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Review

Fungal Infections in Diabetic Patients and Their Management

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Abstract: Diabetes mellitus is a significant risk factor for fungal infections due to persistent hyperglycemia, immune dysfunction, and microvascular complications. Individuals with diabetes are predisposed to a wide spectrum of fungal infections ranging from superficial mucocutaneous candidiasis and dermatophytosis to severe, invasive infections such as aspergillosis and mucormycosis. Impaired neutrophil function, reduced cellular immunity, and an acidic, glucose-rich environment contribute to increased susceptibility, severity, and recurrence of these infections. Delayed diagnosis and inadequate management may result in substantial morbidity and mortality, particularly in cases of invasive fungal disease. Accurate and timely diagnosis relies on clinical assessment supported by microbiological cultures, histopathology, serological tests, and imaging studies when required. Management strategies emphasize optimal glycemic control, early initiation of appropriate antifungal therapy based on the identified pathogen, and correction of underlying risk factors. Surgical intervention may be necessary in refractory or invasive cases. Preventive measures, including patient education, regular follow-up, and early treatment of minor fungal infections, are essential to improve outcomes. A multidisciplinary approach is crucial for the effective management of fungal infections in patients with diabetes.

Keywords: Diabetes, fungal infections, management

INTRODUCTION

Fungal infection usually appears on the skin, as the organisms live on a protein called keratin. This protein makes up the skin, nail and hair. The symptoms of a fungal infection depend on the type of fungus that has caused the infection. Fungi are aerobic and eukaryotic cells and they are more complex than bacteria. (4)

Fungal organisms are broadly classified into

- Hyphae (Moulds): Hyalohyphomycoses, Aspergillus spp., Pseudallescheria bodies, Dermatophytes: Epidermophyton floccosum, Trichophyton spp., Microsporum spp. Phaeohyphomycoses, Alternaria spp., Anthopsis deltoidea, Bipolaris hawaiiensis, Cladosporium spp., Curvularia geniculata, Exophiala spp., Fonsecaea pedrosoi, Phialophora spp., Fusarium spp. Zygomycetes

Absidia corymbifera, Mucor indicus, Rhizomucor pusillus.(9)

- Dimorphic Fungi: Blastomyces spp., Coccidioides spp., Paracoccidioides spp., Histoplasma spp., Sporothrix spp
- Yeasts: Candida spp., Cryptococcus neoformans(6)
- Aspergillosis

  The most common invasive mold infection worldwide, is caused by the ubiquitous fungus Aspergillus spp. Pathogenic species that commonly cause invasive diseases consists of A. fumigatus, A. flavus, A. niger, A.terreus, and A. nidulans. A. fumigatus is the predominant species among them. The rate of progression of invasive disease may be related to the growth rate of the organisms. Macrophage ingestion and killing of the spores and hyphae by neutrophils are the primary host defenses against Aspergillus in the lungs. Corticosteroids can impair the functions of macrophages and neutrophils. (3)T-cell function is thought to be important in the chronic forms of invasive aspergillosis. Signs and symptoms include chronic productive cough, mild to moderate hemoptysis, low-grade fever, malaise, and weight loss. In contrast, patients who are most immunocompromised are least likely to have symptoms and may progress within 7 to 10 days from onset of disease. (1)

Coccidioidomycosis

This is caused by C. immitis, a thermal dimorphic fungus like H. capsulatum and B. dermatitidis. In soil, it grows as a mold with Arthroconidia and converts to a spherule containing 200 to 400 endospores each in host tissues. The number of multiplying coccidioidal organisms is significantly higher than H. capsulatum or B. dermatitidis. Primary infection occurs in the susceptible host when airborne Arthroconidia generated by dust storms or strong winds are inhaled. Patients with diabetes mellitus are more likely to be affected. One third of the cavities may spontaneously within 2 years of discovery. (10)

Candidacies

Candida organisms are yeasts that exist as small (4–6 μm), thin-walled, ovoid cells that reproduce by budding. Other forms, such as pseudo hyphae and hyphae, also can be seen in clinical specimens for most Candida sp except C. glabrata. More than 150 Candida sp have been identified ;however, approximately 10 are considered important human pathogens.(7) The pathogenic species include C.albicans, C. tropicalis, C. parapsilosis, C. glabrata, C. krusei, C. guilliermondii, C. lusitaniae, C. kefyr, C. rugosa, C. dubliniensis, and C. stellatoidea. The pathogens is important owing to the varied pathogenic potential and susceptibility to antifungal agents.

Vulvovaginitis

These are estimated to occur at least once during reproductive years in 75% of women with no recognizable predisposing factors. Identifiable risk factors include broad-spectrum antibiotics, high estrogen-containing oral contraceptives, poorly controlled diabetes, and pregnancy.(1) Among patients infected with AIDS, one large cross-sectional study found similar incidence of vaginal candidiasis compared with patients who do not have AIDS. Signs and symptoms include whitish cheesy discharge, vulvovaginal pruritus, irritation, soreness, dyspareunia, and burning on micturition. (2, 6)

PATHOGENESIS

One of the causes of increased susceptibility to infections in diabetic patients is an impaired immune function. Impaired leukocyte function is associated with inadequate glucose metabolism. Normal phagocytosis requires energy which is a product of glycolysis. (10)Energy supplies used by phagocytes are small, and therefore the substrate must be obtained from external sources. Glucose is transported via the leukocyte cell membrane without the participation of insulin. However, insulin is required to activate enzymes of the glycolytic cycle i.e., glucokinase and pyruvate kinase. Insulin deficiency leads to impaired glycolysis and this impairs the process of phagocytosis. (5)

The disturbed glucose metabolism inside the leukocytes results in a decreased ability of

phagocytes to destroy microorganisms. In the aerobic processes which play a significant role in fungal infections, phagocytosis of microorganisms stimulates respiratory processes within a few minutes, which produces toxic oxidants .(11) The action of the NADPH oxidase and displacement of an electron with NADPH onto the molecular oxygen leads to the formation of a superoxide anion, which gives rise to hydrogen peroxide. With the participation of ferrous ions, hydroxyl radicals are formed from hydrogen peroxide and in the myoloperoxidase-catalyse reaction hypochlorous acid is formed. This in reaction with amines, produces cholaramins. Reactive oxygen compounds are toxic for bacteria, parasitic fungi and tumour cells. (14, 12) A high level of glycaemia in patients with diabetes means that most glucose is metabolized by aldose reductase, by means of NADPH. These transformations lead to the consumption of NADPH which is indispensable in the oxygen processes involved in destroying microorganisms by phagocytes. The significance of these disturbances may be evidenced by the fact that among individuals with myeloperoxidase deficiency who develop severe fungal infections, a large number is represented by patients with diabetes. Patients with diabetes mellitus also have impaired chemotaxis. This seems to have no association with poor metabolic control, but with an independent congenital defect. Impaired chemotaxis has been found in the offspring of patients with diabetes. (8)

Types of fungal infections in patients with diabetes

Significantly increased incidence	Mucormycosis	a. Rhino cerebral, b. Cutaneous
	Candidiasis	a. Vulvovaginal, b. Oral, c. Candiduria, d. Ascending pyelonephritis
	Aspergillosis	a. External otitis
Slightly increased incidence	Candidiasis	a. Cutaneous, b. Prostatic abscess c. Peritonitis in patients undergoing peritoneal dialysis
Possible increased incidence	Dermatophytes	
	Candidiasis	a. Biliary tract infection, b. Postoperative peritonitis
	Pulmonary mucormycosis	
	Invasive aspergillosis	
Incidence similar to that in general population		a. Systemic candidiasis, b. Candidaemia, c. Candida sinusitis

Management

FDA LABELED INDICATIONS	**DOSAGE**	**ADR**
DRUG: CLOTRIMAZOLE		
Candidal vulvovaginitis	Vaginal tablet: Insert 100 mg (1 tablet) INTRAVAGINALLY daily for 7 days or 200 mg (2 tablets) INTRAVAGINALLY daily for 3 days Cream: Insert 1 applicatorful (5 g) of 1% cream INTRAVAGINALLY daily for 7 to 14 days	Pruritus, Skin irritation, Nausea, Vomiting

Candidiasis Oropharyngeal candidiasis	TOPICAL: apply thin layer of 1% cream twice daily for up to 4 wk Slowly dissolve 1 lozenge ORALLY 5 times/day for 14 days	
DRUG: MICONAZOLE		
Oropharyngeal candidiasis	Tablet: 50 mg BUCCALLY against the upper gum above the incisor tooth once daily in the morning for 14 day	Pruritus (2%), Diarrhea (6%), Infectious disease (11.9%), Anaphylaxis reaction
DRUG: ECONAZOLE		
Candidiasis of skin	Apply topically to affected areas twice daily for 2 wk	Erythema, Pruritus, Stinging of skin, Burning sensation
DRUG: ITRACONAZOLE		
Aspergillosis Candidiasis Oropharyngeal candidiasis	Capsule: 200 mg ORALLY every 12 hr for 3 days, followed by 200 mg ORALLY once daily up to a MAX of 200 mg ORALLY twice daily; continue for at least 3 months and until evidence of clinical and laboratory improvement	Rash, Hypokalemia, Diarrhea, Nausea and vomiting Serious: Stevens-Johnson syndrome, Neutropenic disorder
DRUG: FLUCONAZOLE		
Candidiasis	Candidal vulvovaginitis: (uncomplicated) 150 mg ORALLY as a single dose Candidal vulvovaginitis: (complicated) 150 mg ORALLY every 72 hr for 3 doses.	Nausea, Vomiting
		Hepatic: Increased liver enzymes
Candidal vulvovaginitis		Neurologic: Headache
		SERIOUS
Candidemia Candidiasis	loading dose, 800 mg IV or ORALLY, then 400 mg (6 mg/kg) IV or ORALLY daily; continue for 14 days after first negative blood culture result and resolution of signs and symptoms of candidemia Systemic candidiasis, up to 400 mg ORALLY or IV once daily for 4 to 6 wk	Dermatologic: Stevens-Johnson syndrome
Candidiasis of the esophagus		Immunologic: Anaphylaxis (rare
Candidiasis of urogenital site		
Oropharyngeal candidiasis		
DRUG: AMPHOTERACIN		
Candidiasis Fungal infection of central nervous system (Severe) Fungal infection of lung (Severe) Mucormycosis	0.25 to 1 mg/kg/day IV over 2 to 6 hours; MAX of 1.5 mg/kg when given on alternate days	Weight loss; Gastrointestinal: Diarrhea, Indigestion, Loss of appetite, Nausea, Vomiting; Immunologic: Complication of infusion, Chills, fever, headache; Other: Malaise SERIOUS: Cardiovascular: Cardiac dysrhythmia, Hypotension, Thrombophlebitis; Endocrine metabolic: Hypokalemia, Hematologic: Anemia, Thrombocytopenia Immunologic:

		Anaphylaxis Ophthalmic: Blurred vision, Diplopia Renal: Nephrotoxicity
DRUG: NYSTATIN		
Candidal vulvovaginitis Candidiasis of skin, Gastrointestinal candidiasis, Oropharyngeal candidiasis	1 tablet (100,000 units) INTRAVAGINALLY daily for 2 wk Ointment or cream, apply liberally to affected areas TOPICALLY twice daily until healing complete Tablet:1 to 2 tablets (500,000 to 1,000,000 units) ORALLY 3 times per day; continue treatment for at least 48 hr after clinical cure oral suspension, 4 to 6 mL (400,000 to 600,000 units) ORALLY (retained in mouth as long as possible prior to swallowing) 4 times daily; continue treatment for at least 48 hr after perioral symptoms disappear	Nausea and vomiting, With large doses (5 MU/day)
DRUG: GRISEOFULVIN		
Onychomycosis due to dermatophyte, Tinea ungums; onychomycosis	1 g ORALLY once a day for at least 4 months (fingernails) or at least 6 months (toenails)	Dermatologic: Photosensitivity, Rash, Urticaria Gastrointestinal: Diarrhea, Nausea, Vomiting Neurologic: Headache SERIOUS: Neurologic: Acroparesthesia (rare)
DRUG: VORICONAZOLE		
Aspergillosis, Invasive	200 mg ORALLY every 12 hr (guideline dosing)	COMMON Cardiovascular: Peripheral edema (less than 2%), Dermatologic: Rash (7%) Gastrointestinal: Diarrhea (less than 2%), Nausea (5.4%), Vomiting (4.4%), Neurologic: Headache (3%), Ophthalmic: Visual disturbance (21%), Psychiatric:
Candidemia	Maintenance, 200 to 300 mg ORALLY every 12 hr for patients weighing 40 kg or more; 100 to 150 mg ORALLY every 12 hr for patients under 40 kg; treat for a minimum of 14 days following symptom resolution or following last positive culture, whichever is longer	
		Hallucinations (2.4% to 16.6%), Other: Fever (5.7%)
		SERIOUS: Cardiovascular: Prolonged QT interval (less than 2%), Torsades de pointes (less than 2%), Dermatologic: Erythema multiforme (less than 2%), Stevens-Johnson syndrome (less than 2%), Toxic epidermal necrolysis (less than 2%), Gastrointestinal: Pancreatitis (less than 2%); Hepatic: Hepatitis, Increased liver function test, Liver
Candidiasis of the esophagus, Disseminated candidiasis, of the skin and infections in abdomen	200 to 300 mg ORALLY every 12 hr for patients weighing 40 kg or more; 100 to 150 mg ORALLY every 12 hr for patients under 40 kg; treat for a minimum of 14 days following symptom resolution or following last positive culture, whichever is longer	

		failure; Immunologic: Anaphylactoid reaction (less than 2%); Neurologic: Toxic encephalopathy, Ophthalmic: Optic disc edema, Optic neuritis, Renal: Renal failure
DRUG: MICAFUNGIN		
Candidemia Candidiasis of the esophagus Disseminated candidiasis	100 mg/day IV over 1 hr 150 mg/day IV over 1 hr; mean duration of therapy, 15 days (range 10 to 30 days) (manufacturer dosing) 50 mg/day IV over 1 hr ; mean duration for prophylaxis, 19 days (range 6 to 51 days)	COMMON Cardiovascular: Phlebitis (1.6%), Dermatologic: Rash (1.6%) Gastrointestinal: Abdominal pain (1%), Diarrhea (1.6%), Nausea (2.8%), Vomiting (2.4%) , Hematologic: Anemia , Hepatic: Liver function tests abnormal, Neurologic: Headache (2.4%); Other: Fever (1.5%), Rigor (1%) SERIOUS: Hematologic: Febrile neutropenia (36.5%), Hemolysis, Hemolytic anemia, Intravascular hemolysis, Neutropenia, Thrombocytopenia, Thrombotic thrombocytopenic purpura; Hepatic: Hepatitis; Renal: Renal impairment, acute; Other: Drug-induced anaphylaxis

CONCLUSION

Fungal infections constitute an important clinical challenge in patients with diabetes mellitus, owing to altered host immunity, metabolic dysregulation, and associated comorbidities. The spectrum of these infections ranges from common superficial manifestations to aggressive invasive diseases that can significantly increase morbidity and mortality. This review highlights the need for heightened clinical awareness, early and accurate diagnosis, and pathogen-specific antifungal therapy combined with optimal glycemic control. Preventive strategies, including patient education, routine screening, and prompt management of minor infections, play a pivotal role in reducing recurrence and complications. Future research focusing on improved diagnostic modalities, antifungal stewardship, and integrated multidisciplinary care models is essential to enhance outcomes and reduce the burden of fungal infections in the diabetic population.

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