REVIEW ARTICLE

Fungal infections of the oral mucosa

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ABSTRACT

Fungal infections in humans occur as a result of defects in the immune system. An increasing emergence in oral *Candidal* and non-*Candidal* fungal infections is evident in the past decade owing to the rise in the immunodeficient and immunocompromised population globally. Oral *Candidal* infection usually involves a compromised host and the compromise may be local or systemic. Local compromising factors include decreased salivation, poor oral hygiene, wearing dentures among others while systemic factors include diabetes mellitus, nutritional deficiency, HIV infection/AIDS and others. Oral candidiasis is generally a localized infection and rarely appears as a systemic fungal disease whereas oral non-*Candidal* fungal infections are usually signs of disseminated disease. Some of the non-*Candidal* fungi that were once considered exotic and geographically restricted are now seen worldwide, beyond their natural habitat, probably attributed to globalization and travels. Currently infections from these fungi are more prevalent than before and they may present either as primary oral lesions or as oral manifestations of systemic mycoses.

This review discusses the various predisposing factors, clinical presentations, clinical differential diagnosis, diagnosis and management of oral candidiasis, as well as briefly highlights upon a few of the more exotic non-*Candidal* fungi that infect the oral mucosa.

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Key Words: Oral *Candidal* lesions, oral candidiasis, oral mycoses, rare oral fungal infections

INTRODUCTION

Normal oral flora comprises a diverse array of organisms which includes eubacteria, archaea, fungi, mycoplasmas and protozoa.[1] Among these, fungi are classified as eukaryotes, and the most important to dentistry belong to the genus *Candida*. Human infections caused by *Candida albicans* and other related species range from the more common oral thrush to fatal, systemic superinfections in patients who are afflicted with other diseases.[2]

Candida species may be recovered from up to one-third of the mouths of normal individuals and are considered inhabitants of the normal flora of oral and gastrointestinal tract.[3] *C. albicans* is the principal species associated with human oral mycoses and is the most virulent among pathogenic *Candida* spp.[4] The abilities of *C. albicans* to transform from blastospore to the hyphal phase and to form germ tubes, which mark the onset of hyphal growth of *C. albicans*, are the possible factors in the pathogenesis of candidiasis.[5] The advent of the human immunodeficiency virus, the use of wide-spectrum antibiotics, immunosuppressive therapy and increasing incidence of diabetes, are some of the global scenarios that have resulted in the increase in immunocompromised individuals. This in turn has paved way for the increased incidence of opportunistic infections, and oral candidiasis (OC) is clinically the most relevant among them for dental health care providers.[6] The other species of *Candida* (*C.*) besides *C. albicans* encountered in human infections are *C. tropicalis*, *C. glabrata*, *C. parapsilosis*, *C. guillermondii*, *C. krusei* and *C. kyfer* and more recently *C. dubliniensis*.[7,8] [Table 1].

Besides *Candida* spp. other fungi that can cause disease in humans are *Aspergillus fumigatus, Cryptococcus neoformans, Histoplasma capsulatum, Blastomyces dermatitidis, Zygomycetes class, Coccidioides immitis, Paracoccidioides brasiliensis, Penicillium marneffei, Sporotrix schenckii* and *Geotrichum candidum*.[9,10] [Table 1]. The incidence of these rare mycoses has substantially increased in the past decade due to an upsurge in the prevalence of immunocompromised patients, HIV infection and AIDS in particular. The oral

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Table 1: Candidal and non-*Candidal* oral fungal infections and etiologies

Candidiasis	**C. albicans, C. tropicalis, C. glabrata, C. parapsilosis, C. krusei, C. kyfer, C. dubliniensis*
Aspergillosis	*Aspergillus fumigatus*
Cryptococcosis	*Cryptococcus neoformans*
Histoplasmosis	*Histoplasma capsulatum*
Blastomycosis	*Blastomyces dermatitidis*
Zygomycosis	*Orders Mucorales and Entomophthorales*
Coccidioidomycosis	*Coccidioides immitis*
Paracoccidiomycosis	*Paracoccidioides brasiliensis*
Penicilliosis	*Penicillium marneffei*
Sporotrichosis	*Sporothrix schenckii*
Geotrichosis	*Geotrichum candidum*

**Candida*

presentations of these fungi are uncommon and isolated oral lesions are occasionally encountered, generally detected in association with respiratory tract or disseminated fungal infections.[11]

PREDISPOSING FACTORS FOR ORAL CANDIDAL INFECTIONS

Medications

Several drugs are associated with the development of *Candidal* infection. The pharmacological action of these drugs may have a suppressive effect on the normal gastrointestinal and oral bacterial flora that naturally keeps the *Candidal* population at check. This means that there could be *Candidal* overgrowth as a result of such drug effects. The broad spectrum group of antibiotics predispose to *Candidal* infection by this mode of action [12,13]. There are drugs that suppress an individual's resistance to infections by suppressing either the nonspecific inflammatory response or the T-cell-mediated immunity which could in turn predispose individuals to OC. Drugs that come under this category are the corticosteroids used in various inflammatory and immune-mediated diseases.[14] Steroid inhaler medications, intraoral topical steroid preparations[15] and drugs such as azathioprine usually prescribed for treating rejection of organ transplants are known to cause *Candidal* infection.[16]

Drugs that have xerostomic effects often cause OC and drug groups such as antiadrenergics, antidepressants, anticholinergics, antipsychotics and antihypertensives come under this spectrum.[17] The lack of cleansing action of saliva coupled with the reduction in the antifungal salivary components such as lactoferrin, lysozyme, histatins and immunoglobulins would cause the fulminant growth of *Candida* in individuals undergoing such drug therapy.[18]

Nutritional factors

Various nutritional deficiencies have been shown to be involved in the pathogenesis of *Candidal* infections. Among them iron, folate and B12 deficiencies have been suggested to contribute to the prevalence of OC.[19,20] Protein-energy malnutrition, Vitamin C and possibly Vitamin A have all been implicated in the pathogenesis of OC.[21,22] All these deficiencies probably cause infection by reducing host resistance and by causing loss of epithelial integrity of the oral mucosa, both of which could facilitate hyphal invasion by the organism and subsequent infection.[22] Interestingly, an increase in the intake of carbohydrate rich diets have shown to increase the incidence of oral *Candidal* infection. It has been suggested that such diets probably facilitate the adherence of the *Candidal* organism to oral mucosal epithelial cells.[23]

Systemic diseases

Systemic diseases such as diabetes mellitus, hypothyroidism, hypoparathyroidism, Addison's disease, Sjogren's syndrome and others have been associated with O*C.* The primary cause is attributed to reduced salivary flow, which leads to decreased levels of immunoglobulins in the saliva, lowering the efficiency of the humoral-mediated immunological defense mechanism for control of *Candidal* infection.[24] In diabetes mellitus, the level of glycemic control appears to be a more significant factor. Besides decreased salivary flow, poorly controlled diabetic patients exhibit reduced salivary pH and increased salivary glucose levels which facilitate oral *Candidal* overgrowth and colonization.[24]

A wide spectrum of opportunistic infections is seen in immunodeficiency disorders. Hereditary myeloperoxidase deficiency, Chediak-Higashi syndrome, DiGeorge syndrome, human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS) are some of the immunological disorders that can present with OC.[12]

HIV and AIDS

At some point of their illness all HIV seropositive patients develop oral lesions.[25] In the west, the reported prevalence of oral lesions in HIV/AIDS is 56%[26] whereas in India it is reported to be about 64%.[27] In HIV-infected patients candidiasis is reported to occur in over 60% and more than 80% in case of AIDS patients.[28,29] The incidence of oral *Candidal* infection is related to the progressive depletion of the $CD4^+$ T-lymphocytes in HIV-infected patients.[12] Reduced occurrence of the fungal disease is reported in HIV-positive individuals undergoing combination antiretroviral therapy.[30]

Malignancy and cancer therapy

OC has been reported to be prevalent in patients suffering from malignancies such as acute leukemia[10] as well as in cancer patients undergoing chemotherapy and radiotherapy. These patient groups have been reported to have impairment of host resistance, mucosal damage and prolonged neutropenia resulting in an increased prevalence of OC.[31] The incidence of oral *Candidal* infection in these patients can range from 30 to 94%.[32] Radiation therapy to the head

and neck region can cause xerostomia, thereby increasing the incidence of OC.[33]

Other Factors

There have been reports of increased incidence of *Candidal* colonization in denture wearers.[34] Studies have also shown that smokers exhibit more *Candidal* colonies than nonsmokers but the exact mechanism for this is still unclear.[35] Persons with increased blood group H-antigen have reportedly exhibited higher incidence in *Candidal* colonies due to the fact that the H-antigen functions as a receptor for *C. albicans*.[36] Pregnancy, old age and infancy are all important predisposing factors for oral *Candidal* infections.[10] [Table 2].

CLINICAL PRESENTATIONS OF ORAL CANDIDIASIS

Oral Candidal infections may clinically manifest in various forms such as pseudomembranous candidiasis, erythematous candidiasis, denture stomatitis, median rhomboid glossitis, hyperplastic candidiasis, angular cheilitis, chronic muco-cutaneous candidiasis and HIV-associated candidiasis. It can cause oral discomfort, pain, dysguesia and aversion to food. In some patients with HIV infection, OC may also lead to complications such as esophageal candidiasis.[10]

Pseudomembranous candidiasis

Pseudomembranous candidiasis is the most commonly recognized type of candidiasis and is also known as thrush [Figure 1]. It is seen more often in immunocompromised individuals, particularly at extremes of age, those with poorly controlled diabetes mellitus, HIV infection and AIDS, patients on corticosteroids, antiproliferative drugs or psychotropic therapy and prolonged wide-spectrum antibiotics.[37] Clinically thrush presents as confluent white wipeable plaques resembling curdled milk and is usually asymptomatic. The plaques can occur anywhere on the oral mucosa, including the tongue, buccal mucosa and hard palate. Superficially the plaques can be wiped off and the underlying mucosa often exhibits an erythematous appearance.[37] The plaques consist of necrotic material, desquamated epithelial cells, fibrin and fungal hyphae.[36] Histologically, stratum spinosum exhibits hyphal invasion in healthy individuals. However, in immunocompromised patients hyphal extension beyond the spinous layer has been documented. Neutrophils may aggregate to form microabscesses within the epithelium. This feature may not be present in immunocompromised individuals.[37] Other histologically significant features are presence of acanthosis within deeper layers of the epithelium and evidence of lymphocytic infiltration in the surrounding connective tissue.[12]

Erythematous candidiasis

Erythematous candidiasis [Figure 2] clinically presents as localized erythema of the oral mucosa with or without associated symptoms.[12] When symptomatic, it is usually in the form of generalized burning sensation in the mouth accompanied by a loss of filiform papillae on the dorsum of the tongue.[9] It is commonly seen on the dorsal tongue and the palate, and less commonly on the buccal mucosa.[37] This variant was also referred to as "antibiotic sore mouth" by clinicians, due to its association with chronic use of broad-spectrum antibiotics[13] and corticosteroids.[15] Histologically, this lesion is similar to pseudomembranous candidiasis.[12]

Denture stomatitis

This common inflammatory lesion is considered to be a form of erythematous candidiasis and is sometimes referred to as "chronic atrophic candidiasis". The erythema is localized to the fitting surface of the denture bearing areas of maxillary removable dental prosthesis.[9] It is usually asymptomatic; however, may cause mild soreness or burning sensation in some. Poor oral and denture hygiene[38] and ill-fitting dentures[39] have been implicated in the development of this lesion. Besides *Candida*, bacterial infection, mechanical irritation or an allergic reaction to denture base material have all been cited as cofactors in denture stomatitis.[10]

Denture stomatitis has been classified into three subtypes depending on the severity of the lesion as types I, II and III. Type I is the localized simple form with inflammation or pinpoint hyperemia. Type II is a more diffuse erythematous form involving a part or the entire denture-covered mucosa. Type III presents as granular or papillary form involving the central part of the hard palate and alveolar ridge.[10] Interestingly, it has been reported that the histopathology of denture stomatitic specimen rarely demonstrates *Candidal* hyphal penetration within the

Table 2: Predisposing factors for oral candidiasis

Medications	Broad-spectrum antibiotics, systemic steroids, steroid inhalers, cytotoxic agents
Nutritional factors	Iron, folate, B12 deficiencies, high carbohydrate intake, protein energy malnutrition , Vitamins C and A deficiencies
Systemic diseases	Diabetes mellitus, hypothyroidism, hypoparathyroidism, Addison's disease, Sjogren's syndrome, hereditary myeloperoxidase deficiency, Chediak-Higashi syndrome, DiGeorge syndrome
HIV and AIDS	Human immunodeficiency virus infection and acquired immunodeficiency syndrome, related to the progressive depletion of the $CD4^+$ T lymphocytes
Xerostomia	Caused by medications, systemic diseases, irradiation
Malignancy and cancer therapy	Acute leukemia, cancer patients undergoing chemotherapy and radiotherapy
Other factors	Wearing dentures, smoking, persons with increased blood group H antigen, pregnancy, old age and infancy

keratinized layer of the epithelium. However, the fitting surfaces of the dentures have shown heavy colonization of *Candida*.[9]

Median rhomboid glossitis

Median rhomboid glossitis, also known as central papillary atrophy, was originally thought to be a developmental anomaly of the tongue. Now it is considered to be a variant of erythematous candidiasis.[40] Clinically it presents as an erythematous, well-demarcated, rhomboid or elliptical area of atrophy of the papillae of the dorsal tongue. It is usually localized to the posterior aspect of the tongue in front of the circumvallate papillae.[37]

In some patients with median rhomboid glossitis, OC may be seen in other sites such as the oral commissures and hard palate. Palatal "kissing lesion" usually results from direct inoculation that occurs when the dorsal tongue makes contact with the hard palate during deglutition. This presentation of erythematous candidiasis has been referred to as chronic multifocal candidiasis.[9] In some *Candidal* hyphae can be demonstrated histologically in the superficial layers of the epithelium with hyperplastic rete pegs and absence of papillae.[40]

Hyperplastic candidiasis

Hyperplastic candidiasis clinically appears as a white plaque that is non-scrapable, and often mimics leukoplakia.[36] It commonly occurs on the anterior buccal mucosa and is usually well demarcated, slightly elevated and adherent.[12] It can appear as small translucent lesions or as large, dense opaque plaques. It can also present either as an isolated, adherent white plaque (homogeneous type) or as multiple white nodules on an erythematous background (nodular or speckled type).[36] Hyperplastic candidiasis has been associated with a higher degree of dysplasia and malignancy than leukoplakia without any *Candidal* component.[41] It has been suggested that this condition probably represents candidiasis that is superimposed on a preexisting leukoplakia. Some believe that *Candida* alone is capable of inducing a hyperkeratotic lesion.[9] Confirmed diagnosis can be made by the presence of *Candidal* hyphae associated with the lesion histopathologically and by the complete resolution of the lesion with antifungal treatment.[41]

Angular cheilitis

Angular cheilitis is a chronic inflammatory lesion characterized clinically by erythema, maceration, crusting and fissures. It affects the labial commissures and is often symptomatic and bilateral[37] [Figure 3]. It can be associated with multifocal candidiasis but often occurs alone. It has a predilection to occur in older patients with reduced vertical dimension of occlusion and accentuated folds at the angles of the mouth and in persons with habit of constantly licking their lips at the level of the commissures. This could cause pooling of saliva in these areas, rendering them moist and favoring *Candidal* growth.[9] Nutritional factors such as iron deficiency, vitamin B deficiency, high carbohydrate diet intake, have all been implicated in the development of these lesions.[42] Angular cheilitis is now thought to be caused by *Candida* species and/or *Staphylococcus* and *Streptococcus*.[43]

Chronic mucocutaneous candidiasis

This is a heterogenous disorder often characterized by the involvement of mucosa, skin and nails, and exhibiting a poor response to treatment with topical antifungal agents.[44] *Candidal* infections usually present within the first few years of life.[9] Hyperplastic lesion is the most common presentation clinically. Most cases are sporadic, although, an autosomal recessive inheritance pattern has been noted and can be correlated with the severity of the lesion. The underlying immune defect is thought to be cell mediated. Studies have shown that it may involve a defect in cytokine production in response to *Candidal* and bacterial antigens.[45] It has also been associated with various endocrinopathies, including endocrine-candidiasis syndrome and autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED) syndrome, in addition to iron deficiency anemia. The endocrine abnormalities associated include hypothyroidism, hypoparathyroidism, Addison disease and diabetes mellitus.[46]

HIV-associated candidiasis

HIV-infected patients are often diagnosed with OC and it is considered as the most common opportunistic infection in this population.[46] The clinical presentations of this disease are usually in the form of pseudomembranous, erythematous, or hyperplastic candidiasis. The erythematous form more often presents as an early oral manifestation of HIV infection, usually involving the palate and dorsal tongue.[47] The pseudomembranous form frequently involves the tongue, hard and soft palate, and buccal mucosa, although any region of the oral mucosa may be involved.[48]

A unique lesion associated with HIV infection, described as the linear gingival erythema, is a nonplaque-induced gingivitis that presents as an erythematous band along the marginal gingiva. It often results from a combination of *Candidal* and bacterial infections. It appears diffuse or generalized and is usually refractory to plaque control measures. Linear gingival erythema is considered to be an oral manifestation of HIV infection but at times has been reported in HIV-negative population at a much lower frequency.[49]

Clinical differential diagnosis

Chemical burns, traumatic lesions, syphilis and other white keratotic lesions that present a pseudomembrane may clinically mimic pseudomembranous candidiasis. Solitary erythematous lesions such as erythematous candidiasis should be differentiated from thermal traumatic lesions, erosive lichen planus and lichenoid reactions, lupus

erythematosis, erythema multiforme, pernicious anemia and epithelial dysplasia.[12]

Diagnosis

The diagnosis is often made based on clinical examination and thorough history. Additional adjunctive diagnostic methods such as direct examination of smears, culture and biopsy are valuable in confirming the diagnosis.[46]

Laboratory tests

Direct examination of smears

The affected area is scraped with a spatula and the smear spread onto a glass slide. The slide is air dried, fixed in alcohol and stained with periodic acid Schiff reagent (PAS). The PAS stain preferentially stains glycogen in the fungal cell wall and thus renders the *Candidal* organisms magenta under the microscope. A chairside diagnostic procedure that can be performed is by placing a drop of 10% potassium hydroxide (KOH) onto the fixed slide. KOH lyses the keratinocytes, thus allowing the *Candidal* organisms to be more readily seen under the microscope.[46]

Culture

Culture, using Sabourad's dextrose agar (SDA), is useful in a qualitative manner to determine the presence or absence of *Candida*. A sterile cotton swab is placed in contact with the involved mucosa, after which it is used to inoculate the SDA plate. The agar is incubated at 25-30°C for 48-72 hours. The presence of creamy white colonies indicates a positive culture result. For a quantitative analysis, whole saliva, or a 10-ml mouth-swill of sterile water, is collected and the presence and number of colony forming units of *Candida* can be studied after culture on SDA.[1] Further speciation of the cultured fungi can be done by the conventional biotyping techniques or with the aid of commercially available systems like Micronaut-*Candida*, API ID32C, RapID yeast plus system, Auxocolor, Vitek, Api *Candida*, etc.[50,51]

Biopsy

Biopsy is usually performed for the diagnosis of hyperplastic candidiasis. Because this form may mimic other lesions, particularly squamous cell carcinoma, a biopsy is highly recommended. Histopathological examination will reveal epithelial parakeratosis with polymorphonuclear leukocytes in the superficial layers. PAS-stained slides will show the presence of *Candidal* hyphae in these areas.

Management

Management of OC should be directed at identifying the underlying factors that could cause the disease, through clinical examination and history taking.[12] Nutritional deficiencies, diabetes mellitus, immunodeficiencies, usage of pharmacological agents, denture adequacy, oral and denture hygiene status should be identified. Saliva testing to determine hydration and salivary gland function must be done. If alteration or correction of the underlying predisposing factor is not possible or required, drug therapy is initiated.[6] There are different treatment modalities to manage OC using antifungal agents. The mechanism of action of the antifungal agents involves an alteration of RNA and DNA metabolism or an intracellular accumulation of peroxide, which proves to be toxic to the fungal cell.[46] Usually the lesions resolve with the use of topical polyene or azole antifungal agents. The choice of drug is governed by the patient's medical history, oral symptoms and compliance. Nystatin oral suspension (100,000 units⁄mL – 1 mL topically), or nystatin pastilles (100,000 IU) four times daily for 7-14 days is deemed effective in most cases.[6] Amphotericin lozenges (10 mg) or suspension (100 mg⁄mL) four times daily after meals for 14-21 days is also effective.[12] Miconazole 2% gel, 2.5 mL applied topically four times daily after meals for 14-21 days can be used. Miconazole is contraindicated in patients taking Warfarin as it may potentiate their effects.[6] In refractory lesions treatment with systemic antifungal agents such as ketoconazole, fluconazole and itraconazole and amphotericin may be necessary.[6]

NON-CANDIDAL OROFUNGAL DISEASES

In recent times, the prevalence of oral fungal infections other than candidiasis has been on the rise. Immunodeficiency diseases such as HIV infection and AIDS, immunosuppressive therapy, prolonged usage of broad-spectrum antibiotics and corticosteroids, are some of the notable reasons for the disease emergence.[52] Few of the rare fungal organisms that can cause oral diseases in humans are *Aspergillus, Cryptococcus, Histoplasma, Blastomyces, Zygomycetes, Coccidioides* and *Paracoccidioides, Geotrichus, Sporothrix* and *Penicillum*.[10] Some of these fungi once considered exotic and geographically restricted are now seen worldwide, beyond their natural habitat, probably attributed to globalization and travels.[52] Infections from these fungi may present either as primary oral lesions, which are uncommon or as oral manifestations of systemic mycoses. Following is a brief review on some of the rare mycotic diseases that can cause lesions in the oral cavity.

Aspergillosis

Aspergillus organism is ubiquitous and can be found in soil and in decaying vegetation. Most species of *Aspergillus* do not grow at normal human body temperature, only the pathogenic species have the ability to do so.[9] *Aspergillus fumigatus* is the species most often implicated in human diseases.[52] It does not usually cause disease in the immunocompetent population.[53] In immunocompromised patients, however, invasive pulmonary aspergillosis has been reported, following inhalation of the spores.[54] Aspergillosis is reportedly the second most common opportunistic fungal infection after candidiasis.[9] The fungus either invades blood vessels, causing thrombosis and infarction of surrounding tissue or invades the sinuses, progressively causing palatal and tongue lesion.[55]

Oral aspergillosis is predominantly seen in immunocompromised individuals. Marginal gingiva and the gingival sulcus have been cited as the portal of entry of the spores.[9] Painful gingival ulcerations and mucosal soft tissue swellings with gray or violaceous hue have been reported. This can advance to extensive necrosis and present clinically as a yellow or black ulcer with facial swelling.[56] [Figure 4].

The main differential diagnoses for oral aspergillosis are mucormycosis and pseudomonas oral infection.[10] However, palatal ulcerative presentations of aspergillosis could mimic lesions such as tuberculous ulcer, leprosy, syphilitic ulcer, Wegener's granulomatosis, systemic lupus erythematosus, polyarteritis nodosa, Crohn disease, Chug-Strauss syndrome, sarcoidosis, necrotizing sialometaplasia and neoplastic lesions such as squamous cell carcinoma and lymphoma.[57,58]

Diagnosis can be confirmed by PAS-stained sections, culture, serodiagnosis and polymerase chain reaction. In cases of invasive aspergillosis, the detection of circulating antigen galactomannan (GM), a component of the cell wall of *Aspergillus* species, and/or specific antibodies in the serum are useful markers for diagnosis.[58] In primary oral or paranasal sinus lesions, biopsy can provide a definite diagnosis.[52]

Cryptococcosis

Two *Cryptococcus* species can cause diseases in human namely *Cryptococcus neoformans* and *Cryptococcus gattii*. *C. neoformans* infection generally affects immunocompromised hosts. *C. neoformans* is a ubiquitously distributed microorganism. It has been isolated in the soil and in the feces of birds, such as pigeons, canaries, and parrots.[59] *C. gattii* is more often isolated in immunocompetent subjects[52] and the natural reservoir of this fungus is the koala bear and is endemic to Australia and temperate zones, where it is also frequently found on trees of the Eucalyptus genus.[60]

In the immunocompetent population, the fungus usually does not cause any manifestations and the infection may remain subclinical. But in immunocompromised hosts, besides the lungs, Cryptococcosis also infects the central nervous system, skin and the oral mucous membrane.[61] It can produce a variety of cutaneous and oral mucosal manifestations such as superficial ulcers, nodules, granulomas or carcinoma-like lesions.[62] Intraoral sites commonly affected are gingiva, palate and tooth socket after extraction. Violaceous nodules of granulation tissue, swellings and ulcers are the various forms of oral lesions reported.[10]

Diagnosis of cryptococcosis is done by isolation of the organism in sputum samples or broncho-alveolar lavage for pulmonary infection, in cerebrospinal fluid (CSF) and blood for disseminated forms. The ink test on CSF and identification of circulating antigens on serum and/or CSF samples are considered reliable.[63] For cutaneous and subcutaneous forms, histopathological study is useful.

Histoplasmosis

Histoplasmosis is caused by *Histoplasma capsulatum*, a fungus mainly found in the Ohio and Mississippi river valleys of the United States.[52] The disease can affect the lungs and cause acute or chronic respiratory problems in the immunocompromised population.[64] The reticuloendothelial system, gastrointestinal tract and kidneys are also affected by this fungus. Histoplasmosis may sometimes appear in a mucocutaneous form that can manifest as ulcerating erosive or nodular lesions in the oral mucous membrane.[65] The oral lesions may also appear granulomatous and may be painful, localized on the oral mucosa, tongue, palate or lips. The ulcers may often resemble carcinoma or tuberculosis because of the raised and rolled borders, usually covered by a yellow or greyish membrane.[11] [Figure 5]. Diagnosis is usually confirmed by microscopy, culture and serology. The serum immunodiffusion assay that detects antibodies against the H and M antigens of *H. capsulatum* is reported to be a reliable diagnostic method.[66]

Blastomycosis

Blastomycosis is a rare fungal disease caused by *Blastomyces dermatitidis*, a spore found in the soil of the Mississippi, Missouri and Ohio River valleys and the Great lakes region.[10] When inhaled, it can cause disseminated disease or a localized respiratory condition. When the disease affects the oral cavity, it produces ulcerating mucosal lesions[67] as well as sessile projections, granulomatous or verrucous lesions.[10] Small ulcers are characteristic oral manifestation and may present as a primary lesion or secondary to disseminated disease. Oral lesions may resemble actinomycosis without the suppurative element.[68] The isolation of the fungus from clinical specimens and histopathological examination of appropriately stained sections are the definite method for establishing diagnosis. The fungus grows well in standard mycological media such as SDA, potato dextrose agar, potato flake agar or inhibitory mold agar. Classically appears as round to oval, multinucleate yeast cell with a single broad-based bud under the microscope.[69] A specific DNA probe has been developed for faster mycelial identification.[9]

Zygomycosis

The two orders of *Zygomyces* that are of clinical concern are *Mucorales* and *Entomophthorales*. *Mucorales* includes the genera *Rhizopus, Mucor, Absidia and Cunninghamella*, which are more often implicated in human diseases.[70] These microorganisms are capable of causing deep fungal infections that can rapidly deteriorate the condition of immunocompromised patients. *Entomophthorales* causes infections mainly in immunocompetent subjects following trauma, and are far less invasive than *Mucorales*.[52] Infection

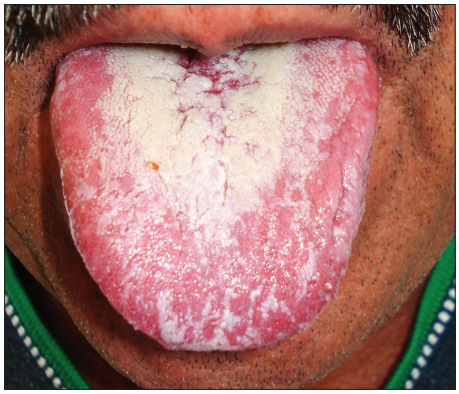

Figure 1: Oral thrush

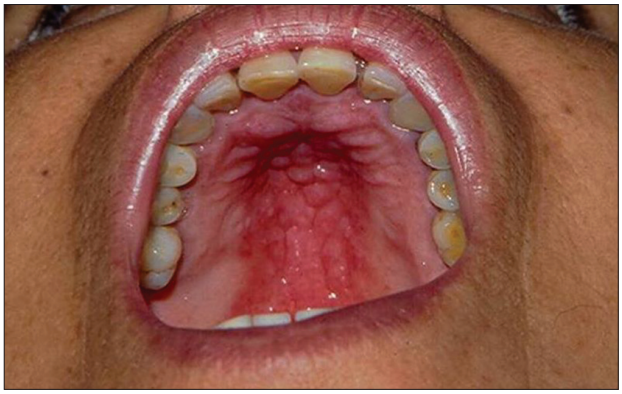

Figure 2: Palatal erythematous candidiasis

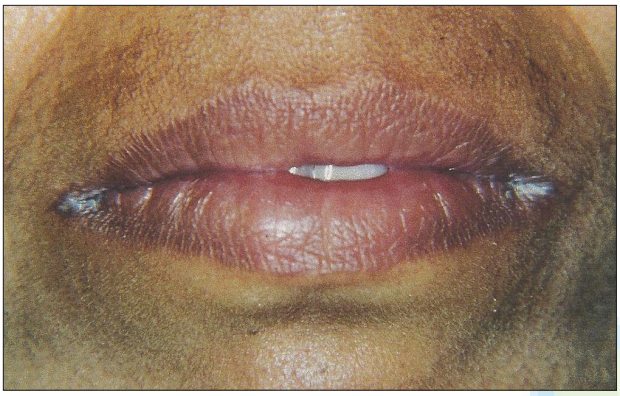

Figure 3: Angular cheilitis

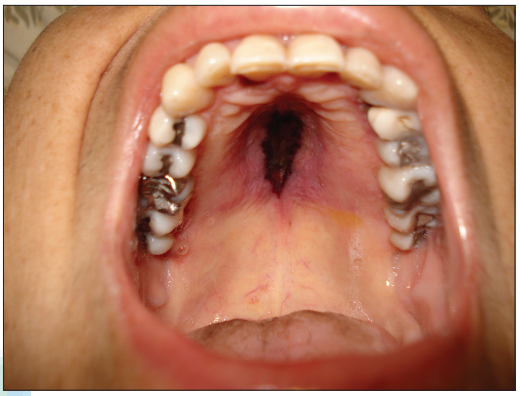

Figure 4: Aspergillosis ulcer

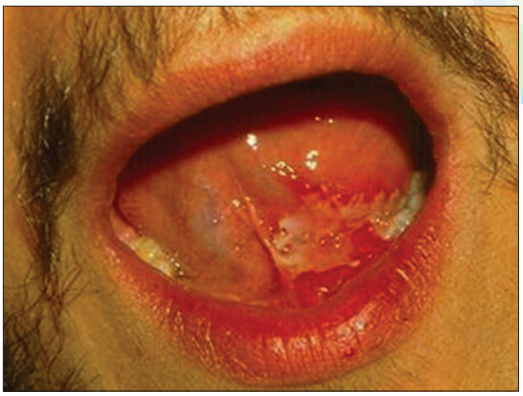

Figure 5: Histoplasmosis ulcer

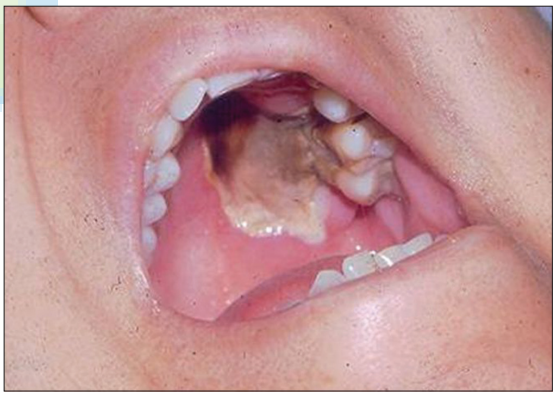

Figure 6: Mucormycosis ulcer

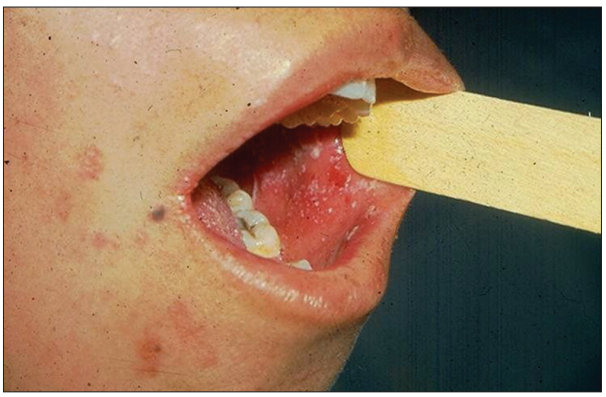

Figure 7: Pseudomembranous geotrichosis

caused by *Mucorales* is known as mucormycosis, while entomophthoramycosis is infection of the *Entomophthoral* fungi.[71]

Oral zygomycosis has more often manifested in immunocompromised patients with blood dyscrasis, diabetes, immunosuppressive therapy, corticosteroid therapy, malignancy, hepatitis, tuberculosis, etc. Prior to HIV/AIDS, diabetic acidosis accounted for about 50-70% of patients reported with mucormycosis. Recently, this infection is encountered in HIV infection more frequently.[10] The most common oral manifestations are palatal ulcers, which are frequently necrotic, well-delimited,

with well-defined borders and may appear as either black or white.[71] [Figure 6]. A definitive diagnosis of zygomycosis is obtained by histology and isolation of the microorganism in culture. The characteristic hyphae are well demonstrated with Gomori-Grocott staining.[72]

Coccidioidomycosis

Coccidioidomycosis, caused by *Coccidioides immitis*, was once confined to the Western hemisphere but is now virtually seen anywhere in the world.[9] It can present with different clinical pictures, and in about 60% of cases, is asymptomatic. Many usually remain benign or may evolve into pulmonary or extrapulmonary disease.[73] Coccidioidomycosis is a recognized opportunistic infection among HIV-positive persons.[52] Oral lesions are uncommon and have been described as ulcerated granulomatous nodules.[9] Clinically the ulcers appear nonspecific and usually heal by hyalinization and scar.[67] Diagnosis is by history and examination supported by histology. The presence of mature spherules, filled with endospores under the microscope is diagnostic.[74] Coccidioidin skin test is also a valuable diagnostic aid.[10]

Paracoccidioidomycosis

Paracoccidioidomycosis is caused by *Paracoccidioides brasiliensis* and is endemic to South and Central America.[75] It manifests with a wide spectrum of clinical presentations, often involving the oral cavity. In many cases, the first and main clinical manifestations are oral lesions.[52] The oral lesions are usually multiple and involve the lip, gingival, buccal mucosa, palate, tongue and floor of the mouth. They are often described as mulberry-like ulcerations. Diagnosis can be made by demonstration of multiple budding daughter yeasts on the parent cells resulting in "Mickey mouse ears" appearance under microscope with Gomori-Grocott methenamine silver or PAS-stained histopathological sections and culture.[9]

Penicilliosis

Penicilliosis is caused by *Penicillium marneffei*, and was considered a rare disease before the advent of HIV/AIDS.[10] *P. marneffei* is endemic to Southeast Asia, southern China, northern Thailand and Hong Kong[76] but its ecology and the mode of transmission to man are unknown.[77] The prevalence of infection has increased considerably in the past decade, especially in persons who are infected with HIV.[78] Currently, penicilliosis is reported to be the third most common opportunistic infection in patients with AIDS in Thailand, followed by tuberculosis and cryptococcosis.[78,79] *P. marneffei* infection may either be disseminated or focal in patients who are otherwise healthy. In HIV seropositive patients the disease usually manifests in the disseminated form.[10] Oral lesions usually appear as shiny papules, erosions, or as shallow ulcers covered with whitish yellow, necrotic slough and are found on the palate, gingiva, labial mucosa, tongue and oropharynx.[52] Diagnosis is usually confirmed by histology and culture. In the presence of lymphadenopathy fine needle aspiration has proven to be a useful diagnostic tool.[80]

Sporotrichosis

Sporotrichosis is caused by *Sporothrix schenckii*, a fungus found in moss, soil and rotting wood with higher incidence of infection among agricultural workers, florists and miners than the general population.[81] It is most frequently found in Central America, Brazil and Mexico, but now has a worldwide distribution. *S. schenckii* gains access to the subcutaneous tissues via traumatic lesions, and proliferation of the fungus leads to the appearance of a nodule or small ulcer.[10]

Rarely primary oral sporotrichosis may be seen but more commonly occurs secondary to disseminated disease from the skin or lung. Oral lesions can manifest in various forms such as erythematous, ulcerative, suppurative, granulomatous, vegetative or papillomatous. The oral lesions are usually painful and heal without scarring. Differential diagnosis of oral sporotrichosis includes aphthous ulcers, lichen planus or secondary cutaneous leishmaniasis owing to its clinical resemblance to these oral lesions.[10] Histology and culture are valuable in confirming the diagnosis. Since the parasitic yeast form of this fungus is difficult to observe under microscope, isolation in mycological media still remains the gold standard diagnostic method.[82]

Geotrichosis

This is caused by the fungus *Geotrichum candidum* which has been isolated from the skin, sputum and feces of humans. It is carried in the alimentary tract of some individuals and can sometimes cause opportunistic infection.[10] The fungus infects the bronchi, lung, mouth and intestine. The oral lesions of geotrichosis are similar clinically to pseudomembranous candidiasis and differentiation can be done only by histolopathological examination and culture of the organism.[10] [Figure 7]. Besides the pseudomembranous form, other clinical presentations of oral geotrichosis have been reported such as the villous hyperplastic and ulcerative forms. Definitive diagnosis is usually confirmed by culture and histology. Demonstration of multiple septate hyphae with rectangular arthroconidia is a useful criterion for diagnosis.[83]

CONCLUSION

Dental clinicians play an important role in the diagnosis and management of oral fungal diseases. Therefore, an adequate knowledge is pivotal in recognizing the various guises of oral *Candidal* and non-*Candidal* infections, which could be markers of immune deterioration. The possibility of systemic mycoses may be considered in cases where chronic oral ulcerations or unusual mouth lesions are observed particularly in the immunocompromised patient population. Awareness of the myriad presenting signs and symptoms of oral fungal diseases could aid in early diagnosis, proper

treatment and prevention of disease dissemination thereby decreasing morbidity.

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