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MAXILLOFACIAL INFECTION: FOCUS ON PERIODONTAL DISEASE

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ABSTRACT

Craniofacial structures, such as the mouth, teeth, and gums, can be subject to both acute and chronic infections. Management of maxillofacial infections, which activate immunity and inflammation, is very important since they can lead to serious complications. The most common pathogens responsible for these infection are viruses, bacteria, and fungi. Maxillofacial infections can be odontogenic, including dental abscesses, jawbone abscesses, and periodontitis, amongst others, and activate the immune system, generating inflammatory molecules. Lipopolysaccharide (LPS) present in Gram-negative bacteria is a large molecule capable of activating immunity and inflammation in the oropharygeal system. Bacterial infections, especially those mediated by Gram-negative bacteria, can cause periodontitis, where the immune response is triggered, resulting in chronic inflammation with gingival damage. Immune molecules, including metalloproteinases (MMPs) and cytokines, such as interleuking (IL)-1, tumor necrosis factor (TNF), and IL-6, are released and mediate inflammation and tissue damage. Upon stimulation, osteoclasts, the precursors of macrophages, lead to the activation of the receptor for nuclear factor kappa B (NF-kB), receptor activator of nuclear factor κ B-ligand (RANKL), and other proteins that contribute to osteoclastogenesis and therefore, bone loss. It can be concluded that bacterial infections can mediate periodontitis in chronic and severe cases.

KEYWORDS: *Infection, maxillofacial, periodontitis, immunity, inflammation, bacteria*

INTRODUCTION

Chronic infections can contribute to local and systemic inflammation and increase the risk of various disorders, including craniofacial diseases affecting the mouth, teeth, and gums (1). Maxillofacial infections involve the facial bones and soft tissues and can lead to significant complications if not properly managed (2). Understanding the mechanisms of infection, inflammation, and immunity in the maxillofacial region is crucial for effective treatment.

Infections begin with pathogens entering the oral mucosa, dental caries, or through spaces created by trauma (3). Infections can affect the face, lymphatic system, or bloodstream (4). The most common pathogens which are responsible for these infections include bacteria (such as *Staphylococcus aureus* and some species of *Streptococcus*), viruses (such as herpes simplex), and fungi (such as Candida) (5).

DISCUSSION

Typical maxillofacial infections are odontogenic infections originating from dental structures (for example, dental abscesses) (6), but also from soft tissue infections such as cellulitis and abscesses, as well as infections of the jaw bones

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(osteomyelitis) (7). Neutrophils and macrophages are innate immune cells that constitute a physical barrier of the tissues and are the first immune cells that intervene in infections for the recognition of pathogens (8). Adaptive immunity involves the intervention of B cells that produce antibodies to try to neutralize the pathogen and facilitate its elimination (9). Helper T cells and cytotoxic T cells are part of cell-mediated immunity and target and kill infected cells, orchestrating the immune response (10). Pathogens defend themselves from the immune process by forming biofilms to resist phagocytosis and antibiotic treatment and can alter host immune responses to evade detection and destruction (11).

Tissue inflammation can be activated by bacterial products such as lipopolysaccharide (LPS) (12). LPS is a large molecule found in the outer membrane of Gram-negative bacteria that plays a key role in the development of inflammation. LPS triggers the immune response in the host by acting as an endotoxin. It causes inflammation and tissue damage, and stimulates the production of cytokines, prostaglandins, and other mediators involved in bone resorption. In addition, LPS can contribute to the progression of periodontitis (13).

Periodontal disease

Periodontitis is a gum infection that damages the soft tissue and can be serious (14). Bacteria are primarily responsible for causing this disease (15). Periodontitis is a chronic infection that occurs in 50% of the adult population in industrialized countries. Bacteria, particularly Gram-negative bacteria containing LPS, proliferate and release LPS, which triggers an immune response, causing chronic inflammation damages gum tissue and bone, which ultimately results in tooth loss if the infection is not treated (16).

In the mouth, certain bacteria can form plaque on the teeth that, if not removed, forms tartar which continues to host the bacteria (17). These bacteria stimulate molecules that attract immune cells and cause inflammation of the gums, i.e. gingivitis, the stage which occurs before periodontitis (18). Gingivitis is commonly caused by poor oral hygiene and leads to the accumulation of bacterial plaque on the gums that, without proper treatment, can destroy the bone that supports the teeth. In this disease, inflammatory molecules such as metalloproteinases (MMPs) are activated, along with others, and enter the inflammatory cascade (19). MMPs participate in the recruitment of migratory immune cells to the inflammatory site (20).

Several lines of experimental and clinical evidence indicate that inflammation in periodontal disease leads to bone loss (21). Periodontal disease is mediated by bacterial antigens that cause the activation of the immune system and inflammatory reactions. The first reaction is the activation of innate immunity with the generation of inflammatory cytokines and arachidonic acid products due to the activation of monocytes/macrophages, dendritic cells, and mast cells (22). The most important inflammatory cytokines that are secreted by the immune reaction are interleukin (IL)-1, tumor necrosis factor (TNF), and IL-6 (23). T and B lymphocytes stimulated by the antigen also activate the adaptive immune response and participate in the inflammatory reaction (24). In these reactions, which stimulate the differentiation of macrophage precursor cells into osteoclasts, there is activation of the receptor for nuclear factor kappa B (RANK) -ligand (RANKL) and other proteins that lead to osteoclastogenesis and thus bone loss (25). Therefore, stimulation of osteoclast maturation leads to bone loss. On osteoclast progenitors, inflammatory cytokines and other proteins participate in bone resorption and act through the RANK to RANKL binding reaction (26). Therefore, RANKL is critical for regulating bone metabolism.

To prevent periodontitis, proper dental hygiene is crucial to avoid the buildup of plaque, and when necessary, antibiotic therapy is important to eliminate bacteria (27). In addition, severe cases of periodontitis may require surgery to clean deeply infected pockets and repair damaged tissue (28).

CONCLUSIONS

Infectious diseases caused by bacteria are treated with antibiotics specifically selected based on the involved pathogens. Surgical interventions are also very common for drainage of abscesses, removal of necrotic tissue, and management of the source of infection. Anti-inflammatory drugs are used against pain and swelling.

In maxillofacial infections, there are complex interactions between pathogens, host immune responses, and inflammatory processes. These reactions are still not fully elucidated and continued research is vital to shed light on mechanisms which could help generate new effective treatment strategies to manage infections and prevent disease. Understanding the role of bacteria in periodontitis is important to prevent the onset and progression of this oral disease.

Conflict of interest

The authors declare that they have no conflict of interest.

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