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# Oral Candidiasis in Immunocompromised Patients: A Review of Current Trends and Future Approaches

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**KEYWORDS:**

Oral candidiasis; Candida albicans; Immunocompromised patients; Antifungal resistance; Diagnostic techniques.

**ABSTRACT:**

Background: Oral candidiasis is a common opportunistic fungal infection predominantly caused by Candida albicans, a yeast-like organism frequently present as a commensal in healthy individuals. Under conditions of local or systemic immunosuppression—such as chronic corticosteroid or antibiotic use, extremes of age, or immunocompromising diseases including HIV/AIDS—the organism proliferates and leads to clinical disease. Other pathogenic species include C. krusei, C. glabrata, and C. tropicalis.

Clinical Presentation: Oral candidiasis, also known as oral thrush, typically manifests as creamy-white plaques on the tongue, inner cheeks, gums, palate, or oropharynx. These lesions may resemble the white chest feathers of the thrush bird, from which the condition derives its name. A Candida load exceeding 400 CFU/mL in the oral cavity is indicative of active infection. Clinically, the disease may present as acute pseudomembranous, acute erythematous, chronic hyperplastic, or chronic erythematous candidiasis.

Diagnosis: Diagnostic evaluation includes microscopy, culture, and molecular methods. C. albicans appears as ovoid or spherical budding yeast cells. Smears may be stained using PAS or GMS, while cultures on Sabouraud's agar yield creamy, smooth colonies with a yeasty odour. The germ tube test provides rapid identification of C. albicans via its ability to form germ tubes in human serum at 37 °C.

Management: Treatment involves plaque removal and antifungal therapy such as nystatin, miconazole, clotrimazole, or ketoconazole. Good oral hygiene practices are essential for prevention. Future advancements, including the development of anti-Candida vaccines, may offer improved control of this infection.

## Introduction:

The most common causative agent of oral candidiasis, *Candida albicans*, is a yeast-like fungus that infects individuals with immunocompromised status. This condition is usually contracted following suppression of the immune complex system; this can become local or

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systemic and is found in chronic systemic steroid and antibiotic usage, extreme age groups, and immune system-compromising diseases (such as HIV/AIDS) [1]. Other Candida species that can cause oral candidiasis are *C. krusei*, *C. glabrata,* and *C. tropicalis* [2]. This infection can also be called oral thrush. The word "thrush" refers to the similarity between the white flecks present in some forms of candidiasis with the chest of the bird named thrush [3]. In the mouth, oral thrush can form creamy white spots or patches, most often on the tongue. It can be present in the inner cheeks and can spread to other parts of the buccal mucosa, such as the gums, the roof of the mouth, the tonsils, and the posterior part of the throat. Around 75% of healthy adults carry *Candida* species in their mouths, and when a count is greater than 400 colony-forming units (CFU) per mL, an ongoing oral candidiasis infection is detected[4]. Based on clinical presentation, oral candidiasis can be categorized into acute pseudomembranous candidosis, acute erythematous candidosis, chronic hyperplastic candidosis, and chronic erythematous candidosis. Diagnosis for this disease includes microscopy, culture and molecular methods. *C. albicans* appears as ovoid or spherical budding cells under the microscope. Staining can be done to smears with Gomori methenamine silver (GMS) stain or Periodic Acid-Schiff (PAS) stain, or by doing a culture with a swab from an oral rinse using Sabouraud's agar [5]. The Gram-stained smears or wet films from the exudates or lesions can be used to find budding Gram-positive cells. The collected specimen must be from an active and fresh lesion, as old lesions often do not contain viable microorganisms [5]. The cultured colonies appear creamy, white and smooth, and they can have a yeasty odour. A rapid test to identify *Candida albicans* is the germ tube test, which makes use of the ability of *C. albicans* to form germ tubes within two hours when they are incubated in human serum at 37 degrees Celsius. This process is also known as the Reynolds-Braude phenomenon. Oral candidiasis can be treated. Removal of heavy candidal plaques can facilitate antifungal action and accelerate healing [5]. Important antifungal drugs include Nystatin and azoles such as miconazole, clotrimazole, and ketoconazole. Efficient oral hygiene, especially for individuals who come under vulnerable age groups, can help prevent such fungal infections from taking over. An anti-Candida vaccine could help improve the current situation for people suffering from this disease.

**Etiopathogenesis:**

Candida species are part of the normal microbial flora of human skin and mucosa. They are known to produce structures called pseudomycelia, which are often used in identification in culture. In cases of immune suppression, most commonly diabetes, these microorganisms are seen to cause infection in the host cells; hence, it is an opportunistic endogenous infection. Other predisposing factors include compromised age groups, prolonged use of systemic steroids or antibiotics, and diseases that affect the immune system, such as HIV/AIDS [1]. Chronic mucocutaneous candidiasis and Candida granuloma are serious manifestations that can occur in immunodeficiencies. Oral thrush is also commonly observed in bottle-fed infants and in debilitated individuals.

The pathogenesis of *C. albicans* begins with its conversion from the yeast-like form to the invasive hyphal form. Since the oral mucosa is moist, it provides a suitable environment for *Candida* species to grow. The *Candida* species adheres to the epithelial cells through reversible weak interactions that include electrostatic and hydrophobic forces, which an important virulence factors for persistence of the infection. [6] (The cell wall-associated glycoproteins that give rise to adhesion of *C. albicans* to the epithelial surfaces are coded for by *Candida* genes of HWP1 (hyphal wall protein) and the ALS (agglutination-like sequence) family. [6] *C. albicans* releases hydrolytic enzymes, toxins, and extracellular vesicles as part of its set of virulence factors. Following this, the immune cells of the host get activated, and the epithelial cells are stimulated. In the later stages, *C. albicans* can form polymicrobial biofilms that are composed of a wide range of bacterial species that are present in the host's oral cavity.

The biofilm can include bacterial species such as:

- *F. nucleatum*
- *L. salivarius*
- *P. gingivalis*
- *P. intermedia*
- *P. aeruginosa*

The fungi present in it can include:

- *C. albicans*

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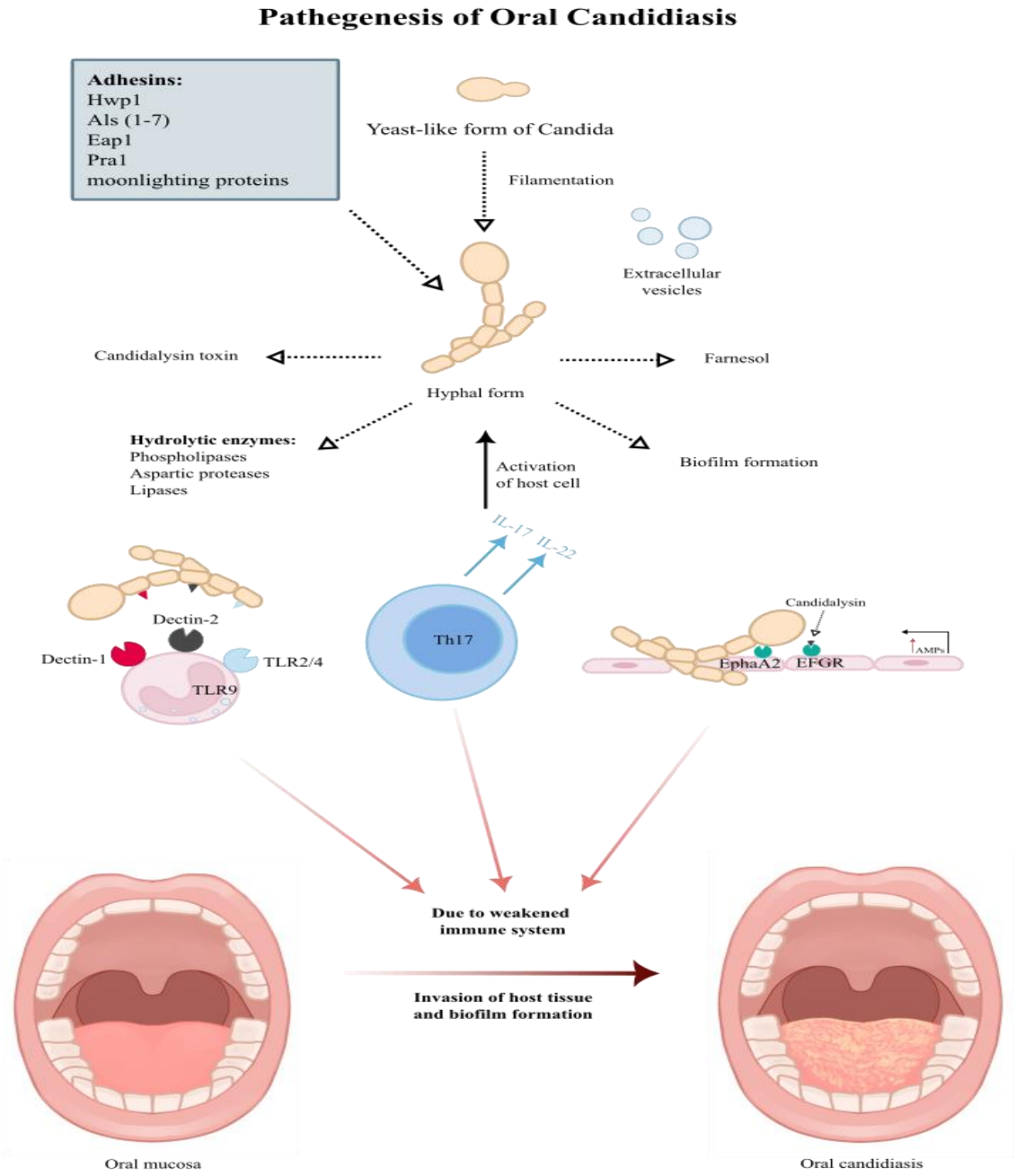
- *C. krusei*
- *C. glabrata*
- *C. tropicalis*
- *C. parapsilosis.*

**Pathogenesis of Oral Candidiasis:**



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The reaction is shown between the virulence factors of *Candida albicans* and the immune response of the host. The conversion of *C. albicans* from the yeast-like form to the hyphal form makes it more invasive to the host. The virulence factors include toxins, the release of hydrolytic enzymes, and extracellular vesicles. As the infection develops, the host's immune cells are activated, and the epithelial cells are stimulated. *C. albicans* can form polymicrobial biofilms with a diverse range of bacterial species that are present in the oral cavity in the later stages.

**Epidemiology and Risk Factors:**

Oral candidiasis is an opportunistic infection that is usually seen in individuals of weakened immunity, such as those who are under immunosuppression or have certain diseases. People who are suffering from diabetes mellitus are at a higher risk of having infections like oral candidiasis and periodontal and gingival diseases [(7).] HIV/AIDS, which is known to compromise the immune system, can also lead to oral candidiasis. The rise of interest and research into these oral infections caused by *Candida* in the 1980s has been greatly associated with the increase in cases of HIV infection and the AIDS epidemic [(6)]. Other factors that can lead to oral candidiasis are extremes of age, chronic steroid use, and chronic antibiotic use. Fungal overgrowth is promoted using antibiotics (broad-spectrum) and corticosteroids, as they disturb the normal flora of the buccal cavity, which can predispose them to oral candidiasis. The use of immunosuppressive therapies, invasive procedures, and head and neck radiation therapy has also been a cause of this issue in the population. [(6)]

**Clinical Manifestations and Classification:**

Oral candidiasis can be categorized into four types based on clinical presentation: acute pseudomembranous candidiasis, acute erythematous candidiasis, chronic hyperplastic candidiasis, and chronic erythematous candidiasis. The clinical manifestation for each of the types included in oral candidiasis is as follows:

**Acute pseudomembranous candidiasis:**

This is the most common type, also called thrush. It includes white patches that can be removed gently, and the area appears red. Immunocompromised individuals contract this type of oral candidiasis. A burning sensation is only expressed by patients, and they are usually asymptomatic.

**Acute erythematous candidiasis:**

In acute atrophic candidiasis, erythematous patches are seen. They can arise newly or when the plaques of thrush, which are white in colour, are shed. This infection may occur when patients are undergoing the course of an antibiotic, as part of the normal flora is lost due to the use of these drugs. In this case, the infection can subside when the treatment is finished.

**Chronic hyperplastic candidiasis:**

This is also called candidal leukoplakia and is uncommon. The plaques are white to translucent and appear to be slightly raised in this case. They are hard to remove by scraping. This is commonly found in male smokers who are in the middle-aged group.

**Chronic erythematous candidiasis:**

An erythematous form called chronic atrophic candidiasis is usually seen in people wearing dentures. Salivary flow is inhibited if the denture is not fitted properly, as the oral mucosa gets occluded. In the blocked areas, erythematous oedema and inflammation are formed. HIV patients may contract this form of chronic erythematous candidiasis.

Other types that can be identified by clinical evaluation include median rhomboid glossitis, angular cheilitis, denture stomatitis, and linear gingival erythema. [(4)]

| Type of Oral Candidiasis | Clinical Manifestations |
|---|---|
| Acute pseudomembranous candidiasis | White patches appear on the oral mucosa that can be removed, and the area appears red. |
| Acute erythematous candidiasis | Erythematous patches are present, which can arise newly or when the plaques of thrush, white in colour, are shed. |

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| | |
|---|---|
| Chronic hyperplastic candidiasis | White to translucent plaques, which are slightly raised, appear. They are hard to remove by scraping. |
| Chronic erythematous candidiasis | Erythematous oedema and inflammation are formed. |

**Diagnostic Approaches:**

Currently, the diagnosis of *Candida* can be done by culture-based and non-culture-based methods. Isolation of *Candida* can be done by taking smears and swabs from the lesion or by using the impression, imprint, or salivary culture technique or oral rinsing. [8] Culture-based methods for diagnosis include the following**:**

**Culture method**

Culture media such as Saboraud's Dextrose Agar (SDA) can be used to grow and identify the presence of *Candida* in samples. Chromogenic agar Candida media is a selective and differential medium that is used for growing *Candida,* which can help identify the different species of *Candida* present in the specimen.

**Chlamydospore formation test**

This test is used to check for chlamydospore-forming species of *Candida*. Cornmeal agar is used in this case, and the species that are known to produce chlamydospores are as follows:

- C. albicans
- C. tropicalis (a few strains)
- C. dubliniensis

**The non-culture-based methods:**

This includes the germ tube test, biochemical methods, and molecular-based Candida detection methods. The germ tube test can be used to detect the presence of *Candida albicans.* The germ tube is a non-septate germinating hypha, which is seen as short outgrowths elongating from the mother cell. [9] The germ tube formation is also known as the Reynolds-Braude phenomenon. The *Candida* species should be incubated in serum at a temperature of 37 degrees Celsius for about 2-4 hours. *C. dubliniensis* is also known to produce germ tubes, and *C. tropicalis* can form pseudo hyphae, which can look like germ tubes. The biochemical tests include EIAs (enzyme immunoassays) for identifying circulating mannans and biochemical tests and (1→3)-β-D-glucan. (9) Polymerase chain reaction remains the gold standard analysis at a molecular level due to its resolution effect on the differentiation of microorganisms. (9) Genes such as the internal transcribed spacer (ITS) region, D1/D2 region, and the IGS1/IGS2 region can be used for molecular diagnosis. MALDI-TOF MS (matrix-assisted laser desorption/ionisation-time of flight mass spectrometry) has gained interest of researchers recently, and it can be used to rapidly identify Candida species.

**Current Therapeutic Strategies:**

**Topical**

In oral candidiasis, topical treatment is considered for mild cases and can include antifungals taken orally, gels, and mouth rinses. Antifungals such as fluconazole (100 mg taken orally, daily for 7 days), ketoconazole (200–400 mg taken orally along with breakfast [requires acidic gastric environment for absorption] for 7–14 days), nystatin mouth rinses (500,000 units [5 mL of 100,000 units/mL] held in the mouth before swallowing, three times daily), and clotrimazole troches (10 mg dissolved and taken orally, five times daily) can be used for treatment. (10) Individuals who wear dentures can use the antifungal Nystatin.

**Systemic**

People who have tested positive for HIV are given longer courses. In this case, antifungals such as fluconazole can be used. Fluconazole can be administered through an IV or by pills.

**Antifungal Resistance and Challenges in Treatment:**

The resistance to antifungal drugs used in treatment can render them useless and lead to the patient receiving an alternate treatment plan. The quantification of the susceptibility of the fungal isolates to the antifungal drugs is done by determining the minimum inhibitory concentration (MIC) through EUCAST or CLSI methods. [11] The MIC can be used to check if the fungal isolate is susceptible, intermediate, resistant, or dependent. Resistance in oral candidiasis can occur

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through mechanisms that include the ERG-11 gene mutation, efflux pump activation, ERG-11 gene expression dysregulation, and the changes that influence the biosynthesis pathway of ergosterol. [12] Certain antifungals, such as azoles, were reported to be involved in antifungal resistance among patients. In a systematic review and meta-analysis, which was aimed at determining the prevalence of drug-resistant oral candidiasis among HIV-positive patients, findings indicated that the pooled prevalence of resistance to azoles and 5-flucytosine was relatively high, ranging between 13.4% and 25.5%. [13] Treatment using azoles and such drugs that are prone to resistance may not be an effective treatment choice. Healthcare providers could prescribe alternative drugs like caspofungin and polyenes in the case of resistance to azoles and 5-flucytosine. [13] Hence, it is vital to examine the resistance profiles of drugs before initiation of treatment to prevent antifungal resistance. Emerging studies have revealed that the oral mycobiome plays a crucial role in maintaining mucosal integrity and controlling fungal colonization. Disruption of this balance, along with immunological dysregulation, increases the risk of chronic and recurrent Candida infections [18]. Moreover, host-targeted therapies involving cytokines such as IL-17 and IL-22, as well as toll-like receptor modulation, are being explored to enhance antifungal immunity without promoting resistance [19]. Recent advances in molecular techniques have highlighted the role of Candida albicans virulence genotype variance in mediating drug resistance and immune evasion. Efflux pump overexpression and ERG11 mutation are central to azole resistance, but emerging studies show that mitochondrial dysfunction and epigenetic modulations also modulate antifungal tolerance [20]. Combination therapies with echinocandins and azoles are being investigated to target both ergosterol synthesis and β-1,3-glucan pathways [21]. These strategies reflect the shift from pathogen-targeted to resistance-aware therapeutics in clinical mycology.

**Innovative and Emerging Therapies:**

As antifungal resistance is a growing threat, certain alternatives are underway for the effective treatment of oral candidiasis. Antifungal drugs that have been approved recently for candidiasis include [14]:

- Caspofungin
- Isavuconazole
- Ibrexafungerp
- Oteseconazole
- Rezafungin

Studies are underway on probiotics, as they have been found to have beneficial effects on the host and help counter fungal infections. Many studies have proven the therapeutic and preventive effects of good bacteria, a few of which include metabolic functions such as fermentation of indigestible fibres, lactose tolerance, production of short-chain fatty acids, vitamin production, and cholesterol level reduction. [4] Probiotics can be defined as live microorganisms that, when administered in sufficient amounts, produce health benefits to the host. [15] Furthermore, good bacteria have antimicrobial activity, such as production of bacteriocins, competitive inhibition of pathogens, antitoxin effects, and enhancement of intestinal barrier function (by increasing mucus production, tight junction proteins, and goblet and Paneth cells). [4] They are also involved in stimulating an immune response in the host. Probiotics used for mucosal candidiasis in an in vivo study by Wagner in 1997 showed antifungal action. [4] Hence, probiotics have the potential to enter treatment regimens for oral candidiasis. Recent advances in nanotechnology have provided novel antifungal drug delivery systems that significantly improve target specificity and bioavailability. Nanoparticle-based formulations of fluconazole and amphotericin B have demonstrated better penetration of biofilms and reduced systemic toxicity [22]. Another promising research area is the development of biofilm-disrupting agents—such as antimicrobial peptides and quorum-sensing inhibitors—which aim to dismantle the protective biofilm matrix that shields Candida from antifungal agents [23].

Photodynamic therapy (PDT) is a method that has promising potential in the treatment of oral candidiasis. This method uses a photosensitizer, which is applied to the diseased tissue, and a photochemical reaction is produced by using irradiation with a specific wavelength light source to achieve a therapeutic effect. [16] The development of nanoparticle-based antifungal delivery systems displays a promising trend for precision medicine. Silver and chitosan nanoparticles have

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demonstrated potent anti-Candida effects with biofilm penetration capability [24]. Liposomal amphotericin B formulations enhance safety profiles while retaining fungicidal efficacy [25]. Additionally, CRISPR-Cas-mediated genetic modulation is an emerging research direction to suppress virulence gene expression and restore antifungal susceptibility [26]. Gene editing strategies have already reduced hyphal transition and phospholipase gene expression in Candida isolates in vitro.

**Prevention, Prophylactic Measures and Future Perspectives:**

The preventive measures primarily include maintaining good oral hygiene. The use of corticosteroids and antibiotics for infections should be done according to the treatment plan of the healthcare provider, and he/she must be notified if there are any symptoms that result due to the medications. Alongside antifungal drug development, vaccines show potential in prevention and reduction in costs. Currently, there are no commercially available vaccines for *Candida* infections, but ongoing research hopes to find the benefits that could be seen with its production. The rise in antifungal resistance pushes the need for an effective vaccine further and has also led researchers to find new antifungal drugs. The successful production of a vaccine can led to significant cost reductions in the healthcare sector, as patients (such as immunocompromised individuals) require extended hospital stays and antifungal medications, which are costly, and may even require surgery. [17] Some vaccines are under clinical trials, among which NDV-3A is showing the most promising results regarding human testing. The challenges of vaccine production include the adaptability and variability of *Candida*, impaired adaptive immunity, and pre-existing immunological tolerance in some individuals. On a global scale, Candida species continue to show geographic variations in prevalence and drug resistance patterns, necessitating the implementation of surveillance-based management strategies. The global incidence of mucosal candidiasis is rising in ageing and immunosuppressed populations, underscoring the need for innovative diagnostic and preventive interventions [17] [27]. Host-microbiome research has identified that the oral mycobiome interacts with bacterial symbionts like Streptococcus mitis and Actinomyces naeslundii, influencing Candida adhesion and immune responses [28]. Maintenance of oral microbial homeostasis may therefore be essential for preventing candidiasis recurrence. Immunotherapeutic interventions targeting IL-17 and IL-22 cytokine pathways show substantial promise in control and recurrence management [29]. Longitudinal surveillance indicates an increasing prevalence of non-albicans Candida species in immunocompromised populations globally [30]. Artificial intelligence-based diagnostic algorithms using MALDI-TOF and metagenomic sequencing have also improved rapid species identification and antifungal stewardship [31]. Finally, antimicrobial peptides (AMPs) derived from human saliva are being explored for their synergistic effects with antifungal nanocarriers [32].

**Conclusion:**

Oral candidiasis is an opportunistic infection that can be seen mainly in individuals with a weakened or compromised immune system. Patients with diseases such as diabetes mellitus or HIV are prone to oral candidiasis. Other factors include age (very young and very old), chronic usage of corticosteroids or antibiotics, radiation, denture wear, and smoking. Antifungals are commercially available, but there is a rising concern for antifungal resistance (especially among azoles) in the healthcare sector. The current research and development for a *Candida* vaccine holds the promise of possibly providing adequate prevention and protection from this fungal infection. Vaccines can be cost-effective, as they reduce medication costs, hospital stays, and the cases that require surgeries. Maintaining good oral hygiene and early detection with treatment can help reduce the occurrence of oral candidiasis among immunocompromised individuals.

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