Review Article

A Comprehensive Review: Risk factors, Pathogenesis, Diagnosis and Management of Mucormycosis

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Abstract

Background: Mucormycosis, also known as black fungus, is a rare yet aggressive opportunistic fungal infection primarily caused by molds of the order *Mucorales*. It predominantly affects immunocompromised individuals, including those with uncontrolled diabetes, malignancies, organ transplants, or prolonged corticosteroid use. The COVID-19 pandemic further escalated mucormycosis incidence, particularly in countries like India, due to widespread steroid use and immune suppression. The pathogenesis involves spore inhalation or inoculation, followed by rapid hyphal growth, angioinvasion, tissue necrosis, and dissemination to organs such as the brain and lungs. Diagnosis relies on clinical suspicion supported by imaging, histopathology, culture, and molecular techniques. Radiological modalities like CT and MRI play crucial roles in early detection and surgical planning. Prompt and aggressive treatment combining antifungal therapy mainly liposomal amphotericin B or posaconazole and surgical debridement is vital for improving outcomes.

Conclusion: Despite advancements, mucormycosis remains associated with high morbidity and mortality, necessitating heightened awareness, early intervention, and risk factor control. This review consolidates current insights into the epidemiology, risk factors, pathogenesis, diagnostic strategies, and treatment modalities of mucormycosis, emphasizing the need for multidisciplinary management to mitigate its severe consequences.

Keywords: mucormycosis, COVID19, risk factors, pathogenesis, diagnosis, management.

Introduction

Mucormycosis, also known as black fungus, is a rare but aggressive fungal infection caused by molds from the Mucorales order. It primarily affects immunocompromised individuals, such as those with uncontrolled diabetes, cancer, organ transplants, or prolonged steroid use. The infection typically invades the sinuses, lungs, and brain but can also affect other organs [1]. Understanding the complex nature of this disease is essential for accurate diagnosis and effective management. Therefore, this review

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provides the information on the epidemiology, risk factors, pathogenesis, diagnosis and management of the mucormycosis.

Epidemiology of Mucormycosis:

Mucormycosis is a globally distributed fungal infection, though its prevalence is higher in regions like India and Southeast Asia due to the large number of patients with uncontrolled diabetes. The incidence of the disease has surged in recent years, particularly during the COVID-19 pandemic, as many immunocompromised patients, especially those with diabetes or treated with steroids, became more susceptible. India has the highest recorded incidence of mucormycosis worldwide, approximately 70 times more than any other country[2]. As per Centers for Disease control and prevention 2022, fungal infections associated with COVID-19 caused serious disease or even death with mortality rates ranging from 40% to 80%, depending on severity and course of treatment. The overall hospital-mortality rate remains higher (48.5%) during the COVID-19 time as compared to non-COVID19 time, depending on the severity of the infection and timely treatment[3,4]. As per the study conducted in June 2021, 55 patients (52.7%) with COVID-19 associated mucormycosis developed the symptoms of infection within 15 to 30 days of developing COVID-19 symptoms [5] Patients with widespread mucormycosis had the highest case fatality rate (68%) while patients with cutaneous disease had the lowest case fatality rate (31%)[2]. Before the COVID-19 pandemic, a computer model estimated an annual average of 171,504 cases (95% CI: 147,688–195,777) and 65,500 deaths in India, resulting in a mortality rate of 38.2% [6]

Risk Factors of Mucormycosis:

Mucormycosis primarily affects individuals with weakened immune systems or underlying health conditions. Several risk factors predispose people to mucormycosis, making them more vulnerable to this opportunistic infection.

a) Diabetes Mellitus:

One of the most significant risk factors for mucormycosis is uncontrolled diabetes mellitus, particularly individuals developing diabetic ketoacidosis. High blood sugar levels create an environment conducive to fungal growth. Additionally, in diabetic ketoacidosis, increased iron availability, due to low pH levels, further promotes the growth of Mucorales fungi. These conditions compromise the body's ability to fight off infections, allowing the fungus to invade tissues[7].

b) Immunosuppression:

Patients with compromised immune systems are at high risk for mucormycosis. This group includes individuals undergoing chemotherapy for cancer, organ transplant recipients on immunosuppressive therapy, and those with haematological malignancies. The body's reduced capacity to fight infections allows Mucorales fungi to thrive and spread quickly[8-12]

c) Corticosteroid Use:

The use of corticosteroids, commonly prescribed to manage conditions like autoimmune diseases, COVID-19, and chronic inflammatory conditions, can increase the risk of mucormycosis. Steroids suppress the immune response and elevate blood sugar levels, creating an ideal environment for fungal infections. [8-12]

d) Prolonged Neutropenia:

Neutropenia, a condition characterized by an abnormally low number of neutrophils (a type of white blood cell), is another important risk factor. Patients undergoing treatments such as chemotherapy or bone marrow transplants are prone to neutropenia, which weakens their immune system and increases their vulnerability to fungal infections, including mucormycosis[8-13].

e) Trauma and Burns:

Invasive mucormycosis can develop in individuals who have suffered severe trauma, such as from burns, surgery, or accidents. The fungi can enter the body through broken skin or wounds, leading to localized or disseminated infection[8-11]

f) Iron Overload and Chelation Therapy:

Excessive iron in the body, whether due to underlying medical conditions or iron

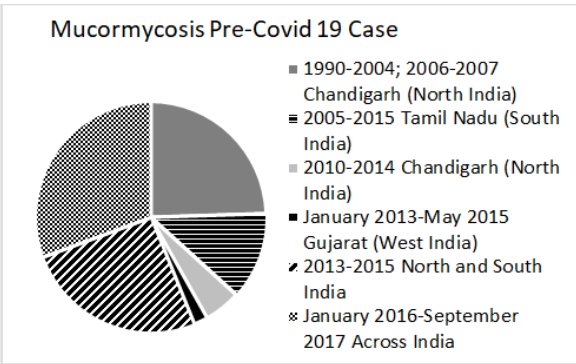
supplementation, also heightens the risk of mucormycosis. The fungi thrive in iron-rich environments, and patients undergoing iron chelation therapy with deferoxamine are particularly susceptible because the drug acts as a siderophore, effectively feeding the fungi[8-12].

g) Malnutrition and Poor Hygiene:

Malnourished individuals and those living in unsanitary conditions are at greater risk for mucormycosis. Poor nutrition weakens the immune system, while unhygienic environments increase the likelihood of exposure to fungal spores[8-12].

h) COVID-19:

COVID-19 has emerged as a significant risk factor for mucormycosis, especially in patients with severe illness or pre-existing conditions like diabetes. The use of corticosteroids in managing severe COVID-19 cases can suppress the immune system and raise blood sugar levels, creating an environment favorable for fungal infections. Additionally, prolonged hospitalization, oxygen therapy, and the overall immune dysregulation caused by COVID-19 can further increase susceptibility. In a study, Kumar et al. 2021 described the ratio of infected mucormycosis (Fig. 1).Other contributing factors like renal disease, older age, secondary infections,poor hygiene, weather, crowding, poverty, as well as co-infection with other diseases, infected ICU interventions, administrations of immunosuppressants, impaired phagocytosis, viral mutations and its new variants, damage to nasal ciliated cells and lung destruction also increase the chance of mucormycosis infection in COVID-19 patients [14,15].



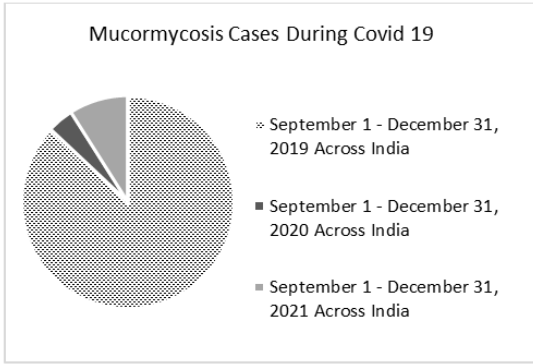


Figure-1: Summary of mucormycosis cases pre- and during COVID19.

Post Covid Scenario:

Mucormycosis has also emerged as a post covid sequalae due to factors like immune dysregulation, steroid use, elevated ferritin levels, and zinc supplementation, particularly in patients with diabetes. Immune dysregulation including a rise in systemic inflammatory markers (including CRP, IL- 6) along with decreased adaptive immune responses is considered a key factor for causing mucormycosis as a secondary infection in recovered COVID-19 patients[16]. Diabetes Mellitus is the most common predisposing condition for post-COVID-19 mucormycosis as observed in multiple case studies and retrospective analysis[17]. In a case series presented by Divit et al, in June 2021, Diabetes Mellitus along with other risk factors predispose patients to increased inflammation leading to long COVID phase defined as increased inflammation for more than 3 months after COVID-19. While it is emphasized that immunosuppression and glucocorticoid use in such patients promote growth of fungus leading to mucormycosis, cases have also reported in young population in absence of such risk factors[18]. Therefore, vigilant monitoring and regular follow-ups are essential for high-risk patients after COVID-19 recovery[19,20]. Patients must be advised to report any symptoms related to the eyes, nose, face, lungs, and skin and immediate medical attention should be received to avoid further complications[17].

Pathogenesis:

The pathogenesis of mucormycosis involves several stages, beginning with the inhalation or inoculation of fungal spores, followed by rapid invasion of host tissues, blood vessel involvement, and extensive tissue necrosis.

Fungal Entry:

The most common route of mucormycosis infection is through the inhalation of fungal spores, which are ubiquitous in the environment and found in soil, decaying organic matter, and air. The respiratory tract, particularly the nasal passages and sinuses, is the primary entry point. The spores can also enter through cutaneous wounds, trauma, or through ingestion, leading to gastrointestinal mucormycosis, although this is less common. To avoid phagocytosis by macrophages, Rhizopus species can transform into hyphae and bind to endothelial cells through specific receptors. One key receptor on the fungal surface is CotH (spore-coating protein family), which interacts with GRP78 (glucose-regulated protein 78) on the endothelium. This binding triggers endocytosis, allowing the fungus to enter the bloodstream, spread via hematogenous routes, cause systemic infection, and invade multiple organs [21].

Host Susceptibility:

Mucormycosis spores when inhaled by healthy individuals, they are usually eliminated without developing an infection. However, these spores survive and proliferate due to weakened immune defences of body in case of immunocompromised individuals.

Clinical and laboratory evidence strongly indicate that individuals with either a deficiency or dysfunction of phagocytic cells are more susceptible to mucormycosis. Notably, patients experiencing severe neutropenia exhibit a heightened risk of developing this fungal infection. In contrast, individuals with AIDS do not appear to share this increased vulnerability, suggesting that neutrophils rather than T lymphocytes play a central role in controlling the growth of fungal spores [22]. In healthy individuals, both mononuclear and polymorphonuclear phagocytes combat Mucorales by producing reactive oxygen species and antimicrobial peptides such as defensins.. However, under conditions commonly seen in diabetic ketoacidosis, such as high blood glucose levels and acidic pH, phagocyte function is significantly impaired. This leads to defective chemotaxis and a reduction in both oxidative and non-oxidative killing capabilities. [23].

Fungal Germination and Hyphal Growth:

Once inside the body, the spores germinate into hyphae, the filamentous structure that penetrate host tissues causing invasion and disrupting normal cellular function. Hyphal growth is the key factor in the pathogenesis of mucormycosis. The fungi prefer an iron-rich environment, which is commonly seen in individuals with diabetic ketoacidosis due to increased serum-free iron. In *Mucor* species, anaerobic conditions and presence of fermentable sugars promote yeast-like growth, whereas oxygen limitation and nutrient scarcity favor hyphal development. The hyphae proliferate, spreading through tissues and infiltrating blood vessels[24,25].

Angioinvasion:

A hallmark of mucormycosis is its tendency for angioinvasion—hyphae penetrate and invade blood vessels. This vascular invasion is a critical aspect of the disease's pathogenesis, as it leads to thrombosis and ischemia. The fungi spread rapidly via blood vessels, causing extensive damage to local tissues. This process is particularly dangerous because it limits the delivery of antifungal drugs and immune cells to the infected area, exacerbating the infection[26].

Tissue Necrosis:

As the hyphae invade blood vessels, the resultant ischemia causes widespread necrosis of the surrounding tissues. This necrotic tissue becomes a fertile ground for fungal growth, allowing the infection to spread further. In rhinocerebral mucormycosis, for example, the infection starts in the nasal passages and sinuses, leading to facial swelling, black necrotic lesions, and potential invasion into the brain. The necrosis is visible as black, dead tissue, which is one of the distinctive signs of mucormycosis, often referred to as "black fungus"[27].

Dissemination:

In severe cases, mucormycosis can become disseminated, spreading to distant organs such as the lungs, brain, and gastrointestinal tract. Pulmonary mucormycosis occurs when the spores reach the lungs, causing pneumonia-like symptoms and often leading to respiratory failure if untreated. When the infection spreads to the brain, it can cause life-threatening complications such as brain abscesses, seizures, or

even coma. Blood vessel invasion by fungal hyphae results in endothelial destruction, blood clots that block the blood arteries, and eventually ischemia and necrosis of surrounding tissues [28,29].

Immune Response:

In mucormycosis, the immune system's ability to respond is critical. Mucorales weaken the immune system by changing the structure of host cells to create huge cell aggregates or clusters or lengthy hyphae that are resistant to leukocyte phagocytosis because they burst from macrophages. Neutrophils play a central role in controlling fungal infections by phagocytosing the spores and hyphae. However, in immunocompromised individuals or those with neutropenia, the neutrophils are either dysfunctional or present in inadequate numbers, allowing the infection to spread uncontrollably [30].

Host Damage:

The combination of direct tissue invasion by fungal hyphae, angioinvasion, and immune system impairment leads to significant host tissue damage. Rhizopus oryzae exists as sporangiophores, which transform into coenocytic hyphae within the host cell. A small number of transformation fungus produce networked hyphae (mycelium) that facilitate the passing on of nutrients and consequently encourage growth [31,32]. The aggressive nature of mucormycosis can result in rapid deterioration of the patient's condition, requiring urgent medical intervention including antifungal therapy and surgical debridement of necrotic tissue.

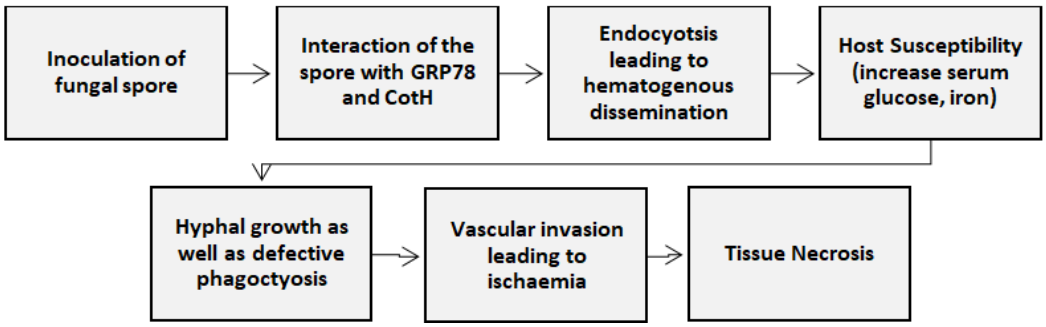


Figure-2: Pathogenesis of mucormycosis.

Types of Mucormycosis:

a) Pulmonary Mucormycosis:

An unusual fungal infection called pulmonary mucormycosis which most commonly observed in persons with impaired immune systems. It generally occurs by inhalation of spores of fungi. Non-productive cough and persistent fever is a common symptom however clinical diagnosis is difficult[33].

b) Rhinocerebral Mucormycos (RCM):

Severe cerebral ischemia and hemorrhagic lesions are possible outcomes of RCM [34]. As per WHO symptoms includelethargy, seizures, slurred speech, partial paralysis, fever, black sores that rapidly worsen on the upper inside of the mouth or on the nasal bridge, headache, nasal or sinus congestion, one-sided facial swelling. Recurrence can occur even after recovery[35].

c) Cutaneous Mucormycosis:

A novel fungal infection caused by an opportunistic fungi belonging to the phylum Glomeromycota, commonly occuring in immunocompromised individuals and those with poorly managed diabetes. Infection typically occurs by direct inoculation through wound[36]. According to the CDC, blisters or ulcers are common signs of cutaneous mucormycosis and the affected area may turn black. Pain, warmth, redness, or swelling around a wound are other signs.

d) Gastrointestinal Mucormycosis:

The gastrointestinal mucormycosis is an uncommon type, most frequently affecting the stomach, then the colon and ileum. Depending on the affected region, it may present with a variety of symptoms, most typical being nonspecific abdominal pain and distention linked to nausea and vomiting. These may be accompanied by fever and hematochezia[37].

e) Disseminated Mucormycosis:

An unusual kind that is frequently observed in people with extremely weakened immune systems is disseminated mucormycosis. The infection grows widespread as it moves to different parts of the body. The skin, heart, spleen, brain, and other organs are among the numerous regions that may be affected. (National Organization for Rare Disorders)[38,39].

Diagnosis of Mucormycosis:

Initial diagnosis of mucormycosis is mainly based on physical examination, symptoms and medical history. However it requires a collaboration of clinical, histopathological, microbiological and radiological approaches for accuracy[40].

Microbiological: A microbiological examination is vital for early diagnosis of mucormycosis with identification of the causative pathogen. For this nasal discharge, excised tissue by endoscopy or during surgery are used[40].

Potassium hydroxide (KOH) wet mount: In KOH wet mount, the Mucorales hyphae reveal coenocytic broad aseptate/sparsely septate hyphae with right-angle branching resembling ribbon-like appearance[40].

Calcofluor white (CFW) stain: is a non-specific fluorochrome dye. Once the sample is stimulated with UV light in a fluorescent microscope, the fungal pathogen appears as the apple green or bluish against the white background, depending on the filter used[40].

Histopathology: On microscopic examination, tissues from the suspected case of mucormycosis show necrosis, inflammatory infiltrate rich in neutrophils and fungal hyphae. The fungal hyphae appear basophilic one, broad, aseptate and show right-angle branching in H and E staining[40].

Molecular Diagnosis:

Different molecular methods like semi-nested PCR, nested PCR with RFLP, real Time PCR targeting the ITS region or specific primers targeting a restricted number of mucoralean genera/species, are being used to diagnose mucormycosis. These molecular methods aid the diagnosis where the fungal load is low and the conditions where other diagnostic tools fail[40].

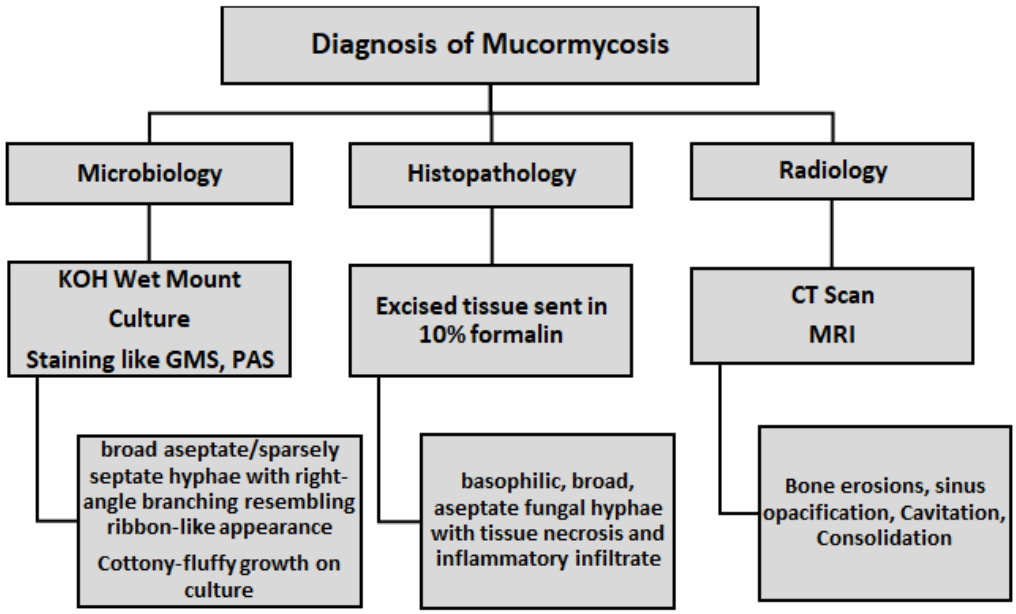


Figure-3: Flow chart for diagnosis of mucormycosis.

Culture:

Mucorales grow quickly (3-5 days) on common fungal culture media (such as potato dextrose agar and Sabouraud agar) when incubated at 25 to 30 °C [41]. It is significant to remember that only 50% of cases are culture positive, even in positive microscopy instances. It is now possible to reliably identify

Mucorales utilising matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALDI-TOF MS) methods using either an in-house database or a commercial filamentous library [42].

Serology:

ELISA is frequently utilised in serology-based methods of detection for recognising antibodies produced in reaction to Mucorales infection. DNA barcoding has been demonstrated to be a highly accurate and efficient method to differentiate between various kinds of fungus[43].

Radiology:

Radiological imaging plays a crucial role in the early diagnosis and assessment of mucormycosis, especially in rhinocerebral and pulmonary forms. Computed tomography (CT) is commonly used to detect bone erosion, sinus opacification, and soft tissue involvement, while magnetic resonance imaging (MRI) offers superior visualization of vascular invasion, intracranial extension, and orbital spread. In pulmonary mucormycosis, CT may reveal nodules, cavitations, consolidation, or the reverse halo sign, which is suggestive of fungal infection. Early imaging is vital for guiding surgical intervention and monitoring treatment response. Radiology, therefore, serves as an essential tool for prompt diagnosis and management of this aggressive infection[44].

Management:

Since mucormycosis is a hazardous infection, it requires prompt treatment with prescription of correct antifungal drugs. Early identification, inversion of risk factors and actual illness, surgical debridement, and rapid intravenous antifungal therapy (typically amphotericin B) are all part of the conventional management for mucormycosis. This means that hyperglycemia and acidosis must be treated immediately, and immunosuppressive medications must be discontinued whenever possible[45]. According to guidelines published by European Conference on Infections in Leukemia (ECIL) in 2017 on mucormycosis treatment and update provided by European Confederation of Medical Mycology (ECMM), liposomal Amphotericin B (L-AmB) is recommended for first-line treatment in adults[46]. The more recent triazoles, isavuconazole (ISAV), which is the active component in the prodrug isavuconazonium sulphate, and posaconazole (POSA) may work effectively for individuals who don't respond well to or are sensitive of lipid-based amphotericin B formulations (LFAB). An essential supplementary function is played by early surgical excision or debridement[47,48]. Other medicines also included as per world health or Centers for Disease Control and Prevention organization are fluconazole, voriconazole, and echinocandins. Patients with diabetes and mucormycosis participated in one retrospective study; those who received the combination medication showed better outcomes than those who received polyene monotherapy[49]. Mucormycosis-related necrosis and thrombosis can impair the absorption of antifungal medications. As a result, the removal of impacted tissue could be a vital treatment to fully get rid of the infection. It has been observed that surgical care produces better results than non-surgical treatment in individuals with rhino-orbito-cerebral mucormycosis and results in local control of the infection[50].

Conclusion

In conclusion, mucormycosis is a rapidly progressing and life-threatening fungal infection, especially in immunocompromised and diabetic individuals. Its pathogenesis is driven by angioinvasion and tissue necrosis, leading to poor outcomes if not treated early. Effective management requires prompt antifungal therapy, surgical debridement, and control of underlying risk factors. Despite therapeutic advances, early diagnosis and intervention remain critical to reducing morbidity and mortality.

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