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Acute Invasive Fungal Rhinosinusitis: An Integrated Review of ENT, Internal Medicine and Radiology

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Abstract
Acute invasive fungal rhinosinusitis (AIFR) is a rapidly progressive and potentially fatal infection characterized by fungal invasion of sinonasal tissue and blood vessels, predominantly affecting patients with impaired host defenses. Diabetes mellitus, diabetic ketoacidosis, hematological malignancy, prolonged neutropenia, transplantation, and corticosteroid-mediated immunosuppression are major predisposing conditions. The disease requires simultaneous contributions from otorhinolaryngology, internal medicine, infectious diseases, hematology, endocrinology, and radiology. ENT provides recognition of suspicious mucosa, urgent tissue diagnosis, and serial surgical debridement. Medicine identifies and reverses systemic conditions that permit fungal invasion, initiates organism-appropriate systemic antifungal therapy, and manages metabolic and hematological abnormalities. Radiology defines the true extent of disease because vascular and perivascular spread may extend beyond the paranasal sinuses before obvious osseous destruction develops. CT is useful for sinonasal anatomy and bone; contrast-enhanced MRI is particularly valuable for devitalized mucosa, perisinus spread, orbital involvement, cavernous sinus disease, vascular complications, and intracranial extension. Literature from 2011-2016 supports a combined strategy of rapid recognition, tissue confirmation, aggressive surgical debridement, immediate systemic antifungal therapy, reversal of predisposing abnormalities, and imaging-guided assessment of disease extent.
Keywords: acute invasive fungal rhinosinusitis; invasive fungal sinusitis; mucormycosis; ENT; internal medicine; radiology; MRI; CT; diabetes mellitus; neutropenia; amphotericin B

1. Introduction
Fungal disease of the nose and paranasal sinuses encompasses a spectrum extending from harmless colonization and fungus balls to allergic fungal rhinosinusitis and invasive disease. Acute invasive

fungal rhinosinusitis represents the most rapidly progressive form and is defined by fungal invasion of sinonasal mucosa, submucosal tissue and, characteristically, vascular structures. Its fulminant course distinguishes it from chronic invasive and granulomatous fungal rhinosinusitis and makes delays in diagnosis particularly dangerous [1]. A systematic review incorporating 807 patients demonstrated an overall survival of approximately 50%, emphasizing that AIFR remains a life-threatening disease even when modern surgical and antifungal treatments are available [2].

The disease is inherently multidisciplinary. In a 15-year institutional series of 41 patients, most affected individuals had serious underlying immunocompromising conditions, and clinical variables such as profound neutropenia and necrosis of the middle turbinate were useful indicators of disease. These observations reinforce the principle that AIFR cannot be managed by sinus surgery alone: ENT findings must be interpreted in the context of systemic immune status, while radiological assessment is necessary to establish how far disease has progressed beyond what is visible endoscopically [3].

This review examines AIFR through three interconnected perspectives: ENT, which provides direct examination, biopsy and surgical source control; Internal Medicine, which determines susceptibility and systemic antifungal management; and Radiology, which maps anatomical and vascular spread. References are restricted to PubMed-indexed literature published between 2011 and 2016.

2. Disease Spectrum and Biological Basis

AIFR is most frequently associated with fungi belonging to the order Mucorales or with Aspergillus species. Mucormycosis has particular clinical importance among patients with diabetes, hematological malignancy, transplantation and other forms of immunosuppression. The anatomic presentation is influenced by the host: rhino-orbital-cerebral disease is especially associated with diabetes, whereas pulmonary or disseminated disease may be more prominent in profoundly neutropenic or transplant populations [1,4].

The biological hallmark of mucormycosis is angioinvasion. Following adherence to and invasion of endothelial cells, fungal hyphae penetrate vessel walls, resulting in endothelial injury, thrombosis, tissue ischemia and coagulative necrosis. This vascular mechanism explains black or necrotic nasal mucosa seen by ENT, nonenhancing tissue on MRI, cranial neuropathies caused by ischemic or perineural extension, and the difficulty of delivering systemic antifungal agents into already infarcted tissue [5].

Hyperglycemia, acidosis, iron availability and impaired phagocyte function create a favorable environment for Mucorales. Diabetic ketoacidosis is particularly important because hyperglycemia and acidemia impair innate immune function while increasing iron availability. Prolonged neutropenia deprives the host of a major cellular defense against filamentous fungi, directly linking systemic disease to local sinonasal invasion [5].

Among patients with hematological disorders, prolonged neutropenia and active leukemia are important determinants of susceptibility and outcome. A 15-year experience in hematological malignancy showed that invasive fungal sinusitis was especially associated with acute myeloid leukemia and prolonged neutropenia; surgical debridement was independently associated with a more favorable prognosis [6].

Geography also influences the clinical population. Indian literature has emphasized the prominent association between mucormycosis and uncontrolled diabetes and the importance of rhino-orbito-cerebral disease. Regional differences in causative Mucorales species suggest that host factors, climate and local epidemiology should inform diagnostic probability [7].

3. Internal Medicine Perspective: Identifying the High-Risk Host

3.1 Diabetes mellitus and metabolic disease

Uncontrolled diabetes is one of the most immediately reversible risk factors. Hyperglycemia may coexist with ketoacidosis, dehydration, electrolyte abnormalities, renal dysfunction and secondary infection, all of which complicate antifungal therapy and surgery. Prognostic studies indicate that correction of reversible predisposing abnormalities is closely related to outcome, illustrating why treatment must extend beyond the infected sinus [8].

The physician should determine serum glucose, acid-base status, renal function, electrolytes and the presence of diabetic ketoacidosis at presentation. Intravenous fluid resuscitation, insulin therapy and correction of electrolyte abnormalities should proceed simultaneously with ENT and radiological investigation rather than waiting for definitive fungal culture because the metabolic abnormality is part of the pathogenetic process [5,8].

3.2 Hematological malignancy and neutropenia

Patients with leukemia, lymphoma, chemotherapy-associated neutropenia and hematopoietic stem-cell transplantation form another major risk group. Persistent fever, facial pain, nasal symptoms or orbital symptoms in a profoundly neutropenic patient should prompt early consideration of invasive fungal disease even when the nasal examination initially appears subtle. International recommendations for hematological malignancy emphasize tissue demonstration or microbiological evidence because a reliable circulating biomarker for mucormycosis was not available during the reviewed period [6,9].

Reversal of neutropenia, where feasible, is an important treatment component. Granulocyte recovery may improve the host's capacity to control infection, whereas persistent refractory leukemia or prolonged neutropenia adversely affects outcome. The physician, hematologist and ENT surgeon must coordinate chemotherapy, growth-factor support, transfusion requirements and surgical procedures [6,9].

3.3 Corticosteroids, transplantation and other immunocompromised states

Systemic corticosteroid exposure, solid-organ transplantation and other causes of impaired cellular immunity also increase susceptibility. Corticosteroids can aggravate hyperglycemia while impairing neutrophil and macrophage function. When possible, reduction of unnecessary immunosuppression should form part of the therapeutic strategy, balanced against the underlying condition [4,10].

The ESCMID/ECMM mucormycosis guideline emphasized correction of predisposing conditions together with surgery and antifungal therapy, including control of hyperglycemia and ketoacidosis, minimization of glucocorticoid exposure when clinically possible and management of persistent neutropenia [10].

4. Internal Medicine Perspective: Systemic Antifungal Therapy

Systemic antifungal therapy should begin promptly when invasive mucormycosis is strongly suspected and should not be unnecessarily delayed while awaiting final culture results. Liposomal amphotericin B or another lipid formulation was recommended as first-line treatment during 2011-2016 because Mucorales are intrinsically resistant to several antifungal drugs used for other mould infections. Lipid formulations are generally preferred to conventional amphotericin B deoxycholate because of a more favorable toxicity profile [9,10].

Renal function requires close attention throughout amphotericin therapy. Serum creatinine, potassium and magnesium should be monitored because amphotericin can produce nephrotoxicity, hypokalemia and hypomagnesemia, particularly relevant in patients with diabetic ketoacidosis, sepsis, hematological malignancy or perioperative instability [10].

Posaconazole had an established role as salvage therapy during this period, particularly when amphotericin B was ineffective or poorly tolerated. Treatment duration is individualized and generally continues until clinical and radiological resolution and adequate reversal of the underlying immunological or metabolic defect [10].

By 2016, isavuconazole had emerged as an additional option. In the VITAL study and matched case-control analysis, isavuconazole showed activity against mucormycosis with outcomes broadly comparable to amphotericin B-treated matched controls, providing an alternative when standard therapy was unsuitable while reinforcing the need for surgical source control whenever disease was resectable [11].

5. ENT Perspective: Clinical Recognition

The earliest ENT manifestations may be nonspecific. Patients can present with nasal obstruction, discharge, facial pain, headache or fever and may initially resemble routine bacterial rhinosinusitis. As disease progresses, facial numbness, palatal abnormalities, epistaxis, visual disturbance, ophthalmoplegia, cranial neuropathy or altered mental status may develop. A 2016 series documented headache and facial pain, facial paresthesia and ophthalmoplegia among important presenting features, with frequent extension to the pterygopalatine fossa and orbit [12].

Because early symptoms are nonspecific, risk status changes the significance of the ENT examination. Mild unilateral congestion in an immunocompetent patient is fundamentally different from the same symptom in a neutropenic patient receiving chemotherapy. The otorhinolaryngologist should integrate medical history, neutrophil count, glucose control, corticosteroid exposure and transplantation history into the decision to perform urgent nasal endoscopy and biopsy [3,6].

6. Nasal Endoscopy and Tissue Diagnosis

Nasal endoscopy provides direct assessment of mucosal viability. Early disease may manifest as edema, discoloration or subtle abnormalities, while advanced vascular invasion may result in pale, dusky, gray or black necrotic tissue. Middle turbinate and septal abnormalities were strongly associated with AIFR in the 15-year series by Payne and colleagues, with middle turbinate necrosis showing particularly high specificity [3].

The absence of dramatic black eschar should not reassure the clinician when systemic risk is high. Tissue necrosis is a relatively advanced manifestation of vascular compromise, and fungal invasion may already be present before grossly black tissue appears. Suspicious mucosa should be biopsied, preferably including tissue from the edge of viable and abnormal regions [3,12].

Histopathological confirmation demonstrates fungal elements invading tissue, vascular structures or both. Histological classification depends fundamentally on whether fungi remain extracellular/noninvasive or invade viable sinonasal tissue, and special stains such as periodic acid-Schiff and Gomori methenamine silver may improve visualization [13].

Mucorales typically produce broad, ribbon-like, pauciseptate hyphae with irregular branching, whereas Aspergillus typically exhibits narrower septate hyphae with more regular acute-angle branching.

Morphological distinction is clinically useful but should be supplemented by microbiological culture or molecular identification when feasible because necrotic tissue can distort morphology [13].

7. ENT Perspective: Surgical Debridement

The surgical objective is to remove devitalized and invaded tissue until viable bleeding margins are encountered, reduce fungal burden and improve the ability of systemic antifungal agents and host immune mechanisms to act on remaining disease. Endoscopic endonasal techniques allow extensive access to the nasal cavity and paranasal sinuses and can be repeated when serial examinations reveal additional necrosis [2,12].

AIFR is dynamic; therefore, a single operation may not provide definitive source control. In the 2016 series by Bakhshaee and colleagues, patients underwent serial debridements together with systemic antifungal therapy and management of underlying disease. Repeated endoscopic examination is consequently integral to postoperative care [12].

The survival literature supports surgical treatment. Turner and colleagues found surgical resection associated with improved survival in their systematic review, while Chen and colleagues identified surgical debridement as a favorable prognostic factor in hematological malignancy [2,6]. These findings support timely removal of surgically accessible invasive disease rather than indiscriminate radicality.

Orbital exenteration is one of the most difficult multidisciplinary decisions. Orbital involvement does not automatically mandate exenteration, and the survival benefit of routine radical orbital surgery remains uncertain. Decisions should consider visual function, orbital necrosis, apex involvement, response to systemic therapy, intracranial spread and the patient's overall prognosis [2].

8. Radiology Perspective: Why Imaging Is Essential

A defining feature of AIFR is that disease can extend through vessels, nerves and soft-tissue planes rather than simply eroding bone sequentially. A patient can therefore have clinically significant extrasinus invasion while osseous sinus walls remain relatively intact, making radiological assessment critical for detecting disease not visible through routine endoscopy [14].

CT and MRI are complementary. CT is fast and excellent for sinonasal anatomy, sinus opacification, bone erosion and surgical landmarks. MRI provides superior soft-tissue contrast and is generally more sensitive for early perisinus, orbital, cavernous sinus, vascular and intracranial involvement [14].

In a comparative study of CT and MRI, MRI was more sensitive than CT for detecting AIFR while both had similar specificity. Extrasinus invasion on MRI was especially useful, clinically important because the survival advantage of early treatment depends on identifying invasion before extensive complications occur [14].

9. CT Findings

CT may demonstrate unilateral or bilateral mucosal thickening, nasal cavity opacification, sinus opacification and soft-tissue infiltration. Areas of high attenuation can occur in fungal sinus disease, although attenuation characteristics alone cannot reliably distinguish invasive fungal infection from noninvasive fungal disease or other causes of sinus opacification [15,16].

One of the most important lessons for radiologists and ENT surgeons is that bone destruction is not required for early extrasinus spread. Angioinvasion and extension along soft-tissue or neurovascular pathways can occur through apparently intact sinus walls. Reliance on bony erosion as the principal radiological marker can therefore delay diagnosis [14,17].

A comprehensive CT analysis published in 2015 identified several findings associated with AIFR and developed a diagnostic model using variables such as periantral fat abnormalities, bone dehiscence, orbital invasion, septal ulceration and nasolacrimal duct involvement. Periantral and adjacent soft-tissue abnormalities are particularly important because they may represent early extension before major osseous destruction is visible [18].

For the ENT surgeon, CT remains valuable for surgical mapping. The relationship of disease to the lamina papyracea, skull base, sphenoid sinus, carotid canal, pterygopalatine fossa, palate and orbital walls can influence the extent and sequence of endoscopic debridement [16,18].

10. MRI Findings

Contrast-enhanced MRI is particularly valuable because fungal angioinvasion causes thrombosis, ischemia and tissue infarction. Devitalized tissue may demonstrate reduced or absent contrast enhancement, providing a radiological correlate of necrotic mucosa observed during nasal endoscopy [14,17].

Seo and colleagues investigated cervicofacial tissue infarction in AIFR and found nonenhancing infarcted tissue in a substantial proportion of patients. Extrasinonasal infarction involving areas such as the orbit and infratemporal fossa was associated with very poor outcomes. Such infarction could occur while bony walls remained preserved on CT [17].

MRI should evaluate the orbit carefully for inflammatory change, infarction, extraocular muscle involvement, optic nerve or orbital-apex involvement and extension through the orbital fissures. Ophthalmoplegia, visual loss or severe retro-orbital pain should therefore prompt urgent MRI assessment when feasible [12,17].

The cavernous sinuses and internal carotid arteries are also critical. Fungal invasion can produce cavernous sinus thrombosis, arterial narrowing, thrombosis, aneurysmal complications or cerebral infarction. Intracranial extension may involve meninges, brain parenchyma or vascular structures and is consistently associated with adverse prognosis [2,14].

11. Radiology as a Prognostic Tool

Imaging is not merely diagnostic; it can help estimate prognosis. The 2013 systematic survival analysis demonstrated that intracranial involvement was an independent adverse prognostic variable. Identifying intracranial spread therefore changes staging, surgical planning and prognostic counseling [2].

Gadolinium-enhanced MRI has also been evaluated for prognostic and surgical purposes. Kim and colleagues demonstrated that areas showing loss of contrast enhancement represented ischemic or devitalized tissue and suggested that residual extrasinonasal nonenhancing lesions after surgery were associated with poor survival. Their analysis also showed that uncontrolled blood glucose and active hematological disease contributed to adverse outcomes, directly linking radiology, systemic medicine and surgery [19].

Radiological reporting should therefore move beyond simply stating 'sinusitis present.' The report should describe the nasal cavity, individual paranasal sinuses, premaxillary and retroantral fat, pterygopalatine fossa, masticator space, palate, orbit, orbital apex, cavernous sinuses, skull base, intracranial compartment and major vascular structures [14,18,19].

12. The ENT-Medicine-Radiology Interface

AIFR should be considered a vascular invasive infection occurring in a susceptible host, not simply an aggressive sinusitis. The internist establishes why invasion has occurred, the ENT surgeon determines what abnormal tissue looks like directly and obtains diagnostic material, and the radiologist determines where invasion has already travelled beyond the visible mucosal surface [3,5,14].

Consider a neutropenic patient with leukemia and mild unilateral nasal discomfort. Internal medicine identifies profound neutropenia and high fungal risk. Endoscopy may reveal subtle middle turbinate discoloration, prompting biopsy. CT may show mild unilateral mucosal disease but subtle periantral fat stranding, while MRI reveals nonenhancing turbinate tissue and early pterygopalatine-fossa extension. Together these observations establish an emergency requiring antifungal therapy and surgery [3,6,18].

Similarly, a patient with uncontrolled diabetes and ketoacidosis may present with facial pain and ophthalmoplegia. Medicine must correct metabolic derangement and begin antifungal therapy; ENT must assess and debride sinonasal necrosis; and MRI must establish whether the orbital apex, cavernous sinus or intracranial compartment has been invaded. Management becomes unsafe if these steps are performed sequentially rather than in parallel [5,8,12,19].

13. Integrated Therapeutic Strategy

The consistent therapeutic message across 2011-2016 is that no single intervention is sufficient. Reviews of treatment outcomes emphasize the combination of early systemic antifungal therapy, surgical debridement and reversal of the underlying predisposing condition [10,20].

Once AIFR is strongly suspected, ENT assessment, contrast-enhanced imaging where appropriate, antifungal initiation and medical correction of systemic abnormalities should proceed urgently and concurrently. Tissue should be obtained as soon as practical, but treatment should not be unnecessarily delayed in a critically ill high-risk patient while definitive culture results are pending [9,10].

Systemic antifungal treatment and surgery interact biologically. Antifungal drugs may penetrate poorly into infarcted tissue because fungal vascular invasion has compromised blood supply. Surgical debridement removes necrotic reservoirs, while systemic therapy treats microscopic or anatomically unresectable disease [5,20].

Underlying disease reversibility is similarly important. A patient whose diabetic ketoacidosis can be rapidly corrected may have a different prognosis from a patient with refractory leukemia and persistent profound neutropenia. Outcomes should therefore be interpreted within the host context rather than attributed exclusively to the extent of sinus surgery [2,8,19].

14. Practical Multidisciplinary Algorithm

Stage 1: Recognize the high-risk patient. Suspect AIFR in patients with diabetes, ketoacidosis, hematological malignancy, profound or prolonged neutropenia, transplantation or significant immunosuppression who develop new sinonasal, facial, orbital or neurological symptoms [4,6,9].

Stage 2: Initiate simultaneous assessment. Obtain urgent ENT examination with nasal endoscopy while assessing complete blood count, neutrophil count, glucose, renal function, electrolytes and acid-base status. Do not allow routine laboratory work to delay direct ENT evaluation [3,8].

Stage 3: Obtain imaging. CT can rapidly define sinus anatomy, soft-tissue abnormality and bone. MRI with contrast should be strongly considered when AIFR is suspected, particularly with orbital, cranial-nerve, vascular or neurological findings or when CT underestimates suspected extrasinus disease [14,18,19].

Stage 4: Obtain tissue. Biopsy abnormal or suspicious mucosa for histopathology and fungal culture. The aim is to demonstrate true tissue invasion rather than simply the presence of fungi within sinus material [13].

Stage 5: Begin systemic antifungal therapy. When mucormycosis is suspected, initiate an appropriate amphotericin B lipid formulation promptly, adjusting subsequent therapy according to organism, response, toxicity and specialist infectious-disease guidance [9,10].

Stage 6: Correct the host abnormality. Treat ketoacidosis and hyperglycemia, correct severe metabolic disturbances, reduce unnecessary immunosuppression when possible and facilitate neutrophil recovery where clinically appropriate [5,10].

Stage 7: Surgically debride invasive disease. Remove necrotic and invaded surgically accessible tissue. Repeat endoscopy and further debridement may be required because progression can continue after the first operation [2,12].

Stage 8: Reassess anatomically and systemically. Persistent fever, new cranial neuropathy, visual change, recurrent endoscopic necrosis or failure to improve should trigger repeat endoscopy, biopsy and/or imaging. Residual nonenhancing extrasinonasal tissue on MRI should raise particular concern [19].

15. Multidisciplinary Correlation Table

Clinical finding	ENT interpretation	Internal Medicine implication	Radiological implication
Middle turbinate discoloration/necrosis	Possible invasive mucosal disease; urgent biopsy	Consider systemic fungal infection in a high-risk host	Search for nonenhancement and extrasinus extension
Diabetic ketoacidosis	Lower threshold for urgent endoscopy	Correct glucose, acidosis, fluids and electrolytes	Assess for rhino-orbital-cerebral spread
Profound neutropenia	Minor nasal abnormalities become highly significant	Consider growth-factor strategy and hematological recovery	Imaging may detect disease before gross necrosis appears
Periantral fat infiltration on CT	Disease may extend beyond visible sinus	Escalates urgency of systemic therapy	Early extrasinus marker even without bone destruction

Loss of enhancement on MRI	Suggests tissue ischemia/necrosis; may influence surgical boundaries	Indicates advanced vascular invasion	Map full extent of nonviable tissue
Ophthalmoplegia or visual loss	Evaluate sinonasal and orbital extension urgently	Indicates severe invasive disease	MRI of orbit, orbital apex and cavernous sinus
Cavernous sinus or intracranial involvement	Limits purely local surgical strategy	Major prognostic deterioration; intensive systemic treatment	Define vascular and intracranial extent
Persistent disease after first debridement	Repeat endoscopy and consider further surgery	Review antifungal adequacy and host recovery	Repeat imaging for residual/progressive disease

The table illustrates that the same observation often has a different but complementary meaning to each specialty. Effective management depends on combining those meanings rather than privileging a single diagnostic modality [2,10,18,19].

16. Prognostic Factors

Several factors identified between 2011 and 2016 can be organized into host, disease-extension and treatment categories. Host-related adverse variables include advanced age, refractory hematological disease, persistent neutropenia, uncontrolled hyperglycemia and major organ dysfunction. Disease-related variables include orbital, vascular and particularly intracranial extension. Treatment-related observations consistently favor surgical source control combined with systemic antifungal therapy [2,6,8,19].

Diabetes itself was associated with better survival than some other underlying diseases in the systematic review, likely because metabolic abnormalities can sometimes be reversed more readily than refractory malignancy or profound persistent immunosuppression. Diabetes is therefore a major risk factor for developing rhino-orbital-cerebral disease but does not necessarily imply the worst prognosis once AIFR develops [2,8].

Radiological extent should also be considered prognostically. Intracranial disease substantially worsens outcome, while residual nonenhancing extrasinonasal tissue after surgery appears particularly concerning. Imaging findings should inform operative planning, intensity of medical therapy, monitoring environment and prognostic discussions [2,19].

17. Challenges in Diagnosis and Management

A major diagnostic problem is the potential mismatch between symptoms and disease extent. Immunocompromised patients may mount little inflammatory response and can therefore have extensive invasion without marked pain, fever or purulent discharge. Conversely, conventional sinus opacification is common in hospitalized and immunocompromised patients and is not synonymous with invasive fungal infection [3,14].

A second challenge is interpreting a negative initial investigation. A single negative biopsy does not completely exclude disease if sampling was superficial or obtained from unaffected tissue, and CT without dramatic bone erosion does not exclude extrasinus spread. Continued clinical deterioration warrants renewed endoscopy, additional tissue sampling or MRI rather than false reassurance from an isolated negative test [13,14,17].

The third challenge is balancing surgical morbidity against inadequate source control. Radical procedures can produce major functional and cosmetic consequences, yet residual necrotic fungal disease is associated with treatment failure. Multidisciplinary interpretation of MRI, endoscopy, systemic prognosis and response to therapy is especially important when deciding the extent of orbital, palatal, skull-base or intracranial surgery [19,20].

18. Conclusion

Acute invasive fungal rhinosinusitis is best understood not as an isolated ENT infection but as a systemic-host problem that becomes anatomically expressed in the sinonasal region. Its aggressive behavior results from fungal tissue invasion and vascular thrombosis and infarction. These biological properties explain why early clinical manifestations can be subtle, why apparently intact bone does not exclude dangerous extrasinus spread, and why devitalized tissue can remain refractory to systemic antifungal therapy [5,14,17].

ENT provides direct visualization, tissue diagnosis, surgical source control and serial endoscopic surveillance. Suspicious turbinate or septal abnormalities in a high-risk patient should trigger urgent investigation, and surgical debridement remains an important component of successful treatment [3,12,20].

Internal Medicine defines and modifies the host environment in which invasion occurs. Control of diabetes and ketoacidosis, management of hematological malignancy and neutropenia, modification of immunosuppression and delivery of systemic antifungal therapy are inseparable from local surgical treatment [6,9,10].

Radiology defines the true anatomical extent of disease. CT provides rapid assessment of sinonasal anatomy and bone, whereas MRI is particularly valuable for detecting tissue devitalization, extrasinus invasion, orbital and cavernous-sinus disease, vascular involvement and intracranial extension. Fungal spread may occur through vascular and soft-tissue pathways before obvious bone destruction develops [14,17-19].

The central management principle is parallel multidisciplinary intervention: high-risk host recognition; urgent ENT endoscopy and tissue sampling; immediate medical stabilization and antifungal therapy; CT/MRI assessment of extent; surgical debridement; correction of underlying disease; and serial endoscopic, clinical and radiological reassessment. In AIFR, pathology may begin in the sinus, but outcome is determined by how effectively ENT, Medicine and Radiology work as a single clinical system [2,10,20].

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