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Review

Classification and Most Common Causative Organisms in Gingivitis

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Abstract

Gingivitis, a reversible periodontal disease inflammation of the gingiva, is primarily caused by dental plaque and can progress to more severe periodontal diseases if left untreated. This review classifies gingivitis and identifies the most common causative organisms. Plaque-induced gingivitis is typically caused by bacterial biofilms containing species such as Streptococcus, Actinomyces, and gram-negative anaerobes like Porphyromonas gingivalis. Non-plaque-induced gingivitis can arise from genetic or developmental abnormalities, bacterial, viral, and fungal infections, Inflammatory or immune conditions, and reactive processes. Necrotizing periodontal diseases and specific bacterial infections, such as those caused by Treponema pallidum, and viral infections, including herpes simplex virus and human papillomavirus, play significant roles. Fungal infections like candidosis also contribute. Autoimmune disorders such as pemphigus vulgaris along with reactive processes like epulides, are notable non-plaque-induced gingivitis causes. Understanding these classifications and causative organisms is critical for developing effective preventive and therapeutic strategies.

Keywords: *Gingivitis, plaque, bacteria, infections, autoimmune, viral, fungal, prevention*

Introduction

The gingiva is the part of the oral mucous membrane covering the alveolar processes and the cervical portions of the teeth. It is traditionally divided into the free and attached gingiva (1). The free gingiva is the tissue coronal to the bottom of the gingival sulcus, while the attached gingiva extends apically from the free gingival groove to the mucogingival junction. Gingival tissues are typically light pink, though the colour can vary based on the individual's complexion, tissue thickness, and degree of keratinization. In young children, gingiva may appear more reddish due to increased vascularity and thinner epithelium, and their gingiva tends to be less stippled, or smoother compared to adults. The marginal gingiva in healthy people has a knife-like edge (2). During the period of tooth eruption in children, however, the gingivae are thicker with rounded margins due to the migration and cervical constriction of the primary teeth. Probing depths around primary teeth are approximately 2 mm, with shallower measurements at facial and lingual probe sites compared to proximal sites (3). Children have a wider periodontal ligament than adults. The width of the attached gingiva is narrower in the mandible than in the maxilla, with both widths increasing as children transitions from primary to permanent dentition. The alveolar bone surrounding primary teeth exhibits fewer trabeculae, less calcification, and larger marrow spaces.

Inflammation of the gingivae caused by a developed tooth plaque biofilm is a sign of gingivitis, a reversible periodontal disease. Gingivitis can develop into chronic periodontitis in susceptible people (4), resulting in irreversible periodontal tissue destruction. Currently, brushing your teeth, cleaning your interdental spaces, and using antimicrobial mouthwashes are the main preventative measures. It is desirable to use alternative preventative methods because many people do not practice dental hygiene to the extent necessary to prevent gingivitis. Recently, there has been an increase in interest in the use of dietary fibers or beneficial bacteria to maintain plaque in a state associated with health and enhance periodontal health (5). A deeper comprehension of the bacterial composition of plaque under healthy conditions and its alterations during the initial stages of gingivitis is essential. The microbiota linked to chronic periodontitis has been studied more thoroughly and was recently reviewed in detail (6).

An experimental gingivitis model was used to initially illustrate the significant role that plaque performs in gingivitis (7, 8). Changes in the major bacterial forms in plaque along the progression from gingivitis to health were observed under a microscope. Researchers found that a comparatively basic bacterial community that included Gram-positive cocci and rods made up early plaque in a healthy state. The bacterial communities became more complicated as plaque and gingivitis progressed, with increased amounts of Gram-negative rods, fusiform, filaments, spirilla, and spirochetes (5).

Methodology

This study is based on a comprehensive literature search conducted on 22 May 2024, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed classification and most common causative organisms in gingivitis. There were no restrictions on date, language, participant age, or type of publication.

Discussion

Gingivitis can be plaque induced typically due to poor oral hygiene or non–plaque induced.

Plaque induced gingivitis

Plaque-induced gingivitis is primarily caused by the accumulation of bacterial plaque on the teeth. The bacteria in dental plaque form a biofilm that adheres to the tooth surfaces and gums. Key bacterial species implicated in plaque-induced gingivitis include *Streptococcus* and *Actinomyces* species, as

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well as gram-negative anaerobes like *Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola* (9). The toxins and enzymes produced by these bacteria cause the gingival tissues to become inflamed. The host's immune response to these bacterial products leads to clinical signs of gingivitis, such as swelling, redness, and bleeding of the gums. Additionally, factors such as hormonal changes, systemic diseases like diabetes, certain medications, and poor nutrition can exacerbate the inflammatory response to the bacterial plaque.

Non–plaque-induced gingivitis

1. Genetic/developmental abnormalities

Clinically, gingival fibromatosis may present gingival overgrowth in varying degrees. Hereditary gingival fibromatosis is an uncommon disorder that can develop as an isolated disease or as part of a syndrome, in contrast to drug-related gingival overgrowth (10).

2. Infections

2.1. Bacterial origin necrotizing periodontal disease

Necrotizing gingivitis (NG), necrotizing periodontitis (NP), and necrotizing stomatitis (NS) are severe inflammatory periodontal diseases caused by bacterial infections in patients with specific underlying risk factors such as poor oral hygiene, smoking, stress, poor nutrition, and compromised immune status (e.g., HIV). The term gingivitis refers to lesions that involve only gingival tissue and are characterized by no loss of periodontal attachment (11). Central necrosis of the papillae may lead to significant tissue destruction with the formation of a crater. If attachment loss is established, the diagnosis becomes NP (12). For lesions with ulceration extending more than 1.0 cm from the gingival margin, including tissue beyond the mucogingival junction, the term NS is used. The three necrotizing diseases represent various stages of the same disease process, and a distinction between the different manifestations has not always been made in literature. Consequently, the term necrotizing periodontal disease (NPD) is proposed as a common term encompassing NG, NP, and NS. The constant flora primarily contains *Treponema spp., Selenomonas spp., Fusobacterium spp., and Prevotella intermedia*, while the variable flora consists of a heterogeneous array of bacterial types (13).

Other bacterial infections

Non–plaque-associated bacterial infections of the gingiva are uncommon. Gingivitis caused by a specific bacterial infection may arise due to a loss of homeostasis between non–plaque-related pathogens and innate host resistance. Other examples of specific bacterial infections of gingiva may include *Neisseria gonorrhoeae* and *Treponema pallidum*. Orofacial tuberculosis, which affects 0.1% to 5% of tuberculosis infections overall, is an uncommon extrapulmonary manifestation of the disease (13).

2.2. Viral infection

The most significant viruses causing gingival manifestations are Coxsackie viruses and herpes viruses, including *herpes simplex virus types 1 (HSV-1) and 2 (HSV-2) and varicella-zoster virus* (14). Primary infections can happen in adults as well, even though these viruses usually infect people in childhood. These infections cause oral mucosal disease, which is followed by periods of latency and reactivation.

2.2.1. Coxsackie Viruses

Coxsackie viruses can cause herpangina and hand-foot-and-mouth disease, also known as vesicular stomatitis with exanthema. While herpangina does not involve the gingiva, hand-foot-and-mouth disease is a common contagious vesicular viral disease that affects the skin and oral mucosa, including the gingiva (15).

2.2.2. HSV-1 and HSV-2

HSV-1 typically causes oral manifestations, whereas HSV-2 is primarily associated with anogenital infections and only occasionally causes oral infections (14).

2.2.3 Herpetic gingivostomatitis

Primary herpetic infection typically occurs in infants with an incubation period of about one week. It may be asymptomatic in early childhood but can

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also cause gingivostomatitis with severe symptoms. A characteristic feature is the formation of vesicles that rupture, coalesce, and leave fibrin-coated ulcers of irregular extension. Recurrent intraoral herpes simplex lesions usually occur in adults and have a less severe course, often remaining undiagnosed or mistaken for aphthous ulcerations. However, aphthous ulcers do not typically affect keratinized mucosa (13).

2.2.4. Varicella-Zoster Virus

Primary infection with varicella-zoster virus causes varicella (chicken pox), which affects children. Later reactivation in adults leads to herpes zoster (shingles), presenting with unilateral lesions along the distribution of an infected nerve. If the second or third branch of the trigeminal nerve is involved, skin lesions may be associated with intraoral lesions, including gingival lesions, and intraoral lesions may occur alone. Initial symptoms include pain and paraesthesia, which may precede the appearance of lesions. The initial lesions are vesicles that soon rupture, leaving fibrin-coated small ulcers that often coalesce into irregular forms (13).

2.2.5. Molluscum Contagiosum Virus

The molluscum contagiosum virus, part of the poxvirus family, causes molluscum contagiosum, a contagious disease with infrequent oral manifestations (16, 17). It primarily affects infants with immature immune systems, manifesting as discrete umbilicated papules on the skin. In adults, the disease appears in the genital areas and is often sexually transmitted.

2.2.6. Human Papilloma Virus (HPV)

More than 100 types of HPV have been identified, with at least 25 types detected in oral lesions, including types 1, 2, 3, 4, 6, 7, 10, 11, 13, 16, 18, 31, 32, 33, 35, 40, 45, 52, 55, 57, 58, 59, 69, 72, and 73. Squamous cell papilloma, condyloma acuminatum, verruca vulgaris, and localised epithelial hyperplasia are examples of benign oral lesions linked to HPV infection. These lesions are linked to several distinctive HPV subtypes. Oral benign HPV lesions are mostly asymptomatic and may persist or regress spontaneously (18).

2.3. Fungal infection

Several fungi can cause oral infections, including candidosis, histoplasmosis, aspergillosis, blastomycosis, coccidioidomycosis, paracoccidioidomycosis, cryptococcosis, geotrichosis, and mucormycosis (19). Many of these infections are uncommon, with oral manifestations more likely to occur in individuals with compromised immune systems (20, 21). Oral mycoses can lead to acute, chronic, and mucocutaneous lesions (22). Candidosis is the most common oral mycosis, while histoplasmosis and aspergillosis are less common.

Candidosis

Several Candida species can be isolated from the human oral cavity, including *C. albicans, C. glabrata, C. krusei, C. tropicalis, C. parapsilosis, and C. guilliermondii.* Candida albicans (C. albicans) is the primary cause of candidosis, the most prevalent fungal infection of the oral mucosa. C. albicans is a normal commensal organism of the oral cavity but also acts as an opportunistic pathogen. While candidal infections can occur anywhere in the oral mucosa, gingival lesions are rarely seen in otherwise healthy individuals. The most common clinical characteristic of gingival candidal infection is the redness of the attached gingiva, often presenting with a granular surface. Nodular gingival lesions, which are uncommon, appear as slightly elevated nodules of a white or reddish colour (13). Diagnosis of candidal infection can be made through culture, smear, and biopsy.

3. Inflammatory and immune conditions and lesions

3.1. Hypersensitivity reactions

3.1.1 Contact allergy

Oral mucosal manifestations of allergy are very uncommon. Most frequently, type IV contact allergies are the cause of these responses, which can also be brought on by meals, mouthwashes, dentifrices, and dental restoration materials (13).

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3.1.2. Plasma Cell Gingivitis

Plasma cell gingivitis is an uncommon inflammatory condition usually affecting the anterior maxillary gingiva and of uncertain aetiology (13).

3.1.3 Erythema Multiforme (EM)

EM is an uncommon, self-limiting, acute immune-inflammatory disorder of the oral mucosa. The aetiology of EM is unclear in most patients, but it appears to be an immunologic hypersensitivity reaction mediated by T-lymphocytes. The disorder may present a diagnostic dilemma because of infections particularly herpes simplex and Mycoplasma pneumoniae and some drugs predispose individuals to developing erythema multiforme, in what are believed to be immune complex disorders (23).

3.2. Autoimmune diseases of skin and mucous membranes

3.2.1 Pemphigus Vulgaris (PV)

PV is an autoimmune vesiculo-bullous disease of the skin and mucous membranes. Involvement of the oral mucosa is common, and in about 54% of cases, the oral cavity is reported to be the primary site of involvement (24). The disease is characterized by intraepithelial bullae in the skin and mucous membranes due to autoantibodies directed against desmosome-associated protein antigens (desmoglein-3). Oral mucosal lesions, including gingival lesions, may precede skin involvement (25). In the literature, gingival localization of PV usually manifests as desquamative gingivitis and/or vesiculo-bullous lesions of the free and attached gingiva; early lesions rarely appear as extensive erythema and erosions (13).

3.2.2 Pemphigoid

Pemphigoid refers to a group of mucocutaneous disorders triggered by autoantibodies targeting basement membrane antigens, leading to the separation of the epithelium from the connective tissue. When only mucous membranes are involved, it is commonly referred to as mucous membrane pemphigoid (MMP) (26). Scarring is an important ocular complication but not for oral mucosal lesions. Any area of the oral mucosa can be affected by MMP, but the primary clinical manifestation is desquamative lesions on the gingiva, which appear as intensely erythematous areas. Usually, the bullae rupture rapidly, leaving fibrin-coated ulcers. The separation of epithelium from connective tissue at the basement membrane area is the main diagnostic feature of MMP and circulating serum antibodies are not always revealed by indirect immunofluorescence (13).

3.2.3 Lichen Planus

Lichen planus is a common mucocutaneous disease frequently affecting the gingiva. Oral involvement alone is common (13). The disease may be associated with severe discomfort. Because it has been shown to possess premalignant potential, it is important to diagnose, treat, and monitor patients through regular oral examinations. A randomized controlled trial showed that a tailored plaque-control regime is beneficial in reducing symptoms of gingival lichen planus and improving overall quality of life (27).

3.2.4 Lupus Erythematosus (LE)

LE is a group of autoimmune disorders characterized by autoantibodies to various cellular constituents, including extractable nuclear antigens and cytoplasmic membrane components. Two major forms are described: discoid LE (DLE) and systemic LE (SLE), which may involve a range of organ systems. DLE is a mild chronic form involving the skin and mucous membranes, sometimes including the gingiva as well as other parts of the oral mucosa. Eight percent of patients with DLE develop SLE, and ulcerations may be a sign of SLE. The systemic type may also include skin lesions on the face, but they tend to spread over the entire body (13).

4. Reactive Processes

4.1. Epulides

The term epulis is often used to describe exophytic processes originating from the gingiva. It is a non-specific term, and histopathology provides a more

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specific diagnosis. Many of these processes are reactive lesions, meaning they are non-neoplastic proliferations that clinically resemble benign neoplastic proliferations (28). Typically, there are no symptoms, although these reactive processes are thought to represent an exaggerated tissue response to local irritation or trauma.

4.2. Fibrous epulis

Among 2,068 cases of reactive lesions of the oral cavity, the attached gingiva was the most frequently affected location, with 1,331 cases (64.36%) (28). Fibrous epulides (focal fibrous hyperplasia, irritation fibroma) are common exophytic smooth-surfaced pink masses of fibrous consistency attached to the gingiva. The size varies from small to large tumour-like processes with a diameter of several centimetres (13).

4.3. Calcifying fibroblastic granuloma

Calcifying fibroblastic granuloma (ossifying fibroid epulis, peripheral ossifying fibroma) occurs exclusively on the gingiva. Although usually smaller than 1.5 cm in diameter, the lesion can reach a larger size and may rarely cause separation of the adjacent teeth and resorption of the alveolar crest (13).

4.4. Pyogenic granuloma

The pyogenic granuloma (telangiectatic granuloma, pregnancy granuloma, pregnancy tumour, vascular epulis) is common and shows a striking predilection for the gingiva, which accounts for 75% of all cases (13). When occurring during pregnancy, the influence of female sex hormones may result in a biologic behaviour distinct from other pyogenic granulomas.

Conclusion

Effective management of gingivitis requires precise classification and identification of causative organisms. Specific approaches for both plaque-induced and non-plaque-induced forms are essential to prevent progression to more severe periodontal diseases, highlighting the importance of understanding the diverse aetiologies and implementing targeted preventive and therapeutic strategies.

Disclosure

Conflict of interest

There is no conflict of interest.

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Ethical Consideration

Not applicable.

Data Availability

Data that supports the findings of this study are embedded within the manuscript which is based on a comprehensive literature search conducted in May 2024, in the Medline and Cochrane databases.

Author Contribution

The authors contributed to conceptualizing, data drafting, collection and final writing of the manuscript.

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