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Human Variation In Gingival Inflammation

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Abstract:

Gingivitis is an inflammatory condition of the gingival tissue, most commonly caused by bacterial infection. Oral commensal bacteria actively participate with gingival tissue to maintain healthy neutrophil surveillance and normal tissue and bone turnover processes. Disruption of this homeostatic host–bacteria relationship occurs during experimental gingivitis studies where it has been clearly established that increases in the bacterial burden increase gingival inflammation.

Gingivitis follows a linear and progressive course when a healthy individual stops oral care. It is not known if and when gingivitis transforms into periodontitis. A very limited number of studies present direct evidence regarding the histological changes over time and how they correlate to the clinical transition from gingivitis to periodontitis.

Periodontitis there is no attachment loss and therefore no migration of the junctional epithelium. The condition is restricted to the soft-tissue area of the gingival epithelium and connective tissue. Among all the periodontal diseases, gingivitis is considered to be the commonest.

There are various forms of gingivitis based on clinical appearance, duration of infection, severity, and etiology. However, the chronic form of gingivitis that is caused by plaque is considered to be the most frequent variant. Clinically, the gingival tissues are characterized by swelling, redness, tenderness, a shiny surface, and bleeding upon gentle probing.

Gingivitis seldom generates spontaneous bleeding and is commonly painless, therefore many patients do not recognize the disease and fail to seek attention.

1. Introduction:

Inflammation underlies a wide variety of physiological and pathological processes. Although the pathological aspects of many types of inflammation are well appreciated, their physiological functions are mostly unknown. The classic instigators of inflammation (infection and tissue injury) are at one end of a large range of adverse conditions that induce inflammation, and they trigger the recruitment of leukocytes and plasma proteins to the affected tissue site **(1).**

Gingiva, with its unique texture and coral pink color**,** is the most delicate tissue in the oral cavity and the first essential component of the periodontium (**Figure 1**).

Two common diseases – gingivitis and periodontitis – affect the periodontium. Symptoms of disease entities are used for distinguishing various forms of gingivitis and periodontitis. Gingivitis follows a linear and progressive course when a healthy individual stops oral care, very limited number of studies present direct evidence regarding the histological changes over time and how they correlate to the clinical transition from gingivitis to periodontitis **(22).**

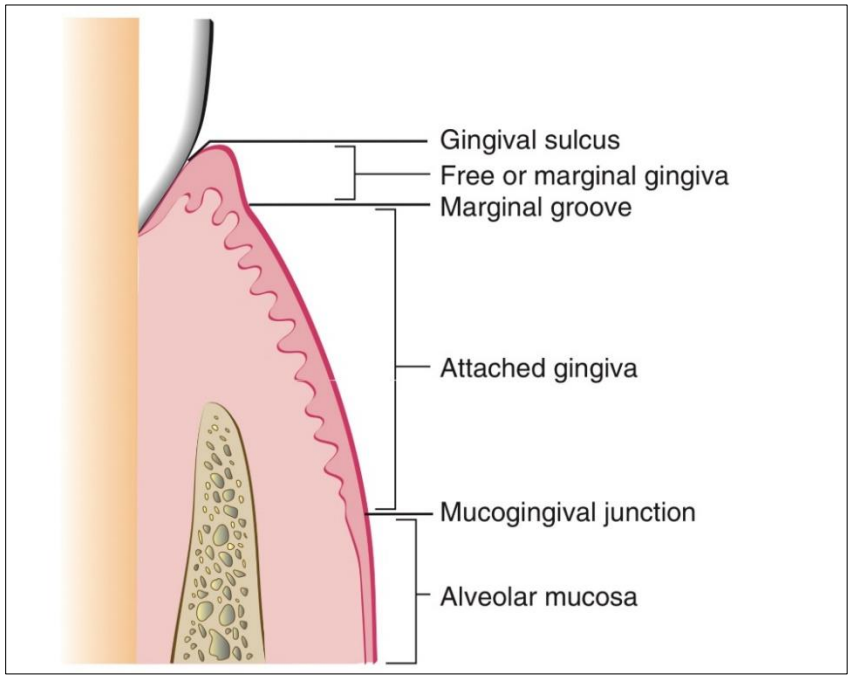

Figure 1. The Periodontium components.

The gingiva, which covers the alveolar bone, is classified as a masticatory portion of oral mucosa. Anatomically, there are three demarcated parts of gingiva. First, the marginal gingiva, which is the free end of gingiva with a smooth surface, enclosing the neck of the teeth as a collar shape to define the gingival sulcus. The second part is the attached gingiva which is stippled, firm, and strongly attached to the alveolar bone and to the cervical area of the tooth by means of junctional epithelium located in the floor of gingival sulcus. The conjunction between the free and attached gingiva is a shallow linear depression called gingival groove **(23).** The attached gingiva extends apically to the oral mucosa, from which it is demarcated by mucogingival junction (**Figure 2**).

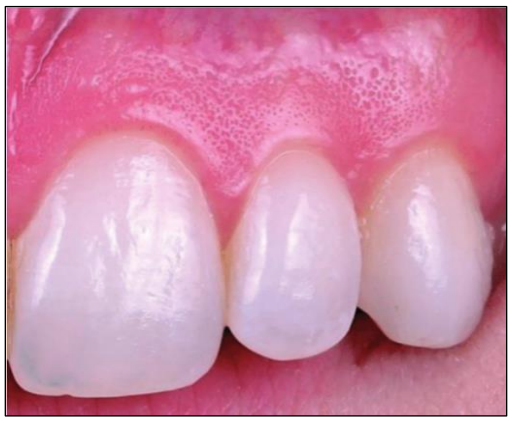

Figure 2. The free and attached gingiva.

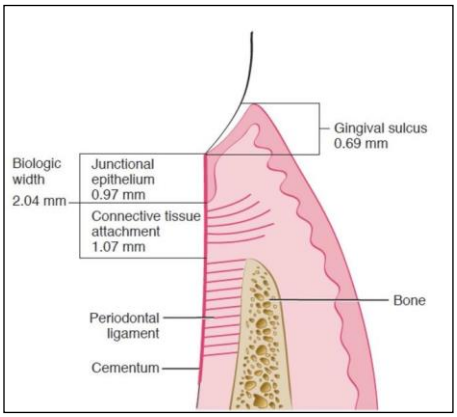

Figure 3. The biological width.

The third part is the interdental zone of gingiva, which is non keratinized and located in the area between the two adjacent teeth beneath the contact point. The biological width or the supracrustal tissue attachment is a natural protective layer, which seals and preserves the periodontium from bacterial invasion, located in the deeper part of gingival sulcus and measuring 2.04 mm in depth (**Figure 3**), which is the sum of junctional epithelium 0.97 mm and supracrestal connective tissue attach meant 1.07 mm **(2).**

2. Review

2.1. Etiology:

Gingivitis is caused by the microbial plaque deposits located in or close to the gingival sulcus. The microorganisms more strongly associated with the etiology of gingivitis include species of Streptococcus, Fusobacterium, Actinomyces, Veillonella, and Treponema. Bacteroides, Capnocytophaga, and Eikenella are also potentially linked to the etiology of the disease **(2,3)**.
There may be other local or systemic etiologic factors that intensify plaque deposition or the vulnerability of the tissue to the microbial attack **(3)**.

Based on the etiology, gingivitis can be classified into different types:

2.1.1. Plaque Induced Gingivitis:

This is the most common cause of gingivitis. Plaque is a thin film that forms on the tooth surface due to poor oral hygiene. If not regularly removed, it can harden up and form calculus.
As plaque harbors a large number of bacteria, inflammation can occur in the gingival tissue. Some local factors can contribute to the formation of plaque, such as crowding of teeth due to which plaque removal becomes difficult **(4).**

As misaligned teeth often require orthodontic correction, cleaning difficulty increases accumulating more plaque. Furthermore, a dental prosthesis that does not have an adequate fit or is not properly finished can also act as a nidus for plaque accumulation.
In children, tooth eruption is frequently associated with gingivitis as plaque accumulation tends to increase in the area where primary teeth are exfoliating, and permanent teeth are erupting as oral hygiene may be difficult to be maintained in these areas (**Figure 4**). This is referred to as eruption gingivitis **(5).**

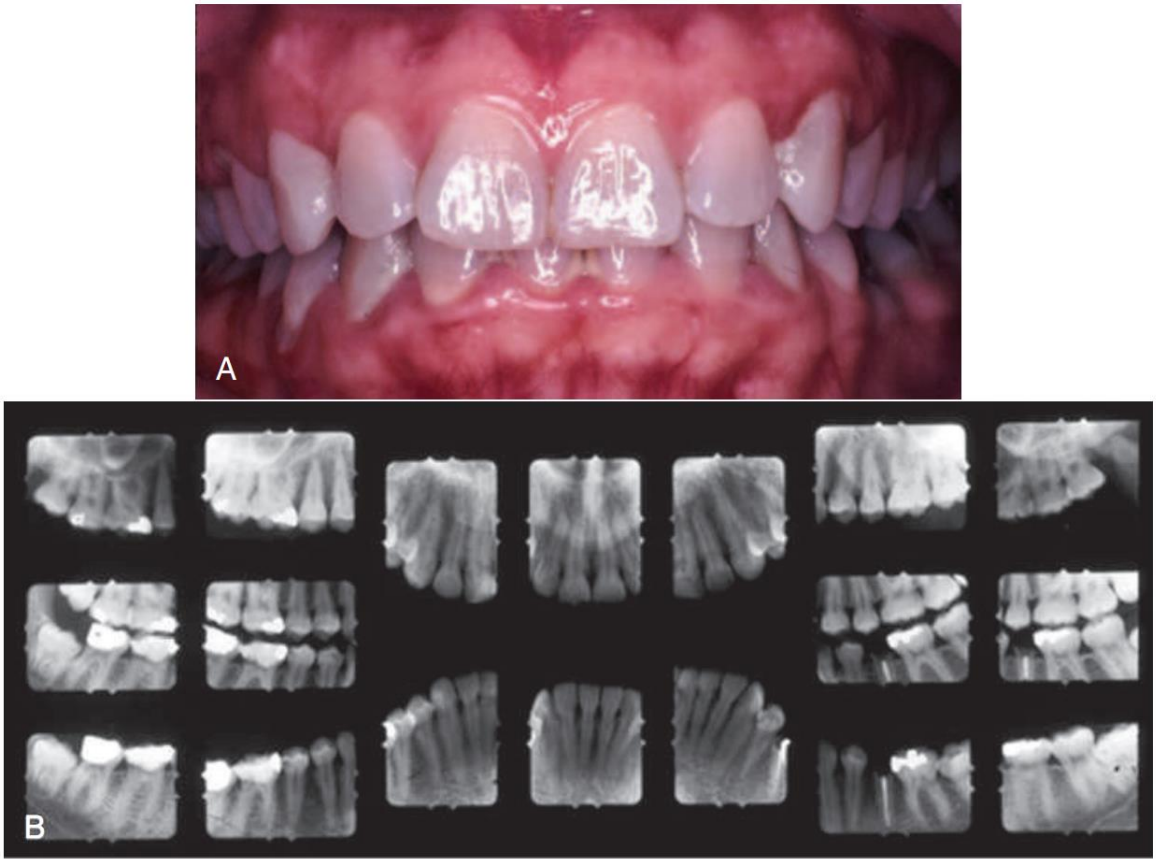

Figure 4. (A) Plaque-related gingivitis depicts marginal and papillary inflammation, with 1- to 4-mm probing depths and generalized zero clinical attachment loss, except recession in tooth.

(B) Radiographic images of the patient.

2.1.2 Nutritional Gingivitis:

This may occur due to a deficiency of vitamin C. It has been found that a modern lifestyle with the intake of an increased amount of refined carbohydrates and an increased ratio of omega-6 to omega-3 fatty acids can promote the inflammatory process. The mechanism by which carbohydrates with a high glycemic index promote the inflammatory process is through activation of NFkB and oxidative stress **(6)**.

2.1.3 Gingival Diseases Modified by Systemic Factors:

Systemic factors that contribute to gingivitis—such as the endo-crine changes associated with puberty (**Figure 5**), the menstrual cycle, pregnancy (**Figure 6**), and diabetes—may exacerbate the gingival inflammatory response to plaque. This altered response appears to result from the effects of systemic conditions on the host's cellular and immunologic functions, but the primary etiologic factor is still considered to be microbial plaque **(7 and 23).** One example of altered host response due to systemic factors is apparent during pregnancy when the incidence and severity of gingival inflammation may increase even in the presence of low levels of plaque **(22).** In blood dyscrasias (e.g., leukemia), the reduced number of immunocompetent lymphocytes in the periodontal tissues is associated with increased edema, erythema, and bleeding of the gingiva as well as gingival enlargement that may be associated with the swollen, spongy gingival tissues caused by the excessive infiltration of malignant blood cells (**Figure 7**) **(7,8 and 23).**

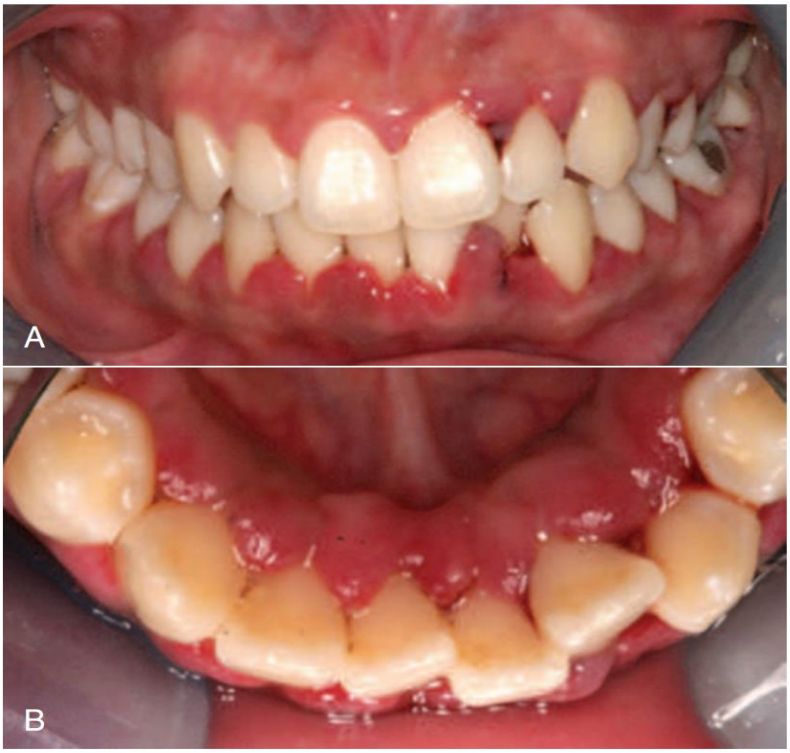

Figure 5. 13-year-old female with hormone-exaggerated marginal and papillary inflammation, with (1-4mm) probing depths yet minimal clinical attachment loss. (A) Facial view. (B) Lingual view.

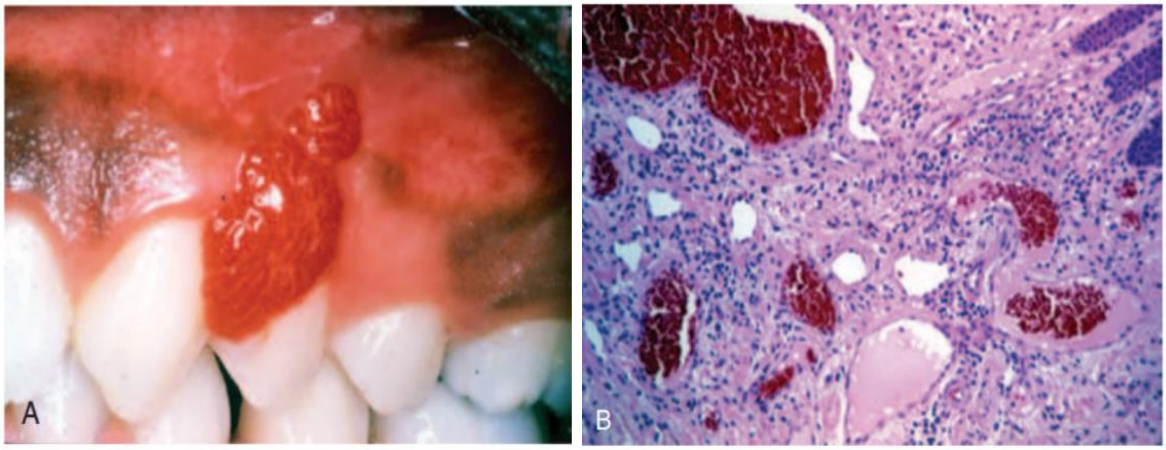

Figure 6. **(A) Clinical image of pyogenic granuloma in a 27-year-old pregnant female. (B) Histologic image depicts dense inflammatory infiltrate and prominent vessels.**

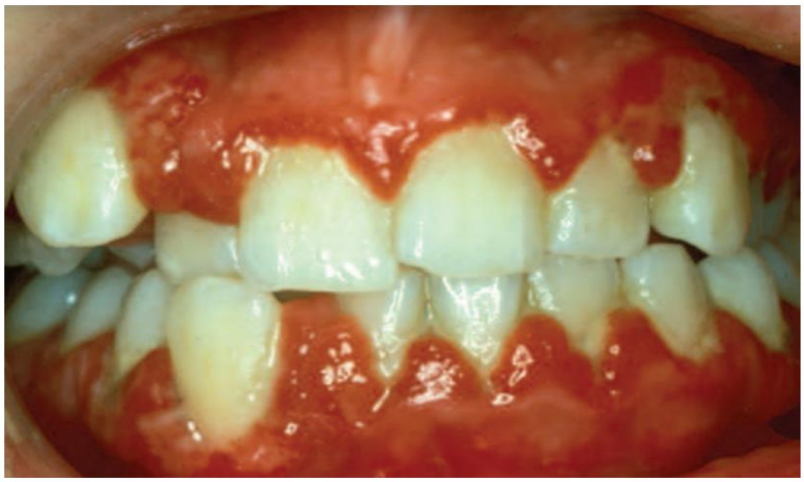

Figure 7. **A 12-year-old female with a primary medical diagnosis of leukemia that exhibits swollen/spongy gingiva.**

2.1.4 Drug-Induced Gingivitis:

Various drugs used for systemic conditions can cause gingivitis as a side effect such as phenytoin (used for epileptic seizures), calcium channel blockers (used for angina, high blood pressure), anticoagulants, and fibrinolytic agents, oral contraceptive agents, protease inhibitors, vitamin A and analogs. The mechanism behind this gingival inflammation is thought to be the ability of the metabolites of these drugs to induce the proliferation of fibroblasts **(9).**

Drug-induced gingival overgrowth remains a significant problem for the periodontologist. Many patients medicated with the drugs implicated in this unwanted effect experience significant, recurrent gingival problems that require repeated surgical excisions **(24).**

An imbalance between the synthesis and the degradation of the extracellular matrix leads to the accumulation of immature proteins in the extracellular matrix, particularly collagen. This, in turn, results in gingivitis (**Figure 8,9**) **(9).**

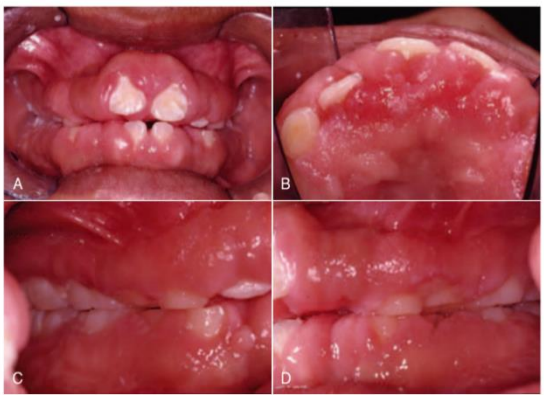

Figure 8. Clinical images of a 9-year-old male with severe gingival overgrowth secondary to heart transplant and cyclosporine therapy.

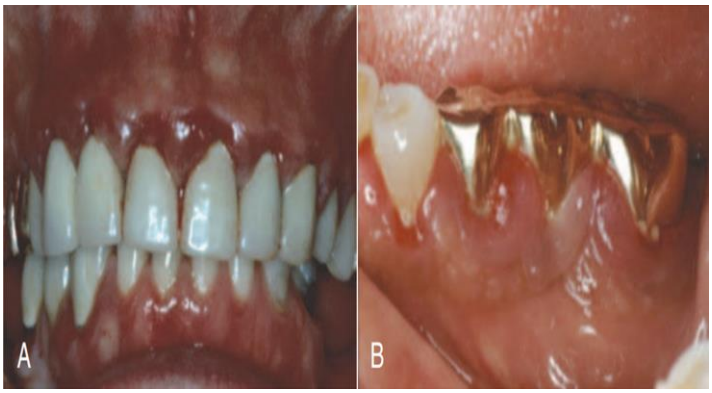

Figure 9. Clinical images of gingival overgrowth following use a of calcium channel blocker to control hypertension.

2.2.Pathophysiology:

Periodontal disease undergoes four different stages that were first explained by Page and Schroeder in 1976. Pathophysiologically, gingivitis has been divided into initial, early, and established stages, and periodontitis has been indicated as the advanced stage. Chronic periodontitis, a common disease of microbial origin, is the major cause of tooth loss in adult humans. The disease serves as a convenient experimental model for analysis of many aspects of chronic inflammation **(10).** Clinical gingival health is generally associated with an inflammatory infiltrate and a host response consistent with homeostasis. The molecules that play a role in the pathogenesis are divided into two main groups: those derived from the subgingival microbiota (i.e., microbial virulence factors) and those derived from the host immune-inflammatory response. The immune system is essential for the maintenance of periodontal health and is categorized as innate immune system and the adaptive immune system. Innate immunity reflects the capacity of the host to defend against infectious attacks. Understanding the disease processes is important for the development of improved treatment strategies **(25).**

2.3.The Gingival Index (GI) and Bleeding on Probing Review:

Two common measures of gingival inflammation are the Gingival Index (GI) and bleeding on probing.

2.3.1 The Gingival Index (GI):

Is proposed in 1963 as a method for assessing the severity and quantity of gingival inflammation. With this particular index, only gingival tissues are assessed. Each of the four gingival areas of the tooth (i.e., facial, mesial, distal, and lingual) are assessed for inflammation and rated as: normal gingiva (a score of 0) to severely inflamed gingiva with a tendency to spontaneously bleed (a score of 3) **(26).**

Gingiva that is mildly inflamed but without bleeding on probing is given a score of 1, whereas moderately inflamed gingiva with bleeding is given a score of 2. The scores can be averaged for each patient to provide patient means. Alternatively, site-specific analyses can relate local and patient-speciic factors to the GI that is measured at individual sites **(11).**

2.3.2. Bleeding on Probing:

is another measure of periodontal inflammation. The specific approach to obtain a bleeding measure can vary from one study to the next as well as from one clinician to another , for example, in the third National Health and Nutrition Examination Survey (NHANES III),52 bleeding measures were obtained as follows **(26).**

First, the facial and mesiofacial sites of teeth in two randomly selected quadrants—one maxillary and one mandibular—were selected. a special probe known as the National Institute of Dental Research probe was used in these assessments. This color-coded probe is marked at 2, 4, 6, 8, 10, and 12 mm **(11).**

To begin the assessment, the examiner dried a quadrant of teeth with air. Then, starting with the most posterior tooth in the quadrant (excluding the third molar), the examiner placed a periodontal probe 2 mm into the gingival sulcus at the facial site and carefully swept the probe from the mesiofacial to the mesial interproximal area. After probing the sites in the quadrant, the examiner assesses the presence or absence of bleeding at each probed site. The same procedure was repeated for the remaining quadrant **(11).**

2.4.Evaluation and Differential Diagnosis:

As gingivitis is a soft tissue disease, radiographic evaluation is not usually necessary; however, it may be of help for differentiating gingivitis from periodontitis in some cases. Lab investigations are also routinely not required.

Gingivitis can be differentiated from periodontitis by the attachment loss undergone in the latter that can be clinically noticed during periodontal probing. They can also be differentiated histologically and radiographically **[12].**

2.5.Treatment / Management:

The prime objective of treating gingivitis is to reduce inflammation. This is achieved by the use of different instruments to remove dental plaque deposits **.** Gingivitis, in its initial stages, can be easily managed if the patient starts following oral hygiene protocol, which includes regular tooth brushing with an appropriate technique and interproximal hygiene, such as dental flossing, or the use of interproximal brushes **(13).**

The removal of plaque and calculus is also professionally achieved by scaling and root planning according to the severity of the condition. If it is a drug-induced gingival overgrowth, the physician can change the medication to improve the outcome of treatment of the condition. If it is due to nutritional deficiency, supplements can be prescribed. Medications in the form of antiseptic mouthwash that contains chlorhexidine can also be prescribed in conjunction with the mechanical removal of plaque **(13).**

It has been suggested that the use of chlorhexidine mouthwashes in addition to the usual toothbrushing and interproximal cleaning leads to a significant decrease in the build-up of dental biofilm. The concentration of the chlorhexidine rinse does not affect its effectiveness **(14).**

There are studies on the effect of medicinal or herbal plants on the management of gingivitis. The mechanism of action of these plants on gingivitis is due to their anti-inflammatory property. Such medicinal plants include pomegranate, tea, and chamomile. The flavonoids and tannins present in these plants are potent anti-inflammatory and astringent phytochemicals. Therefore, they can resolve both gingival bleeding and inflammation **(15).**

Some studies proved that there is a synergistic effect when the herbal plants are prescribed along with conventional mechanical procedures of plaque removal, such as scaling **(16).**

To improve the treatment outcome of gingivitis, an interprofessional approach is required to identify the causes of the disease and to intervene at an early stage. Also, a thorough knowledge of the epidemiological pattern is required for planning the public-health services as plaque-induced gingivitis can be seen at any age of the dentate population. Periodontal disease is not just limited to the destruction of the periodontium, it affects general systemic health too. Thus, both dentists and physicians must be aware of the close link between periodontal disease and systemic diseases, such as diabetes mellitus, cardiovascular diseases, and preterm birth or low birth weight (PLBW) **(17,18 and 19).**

2.6.Prognosis and Complications:

Gingivitis, if identified and treated, can easily be resolved as the condition is reversible and the altered tissues can return to normal once the dental biofilm has been removed. If gingivitis progress to periodontitis, connective tissue attachment loss, and bone destruction will occur, which may ultimately result in tooth loss **(20).**

The most common complication or sequelae of chronic gingivitis is the progression of the inflammation towards the underlying tissue and bone, resulting in periodontitis.The ultimate consequence of such an event is tooth loss. Gingivitis is a precursor of periodontitis. However, gingivitis does not always progress to periodontitis **(21).**

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