Periodontal Health, Gingival Diseases and Conditions

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Clinical Application
Examination of the gingiva is part of every patient visit. In this context, a thorough clinical and radiographic assessment of the patient’s gingival tissues provides the dental practitioner with invaluable diagnostic information that is critical to determining the health status of the gingiva. The dental hygienist is often the first member of the dental team to be able to detect the early signs of periodontal disease. In 2017, the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) developed a new worldwide classification scheme for periodontal and peri-implant diseases and conditions. Included in the new classification scheme is the category called "periodontal health, gingival diseases/conditions." Therefore, this chapter will first review the parameters that define periodontal health. Appreciating what constitutes as periodontal health serves as the basis for the dental provider to have a stronger understanding of the different categories of gingival diseases and conditions that are commonly encountered in clinical practice.

Learning Objectives
- Define periodontal health and be able to describe the clinical features that are consistent with signs of periodontal health.
- List the two major subdivisions of gingival disease as established by the American Academy of Periodontology and the European Federation of Periodontology.
- Compare and contrast the etiologic factors associated with dental biofilm-induced gingivitis and non–plaque-induced gingival diseases.
- List the conditions that are classified under the non–plaque-induced gingival diseases category.
- Describe the differences between an intact periodontium and a reduced periodontium.
- Differentiate papillary gingivitis, marginal gingivitis, and diffuse gingivitis.
- Describe the clinical signs of inflammation characteristic of moderate plaque-induced gingivitis.
- Describe how systemic factors can modify the host response to plaque biofilm and lead to gingival inflammation.

Key Terms
- Periodontal health
- Intact periodontium
- Reduced periodontium
- Periodontal health on an intact periodontium
- Periodontal health on a reduced periodontium in a non-periodontitis patient
- Periodontal health on a reduced periodontium in a successfully treated stable periodontitis patient
- Plaque-induced gingivitis
- Papillary gingivitis
- Marginal gingivitis
- Diffuse gingivitis
- Acute gingivitis
- Chronic gingivitis
- Gingivitis on an intact periodontium
- Gingival inflammation on a reduced periodontium in a successfully treated stable periodontitis patient
- Pregnancy-associated pyogenic granuloma
- Hyperglycemia
- Drug-influenced gingival enlargements
- Non–plaque-induced gingival diseases
- Lichen planus
Section 1
Periodontal Health

The 2017 World Workshop defines periodontal health as simply “a state free from inflammatory periodontal disease that allows an individual to function normally and avoid consequences (mental or physical) due to current or past disease.”

1. Characteristics of Periodontal Health. Clinically, periodontal health is characterized by the absence of bleeding on probing, erythema, edema, patient symptoms and attachment and bone loss.

2. Categories of Periodontal Health
   A. Normal versus Reduced Periodontium. The 2017 classification system recognizes that periodontal health can occur either on an intact periodontium or on a reduced periodontium.
      1. An intact periodontium—a periodontium with no loss of periodontal tissue (no loss of connective tissue or alveolar bone).
      2. A reduced periodontium—a periodontium with pre-existing loss of periodontal tissue but, is not currently undergoing loss of connective tissue/alveolar bone.
   B. Three Categories of Periodontal Health. The following describes the three categories of periodontal health as described by the 2017 World Workshop.
      1. Periodontal health on an intact periodontium—clinical signs characteristic of periodontal health coupled with an intact periodontium (Box 6-1, Fig. 6-1).
      2. Periodontal health on a reduced periodontium in a non-periodontitis patient—clinical signs of periodontal health on a periodontium with a pre-existing loss of connective tissue and/or loss of alveolar bone which is attributed to non-periodontitis reasons (Box 6-2, Fig. 6-2). For example, recession of the gingival margin that occurs from a history of traumatic toothbrushing or the deliberate removal of alveolar bone that occurs in surgical crown lengthening.
      3. Periodontal health on a reduced periodontium in a successfully treated stable periodontitis patient—clinical signs of periodontal health on a periodontium with a pre-existing loss of connective tissue and alveolar bone which is attributed to periodontitis but, has been successfully treated and is currently stable (Box 6-3, Fig. 6-3).

Box 6-1. Periodontal Health on an Intact Periodontium

Figure 6-1. Periodontal Health on an Intact Periodontium. This patient exhibits no clinical signs of gingival inflammation and no previous loss of periodontal tissues. The dental radiograph does not reveal any changes in either the alveolar bone height or the architecture and morphology of the alveolar bone.
Part 2 Diseases Affecting the Periodontium

Box 6-2. Periodontal Health on a Reduced Periodontium in a Non-periodontitis Patient

Figure 6-2. Periodontal Health on a Reduced Periodontium in a Non-periodontitis Patient. This is a 54-year-old male patient with a history of excessive frequent daily toothbrushing (he brushed five to six times a day with a hard-bristled toothbrush).

- Note the generalized recession of the gingival margin. However, there are no evident signs of clinical inflammation.
- The dental radiograph does not reveal any changes in either the alveolar bone height or the architecture and morphology of the alveolar bone.
- Treatment objectives for this case are to (1) address the patient’s self-care habits and reinforce proper oral hygiene and (2) periodically monitor the patient’s periodontal status to prevent further loss of tissue.

Box 6-3. Periodontal Health on a Reduced Periodontium in a Successfully Treated Periodontitis Patient

Figure 6-3. Periodontal Health on a Reduced Periodontium in a Successfully Treated Stable Periodontitis Patient. In 2016, this generalized periodontitis patient underwent nonsurgical and surgical periodontal therapy to control the disease. Eighteen months later, the above photo was taken to illustrate how healthy her gingiva looks following therapy and meticulous home care.

- The dental radiograph reveals loss of alveolar bone height.
- Future treatment objectives should be to (1) maintain the reduced periodontium in a healthy and stable state and (2) prevent the reactivation of periodontitis.
Section 2

Dental Plaque-Induced Gingival Conditions

As illustrated in Figure 6-4, gingival diseases and conditions are broadly classified as either (1) gingivitis that is dental biofilm-induced or (2) gingival diseases that are not dental biofilm-induced. This section discusses the first and most common of these, gingivitis that is dental biofilm-induced.2

![Gingival Diseases and Conditions](image)

**Figure 6-4. Gingival Diseases and Conditions.** Gingival diseases and conditions are classified as either dental biofilm-induced or non–dental biofilm-induced.

**CLASSIFICATION OF PLAQUE-INDUCED GINGIVITIS AND MODIFYING FACTORS**

Plaque-induced gingivitis is an inflammatory response of the gingival tissues resulting from bacterial plaque biofilm accumulation located at and below the gingival margin. Löe and colleagues first described gingivitis in their landmark research in 1965.3 The 2017 classification scheme for plaque-induced gingivitis is outlined in Table 6-1.2 The severity of plaque-induced gingivitis can be influenced by various systemic and tooth-related factors.

<table>
<thead>
<tr>
<th>TABLE 6-1</th>
<th>CLASSIFICATION OF PLAQUE-INDUCED GINGIVITIS AND MODIFYING FACTORS</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.</td>
<td>Associated with bacterial dental biofilm only</td>
</tr>
<tr>
<td>B.</td>
<td>Potential modifying factors of plaque-induced gingivitis</td>
</tr>
<tr>
<td>1.</td>
<td>Systemic conditions</td>
</tr>
<tr>
<td>a)</td>
<td>Sex and steroid hormones</td>
</tr>
<tr>
<td>1)</td>
<td>Puberty</td>
</tr>
<tr>
<td>2)</td>
<td>Menstrual cycle</td>
</tr>
<tr>
<td>3)</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>4)</td>
<td>Oral contraceptives</td>
</tr>
<tr>
<td>b)</td>
<td>Hyperglycemia</td>
</tr>
<tr>
<td>c)</td>
<td>Leukemia</td>
</tr>
<tr>
<td>d)</td>
<td>Smoking</td>
</tr>
<tr>
<td>e)</td>
<td>Malnutrition</td>
</tr>
<tr>
<td>2.</td>
<td>Oral factors enhancing plaque biofilm accumulation</td>
</tr>
<tr>
<td>a)</td>
<td>Prominent subgingival restoration margins</td>
</tr>
<tr>
<td>b)</td>
<td>Hyposalivation (reduced saliva production)</td>
</tr>
<tr>
<td>C.</td>
<td>Drug-influenced gingival enlargements</td>
</tr>
</tbody>
</table>
PLAQUE-INDUCED GINGIVITIS

1. Characteristics of Plaque-Induced Gingivitis
   A. The Most Common Form of Periodontal Disease. Plaque-induced gingivitis is by far the most common type of periodontal disease. It does not directly cause tooth loss; however, managing gingivitis is the primary strategy for preventing periodontitis.\(^4\) Epidemiologic data show that plaque-induced gingivitis is prevalent at all ages in the population.\(^5,6\)

   1. The intensity of the clinical signs and symptoms of gingivitis may vary between individuals and within the dentition of an individual.\(^7\)

   2. Plaque-induced gingivitis differs between children and adults (for more detailed information regarding the periodontium of a child patient, refer to Chapter 31, Periodontal Disease in the Pediatric Population).
      a. Inflammation is not as intense in children versus young adults with the same quantity of plaque biofilm.\(^5,10\)
      b. Adolescents may have elevated levels of certain bacteria: *Actinomyces*, *Capnocytophaga*, *Leptotrichia*, and *Selenomonas* species.\(^11\)
      c. Children may have fewer pathogenic bacteria in their plaque biofilm, a thicker junctional epithelium, and a less developed immune response.\(^12,13\)
      d. Gingival inflammation in adults is more pronounced even when similar amounts of plaque biofilms are present, perhaps attributed to age-related differences in cellular inflammatory response to plaque biofilm.\(^14,15\)

   3. Local factors—such as dental restorations, appliances, root fractures, and tooth anatomy—act as sites for plaque biofilm retention and may contribute to progression of the disease.

   B. Clinical Signs of Plaque-Induced Gingivitis
      1. Common clinical signs of plaque-induced gingivitis include changes in gingival color, contour, and consistency. These common signs include redness (erythema), swelling (edema), bleeding, increased gingival crevicular fluid, and tenderness.\(^4,16\)

      2. The earliest signs of gingivitis are seen in the papillary region. This is known as *papillary gingivitis*. Papillary gingivitis is gingival inflammation that involves the interdental papilla. If the papillary gingival inflammation extends into the adjacent gingival margin, the condition is known as *marginal gingivitis*. Marginal gingivitis involves the gingival margin and a portion of the contiguous attached gingiva. If the gingival inflammation affects all three parts of the gingiva—the interdental papilla, the marginal gingiva, and the attached gingiva—this is known as *diffuse gingivitis*.

      3. It should be noted that gingivitis is a clinical diagnosis. Thus, radiographs, alone, cannot be used to diagnose gingivitis.

   C. Duration of Plaque-Induced Gingivitis
      1. Acute gingivitis—gingivitis of a sudden onset and short duration, after which professional care and patient self-care returns the gingiva to a healthy state.

      2. Chronic gingivitis—long-lasting gingivitis; gingivitis may exist for years without ever progressing to periodontitis. Chronic gingivitis is typically painless and is more commonly encountered than acute gingivitis.

2. Categories of Plaque-Induced Gingivitis
   A. Three Categories of Plaque-Induced Gingivitis. The 2017 classification system recognizes that plaque-induced gingivitis can occur either on an intact periodontium or on a reduced periodontium. Table 6-2 summarizes the key clinical features for each category.
1. **Gingivitis on an intact periodontium** (Box 6-4, Fig. 6-5)—the presence of plaque-induced inflammation on an intact periodontium.

2. **Gingivitis on a reduced periodontium in a non-periodontitis patient** (Box 6-5, Fig. 6-6)—the presence of plaque-induced inflammation on a periodontium with pre-existing loss of connective tissue and/or loss of alveolar bone that can be attributed to non-periodontitis reasons.

3. **Gingival inflammation on a reduced periodontium in a successfully treated stable periodontitis patient** (Box 6-6, Fig. 6-7).

**B. Plaque-Induced Gingivitis on a Reduced Periodontium After Successful Treatment**

1. In someone successfully treated for periodontitis, if the extent of attachment loss present remains stable over months or years, then the presence of attachment loss is not an indication of active periodontitis.

2. Plaque-induced gingivitis on a reduced periodontium in a successfully treated periodontitis case is characterized by the return of bacteria-induced inflammation to the gingival margin on a reduced periodontium with no evidence of progressive attachment loss. If a clinician is going to make the diagnosis of “gingivitis on a reduced but stable periodontium,” it is necessary to demonstrate that attachment loss (loss of connective tissue and alveolar bone) is not ongoing.

3. The common clinical findings on a reduced periodontium are the same as plaque-induced gingivitis except for the presence of pre-existing attachment loss and therefore a higher risk of periodontitis. Due to this higher risk for recurrence of periodontitis, individualized professional care is of utmost importance for the patient with plaque-induced gingivitis on a reduced periodontium.

4. Although a successfully treated periodontitis patient exhibits an absence of ongoing, progressive periodontitis, the patient cannot revert to being classified as a “gingivitis case on an intact periodontium.”
   a. Periodontitis is an irreversible condition that results in a permanent loss of attachment and alveolar bone.
   b. A periodontitis patient remains a periodontitis patient for life—even following successful periodontal therapy—and requires lifelong supportive care to prevent recurrence of disease.

**TABLE 6-2**  **KEY FEATURES OF PERIODONTAL HEALTH AND DENTAL PLAQUE-INDUCED GINGIVITIS**

<table>
<thead>
<tr>
<th>Intact Periodontium</th>
<th>Health</th>
<th>Gingivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical attachment loss</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Radiological bone loss</td>
<td>No</td>
<td>No</td>
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</table>

<table>
<thead>
<tr>
<th>Reduced Periodontium, Non-periodontitis Patient</th>
<th>Health</th>
<th>Gingivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical attachment loss</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Radiological bone loss</td>
<td>Possible⁴</td>
<td>Possible⁴</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Successfully Treated Stable Periodontitis Patient</th>
<th>Health</th>
<th>Gingivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical attachment loss</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Radiological bone loss</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*Clinical attachment loss would be present on a reduced periodontium; however, bone loss may not be detectible on a radiograph.
Box 6-4. Plaque-Induced Gingivitis on an Intact Periodontium

Figure 6-5. Plaque-Induced Gingivitis. Plaque-induced gingivitis on an intact periodontium in this patient has resulted in rolled gingival margins and enlarged papillae.

- Note how this plaque-induced gingivitis is affecting the interdental papilla and the marginal gingiva. This type of plaque-induced gingivitis would be classified as marginal gingivitis.
- The dental radiograph does not reveal any changes in either the alveolar bone height or the architecture and morphology of the alveolar bone.

Box 6-5. Plaque-Induced Gingivitis on a Reduced Periodontium in a Non-periodontitis Patient

Figure 6-6. Plaque-Induced Gingivitis on a Reduced Periodontium in a Non-periodontitis Patient. On the clinical photograph, note all the visual signs of gingival inflammation, such as bleeding, swelling, and redness. This is a 17-year-old male patient who just completed orthodontic therapy to straighten teeth that were previously malaligned (crowded). The dental radiograph does not reveal any changes in either the alveolar bone height or the architecture and morphology of the alveolar bone.

- Orthodontists use different strategies to move teeth to an ideal alignment and position. However, one undesired consequence is that changes in the periodontal tissues may occur as a result of orthodontically induced movement of the teeth. This is apparent in cases where orthodontic therapy displaces the teeth in a buccal direction causing resorption of the overlying thin, delicate outer cortical plate. In the case above, post-orthodontic movement of the lower right central incisor and both canines have led to recession of the gingival margin (permanent loss of the marginal gingiva).
- Since the recession of the gingival margin is not due to periodontitis, the treatment objectives are to (1) remove the etiologic factors responsible for gingival inflammation, (2) reinforce oral hygiene, (3) avoid further loss of periodontal tissues (i.e., recession of the gingival margin), and (4) minimize the risk of the gingivitis converting into periodontitis.
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MODIFYING FACTORS OF PLAQUE-INDUCED GINGIVITIS

Several modifying factors may play a role in exacerbating the host inflammatory response to the plaque biofilm. These modifying factors can be subdivided into systemic factors that alter the host response to plaque biofilm or oral factors that contribute to increased plaque retention.

1. Systemic Conditions as Modifying Factors

   A. Sex and Steroid Hormones. The maintenance of a healthy balance within the periodontium involves a complex relationship with the endocrine system. Evidence suggests that tissue responses in the periodontium are influenced by the levels of sex hormones present at one time or another throughout a person’s life cycle.18,19 The following conditions may modify plaque-induced gingivitis but are not considered diagnoses in and of themselves.

   1. Puberty

      a. The dramatic rise in steroid hormone levels during puberty has a temporary effect on the inflammatory status of the gingiva.18,19

      b. Studies demonstrate an increase in gingival inflammation around the time of puberty in both genders.20,21

   Box 6-6. Plaque-Induced Gingivitis on a Reduced Periodontium in a Successfully Treated Periodontitis Case

   Figure 6-7. Plaque-Induced Gingivitis on a Reduced Periodontium in a Successfully Treated Stable Periodontitis Patient. This is a 68-year-old female patient who was successfully treated in the past for periodontitis. The dental radiograph reveals loss of alveolar bone height.

   • By performing a comprehensive periodontal examination and comparing the present information with previous periodontal findings (such as past probing depths, CAL, bleeding upon probing scores, etc.), it is apparent that the periodontitis is not ongoing, but rather arrested (no additional loss of alveolar bone or connective tissue).

   • However, note the visual signs of gingival inflammation—such as thickened marginal gingiva and slight gingival bleeding from the lower left incisors.

   • In such a case, the treatment objectives are to (1) maintain the reduced periodontium in a healthy and stable state by removing the etiologic agents, (2) reinforce patient self-care and professional care to prevent the reactivation of periodontitis.
c. Puberty-associated gingivitis has many of the clinical features of plaque-induced gingivitis; however, it is distinguished by clinical signs of *gingival inflammation in the presence of relatively small amounts of plaque biofilm.* Thus, puberty-associated gingivitis is characterized by an exaggerated inflammatory response of the gingiva to a relatively small amount of plaque biofilm around the time of puberty. Figure 6-8 shows one example of puberty-associated gingivitis.

2. Menstrual Cycle
   a. Most clinical studies show there to be only modest observable inflammatory changes in the gingiva during ovulation. Although there may be a few women who are extremely sensitive to hormonal changes in the gingiva during the menstrual cycle, in most women there will be no clinically evident inflammatory changes to the gingiva.

3. Pregnancy
   a. During pregnancy, the levels of estrogen and progesterone continue to rise and reach their peak in the eighth month of gestation. High levels in both blood and saliva cause an exaggerated tissue response to plaque biofilm. Increased quantities of hormones also trigger gingival crevicular fluid flow, which may precipitate gingival inflammation.
   b. Gingival inflammation is significantly higher in the pregnant patient in response to even relatively small amounts of plaque biofilm and bleeding on probing or with toothbrushing is also increased. Figure 6-9 shows a case of gingivitis associated with pregnancy.
   c. Gingivitis associated with pregnancy can spontaneously resolve postpartum.
   d. In some cases, a gingival papilla can react so strongly to plaque biofilm that a large, localized overgrowth of gingival tissue called a *pregnancy-associated pyogenic granuloma* (pregnancy tumor), may form on the interdental gingiva or on the gingival margin. This condition is the result of an exaggerated tissue response to plaque biofilm or other irritants that usually occurs after the first trimester of pregnancy.
      1) The gingival mass is characterized by a mushroom-like tissue mass that most commonly occurs in the maxilla and interproximally (Fig. 6-10).
      2) A pregnancy-associated pyogenic granuloma is painless and noncancerous. However, if it grows to interfere with occlusion, painful ulceration of the pyogenic granuloma may occur.
      3) The tissue mass bleeds easily if disturbed and may appear to be covered with dark red pinpoint markings.
      4) The growth usually resolves after childbirth. Even though the growth spontaneously resolves after childbirth, the granuloma can be completely eliminated during the pregnancy stage by removing the plaque biofilm responsible for the enlargement.

4. Oral Contraceptives
   a. Early conceptive agents had high hormone concentrations that were associated with gingival inflammation.
   b. Current oral contraceptive concentrations are much lower than the original doses and current formulations of oral contraceptives do not induce clinical changes in the gingiva.
B. Hyperglycemia. **Hyperglycemia** is the presence of an abnormally high concentration of glucose in the circulating blood that occurs especially in individuals with diabetes mellitus.

1. Gingivitis is often seen in children with poorly controlled diabetes mellitus.
2. The level of glycemic control may be more important in determining the severity of the gingival inflammation than the amount of plaque biofilm present. In other words, the inflammatory response of the gingiva to plaque biofilm is exacerbated by the high blood glucose levels.

C. Leukemia. Leukemia is cancer of the body’s blood-forming tissues, including the bone marrow and the lymphatic system. Leukemia usually involves the white blood cells. In people with leukemia, the bone marrow produces abnormal white
blood cells, which do not function properly. Many types of leukemia exist. Some forms of leukemia are more common in children. Other forms of leukemia occur mostly in adults.

1. Gingivitis associated with leukemia is an exaggerated inflammatory response of the gingiva to plaque biofilm resulting in increased bleeding and tissue enlargement. Oral lesions may be the first clinical signs of leukemia; therefore, dental health care providers can be the first to suspect that a patient may have leukemia.

2. Gingival tissues appear swollen, spongy, shiny, and red to deep purple in appearance. Figure 6-11 shows an example of gingivitis associated with leukemia. Typically, leukemia-associated gingivitis begins in the papillae and spreads to the marginal and then, the attached gingiva.

3. Tissues are very friable (tear easily) and tend to bleed with slight provocation.

4. Although plaque biofilm can exacerbate the gingival response to leukemia, the presence of biofilm is NOT a prerequisite for gingivitis in patients with leukemia.

D. Smoking

1. Epidemiologic studies indicate that smoking is one of the major lifestyle risk factors for periodontal disease.

2. Plaque biofilm accumulation and disease progression are exacerbated in smokers. Gingival fibrosis—the formation of an abnormal amount of fibrous tissue—is often observed in smokers.

3. Smokers have fewer clinical signs and symptoms of gingival inflammation than nonsmokers. Therefore, clinicians should be aware that smoking can mask gingivitis.

E. Malnutrition

1. Even with our adequate food supply in North America, infants, institutionalized individuals, and alcoholics are all at risk for vitamin deficiencies. However, the precise relationship between nutrition and periodontal disease is not fully understood.

2. The one nutritional deficiency that has well-documented effects on the periodontium involves the depletion of plasma ascorbic acid (vitamin C).
   a. Vitamin C—a substance found in many fruits and vegetables—is essential for the formation of collagen and fibrous tissue for normal intercellular matrices in teeth, bone, cartilage, connective tissue, and skin, and for the structural integrity of capillary walls.
   b. A lack of vitamin C can lead to scurvy, or less severe conditions, such as delayed healing of wounds.

3. Gingivitis associated with declining ascorbic acid levels may be difficult to detect clinically, and when it is detected, it usually has characteristics similar to plaque-induced gingivitis. Figure 6-12 shows an example of gingivitis in a patient with scurvy.

2. Oral Factors That Enhance Plaque Biofilm Accumulation

A. Prominent Subgingival Restoration Margins

1. A 26-year longitudinal study confirms that dental restorations margins placed apical to the gingival margin are detrimental to gingival health.
   a. The margins of prominent subgingival restorations promote gingivitis by increasing the local accumulation of bacterial plaque biofilms. Therefore, subgingival restoration margins need to be carefully designed in order to minimize biofilm retention.
B. Hyposalivation

1. Hyposalivation—a decreased flow of saliva—may be caused by some health conditions/diseases such as Sjögren syndrome, anxiety, and poorly controlled diabetes. In addition, it is frequently observed as a side effect of medications, such as antihistamines, decongestants, antidepressant, and antihypertensive medications.

2. Hyposalivation may cause progressive dental caries, taste disorders, halitosis, and inflammation of the oral mucosa, tongue, and gingiva. Dryness of the mouth may make patient self-care more difficult and worsen gingival inflammation.

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**Figure 6-11.** Gingivitis in an Adult with Leukemia. Note the red, swollen appearance of the gingiva in this patient with leukemia. (Courtesy of Dr. Ralph Arnold.)

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**Figure 6-12.** Ascorbic Acid-Deficiency Gingivitis. A photograph of a patient with scurvy. Scurvy is the clinical state arising from dietary deficiency of vitamin C (ascorbic acid). Note the bright red, swollen, and ulcerated gingival tissue. (Courtesy of Mediscan Company.)

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**DRUG-INFLUENCED GINGIVAL ENLARGEMENTS**

1. **Drug-influenced gingival enlargements** are an increase in size of the gingiva associated with certain systemic medications, most commonly anticonvulsants, calcium channel blockers, and immunosuppressants.

   A. **Medications Associated with Gingival Enlargement**

   1. Anticonvulsants (e.g., Phenytoin, Celontin, Depakote). Anticonvulsants are used to manage epileptic seizures. In addition, some anticonvulsants are now used in the treatment of bipolar disorder.

   2. Immunosuppressants (e.g., cyclosporine). Immunosuppressant drugs suppress the natural immune responses. Immunosuppressants are given to transplant patients to prevent organ rejection or to patients with autoimmune diseases. The immunosuppressant stimulates fibroblast proliferation with excessive extracellular matrix accumulation in gingival tissues.

   3. Calcium Channel Blocking Agents (e.g., amlodipine, nifedipine, verapamil). Calcium channel blocking agents relax the blood vessels and increase the supply of blood and oxygen to the heart while reducing its workload. Some
of the calcium channel blocking agents are used to relieve and control angina pectoris (chest pain). Some are also used to treat high blood pressure (hypertension). These drugs affect gingival connective tissues by stimulating an increase of fibroblasts and increasing the production of connective tissue matrix.

B. Etiology
1. For drug-influenced gingival conditions, plaque biofilms in conjunction with the drug are necessary to produce gingival enlargements.
2. However, not all individuals who take these medications will develop enlargements of the gingival tissues.

2. Common Characteristics of Drug-Influenced Gingival Enlargements
   A. Onset of Tissue Enlargements
      1. The onset of tissue enlargement usually occurs within 3 months of medication use. The severity of overgrowth is directly affected by level of patient self-care; scrupulous patient self-care can reduce the severity of the overgrowth but may not eliminate it.
      2. There is a higher prevalence of drug-influenced gingival enlargements in younger age groups.
   B. Clinical Appearance
      1. Gingival tissues in anterior sextants are most commonly affected, however, tissue enlargements can occur in posterior sextants (Figs. 6-13 and 6-14).
      2. The pattern of tissue enlargement is irregular, usually first observed in the papillae, beginning as a painless area of enlargement on the papilla and then proceeding to the marginal gingiva.
      3. Gingival enlargement is characterized by an increased flow of crevicular fluid from the sulcus and bleeding upon probing with no attachment loss.

Figure 6-13. Phenytoin-Induced Gingival Enlargement. Massive-tissue overgrowth may be seen in phenytoin-induced gingival enlargement.

Figure 6-14. Cyclosporine-Induced Gingival Enlargement. Gingival changes seen in cyclosporine-induced gingival enlargement.
Section 3
Non–Plaque-Induced Gingival Diseases

A small percentage of gingival diseases—non–plaque-induced gingival diseases—are not caused by plaque biofilm and do not resolve after plaque biofilm removal. It should be emphasized, however, that the presence of plaque biofilm could increase the severity of the gingival inflammation in non–plaque-induced lesions.

Although non–plaque-induced gingival diseases are less common, these conditions are often painful and of major significance for patients. This section presents some examples of the small percentage of gingival disease in which plaque biofilm does not play a primary etiologic role. The 2017 classification scheme for non–plaque-induced gingival diseases and conditions is outlined in Table 6-3.

<table>
<thead>
<tr>
<th>TABLE 6-3</th>
<th>CLASSIFICATION OF NON–PLAQUE-INDUCED GINGIVAL DISEASES AND CONDITIONS</th>
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</thead>
<tbody>
<tr>
<td>1. Genetic/developmental disorders</td>
<td></td>
</tr>
<tr>
<td>1.1 Hereditary gingival fibromatosis (HGF)</td>
<td></td>
</tr>
<tr>
<td>2. Specific infections</td>
<td></td>
</tr>
<tr>
<td>2.1 Bacterial origin</td>
<td></td>
</tr>
<tr>
<td>• Necrotizing periodontal diseases (Treponema spp., Selenomonas spp., Fusobacterium spp., Prevotella intermedia, and others)</td>
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<tr>
<td>• Neisseria gonorrhoeae (gonorrhea)</td>
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<tr>
<td>• Treponema pallidum (syphilis)</td>
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<tr>
<td>• Mycobacterium tuberculosis (tuberculosis)</td>
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<tr>
<td>• Streptococcal gingivitis (strains of streptococcus)</td>
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<tr>
<td>2.2 Viral origin</td>
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<tr>
<td>• Coxsackie virus (hand-foot-and-mouth disease)</td>
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<tr>
<td>• Herpes simplex 1/2 (primary or recurrent)</td>
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<tr>
<td>• Varicella-zoster virus (chicken pox or shingles affecting V nerve)</td>
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<td>• Molluscum contagiosum virus</td>
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<tr>
<td>• Human papilloma virus (squamous cell papilloma, condyloma acuminatum, verruca vulgaris, and focal epithelial hyperplasia)</td>
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<td>2.3 Fungal</td>
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<tr>
<td>• Candidosis</td>
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<tr>
<td>• Other mycoses (e.g., histoplasmosis, aspergillosis)</td>
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<td>3. Inflammatory and immune conditions and lesions</td>
<td></td>
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<tr>
<td>3.1 Hypersensitivity reactions</td>
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<tr>
<td>• Contact allergy</td>
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<td>• Plasma cell gingivitis</td>
<td></td>
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<td>• Erythema multiforme</td>
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<tr>
<td>3.2 Autoimmune diseases of skin and mucous membranes</td>
<td></td>
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<tr>
<td>• Pemphigus vulgaris</td>
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<tr>
<td>• Pemphigoid</td>
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<td>• Lichen planus</td>
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<td>• Lupus erythematosus</td>
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<td>3.3 Granulomatous inflammatory conditions (orofacial granulomatosis)</td>
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<tr>
<td>• Crohn’s disease</td>
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<tr>
<td>• Sarcoidosis</td>
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(continued)
TABLE 6-3  CLASSIFICATION OF NON–PLAQUE-INDUCED GINGIVAL DISEASES AND CONDITIONS (Continued)

4. Reactive processes
   4.1 Epulides
      • Fibrous epulis
      • Calcifying fibroblastic granuloma
      • Pyogenic granuloma (vascular epulis)
      • Peripheral giant cell granuloma (or central)

5. Neoplasms
   5.1 Premalignant
      • Leukoplakia
      • Erythroplakia
   5.2 Malignant
      • Squamous cell carcinoma
      • Leukemia
      • Lymphoma

6. Endocrine, nutritional, and metabolic diseases
   6.1 Vitamin deficiencies
      • Vitamin C deficiency (scurvy)

7. Traumatic lesions
   7.1 Physical/mechanical insults
      • Frictional keratosis
      • Toothbrushing-induced gingival ulceration
      • Factitious injury (self-harm)
   7.2 Chemical (toxic) insults
      • Etching
      • Chlorhexidine
      • Acetylsalicylic acid
      • Cocaine
      • Hydrogen peroxide
      • Dentifrice detergents
      • Paraformaldehyde or calcium hydroxide
   7.3 Thermal insults
      • Burns of mucosa

8. Gingival pigmentation
   • Gingival pigmentation/melanoplakia
   • Smoker’s melanosis
   • Drug-induced pigmentation (antimalarials; minocycline)
   • Amalgam tattoo

DESCRIPTION OF SELECTED DISEASE DISORDERS

1. Genetic/Developmental Abnormalities
   A. Hereditary Gingival Fibromatosis. Hereditary gingival fibromatosis is a rare benign oral condition characterized by slow and progressive enlargement of both maxillary and mandibular attached gingiva. One example of a patient with hereditary gingival fibromatosis is pictured in Figure 6-15.\textsuperscript{42} It may develop as an isolated disorder but can feature along with a syndrome. Compared to drug-related gingival overgrowth, hereditary gingival fibromatosis is a rare disease.
2. Infections of Bacterial Origin
   A. Necrotizing Periodontal Disease
      1. Necrotizing gingivitis, necrotizing periodontitis, and necrotizing stomatitis are severe inflammatory periodontal diseases caused by bacterial infection in patients with specific underlying risk factors (poor oral self-care, smoking, stress, poor nutrition, compromised immune status). Necrotizing periodontal disease is the term encompassing necrotizing gingivitis, necrotizing periodontitis, and necrotizing stomatitis.
      2. Necrotizing gingivitis involves only gingival tissue and is characterized by no loss of periodontal attachment.\(^43\)
      3. The clinical appearance of necrotizing periodontal disease is noticeably different than that of any other periodontal disease. Necrotizing periodontal disease is characterized by ulcerated and necrotic papillae and gingival margins, giving the appearance that the papillae and gingival margins have been “punched-out” or “cratered” (Fig. 6-16).
      4. Detailed content on necrotizing periodontal disease is found in Chapter 8, Other Periodontal Conditions.
   B. Other Bacterial Infections. Non–plaque-associated bacterial infections of the gingiva are uncommon. Gingival diseases in this category are characterized by a bacterial infection of the gingiva by a specific bacterium that is not commonly found in a typical bacterial plaque biofilm.
      1. Gingival diseases of specific bacterial origin occur on rare occasions when a bacterial infection overwhems the host resistance. Examples include infections with \textit{Neisseria gonorrhoeae}, \textit{Treponema pallidum}, and streptococcal species.\(^44\)
      2. The gingival lesions manifest as painful ulcerations, chancres or mucous patches, or atypical gingival inflammation (Fig. 6-17).
DESCRIPTION OF SELECTED INFLAMMATORY AND IMMUNE CONDITIONS AND LESIONS

1. Hypersensitivity Reactions: Intraoral Allergic Reactions. Intraoral allergic reactions can be caused by ingredients in toothpastes, mouthwashes, or chewing gum. These reactions are usually the result of a flavor additive or preservatives in the product. Flavor additives known to cause gingival reactions are cinnamon and carvone.

A. Occurrence of Intraoral Allergic Reactions
   1. Allergic reactions occur most commonly in patients who have a history of allergic conditions such as hay fever, allergic skin rashes, or asthma.
   2. Allergic patients seem to be particularly sensitive to the flavoring agent. The flavoring agent in toothpastes and mouthwashes is usually the most allergenic component.

B. Clinical Manifestations. The clinical manifestations of allergy are a diffuse fiery red gingivitis sometimes with ulcerations (Fig. 6-18).

C. Recognition and Treatment of Allergic Reaction
   1. The dental clinician might suspect an intraoral allergic reaction in a patient with good self-care who previously has had healthy gingiva (especially if the patient has a history of allergies). The dental clinician should inquire if the patient is using a new toothpaste, mouthwash, or chewing gum.
   2. Advise the patient to change brands or flavors of gum, toothpaste, or mouthwash. Cessation of the allergen-containing product should result in a resolution of gingival inflammation.
   3. If necessary, the diagnosis of allergic response can be confirmed by a biopsy with a diagnosis of plasma cell gingivitis.
   4. When the manufacturer becomes aware of allergic reactions, the flavoring agent or additive causing the problem is usually altered. For this reason, the patient sometimes can switch back to the original product (after 6 to 12 months) and use it without problem.

2. Erythema Multiforme
   A. Disease Characteristics
      1. Erythema multiforme is an uncommon acute immune inflammatory disorder of the skin and/or oral mucosa. The characteristic hallmarks of this condition are large, symmetrical red blotches, resembling a target, that appear on the skin in a circular pattern.
2. On mucous membranes, it begins as blisters and progresses to ulcers (Fig. 6-19). Oral involvement occurs in as many as 25% to 60% of cases and is sometimes the only involved site.

3. The exact cause is unknown, though it may involve a hypersensitivity reaction.

3. Oral Lichen Planus
   A. Disease Characteristics
      1. **Lichen planus** (LIE-kun Play-nus) is a common inflammatory condition that can affect the skin, hair, nails, and mucous membranes. It is important to diagnose, treat, and follow patients through regular oral examinations.
      4. Symptoms usually can be managed, but people who have oral lichen planus need regular monitoring because they may be at risk of developing oral cancer in the affected areas. It is important to diagnose, treat, and follow patients through regular oral examinations.

B. Clinical Manifestations
   1. Six types of clinical manifestations of oral lichen planus have been described: papular, reticular, plaque type, erythematous, ulcerative, and bulbous lesions.
a. The reticular pattern is commonly found bilaterally on the buccal mucosa as lacy web-like, white threads that are slightly raised. These lines are sometimes referred to as Wickham's striae (Fig. 6-20).

b. The ulcerative pattern can affect any mucosal surface, including the buccal mucosa, tongue, and gingiva. Ulcerative lichen planus is painful and associated with intense erythema of the gingiva.

2. Since the clinical appearance of lichen planus may mimic other types of non-plaque-induced gingival lesions, a biopsy and an oral pathology consultation will be required to determine the appropriate diagnosis.

Figure 6-20. Lichen Planus of Buccal Mucosa. These delicate white lines arranged in a lacy web-like network are characteristic of the reticular pattern of lichen planus. (Used with permission from Langlais, RP, Color Atlas of Common Oral Diseases. Philadelphia, PA: Wolters Kluwer; 2016.)

Figure 6-21. Oral Lichen Planus. Oral lichen planus of the maxillary gingiva. The gingival tissues are erythematous, ulcerated, and painful. (Courtesy of Dr. Ralph Arnold, San Antonio, TX.)

Chapter Summary Statement

This chapter reviews the criteria for periodontal health and gingival diseases/conditions as developed by the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. Periodontal health is defined as the absence of any clinical signs of inflammation. Periodontal health can occur on an intact periodontium, a reduced periodontium due to non-periodontitis reasons, or a reduced periodontium due to periodontitis.

Plaque-induced gingivitis is the most common of the periodontal diseases. Clinically, plaque-induced gingivitis is characterized by gingiva that is red, swollen, bleeds easily, and is slightly tender. Gingival diseases are the mildest form of periodontal disease and patients with gingivitis can revert to a state of health. Like periodontal health, plaque-induced gingivitis can occur on an intact periodontium, reduced periodontium due to non-periodontitis reasons, or a reduced periodontium due to periodontitis. Plaque-induced gingivitis may also be modified by systemic factors, medications, or malnutrition.

Non-plaque-induced gingival diseases are a group of uncommon gingival lesions that are not caused by plaque biofilm. Non-plaque-induced gingivitis can result from such diverse causes as bacterial, viral, or fungal infection, inflammatory conditions, allergic reactions, or trauma.
Section 4
Focus on Patients

Clinical Patient Care

CASE 1
You are scheduled to perform nonsurgical periodontal instrumentation on a patient with a diagnosis of localized severe plaque-induced gingivitis. At the time of the appointment, the patient informs you that she is pregnant. With this information in hand, what would be the most appropriate periodontal diagnosis?

CASE 2
A patient who has been cared for by your dental team suddenly exhibits poor self-care with quite a bit of plaque biofilm accumulation. This is unusual for this patient. Discussions reveal that the patient is having difficulty with brushing and flossing due to soreness of the mouth. Examination reveals numerous small mucosal ulcers. Further discussions reveal that the patient has been experiencing this soreness since she began using tartar control toothpaste. How might your dental team manage this patient’s diminished effectiveness of self-care?

CASE 3
Your patient is a 12-year-old male, who despite good oral hygiene practices, presents with generalized marginal redness and bleeding upon probing. His demonstration of toothbrushing and flossing indicates high dexterity and an ability to remove plaque biofilm. In talking with his mother, she confirms that he practices daily oral hygiene. How would you explain the presence of gingival disease to this patient and what would you recommend to improve his gingival health?

CASE 4
Your patient reports a change in his medical history from last visit; he is now taking Depakote as a mood stabilizer. The drug reference manual states it is an anticonvulsant and may cause gingival enlargement. How will this new information alter your plan for dental hygiene care and patient education?
Ethical Dilemma

Lily E, a 17-year-old high school senior, who is a routine 6-month recall patient, is your first patient of the afternoon. She received her driver’s license 3 months ago and has driven herself to today’s appointment. You review her medical history, and she states there are “no changes and she has no chief complaint.”

As you are performing your intraoral examination, you notice that the tissue between the maxillary central incisors does not appear to be normal. You observe a mushroom-shaped gingival mass projecting from the gingival papilla. It appears red, and bleeds easily upon digital palpation. You ask Lily if it bothers her, but she denies any discomfort. She also states that she was not aware of any problem.

You are concerned that the lesion may be a pyogenic granuloma associated with pregnancy. You would like to discuss the possible implication of this lesion with Lily, but you are not sure how to proceed.

1. What is the best way for you to handle this ethical dilemma?
2. What is the best way to address/discuss Lily’s treatment plan with her?
3. Under the ethical principle of confidentiality, can you discuss this with your employer dentist, without violating Lily’s confidentiality?
4. Do you have the right to divulge your findings and concerns to her parents?
5. Can a 17-year-old consent to treatment, or must you receive parental consent?

References


