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ROLE OF FUNGI IN ORAL INFECTIONS – A COMPREHENSIVE LITERATURE REVIEW FOR DENTAL PROFESSIONALS

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ABSTRACT

For thousands of years, oral fungal infections have existed in human history. Hippocrates, who lived between 460 and 370 BCE, was the first person to report occurrences of oral aphthae that could have been oral candidiasis. While infections like cryptococcosis, aspergillosis, and mucormycosis are relatively rare in the mouth, oral candidiasis became widely recognized during the AIDS epidemic. It was a major opportunistic infection, signaling the progression from HIV to AIDS. This highlighted the vital role of our immune system in preventing fungal infections. Over time, research has revealed that our oral immune defenses respond to fungal infections in unique ways. Scientists now understand that these infections don't just result from interactions between the host and the fungus, but they are also influenced by other microbes in the mouth. Recent findings suggest that different microorganisms communicate with each other, affecting how infections develop and progress. In the past decade, there have been exciting breakthroughs in analyzing the oral microbiome. We can now study fungi, bacteria, archaea, protozoa, and even viruses like SARS-CoV-2 more precisely than ever before. At the same time, we can measure immune responses, including immune cells, cytokines, and microbial signals, giving us a more complete picture of oral health. With the rise of artificial intelligence and machine learning, there's a real opportunity to refine our understanding of oral fungal infections. By combining all this knowledge, we can improve diagnostics, predict who may be more vulnerable, and ultimately help people stay healthier by detecting infections earlier.

INTRODUCTION

The oral cavity constitutes a distinct ecological niche for microbial colonization, which serves as a primary entry point to the human body. It presents a diverse array of surfaces for microbial attachment, ranging from the hard, non-shedding surfaces of the teeth to desquamating keratinized and non-keratinized epithelia. In certain individuals, dental prosthetics introduce additional materials, such as acrylic, polyurethane, ceramic, and metal alloys, all susceptible to colonization. The continuous saliva flow across these surfaces maintains warmth and moisture, further promoting microbial habitation. As a result, the oral cavity harbors a complex and dynamic microbiota1-3, which under normal conditions, is largely non-pathogenic and contributes to inhibiting the colonization of pathogenic microorganisms. However, disturbances in the equilibrium of this microbial community, known as microbial dysbiosis, can precipitate disease and tissue damage, mediated by various microorganisms, including bacteria, archaea, fungi, and viruses.

Oral fungal infections, though less common, can still lead to significant morbidity[4,5], with Candida species being the primary etiological agents. Other less frequent fungal pathogens with oral manifestations include mucormycosis, aspergillosis, blastomycosis, histoplasmosis, geotrichosis, rhinosporidiosis,

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cryptococcosis, and coccidioidomycosis[5]. Effective treatment of these infections relies on a thorough understanding of their etiology. While visual examination of symptoms may provide initial clues, accurate treatment demands precise pathogen identification and a deep understanding of the mechanisms driving disease.

In recent years, the study of oral microorganisms has advanced considerably, shifting from traditional culture-based methods to modern DNA detection and sequencing technologies[6]. This progress has allowed researchers to better associate disease pathogenesis with specific virulence factors, such as the tissue-degrading enzymes and cytotoxic compounds produced by Candida albicans[7, 8.] However, it has become increasingly clear that the development of oral fungal infections is not solely dependent on fungal virulence but is also shaped by the host's immune defenses. Moreover, these infections may not always be caused by a single strain but could involve the coordinated activity of multiple fungal species, driven by interspecies communication and influenced by the host environment. As the causes of immune compromise continue to change and comorbidities become more prevalent, the need for advanced methods to detect, manage, and ideally prevent oral fungal diseases has become more pressing. This mini-review will examine recent progress in understanding the causes of these infections, provide an assessment of current research, and highlight key areas for future exploration.

Etiology of oral fungal infections

Oral fungal infections are most frequently caused by Candida species, with Candida albicans being the most prevalent. Candida tropicalis, Candida glabrata, Candida parapsilosis, Candida krusei, and Candida guilliermondii are among the non-Albicans species that are on the rise, though. Other fungi can also lead to oral lesions, including Histoplasma capsulatum (histoplasmosis), Cryptococcus neoformans (cryptococcosis), Aspergillus species (aspergillosis), and Mucor species (mucormycosis). Additionally, Geotrichum candidum can cause geotrichosis, while Blastomyces dermatitidis is responsible for blastomycosis. Understanding these pathogens helps in early diagnosis and better treatment.[7, 9]

Predisposing Factors for Oral Fungal Infections

Oral fungal infections, particularly oral candidiasis, occur when the balance of microorganisms in the mouth is disrupted, allowing fungal overgrowth. Several factors can predispose individuals to these infections, which are discussed below.

Reduced saliva production decreases antimicrobial activity and promotes fungal colonization. Common causes are Aging, Sjögren's syndrome, radiation therapy, polypharmacy (multiple medications), and dehydration. Salivary hypo function, or reduced saliva production, increases the risk of oral fungal infections, particularly oral candidiasis, by disrupting the natural balance of the oral environment. Saliva contains antimicrobial proteins like lysozyme, lactoferrin, and histatins, which help control fungal growth, and its reduction weakens these defences, allowing Candida to thrive. A dry mouth also alters the oral microbiome, shifting the balance in favour of fungal colonization. Additionally, saliva plays a crucial role in lubricating oral tissues, and its absence leads to mucosal fragility, increasing the risk of irritation, ulceration, and fungal invasion. Saliva also helps clear excess microorganisms from the oral cavity, and without sufficient flow, fungi can adhere to mucosal surfaces and form biofilms, making infections more persistent. Furthermore, saliva supports immune function by transporting immunoglobulins, particularly IgA, and its deficiency weakens immune surveillance, making the body more susceptible to opportunistic infections. Denture wearers with dry mouth are especially vulnerable, as reduced saliva creates an ideal environment for fungal colonization under prostheses, leading to denture-related stomatitis. Ensuring proper hydration and maintaining good oral hygiene can help mitigate these risks and support overall oral health.[6,7,8,10]

Weakened immune defences allow opportunistic fungi like *Candida albicans* to thrive. Causes include HIV/AIDS, chemotherapy, organ transplantation, corticosteroid therapy, uncontrolled diabetes. Accumulation of plaque and food debris provides a favourable environment for fungal growth. Infrequent brushing, flossing, and tongue cleaning increase fungal colonization. Dentures create a moist, oxygen-limited environment ideal for *Candida* biofilm formation. Poorly fitting or unclean dentures increase fungal adhesion and infection risk. Broad-spectrum antibiotics disrupt the natural bacterial balance, reducing competition and allowing fungi to overgrow. Fungi like *Candida* thrive on sugar, and excessive carbohydrate consumption can promote fungal overgrowth. Smoking alters the oral microbiome and damages mucosal defences, increasing fungal colonization. Alcohol can contribute to dry mouth and disrupt oral microbial balance.[8,9]

Some of the other factors are high blood sugar levels which enhance fungal growth and impair immune response. Hormonal changes like pregnancy, menopause, or hormonal therapy may alter oral conditions, increasing fungal susceptibility. Asthma and COPD patients using steroid inhalers are at risk of developing oropharyngeal candidiasis (thrush).Fungal development is accelerated by improper use, such as failing to rinse the mouth after use. Radiation therapy (especially to the head and neck) can damage salivary glands, causing dry mouth. Chemotherapy weakens the immune system, making fungal infections more likely.[10,11]

Pathogenesis of oral fungal infections

1. Oral candidiasis

The following stages make up the pathophysiology of oral candidiasis. Using adhesins and extracellular polymeric molecules, Candida cells attach themselves to oral epithelial cells to promote colonization [12.] *C. albicans* can switch from a yeast form to a hyphal form, which is associated with tissue invasion and increased pathogenicity[12]. The formation of biofilms on oral surfaces, such as dentures, provides a protective environment for Candida, enhancing its resistance to antifungal agents. Candida has a number of strategies to get around the host immune system, such as modifying immunological responses and secreting enzymes that break down host proteins. Patients with oral candidiasis may exhibit creamy white lesions on their tonsils, gums, inner cheeks, or tongue as a clinical symptom. It is frequently possible to wipe these patches off, leaving behind red, swollen, and perhaps bleeding spots. Symptoms can include soreness, a cottony feeling in the mouth, and loss of taste.[13]

2.Aspergillosis

Aspergillus spores (conidia) are ubiquitous in the environment and can enter the oral cavity through inhalation or direct contamination. In immunocompetent individuals, saliva, mucosal immunity, and mechanical cleansing prevent fungal adherence. However, in susceptible individuals (e.g., those with diabetes, prolonged corticosteroid use, or immunosuppression), the fungus can colonize oral tissues[14]. The fungus adheres to damaged oral mucosa, especially in cases of trauma, recent dental extractions, or prolonged denture use. Hyphae penetrate deeper into the epithelium and connective tissues, leading to necrosis. Invasive forms may involve blood vessels (angioinvasion), leading to ischemia and tissue destruction[15]. The host immune system uses neutrophils and macrophages to try to regulate fungal development. In immunocompromised patients (e.g., those with HIV, leukaemia, or undergoing chemotherapy), the immune response is ineffective, allowing rapid fungal spread. Tissue necrosis, ulceration, and secondary bacterial infections can develop. Severe cases may extend to the maxillary sinus, palate, or even the brain (rhinocerebral aspergillosis) [16].

Clinically, oral aspergillosis presents as painful ulcerative lesions, usually on the palate or gingiva, with greyish-black necrotic tissue due to vascular invasion. Symptoms may include swelling, erythema, and occasional bleeding[17]. Antifungal treatment and early detection are crucial to preventing issues.

3. Mucormycosis

Mucormycosis is a rare but aggressive fungal infection caused by fungi in the order Mucorales, with *Rhizopus* species being the most common culprits. The Pathogenesis of Oral Mucormycosis is given below.

The spores can be inhaled or consumed by humans, and the fungi are found throughout the environment. In sensitive individuals, the spores attach to the oral mucosa, while in immunocompetent persons, they are typically eliminated without producing illness. The fungi exhibit a propensity for vascular invasion, leading to thrombosis (blood clot formation) and subsequent tissue necrosis (death). This vascular invasion is a hallmark of mucormycosis and contributes to its rapid progression. The fungal hyphae's invasion of blood arteries reduces blood flow, which leads to tissue ischemia and necrosis. Clinically, this presents as black necrotic lesions in the oral cavity, often accompanied by pain and swelling.

Uncontrolled diabetes, especially when associated with ketoacidosis, is a significant risk factor. The acidic environment and elevated blood glucose levels promote fungal growth and impair the immune response. Patients undergoing chemotherapy, organ transplantation, or those with conditions like HIV/AIDS have weakened immune defences, making them more susceptible to mucormycosis. Long-term corticosteroid use can weaken the immune system, which raises the possibility of fungal infections. There has been an observed increase in mucormycosis cases among individuals recovering from COVID-19, particularly those treated with corticosteroids or those with pre-existing diabetes[18].

Clinically, it is seen as painful ulcers with a characteristic black eschar (dead tissue) may develop on the palate or other areas of the oral mucosa. Redness and swelling may be seen in affected areas, which frequently worsen quickly. Teeth loss or loosening may result from maxillary involvement. Facial pain and edema may result from the infection spreading to the sinuses. Prompt recognition of symptoms and immediate medical attention are crucial due to the rapid progression of the disease. Biopsy of the affected tissue, followed by microscopic examination, can confirm the presence of broad, non-septate hyphae characteristic of mucormycosis. The cornerstone of treatment is the administration of antifungal medications, such as amphotericin B. Removal of necrotic tissue is often necessary to control the spread of the infection. Understanding the pathogenesis of oral mucormycosis is vital for early diagnosis and effective management, especially in high-risk populations.

4. Histoplasmosis

This fungal infection is brought on by Histoplasma capsulatum, a dimorphic fungus that is common in areas with a lot of bird or bat droppings. While the primary route of infection is through inhalation of spores leading to pulmonary involvement, the pathogen can disseminate and manifest in various body sites, including the oral cavity.

The pathogenesis of oral histoplasmosis includes the following. Inhalation of *H. capsulatum* spores occurs when contaminated soil or droppings are disturbed, releasing microconidia into the air. When these spores get to the lungs' alveoli, macrophages phagocytose them. Within these immune cells, the spores convert into their yeast form, enabling survival and replication[19]. In individuals with weakened immune systems, the yeast can evade the host's immune defences and disseminate via hematogenousroute to various organs, including the oral mucosa. The propensity for dissemination is influenced by factors such as the number of inhaled spores, host immune status, and specific fungal strain virulence[19]. Once in the oral cavity, *H. capsulatum* can cause lesions that may present as non-healing ulcers, nodules, or granulomatous masses. These lesions are often painful and can mimic other conditions, such as malignancies or other infections, making diagnosis challenging[19]. Individuals with compromised immune systems, such as those with HIV/AIDS, organ transplant recipients, or patients on immunosuppressive therapies, are at heightened risk for disseminated histoplasmosis with oral involvement[19]. Exposure to H. capsulatum-endemic environments, particularly those with large concentrations of bird or bat droppings, increases the chance of a first infection[20].

Clinical presentation shows lesions may appear as painful ulcers with irregular borders, commonly affecting the tongue, palate, buccal mucosa, and gingiva. These lesions can persist for weeks and may be mistaken for malignant ulcers[21]. Definitive diagnosis is achieved through biopsy of the lesion, with histopathological examination revealing granulomatous inflammation and identification of the yeast forms of *H. capsulatum* within macrophages[21]. Treatment typically involves antifungal agents such as itraconazole for mild to moderate cases, while more severe or disseminated infections may require amphotericin B. To avoid problems, early detection and the right treatment are essential [21]. Understanding the pathogenesis of oral histoplasmosis is essential for timely diagnosis and effective management, especially in individuals with risk factors for dissemination

5. Blastomycosis

Once in the oral mucosa, *Blastomyces* yeast form induces a granulomatous inflammatory response, leading to tissue destruction. Oral lesions may appear asulcerated nodules or plaques,verrucous (wart-like) growths and painful, necrotic ulcers with irregular borders. The lesions mimic squamous cell carcinoma, tuberculosis, or deep fungal infections like histoplasmosis. Innate immunity (macrophages and neutrophils) attempts to contain the fungus, but *B. dermatitidis* can survive intracellularly. Cell-mediated immunity (T-helper 1 response) plays a key role in controlling infection by activating macrophages to kill yeast cells. In immunocompromised individuals, the infection becomes severe, leading to widespread tissue destruction and deeper bone involvement (osteomyelitis of the jaw).

6. Cryptococcosis

Cryptococcal infection begins when tiny fungal spores are inhaled into the lungs. As the immune system responds immediately, the spores are engulfed by alveolar macrophages, the body's first line of defense in the lungs. These macrophages then signal other immune cells, triggering a helper T-cell response to combat the infection. However, Cryptococcus has evolved remarkable survival strategies to outwit the immune system. Once inside the macrophages, the fungi can remain dormant within specialized compartments called phagolysosomes, sometimes staying latent for years in thoracic lymph nodes without causing any symptoms. This dormancy is largely due to the fungus's prominent mucopolysaccharide capsule, which serves as a protective shield against the host's immune defenses. Other virulence factors, such as melanin production, phospholipase, and urease activity, also enhance the fungus's ability to survive and thrive within the hostile environment of the macrophages. Despite the immune system's best efforts, Cryptococcus can produce mucin, which damages and ultimately bursts the macrophages. This process leads to granulomatous inflammation[22], where the immune system walls off the infection to contain it. However, in some cases, the infection can progress without this protective granulomatous response, either becoming symptomatic or spreading to other parts of the body. For individuals with weakened immune systems, the situation becomes even more concerning. In certain situations, the yeast grows out of control, entering the bloodstream and passing past the blood-brain barrier to enter the brain[23]. This can lead to severe complications, such as cryptococcal meningitis. The ability of Cryptococcus to adapt and survive in the body highlights the intricate interplay between pathogens and the immune system. While the fungus can pose significant challenges, understanding its mechanisms helps healthcare providers devise better strategies for managing and treating cryptococcosis. For individuals, maintaining overall health and promptly addressing symptoms can make a significant difference in outcomes, particularly for those at higher risk due to immunodeficiencies.

7. Geotrichosis

Geotrichum is a natural part of the normal oral flora[24]. For individuals with a healthy immune system, this fungus poses no threat and does not cause disease. It's simply one of the many microorganisms contributing to the oral cavity's balance. This harmless relationship underscores the remarkable resilience of the human body and its ability to maintain harmony with countless microorganisms that live on and within us. However, it's always important to keep overall health in check, as a strong immune system plays a key role in ensuring that Geotrichum and other microorganisms remain benign and non-pathogenic.

8. Rhinosporidiosis

The infection caused by R. seeberi is typically thought to occur through the epithelium, especially in areas like the nose, where it's believed that the infection begins when free endospores, released from ruptured sporangia, come into contact with previously damaged epithelial tissue[25]. These endospores then become activated and continue their journey of invasion, maturing into trophocytes (both early and late forms), and the cycle starts over again[26].Interestingly, the infection can sometimes spread to distant skin areas, far from the initial site (such as the nose), through mechanisms like hematologic or lymphatic spread, autoinoculation, or direct inoculation. This can result in skin lesions appearing in places that weren't originally affected. R. seeberi has been detected in people, birds, and other mammals, despite having no particular host. It is currently uncertain exactly how the sickness spreads. It's still unknown how exactly the illness spreads. There's been no confirmed cross-infection between humans in the same family or between animals and humans[27]. However, it's possible that contact with free spores in aquatic or marshy areas could explain the infection in both humans and animals. In drier areas, it has also been suggested that spores may be transmitted through the air. When it comes to the immune response, some studies suggest that both the endospores and the layers of the sporangia play a significant role in triggering the immune system in the host. Additionally, the virulence of the infection may differ depending on the genetic makeup of the strain, which highlights the complexity and variation in the response to R. seeberi[76]. Both cellular and humoral immune responses are activated in mammals, further illustrating the body's efforts to combat this microorganism[28].

Laboratory diagnosis of Oral Fungal infections

Laboratory diagnosis of fungal infections typically includes specimen collection, microscopy, cultivation and identification. Using sterile swabs, the samples were taken from the denture surfaces, palate, tongue, or lesions. In oral rinse technique the patient rinses with 10 mL phosphate-buffered saline (PBS), and then expectorates for analysis. For persistent infections, histopathological examination may be needed. Potassium hydroxide (KOH) mount (10–20%) dissolves keratin, making fungal elements (hyphae, pseudohyphae, yeast cells) visible under a light microscope. Useful for rapid presumptive diagnosis (Williams & Lewis, 2011).Candida species are characterized by gram-positive, oval, budding yeast cells with pseudohyphae. Not species-specific but aids in initial identification (Martins et al., 2014). In Calcofluor white staining fluorescent stain binds to fungal cell walls, enhancing visibility under a fluorescence microscope (Kumar et al., 2019). Sabouraud Dextrose Agar (SDA) supports fungal growth; colonies appear cream-colored and pasty. Requires 24–48 hours at 37°C (Samaranayake et al., 2009). CHROMagarTM Candida sets itself apart Species of Candida according to colony color: According to Pincus et al. (2007), Candida albicans: Green, Candida tropicalis: Blue, and Candida krusei: Pink rough colonies. Tween 80 added to

cornmeal agar promotes chlamydospores development, which helps identify Candida albicans.

The following are examples of identification tests. Ingerm tube test, *C. albicans* and *C. dubliniensis* form germ tubes in serum or plasma within 2–3 hours at 37°C. The test is negative in non-*albicans* species. Carbohydrate assimilation & fermentation test determines metabolic patterns of *Candida* spp. Automated systems like API 20C AUX aid identification. In urease test,*C. krusei* and *C. glabrata* are urease-positive; *C. albicans* is negative. Polymerase chain reaction (PCR) detects fungal DNA, allowing species-specific identification. Real-time PCR (qPCR) quantifies fungal load and detects antifungal resistance genes. Periodic acid-Schiff (PAS) & Gomori Methenamine Silver (GMS) Stains stain fungal hyphae and yeast cells in tissue samples (Naglik et al., 2008).- B-D-Glucandetection indicates systemic fungal infections (Richardson & Lass-Flörl, 2008). Mannan antigen test helps diagnose invasive candidiasis[29-33].

Future prospects and the clinical implications of fungal infections

The number of immunocompromised individuals is increasing due to aging, cancer, and various medical treatments. Aging weakens immune responses, while cancer treatments often suppress immunity, increasing susceptibility to fungal infections. The global cancer burden is expected to rise to 28.4 million cases by 2040, a 47% increase from 2020[34]. Additionally, the use of immunosuppressive drugs like dexamethasone for COVID-19 has led to a rise in mucormycosis cases, particularly in India[35, 36]. This serious fungal infection mainly affects the nose, sinuses, eyes, and brain. A systematic review of post-COVID-19 fungal infections in the maxillofacial region highlights significant cases of candidiasis, mucormycosis, and aspergillosis, indicating a broader spectrum of oral fungal infections in immunocompromised individuals[37]. The necessity for more thorough research on oral fungal infections has been highlighted by the COVID-19 epidemic. The oral cavity, connected to the respiratory tract, interacts with viruses and the microbiome. Research suggests that viruses can influence fungal infections, as seen with mycoviral genetic material found in Aspergillus and Fusarium species[38]. Just as bacteriophages are used to treat bacterial infections, mycoviruses may have potential for antifungal treatments, though their safety must be carefully evaluated. Additionally, mammalian viruses such as SARS-CoV-2, herpesviruses, and Epstein-Barr virus can alter immune responses, potentially increasing fungal infection risks. Over the past forty years, research on host-microbial interactions has expanded from studying individual microorganisms to exploring complex interactions between bacteria, fungi, and immune responses. However, there is a need to explore viruses, archaea, and protozoa in this context. These interactions generate vast amounts of data, making interpretation challenging. Artificial intelligence (AI) has become a useful tool in analyzing such complex data. In dentistry, AI aids in diagnosing diseases by integrating microbiome data with immune profiling. Machine learning has been used to predict caries and classify peri-implantitis patients based on clinical outcomes[39]. Future AI-driven approaches hold promise for improving oral fungal infection diagnosis and risk prediction. Despite progress, key challenges remain in detecting, diagnosing, and treating oral fungal infections. Addressing these challenges may lead to new therapeutic strategies that leverage microbial and host interactions for better patient care[39-40].

References

1. Ananthanarayan and Paniker's Textbook of Microbiology, 8th Edition, Chapter-3: Sterilization and Disinfection, 2009, 30-38
2. Sterilization and Disinfection of Patient-Care Items and Devices. Summary of Infection Prevention Practices in Dental Settings: Basic Expectations for Safe Care Centers for Disease Control and Prevention(CDC),1998: 1–26.
3. Patel PR. Developing a broader approach to management of infection control breaches in health care settings. Am J Infect Control2008;36:685–690.
4. Rutala WA. How to assess risk of disease transmission to patients when there is a failure to follow recommended disinfection and sterilization guidelines. Infect Control Hosp Epidemiol2007;28:146–155.
5. Reddy V, Reddy G, Priyadarshini A. Sterilization Methods in Orthodontics - A Review. International journal of dental clinics 2011;3(1)
6. Condrin AK. Disinfection and sterilization in dentistry. Texas Dental Journal 2014;131(8):604–608
7. M. Saccucci, G. Ierardo, C. Protano, M. Vitali, and A. Polimeni, "How to manage the biological risk in a dental clinic: current and future perspectives," Minerva stomatologica 2017;66(5):232–9.
8. Gyorfiand A and Fazekas A. Significance of infection control in dentistry: a review. FogorvosiSzemle2007;100(4):141–52.
9. M. L. Ling, P. Ching, A. Widitaputra, A. Stewart, N. Sirijindadirat, and L. T. A. Thu. "APSIC guidelines for disinfection and sterilization of instruments in healthcare facilities," Antimicrobial Resistance and Infection Control 2018;7(1):25-39.
10. 10. G. Smith and A. Smith, "Microbial contamination of used dental handpieces," American Journal of Infection Control 2014;42(9):1019–21.
11. V. Barbot, A. Robert, M.-H. Rodier, and C. Imbert, Williams, D.; Lewis, M. Pathogenesis and treatment of oral candidosis. J. Oral Microbiol. 2011, 3. .
12. Greenberg MS, Glick M, Ship JA (2008). Burket's oral medicine (11th ed.). Hamilton, Ont.: BC Decker. pp. 79–84.
13. Vazquez J. Overview of Candida infections. https://www.uptodate.com/contents/search. Accessed Oct. 25, 2024.
14. Centers for Disease Control and Prevention. Clinical Features of aspergillosis. (https://www.cdc.gov/fungal/diseases/aspergillosis/symptoms/clinical-features.htm) Accessed 01/16/2023.
15. Young RC, Bennett JE, Vogel CL, Carbone PP, DeVita VT. Aspergillosis. The spectrum of the disease in 98 patients. Medicine (Baltimore). 1970;49(2):147–73.
16. Allen, M. J., R. Harbeck, B. Smith, D. R. Voelker, and R. J. Mason. 1999. Binding of rat and human surfactant proteins A and D to Aspergillus fumigatus conidia. In-

fect. Immun. 67:4563-4569.

17. Schubert MM, Peterson DE, Meyers JD, Hackman R, Thomas ED. Head and neck aspergillosis in patients undergoing bone marrow transplantation. Report of four cases and review of the literature. Cancer. 1986 Mar 15;57(6):1092-6 .
18. Wiersinga WJ, Rhodes A, Cheng AC, Peacock SJ, Prescott HC. Pathophysiology, transmission diagnosis, and treatment of coronavirus disease 2019 (COVID-19):A review. JAMA. 2020;324:782–93. doi: 10.1001/jama.2020.12839.
19. Huffnagle GB, Noverr MC (2008) GI microbiota and regulation of the immune system In: Advances in experimental medicine and biology, vol 635 Springer Science + Business Media; Landes Bioscience, New York; Austin, TX: .
20. Pendergast M (2010). Inside the Outbreaks: The Elite Medical Detectives of the Epidemic Intelligence Service. Houghton Mifflin Harcourt. pp. 13–14.
21. Viswanathan S, Chawla N, D'Cruz A, Kane SV. Head and neck histoplasmosis—a nightmare for clinicians and pathologists! Experience at a Tertiary Referral Cancer Centre. Head Neck Pathol. 2007;1(2):169–172. doi: 10.1007/s12105-007-0034-1.
22. Mehrabi M, Bagheri S, Leonard MK, Perciaccante VJ. Mucocutaneous manifestation of cryptococcal infection: report of a case and review of the literature. J Oral Maxillofac Surg. 2005 Oct;63(10):1543-9. .
23. Sabiiti W, May RC. Mechanisms of infection by the human fungal pathogen Cryptococcus neoformans.Future Microbiol. 2012 Nov;7(11):1297-313. .
24. Bonifaz A, Vázquez-González D, Macías B, Paredes-Farrera F, Hernández MA, Araiza J, Ponce RM. Oral geotrichosis: report of 12 cases. J Oral Sci. 2010 Sep;52(3):477-83. .
25. Arseculeratne SN, Panabokke RG, Atapattu DN, 2002. Lymphadenitis, trans-epidermal elimination and unusualhistopathology in human rhinosporidiosis. Mycopathologia 153: 57–69.
26. Lupi O, Tyring SK, McGinnis MR, 2005. Tropical dermatology: fungal tropical diseases. J Am AcadDermatol 53: 931–951.
27. Venkatachalam V, Anand N, Bhooshan O, 2007. Rhinosporidiosis: its varied presentations. Indian J Otolaryngol Head Neck Surg 59: 142–144.
28. De Silva NR, Huegel H, Atapattu DN, Arseculeratne SN, Kumarasiri R, Gunawardena S, Balasooriya P, Fernando R, 2001. Cell-mediated immune responses (CMIR) in human rhinosporidiosis. Mycopathologia 152: 59–68.
29. Akpan, A., & Morgan, R. (2002). *Oral candidiasis*. Postgraduate Medical Journal, 78(922), 455-459.
30. Kumar, S. et al. (2019). *Comparison of fungal staining techniques in oral samples*. Mycoses, 62(8), 693-699.
31. McCullough, M. J., et al. (1996). *Germ tube test for Candida identification*. Journal of Clinical Microbiology, 34(6), 1476-1479.
32. Melo, N. R., et al. (2020). *Molecular methods in fungal diagnostics*.Journal of Fungi, 6(2), 91.
33. Pincus, D. H., et al. (2007). *CHROMagar™ Candida for yeast identification*. Journal of Clinical Microbiology, 45(11), 3784-3786.
34. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for cancers in 185 countries. CA Cancer J Clin.(2021) 71:209–49.
35. Gambhir RS, Aggarwal A, Bhardwaj A, Kaur A, Sohi RK, Mehta S. Covid-19 and mucormycosis (Black Fungus): an epidemic within the pandemic. RoczPanstwZaklHig. (2021) 72:239–44.
36. Patel A, Agarwal R, Rudramurthy SM, Shevkani M, XessI,Sharma R, et al. Multicenter epidemiologic study of coronavirus disease-associated mucormycosis, India. Emerg Infect Dis. (2021)27:2349–59.
37. Jain A, Taneja S. Post-COVID fungal infections of maxillofacial region: a systematic review. Oral Maxillofac Surg.(2021)7:1–7.
38. Bhatti MF, Jamal A, Bignell EM, Petrou MA, Coutts RH. Incidence of dsRNAmycoviruses in a collection of Aspergillus fumigatus isolates. Mycopathologia.
39. Reyes LT, Knorst JK, Ortiz FR, Ardenghi TM. Scope and challenges of machinelearning-based diagnosis and prognosis in clinical dentistry: a literaturereview. J Clin Transl Res. (2021) 7:523–39.
40. Wu TT, Xiao J, Sohn MB, Fiscella KA, Gilbert C, Grier A, et al. Machine learning approach identified multi-platform factors for caries prediction in child-mother dyads. Front Cell Infect Microbiol. (2021)

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