Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202631782

Early Recognition of Mucormycosis in High-Risk Patients: The Role of Nursing Assessment and Clinical Monitoring-An Updated Review

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Abstract

Background: Mucormycosis is a rapidly progressive invasive fungal infection caused by Mucorales and is associated with substantial morbidity and mortality, particularly among patients with diabetes mellitus, hematological malignancies, transplantation, neutropenia, severe COVID-19, and other states of impaired host defense. Its angioinvasive nature promotes vascular thrombosis, tissue ischemia, infarction, and necrosis, making early recognition and treatment essential.

Aim: This review aims to provide a comprehensive clinical overview of mucormycosis, emphasizing its etiology, epidemiology, pathophysiology, histopathological characteristics, clinical manifestations, diagnostic evaluation, and current principles of treatment and nursing management.

Methods: A narrative review was developed using established medical literature addressing the microbiology, epidemiology, mechanisms of disease, clinical presentations, diagnostic approaches, and therapeutic management of mucormycosis. Particular attention was given to histopathological findings, imaging characteristics, antifungal therapy, surgical intervention, correction of predisposing conditions, and supportive multidisciplinary care.

Results: Mucormycosis primarily affects individuals with impaired immune or metabolic defenses. *Rhizopus* represents the most frequently implicated genus. Disease commonly involves the rhino-orbital-cerebral region, lungs, skin, gastrointestinal tract, and disseminated sites. Histopathology demonstrates broad, pauciseptate or nonseptate hyphae with irregular right-angle branching and tissue or vascular invasion. Diagnosis requires integration of clinical findings, imaging, histopathology, culture, and emerging molecular methods. Management depends on prompt antifungal therapy, extensive surgical debridement when feasible, correction of underlying risk factors, and selected use of combination or adjunctive therapies.

Conclusion: Early recognition, rapid diagnostic evaluation, aggressive source control, appropriate antifungal therapy, and correction of predisposing conditions are central to mucormycosis management. Nurses contribute substantially through risk assessment, clinical monitoring, medication management, wound care, and multidisciplinary coordination.

Keywords: Mucormycosis; Mucorales; Invasive fungal infection; Histopathology; Antifungal therapy; Surgical debridement; Nursing management; Angioinvasion.

Introduction

Mucormycosis is an invasive and potentially life-threatening fungal infection caused by members of the order Mucorales. Although these organisms are widely distributed in the environment and are generally regarded as low-virulence molds with limited pathogenic potential in healthy individuals, they can produce rapidly progressive disease when host defense mechanisms are impaired. A defining characteristic of mucormycosis is its marked angioinvasive capacity, whereby fungal hyphae penetrate vascular structures and compromise local blood flow. This vascular invasion promotes thrombosis, ischemia, tissue infarction, and extensive necrosis, allowing the infection to progress rapidly and increasing the risk of organ dysfunction and

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Receive Date: 30 August 2026, Revise Date: 15 September 2026, Accept Date: 20 September 2026

mortality. The clinical severity of mucormycosis therefore reflects an interaction between fungal invasion and the underlying condition of the affected host. Early recognition is particularly important because the pathological process can advance considerably before characteristic clinical manifestations become apparent.[1] Mucormycosis occurs predominantly among individuals with impaired immune or metabolic defenses. Patients with hematologic malignancies represent an important high-risk population, particularly when the disease or its treatment results in prolonged neutropenia or other forms of immune dysfunction. Recipients of solid-organ or hematopoietic stem-cell transplantation are also susceptible because of immunosuppressive therapy and compromised host immunity. Poorly controlled diabetes mellitus constitutes another major predisposing condition, particularly when metabolic abnormalities impair innate immune responses and create physiological conditions that favor fungal proliferation. Nevertheless, mucormycosis is not restricted to conventionally immunocompromised populations. Invasive disease has also been reported in individuals who develop substantial tissue injury, including those exposed to traumatic wounds or severe burns. Furthermore, severe COVID-19 infection has been associated with mucormycosis, particularly in patients with additional metabolic or immunological risk factors. These observations emphasize the importance of assessing individual susceptibility rather than relying exclusively on a single underlying diagnosis.[1]

The anatomical presentation of mucormycosis depends on the route of infection, host characteristics, and underlying risk factors. The rhino-orbital-cerebral form is particularly important because infection involving the paranasal sinuses can extend into the orbit and intracranial structures, resulting in substantial neurological and visual morbidity. Pulmonary mucormycosis represents another major clinical presentation and is frequently encountered in patients with hematological or immunological disorders. Although the sinuses, lungs, orbits, and brain are among the most frequently affected sites, Mucorales infection can involve almost any organ system, including the skin and gastrointestinal tract. The possibility of extrapulmonary or disseminated disease requires clinicians and nurses to maintain a high level of clinical vigilance when unexplained tissue necrosis, systemic deterioration, or localized symptoms occur in a susceptible patient. From a nursing perspective, continuous assessment of changes in neurological, respiratory, ocular, dermatological, and systemic status can contribute to earlier recognition of clinical deterioration.[1] Successful management of mucormycosis depends on the rapid coordination of diagnostic, medical, surgical, and nursing interventions. Management should address the underlying predisposing condition whenever correction is possible, while antifungal treatment should be initiated without unnecessary delay when invasive disease is suspected or established. Surgical intervention, including aggressive debridement of devitalized tissue, may be required to reduce the fungal burden and limit further local extension. The complexity and rapid progression of the disease make interdisciplinary collaboration essential, involving physicians, surgeons, infectious disease specialists, pharmacists, microbiology and pathology teams, and nurses. Nursing care has an important role throughout this process through systematic patient assessment, monitoring for clinical deterioration, administration and monitoring of prescribed antifungal therapy, wound and postoperative care, infection-prevention practices, and patient and family education. Early identification of risk factors and subtle changes in clinical condition can facilitate timely escalation of care and support the broader multidisciplinary strategy required to manage this severe invasive fungal infection.[1]

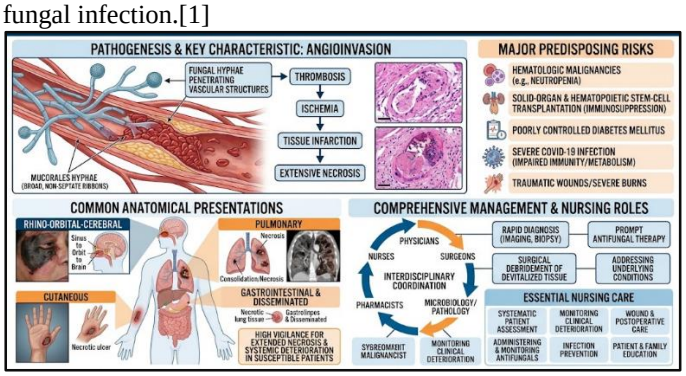

Figure-1: Mucormycosis.

Etiology

Mucormycosis is an invasive fungal infection caused by organisms belonging to the order Mucorales within the class Mucoromycetes. The order encompasses a diverse group of environmental molds, comprising more than 300 species distributed among 56 genera and 11 families, of which at least 39 species have been associated with human disease.[2] These fungi are widely distributed in the natural environment and primarily function as saprophytic organisms involved in the decomposition of organic material. They can be found in soil, decaying vegetation, plant debris, and animal dung, providing continuous environmental exposure to humans.[3] Despite their widespread presence, exposure does not usually result in disease in individuals with intact host defenses. The development of invasive mucormycosis therefore depends largely on the interaction between fungal characteristics and host susceptibility, particularly when immune, vascular, or metabolic defenses become compromised. Members of Mucorales possess morphological characteristics that distinguish them from many other pathogenic filamentous fungi.

They are rapidly growing, filamentous molds that reproduce predominantly through asexual production of sporangiospores. Their hyphae are characteristically broad and ribbon-like and generally demonstrate few or no septations. Histologically, the hyphae commonly exhibit irregular branching at approximately right angles. These morphological characteristics have considerable diagnostic importance because tissue examination may provide an early indication of invasive infection, particularly when fungal elements are identified within necrotic or ischemic tissue. The ability of these organisms to invade blood vessels represents a major pathogenic feature and contributes to thrombosis, tissue ischemia, infarction, and necrosis. Consequently, the biological characteristics of Mucorales are closely associated with the rapid clinical progression that can occur once invasive infection becomes established.[2]

Several genera within the order Mucorales have been implicated in human disease. The most clinically significant include *Rhizopus*, particularly *Rhizopus arrhizus* and *Rhizopus microsporus*, *Mucor*, including *Mucor circinelloides*, and *Rhizomucor*, such as *Rhizomucor pusillus*. Additional genera associated with human infection include *Lichtheimia*, formerly classified within *Absidia*, as well as *Cunninghamella*, *Apophysomyces*, *Saksenaea*, *Actinomucor*, *Cokeromyces*, *Tahmnostylum*, and *Syncephalastrum*.[4] The distribution of these organisms and their association with particular clinical presentations may vary according to geographic region, host characteristics, and environmental exposure. Recognition of the causative genus and species can therefore contribute to epidemiological characterization and may provide clinically relevant information during the diagnostic evaluation of invasive fungal disease. Among the Mucorales, *Rhizopus* is the predominant genus associated with human mucormycosis and is estimated to account for up to 70% of cases worldwide.[5] *Rhizopus* species are particularly important in patients with diabetes mellitus, hematological malignancies, transplantation, and other conditions associated with impaired host defenses. Nevertheless, the presence of Mucorales in the environment should not be interpreted as an indication of inherent pathogenicity. These organisms are generally regarded as nonpathogenic or of low pathogenic potential in immunocompetent individuals because effective innate immune mechanisms can prevent fungal germination and tissue invasion.[6] Disease develops when these protective mechanisms are disrupted, allowing fungal spores to germinate, hyphae to proliferate, and vascular invasion to occur. Accordingly, the etiology of mucormycosis should be understood as the result of both environmental exposure to ubiquitous Mucorales and the presence of host-related conditions that permit these normally low-pathogenic organisms to establish invasive infection.[6]

Epidemiology

Mucormycosis is an uncommon but severe invasive fungal infection with a substantial clinical burden because of its rapid progression, tissue-invasive behavior, and high mortality among susceptible patients. The estimated global incidence is approximately 2.7 cases per 100,000 population, corresponding to nearly 211,000 cases annually. In most countries, the estimated incidence is approximately 2 cases per million population, whereas substantially higher rates have been reported in India and Sri Lanka, where the estimated incidence reaches approximately 14 cases per 100,000 population.[7] The marked geographic variation in disease occurrence reflects differences in the prevalence of underlying medical conditions, access to healthcare, diagnostic capacity, corticosteroid exposure, environmental conditions, and population-level metabolic risk. India has reported a particularly high burden of mucormycosis, which has been attributed in part to the high prevalence of poorly controlled diabetes mellitus, widespread corticosteroid exposure, and environmental factors that may facilitate infection and influence clinical outcomes.[8][9] The epidemiological profile of mucormycosis is strongly associated with conditions that impair innate immune defense or alter normal metabolic and vascular physiology. Diabetes mellitus, particularly when complicated by diabetic ketoacidosis, represents one of the most important predisposing conditions. Hematological malignant neoplasms, especially acute myeloid leukemia, constitute another major risk group, together with recipients of hematopoietic stem-cell or solid-organ transplantation. Profound and prolonged neutropenia also markedly increases susceptibility, particularly when the absolute neutrophil count remains below 500 cells/μL for more than 7 days. Additional recognized risk factors include severe COVID-19 infection, iron overload, systemic glucocorticoid therapy, trauma, extensive burns, and advanced HIV infection accompanied by severe CD4+ lymphocyte depletion. These factors can compromise cellular immunity, disrupt tissue integrity, alter iron availability, or impair the mechanisms responsible for limiting fungal germination and invasion.[10][12][13][14][15][16]

The contribution of individual risk factors varies considerably between geographic regions and patient populations. Poorly controlled diabetes mellitus is considered the most prominent predisposing factor worldwide and has been associated with approximately 17% to 88% of mucormycosis cases depending on the clinical and geographical setting. In published case series from India, diabetes mellitus has been identified in more than half of affected patients. In contrast, hematological malignant neoplasms represent the leading underlying risk factor in Europe and the United States, accounting for approximately 38% to

62% of reported cases. Solid-organ transplantation contributes to approximately 2% to 15% of mucormycosis cases.[10] These differences indicate that the epidemiology of mucormycosis is closely linked to the structure of local healthcare populations and the distribution of chronic disease, immunosuppressive treatment, transplantation, and hematological disorders. The COVID-19 pandemic produced a substantial increase in reported cases of COVID-19-associated mucormycosis, particularly among patients with diabetes and those receiving corticosteroid therapy. More than half of the reported COVID-19-associated mucormycosis cases occurred in India, which accounted for approximately 57% of reported cases, followed by Iran at 11%, Egypt at 6%, and France and Türkiye at approximately 3% each.[11] This distribution illustrates the interaction between infectious disease, metabolic disorders, immunomodulatory treatment, and healthcare-related factors in shaping the epidemiology of mucormycosis. Beyond these established associations, emerging populations at risk include patients receiving chimeric antigen receptor T-cell therapy, individuals exposed to severe trauma or combat-related blast injuries, and patients with extensive burns.[12][13][14][15][16] For nursing practice, recognition of these epidemiological patterns is relevant because patients with multiple risk factors require close surveillance for early clinical manifestations, prompt escalation of suspected infection, and coordinated multidisciplinary management.

Pathophysiology

Mucormycosis develops through a sequence of events that begins with exposure to ubiquitous environmental Mucorales spores and progresses to fungal germination, tissue invasion, vascular involvement, and ischemic necrosis when host defenses fail to contain the organism. Inhalation represents the principal route of acquisition, with sporangiospores entering the respiratory tract and reaching the nasal passages, paranasal sinuses, or lower respiratory system. In individuals with intact immune function, the respiratory tract provides several effective mechanisms of protection. Mucociliary clearance removes inhaled particles from the airway, with expelled material subsequently eliminated through sneezing, coughing, or swallowing. Spores that bypass these mechanisms and reach the alveoli are exposed to pulmonary macrophages, which contribute to their recognition and elimination. These coordinated physical and cellular defenses generally prevent fungal germination and subsequent tissue invasion in immunocompetent individuals. However, when innate immune function is compromised, inhaled sporangiospores may evade clearance, undergo intracellular swelling, germinate, and develop into invasive hyphal structures. Neutrophils represent a major component of the innate immune response against Mucorales and are particularly important in preventing the transition from spore exposure to invasive fungal disease. They contribute to fungal killing through oxidative and nonoxidative mechanisms and help restrict hyphal proliferation within tissues. Consequently, patients with profound or prolonged neutropenia, as well as those with impaired neutrophil function, are highly susceptible to invasive mucormycosis. Once germination occurs, developing hyphae can proliferate rapidly and penetrate surrounding tissues. This process becomes particularly aggressive when metabolic conditions favor fungal growth and simultaneously weaken host immunity. Hyperglycemia is an important example because elevated glucose concentrations can support fungal proliferation while impairing several components of innate immune function. Metabolic acidosis further compromises host defenses and creates conditions that favor fungal survival. Experimental findings involving *Rhizopus arrhizus* indicate that ketone bodies can be metabolized through fungal ketone reductase activity, allowing the organism to persist and grow under acidic conditions. This capacity is particularly relevant to patients with diabetic ketoacidosis, in whom hyperglycemia and acidosis may act together to promote invasive infection [17].

The progression from tissue invasion to vascular invasion represents a central pathological feature of mucormycosis. Following germination, Mucorales hyphae invade blood vessels and produce extensive endothelial and vascular injury. Angioinvasion promotes thrombosis and obstruction of local blood flow, resulting in tissue hypoxia, ischemia, and infarction. Progressive vascular compromise ultimately produces extensive tissue necrosis, which can further reduce penetration of immune cells and antifungal agents into the infected region. This process helps explain the rapid progression of mucormycosis and the characteristic presence of necrotic tissue in advanced disease. In rhino-orbital-cerebral infection, for example, vascular invasion can permit extension from sinonasal tissues into the orbit and intracranial structures. In pulmonary disease, similar vascular and parenchymal invasion can contribute to pulmonary infarction, hemorrhage, and progressive respiratory deterioration. The capacity of Mucorales to invade vessels therefore represents a major determinant of disease severity and clinical progression. The metabolic environment of the host also has an important influence on fungal proliferation. Poorly controlled diabetes mellitus produces increased circulating glucose and can increase the availability of free iron, creating conditions that support rapid filamentous fungal growth. Iron is an essential nutrient for Mucorales, and increased availability of free iron can enhance fungal proliferation and

virulence. Iron overload states therefore represent an additional risk factor for invasive disease. Acidosis can intensify this susceptibility by impairing polymorphonuclear leukocyte chemotaxis, reducing phagocytic activity, and weakening the local inflammatory response.

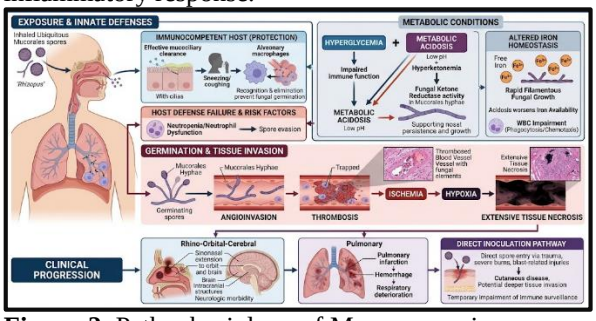

Figure-2: Pathophysiology of Mucormycosis.

The combined effects of hyperglycemia, acidosis, altered iron homeostasis, and impaired innate immunity create a physiological environment in which fungal germination and tissue invasion can proceed with limited host resistance. These mechanisms demonstrate why metabolic control is an important component of preventing progression in susceptible patients. Mucormycosis can also develop through direct inoculation rather than inhalation, particularly following substantial tissue injury. Individuals who are otherwise immunocompetent may experience temporary impairment of immune function after major trauma, extensive burns, or blast-related injuries. Tissue disruption can provide an entry site through which Mucorales spores gain direct access to damaged skin and subcutaneous tissues. When spores are introduced into injured tissue, local ischemia, necrosis, inflammation, and impaired immune surveillance can facilitate rapid fungal germination and hyphal extension. Localized immune suppression may therefore allow infection to develop even in the absence of chronic systemic immunodeficiency. The combination of direct fungal inoculation and compromised tissue defenses can result in aggressive cutaneous disease and, in severe cases, progression into deeper structures or dissemination.[17] Overall, the pathophysiology of mucormycosis reflects the interaction between fungal virulence, impaired innate immunity, metabolic abnormalities, iron availability, tissue injury, and vascular invasion, with angioinvasion and thrombosis ultimately driving much of the characteristic tissue destruction associated with this infection.[17]

Histopathology

Histopathological examination remains a central diagnostic method for mucormycosis and is widely regarded as the gold standard for establishing definitive tissue invasion by Mucorales. The diagnostic value of histology lies not only in demonstrating fungal elements but also in confirming their invasion of viable or necrotic tissue and vascular structures. The characteristic microscopic appearance consists of broad, ribbon-like fungal hyphae that generally measure approximately 5 to 10 µm in diameter. These hyphae are typically nonseptate or pauciseptate and demonstrate irregular branching, frequently approaching a right angle. Identification of fungal structures within tissue is particularly important because Mucorales may be present in environmental samples or colonized surfaces without necessarily producing invasive disease. Demonstration of invasion into the vascular wall or surrounding tissue therefore provides important evidence that the organism is contributing directly to the pathological process. Vascular invasion is especially significant because it is associated with thrombosis, ischemia, infarction, and tissue necrosis, which are characteristic consequences of invasive mucormycosis.[18] An important diagnostic challenge is the morphological distinction between Mucorales and other filamentous fungi, particularly *Aspergillus* species. Patients who develop mucormycosis frequently have severe immunosuppression and may simultaneously be susceptible to several invasive fungal pathogens. Coinfection can therefore occur, and accurate identification of the organism has direct therapeutic implications because antifungal susceptibility and treatment strategies differ between fungal pathogens. Mucorales typically demonstrate broad hyphae measuring approximately 5 to 10 µm, with absent or limited septation and irregular branching that commonly occurs at approximately 90°. In contrast, *Aspergillus* species generally produce narrower hyphae measuring approximately 3 to 5 µm, with regular septation and characteristic acute-angle branching of approximately 45°. Mucorales also tend to have a ribbon-like and irregular appearance, whereas *Aspergillus* hyphae are more uniform and tubular. Recognition of these morphological distinctions can assist pathologists and clinicians in differentiating the two infections and selecting appropriate antifungal management.[18]

Routine histological staining can demonstrate fungal structures, although the degree of visualization may vary according to tissue quality, fungal burden, and the characteristics of the specimen. Hematoxylin and eosin staining may reveal the general outline of fungal hyphae and their relationship with surrounding tissues, but internal fungal structures may not always be clearly defined. Special fungal stains can increase the visibility and diagnostic sensitivity of tissue examination. Grocott methenamine silver staining is particularly useful because it highlights fungal cell walls in black or dark brown, providing clear contrast against the surrounding tissue. Periodic acid-Schiff staining also facilitates visualization of fungal structures by emphasizing morphological features of the fungal cell wall and cytoplasmic components. The use of these stains can be particularly valuable when fungal elements are sparse, fragmented, or difficult to distinguish from surrounding necrotic material.

Histopathological interpretation should therefore integrate routine staining with appropriate special stains and the clinical context of the patient.[19][20][21] The approach to obtaining tissue for histopathological examination depends largely on the anatomical site involved and the patient's clinical condition. Because the paranasal sinuses represent a common site of mucormycosis, tissue sampling can often be performed through direct biopsy or endoscopic procedures. Endoscopic sampling may permit visualization of abnormal tissue while providing material for histological, microbiological, and molecular investigations. Pulmonary and gastrointestinal disease may require more invasive endoscopic procedures to obtain adequate tissue specimens, particularly when the suspected infection is located in regions that cannot be accessed through less invasive approaches. Interventional radiological techniques may also assist in obtaining tissue from selected anatomical locations when conventional biopsy is technically difficult or carries substantial procedural risk. The quality, depth, and anatomical representativeness of the specimen are important because superficial sampling may fail to capture areas of invasive disease. Importantly, a negative or inconclusive histopathological finding does not necessarily exclude mucormycosis. Fungal elements may be absent from a limited tissue specimen because of sampling error, extensive necrosis, low fungal burden, prior antifungal exposure, or the heterogeneous distribution of infection. Consequently, clinicians should interpret histological findings together with the patient's underlying risk factors, clinical manifestations, radiological findings, and microbiological investigations. The absence of recognizable hyphae should not automatically discourage consideration of mucormycosis when the clinical probability remains high. In some specimens, only yeast-like forms or poorly defined fungal structures may be observed, while the characteristic hyphal morphology may not be apparent. Clinical management may therefore need to proceed on the basis of the overall clinical picture when invasive mucormycosis remains strongly suspected, rather than waiting for unequivocal histological confirmation.[19][20][21] This principle is particularly relevant to nursing practice because nurses frequently participate in early recognition of deterioration, preparation for diagnostic procedures, specimen-related care, and continuous monitoring of patients receiving treatment for suspected invasive fungal infection.

History and Physical

Mucormycosis is characterized by a rapidly progressive clinical course and can involve almost any anatomical site. The distribution of infection depends on the route of fungal entry, the underlying host condition, and the degree of impairment of immune and metabolic defenses. The paranasal sinuses, orbit, lungs, gastrointestinal tract, skin, and central nervous system are among the most frequently affected sites, while disseminated disease may involve multiple organs simultaneously. Clinical manifestations can initially be nonspecific, particularly in patients with profound immunosuppression, and may therefore resemble bacterial infection, other invasive fungal diseases, or noninfectious inflammatory conditions. Progressive tissue invasion and vascular thrombosis can result in ischemia, infarction, necrosis, and rapid deterioration. A careful history should therefore identify predisposing conditions such as uncontrolled diabetes mellitus, diabetic ketoacidosis, neutropenia, hematological malignancy, transplantation, corticosteroid exposure, severe COVID-19 infection, trauma, burns, and other states associated with impaired host defense. Rhino-orbital-cerebral mucormycosis represents a major clinical presentation and is particularly associated with poorly controlled diabetes mellitus and renal transplantation. It may also develop in patients with neutropenia and in recipients of hematopoietic stem-cell or solid-organ transplantation. Infection generally begins following inhalation of Mucorales spores into the nasal cavity and paranasal sinuses. The organisms subsequently invade adjacent tissues and blood vessels, allowing the infection to extend toward the orbit and intracranial structures. Early manifestations may include persistent nasal obstruction or congestion, facial or sinus pain, nasal discharge, headache, and fever. As vascular invasion and tissue destruction progress, patients may develop facial swelling, facial numbness, visual disturbance, blurred vision, ptosis, proptosis, and impaired extraocular muscle function. Intranasal examination may demonstrate ulceration, purulent or bloody exudate, and areas of necrotic tissue. These lesions may progress within a short period because of angioinvasion and tissue infarction. Persistent sinonasal symptoms in an immunocompromised patient should therefore prompt urgent clinical evaluation and consideration of tissue biopsy when mucormycosis is suspected.

Pulmonary mucormycosis is particularly important among recipients of hematopoietic stem-cell transplantation and patients experiencing prolonged or profound neutropenia. It usually follows inhalation of infectious spores, with subsequent germination and invasion of pulmonary tissue and vascular structures. The clinical presentation is frequently nonspecific and may include persistent fever, cough, dyspnea, pleuritic or nonspecific chest pain, and hemoptysis. Radiological and clinical findings may overlap with other invasive pulmonary infections, making early recognition challenging. Pulmonary mucormycosis can produce cavitary lesions and areas of pulmonary tissue destruction and may clinically resemble pulmonary tuberculosis.

However, the progression of invasive mucormycosis can be considerably more rapid. Sinonasal disease may coexist with pulmonary involvement, particularly in patients with extensive invasive infection. Nurses caring for high-risk patients should monitor changes in respiratory status, oxygenation, temperature, cough, chest discomfort, and hemoptysis and should promptly communicate clinical deterioration to the treating team. Cutaneous and musculoskeletal mucormycosis may occur as either primary or secondary infection. Primary cutaneous disease generally follows direct inoculation of Mucorales into damaged skin and is particularly associated with burns, traumatic wounds, surgical sites, and other forms of tissue disruption. The initial lesion may resemble cellulitis, with localized erythema, induration, swelling, pain, or tenderness. As fungal invasion progresses, vascular thrombosis and tissue ischemia can produce discoloration followed by brown or black eschar and deep necrotizing infection. Extension into muscles, fascia, and bone may occur, and osteoarticular involvement can produce extensive lytic destruction at the site of inoculation. Secondary cutaneous mucormycosis results from dissemination of infection from another anatomical site, usually through hematogenous spread. In contrast to primary localized disease, secondary infection is more likely to produce multiple cutaneous lesions. Assessment of wound appearance, tissue viability, pain, drainage, and progression of necrosis is therefore important in patients with relevant risk factors.

Gastrointestinal mucormycosis is less common than rhino-orbital-cerebral, pulmonary, or cutaneous disease and is particularly associated with malnutrition and premature infancy. Infection may follow ingestion of Mucorales organisms and has been reported in association with contaminated dietary supplements,[22] nasogastric intubation, infected wooden tongue depressors, and contaminated food products, including fermented milk and other foods,[23] as well as dried bread products and alcohol derived from contaminated corn.[24] Clinical manifestations depend on the gastrointestinal region involved and the extent of tissue invasion. Abdominal pain, nausea, and vomiting may occur, while progressive vascular invasion can produce mucosal ischemia and necrotic ulceration. Gastrointestinal bleeding represents an important complication, and progression through the intestinal wall can result in perforation, peritonitis, and systemic deterioration. Because early gastrointestinal manifestations may be nonspecific, persistent gastrointestinal symptoms in a high-risk patient require careful assessment for evidence of invasive disease. Disseminated mucormycosis generally occurs in patients with severe immune impairment, particularly those with profound or prolonged neutropenia. The lungs commonly serve as the initial site of infection, although dissemination can originate from other anatomical locations. Hematogenous spread allows Mucorales to reach distant organs and produce multifocal tissue injury. Central nervous system involvement may develop through direct extension from infected paranasal sinuses, while renal involvement is more commonly associated with hematogenous dissemination. The clinical manifestations of disseminated disease depend on the organs affected and may include respiratory deterioration, neurological abnormalities, renal dysfunction, fever, cutaneous lesions, abdominal symptoms, or other organ-specific findings. Because disseminated mucormycosis can progress rapidly and involve multiple organ systems, comprehensive physical assessment and repeated monitoring are essential. Early recognition of new neurological, respiratory, renal, gastrointestinal, or cutaneous abnormalities can support timely diagnostic investigation and multidisciplinary intervention.

Evaluation and Diagnosis:

The evaluation of mucormycosis requires a high level of clinical suspicion, rapid diagnostic investigation, and integration of histopathological, microbiological, molecular, and radiological findings. Because Mucorales infections can progress rapidly through vascular invasion and tissue necrosis, diagnostic assessment should proceed without unnecessary delay in patients who have compatible clinical manifestations and recognized predisposing conditions. Definitive diagnosis generally depends on demonstrating Mucorales in infected tissue through histopathological examination and, when possible, microbiological culture. Culture remains useful for identifying the causative organism and supporting species-level characterization, but its diagnostic sensitivity is limited and is estimated to be below 50%.[25] Consequently, a negative culture does not exclude mucormycosis, particularly when the clinical and radiological findings strongly suggest invasive fungal disease. The diagnosis is strengthened when compatible imaging abnormalities and clinical manifestations occur together with histopathological or microbiological evidence of Mucorales invasion. Conventional serum fungal biomarkers have limited diagnostic value in mucormycosis. The serum (1,3)-β-D-glucan assay and the *Aspergillus* galactomannan assay are not considered reliable diagnostic tests for Mucorales infection because these organisms lack the relevant fungal cell-wall components detected by these assays. Consequently, negative results from these biomarkers should not be interpreted as evidence against mucormycosis when clinical suspicion remains high. This distinction is important because patients at risk for mucormycosis frequently undergo evaluation for invasive fungal infection using conventional biomarkers, potentially creating false reassurance when the results are negative. Molecular diagnostic approaches are increasingly being investigated to address limitations associated

with conventional culture. Polymerase chain reaction-based assays targeting multiple Mucorales species can detect fungal DNA in blood, tissue, and other biological specimens and may provide useful diagnostic information even when conventional cultures remain negative.[26] These approaches may be particularly valuable in immunocompromised patients in whom early diagnosis is essential.

Next-generation sequencing represents another emerging approach with potential application in the diagnosis of invasive fungal infections. By detecting and characterizing microbial genetic material within clinical specimens, sequencing-based methods may facilitate identification of infectious organisms when conventional culture or targeted testing provides inconclusive results.[27] Although the availability, standardization, interpretation, and clinical integration of these molecular technologies continue to evolve, they may complement established diagnostic methods in selected patients. Importantly, molecular testing should not be considered a replacement for appropriate tissue sampling when invasive mucormycosis is suspected. Histopathological assessment remains essential because it can demonstrate the anatomical relationship between fungal elements and damaged or viable tissue and can provide direct evidence of invasive disease. Imaging has a central role in identifying suspected sites of infection, determining the anatomical extent of disease, and guiding subsequent diagnostic procedures. In patients with suspected rhino-orbital mucormycosis, computed tomography of the paranasal sinuses is commonly used as an initial imaging examination. CT may demonstrate mucosal thickening, frequently involving the maxillary sinuses, together with extension into surrounding structures. Disease may involve adjacent bone, the nasolacrimal ducts, the lacrimal sac, and surrounding soft tissues. Magnetic resonance imaging provides greater sensitivity for evaluating soft-tissue involvement and intracranial extension and is particularly valuable when there is concern for orbital, cerebral, or deep facial involvement. When imaging findings raise suspicion for invasive fungal sinusitis, prompt endoscopic assessment is warranted. Endoscopy allows direct visualization of abnormal mucosa and can facilitate targeted tissue acquisition for histopathological and microbiological evaluation. Mucosal necrosis is an important clinical finding that increases suspicion for invasive mucormycosis, particularly in patients with compatible risk factors. Necrotic tissue should prompt consideration of biopsy because histopathological examination may demonstrate the characteristic broad, ribbon-like, pauciseptate or nonseptate hyphae with irregular right-angle branching associated with Mucorales. Demonstration of tissue invasion provides important evidence that the fungal organism represents invasive disease rather than superficial colonization. In suspected pulmonary mucormycosis, chest CT may reveal a broad range of abnormalities, including multifocal pulmonary nodules, mass-like consolidations, pleural effusions, and ground-glass opacities. Larger lesions, particularly those exceeding 4 cm, may increase clinical suspicion. Radiological signs associated with pulmonary necrosis, including the halo sign, reverse halo sign, cavitation, and air crescent sign, can provide additional diagnostic clues. However, these findings are not individually specific for mucormycosis and must be interpreted within the patient's clinical context.

Bronchoalveolar lavage may demonstrate broad, nonseptate fungal hyphae and can contribute to the diagnostic assessment of pulmonary disease. Nevertheless, lavage findings alone may not establish definitive invasive mucormycosis because the detection of fungal elements does not always demonstrate tissue invasion. Transbronchial or percutaneous biopsy may therefore be required to obtain tissue for histopathological confirmation when clinically appropriate. For suspected gastrointestinal mucormycosis, endoscopic evaluation using procedures such as esophagogastroduodenoscopy or colonoscopy can identify ulcerative, necrotic, or otherwise abnormal mucosal lesions. Biopsy of these lesions may demonstrate characteristic Mucorales hyphae and provide material for culture and molecular testing. Similarly, patients with suspected cutaneous or osteoarticular mucormycosis require tissue biopsy from the involved area to establish the diagnosis and determine the depth and extent of invasion.

Treatment / Management

The management of mucormycosis requires an immediate, coordinated, and multimodal therapeutic strategy because the infection can progress rapidly through angioinvasion, vascular thrombosis, tissue ischemia, and extensive necrosis. Successful treatment depends on the simultaneous use of antifungal therapy, surgical source control, correction of reversible predisposing conditions, and restoration of immune function whenever feasible. Treatment should begin as soon as mucormycosis is clinically suspected rather than being postponed until microbiological or histopathological confirmation when the probability of invasive disease is high. Delays in antifungal treatment or surgical intervention can permit continued fungal proliferation and irreversible tissue destruction. Management therefore requires close collaboration among infectious disease specialists, surgeons, intensivists, pharmacists, microbiologists, radiologists, and nurses. Nursing care is integral to this approach through continuous monitoring, medication administration, assessment of treatment response, wound care, recognition of adverse effects, and coordination of multidisciplinary interventions.

Correction of the underlying predisposing condition represents a fundamental component of treatment. In patients with diabetes mellitus, prompt control of hyperglycemia and correction of metabolic abnormalities, particularly diabetic ketoacidosis, are important because hyperglycemia and acidosis promote fungal growth while impairing host immune defenses. When clinically appropriate, reduction of immunosuppressive therapy should be considered, although this decision must be balanced against the risk of rejection in transplant recipients or complications associated with the underlying disease. Patients with hematological malignancies may require treatment directed toward the underlying neoplasm and restoration of neutrophil function or recovery of neutrophil counts whenever possible. Reversal of the factors that enabled fungal invasion is important because antifungal therapy alone may be insufficient when profound immunosuppression or metabolic dysfunction persists. Prognosis remains particularly poor when meaningful immune recovery cannot be achieved despite appropriate antifungal treatment and surgical source control.

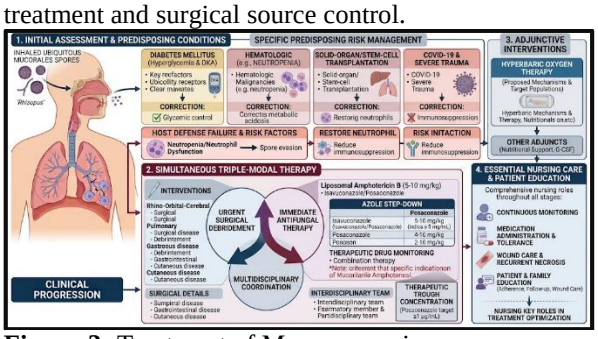

Figure-3: Treatment of Mucormycosis.

Surgical intervention represents another central component of management because necrotic tissue has poor vascular supply and therefore limits penetration of both immune cells and antifungal agents. Urgent and extensive debridement should be considered when anatomically feasible to remove infected and devitalized tissue and prevent continued local extension. The extent of surgery depends on the anatomical site, severity of invasion, and involvement of adjacent structures. In extensive rhino-orbital disease, surgical procedures may range from endoscopic debridement to more radical facial resections when required to achieve adequate source control. Pulmonary disease may necessitate procedures such as lobectomy or pneumonectomy in selected patients with localized but extensive tissue destruction. Gastrointestinal involvement may require bowel resection when necrosis, perforation, or extensive invasive disease is present. Cutaneous and musculoskeletal disease may require repeated excision of necrotic tissue and, in severe cases, extensive reconstructive or ablative procedures. Similar to other necrotizing infections, repeated surgical assessment and serial debridement may be necessary because disease can extend beyond the initially recognized margins.[28] Antifungal therapy should be initiated immediately when mucormycosis is suspected. Liposomal amphotericin B is generally considered the preferred initial antifungal agent in most patients. A commonly recommended starting dose is 5 mg/kg administered intravenously once daily. Higher dosing is generally considered in selected high-risk circumstances, including suspected central nervous system involvement and certain transplant recipients, where a dose of approximately 10 mg/kg daily may be initiated. Dose escalation to 10 mg/kg daily can also be considered when there is inadequate clinical response after approximately one week of treatment.[28] The choice and intensity of antifungal treatment should account for disease severity, anatomical involvement, renal function, concomitant medications, transplant status, and the patient's overall clinical condition. Nurses play an important role in monitoring intravenous administration, renal function, electrolyte abnormalities, infusion-related reactions, fluid balance, and other potential complications associated with amphotericin B therapy.

Once clinical improvement has been achieved and the patient's condition permits transition from intensive treatment, oral azole therapy can be used as step-down or maintenance treatment. Isavuconazole and posaconazole are commonly used options for prolonged therapy.[28] These agents can provide a practical strategy for continuing antifungal treatment after stabilization and may reduce the need for prolonged intravenous therapy. However, treatment selection should consider drug interactions, gastrointestinal absorption, hepatic function, comorbidities, and the potential for breakthrough infection. Posaconazole requires particular attention to therapeutic drug exposure because inadequate concentrations may compromise treatment effectiveness. Therapeutic drug monitoring is recommended for patients receiving posaconazole, with a target serum trough concentration of at least 1 µg/mL. Routine therapeutic drug monitoring is generally not required for isavuconazole, although measurement may be considered when treatment failure, inadequate exposure, or breakthrough infection is suspected.[29] The use of combination antifungal therapy remains an area of clinical uncertainty. In vitro experiments, animal models, and retrospective human studies have suggested that combining antifungal agents may provide benefit in selected patients with severe mucormycosis. However, large randomized controlled trials demonstrating a clear benefit of routine combination therapy remain lacking. Some experts therefore consider combination therapy in patients with disseminated infection, severe immunocompromise, or inadequate response to high-dose liposomal amphotericin B. Combination treatment may also be considered when there is no satisfactory response after approximately one week of liposomal amphotericin B at 10 mg/kg daily. When

combination therapy is selected, posaconazole or isavuconazole may be administered together with amphotericin B.[29] Decisions regarding combination treatment should therefore be individualized and should account for disease severity, anatomical extent, immune status, treatment response, drug interactions, and the risks associated with additional antifungal exposure.

The duration of treatment is not defined by a single fixed interval and should be individualized according to the patient's clinical and radiological response and the extent to which predisposing factors have been corrected. Therapy commonly continues for several weeks to several months and should generally persist until there is clear clinical improvement, evidence of radiological stabilization or resolution, and adequate reversal of the underlying immunological or metabolic risk factors. Persistent immunosuppression may require prolonged treatment or secondary prophylaxis after completion of the initial therapeutic course. Posaconazole is recommended for primary prophylaxis in selected high-risk patients with prolonged neutropenia and in hematopoietic stem-cell transplant recipients with graft-versus-host disease. Isavuconazole may provide an alternative when posaconazole cannot be tolerated or is contraindicated. Secondary prophylaxis should generally continue in patients who remain immunosuppressed following treatment of mucormycosis, with duration determined by the persistence and severity of the underlying risk factor. Hyperbaric oxygen therapy has also been investigated as an adjunctive intervention, particularly when combined with aggressive surgical debridement. Reported experience has associated hyperbaric oxygen therapy with lower mortality in selected patients.[30] The proposed therapeutic mechanisms include correction of tissue hypoxia, improvement of oxygen delivery to compromised tissues, promotion of healing and vasculogenesis, and possible enhancement of oxygen-dependent antifungal mechanisms. Increased tissue oxygenation may also increase the generation of oxygen-derived free radicals that can contribute to fungal killing. Despite these potential mechanisms, evidence supporting routine use remains limited because much of the available evidence derives from case reports and small case series rather than large controlled trials. Reported benefits have been most frequently described in patients with diabetes-associated mucormycosis and those with cutaneous or musculoskeletal involvement.[31] Hyperbaric oxygen therapy should therefore be considered as an adjunct to, rather than a replacement for, antifungal treatment, surgical debridement, and correction of underlying risk factors.

Nursing management extends throughout every stage of treatment. Nurses should monitor temperature, hemodynamic status, respiratory function, neurological status, wound characteristics, pain, nutritional status, fluid balance, and relevant laboratory parameters. Patients receiving amphotericin B require close monitoring of renal function and serum electrolytes, while patients receiving azole therapy require assessment of hepatic function, drug interactions, adherence, and treatment tolerance. Surgical wounds require repeated assessment for recurrent necrosis, drainage, bleeding, and evidence of infection. Patients with rhino-orbital disease require careful monitoring of visual changes, ocular movement, facial sensation, and neurological status. Patients with pulmonary disease require assessment of oxygenation, respiratory effort, hemoptysis, and progression of respiratory symptoms. Education should address medication adherence, recognition of worsening symptoms, glycemic control when applicable, wound care, follow-up imaging, and the importance of completing prolonged antifungal therapy. Through systematic assessment and early recognition of deterioration, nursing care supports the broader multidisciplinary strategy required to control this rapidly progressive invasive fungal infection.

Conclusion:

Mucormycosis represents a severe invasive fungal infection in which rapid fungal proliferation and angioinvasion can produce vascular thrombosis, tissue infarction, and extensive necrosis. The disease predominantly affects patients with impaired immune or metabolic defenses, particularly those with poorly controlled diabetes, hematological malignancies, transplantation, neutropenia, or other predisposing conditions. Accurate diagnosis requires integration of clinical assessment, imaging, histopathology, microbiological culture, and emerging molecular techniques. Management should begin promptly and combine appropriate antifungal therapy with surgical debridement and correction of reversible underlying risk factors. Liposomal amphotericin B remains central to initial treatment, while isavuconazole or posaconazole may support subsequent therapy. Nursing professionals have an important role in early recognition, continuous assessment, treatment monitoring, wound management, patient education, and multidisciplinary coordination. Improving awareness of risk factors and early clinical manifestations may facilitate timely intervention and contribute to better outcomes in affected patients.

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