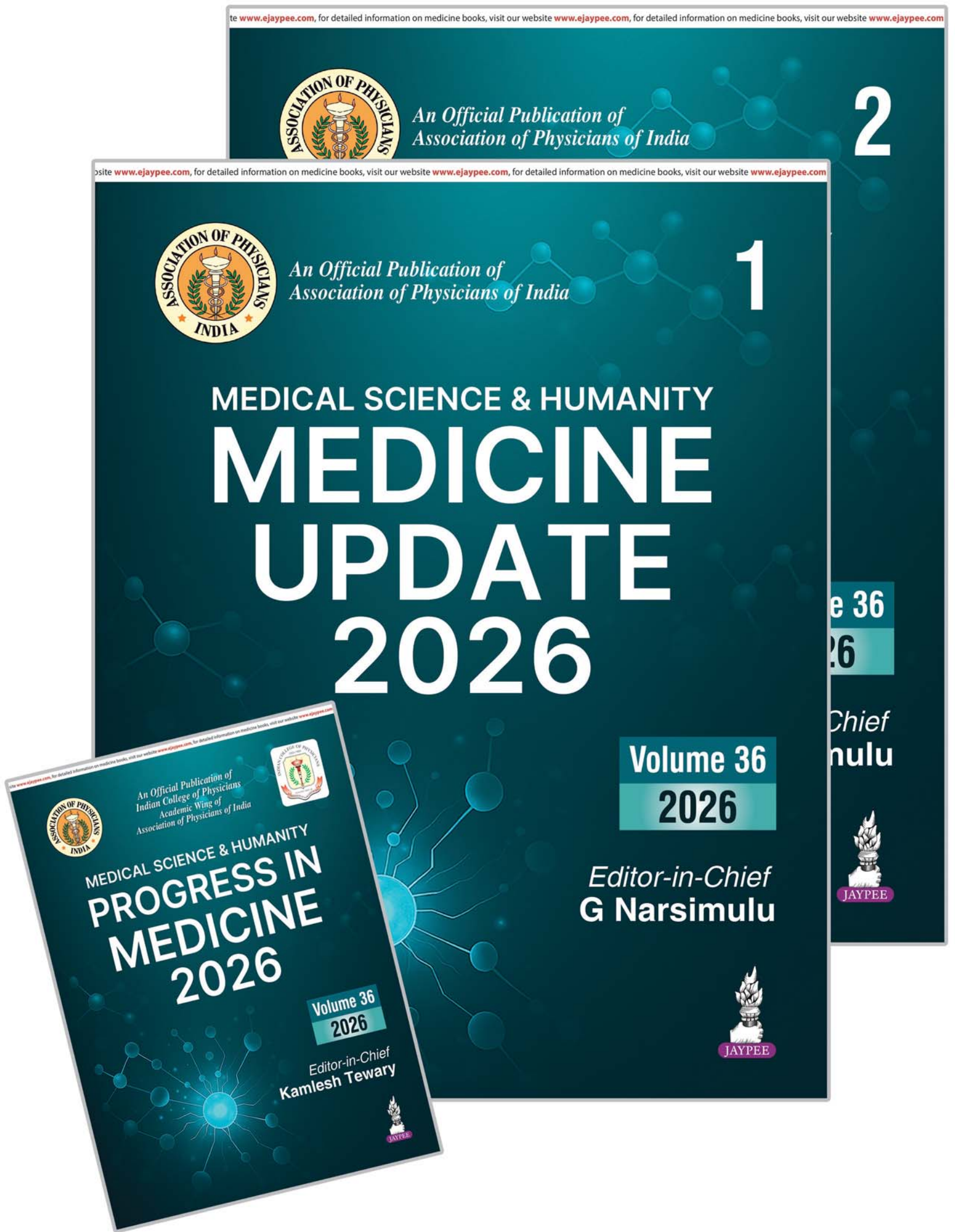

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Clinical Spectrum of Aspergillosis

Bibhuti Saha, Navin Kumar Yadav

ABSTRACT

Aspergillosis encompasses a broad spectrum of diseases caused predominantly by *Aspergillus fumigatus*, ranging from allergic reactions to life-threatening invasive infections. Rising prevalence is linked to chronic lung disease and increasing immunosuppression from conditions such as hematologic malignancies, hematopoietic stem cell transplantation (HSCT), and solid organ transplantation (SOT). This review summarizes the clinical manifestations, diagnostic criteria, and management of key entities: allergic bronchopulmonary aspergillosis (ABPA), chronic pulmonary aspergillosis (CPA), and invasive aspergillosis (IA). Diagnosis combines host risk assessment, imaging, histopathology, and fungal biomarkers such as galactomannan. Early recognition, targeted antifungal therapy, and timely surgical intervention remain essential to improve outcomes.

Keywords: Aspergillosis, ABPA, CPA, IA, galactomannan

INTRODUCTION

The ubiquitous mold *Aspergillus* was first recognized in 1729 by Micheli, in Florence, who noted the resemblance with aspergillum used to sprinkle holy water. Globally, the most common and most pathogenic species is *A. fumigatus*. Indian studies have shown rising cases of *A. flavus* and *A. niger*. It is typically seen in individuals with structural lung disease or immunocompromised states. The spectrum ranges from asymptomatic colonization, allergic reactions, and superficial infection to life-threatening invasive infections.

ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS

Allergic bronchopulmonary aspergillosis (ABPA) is hypersensitivity reaction to *A. fumigatus*, commonly seen in asthma (1–2%) or cystic fibrosis (7–15%). It is characterized by recurrent exacerbation, mucoid impaction, and eosinophilic pneumonia causing central bronchiectasis. Revised ISHAM-ABPA working group has defined diagnostic criteria **(Box 1)**, clinical classification, and treatment response criteria.

BOX 1: ABPA diagnostic criteria.

Predisposing conditions: Asthma, CF, COPD, bronchiectasis *or* compatible clinicoradiological presentation

Essential components:

- *A. fumigatus* specific serum IgE ≥0.35 kU/L or positive *Aspergillus* skin test
- Serum total IgE ≥500 IU/mL

Other components (any two):

- Positive *A. fumigatus*-specific IgG
- AEC ≥500 cells/µL
- Imaging consistent with ABPA
- Mucus plugs expectoration, finger-in-glove and fleeting opacities on chest radiograph, and lung collapse
- <500 IU/mL may be acceptable if all other criteria are fulfilled
- Bronchiectasis, mucus plugging, high-attenuation mucus (HAM), chronic pleuro-pulmonary fibrosis. HAM is pathognomonic and diagnostic of ABPA even if other criteria are not fulfilled

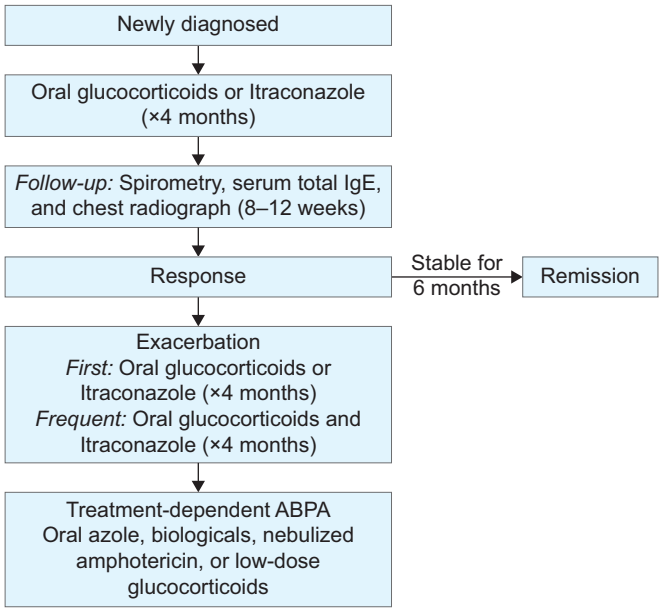
TREATMENT OF ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS (FLOWCHART 1)

Voriconazole, isavuconazole, posaconazole, or biologicals are not recommended as first-line agents for acute ABPA. Biologicals options are omalizumab, tezepelumab, mepolizumab, dupilumab, and benralizumab. Systemic therapy is not recommended for asymptomatic ABPA.

CHRONIC PULMONARY ASPERGILLOSIS

CPA is a progressive disease with 5-year mortality rate of 40–50%. Local immune response to *Aspergillus* colonization is the major driving factor.

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FLOWCHART 1: ABPA treatment.

- *Predisposing factors: Past TB, COPD, smoking, diabetes.*
- *Common symptoms:* cough, fatigue, expectoration, fever, hemoptysis, weight loss, dyspnea, and chest pain.
- *Forms of CPA:*
  - *Localized noninvasive:* Aspergilloma **(Fig. 1)**, nodules
  - *Extensive inflammation:* Chronic cavitary (CCPA), chronic fibrosing (CFPA)
  - *Invasive:* Subacute invasive (SAIA)

SAIA is similar to CCPA except fever is more prominent and having slow progression over 1–3 months as compared to IA. For SAIA preexisting structural lung disease is not a prerequisite. It is commonly associated with mild-to-moderate immunosuppression.

Radiological features: ≥1 lung cavities, typically with irregular or thick wall which progress over years forming pericavitary infiltrates and pleural perforation **(Fig. 2)**.

ESCMID/ERS diagnostic criteria for CPA is outlined in **Figure 3**.

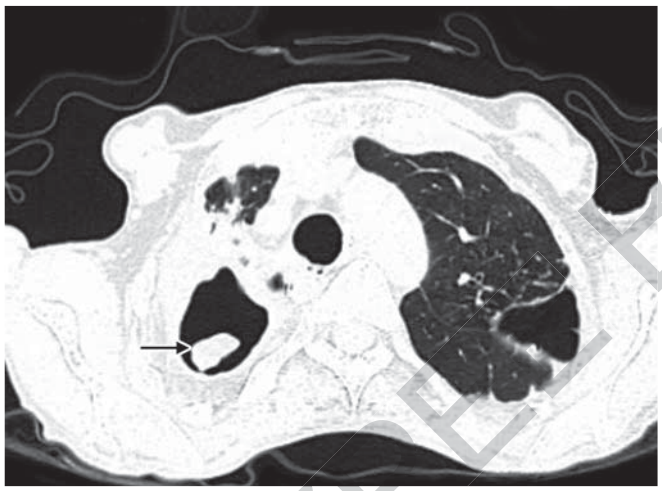


FIG. 1: Thin-walled cavities in both lung apices with soft tissue density (arrow) in the right lung cavity, representing aspergilloma.
Courtesy: Dr Hidayatullah Hamidi and Radiopaedia.

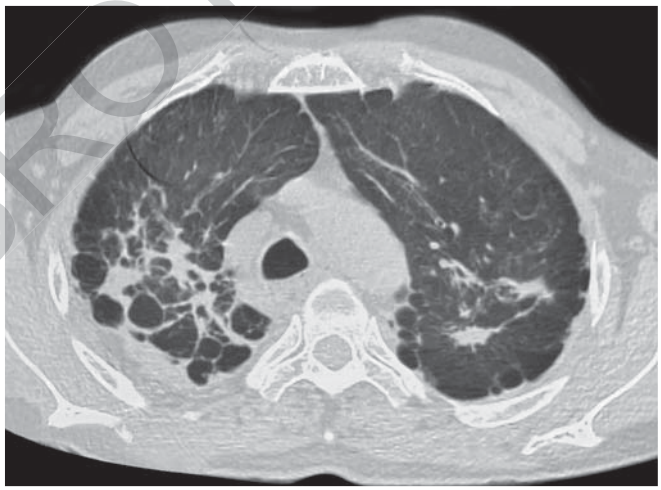


FIG. 2: CPA—multiple lung cavities right > left upper lobes in a patient with prior TB and *Aspergillus* growth from BAL fluid.

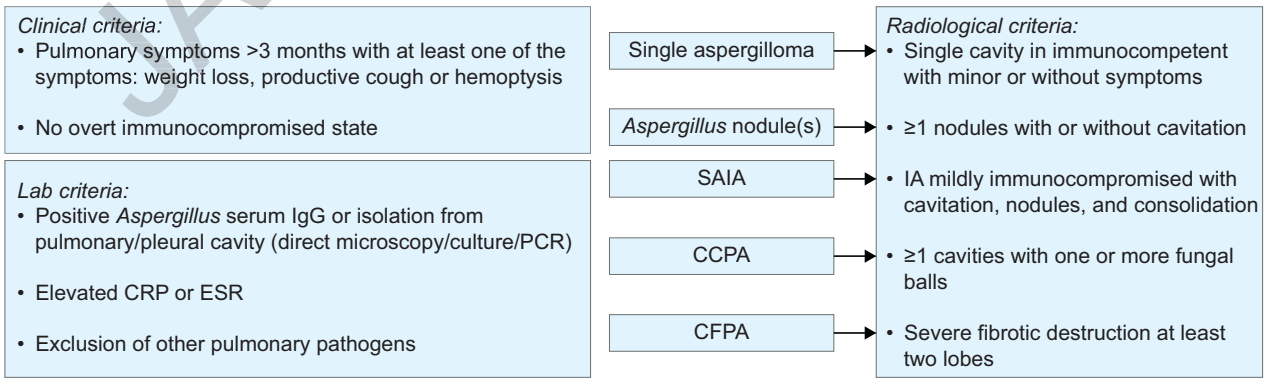


FIG. 3: ESCMID/ERS diagnostic criteria for CPA.

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Treatment of Aspergilloma

Refer to **Table 1**.

Treatment of *Aspergillus* Nodule

- *Immunocompetent:*
  Surgical resection. No upfront antifungal therapy.
  Repeat CT scan at 6-month and 1-year → increased size → 03 months oral antifungal → further management as per CT-scan.
- *Mildly immunocompromised:* DM, low-dose corticosteroids, PLHIV with CD4 > 200 cells/μL, malnutrition.
  - Surgical resection
  - 3 months oral antifungal → further management as per CT-scan.
- *Severely immunocompromised:* Neutropenia, transplant recipient, prolonged high-dose corticosteroids.
  - Manage as IA.

TREATMENT OF CHRONIC CAVITARY PULMONARY ASPERGILLOSIS (TABLE 2)

Aspergillus Sinusitis

Noninvasive: Most commonly affect maxillary sinus. Clinical presentation includes purulent nasal discharge and chronic nasal obstruction.

Invasive form in neutropenic hosts may not show features of inflammation. Common clinical presentation includes nasal congestion, epistaxis, headache, and fever and facial/periorbital pain.

TABLE 1: Aspergilloma treatment.

Immunocompromised	Management as IA	
Immunocompetent	Life-threatening hemoptysis or worsening course	• Surgery + oral azole × 3 months • Bronchial artery embolization
	Asymptomatic or mild hemoptysis	3 monthly radiological monitoring. No therapy if stable disease

TABLE 2: CCPA treatment.

Goals	Clinicoradiological stability or improvement Hemoptysis prevention	
Antifungal therapy: To be started at the time of diagnosis	*Preferred azole*: • Voriconazole • Itraconazole	*Other azole options:* • Posaconazole • Isavuconazole
	Alternative: • Echinocandins • Liposomal amphotericin B	*Duration of azole therapy:* Assess response at 6-month. *Usually duration*: 12 months

Invasive Aspergillosis

IA occurs primarily in severely immunocompromised patients. Predisposing factors include prolonged neutropenia, hematologic malignancy, HSCT/SOT/CAR T-cell therapy, high-dose corticosteroids, immunosuppressants, GvHD, COVID-19, and HIV (CD4 < 50 cells/μL).

Invasive Pulmonary Aspergillosis

Most common form of IA with classic triad of fever, pleuritic chest pain, and hemoptysis in neutropenic patients. IPA rarely manifests before 10–12 days of profound neutropenia. Radiographic findings vary as per host and type of disease-bronchopneumonia, angioinvasive, tracheobronchitis, or chronic necrotizing aspergillosis. Typical lesions in IPA are single or multiple nodules with or without cavitation, patchy or segmental consolidation, or peribronchial infiltration. Pulmonary nodules with surrounding ground-glass infiltrates are classically described as "halo sign" reflecting surrounding hemorrhage. "Air-crescent sign" often appears concurrently after neutropenic recovery **(Fig. 4)**.

Influenza-associated pulmonary aspergillosis (IAPA) and COVID-19-associated pulmonary aspergillosis (CAPA) are increasingly recognized (~10–20%) as important complications in ICU with ~50% mortality.

Disseminated Aspergillosis

It has an extremely high mortality, even with advances in antifungal therapy. *Aspergillus* species can disseminate to brain, eyes, bones, skin, heart, liver, kidneys, and GI tract.

CNS Aspergillosis

It has the highest mortality of IA syndromes. Concomitant pulmonary infection may be absent. Presentation: seizures or focal neurological signs. Mycotic aneurysmal rupture can cause intracranial/subarachnoid hemorrhage or empyema.

Usually, three common radiological patterns are seen: (1) ring-enhancing lesions **(Fig. 5)**, (2) cerebral infarction with or without hematoma, and (3) paranasal sinus mucosal thickening with secondary dural enhancement.

Other Forms of IA

- *Endophthalmitis:* Hematogenous spread and following direct inoculation after ocular trauma.
- *Endocarditis:* Second most common fungal endocarditis after *Candida* spp. Primarily seen with prosthetic heart valves due to fungal contamination. Other risk factors: indwelling CVC and IV drug use.
- Lumbar vertebral osteomyelitis is the most common site for hematogenous spread to bone. *Aspergillus* peritonitis can occur in patients undergoing peritoneal dialysis.

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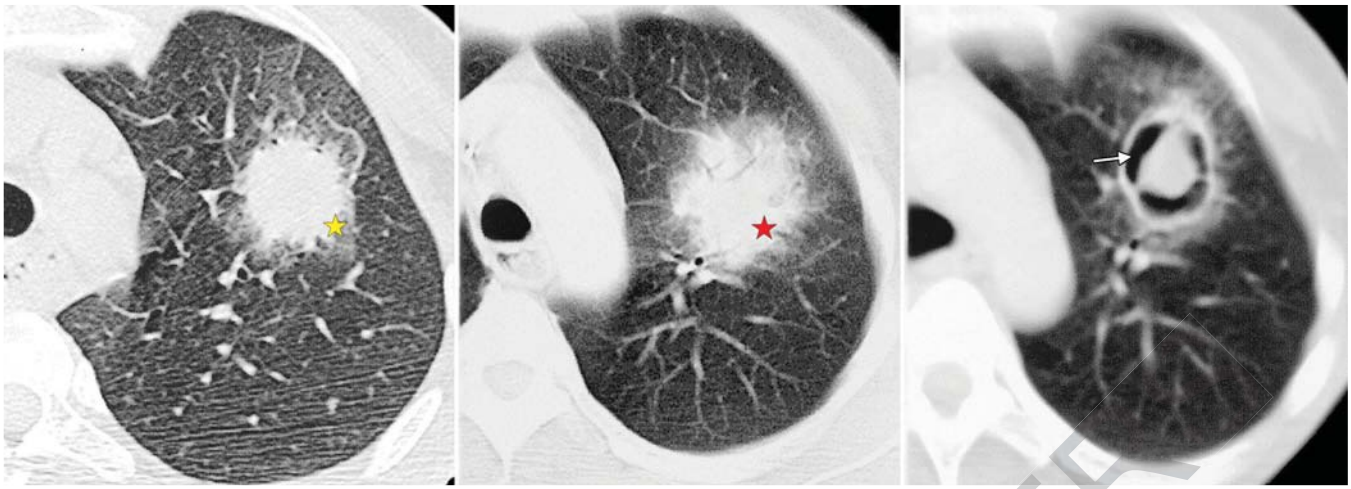


FIG. 4: IPA in febrile neutropenia—Halo sign (yellow star) at day 0 and progression at day 4 (red star). Air-crescent sign (arrow) at day 7 after neutropenic recovery.

Courtesy: Caillot D, Couaillier JF, Bernard A, et al. Increasing volume and changing characteristics of invasive pulmonary aspergillosis on sequential thoracic computed tomography scans in patients with neutropenia. J Clin Oncol. 2001;19(1):253-9.

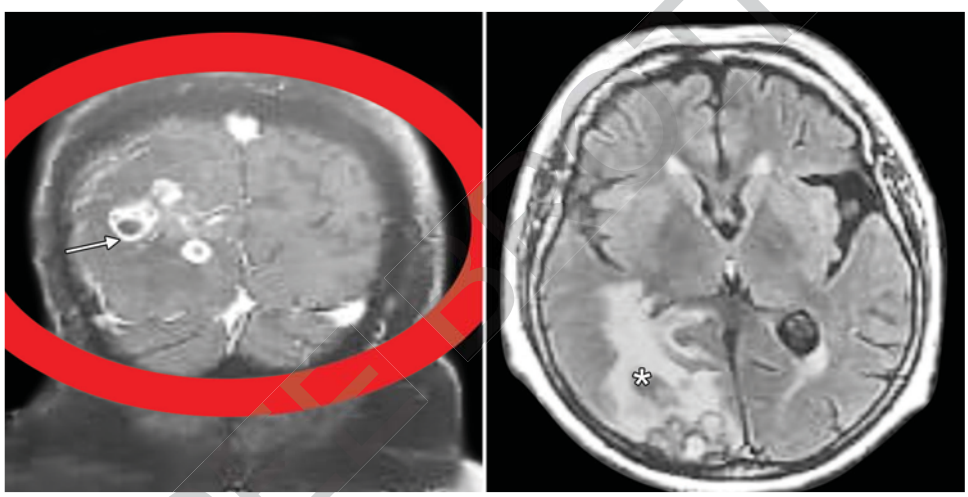


FIG. 5: MRI brain showing multiple abscesses (arrow) with perilesional edema (*) in an elderly lady on long-term steroids and TB empirical therapy. Stereotactic brain abscess aspirate grew *A. fumigatus.*

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DIAGNOSIS

EORTC/MSG revised criteria for:

- *Proven:* IA includes definitive evidence of filamentous growth plus associated tissue damage and should include any one of the following:
  - Histopathology or direct microscopic examination of needle aspiration or biopsy samples demonstrating hyphae compatible with *Aspergillus* along with tissue damage.
  - *Aspergillus* growth on culture of a specimen obtained by a sterile procedure.

Probable: IA includes mycological evidence of *Aspergillus* **(Box 2)**.

On GMS stain, *Aspergillus* is seen as narrow, septate hyaline hyphae with dichotomous acute angle (45°) branching. It grows within 2–5 days of incubation on SDA culture media.

GM is a major component of *Aspergillus* cell wall. Higher baseline/peak serum GM and dual (serum + BAL) positivity predict poor prognosis.

Sensitivity of serum GM decreases with concurrent mold-active antifungal therapy or prophylaxis. Serial GM helps in treatment monitoring **(Table 3)**.

Serum β-D-glucan can be combined with GM but it is nonspecific for *Aspergillus*. Currently, *Aspergillus* PCR cannot be recommended in clinical practice due to lack of standardization. Next-generation sequencing is an emerging tool for diagnostics. 2-Pentylfuran, a volatile compound

BOX 2: Criteria for diagnosing invasive *Aspergillus*.

Any one (provided clinical and host criteria are met):

- Cytology, direct microscopy and/or culture indicating presence of *Aspergillus* spp. in a lower respiratory tract specimen
- Galactomannan (GM) optical density index (ODI) (any one):
  - Single serum or BAL ≥1.0
  - Single serum ≥0.7 and BAL ≥0.8
  - Two BAL ≥0.5 (within 1 week)
  - Single CSF ≥1.0
- *Aspergillus* PCR +ve (any one):
  - ≥2 consecutive blood
  - >2 duplicate BALF
  - >1 in blood and BALF

Any one clinicoradiological abnormality:

- Dense, well-circumscribed lesions with/without halo sign
- Air-crescent sign
- Cavity
- Wedge-shaped and segmental or lobar consolidation
- *Aspergillus* tracheobronchitis

Plus any one (host factors):

- Prednisone equivalent therapy of ≥20 mg/day
- Qualitative or quantitative neutrophil abnormality (inherited deficiency, ANC ≤500 cells/μL)
- Chronic respiratory airway abnormality
- Decompensated cirrhosis
- Immunosuppressants therapy during past 90 days
- Hematological malignancies/HSCT/SOT
- HIV
- Severe influenza or severe COVID-19

produced by *A. fumigatus* can be detected in breath using gas chromatography/mass spectroscopy.

TREATMENT OF INVASIVE ASPERGILLOSIS/ DISSEMINATED ASPERGILLOSIS

Refer to **Table 4**.

CONCLUSION

Along with global warming, rising trend of various predisposing conditions/immunocompromised states, occurrence of cases with different spectrum of aspergillosis are likely to increase. Diagnosis, treatment, and prophylaxis should be adhered with the existing guidelines to enhance antifungal stewardship. Surgery has a definite role in selected cases. Affordable and accurate diagnostics as well as TDM are the need of the hour.

TABLE 3: Diagnosis and prognosis of *Aspergillus infections with Galactomannan* testing.

Specimen	ODI cutoff	Sensitivity (%)	Specificity (%)
Serum	≥0.5 (single)	92	84
	≥1 (single)	71	90
	≥0.5 (two consecutive within a week)	66	95
BAL	≥0.5	75	88
	≥1	75	96

TABLE 4: Treatment of IA/disseminated aspergillosis.

Drug	Dosing	Comments
Primary therapy		
Voriconazole	6 mg/kg q12h × 2 doses → 4 mg/kg q12h IV 200 mg PO BD	• Survival benefit • TDM is recommended • Potential hepatotoxicity
Alternative primary therapy		
Isavuconazole	372 mg q8h × 6 doses IV/PO → 372 mg OD	--
Liposomal amphotericin B	3–5 mg/kg/day IV	Monitor renal function, K^+
Other agents		
Amphotericin B deoxycholate	1–1.5 mg/kg/day IV	• Nephrotoxicity • Limited efficacy in high-risk
Posaconazole	300 mg IV/PO q12h × 2 doses → 300 mg/day	Efficacy in salvage therapy and prophylaxis
Caspofungin	70 mg loading on Day 1 → 50 mg/day IV	Combination with azoles as salvage therapy
Micafungin	100–150 mg/day IV	-"-
Anidulafungin	200 mg loading on Day 1 → 100 mg IV daily	-"-
Duration		
• Location of infection • Underlying disease • Status of immunosuppression • Response to therapy		• *Minimum duration:* 6–12 weeks • Continued till all signs/symptoms have resolved • Radiological resolution or stable disease

Continued

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Continued

Drug	Dosing	Comments
Other measures		
• *Surgery:* Source control • If feasible, immunosuppression reduction • *Primary prophylaxis:* Posaconazole • *Secondary prophylaxis:* Voriconazole • Glycemic control • *Aspergillus endophthalmitis*: Systemic oral/IV voriconazole + intravitreal voriconazole or intravitreal AmB deoxycholate.		

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G Narsimulu MD FICP FIACM is a Senior Consultant–Rheumatologist with an illustrious career spanning over three decades in clinical practice, medical education, and institutional leadership. He served as Professor and Head of the Department of Rheumatology at NIMS, Hyderabad, Telangana, India, where he played a pivotal role in training generations of physicians and advancing rheumatology services in India.

He has held several prestigious positions, including Dean of the Indian College of Physicians, Former President of Indian Association of Clinical Medicine (IACM), and Past President of the Indian Rheumatology Association. He organized Department of Rheumatology, Fellowship courses as well as DNB and DM Rheumatology courses in NIMS first in India. His leadership at ESI Medical College, Hyderabad, led to the establishment of the first DNB course in Rheumatology in ESI Hospitals of India.

A distinguished academician, Dr Narsimulu has authored numerous research papers and contributed chapters to medical updates. He has been a postgraduate teacher and examiner for more than 30 years and is the recipient of multiple national and international honors, including the API Distinguished Member Award, ICP Masters Award, and APLAR Master Award, and has delivered several orations.

Currently serving as the President-Elect of Association of Physicians of India (API), Dr Narsimulu continues to be a guiding force in the medical community. His dedication, vision, and contributions to healthcare, especially in Telangana, made him an invaluable asset to the profession.

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