Scale was 15. Magnetic Resonance Imaging (MRI) of the brain revealed a gadolinium-enhancing lesion adjacent to the right basal ganglia and thalamus consistent with an abscess, with surrounding vasogenic edema, mass effect causing a 6-millimeter right-to-left midline shift involving the right lateral ventricle.

The patient underwent stereotactic biopsy of the lesion which revealed multinucleated giant cells and scattered groupings of branching fungal forms consistent with zygomycete infection. Culture of the biopsy site was positive for *Rhizopus oryzae*. Her remaining infectious and immunologic workup was unremarkable, including undetectable Hepatitis C RNA and absent HIV antibodies. At this time, she was started on a regimen of amphotericin B and voriconazole.

Post-operative MRI revealed remaining ring-enhancing lesions in the right basal ganglia, worsening vasogenic edema, and hemorrhage within the lesions. Subsequently, amphotericin B dosage was increased and isavuconazole was added. During her hospital course, she exhibited new neurological findings including right lateral gaze palsy, left facial weakness, left hemi-neglect, and left ankle clonus. This was attributed to worsening vasogenic edema despite aggressive antifungal therapy. Four weeks after initial presentation, the decision was made for the patient to undergo hemicraniotomy for abscess debridement.

Once stabilized, she was discharged to an inpatient rehabilitation center on amphotericin B and andulafungin therapy for a 6-month course. On follow up examinations, she exhibited gradual improvement in her strength, however she continued to have visual field deficits and requires assistance with activities of daily living.



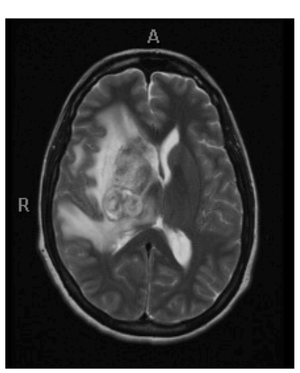


Figure 1 (Left): T1. Multiple solid and ring-enhancing masses scattered throughout the right basal ganglia extending into the right thalamus. Figure 2 (Right): T2. Extensive vasogenic edema with mass effect and midline shift.

Discussion

Mucormycosis is a rare, life-threatening fungal infection predominantly affecting immunocompromised hosts.[1] The most significant risk factors for this infection include diabetic ketoacidosis, neutropenia, prolonged high-dose glucocorticoid therapy, bone marrow or solid organ transplantation, iron overload, and IVDA.[4] Clinical presentation often reflects underlying risk factors for disease. For example, diabetic patients are likely to present with sinus involvement whereas patients with transplanted organs and malignancy more commonly present with pulmonary infection. Disseminated disease is most often seen in patients in an iron-overloaded state and in those treated with deferoxamine.[1,3]

Isolated cerebral infection constitutes just 5% of all mucormyosis infections, but has a fatality rate exceeding 60%.[3] Despite its rarity, however, isolated cerebral mucormycosis is the most common presentation among IVDA, constituting 62% of mucormycosis infections in this subgroup.[3] Brain lesions are most commonly located within the basal ganglia, likely secondary to hematogenous seeding of the perforating branches of the Middle Cerebral Artery.[4] It has been hypothesized that illicit injection drugs contaminated with mucormyosis spores enter the systemic arterial circulation before seeding the brain.[2] Improved survival rates are seen among those patients treated with early stereotactic biopsy and aggressive amphotericin B therapy.[4,5,6]

Conclusion

For patients with a history of IVDA who present with brain abscess (especially in the basal ganglia), mucormycosis should be on the clinicians' differential diagnosis. Early intervention with stereotactic brain biopsy and amphotericin B, as well as abscess debridement, can dramatically increase the likelihood of survival and can minimize permanent neurologic sequelae.

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