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Mucormycosis (Zygomycosis): An overview

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Abstract

Mucormycosis is an emerging angioinvasive disease caused by saprophytic fungi of the order Mucorales. Spores of these widespread filamentous fungus (found in soil, fallen leaves, compost, animal dung, and air) can be inhaled and infect the lungs, sinuses, brain, and eyes. Infection occurs less frequently when spores enter the body through a cut or an open wound. Mucormycosis is not an infectious disease that may be passed from person to person. The hyphae infiltrate blood vessels, producing tissue infarction and necrosis, regardless of the mode of infection (inhalation of airborne spores, ingestion, or direct skin inoculation). Hematological malignancies (leukaemia, lymphoma, and multiple myeloma), aplastic anaemia, myelodysplastic syndromes, solid organ or hematopoietic stem cell transplantation, human immunodeficiency virus (HIV) infection, diabetic and metabolic acidosis, intravenous drug abuse, prematurity, and advanced age are all risk factors.The underlying conditions can influence clinical presentation and outcome. Finally, with mucormycosis in India, the rise in the number of cases, the advent of new risk factors and causative agents, and the problems in controlling the disease are all major issues.This review describes the emerging epidemiology and the clinical manifestations of mucormycosis.

Keywords: Mucormycosis, Epidemiology, Incidence, Zygomycosis.

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INTRODUCTION

Mucormycoses are life-threatening fungal infections that commonly affect haematological, solid organ transplant, and diabetes patients; however, they can also harm immunocompetent patients after a trauma or burn [1]. R.D. Baker, an American pathologist, coined the name Mucormycosis, which can also be referred to as Zygomycosis. Mucormycosis is a sneaky fungal infection spread by members of the Mucorales and zygomycotic species. Mucormycotina is a type of saprobe that can be found in rotting waste or soil. Mucorales infections are classified according to how quickly they progress. The first instance of Mucomycosis was documented as Mycosis Mucorina by the German doctor Paltauf in 1885. In the 1980s and 1990s, the rate of mucormycosis climbed substantially, primarily in immunocompromised people [2].

Inhaled Spores of these common fungi (found in soil, fallen leaves, compost, animal dung, and air) can infect the lungs, sinuses, and extend into the brain and eyes. Infection occurs less frequently when spores enter the body through a cut or an open wound[3]. Patients with severe COVID-19 or those recovering from the condition have been documented to develop fungal infections such as mucormycosis, aspergillosis, and invasive candidiasis, which have been linked to serious sickness and mortality [3].

During COVID -19, India saw an increase in mucormycosis cases. Prevention of COVID-19 related mucormycosis need to cognizance on aiming for better glycaemic manipulate in COVID-19 sufferers and tracking the usage of systemic corticosteroids in treating intense cases [3].

Outpatient use of systemic corticosteroids/different immunomodulating tablets for slight or mild sufferers with COVID-19 must be avoided. Health care centres want to bolster their contamination prevention and control (IPC) programmes to prevent healthcare-related outbreaks [3].

Early identity and instant clinical and surgical interventions are important to save the excessive morbidity and mortality related to this infection. Although rare, there are suggested instances of an indolent sickness route for mucormycosis [4].

What is mucormycosis?

Mucormycosis (also known as zygomycosis) is a dangerous but uncommon fungus caused by a group of moulds known as mucormycetes. These fungus can be found all over the place. They live in soil and decomposing organic waste including leaves, compost piles, and rotten wood [5].

Prevalence and incidence

Mucormycosis is becoming more common over the world[5]. Mucormycosis has an incidence rate that ranges from 0.005 to 1.7 per million people worldwide. A systematic review and meta-analysis of 851 case studies was conducted in 2018 and the results revealed that 389/851 (46%) of patients died. Patients with disseminated mucormycosis (68%) had the greatest case fatality rate, whereas those with cutaneous disease had the lowest (31 percent) [3].

Mucormycosis prevalence in India is estimated to be 140 per million people, which is nearly 80 times higher than in developed countries. The most common risk factor is diabetes mellitus, which is followed by haematological malignancy and solid-organ transplantation. In this country, patients with postpulmonary tuberculosis and chronic renal illness are at an increased risk of developing mucormycosis. Trauma increases the risk of cutaneous mucormycosis. In India, isolated renal mucormycosis in an immunocompetent host is a rare occurrence [6].

Types and clinical manifestations of mucormycosis

- Rhinocerebral mucormycosis (sinus and brain) is a sinus infection that can extend to the brain. People with uncontrolled diabetes and those who have received a kidney transplant are more likely to develop this condition.. Symptoms as follows:
    - One-sided facial swelling
    - Headache
    - Nasal or sinus congestion,
    - bleeding from nose,
- Severe black lesions on the bridge of the nose or the upper interior of the mouth
- Fever is frequently accompanied by blindness, exophthalmos, facial paralysis, and indications of trigeminal nerve invasion.
    - Cavernous sinus thrombosis
- The most prevalent type of mucormycosis in persons with cancer and those who have undergone an organ transplant or a stem cell transplant is pulmonary (lung) mucormycosis.
    - Fever
    - Cough
    - Chest pain
- Mucormycosis of the gastrointestinal tract is more frequent in young children than in adults. Antibiotics, surgeries, or medications that impair the body's ability to fight germs and sickness put premature and low-birth-weight infants under one month of age at danger [6-7].
    - Abdominal pain
    - Nausea and vomiting
    - Gastrointestinal bleedings
- Cutaneous (skin) mucormycosis happens when fungus get into the body through a skin crack. After a burn, scrape, cut, surgery, or other sorts of skin trauma, this type of infection can develop. This is the most common form of mucormycosis among people who do not have weakened immune systems.
    - Blisters or ulcers,
    - Infected area may turn black.
- Other signs and symptoms include pain, warmth, redness, and swelling around the incision.

Disseminated mucormycosis develops when an infection spreads from one part of the body to another through the bloodstream. The infection is most usually found in the brain, although it can also damage the spleen, heart, and skin [7].

Transmission of mucormycosis

- It is noncontagious and it does not transmit from person to person..
- This fungus is found in the wild. Transmission occurs through inhalation, injection, or ingestion of spores from the environment.
- Sticky bandages, wooden tongue depressors, hospital linens, negative pressure rooms, water leaks, inadequate air filtration, non-sterile medical instruments, and building architecture have all been linked to healthcare-related epidemics, despite the fact that most incidences are rare.
- The sinuses and lungs are most commonly affected after inhaling fungal spores from the air. In such cases, it may spread to the brain and eyes.
- When a cut, burn, or other type of skin damage becomes infected, it can lead to infection

Diagnosis

Early identification of mucormycosis is important since it can improve prognosis.It has been found in studies to improve survival as well as lessen the necessity for or extent of surgical resection, deformity, and pain. Because the condition is so uncommon, having a strong index of suspicion is crucial. Recognition of risk factors, evaluation of clinical manifestations, early use of imaging modalities, and fast implementation of diagnostic approaches based on histology, cultures, and advanced molecular techniques are all part of the diagnosis process.

3.1. Clinical Diagnosis

The sensitivity and specificity of the clinical approach to diagnosis are both low. Mucormycosis is characterised by tissue necrosis produced by angioinvasion and thrombosis; nevertheless, the absence of a necrotic eschar does not rule out the diagnosis. In immunocompromised patients, mucormycosis can induce necrotic cutaneous lesions; however, other infections such as Aspergillus, Fusarium, Pseudallescheria, and Scedosporium species should be examined in the differential diagnosis.

3.2. Laboratory Diagnosis on a Regular Basis

In clinical practise, histopathology, direct examination of wetmounts, and culture are used to diagnose mucormycosis in the laboratory.

3.2.1. Histopathology

In patients with pulmonary mucormycosis, a definite diagnosis is predicated on the presence of fungal hyphae typical of mucormycetes in biopsies of afflicted tissues or bronchoalveolar lavage (BAL). Histopathology is an important diagnostic tool because it may detect the presence of fungus as a pathogen in a culture contaminated sample and assess whether blood vessel invasion has occurred.

3.2.2. Direct Microscopy

Direct microscopy of KOH wet mounts can be employed for a quick presumptive diagnosis of mucormycosis. It can be used on all materials delivered to the clinical laboratory, preferably with fluorescent brighteners as Blankophor and Calcofluor White in combination with KOH to facilitate visualisation of the typical fungal hyphae, which in this instance necessitates the use of a fluorescent microscope.

3.2.3. Culture

Culture of specimens is required for the diagnosis of mucormycosis since it enables for genus and species identification, as well as antifungal susceptibility testing. Mucorales, the most medically significant of the Mucorales, are thermotolerant and may grow fast at temperatures as low as 37°C. Colonies emerge within 24–48 hours on nearly any carbohydrate substrate, and identification is based on colonial and microscopic morphology as well as growth temperature.

3.3. Applied and Emerging Molecular Methods

Molecular approaches have become an important tool for confirming infections and identifying the strains involved. As a result, methods have been developed to properly identify strains that have previously grown in cultures to the species level, as well as methods to detect mucormycetes in tissues.

3.4. Non-Invasive Diagnostic Methods: A Look Ahead?

3.4.1. Molecular

Most of the above-mentioned approaches employ samples that need invasive procedures, which may not be appropriate for specific patient groups (hematologic malignancies with thrombocytopenia, ICU patients, etc.). Blood cultures are negative despite the infection's angioinvasive nature. Only a few cases with a positive blood culture have been documented thus far. Fungal DNA, on the other hand, circulates in the blood. As a result, a lot of research is going on right now, focused on non-invasive approaches like qPCR for detecting circulating mucoralean DNA in blood (plasma or serum) or urine.

3.4.2. Serology

Antigen markers for Mucorales, such as galactomannan (GM) for Aspergillus, are not commercially available. GM blood tests and BAL in haematological patients or patients with compatible chest CT imaging results, on the other hand, may be employed to reduce the risk of mucormycosis.

3.4.3. Metabolomics-Breath Test

Koshy et al. used thermal desorption gas chromatography/tandem mass spectrometry (GC–MS) to examine breath volatile metabolite profiles in an experimental murine model of invasive mucormycosis (IM). They used the three Mucorales species that most commonly cause human IM—Rhizopus arrhizus var. arrhizus, R. arrhizus var. delemar, and R. microporus.

MANAGEMENT

General principles

The following are the four steps in the successful treatment of mucormycosis:

(1) Early diagnosis

Starting polyene therapy within 5 days of a mucormycosis diagnosis was linked to a higher chance of survival than starting polyene therapy 6 days after a diagnosis (83 percent vs. 49 percent survival). As a result, obtaining an early diagnosis of mucormycosis is important in order to begin aggressive antifungal medication as soon as possible.

(2) Reversal of underlying predisposing risk factors

When treating individuals with mucormycosis, it's important to correct or avoid underlying abnormalities in host defence. Immunosuppressive drugs, especially corticosteroids, should be used at lower doses or avoided altogether if at all feasible. In diabetics in ketoacidosis, aggressive therapy to quickly restore euglycemia and normal acid-base balance is important.

(3) Surgical debridement where applicable

During mucormycosis, blood artery thrombosis and the accompanying tissue necrosis can prevent antifungal medicines from reaching the infection site. As a result, debridement of necrotic tissues may be necessary for full mucormycosis eradication.

(4) Prompt antifungal therapy

Unless patients refuse polyene medication or, conceivably, in cases of lesser sickness in immunocompetent hosts who have had the disease surgically removed, the first antifungal therapy for mucormycosis should be based on a polyene.The only antifungal medications having in vitro action against mucorales were amphotericin B (Amb) and its lipid formulations, as well as posaconazole. Isavuconazole, a new antifungal drug, has just been added to the arsenal. Liposomal Amb (L-Amb) or Amb lipid complex is the first-line antifungal drug indicated (ABLC). The length of first-line antifungal therapy should be chosen on a case-by-case basis and modified according to the underlying disease.

PROGNOSIS AND MORBIDITY RATE

The depth of the ailment and the successful treatment offered in response to it influence the prognosis.The survival rate varies depending on the type of infection: rhino cerebral mucormycosis has a 45 percent survival rate, focal cerebral mucormycosis has a 33 percent survival rate, pulmonary forms have a 36 percent survival rate, sinusitis without cerebral involvement has an 87 percent survival rate, cutaneous isolated disease has a 90 percent survival rate, disseminated disease has a 16 percent survival rate, and gastro intestinal involvement has a 10 percent survival rate. Patients with low baseline serum iron / ferritin concentrations, neutropenia, and malignant diseases that aren't caused by infection have a higher chance of survival.

CONCLUSION

Though the etiopathogenesis of this illness varies from country to country, its presentation can be quite aggressive, with a high death rate if not treated promptly. As a result, it presents a problem to many doctors. Keeping the high death rate in mind, the key to effectively treating this illness is early and fast diagnosis, as well as an attempt to recover from the predisposing conditions. The status of this fatal disease can also be improved with early intervention, such as surgical debridement and medicinal medicines.

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