

Fungal Infection	Histoplasmosis	Zygomycosis	Pneumocystosis
Causative agent	Histoplasma capsulatum, a dimorphic fungus (filamentous mold in environment, budding yeast in human)	Zygomycetes (broad, non-septate hyphae) e.g. Rhizopus, Mucor (black fungus)	Pneumocystis jiroveci (Yeast)
Pathogenesis	● intracellular mycoses of the Reticuloendothelial system. ● primary reservoir: soil ● acquired by inhalation of spores which are engulfed by alveolar macrophages ● Acute: similar to pneumonia. ● Chronic: similar to pulmonary T.B ● Symptoms: fever, headache, weakness with some chest discomfort, lymphadenopathy, enlarged liver & spleen, anemia ● not communicable	● Pulmonary type (Less common) ● Rhinocerebral type (Most common, starts as acute sinusitis) ● Rarely occurs in immunocompetent individuals	Interstitial pneumonia of the alveolar area (common in AIDS patients, opportunistic)
Laboratory diagnosis	1) Specimen: sputum OR bone marrow aspirate and blood buffy coat in disseminated infections. 2) Microscopy: Giemsa: yeast form in the cytoplasm of mononuclear cells as budding cells, small, round or oval, uninucleate. 3) Culture: - sabouraud's agar: mycelial growth with spores. -lactophenol cotton blue: septate hyphae with both microconidia and macroconidia which are: round, thick walled, finger like projections. -glucose-cystein blood agar: yeast form (budding cells) 4) Serology: positive tests for Abs. obtained within 2-4 weeks OR Histoplasmin skin test (1:100) which becomes +ve after infection and remain so for years. In disseminated infection, it may be -ve.	1) Respiratory specimens: Sputum - BAL - Lung biopsy 2) Microscopy: broad non-septate fungal hyphae. 3) Culture on SDA	Histological sections or smears stained by: ❖ GMS stain. ❖ Immunofluorescence (better sensitivity) • If positive —> cysts of hat-shape, Cup shape, crescent • Does not grow in laboratory media e.g. SDA
Treatment	● Moderate infection: Oral Ketoconazole. ● Disseminated disease: I.V. Amphotericine B.	● Amphotericin B ● Surgery	● Trimethoprim (drug of choice) ● Sulfamethoxazole ● Dapsone Note that it is treated by antibiotics, not antifungals.

TYPE	Aspergillus Fumigatus	Aspergillus Flavus	Aspergillus Niger
Pathogenesis	Pulmonary aspergillosis: 1) Aspergilloma: fungus do not invade tissues but grow in a pre-existing cavity, e.g. T.B. cavity or bronchiectasis. X- ray shows fungus ball. 2) Allergic: asthmatic attacks with high level of IgE in serum (hypersensitive people) 3) Invasive Aspergillosis: immunocompromised people. Fungus spread and invades lung tissues--> pneumonia and haemoptsis. Fungal hyphae present in lung tissue.	fungal sinusitis	otomycosis and chronic infection of the external auditory meatus.
Laboratory Diagnosis	Aspergillus species are filamentous septate moulds. Identification depends on: shape of conidiphore and conidia (Aspergillus head), pigmentation of aerial growth of the colony. 1) Specimen: • Respiratory specimens: Sputum, BAL, Lung biopsy. • Other samples: blood for serology & detection of antigens 2) Direct Microscopy: filamentous septate hyphae with characteristic aspergillus head in lactophenol cotton blue preparations 3) Culture: Sabouraud Dextrose agar at 3 different temp. 37 C, 27C and 45C. 4) Serology: Test for Antibody, ELISA test for galactomannan Antigen (specific). 5) PCR 6) Skin test which is exaggerated in allergic conditions. 7) For invasive disease: tissue biopsy 8) For allergic disease, IgE.		
Direct Microscopy	Flask-shaped	Hemi-spherical	Rounded
Culture	White filaments with smoky green spores.	White filaments with yellowish green spores	White filaments with black spores
Treatment	● Voriconazole (Gold-standard) ● Alternative therapy: 1- Amphotericin B 2- Itraconazole 3- Caspofungin ● In invasive pulmonary Aspergillosis and disseminated diseases: Combined treatment with: I.V. Amphotericin B & oral Flucytosin (Synergism)		