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Oral, Neck and Orofacial Infections and Non -Bacterial Infection – A Review

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Abstract: This study aimed to determine the advancement of the medical field to cure diseases using drug and therapeutic agents. Therapeutic agents used in advancement are important in the combat of diseases. Multitargeted combination therapy has also been targeted by many clinicians due to the synergism interaction existing between many antifungal agents proven by in vitro studies. Bacterial infection which is commonly caused in the oral, neck and orofacial region. The knowledge of current concepts of orofacial infections, causative agents, their classification and clinical manifestations and a basis for management is essential for providing appropriate dental care to Patients

Keywords: Medical, infections, antifungal, bacterial, disease

INTRODUCTION

Advancement of the medical field is proportional to the emergence of drugs and therapeutic agents to cure diseases. However, the increased use of such therapeutic agents are not only efficient in the combat of diseases but also develops resistance of pathogenic microorganisms causing much invasive infections especially among immunocompromised patients, recurrence of opportunistic infections, limited availability of effective antimicrobial agents that specific in targeting certain infections. The need to develop antimicrobial drugs with mechanistic targets rises along with the prevalence of diseases(Barakate *et al.*, 2001). Multitargeted combination therapy is also targeted by many clinicians due to the synergism interaction existing between many antifungal agents proven by in vitro studies.

These combinations can broaden the spectrum and coverage of inhibition zones besides increasing the microbial effect and lowering the chances of resistance along with reduced toxicity and mortality level of humans. Under certain circumstances, some microorganisms may actually be commensal inhabitants whereby one exists with another type of microorganism under the same oral or environment's flora and do not cause diseases till the balanced commensalism flora is triggered by drugs or diseases resulting in the overgrowth of the microorganism species, now leading to the various invasive infections.(Bensinger *et al.*, 2008). Antifungals agents from various different classes targets fungal cells that are made up of chitin, glucan, mannans and glycoproteins by targeting the site of action which are intracellular, cell membrane and cell wall.(Brescó-Salinas *et al.*, 2006) This article reviews on the antimicrobial therapy for the management of bacterial and non-bacterial infections in head and neck.

Previously our university had conducted numerous clinical trials(Venugopalan *et al.*, 2014; Ganapathy *et al.*, 2016; Jyothi *et al.*, 2017) and in-vitro studies(Ajay *et al.*, 2017; Duraisamy *et al.*, 2019) and case reports(Ashok *et al.*, 2014; Ranganathan, Ganapathy and Jain, 2017) and systemic review(Selvan and Ganapathy, 2016; Subasree, Murthykumar and Others, 2016; Vijayalakshmi and Ganapathy, 2016; Ganapathy, Kannan and Venugopalan, 2017; Jain *et al.*, 2018; Kannan and Venugopalan, 2018) and surveys(Ashok and Suvitha, 2016; Basha, Ganapathy and Venugopalan, 2018) over the past 5 years. Now we are focusing on literature review. Our team has rich experience in research and we have collaborated with numerous authors over various topics in the past decade (Deogade, Gupta and Ariga, 2018; Ezhilarasan, 2018; Ezhilarasan, Sokal and Najimi, 2018; Jeevanandan and Govindaraju, 2018; J *et al.*, 2018; Menon *et al.*, 2018; Prabakar *et al.*, 2018; Rajeshkumar *et al.*, 2018, 2019; Vishnu Prasad *et al.*, 2018; Wahab *et al.*, 2018; Dua *et al.*, 2019; Duraisamy *et al.*, 2019; Ezhilarasan, Apoorva and Ashok Vardhan, 2019; Gheena and Ezhilarasan, 2019; Malli Sureshbabu *et al.*, 2019; Mehta *et al.*, 2019; Panchal, Jeevanandan and Subramanian, 2019; Rajendran *et al.*, 2019; Ramakrishnan, Dhanalakshmi and Subramanian, 2019; Sharma *et al.*, 2019; Varghese, Ramesh and Veeraiyan, 2019; Gomathi *et al.*, 2020; Samuel, Acharya and Rao, 2020)

Therefore the current article was to review the oral,neck and orofacial infections.

Microbial Etiology

The microbial etiology of orofacial contaminations may foresee from the normal occupant flora of the contiguous mucosal surfaces from which the infection originated. Due to the close anatomic relationship, the resident flora of the oral cavity, upper respiratory tract, and certain parts of the ears and eyes share many common organisms. Anaerobes generally outnumber aerobes at all sites by a factor of 10:1(Brook, 2003). Important differences in bacterial compositions have been noted from various sites within the oral cavity. Although less is known about the pathogenic potential of individual species, it is clear that as a group these endogenous organisms are structural opportunists and may invade deep tissues when normal mucosal barriers are disrupted. Invasiveness is often influenced by synergistic interactions of multiple species, both aerobic and anaerobic (Brook, 2007).

In addition, certain species or combinations may be more invasive or more resistant to treatment than others. While anaerobes are likely the predominant pathogens in most orofacial infections, other pathogens such as Staphylococcus aureus and facultative gram-negative rods including Pseudomonas aeruginosa may be present in a small but significant proportion of cases, particularly in immunocompromised patients (Brook and Gober, 2006) . In contrast to odontogenic infections, suppurative infections arising from the pharynx contain both oral anaerobes and facultative streptococci, particularly Streptococcus pyogenes, whereas rhinogenic or otogenic infections may harbor , facultative gram-negative rods as well as anaerobes (Caufield and Griffen, 2000).

Microbial Etiology Of Non-Bacterial Infections

The microbial etiology of Non-Bacterial Infections include viral infections and fungal infections. Orofacial viral infections may be less common but appear in different clinical forms. Often these infections initially get treated with antibiotics which will have no effect on the course of the primary infection. Most viral infections do not require any specific treatment except in patients who are immunosuppressed or immunodeficient(Yoshikawa and Norman, no date). Appropriate diagnosis and timely management of orofacial viral lesions are important irrespective of whether it is localized or a manifestation of a systemic infection. Viruses causing orofacial infections. There are a number of viruses that may produce a subclinical or an overt infection of the perioral, oral, and oropharyngeal region, the most common being a group belonging to the Herpesviridae and the Papillomaviridae family.(Chow, 2010)

Bacterial Infection

There are over 500 bacteria present in the oral cavity, most of which are nonpathogenic and are in symbiosis with other bacteria. Using various molecular methods and types of cultivation, the isolation of periodontal and cariogenic bacteria has been accomplished using various oral cavity samples.(Chow, no date) Routinely used methods do not enable detection of primary pathogenic bacteria which cause diseases of the lungs and upper and lower respiratory tracts. For example, a positive test for *Mycobacterium tuberculosis* was detected in oral fluid by the PCR method at 98 %, but using regular cultivation, it was only 17.3 % . (Dahlén, 2002) .Only when this infectious disease is in an acute state and the level of bacteria is very high, do Mycobacteria appear in the oral cavity in a greater number.

Helicobacter pylori is considered to be the most common cause of peptic ulcers and is also currently a risk factor for carcinoma and mucosa-associated lymphoid tissue lymphoma. The bacteria is transmitted via the fecal-oral route, but there exists very little work aimed at cultivating this bacteria from oral or fecal samples44. The golden standard of cultivation is thus still a biopsy of stomach mucous membrane and subsequent cultivation. Detection of specific *Helicobacter pylori* DNA with the help of the PCR method in saliva samples in symptomatic patients enables detection of infection in its active state. (Dale and Fredericks, 2005) .Methods of detecting Mycobacterium tuberculosis and Helicobacter pylori show that although healthy bacteria are not directly detectable in the oral fluid, saliva contains markers of these pathogens. In addition to DNA, other markers such as 16S RNA sequences have also been described in earlier studies. In diagnosing streptococcal pneumonia, the detection of pneumococcal C-polysaccharides is used in saliva without the detection of nucleic acids.(Chow, 2010)

Odontogenic Infections Of Head And Neck

Dental Caries and Pulpitis

Streptococcus pneumoniae,Haemophilus influenzae are responsible for Dental Caries .Dental caries remain prevalent in all age groups today, even though there has been a decline in carious tooth surfaces in the 24-44 years age-group within the past decade. In the adolescent population, 25% of children in the 5-17 years age-group accounted for 80% of all caries involving permanent dentition. Because of gingival recession and constant exposure of dentin, the elderly people has a higher recurrence of root caries, while enamel-related caries tend to occur at enduring throughout life (Hicks, Garcia-Godoy and Flaitz, 2003) .Changes in dental practice have significantly impacted the geriatric population in that whereas 70% of adults over 75 years of age had no natural teeth in 1957, fewer than 35% of this age group lack all teeth by 1995. (Hull and Chow, 2005)

Periodontal Diseases

Periodontal disease affects the connective tissues supporting the tooth, including the gingiva, periodontal ligament and the alveolar bone. Infection may be confined to the soft tissues around the tooth (gingivitis), or involve deeper structures (periodontitis) with loss of alveolar support for the tooth and eventually tooth loss. Gingivitis and periodontitis are prevalent in the general population. Earlier national surveys revealed that over 90% of the study population 13 years or older had evidence of gingival loss of attachment, and a third had periodontitis. Roughly 3-4% of patients will develop aggressive disease with beginning of 14 and 35 years of age. In addition, certain factors such as advancing age, smoking and diabetes mellitus are associated with an increased risk of periodontal disease. (Hicks, Garcia-Godoy and Flaitz, 2003; Hull and Chow, 2007) In addition to plaque induced inflammation, different conditions that can trigger gingival irritation include hormonal changes for example as during pregnancy, and medications such as dilantin or cyclosporin. The inflammatory host reaction at last outcomes in destruction of the periodontium surrounding the root of the tooth. It is this loss of connective tissue and bony support resulting in deepening of the gingival sulcus which defines active periodontitis. (Hull and Chow, 2007)

Suppurative Odontogenic And Deep Fascial Space Infections

Suppurative odontogenic diseases may prompt mandibular osteomyelitis, or deep fascial space infections of the oral cavity, head and neck. Soft tissue infections of odontogenic origin tend to spread along planes of least resistance from the supporting structures of the affected tooth to various potential spaces in the vicinity. Accumulated pus subsequently must puncture the bone for the most part at the site where it is thinnest and weakest before stretching out into the periapical areas or deeper fascial spaces. In the mandible, the bone is weakest and perforation tends to occur on the lingual aspect in the region of the molar teeth, and on the buccal aspect when more anterior teeth are involved. In the maxilla, the bone is weakest on the buccal aspect all through and it is thicker on the palatal aspect. If pus perforates through either the maxillary or mandibular buccal plate, it will present intraorally if inside the attachment of the buccinator muscle to the maxilla or mandible, and extraorally if outside this muscle connection .(Hurley and Heran, 2007).Therefore, contamination of the upper and lower molars, lower incisors, and lower canine teeth is often joined by extraoral sign. When a mandibular infection perforates lingually, it presents in the sublingual space if the apices of the involved teeth lie above the attachment of the mylohyoid muscle (e.g., mandibular first molar, premolar, incisor, canine and in the submandibular space is beneath the attachment of the muscle (e.g., the third molars and second molars) . A thorough understanding of the "anatomic routes" of infection not only provides valuable information about the nature and extent of infection, but also suggests the optimal surgical approach for drainage.(Hurley and Heran, 2007)

Microbiology

Culture of release is the best quality for microbiological discoveries. However, sampling may be contaminated with flora from saliva or fistula tracts. The microorganisms associated with dentoalveolar abscess are generally not the organisms associated with dental caries, but tend to be predominantly anaerobic species from deep within the gingival sulcus. Cultures of pus from periapical abscess demonstrate gram negative bacilli for example Prevotella, Porphyromonas And Fusobacterium and additionally gram-positive cocci including Peptostreptococcus and Streptococcus species. Cultures from periodontal abscesses show a similar polymicrobial microflora.(Keefe *et al.*, 2007)

Definitive treatment of suppurative odontogenic and deep space infections of the head and neck needs surgical drainage of purulent collections, and removal of the source of infection. Endodontic (root canal) intervention or extractions may be required. Systemic antibiotic therapy is not generally required for dentoalveolar or periodontal abscesses that are superficial and adequately drained surgically. For more serious infections involving deeper tissues, and in antimicrobial therapy immunocompromised hosts are important in halting the local spread of infection and preventing hematogenous dissemination. Although penicillin monotherapy has been the treatment of choice for odontogenic infections in the past, the emergence of ß-lactamase production among certain oral anaerobes, particularly pigmented Prevotella species and Fusobacterium nucleatum, is increasingly recognized and treatment failure with penicillin alone has been well documented. Thus, penicillin monotherapy is no longer recommended. Metronidazole is active against anaerobic gram-negative bacilli and recommended in combination with penicillin.[20] Stable outpatients may be treated with amoxicillin-clavulanic acid. Penicillin allergic patients should be treated with clindamycin which has excellent activity against the majority of oral pathogens. Gastro-intestinal upset and the association with Clostridium difficile-associated diarrhea are the main adverse effects. In more complicated infections, particularly in immunocompromised hosts, the choice of antibiotic therapy is primarily directed at presumed etiologic agents in the oral microflora, and broad-spectrum coverage with a second or third generation cephalosporin, a carbapenem, or ureidopenicillin

may be given. The role of newer fluoroquinolones with activity against oral streptococci and anaerobes, such as gatifloxacin and moxifloxacin, for the treatment of severe infections in immunocompromised hosts remains to be determined.(Stoopler, 2005; Loesche, 2007)

Cellulitis

Cellulitis is an acute infection of the skin which extends deep into subcutaneous tissues where underlying the dermis. It can result either by inoculation of organisms through an open wound, contiguous spread of infection from a more superficial source, or by hematogenous seeding. The involved tissue is called erythematous, the clinical feature is that it is warm in tenderness. In contrast to erysipelas, the margins of the lesion in cellulitis are non-elevated and poorly defined. The patient usually demonstrates constitutional signs of illness such as fever, chills, and malaise. In adults, Streptococcus pyogenes is the most common agent of cellulitis in the maxillofacial region which usually causes minor wounds and punctures. Once established, the infection progresses rapidly via the involved lymphatics, causing destruction of the superficial layers of the skin which may progress to streptococcal gangrene. Treatment usually requires surgical debridement of the gangrenous skin, incision and drainage at the underlying the facial planes and tissue, as well as appropriate systemic antibiotics.S. aureus can also induce a cellulitis or pyoderma, but in contrast to streptococcal cellulitis, the infection usually arises from a furuncle and tends to be more slowly progressive.(Ghodratnama, Wray and Bagg, 1999).Cellulitis caused by H. influenzae was common in children below two years of age, and the infected site may take on a bluish or purplish hue. This condition is less common nowadays. Periorbital cellulitis is more commonly infected in children and is continued by otitis media,trauma, sinusitis, or other upper respiratory tract infections. There is diffuse swelling of the eyelid that may spread across the nasal bridge. Additional manifestations may include conjunctival edema, proptosis, limitation of ocular motility, and decreased vision. Antibiotic treatment should be directed at H. influenzae, staphylococci, and streptococci, the most common pathogens in this entity. (Renne *et al.*, 1996)

Infections of The Oral Mucosa

These include aphthous stomatitis,acute herpetic gingivostomatitis, oropharyngeal candidiasis, herpangina, gangrenous stomatitis or noma, and mucositis in the severely immunocompromised host. (Gnann, 2002)

Acute Herpetic Gingivostomatitis

This condition is usually caused by a primary infection. These primary infections include Herpes simplex virus which most commonly in children between two and five years of age and less frequently in adults. (Webster-Cyriaque *et al.*, 1997).The initial symptoms consist of an enlarged submandibular lymph nodes sore throat, and a burning sensation of the oral mucosa. This vesicular eruption is caused in a rapid progress of the oral mucosa which soon becomes ulcerated. The ulcers seen in the mucosa in the early stages are small but turn into large shallow lesions with rigid borders and become covered by a yellowish, fibrinous, firmly adherent membrane. The ulcer is very painful and the patient is febrile and has considerable difficulty swallowing talking and eating. The diagnosis is confirmed by a positive Tzanck smear, and by immunofluorescence staining or virus isolation. Treatment requires topical analgesics and systemic antiviral agents (acyclovir, vidarabine, or foscarnet) .(Webster-Cyriaque *et al.*, 1997)

Aphthous Stomatitis

Recurrent oral lesions are most commonly due to aphthous ulcers, and must be differentiated from other conditions such as Behcet's disease, herpes simplex virus or coxsackie virus infections and agranulocytosis. The etiology of aphthous ulcers remains uncertain, although a number of infectious agents including viruses have been implicated. The most prevailing hypothesis suggests that the mechanisms for mucosal ulceration is autoimmune in nature.(Samonis, Mantadakis and Maraki, 2000).

Three major clinical variants are recognized:

1. minor aphthous ulcers,
2. major aphthous ulcers, and
3. herpetiform aphthous ulcers.

Minor aphthous ulcers appear as a number of small ulcers on the buccal and labial mucosa, the floor of the mouth, or the tongue. The rarely involved sites are tonsillar fauces,palatal soft tissues and pharynx. A prodromal stage is usually present. The ulcers appear gray-yellow, often with a raised and erythematous margin, and are exquisitely painful. Lymph node enlargement is seen only with secondary bacterial infection. The course of ulceration varies from a few days to several weeks, and is followed by spontaneous healing. Major aphthous ulcers last up to several months. The soft palate and tonsillar areas may also be involved. .Prolonged periods of remission may be followed by intervals of intense ulcer activity. Herpetiform aphthous ulcers appear small and multiple in numbers and characteristically affect the lateral margins and tips of the tongue. The ulcers are gray with a delineating erythematous border, and are extremely painful which makes eating and speaking difficult.

The treatment is aimed to primarily relieve the symptoms with antiseptic mouthwashes and local anaesthetic lozenges or gels. Topical or systemic steroids may be beneficial in selected individuals with extensive disease.

Oropharyngeal Candidiasis

The colonies of candida species are most commonly seen in patients in old age and the presence of the colonies of candida species is found to be independent of denture use. Diseases seen clinically can be caused by the use of broad spectrum antibiotics or ill fitting and unclean dentures. Oral candidiasis is common in the later stages of HIV disease. The oral manifestation that is most commonly seen is the pseudomembranous candidiasis (thrush). It is characterized by soft white, slightly raised adherent plaques which can be wiped off leaving an erythematous or bleeding surface. Acute erythemic or atrophic candidiasis is characterized by painful erythematous mucosal lesions and a "bald" (depapillated) appearance of the tongue, with a matching lesion on the opposing surface of the palate. Chronic atrophic candidiasis or denture-induced stomatitis is commonly found in denture wearers and elderly persons with diffuse inflammation of denture-bearing areas due to prolonged irritation. Chronic hyperplastic candidiasis is a leukoplakic or keratotic lesion that cannot be removed by scraping, and is usually located on the anterior buccal mucosa. Treatment options include topical antifungal agents, such as nystatin oral suspensions or clotrimazole troches, and systemic agents with azoles (fluconazole, itraconazole, voriconazole) or caspofungin . (Brook, 2003; Kolokotronis and Doumas, 2006; Chayavichitsilp *et al.*, 2009)

Non-Bacterial Infections of Viral Etiology

Herpes Group of Viruses

Herpes group of virusesThe herpes group of the Herpesviridae family includes eight viruses in three subclasses as Alpha, Beta and Gamma herpes viruses. Alpha herpes viruses include the herpes simplex virus-1 (HSV-1), herpes simplex virus-2 (HSV-2) and varicella zoster virus (VZV, HHV-3). Beta herpes viruses are grouped as cytomegalovirus (CMV, HHV-5), human herpesvirus-6 (HHV-6) and human herpesvirus-7 (HHV-7). Gamma herpes viruses consist of Epstein-Barr virus (EBV, HHV-4) and human herpesvirus-8 (HHV-8).Interestingly, most of the viruses in the Herpesviridae family are known to cause oral and peri-oral infections, although there is controversy as to the true causative agent of some of these orofacial infections.1 Orofacial viral infections are common among immunocompromised patients; the most common being those caused by the herpes simplex virus (HSV). Human herpesvirus-6 has been proposed as an aetiologic factor in recurrent aphthous stomatitis.2 Human herpesvirus-8 (HHV-8) is the aetiopathogenesis of Kaposi's sarcoma.3 Varicella-zoster virus is less common but occurs in severe forms, as in herpes zoster.4 Epstein-Barr virus in oral hairy leukoplakia (OHL)5 and many of the herpes viruses and certain human papilloma viruses (HPV) are known for their association in malignant neoplasms. (Arduino and Porter, 2008)

Human papillomavirus (HPV)

The Papillomaviridae family are a gathering of double-stranded circular DNA infections viruses commonly found in the oral and oropharyngeal mucosa, ano-genital region and tracheo-bronchial mucosa. They are grouped into more than a hundred types and HPV type 16 and 18 have been implicated in oral, oropharyngeal and tonsillar carcinomas.27 More recently, there has been an increasing understanding of the risk factors of HPV in oral cancers, especially the risk of oro-genital sexual activity.28-30Orofacial manifestations of HPV are: Verruca vulgaris or the common wart on the perioral skin; Oral papilloma (squamous cell papilloma) of the oral mucosa (Figure 4); Focal epithelial hyperplasia; and Condyloma accuminatum, a sexually transmitted disease.31Management of HPV infection depends on the clinical presentation, such as papilloma, usually using complete surgical excision and/or topical drug therapy. Laser and cryotherapy are not recommended owing to lack of a tissue for histopathological evaluation and a possible seeding of the lesion to the surrounding area in the process.22,23 Two HPV vaccines are currently available and have a clear role in preventing many ano-genital cancers and conditions related to HPV infection. The effectiveness of HPV vaccines in preventing oral HPV infection and cancer is unknown. Studies are underway to evaluate the long-term efficacy of the vaccine against both ano-genital and non-ano-genital endpoints.[25,26,29,30](Samonis, Mantadakis and Maraki, 2000; Malkin, 2002; Arduino and Porter, 2008; Wittek, Doerr and Allwinn, 2010)

Human immunodeficiency virus

Human immunodeficiency virus infection is pandemic and the disease has become more of a chronic viral infection due to the advent of multiple drug therapy using ART or HAART. The disease may progress to a serious and debilitating stage of AIDS with increased viral load and a significant decrease in the CD4 cells, leading to several opportunist .(Wittek, Doerr and Allwinn, 2010) .There are several orofacial manifestations associated with HIV disease and these are among the earliest manifestations and considered important indicators of HIV infection, with some carrying a prognostic value. Oral manifestations of HIV/AIDS have been classified into three groups, based on the clinical features and intensity. Lesions that are 'strongly associated' with HIV

infection such as hairy leukoplakia, Kaposi's sarcoma and Non-Hodgkin's lymphoma(Mustafa, Arduino and Porter, 2009)

Coxsackie virus

Coxsackie virus causes hand, foot and mouth disease (strain A16) and herpangina. These viruses can pass through the oral mucosa and small intestine and the regional lymph nodes. Clinical features of hand, foot and mouth disease include a mild prodrome followed by sparse distribution of vesicles with an erythematous halo affecting the oral mucosa, hands and feet. Painful ulcerative lesions occur anywhere in the oral cavity, but are commonly found on the hard palate, tongue and buccal mucosa. The exanthem (mucosal lesions) begins as 2−8 mm erythematous papules, a short vesicular stage and yellow-grey ulcers with an erythematous halo. Lesions may coalesce, the tongue may become red and oedematous and painful, interfering with oral intake. Oral lesions heal without treatment within 5−7 days.34 No specific treatment is necessary except for isolation of the patient, especially children, to avoid spread of the disease in a community.34Herpangina is also a disease of the early life, with an incubation period of 4 days followed by an abrupt onset of fever with malaise, headache, neck or back pain. The oral mucosal lesions consist of 1−2 mm grey-white papulo-vesicular lesions that progress to ulcers surrounded by an erythematous halo or rim and the oropharynx may appear diffusely hyperaemic. Lesions are distributed on the anterior tonsillar pillars, soft palate, uvula and tonsils and usually last for a week. Common complaints of affected patients are anorexia, dysphagia and sore throat. No associated cutaneous lesion is typically seen. Only symptomatic treatment is necessary, such as antipyretics, analgesics and anti-inflammatories, if necessary (Bravender, 2010)Our institution is passionate about high quality evidence based research and has excelled in various fields ((Pc, Marimuthu and Devadoss, 2018; Ramesh *et al.*, 2018; Vijayashree Priyadharsini, Smiline Girija and Paramasivam, 2018; Ezhilarasan, Apoorva and Ashok Vardhan, 2019; Ramadurai *et al.*, 2019; Sridharan *et al.*, 2019; Vijayashree Priyadharsini, 2019; Chandrasekar *et al.*, 2020; Mathew *et al.*, 2020; R *et al.*, 2020; Samuel, 2021)

CONCLUSION

The knowledge of current concepts of orofacial infections, causative agents, their classification and clinical manifestations and a basis for management is essential for providing appropriate dental care to Patients.Hence we conclude that Clinicians and readers should understand that management of orofacial infections of both bacterial and non Bacterial origin is critical in primary care, either in dental or medical practise.

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CONFLICT OF INTEREST

The authors declare that there was no conflict of interest.

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