Review Article

Orofacial Bacterial Infectious Diseases: An Update

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Abstract

Objectives: Most of the oral infections with odontogenic origin are very common and can be treated by tooth extraction, endodontic therapy, or surgical treatment. Other infectious lesions are the manifestations of systemic diseases such as tuberculosis and syphilis. Skin and underlying subcutaneous tissue, fascia, or muscle is also involved with infectious diseases which range from superficial epidermal infections to very serious necrotizing fasciitis.

Materials and Methods: An extensive literature in PubMed, Google Scholar, and Scopus search was performed from 1980 to 2017. All related articles were analyzed.

Results: Most oral infections have odontogenic origin. Skin and the underlying subcutaneous tissue, fascia, or muscles are also involved with infectious diseases which range from superficial epidermal infections to very serious necrotizing fasciitis.

Conclusions: These facts prove that the interaction between the oral cavity, face skin, and the other organs can risk the people's life. The establishment of a correct diagnosis and recognition of clinical findings are the crucial steps to support and improve professional orofacial health status.

Keywords: *Bacterial, infectious diseases, orofacial, skin*

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Introduction

Orofacial infections have threatened humans since the rise of human existence. Most oral infections which have the odontogenic origin are very common and can be treated by tooth extraction, endodontic therapy, or surgical treatment.[1] However, prescribing an antibiotic may be necessary.[2] Other infectious lesions are the manifestations of systemic diseases such as tuberculosis and syphilis.[3] A variety of bacteria which have synergistic or antagonistic interactions shape the oral biofilm.[4-8] Teeth provide hard, nonshedding surfaces for the deposition of oral microorganisms which remain within dental plaque. The accumulation and metabolism of the bacteria on hard oral surfaces is the primary cause of dental caries, gingivitis, periodontitis, peri-implant infections, and stomatitis.[9] Oral epithelium is a barrier which separates the oral cavity from its environment. The main functions of oral mucosa are resistance to microorganisms, trauma, and exogenous substances.[10]

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Skin and the underlying subcutaneous tissue, fascia, or muscle is also involved with the infectious diseases which range from superficial epidermal infections to very serious necrotizing fasciitis. The incidence is about 24.6/1000 person-years in developed societies. As the diseases are variable in etiology, clinical presentation, and the diagnosis and therapy, it is critical to identify and differentiate them.[11] The aim of this study was to review the bacterial profiles associated with the orofacial infections.

Materials and Methods

In this review article, a relevant English Literature search in PubMed, ScienceDirect, and Google Scholar from 2000 to mid-2017 was performed. All relevant articles were selected and reviewed. The diseases were classified into oral infections and facial infections.

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Results

Oral infectious diseases

Odontogenic infectious diseases

Dental caries and pulpitis

Dental caries is considered to be the most prevalent human disease, affecting 80%–90% of the world population[12] and mostly the children aged 5–17 years. A published article found a higher prevalence of dental caries in males compared to females (73% vs. 27%).[13] Both primary dentition and permanent dentition can be affected[14] with more prevalence in molar teeth.[13,15] Based on the degree of pulp inflammation, patient's clinical symptoms differ. The sensibility tests such as thermal test can assess the response of nerve fibers in dental pulp, but due to resistance of the nerve fibers to inflammation, they may still remain active even after the degeneration of pulp tissue.[16] Over the time, the patient suffers from pain caused by irreversible pulpitis.[17] Pulpitis, the inflammation of the dental pulp, is a common sequel to caries. Bacteria are involved in inducing dental tissue damage and inflammation in the pulp tissue. The pulp is affected by bacterial virulence factors and antigens diffused by the dentinal fluid or bacterial cells. Clinically, pulpitis is classified as reversible or irreversible. In the reversible form, removing the causative agent leads to the return to normal condition, whereas in the irreversible pulpitis, root canal treatment is required. In the cases of deep dentinal caries, the bacterial composition differs from enamel caries. *Streptococcus mutans* was isolated in the 1920s from carious lesions and considered as the etiological agent of dental caries. The other species playing a key role in the microbial community and dentin caries include *Veillonella*, *Rothia*, and *Leptotrichia* in enamel caries and *Streptococcus sanguinis*, *Atopobium*, Schlegelella, *Pseudoramibacter*, and *Lactobacillus*.[18] Besides lactobacillus which is a very prevalent bacterium in dental caries, asaccharolytic and/or proteolytic anaerobic bacteria have been frequently detected in pulpitis. Most microorganisms in dental caries initiate endodontic infections. *Atopobium*, *Pseudoramibacter alactolyticus, Streptococcus* species, and *S. mutans* are the most frequent bacteria found in teeth with irreversible pulpitis.[19]

Gingivitis and periodontitis

Gingivitis is the reversible inflammation of only the gingiva. The prevalence of gingivitis has been reported to be as high as 90%–100% among children 7–14 years of age.[20] Clinically, inflammation begins in childhood and increases with age.[21] Redness, hypertrophy, and bleeding are the most common symptoms of gingivitis.[22] Gingivitis is initiated by the enzymatic effects and toxins released by the pathogenic microorganisms of the supragingival dental calculus and plaque. Spirochaetes, numerous Gram-positive cocci and Gram-negative bacilli as well as a variety of aerobic and capnophilic microorganisms present in the supragingival calculus.[23] The host-bacteria interaction results in the destruction of gingiva and periodontal tissues.

Necrotizing ulcerative gingivitis is a different pattern of gingivitis. It may occur in any age, but it is more frequent in young and middle-aged adults.[21] In this case, the interdental papillae are edematous, inflamed, and hemorrhagic. The blunted "punched-out" appearance of the affected papillae and craterlike necrosis of papillae covered with a gray pseudomembrane are the clinical features. Both necrotizing ulcerative periodontitis and necrotizing ulcerative mucositis (necrotizing stomatitis) are developed by the extension of the infection to the adjacent tissues. A fusiform bacterium, *Bacillus fusiformis* (currently *Fusobacterium nucleatum*), and a spirochete, *Borrelia vincentii,* are the causative microorganisms.[21]

Periodontitis is a chronic inflammation of supporting structures of teeth related to the oral biofilm which causes the destruction of connective tissue attachment to the tooth, alveolar bone resorption, and tooth loss.[24] The patient's age ranges between 18 and 81.[25] The most frequent areas are the first and second molars.[26] *Porphyromonas gingivalis* is the main etiology of periodontitis. It has the ability to colonize on the oral soft-tissue surfaces and interacts with other oral bacteria inducing immune response and finally invades host cells. *P. gingivalis* invasion protects the bacteria from immune system and antibiotic treatment.[27] The other more prevalent pathogens in the periodontal diseases are *Prevotella intermedia, Bacteroides forsythus, Aggregatibacter actinomycetemcomitans, F. nucleatum,* and *Capnocytophaga*.[28]

Pericoronitis

Pericoronitis is an inflammatory process arising within the tissues surrounding the crown of a partially erupted tooth, mostly third molar.[21,29] The main causes of inflammation are food debris and bacteria beneath the gingival flap overlying the crown. Abscess develops most frequently in association with the mandibular third molars.[21] The third molar impaction usually occurs between the ages of 17 and 21 years. Previous studies suggest that impaction of mandibular third molar has a higher incidence in females compared to males.[30] Periodontitis, pain (in 5%–53% of cases), cellulitis, and osteomyelitis (in 5% of cases) are the most common clinical features.[30] *Hemolytic streptococci*, obligate anaerobes, the genus *Prevotella*, *Veillonella* are the most common microorganisms which have been detected in

pericoronitis patients.[29] In addition, in a published study, *Parvimonas micra* was found in 66.7% of pericoronitis samples.[31]

Endodontic infections

Endodontic disease is initiated by the infection with multiple microorganisms, and later, the infiltration of inflammatory cells leads to pulpitis and periapical periodontitis. The results of a previous investigation have shown that the endodontic treatment is the most prevalent in female and the patient's age ranges between 46 and 60 years old. Besides, this study indicated that maxillary premolars and molars are the most prevalent teeth for the endodontic treatments.[32] The most common clinical features are pain, sensitivity of the tooth to pressure, and the swelling of surrounding tissues.[33]

Black-pigmented organisms such as *Prevotella* followed by *Porphyromonas*, especially, *P. gingivalis* are the most isolated microorganisms through culture of samples obtained from necrotic pulps.[34] While *Fusobacterium, Parvimonas, and Peptostreptococcus* were indicated as the most prevalent microorganisms in acute endodontic infections, *phyla Firmicutes, Bacteroidetes*, and *Actinobacteria* were the most prevalent microorganisms in chronic endodontic infections.[35]

Nonodontogenic infectious diseases

Peri-implantitis

An inflammation and destruction of hard and soft tissues surrounding dental implants is called peri-implantitis. In cases where infection is limited to the peri-implant mucosa, it is named peri-implant mucositis.[36] It is suggested that a disturbance of the balance between the microbiological challenge and the host response may result in peri-implantitis. Loss of supporting bone is the main characteristic of peri-implantitis. The lesion is a multifactorial process as all microorganisms; immunological, environmental, iatrogenic, mechanical, anatomical, genetic factors are involved in developing of the lesion.[37] The history of periodontitis is the most common risk factor.[38] As the attachment between the titanium surface of an implant and supra-alveolar connective tissue is weak, it is easily destroyed leading to bacterial contamination spreading to the bone.[39,40] The lesion is more frequent in males older than 65 years.[41,42] The submucosal presence of *P. gingivalis, P. intermedia, T. forsythia,* and *F. nucleatum* has been demonstrated around implants with peri-implantitis.[37,43]

Syphilis

Syphilis is an acute and chronic sexually transmitted disease which is caused by an anaerobic tightly coiled helical bacterial species, *Treponema pallidum*. The disease is most common in males older than 15 years.[44] Oral lesions include chancre mostly in the lower lip (primary syphilis) and mucous patches mainly on the tongue and associated with secondary syphilis.[21] The other lesion is a white plaque with verrucous aspect, so-called "leukoplakia-like" lesion.[45] One-third of patients with secondary syphilis and the characteristic skin rash present oral manifestations.[46-48]

Tuberculosis

Tuberculosis is a chronic granulomatous affecting various systems of the body. It is caused mainly by *Mycobacterium tuberculosis*, *Mycobacterium bovis*, and other atypical mycobacterial species.[49] The disease can be found in all age groups and both genders.[50] The lungs are the most common primary sites of disease. The oral cavity lesions are rare and appear secondary to pulmonary tuberculosis.[51] Oral lesions seem to occur as chronic ulcers, nodular or granular areas, and rare, firm leukoplakia regions.[52] Oral lesions are painless in most cases.[51]

Leprosy

Leprosy is a chronic infectious contagious disease produced by *Mycobacterium leprae*. It is more common in males older than 15 years old.[53] Clinical presentations of leprosy are related to the immune response against *M. leprae*. Orofacial lesions (oral and nasal lesions) are the most frequent source of the spread of pathogen. Well-circumscribed hypopigmented lesions in tuberculoid leprosy and ill-defined hypopigmented macules or papules on the skin particularly on the face of patients with lepromatous leprosy are the common skin lesions.[21] Severe destruction of the anterior face including the maxilla occurs in the patients.[54] Initially, the nasal mucosa is affected, usually preceding skin lesions. The oral cavity may be contaminated in advanced stages of disease when the microorganism invades the oral cavity due to bacterial dissemination in the circulating blood.[55] Oral lesions are rare occurring in a lepromatous form. These lesions initially appear yellowish to red, sessile, firm, and papule or as nodules that develop ulceration and necrosis. According to the WHO, the most affected sites of oral cavity in leprosy patients are: the hard palate, soft palate, labial maxillary gingiva, and buccal mucosa.[56]

Scarlet fever

Scarlet fever is a systemic infection caused by group A, β-*H. streptococci*. The disease is the most prevalent in children between 3 and 12 years old.[21] First, the disease begins as a streptococcal tonsillitis with pharyngitis. During the first 2 days, a white coat covers the dorsum of tongue (white strawberry tongue). After desquamating of white coat, erythematous dorsal surface of tongue appears (red strawberry tongue). Within the first

2 days, the disease is associated with elaboration of an erythrogenic toxin which attacks the blood vessels and develops the cutaneous rash.[57]

Gonorrhea

Gonorrhea is caused by the bacterium *Neisseria gonorrhoeae.* Gnorrhea is a young person's disease and the male:female ratio is 1.5:1.[58] Oral lesions are common on the soft palate and oropharynx which clinically present as aphthous ulcers.[59]

Facial bacterial infectious diseases

Cellulitis

Cellulitis is a bacterial skin infection which can spread to other parts of body in severe cases.[60] The most common causative organisms in adults are streptococci (particularly *Streptococcus pyogenes*) and *Staphylococcus aureus* in children.[61] Orbital cellulitis is caused by primary infection of the skin, sinuses, or teeth. The most common cause is the primary sinus bacterial infection (64%). Some cases (16%) arisen from cutaneous lesions, such as eczema, furuncles, or facial cellulitis. Odontogenic infection is the least frequent cause of orbital cellulitis, however, is very important due to poor prognosis.[62] Redness of the skin, warmth, and fever are the clinical symptoms. The other clinical manifestations include abscess formation, blindness, cavernous sinus thrombosis, pulmonary embolism, and death.[60]

Impetigo

Impetigo is a superficial infection of the skin caused by *Staphylococcus* aureus/or in combination with *Streptococcus pyogenes* (group A, β-hemolytic). The scalp and face are the most commonly involved sites in the maxillofacial area. The facial lesions usually appear around the nose and mouth. It is believed that the pathogenic bacteria which are harbored in the nose, spread onto the previously damaged skin.[21] The disease is most common in children aged 2–5 years. The lesions are divided into bullous and nonbullous types. The bullous type is almost caused by *S. aureus*. The lesion starts as a small blister which gradually becomes a flaccid blister, measuring up to 2 cm in diameter. However, the nonbullous type mainly occurs in adult. In majority of cases *S. aureus* alone or in combination of group A, β-hemolytic Streptococci is the main causative pathogen. Initially, the disease starts with a vesicle, located on erythematous base. The rupture of the vesicle results in ulcer formation. Finally, the ulcer turns to a yellowish crust.[63]

Erysipelas

Erysipelas involves the upper dermis and superficial lymphatics.[64] It is mostly found in immunosuppressed males older than 50 years.[65] The affected areas appear as painful, red well-circumscribed, and swollen regions.[21] The most common causative pathogens are β-*H. streptococci* (usually group A, such as *S. pyogenes*). Other microorganisms, such as *Staphylococcus aureus*, may be involved in the development of the lesions. On the face, the lesions appear on the cheeks, eyelids, and bridge of the nose and look like the lesions in lupus erythematosus. High fever and lymphadenopathy are other clinical features.[21]

Tables 1 and 2 summarize the clinical features and the most prevalent pathogens of orofacial bacterial infections.

Table 1: Clinical characteristics of orofacial bacterial infectious diseases

Lesion	Age (years)	Sex	Most prevalent site	Clinical symptoms	Reference
Dental caries and pulpitis	5-17	Male	Molar teeth	Different response to sensibility tests, pain in advanced stages	[13-17]
Gingivitis	7-14	Female	No data	Redness, hypertrophy, and bleeding	[20,22]
Necrotizing ulcerative gingivitis	>20	Both	No data	Blunted "punched-out" papillae, and craterlike necrosis of papillae covered with a gray pseudomembrane	[21]
Periodontitis	18-81	Both	Molar teeth	Destruction of alveolar bone and tooth loss	[24,25]
Pericoronitis	17-21	Female	Mandibular third molar	Periodontitis, pain, cellulitis, osteomyelitis	[30]
Acute and chronic endodontic infections	46-60	Female	Maxillary premolars and molars	Pain, sensitivity of the tooth to pressure, and swelling of surrounding tissues	[32,33]
Peri-implantitis	>65	Male	Maxilla	Bone resorption, decreased osseointegration, pocket formation, and purulence	[36,42]
Syphilis	>15	Male	Lower lip, tongue	Chancre, mucous patch	[21]
Tuberculosis	Any age	Both	Tongue	Ulcer, nodular, and granular areas	[21]
Leprosy	>15	Male	Hard and soft palates	Papules	[21]
Scarlet fever	3-12	Both	Skin and oral cavity	Rash and erythema	[21,57]
Gonorrhea	16-19	Male	Soft palate and oropharynx	Aphthous ulcers	[21,58]
Cellulitis	<10	Both	Face (orbital region)	Redness, fever	[60]
Impetigo	<2-3	Both	Scalp and face	Small blisters	[21]
Erysipelas	>50	Male	Face	Pain, redness, and swollen	[65]

Table 2: The most prevalent bacterial pathogens in orofacial infections

Lesion	The causative pathogen (s) (reference)
Dental caries	*S. mutans*[18]
Pulpitis	*Atopobium, P. alactolyticus, Streptococcus* species, and *S. mutans*[19]
Gingivitis	*Spirochaetes*, Gram-positive cocci, and Gram-negative bacilli[23]
Necrotizing ulcerative gingivitis	*F. nucleatum*, *B. vincentii*[21]
Periodontitis	*P. gingivalis*, *P. intermedia*, *B. forsythus*, *A. actinomycetemcomitans*, *F. nucleatum*, and *Capnocytophaga*[27,28]
Pericoronitis	*Hemolytic streptococci*, *Prevotella*, *Veillonella*[29]
Acute endodontic infections	*Fusobacterium*, *Parvimonas*, *Peptostreptococcus*[35]
Chronic endodontic infections	*Phyla firmicutes*, *Bacteroidetes*, *Actinobacteria*[35]
Peri-implantitis	*P. gingivalis*, *P. intermedia*, *T. forsythia*, and *F. nucleatum*[37,43]
Syphilis	*T. pallidum*[44]
Tuberculosis	*M. tuberculosis*, *M. bovis*[49]
Leprosy	*M. leprae*[53]
Scarlet fever	Group A, β-*hemolytic streptococci*[21]
Gonorrhea	*N. gonorrhoeae*[58]
Cellulitis	Streptococci; *S. pyogenes* and *S. aureus*[61]
Impetigo	*S. aureus*/or in combination with *S. pyogenes* (Group A, β-hemolytic)[21]
Erysipelas	β-*hemolytic streptococci* mostly group A, such as *S. pyogenes*[21]

S. mutans=Streptococcus mutans, *P. alactolyticus=Pseudoramibacter alactolyticus*, *F. nucleatum=Fusobacterium nucleatum*, *B. vincentii=Borrelia vincentii*, *P. gingivalis=Porphyromonas gingivalis*, *P. intermedia=Prevotella intermedia*, *B. forsythus=Bacteroides forsythus*, *A. actinomycetemcomitans=Aggregatibacter actinomycetemcomitans*, *T. forsythia=Tannerella forsythia*, *T. pallidum=Treponema pallidum*, *M. bovis=Mycobacterium bovis*, *M. tuberculosis=Mycobacterium tuberculosis*, *M. leprae=Mycobacterium leprae*, *N. gonorrhoeae=Neisseria gonorrhoeae, S. pyogenes=Streptococcus pyogenes*, *S. aureus=Staphylococcus aureus*

Discussion and Conclusions

This study provided details regarding orofacial bacterial infectious diseases. Several local or systemic diseases lead to orofacial lesions. These facts prove that the interaction between the oral cavity, face skin, and the other organs can risk the people's life. The establishment of a correct diagnosis and recognition of clinical findings are the crucial steps to support and improve professional orofacial health status. More work has to be performed to identify causative pathogens.

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Conflicts of interest

There are no conflicts of interest.

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