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Black Mucormycosis (Black Fungus)

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Abstract: *Mucormycosis is a rare but severe fungal infection caused by species of the order Mucorales, including Rhizopus, Mucor, Lichtheimia, Rhizomucor, Apophysomyces, and Absidia. These fungi are widespread in the environment and usually harmless but can become life- threatening in people with weakened immunity, such as those with uncontrolled diabetes, organ transplants, or prolonged steroid use. The infection can affect various body parts — most commonly the sinuses, brain, lungs, skin, or gastrointestinal tract — and often spreads rapidly through blood vessels, leading to tissue damage and necrosis.*

The disease develops when fungal spores enter the body through inhalation or open wounds. Conditions like acidosis and high iron levels, especially in diabetic ketoacidosis, promote fungal growth. Early diagnosis is crucial and relies on clinical suspicion, imaging, microscopy, and culture studies. Typical findings include broad, ribbon-like aseptate hyphae and tissue invasion.

Effective management requires a combination of prompt surgical removal of infected tissue, antifungal therapy (mainly liposomal Amphotericin B), and control of underlying conditions. Posaconazole and Isavuconazole serve as alternative or supportive treatments. Early recognition and a multidisciplinary approach are vital to improve survival rates.

Keywords*:* Absida, Apophysomyces, Lichtheimia, Mucor, Rhizomu Scor, Rhizopus

I. INTRODUCTION

Invasive mucormycosis is very serious and dangerous fungal infection . This fungal infection mostly happen to the person suffering from health issue like uncontrolled diabetes and thos person who ha organtransplant . It usually affect the lung and can spread the body (Disseminated infection) It is caused by fungi from the class Zygomycetes, mainly from the order Mucorales (such as Rhizopus, Mucor, Absidia, and Cunninghamella). The most dangerous one is Rhizopus oryzae. [1,2,3,4,5.

Another group, Entomophthorales (like Conidiobolus and Basidiobolus), causes milder infections in tropical areas, usually affecting the skin and tissues.Mucormycosis can affect different parts of the body — nose and brain (rhino-cerebral), lungs (pulmonary), skin (cutaneous), gut (gastrointestinal), or can spread throughout the body (disseminated) — mostly in people with poor immunity . [6,7,8,9,.

These fungi are very common in the environment — they live in soil, manure, plants, fruits, vegetables, and even in the air. Small amounts can also be found on the skin or in the nose of healthy people without causing harm. [10,11.

However, when a person's immune system is weak — for example, if they have uncontrolled diabetes or a serious immune disorder — the fungus can invade the body and cause infection. It mainly affects the sinuses, brain, and lungs, and can become life-threatening if not treated quickly. [12,13]

The term ―mucormycosis‖ was first used by R. D. Baker, an American pathologist, to describe infections caused by fungi belonging to the Mucorales group. The first recorded case was reported in 1885 by a German doctor named Paltauf, who called it ―Mycosis Mucorina.‖ Among all species, Rhizopus oryzae is the most common fungus responsible — it causes about 70% of all mucormycosis cases. During the COVID-19 pandemic, doctors noticed a rise in fungal co-infections. The most frequent ones were caused by Candida and Aspergillus, but many COVID-19 patients also developed mucormycosis, especially in India, where thousands of cases were reported. [14,15,16.

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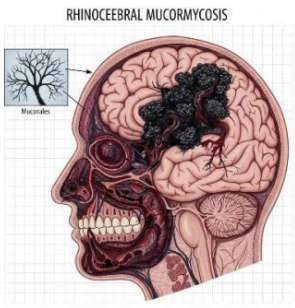
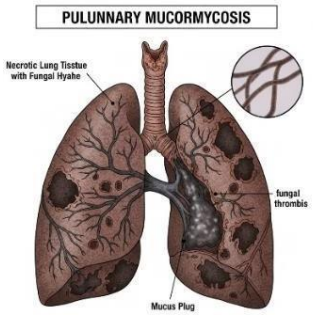
TYPES :

1 Rhinocerebral (Sinus and Brain) Mucormycosis :



This type starts in the sinuses (nose area) and can spread to the brain.It is most common in people with uncontrolled diabetes or those who have had a kidney transplant.

2.Pulmonary (Lung) Mucormycosis :



This is a serious lung infection caused by the fungus. It mainly affects people with weak immune systems, such as organ or stem cell transplant patients, or those with blood cancers or low white blood cell counts (neutropenia). It can lead to severe breathing problems if not treated in time. [17].

3. Gastrointestinal (Stomach and Intestine) Mucormycosis :

This form affects the digestive system, especially the stomach, colon, or intestines. It is more common in young children than adults — especially premature or low-birth-weight babies who have had antibiotics, surgery, or medicines that weaken the immune system. [18].

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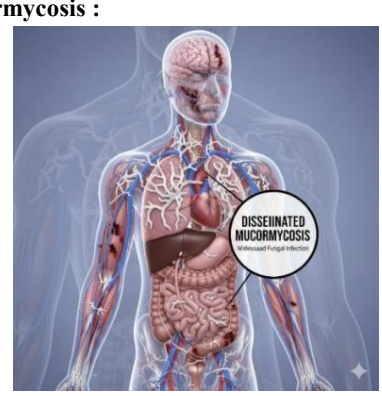
4. Cutaneous (Skin) Mucormycosis :

This type happens when the fungus enters through a cut, burn, or wound in the skin. It can develop after injuries, surgeries, or burns.It is the most common type found in healthy people who don't have immune problems.[19].

5. Disseminated (Widespread) Mucormycosis :



This occurs when the infection spreads through the blood to other parts of the body.It usually affects the brain, but can also involve the heart, spleen, skin, and other organs. This is the most dangerous form, as it affects multiple organs at once. [20]

Epidemiology:

Mucormycosis is a rare but increasingly common fungal infection seen more often in recent year .Majorlly in begium France , Switzerland and india studies show that about 1.5% of serious fungal infection in France . people taking immunosuppressive drug or voriconazole are more at risk.

The fungi that cause by the Mucorales group they are found everywhere in the environment and can grow at body temperature infection tend to increses in late summer and autumn .

infection happen when spores are brethed in or enter through cuts . [22 ,23.

In development countries it mainly affect people with diabetes blood disorder or those who have had stem cell to uncontrolled diabetes or stroke.

Mucorales mainly infect people with weak immune system such as those with diabetes ketoacidosis ,burns , injuries or those receiving iron theraphy or chemotherapy.

The order Mucorales includes many species, with Rhizopus, Mucor, and Lichtheimia* causing about 90% of mucormycosis cases. Globally, Rhizopus spp. are the most common (>70%), followed by Mucor, Lichtheimia, Cunninghamella, Apophysomyces, Rhizomucor, and Saksenaea. In India, Apophysomyces is a notable secondary pathogen, and rare species like Rhizopus homothallicus and Mucor irregularis have also been reported. [24 ,25.

Global prevalence is not well-defined due to limited population studies. In the US, incidence increased from 1.7 per million person-years to 16 per million by 2016. In India, the prevalence is ~70 times higher than the global average,

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and cases surged during the COVID- 19 pandemic. Mucormycosis incidence has also risen in China, Pakistan, Iran, and several European countries (France, Belgium, Switzerland, Spain).

Major risk factors include diabetes mellitus, hematological malignancies, stem cell or organ transplants, long-term neutropenia, corticosteroid use, and deferoxamine therapy. Post- tuberculosis patients and those with chronic kidney disease are emerging risk groups. In developed countries, hematological malignancies and transplant recipients are most affected, whereas in developing countries, uncontrolled diabetes and steroid misuse are the primary risks. Use of antifungals ineffective against Mucorales (e.g., fluconazole, voriconazole, caspofungin) may also contribute.

These fungi can cause serious infections in susceptible people, including gastrointestinal, skin, respiratory, and rhinocerebral infections. The symptoms usually depend on the person's underlying health conditions. Although these fungi are found in many environments, they rarely cause disease in healthy people, showing their low pathogenicity.

Infections mainly occur in individuals with trauma, diabetes, or severe illnesses. The respiratory system is the most common entry route, where spores land in the nasal passages or are inhaled into the lungs. In cutaneous mucormycosis, spores enter through broken or damaged skin multiply, and can spread.

While less common than Aspergillus, hospital-acquired (nosocomial) mucormycosis has been linked to construction work, contaminated ventilation systems, biomedical equipment, or non-sterile surgical dressings, particularly involving Rhizopus species. [26,27.

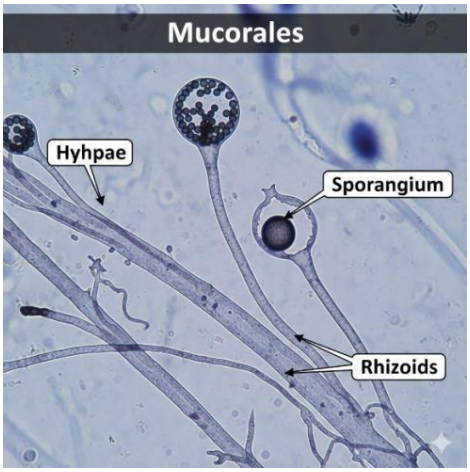
Pathogenesis:

1) How Mucorales Fungi Enter and Spread in the Body

When spores of Mucorales fungi enter the human body from the environment, the body' s defense cells called polymorphonuclear phagocytes try to destroy them through a process called phagocytosis, where immune cells —eat ‖ harmful particles.

However, sometimes the fungus can survive and even kill these immune cells. Certain health problems like acidosis (too much acid in the blood) and hyperglycemia (high blood sugar) make it easier for the fungus to grow. This happens because the fungus produces an enzyme called ketone reductase, which helps it live and multiply in acidic conditions.Once it starts growing, the fungus enters the blood vessels, where it uses the iron in the blood for growth. This can cause tissue damage, blood clots, and a serious condition known as angioinvasion, when the infection spreads through the blood vessels. [28.

After this, the fungus attacks the endothelial cells (cells lining the blood vessels) and the extracellular matrix (the structure that supports cells). This is a key stage in the disease process.



When spores first enter the body, epithelial cells such as those in the lungs (alveoli) and skin are the first to react. But the fungi can stick to the basement membrane of these cells and start growing. A protein called Glucose-Regulated Protein 78 (GRP78) acts like a receptor that allows the fungus to enter and damage the cells.Because there are

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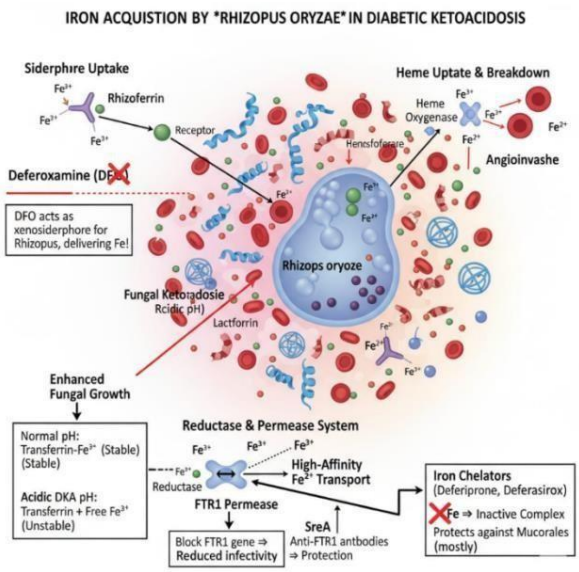
different species of Mucorales fungi, the exact way the disease develops, called pathogenesis, may vary slightly depending on the fungal type. [29].

2) Why Mucormycosis Mostly Affects Weak Immune Systems

Spores from Mucorales fungi can sometimes cause inflammation even in healthy people. However, mucormycosis usually occurs in people with weak immune systems, especially when their phagocytes (immune cells that destroy germs) don't work properly.The disease can also develop due to drug abuse or injuries to the skin or soft tissues, which allow the fungus to enter the body. Once inside, it grows very fast because it has several special features. It can survive at normal body temperature (thermotolerant) and can change its cell wall to adapt and protect itself from harsh conditions inside the body. [31 ,32.

The fungus also needs iron to grow. People with higher iron levels in their blood are more likely to get infected because iron helps the fungus multiply.For this reason, scientists are testing medicines called iron chelators, which remove extra iron from the body, to see if they can stop the infection. Some of these new medicines have shown good results in animals, but they may not work as well in people who have neutropenia (very low white blood cell counts). [33].

Mechanism :



Iron is an essential element for the growth and development of all living cells. It helps in many vital cell processes. Normally, in our body, iron is safely stored and transported by proteins like transferrin, ferritin, and lactoferrin, which prevent harmful free iron from circulating in the blood.However, research has shown that the amount of free (available) iron in the blood plays a key role in the risk of mucormycosis, especially in people with diabetic ketoacidosis (DKA).

Under healthy conditions, Rhizopus oryzae (a fungus that causes mucormycosis) does not grow well in normal human serum unless extra iron is added. This means that limiting iron availability is one of the body's natural defenses against infections, including those caused by Mucorales fungi.

In people with DKA, the blood becomes acidic, and this reduces the ability of transferrin to bind iron. As a result, more free iron becomes available, which helps the fungus grow. Experiments have shown that R. oryzae grows well when serum is made acidic and iron is added, but not in normal (alkaline) conditions.

Animal studies also found that treating DKA mice with certain iron chelators—chemicals that bind iron—such as deferiprone or deferasirox, protected them from fungal infection. These drugs are safe because Mucorales cannot use them as a source of iron. But not all species of Mucorales respond to these chelators in the same way.

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Another important finding comes from patients who are on dialysis and are treated with the iron chelator deferoxamine. These patients are more likely to get mucormycosis because deferoxamine actually acts as a siderophore (iron carrier) for the fungus. In simple terms, deferoxamine binds to iron and delivers it to Rhizopus, helping it grow. The fungus recognizes deferoxamine through a special receptor and takes up the iron, first converting ferric iron (Fe^{3+}) to ferrous iron (Fe^{2+}), which is more soluble and useful for fungal growth. Interestingly, this effect happens only with Rhizopus, not with other fungi like Candida albicans.iron overload from repeated blood transfusions (as seen in patients with myelodysplastic syndrome) also increases the risk of mucormycosis.

To obtain iron, fungi use three main mechanisms:

1. Iron permeases and reductases – surface proteins that convert ferric iron (Fe^{3+}) to ferrous iron (Fe^{2+}) and transport it into the cell.
2. Siderophores – small molecules that bind and carry iron.
3. Heme uptake – using heme (from hemoglobin) as a source of iron.

The FTR1 gene in R. oryzae encodes a high-affinity iron permease, which is crucial for iron uptake. Experiments show that when this gene is blocked or reduced, the fungus becomes less infectious in animals. Vaccinating mice with anti-FTR1 antibodies protected them from infection, proving that FTR1 is a key virulence factor.

R. oryzae also makes its own siderophore called rhizoferrin, but it is not very effective at taking iron from human serum, so its role in infection may be small.

Additionally, the fungus can obtain iron from heme (found in blood). Genes for heme oxygenase—enzymes that break down heme to release iron—were found in R. oryzae. This might explain why the fungus is angioinvasive (invades blood vessels). Even when FTR1 is reduced, R. oryzae can still grow if heme is available, suggesting that heme uptake is another pathway for getting iron.Finally, genes like SreA, known from other fungi such as Aspergillus fumigatus, may help R.oryzae adapt to different iron levels in its environment. [34].

Clinical diagnosis :

To diagnose mucormycosis early, doctors must be alert and suspicious, especially in people with risk factors like diabetes or a weakened immune system.

For example:

* Double vision (diplopia) in a diabetic person, or
* Chest pain (pleuritic pain) in someone with low white blood cells (neutropenia)

These could be warning signs of mucormycosis.In such cases, doctors should quickly perform imaging tests (like CT scans) and collect samples for lab testing using:

* Microscopy (histology)
* Microbiology
* Molecular tests (like PCR) # Common Types of Mucormycosis

The infection most often affects:

* Sinuses and brain (rhinocerebral form)
* Lungs (pulmonary form)
* Skin and soft tissues
* Multiple organs (disseminated form)

But it can infect almost any organ in the body.

The main sign of mucormycosis is tissue death (necrosis) caused by blood vessel invasion.

However, symptoms alone are not enough for diagnosis because other fungi (like Aspergillus or Fusarium) can cause similar signs.

In areas where tuberculosis (TB) is common, both infections may occur together — especially in diabetic patients.

Red Flag Symptoms (Warning Signs) :

Doctors should strongly suspect mucormycosis if these signs appear:

* Cranial nerve palsy (weakness of face or eye muscles)
* Double vision (diplopia)
* Sinus pain

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* Eye bulging (proptosis)
* Swelling around the eyes (periorbital swelling)
* Orbital apex syndrome (severe eye and vision symptoms)
* Palate ulcers (roof of mouth sores) # Imaging Findings :

CT scans can help detect lung involvement.
Some key findings are:
* Multiple nodules in the lungs
* Pleural effusion (fluid around the lungs)

Reverse Halo Sign (RHS): a round area on the CT scan where a dark center is surrounded by a lighter ring — strongly linked to mucormycosis.
In one study, 94% of leukemia patients with mucormycosis showed the RHS in their early scans.
Comparing infections:
* RHS was more common in mucormycosis (54%) than in aspergillosis (6%).

Aspergillosis showed more airway-centered changes (like small clusters of nodules or mycosis early and perform lab confirmation tests quickly.
Other Diagnostic Tools
* PET/CT scan using a special dye (FDG) can help identify infection areas. Endobronchil ultrasound-guided fine needle aspiration (EBUS-FNA) collect lung samples safely for diagnosis. [35].

When samples from infected patients are grown in the lab, the fungus grows well at 37°C and forms fluffy white, gray, or brown colonies that can fill the petri dish within 1 to 7 days.
If tissue samples are crushed too much during processing (mechanical homogenization), the chances of the fungus growing in culture may decrease.
To directly detect the fungus under a microscope, different staining methods can be used, such as:
* 20% potassium hydroxide (KOH) preparation
* Gomori's methenamine silver (GMS) stain
* Hematoxylin and eosin (H&E) stain
* Periodic acid–Schiff (PAS) stain

Under the microscope, Mucorales fungi usually appear as broad (10–50 µm), ribbon-like hyphae that have few or no cross walls (aseptate or pauciseptate) and branch at wide angles (45° to 90°).
In tissue sections (histopathology), these fungi often show angioinvasion (they invade blood vessels), which leads to tissue death (infarction) around the infected area.- [36].

Treatment :

1. General Management

Mucormycosis requires immediate and intensive care. Treatment usually combines:
Surgical removal of infected tissue
Potent antifungal medications
Supportive management
Control of underlying conditions, such as diabetes or steroid overuse
There isn't a fixed treatment duration — therapy continues until clinical symptoms resolve, imaging improves, lab results normalize, and the patient's immunity stabilizes. [37.

2. Surgical Management

Surgery is often the first and most critical step in treating mucormycosis. Early and complete excision of infected tissue greatly improves outcomes.
Some patients may need more than one operation.
If the infection extends to the brain or skull base, prognosis worsens.
In pulmonary cases, procedures like lobectomy or thoracotomy may be done.
Cutaneous mucormycosis usually responds well to debridement combined with antifungal therapy.

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Surgery may offer limited benefit for patients with very low platelets or severe systemic illness. [38,39,]

3. Antifungal Therapy

Amphotericin B

Remains the first-line drug for mucormycosis.

The liposomal form causes fewer kidney-related side effects. Typical dose: 5–10 mg/kg per day (higher for brain involvement).

Side effects include renal toxicity and low potassium.

A newer oral version, MAT2203, is under evaluation.

Posaconazole

Effective against mucormycosis, available as tablets, IV, and suspension (tablet and IV are preferred).

Used when Amphotericin B is ineffective or unsuitable. Also helpful for preventing infection in high-risk individuals. Blood level monitoring is advised for optimal dosing.

Isavuconazole

Has good brain penetration and fewer adverse effects. Used mainly as salvage therapy when other drugs fail.

Therapeutic drug monitoring is not necessary.

Echinocandins

Have limited activity alone but may enhance Amphotericin B's effect by disrupting the fungal cell wall. [40,41,42,.

4. Combination Therapy

Evidence is limited, but Amphotericin B with Posaconazole may be more effective in certain severe or resistant infections.

Routine use is not recommended until stronger evidence supports it. [43,44.

5. Supportive and Adjunctive Treatments

Additional measures can help strengthen the immune system, such as:

Interferon-gamma therapy

Immune checkpoint inhibitors

Hyperbaric oxygen

Iron chelators (e.g., Deferasirox)

Growth factors to boost white blood cells

These are reserved for selected cases due to limited supporting data. [45,46.

6. Emerging and Experimental Drugs

Several new antifungal agents are being studied, including:

VT-1161

APX001

Hemofungin

These are not yet approved and remain in experimental stages.

7. Multidisciplinary Approach

Optimal care requires collaboration among ENT surgeons, ophthalmologists, neurosurgeons, infectious disease specialists, radiologists, pathologists, critical care teams, and clinical pharmacists.

This team approach enables early diagnosis, precise surgical planning, and effective therapy coordination. [47,48].

Conclusion :

Mucormycosis is a dangerous and fast-spreading fungal infection that mainly affects people with weak immunity, especially those with uncontrolled diabetes or on long-term steroids. Early diagnosis is critical because the infection causes rapid tissue damage through blood vessel invasion. Effective treatment requires quick surgical removal of infected tissue, strong antifungal medicines like liposomal Amphotericin B, and proper control of underlying diseases. Newer drugs and supportive therapies are improving outcomes, but prevention, awareness, and early medical attention remain the most important steps to reduce serious complications and deaths.

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