

A REVIEW ARTICLE ON BLACK FUNGUS**Devyani S. Kutemate***

India.

Article Received on 15 Feb. 2026,
Article Revised on 05 March 2026,
Article Published on 16 March 2026,

<https://doi.org/10.5281/zenodo.19043962>

Corresponding Author*Devyani S. Kutemate**

India.



How to cite this Article: Devyani S. Kutemate*. (2026). A Review Article on Black Fungus. World Journal of Pharmaceutical Research, 15(6), 270–285.

This work is licensed under Creative Commons Attribution 4.0 International license.

ABSTRACT

As a rare "opportunistic" fungal disease, the black fungus infection has had a significant impact on post-COVID-19 recoveries and has placed an additional strain on our medical and healthcare management systems. Following the initial phase of COVID-19, the second wave affected many Indians with a mysterious fungal infection known as Mucormycosis. In this review, we examined the clinical pathogenesis, signs, symptoms, and treatment options for black fungus. The findings indicated that the use of immunosuppressants to treat COVID-19 also heightens the risk of infection with mucormycosis. Patients suffering from hyperglycemia, ketoacidosis, solid organ or bone marrow transplants, liver cirrhosis, and neutropenia are particularly vulnerable to

Mucormycosis molds. Early diagnosis, elimination of predisposing factors, prompt antifungal treatment combined with surgical removal of all infected tissues, and adjunctive therapies are four critical components in the fight against Mucormycosis. Consequently, millions of lives have already been lost. Due to mutations, the virus is continuously altering its characteristics, including the transmission rate, virulence, pathogenesis, and clinical manifestations. A recent study found that some COVID-19 patients were also co-infected with a fungal disease known as mucormycosis (black fungus). India has officially classified the black fungus outbreak among COVID-19 patients as an epidemic, with only a few cases reported in other nations. The immune system is compromised by COVID-19 treatments, making it more susceptible to infections such as black fungus (mucormycosis). COVID-19, caused by the B.1.617 strain of the SARS-CoV-2 virus, has been prevalent in India since April 2021. Mucormycosis is a rare fungal infection triggered by exposure to a fungus known as mucormycete.

KEYWORDS: Mucorales; Mucormycosis; Diabetes Mellitus; Black Fungus; Pathogenesis; Diagnosis; Pathophysiology; Treatment.

INTRODUCTION

Mucormycosis is a fungal infection that arises from Mucorales. Among the three genera, namely *Rhizopus*, *Rhizomucor*, and *Mucor*, they account for 75% of mucormycosis cases (Kontoyiannis et al., 2010). This infection manifests in the upper airways as granulomatous invasion and may progressively extend into the sinuses and/or brain tissue. As a rare "opportunistic" fungal disease, the black fungus infection has significantly affected post-COVID-19 recovery efforts and has placed an additional strain on our medical and healthcare management systems. Coupled with the uncertain treatment options available at the onset of the pandemic, the indiscriminate use of various medications, including steroids and antibiotics, has contributed to the rising incidence of complications associated with black fungus. However, the ongoing low oxygen levels in the blood, combined with elevated iron levels and the extended hospitalization of COVID-19 patients reliant on mechanical ventilation, are primary factors leading to the contraction of the black fungus infection. Black fungus refers to a group of molds commonly identified as mucormycosis, while the resulting infection is known as mucormycosis. Reports indicate that mucormycosis is more frequently observed in COVID-19 patients with pre-existing medical conditions such as hyperglycemia, particularly those receiving steroid treatments. Mucormycosis typically spreads via the respiratory tract, primarily damaging facial structures, resulting in discoloration or blackening of the nose, and may lead to blurred or double vision. Additionally, infected individuals often experience chest pain, difficulty breathing, and hemoptysis (coughing up blood). Although it is not contagious, the prognosis of the disease can be dire, as it may lead to gastrointestinal bleeding and severe respiratory distress. Furthermore, if the infection spreads systemically, there is a significantly increased risk of impacting vital organs such as the spleen and heart. Given the significant population density and the rapid spread of mucormycosis, we have endeavored to present an epidemiological overview of black fungus infections in India in the initial section of the review. In the subsequent section, we have concentrated on compiling a thorough fact check regarding the current circumstances from an immunological standpoint.^[1]

Mucormycosis and COVID-19

Mucormycosis is a fungal infection that impacts the sinuses and lungs following the

inhalation of fungal spores present in the air. Historically, this infection has been infrequent, primarily affecting individuals with poorly managed diabetes. However, numerous hospitals throughout India have recently reported an increase in cases of COVID-19-associated mucormycosis (CAM). Due to the COVID-19 virus's ability to infiltrate the pancreas and elevate blood sugar levels, which promotes fungal proliferation, CAM has also been observed in individuals who were recently diagnosed with diabetes. The virus may penetrate and subsequently damage insulin-producing cells, as the pancreas, which is responsible for regulating blood sugar, contains a high concentration of angiotensin-converting enzyme 2 (ACE2). The ACE2 protein is an enzyme that plays a role in blood pressure regulation and is believed to serve as the virus's "entry point" into the host. The journal Nature recounts, among other cases, the story of an 18-year-old student who was infected by his parents but remained asymptomatic until a few days later when he experienced severe fatigue and an intense thirst. The diagnosis revealed that he had developed Type 1 diabetes.^[2]

Mucormycosis is a rare, invasive fungal opportunistic infection that poses a significant risk to life. It arises from exposure to mucor mold, which is present in soil, air, and even within human nasal passages and mucus. As the infection advances through the respiratory system, it damages facial structures. Patients may succumb to the illness within days of its onset, and in some instances, medical professionals have been compelled to surgically remove eyes and upper jaws to halt the potentially fatal spread of the infection. Treating mucormycosis requires a coordinated effort among eye surgeons, ENT specialists, general surgeons, and neurosurgeons. Symptoms of rhinocerebral mucormycosis (affecting the sinuses and brain) include unilateral facial swelling, lesions, nasal or sinus congestion, and fever.^[3]



Fig. 1: Mucormycosis.

Types of Mucormycosis

1. Rhinocerebral (sinus and brain) mucormycosis
2. Pulmonary (lung) mucormycosis
3. Cutaneous (skin) mucormycosis
4. Gastrointestinal mucormycosis
5. Disseminated mucormycosis.

- **Rhinocerebral (sinus and brain) mucormycosis**

Rhinocerebral mucormycosis, which is also referred to as zygomycosis, is an infection that impacts the nose and paranasal sinuses, and it has the potential to extend to the brain.^[4] This type of mucormycosis is prevalent among individuals with uncontrolled diabetes and those who have received a kidney transplant. Initially, the infection begins in the nasal cavity and subsequently spreads to the adjacent paranasal sinuses. Following this, implantation occurs in that region, leading to a more rapid growth rate within the sinuses and nasal cavity. The damp and humid conditions present in the nasal cavity and paranasal sinuses are conducive to the proliferation and invasion of fungi. Angioinvasion represents another pathway through which the infection can access the brain. In such instances, the progression is exceedingly swift and exhibits a distinct model of pathogenesis. Symptoms of rhino cerebral mucormycosis include the presence of black lesions on the upper interior of the buccal cavity, accompanied by mild to severe fever. More severe outcomes, such as brain infarction, hematoma, and orbital apex syndrome, are not uncommon.^[5] Given that vascular invasion is a significant feature of this type of infection, the development of intravascular thrombi frequently results in brain infarction and ischemia.^[6]

Pulmonary (lung) mucormycosis

Pulmonary mucormycosis primarily affects the lungs of individuals suffering from black fungus. This form of mucormycosis is the most prevalent among those who have undergone stem cell or organ transplants, including cancer patients.^[7] The condition may arise from the inhalation of spores or through lymphatic and hematogenous dissemination. Mucorales typically enter the body via the respiratory tract, where the fungi can easily invade veins, arteries, and lymphatics, leading to infarction and thrombosis, which can be life-threatening.^[8] Individuals with hematological malignancies, diabetes mellitus, or those who have received organ transplants and hematopoietic stem cell transplants are particularly susceptible to invasive mucormycosis. Additionally, patients undergoing corticosteroid-based

therapy, chelation therapy, and those who are neonatally premature may also be at risk for infection. Furthermore, in low-income countries such as India, malnutrition poses a significant challenge that can contribute to the development of mucormycosis infection.^[9]



Fig. 2: Pulmonary mucormycosis.

- **Gastrointestinal mucormycosis**

Gastrointestinal mucormycosis primarily impacts the stomach and intestines, with a notably high incidence among newborns, particularly those who are premature and under 1 month old.^[10] There has been a documented prevalence of gastrointestinal mucormycosis in developed countries. Nevertheless, in recent years, the global incidence of gastric and gastrointestinal mucormycosis has risen.^[11] Additionally, other uncommon instances of gastrointestinal mucormycosis have been observed in immunocompromised individuals, including those with AIDS, systemic lupus erythematosus, and those who have received organ transplants.^[12]

Some patients exhibiting hepatic mucormycosis indicate a potential link to the consumption of herbal medications.^[13] A study conducted by Morton and colleagues revealed a significant rise in this infection during the 21st century.^[14] Recently, an outbreak of gastric mucormycosis was reported, attributed to the contamination of wooden applicators used for mixing medications for patients with nasogastric feeding tubes.^[15] These patients experienced severe gastric hemorrhages.



Fig. 3: Gastrointestinal mucormycosis.

- **Cutaneous (skin) mucormycosis**

This particular infection predominantly impacts the skin, particularly in instances of skin injury or surgical procedures. It is recognized as one of the most prevalent forms of mucormycosis, occurring even among individuals with a history of immunocompromising conditions.^[16]

Cutaneous mucormycosis is relatively a newly emerging fungal infection caused by fungi from the phylum Glomeromycota. The clinical manifestations are generally nonspecific; however, a rapidly progressing necrosis suggests the presence of this infection. In relation to cutaneous mucormycosis, the strains that are most commonly isolated include *Rhizopus oryzae*, *Apophysomyces elegans*, and *Lichtheimia corymbifera*.^[17] Other identified isolates responsible for this condition include *Saksenaea vasiformis*, *Mucor* sp, *Cunninghamella bertholletiae*, *Rhizopus microspores*, and *Rhizomucor* spp.^[18] There are reports indicating that this type of fungus is linked to nitroglycerine patches and vascular devices.^[19]

Symptoms of Mucormycosis

The condition is characterized by black fungus, accompanied by nasal congestion and nosebleeds, eye inflammation, and drooping eyelids, all of which can ultimately result in vision loss. In such cases, it may be necessary to remove the eye to avert the infection from advancing to the brain. Furthermore, in certain instances, the jaw may also need to be excised to hinder the spread of the infection, alongside symptoms such as elevated temperature, facial swelling, skin ulcers, darkening around the mouth, chest pain, difficulty in breathing, double vision, and expectoration of blood, as illustrated in Figure 2.^[20]

Symptoms can differ based on the organ that is impacted.

In instances of Rhinocerebral mucormycosis, one may experience swelling on one side of the face, head pain, nasal or sinus congestion, and darkening of the nose's tip.^[21] In cases of gastrointestinal mucormycosis, symptoms manifest as abdominal pain, nausea, and vomiting, along with gastrointestinal bleeding.^[22] In cutaneous mucormycosis, the infection manifests as blisters and ulcers that rapidly enlarge and become red, often accompanied by a high fever. Their color may alter, signifying decay due to insufficient blood circulation, which necessitates their excision to prevent damage to surrounding organs.^[23]



Fig. 4: Black fungus symptoms.

The Mechanism of Mucormycosis

Steel is a crucial element for cellular growth and innovation, as it plays a role in various essential processes within the battery. Recent evidence has demonstrated that the level of available, unrestricted steel in plasma is recognized as a vital component in the development of specific predisposing factors that lead individuals to ketoacidosis. Metal is associated with complete blood, which includes peptides found in mammalian serum, such as serum ferritin, body iron, and lactoferrin, which also facilitate the transfer of steel throughout the body.^[24]

The absorption characteristics of metals inhibit similar negative effects as unrestricted metals. This occurs after certain events. It has been shown that the development of specific geobacillus isolates is reduced in normal blood unless metals are introd Limiting metal availability in circulating blood serves as a protective measure for the host group, particularly against normal pathogenic organisms and their activities. This is due to the fact that individuals with certain functions are now more susceptible to processes in which traditional healthy individuals are involved. The diagnostic findings indicate that a specific iron absorption plays a role in the pathobiology of diseases, lending more credence to this theory.

The injection of plasma containing extracellular metals has been permitted. *Oryzae* has successfully thrived in significantly acidified conditions, but not under potassium hydroxide conditions in an acidic environment. This same iron-binding potential has diminished, suggesting that an acidic environment reduces transferrin's ability to bind metals while also affecting its capacity to convert ferrous ions from transferrin through proton-mediated transfer. Researchers studying living organisms have indicated that treating rats with metal chelating agents such as deferiprone and deferasirox, which are not utilized by various xenosiderophores, has provided protection to individuals previously exposed to infectious diseases. Conversely, not all activities encompass the complete system, which has proven to be extremely vulnerable in order to render metals functionally equivalent^[25] diseases.

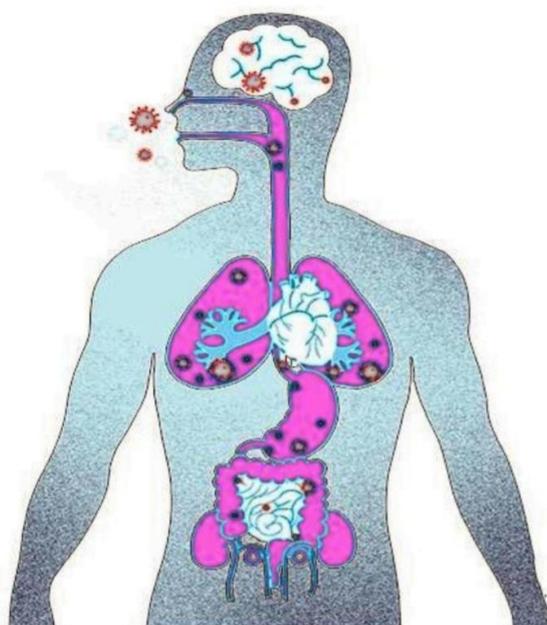


Fig. 5: Transmission of mucormycosis.

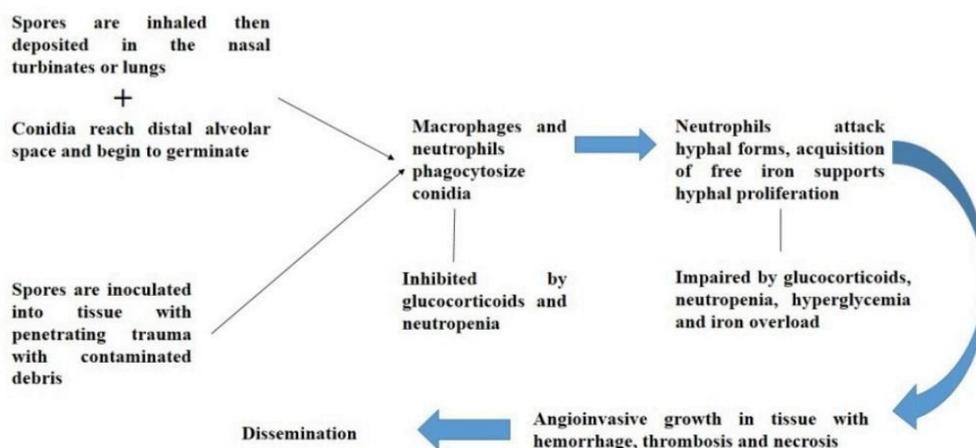


Fig. 6: Mechanism of Mucormycosis.

Clinical pathogenesis

Mucormycetes spores can indeed penetrate the body of a susceptible individual through the nose, mouth, tongue, or damaged skin, resulting in rhino-orbito-cerebral, digestive, and cutaneous wound infections. Furthermore, mucormycosis can lead to the formation of capillary blood clots, which may result in tissue necrosis. The analysis of horn brain mucormycosis remains the most prevalent among all cases of mucormycosis. This condition is particularly common in patients suffering from uncontrolled diabetes and blood cancer. Occasionally, the progression of rhino-cerebral mucormycosis can affect the nervous system, potentially leading to death. The respiratory system and nasal passages are also common sites of infection. The fatality rate associated with lung infections exceeds 60%. Depending on the severity of hyperinflammation and virological factors, clinicians often resort to the use of immunosuppressants and steroids as a potentially life-saving intervention for critically ill patient populations. Steroids help alleviate inflammation in the respiratory system, and these performance-enhancing drugs also diminish the innate immune response, which results in increased blood serum glucose levels in both diabetic and non-diabetic individuals. According to medical professionals, immunosuppressed patients are generally more susceptible to mucormycosis and black fungus.^[26] This was identified as a specific muscle and adipose connective tissue that discharges plates, which were inoculated to modulate glucose metabolism. Additionally, there is an extreme discharge of inflammation cytokines that leads to hyperinflammation. Adipocytes in connective tissue, particularly in overweight individuals, stimulate mitochondrial production and reactive oxygen species (ROS). In terms of glycaemic control, higher levels of ROS can actually increase the quantity of beneficial proteins and initiate protein kinase activity. Consequently, COVID-19 patients who are overweight are more vulnerable to attacks by mucormycosis. Individuals with severe inner ear conditions and those undergoing transplantation, as well as those with cirrhosis or neutropenia, are at a greater risk of being affected by mucormycosis. Although these patients exhibit a lower variety of myocytes and white blood cells capable of inhibiting mucormycetes mold, it is noteworthy that COVID-19 patients with a reduced number of monocytes and neutrophils have a higher likelihood of contamination with mucormycosis. As previously discussed, the risk of developing mucormycosis is primarily associated with individuals suffering from diabetes, ketoacidosis, decreased immunity, and those receiving immunosuppressants or corticosteroids, even in the context of COVID-19. The research aimed at developing an immunization against mucormycosis microbes is largely linked to groundwater pollution affecting soil and water. If COVID-19 is indeed a potential source, it

may be transmitted through liquids such as humidifiers used during supplemental oxygen therapy, especially when patients are breathing in that environment. This infectious disease can be extremely dangerous, with a mortality rate ranging from 38% to 80%.^[27]

Diagnosis

The diagnosis of mucormycosis presents a significant challenge; however, it can be achieved through the identification of distinctive symptoms, a comprehensive patient history, an extensive clinical assessment, and specialized testing. A dedicated physician is capable of making this diagnosis. It is important to note that mucormycosis does not yield results in any antigen detection tests, as the galactomannan antigen test is specifically designed for the detection of aspergillosis.^[28]

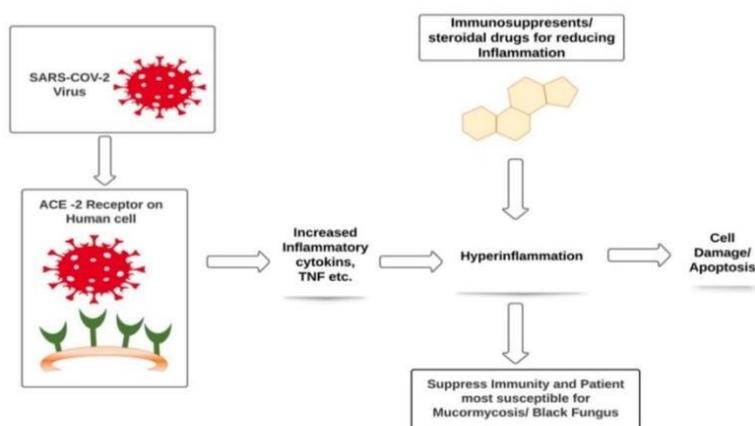


Fig. 7: In severe Covid-19 situation patient could develop dysfunction of immune system with decrease in lymphocyte counts and exponentially rise in inflammatory.

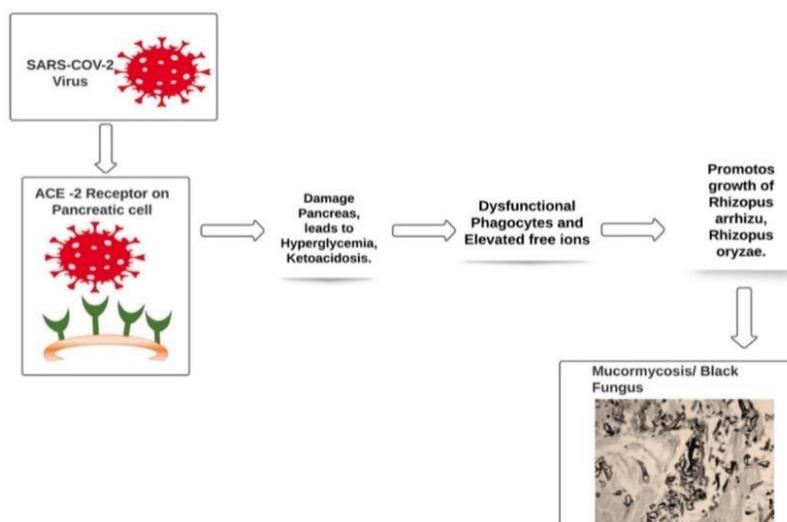


Fig. 8: The entry of SARS-CoV-2 is possible via spike protein available on the envelope which binds with angiotensin converting enzyme 2 (ACE 2), which are.

Direct microscopy (wet mounts method) of infected tissue using fluorescent brighteners such as Blankophor and Calcofluor white combined with potassium hydroxide is also employed for the swift diagnosis and visualization of characteristic fungal hyphae. Culturing the specimen is another crucial method, as mucorales can grow rapidly at temperatures ranging from 24 to 37°C within a period of 24 to 48 hours. The tissue culture method facilitates identification down to the genus and species level.^[29]

Treatment

Table 1: First-line anti fungal agents for mucormycosis.

Drug	Recommend doses	Advantage and supporting studies	Disadvantage
AMB	1.0-1.5 mg/kg/ day	>5 decades clinical ex-perience, only licensed agent for treatment of mucormycosis	Highly toxic, poor CNS penetration
LAMB	5-10 mg/kg/day	Improved CNS pen-etration compared to AMB	Expensive
ABLC	5-7.5 mg/kg/ day	Less nephrotoxic than AMB; murine and retrospective clinical data suggest benefit of combination therapy with echinocandins	More nephrotoxic than LAMB

The effective management of mucormycosis necessitates early detection, addressing risk factors and underlying health conditions, surgical debridement, and the swift administration of intravenous antifungals, primarily.

Amphotericin B: first line drug.

This process involves the immediate management of hyperglycemia, acidosis, and the discontinuation of immunosuppressive medications whenever feasible. Amphotericin B is regarded as the primary treatment for mucormycosis. The lipid formulation of amphotericin B is given in high doses intravenously once daily as the initial treatment. The starting dose is typically 5 mg/kg IV daily, with a maximum allowable dose of 10 mg/kg IV. The length of treatment is contingent upon the clinical status of the patient. Surgical debridement of the infected tissue must be conducted urgently to prevent further infection spread. Immediate aggressive surgical debridement of necrotic tissue is essential. This may require radical facial resections, partial pneumonectomy, colectomy, etc., depending on the location of the disease. Similar to necrotizing fasciitis, this situation demands very aggressive surgical intervention and often results in significant morbidity. Unless the immune status is restored, the prognosis remains unfortunately poor, even with the most intensive therapies and drastic surgical measures.

Posaconazole or Isavuconazole: Second line drug. has shown some promise as a second-line treatment for mucormycosis. For salvage therapy, posaconazole 200 mg IV four times daily is advised. The guidelines do not endorse the combination of amphotericin and posaconazole. Additional adjunctive treatments include hyperbaric oxygen therapy. The elevated oxygen pressure enhances the capacity of neutrophils to eliminate the organism and promotes wound healing.^{[30][31]}



Fig. 9: Treatment of black fungus.



Fig. 10: Before and after of black fungus.

PATHOPHYSIOLOGY

Mucormycosis commonly referred to as "black fungus," is a potentially fatal condition that can lead to visual disturbances, heart failure, and respiratory complications. The emergence

of new health risks and causative organisms, along with the challenges in managing the disease, pose significant concerns regarding mucormycosis nationwide. It is particularly prevalent among individuals suffering from COVID-19. Mucormycosis is a specific fungal infection that primarily affects immunocompromised patient populations, primarily caused by mold species such as *Mucor*, *Rhizopus*, and *Rhizomucor*, which belong to the Zygomycetes class and the Mucorales order. The most common health risk associated with this condition is diabetes mellitus, followed by hematological malignancies and solid organ transplants. A thorough understanding of underlying genetic factors, surgical excision of necrotic tissue, and effective antimicrobial therapy are essential for the diagnosis of mucormycosis. This evaluation discusses epidemiological studies, pathogenicity, symptoms of black fungus, its association with COVID-19, as well as diagnosis and treatment strategies.^[32] Mucormycosis is recognized as one of the most effectively functioning yet also severe forms of fungal infectious disease. It typically initiates within the oral cavity and nasal sinus passages following the inhalation of pathogenic organisms. This condition is attributed to life forms classified under the mucormycotina, including various taxonomic classifications such as *pleurotus* and *rhizomucor*. The reported prevalence of mucormycosis is approximately 1.7 cases per 1,000,000 individuals annually.

Mucormycosis affecting the zygomatic arch is considered rare due to the abundant blood supply in maxillofacial regions; however, more virulent fungi, such as *pleurotus*, may overcome this challenge. The common manifestation of this infectious disease can be observed in the rhinomaxillary region, particularly in immunosuppressed patients, including those with diabetes mellitus. Therefore, early detection of this potentially life-threatening condition and prompt diagnosis is crucial for reducing the mortality rate associated with it.^[33]

REFERENCE

1. Mondal A, Gireeshwar M, Govinderaj L. Black Fungus Mutilating COVID-19 Pandemic in India: Facts and Immunological Perspectives. *Eur J Biol.*, 2022; 81(1): 96-106.
2. Wan Y, Shang J, Graham R, Baric RS, Li F. Receptor Recognition by the Novel Coronavirus from Wuhan: An Analysis Based on Decade Long Structural Studies of SARS Coronavirus. *J Virol*, 2020; 94: 41-47. CDC. Available from: <https://www.cdc.gov/fungal/diseases/mucormycosis/index.html>. [Last accessed on 2020 Feb 25].
3. Cuervo NZ, Grandvaux N. Ace2: Evidence of role as an entry receptor for sars -cov-2 and

- implications in comorbidities. *Elife*, 2020; 9: e61390.
4. Cornely OA, Alastruey-Izquierdo A, Arenz D, Chen SCA, Dannaoui E, Hochhegger B, et al. Global guideline for the diagnosis and management of mucormycosis: an initiative of the European Confederation of Medical Mycology in cooperation with the Mycoses Study Group Education and Research Consortium. *Lancet Infect Dis.*, 2019; 19(12): e405-e421.
 5. Azhar EI, Hui DSC, Memish ZA, Drosten C, Zumla A. The Middle East Respiratory Syndrome (MERS). *Infect Dis Clin North Am.*, 2019; 33(4): 891-905.
 6. Lionakis MS, Iliev ID, Hohl TM. Immunity against fungi. *JCI Insight*, 2017; 2(11): e93156.
 7. Schroeder MR, Stephens DS. Macrolide resistance in *Streptococcus pneumoniae*. *Front Cell Infect Microbiol*, 2016; 6: 98.
 8. Gebremariam T, Lin L, Liu M, Kontoyiannis DP, French S, Edwards JE, et al. Bicarbonate correction of ketoacidosis alters host-pathogen interactions and alleviates mucormycosis. *J Clin Invest*, 2016; 126(6): 2280-94.
 9. Yang HN, Wang CL. Looks like tuberculous meningitis, but not: A case of rhinocerebral mucormycosis with garcin syndrome. *Front Neurol*, 2016; 7: 181.
 10. Mattingly JK, Ramakrishnan VR. Rhinocerebral Mucormycosis of the Optic Nerve. *Otolaryngol Head Neck Surg*, 2016; 155(5): 888-9.
 11. Paczosa MK, Meccas J. *Klebsiella pneumoniae*: Going on the Offense with a Strong Defense. *Microbiol Mol Biol Rev.*, 2016; 80(3): 629-61.
 12. Alaa Abdul-Hussein Al-Daamy, Haider Abd-Al Ameer, Hasan Zuher, Hussein Monather, Bashaer Ahmmad, Niesreen Kadhim. Antifungal activity of propolis against Dermatophytes and *Candida albicans* isolated from human mouth. *J contemp med sci.*, 2015; 1(3).
 13. Deng Z, Ma S, Zhou H, Zang A, Fang Y, Li T, et al. Tyrosine phosphatase SHP-2 mediates C-type lectin receptor-induced activation of the kinase Syk and anti-fungal T H 17 responses. *Nat Immunol*, 2015; 16(6): 642-52.
 14. Underhill DM, Pearlman E. Immune Interactions with Pathogenic and Commensal Fungi: A Two -Way Street. *Immunity*, 2015; 43(5): 845-58.
 15. Shin S, Jung S, Menzel F, Heller K, Lee H, Lee S. Molecular phylogeny of black fungus gnats (Diptera: Sciaroidea: Sciaridae) and the evolution of larval habitats. *Mol Phylogenet Evol*, 2013; 66(3): 833-46.
 16. Guymer C, Khurana S, Suppiah R, Hennessey I, Cooper C. Successful treatment of

- disseminated mucormycosis in a neutropenic patient with T-cell acute lymphoblastic leukemia. *BMJ Case Rep.*, 2013; 2013: bcr2013009577.
17. Lewis RE, Kontoyiannis DP. Epidemiology and treatment of mucormycosis. *Future Microbiol*, 2013; 8(9): 1163-75.
 18. Ibrahim AS, Spellberg B, Walsh TJ, Kontoyiannis DP. Pathogenesis of mucormycosis. *Clin Infect Dis.*, 2012; 54(1): S16-22.
 19. Aroch I, Ofri R, Sutton GA. Ocular Manifestations of Systemic Dis-eases. *Slatter's Fundamentals of Veterinary Ophthalmology*, 2008; 374-418.
 20. Kwon-Chung KJ. Taxonomy of fungi causing mucormycosis and entomophthoromycosis (zygomycosis) and nomenclature of the disease: molecular mycologic perspectives. *Clin Infect Dis.*, 2012; 54: 8-15.
 21. Walaa F. Antibiotic Drugs and Multidrug Resistance Bacteria. *Int J Pub Health Safe.*, 2017; 2(131): 1-3.
 22. Spellberg B. Gastrointestinal mucormycosis: an evolving disease. *Gastroenterol Hepatol (N Y)*, 2012; 8(2): 140-2.
 23. Donthukenkatateja, Yerikala Ramesh, PenabakaVenugopalaiah, Y Prapurna Chandra, The pharmacist's attitudes and knowledge of pharmacogenomics and the factors that may predict future engagement, *World Journal of Advanced Research and Reviews*, 2023; 20(01): 939–945. <http://doi.org/10.30574/wjarr.2023.20.1.2109>.
 24. Rohith A, Niharika K J, Ravindra B N, C Robin George. Case Report on Myxedema Coma Associated with End-Stage Cardiac Disease. *International Journal of Clinical Pharmacokinetics and Medical Sciences*, 2023; 3(2): 54–58.
 25. K bansal. A review on mucormycosis (black fungus) epidemiology, risk factors, treatment. *Curr. Pharm. Biotechnol*, 2021; 15(7): 389-397.
 26. Madhu M, Robin George, Manaswi N, Ashik P T, C Thasvin U R. Case Report on Henoch-SchonleinPurpuraVasculitis. *International Journal of Clinical Pharmacokinetics and Medical Sciences*, 2023; 3(2): 59–62.
 27. Ribes JA, Vanover-Sams CL, Baker DJ. Zygomycetes in human disease. *Clin Microbiol Rev.*, 2000; 13: 236–301. <https://doi.org/10.1128/CMR.13.2.236>.
 28. Walsh TJ, Gamaletsou MN, McGinnis MR, Hayden RT, Kontoyiannis DP. Early clinical and laboratory diagnosis of invasive pulmonary, extrapulmonary and disseminated mucormycosis (zygomycosis). *Clin. Infect. Dis.*, 2012; 54: S55–S60. <https://doi.org/10.1093/cid/cir868>.
 29. Skiada A, Lass-Floerl C, Klimko N, Ibrahim A, Roilides E, Petrikos G. Challenges in

- the diagnosis and treatment of mucormycosis. *Med Mycol*, Apr. 01, 2018; 56(1): 93-101. [PMC free article: PMC6251532] [PubMed: 29538730]
30. Farmakiotis D, Kontoyiannis DP. Mucormycoses. *Infect Dis Clin North Am.*, Mar. 2016; 30(1): 143-63. [PubMed: 26897065]
31. Robin George, Bhavana Sree G, Akshaya S, Bhargav C, C Sunil Kumar E. Study on the Prescription Pattern and Antibiogram in the Management of Cellulitis. *Future Journal of Pharmaceuticals and Health Sciences*, 2023; 3(2): 157–168. <https://doi.org/10.26452/fjphs.v3i2.463>
32. NK choudhary. A deadly black fungus infection among covid - 19 patients in India. *Clin epidemiol glob health*, 2021; 12(1): 423-429.