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Mucormycosis (Black Fungus): Interplay between Immunosuppression, Diabetes Mellitus, and Genetic Susceptibility: A review

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ABSTRACT

Objective: *Moulds called mucormycetes are the source of the uncommon but dangerous fungal infection known as mucormycosis, or zygomycosis.* ***Method:*** *Immunocompromised people, such as those with untreated diabetes, haematologic malignancies, and immunological dysregulation brought on by COVID-19, are the main victims.* ***Results:*** *Rapid progression, angioinvasion, tissue necrosis, and a high death rate if left untreated are the disease's hallmarks.* ***Novelty:*** *This study highlights the emerging association of mucormycosis with COVID-19-induced immunological dysregulation, emphasizing its significance in recent patient populations and the urgent need for clinical awareness.*

INTRODUCTION

Mucormycosis, often known as black fungus, is a potentially lethal opportunistic infection caused by a group of moulds called mucormycetes, which belong to the class Zygomycetes and the order Mucorales [1]. These fungi are widely distributed in the environment and are usually found in soil, decomposing vegetation, seeds, grains, and air. Under normal conditions, they pose little threat to healthy individuals, but they can cause serious infections in hosts with impaired immune systems [2]. The disease has a reputation for being angioinvasive; if the infection is not detected and treated quickly, it can become fatal and often results in tissue necrosis due to vascular invasion and subsequent thrombosis [3].

A dramatic increase in mucormycosis instances among COVID-19 patients in nations like India and Iran in recent years has drawn attention from all around the world, especially among those with poorly managed diabetes mellitus and those undergoing corticosteroid therapy [4]. Fungal spores are believed to be inhaled into the paranasal sinuses, causing rhino orbito cerebral mucormycosis, the most prevalent form of mucormycosis. Mucormycosis associated with COVID-19 primarily manifests as a rhino orbital cerebral infection, which affects the nose, eyes, and even the brain. [5]. Clinical infections can take many different forms, including pulmonary, gastrointestinal, cutaneous, encephalic, and rhinocerebral [6].

This study aims to explore the underlying immunological and genetic mechanisms that predispose certain individuals to mucormycosis, with an emphasis on recent findings linking immune suppression, hyperglycemia, and host genetic susceptibility to increased fungal pathogenicity.

RESEARCH METHOD

This article was prepared through a literature review by collecting and analyzing studies related to mucormycosis, including its pathogenesis, risk factors, clinical features, diagnosis, and management. Relevant publications in English from 2000 to 2025 were identified through databases such as PubMed, Scopus, and Google Scholar using keywords including *mucormycosis*, *zygomycosis*, *fungal infection*, and *COVID-19-associated mucormycosis*. Only peer-reviewed articles focusing on human cases were considered to ensure the accuracy and reliability of the discussion.

RESULTS AND DISCUSSION

Causative Organisms

Mucormycetes, the mould species that cause mucormycosis, are present in the atmosphere and emit spores that are easily aerosolised and dispersed. The most frequently isolated species from patients are Apophysomyces (*A. variabilis*), Cunninghamella (*C. bertholletiae*), Lichtheimia [Absidia] (L. corymbifera L. raosa), and Mucor (*M. circinelloides*). Saksenaea (*S. vasiformis*), Rhizopus, and Rhizomucor (*R. pusillus*) – The most common genus, especially *Rhizopus oryzae* (also known as *Rhizopus arrhizus*), which is responsible for the majority of human mucormycosis cases[7]. These fungi are thermo tolerant, growing rapidly at human body temperature (37°C) and producing broad, ribbon-like, non-septate or sparsely septate hyphae[8].

Pathogenesis (The Angio-Invader)

Angioinvasion: Mucormycetes' capacity to enter blood arteries and cause thrombosis, tissue ischaemia, and necrosis is a defining feature of mucormycosis. An essential stage in the pathophysiology is damage and penetration through the extracellular matrix proteins or endothelial cells that line blood vessels [9]. A possible explanation for this angioinvasion is that the fungal surface's By interacting with the glucose regulator protein 78 (GRP78) receptor on the surface of endothelial cells, the spore-coating protein family, or CotH, damages endothelial cells and causes haemorrhage [10]. Additionally, army leukocytes and antifungal medications cannot reach the target due to the ischaemic necrosis in the infected tissues.

Clinical Manifestations

The site of fungal invasion, the host's immune system, the infection pathway, and predisposing factors all affect the clinical symptoms.

1. Rhino-Orbital-Cerebral Mucormycosis (ROCM)

The most prevalent and severe type, especially in diabetic individuals, starts with fever, headache, facial pain, and congestion of the nose [11]. The progression causes periorbital oedema, impaired vision, ophthalmoplegia, facial numbness, and black necrotic eschar in the nasal or oral mucosa. Hemiparesis, convulsions, or altered mental status can be caused by cerebral involvement. Via vascular invasion, the infection frequently moves from the paranasal sinuses to the orbit and brain [12].

2. Pulmonary Mucormycosis

Frequently seen in individuals who have post-transplant, neutropenia, or haematologic malignancies. In immunocompromised patients, the pulmonary alveolar macrophages are ineffective at combating the fungus. Fever, haemoptysis, dyspnoea, and pleural pain are indicators of pulmonary involvement that are difficult to differentiate from TB or aspergillosis symptoms. Endobronchial and tracheal invasion, along with the involvement of major arteries, can cause fatal haemoptysis [13].

3. Cutaneous Mucormycosis

The main risk factors for the cutaneous form are burn contamination, surgery, and penetrating trauma. appears as skin lesions that are violaceous, erythematous, or painful. rapid development of black necrotic ulcers surrounded by oedema and erythema. may spread to deeper tissues and the circulation or remain localised [14].

4. Gastrointestinal Mucormycosis

More prevalent in immunocompromised patients, malnourished people, and newborns. The stomach, small intestine, and large intestine can all be affected by intestinal mucormycosis. symptoms that are not specific, like diarrhoea, vomiting, gastrointestinal bleeding, or stomach pain. has a bad prognosis and is frequently diagnosed late [15].

5. Disseminated Mucormycosis

Occurs when the fungus spreads hematogenously from the initial site of infection, involve: the brain, heart, spleen, kidneys, or skin. Presents with multi-organ failure, sepsis, or altered consciousness. High fatality rate due to diagnostic delay and difficulty in controlling systemic spread. Due to its angioinvasive nature, mucormycosis often leads to vascular thrombosis, tissue infarction, and necrosis, which are hallmark features in all clinical forms. Early recognition of these manifestations, particularly in high-risk populations, is essential for improving outcomes [16].

Risk Factors

The majority of people with mucormycosis have weakened immune systems. Susceptibility to this severe fungal infection can be considerably increased by a number of underlying diseases and extrinsic factors. The most commonly recognized risk factors include:

1. Uncontrolled Diabetes Mellitus

Diabetes, especially when poorly controlled or associated with diabetic ketoacidosis (DKA), is the most prevalent risk factor for mucormycosis. Hyperglycemia impairs neutrophil function, decreases chemotaxis and phagocytosis, and provides a favorable environment for fungal proliferation due to increased free iron availability during acidosis [17].

2. Corticosteroid Use

Corticosteroids, widely used in managing autoimmune diseases and severe COVID-19 cases, suppress immune function and elevate blood glucose levels. Their prolonged use can predispose patients to opportunistic infections such as mucormycosis by impairing macrophage and neutrophil responses [18].

3. Hematologic Malignancies and Organ Transplantation

Patients with leukemia, lymphoma, or those undergoing hematopoietic stem cell or solid organ transplantation are at high risk due to prolonged neutropenia and immunosuppressive therapy. These conditions significantly reduce the body's ability to mount an effective immune response against fungal invasion [19].

4. COVID-19 Infection

An increase in mucormycosis cases has been connected to the COVID-19 pandemic, especially in nations where diabetes is highly prevalent. This is caused by a number of factors, including as immunological dysregulation brought on by viruses, extensive use of corticosteroids, mechanical ventilation, and high-dose oxygen therapy administered in non-sterile environments [20].

5. Iron Overload and Deferoxamine Therapy

For Mucorales to flourish, iron is necessary. Individuals with iron overload are more vulnerable, especially those taking the iron-chelating medication deferoxamine. As a siderophore for Mucorales, deferoxamine helps the fungus absorb iron [21].

6. Traumatic Injuries and Burns

Cutaneous mucormycosis can occur in individuals with open wounds, burns, or post-surgical infections, especially in healthcare settings with poor hygiene or contamination[22].

7. Immunodeficiency States

Patients with AIDS, chronic immunosuppressive therapy (e.g., for autoimmune diseases), or inherited immunodeficiencies have a weakened defense system, making them vulnerable to mucormycosis and other invasive fungal infections [23].

Immunological Mechanism

The development of mucormycosis is strongly influenced by the status of the host's immune system. In healthy individuals, innate immune defenses are usually sufficient to eliminate Mucorales spores. However, in immunocompromised individuals, these defenses are impaired, allowing the fungus to invade and proliferate. Macrophages and neutrophils play a central role in clearing Mucorales spores, Macrophages phagocytose and inhibit spore germination. Neutrophils produce reactive oxygen species (ROS) and antifungal peptides to destroy hyphae. In individuals with neutropenia or impaired neutrophil function (e.g., due to chemotherapy, corticosteroids, or diabetes), fungal spores can evade destruction, germinate, and invade tissues [24]. Pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1β are important for activating antifungal responses. However, dysregulated inflammation (as seen in COVID-19 or in corticosteroid therapy) can impair effective immune clearance or suppress critical signaling pathways [25]. The immune system recognizes fungal components via PRRs such as: These Dectin-1and Toll-like receptors (TLR2 and TLR4) receptors detect fungal cell wall components and activate signaling pathways (e.g., NF-κB) to promote inflammation and phagocyte recruitment. Genetic polymorphisms in these receptors may impair recognition of the fungus, leading to increased susceptibility [26]. Also corticosteroids, used commonly in treating autoimmune diseases or COVID-19, impair

phagocyte function and cytokine signaling. And Lymphopenia, particularly T-cell depletion, also weakens mucosal immunity and facilitates fungal dissemination [27].

Genetic Susceptibility

While environmental exposure and immunosuppression are well-known risk factors for mucormycosis, not all exposed or immunocompromised individuals develop the disease. This suggests that host genetic factors may influence individual susceptibility to mucormycosis. Recent studies have begun to identify genetic polymorphisms and inherited immune deficiencies that contribute to increased susceptibility. Genetic variations in pattern recognition receptors (PRRs), which detect fungal pathogens, may impair immune recognition and response to Mucorales fungi. Dectin-1 (CLEC7A gene), A C-type lectin receptor involved in detecting fungal β-glucans. Although Mucorales have minimal β-glucan, Dectin-1 contributes to broader antifungal immunity. Mutations in the CLEC7A gene can reduce phagocyte activation and fungal clearance [28]. Toll-Like Receptors (TLR2 and TLR4), These receptors recognize fungal components and activate innate immunity. A higher incidence of invasive fungal infections, such as mucormycosis, has been linked to polymorphisms in TLR2 and TLR4 [29]. Mutations in the HFE gene and genes related to iron transport and storage may contribute to the risk of mucormycosis. (e.g., C282Y and H63D) are associated with hereditary hemochromatosis and increased iron availability, which enhances Mucorales growth [30].Altered expression of ferritin and transferrin may also influence fungal virulence in the host[31].Pentraxin-3 (PTX3) is a soluble pattern recognition molecule involved in opsonization and immune regulation during fungal infections,PTX3 deficiency has been linked to increased susceptibility to invasive pulmonary aspergillosis and is under investigation for its role in mucormycosis [32].

Diagnosis

Given its high mortality rate and quick progression, mucormycosis requires an early and precise diagnosis. A high index of clinical suspicion precedes the diagnosis, particularly in high-risk people like : Diabetic patients with sinusitis or orbital symptoms, immunocompromised individuals presenting with necrotic lesions or pulmonary symptoms,COVID-19 patients showing facial swelling, black eschar, or vision loss, nasal congestion, facial pain or swelling, black discoloration of nasal or oral mucosa, fever, headache, and orbital swelling, neurological symptoms if the infection spreads to the brain. A cheap yet useful method for quickly identifying the fungi is direct microscopy on Giemsa stain or KOH mount, which is used in laboratory diagnosis of mucormycosis [8]. Particularly sensitive specimens are nose swabs taken during diagnostic nasal endoscopy. The hyphae of Mucorales are usually nonpigmentedbroad (6–25 μm), flat, nonseptate, ribbon-like, and thin-walled, and irregularly branch at right angles [33]. Fungal culture on Sabouraud dextrose agar can help identify the specific fungal species, though cultures may take time and sometimes yield negative results even in confirmed cases. Tissue biopsy and staining (e.g., The definitive methods for diagnosis are Gomori methenamine silver, Haematoxylin and Eosin, or Periodic acid–Schiff [34]. Pathognomonic features include: Right-angle branching, broad, non-septate hyphae, and

signs of tissue necrosis and angioinvasion. Fungal DNA can be found in tissue or blood samples using molecular diagnostic methods such PCR tests, albeit these methods are not commonly accessible [35]. Aspergillosis and other fungal infections can be diagnosed more accurately with serologic markers, such as galactomannan and β-D-glucan, than with mucormycosis [36].

Treatment and Management

Mucormycosis management necessitates a multimodal strategy that includes vigorous surgical intervention, timely antifungal therapy, and addressing underlying risk factors. Inadequate or delayed treatment is linked to a markedly higher death rate. The first line of treatment for antifungal medication is liposomal amphotericin B ; it is recommended since it is efficacious and has less nephrotoxicity than traditional formulations. Following an initial improvement, isavuconazole is used as a step-down oral therapy or as a second-line medication [37]. Mucorales fungi are resistant to many antifungals used for other fungal infections, including fluconazole and voriconazole. Surgical removal of infected and necrotic tissues is often essential, especially in cases of rhino-orbital or cutaneous mucormycosis [38]. Glycemic control: Tight control of blood glucose in diabetic patients is crucial. Reduction of immunosuppressive therapy, including corticosteroids, if possible. Treatment of ketoacidosis, neutropenia, or iron overload should be initiated promptly [39]. Hyperbaric oxygen therapy: May enhance wound healing and neutrophil function. Iron chelators like deferasirox (not deferoxamine) are under investigation as potential adjunctive agents to inhibit fungal growth [40]. Patients require close monitoring through: Repeat imaging to assess response to therapy, renal function tests due to nephrotoxicity risk with Amphotericin B, long-term follow-up, especially in immunocompromised individuals, to detect recurrence.

CONCLUSION

Fundamental Finding : Mucormycosis, though rare, represents a life-threatening fungal infection that demands urgent clinical attention, especially in immunocompromised individuals and patients with uncontrolled diabetes mellitus. The recent surge in cases during The importance of immunological dysfunction and metabolic abnormalities in disease vulnerability has been brought to light by the COVID-19 pandemic. **Implication :** For prompt diagnosis and successful therapy, it is crucial to comprehend how immunosuppression, hyperglycemia, and genetic predisposition interact. **Limitation :** The current understanding is limited because although the disease has been recognized as severe, there remains a lack of clarity on the precise mechanisms linking immunological dysfunction, metabolic abnormalities, and the sudden surge observed during the COVID-19 pandemic. **Future Research :** To reduce the morbidity and mortality rate associated with this serious infection, future studies should concentrate on early detection techniques, tailored antifungal treatments, and prophylactic measures.

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