Clinical Cases in Periodontics

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Examination and Diagnosis

Clinical Cases in Periodontics, First Edition. Nadeem Karimbux.

Case 1

Examination and Documentation

CASE STORY

A 40-year-old African-American male (LD) in no apparent distress presented with a chief complaint of: "My dentist told me I have gum disease and I should see a periodontist." Figures 1–5 are the patient's intraoral photographs.

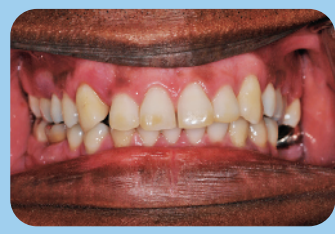

Figure 1: Frontal view.

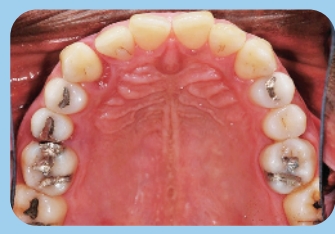

Figure 2: Maxillary view.

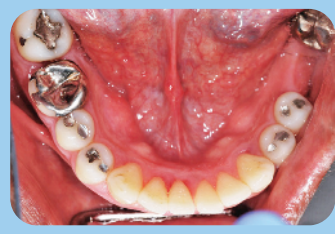

Figure 3: Mandibular view.

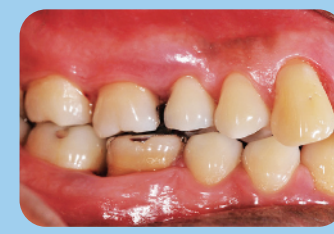

Figure 4: Right occlusal view.

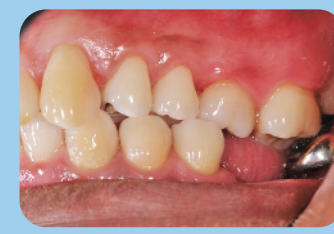

Figure 5: Left occlusal view.

LEARNING GOALS AND OBJECTIVES

- The patient's chief complaint
- Medical and dental history
- Soft tissue and gingival examination
- Periodontal charting
- Radiographic interpretation and diagnosis of periodontal condition

Past Dental History

This patient denied having bleeding gums during brushing or flossing, he had no loose teeth, and he was not in pain. The patient claimed to brush twice a day and flossed sporadically. His last dental visit was a year ago for a cleaning.

This particular patient presented with a blood pressure of 140/90, a pulse of 70 beats per minute, and a respiration rate of 14 breaths per minute. The patient denied having any significant health problems, had no known allergies, and denied taking any medications.

Soft Tissue and Gingival Examination

The patient had no pathologic masses or lesions upon extraoral and intraoral examination (Figures 1–5). He presented with generalized coral pink gingiva with normal pigmentation, scalloped gingival contour with knife-edged margins, pyramidal papillae with localized areas of blunted papilla, stippling, localized areas of recession, and firm consistency with localized edematous areas. There was mild plaque, with no significant supra/subgingival calculus. There was some normal pigmentation associated with the attached gingiva (Figures 1, 4, and 5).

Periodontal Charting

A thorough periodontal examination was completed. The periodontal chart (Figures 6 and 7) shows that the patient had generalized bleeding on probing, minimal plaque, pocket depths ranging from 2 to 15 mm with the more severe probing depths in the posterior teeth, class 1–2 furcations, class 1–3 mobility, and localized recession.

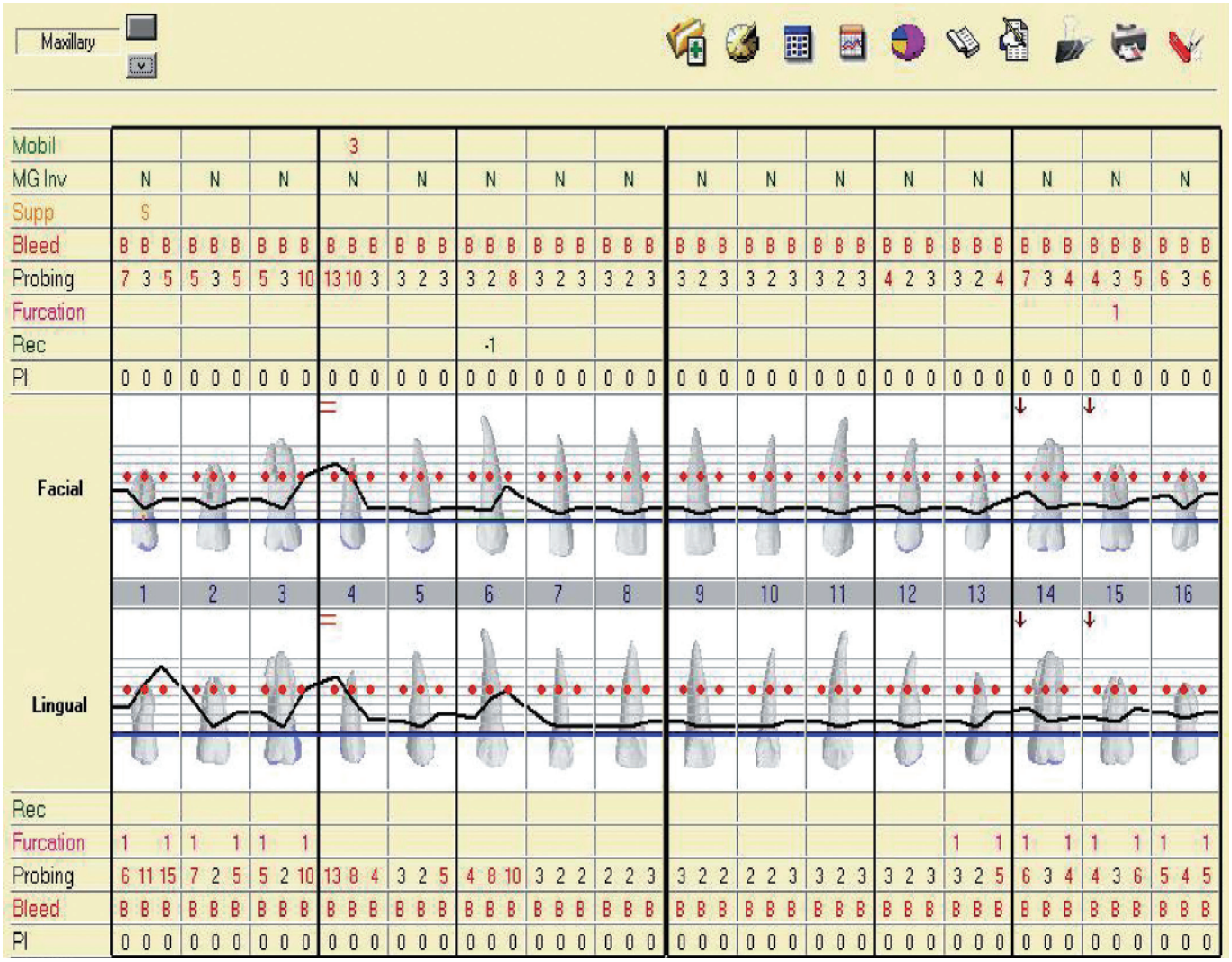

Figure 6: Maxillary periodontal charting.

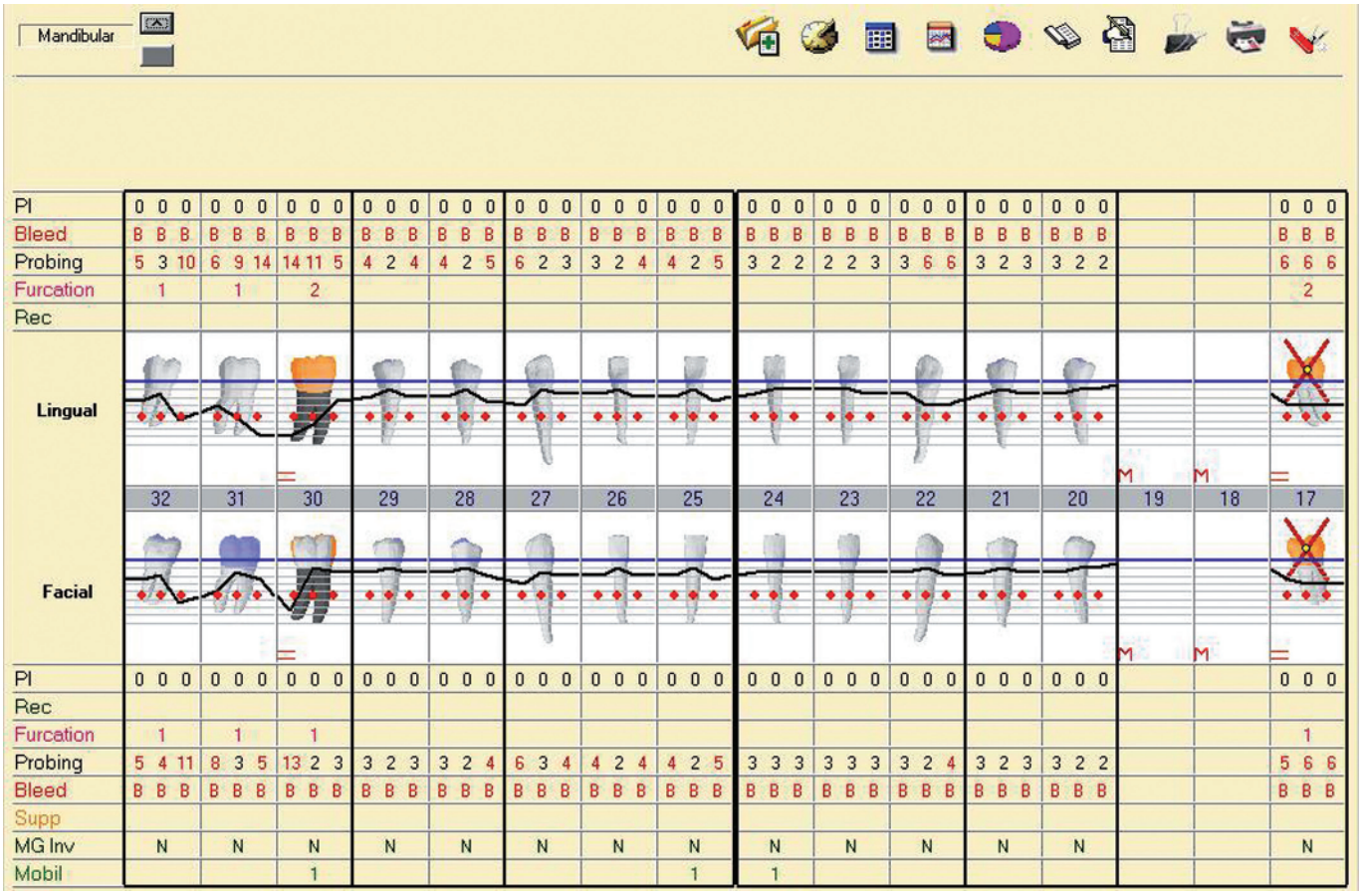

Figure 7: Mandibular periodontal charting.

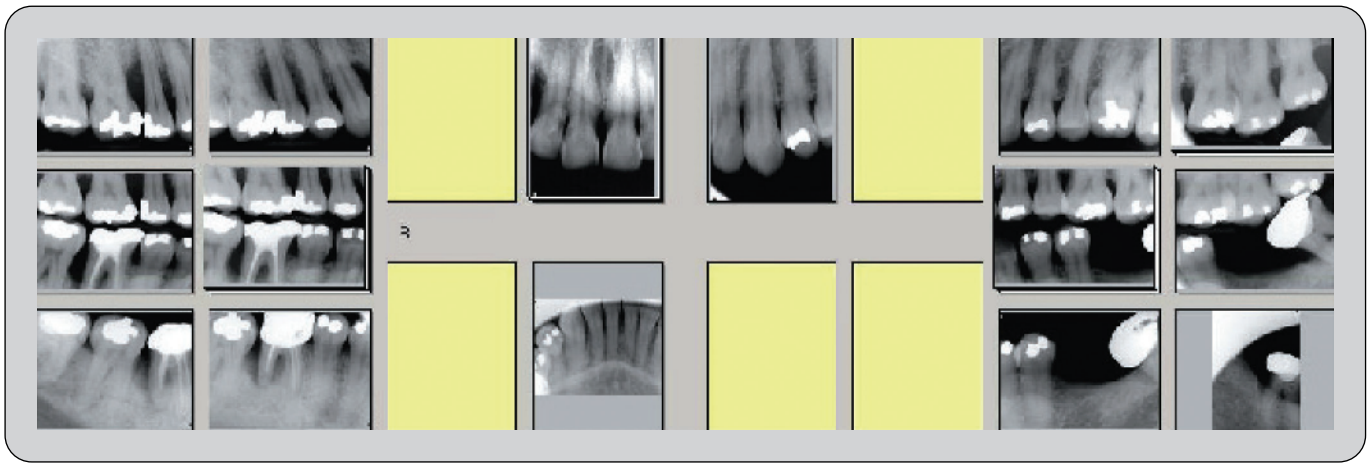

Figure 8: Full mouth series radiographs.

Radiographic Examination

From this full mouth radiograph (Figure 8), generalized bone loss is evident with severe bone loss surrounding #4, #6, #7, and #30. Bone loss in the furcation area is evident for #17 and #30.

Diagnosis

According to the American Academy of Periodontology (AAP), patient LD would be diagnosed with generalized severe chronic periodontitis, which corresponds to an ADA diagnosis of Case Type IV.

Self-Study Questions

A. What dental history questions are important to consider for a periodontal patient?

B. What medical history questions are important to consider for a periodontal patient?

1. From a medical perspective for medical management of the patient
2. From a perspective of conditions that might affect the gingiva/periodontium

C. What constitutes a thorough periodontal examination?

D. How often and what type of radiographs should be exposed for a periodontal examination?

E. How does one come to a diagnosis for gingival and periodontal diseases?

Answers located at the end of the chapter.

Diagnosis (ADA)

Case Type I. Gingivitis

- No attachment loss
- Bleeding may or may not be present
- Pseudopockets may be present
- Only the gingival tissues have been affected by the inflammatory process
- No radiographic evidence of bone loss
- The crestal lamina dura is present
- The alveolar bone level is within 1–2 mm of the CEJ area

Case Type II. Early Periodontitis

- Bleeding on probing may be present in the active phase
- Pocket depths or attachment loss of 3–4 mm
- Localized areas of recession
- Possible class I furcation invasion areas
- Horizontal type of bone loss is most common
- Slight loss of the interdental septum
- Alveolar bone level is 3–4 mm from the CEJ area

Case Type III. Moderate Periodontitis

- Pocket depths or attachment loss of 4–6 mm
- Bleeding on probing
- Grade I and/or grade II furcation invasion areas
- Tooth mobility of class I
- Horizontal or vertical bone loss may be present
- Alveolar bone level is 4–6 mm from the CEJ area
- Radiographic furcations of grade I and/or grade II
- Crown to root ratio is 1:1 (loss of a third of supporting alveolar bone)

Case Type IV. Advanced Periodontitis

- Bleeding on probing
- Pocket depths or attachment loss >6 mm
- Grade II, grade III furcation invasion areas
- Mobility of class II or class III
- Horizontal and vertical bone loss
- Alveolar bone level is ≥6 mm from the CEJ area
- Radiographic furcations
- Crown to root ratio is ≥2:1 (loss of more than a third of the supporting alveolar bone)

Source: American Academy of Periodontology: Current Procedural Terminology for Periodontics and Insurance Reporting Manual, 7th ed.

References

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3. Emrich LJ, Shlossman M, et al. Periodontal disease in non-insulin dependent diabetes mellitus. J Periodontol 1991;62:123–131.
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5. Academy Reports informational paper: Drug associated gingival enlargement. J Periodontol 2004;75:1424–1431.

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7. Association Report: The use of dental radiographs. Update and Recommendations. JADA 2006;137:1304–1312.
8. American Academy of Periodontology. Current procedural terminology for periodontics and insurance reporting manual. 7th ed. Chicago, IL: AAP, 1995.
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10. Armitage G. Periodontal diagnoses and classification of periodontal disease. Periodontology 2000 2004;34:9–21.

TAKE-HOME POINTS

A. The patient should be asked if he or she has a history of bleeding gums, loose teeth, or if there is a history of periodontal disease in the family. Bleeding of the gums spontaneously or on brushing or flossing can either indicate a systemic problem or a gingival/periodontal problem. Most patients with "bleeding gums" most likely have plaque-associated gingivitis or a form of aggressive/chronic periodontitis. If the bleeding does not resolve after initial phase therapy and home care instructions, the dentist/periodontist should refer the patient for a medical workup. Examples of diseases that may involve bleeding gums include leukemia or hemophilia. Loose teeth can either indicate an advanced stage of periodontitis or trauma from occlusion. A family history of periodontal disease should be considered in cases of localized aggressive periodontitis. Patients should also be asked if they have had prior periodontal therapy and when the last visit to a dentist took place. This history can indicate a past history of treatment for periodontal problems, or it might indicate the patient has had irregular care in the past. One must also ask patients how regularly they brush/floss and if they use any other aids. This can indicate dental neglect or good oral hygiene practices.

For this portion of the examination, it is also important to ask patients if they have a history of smoking or are currently smoking, if they have diabetes or a family history of diabetes, and if they are taking any medications. Smoking and uncontrolled diabetes are both factors that put patients at higher risk for getting periodontitis [1–4]. Because smoking and diabetes are known to affect periodontal health, it is vital that we question patients about these issues. If the patients smoke, they should be given information about how smoking affects oral health and advised to consider smoking cessation. If a patient has diabetes, he or she should be under the care of a physician who regularly monitors the condition.

B. (1) As discussed, a history of smoking and uncontrolled diabetes can reveal risk factors for periodontal disease. Any major systemic conditions or illnesses that a patient has need to be incorporated in the treatment plan. For example, complex medical issues or conditions may need to be treated in a hospital setting. If a patient is hypertensive, the AHA guidelines should be followed before any treatment. If a patient has bleeding disorders or is taking blood thinners (Coumadin, Plavix), a consultation with a physician before any surgeries should be completed. **(2)** Certain drugs such as nifedipine, cyclosporine, and phenytoin are known to cause gingival hyperplasia [5]. Oral contraceptives can affect the gingiva as well. If patients are taking any medications such as these, they should be informed that their prescription medication may cause changes in their gingiva.

C. A complete periodontal examination involves filling out a periodontal chart. This chart can include information on (1) probing depths (PDs), (2) bleeding on probing (BOP), (3) distance from the gingival margin to the cementoenamel junction (GM-CEJ), (4) clinical attachment loss (CAL), (5) recession, (6) furcation, (7) mobility, (8) ridge defects, and (9) plaque and calculus [6].

1. Probing Depths

To obtain probing depths, the periodontal probe is placed in the sulcus in six different locations

around each tooth. These locations are the mesiobuccal, straight buccal, distobuccal, distolingual, straight lingual, and mesiolingual. The probe is gently placed to the depth of the gingival sulcus, and the distance from the base of the pocket to the gingival margin is measured in millimeters using the markings on the instrument.

2. Bleeding on Probing

Following periodontal probing, bleeding on probing should be noted. Some sites will experience bleeding immediately or a few moments afterward. Bleeding on probing or the lack of bleeding should be recorded on the periodontal chart.

3. Distance from Gingival Margin to Cementoenamel Junction

With the use of the periodontal probe, the distance from the CEJ to the gingival margin must be measured in the same locations as the periodontal probing depths. If there is recession, this distance should be measured and recorded as a positive number. If the gingival margin is above the CEJ, which occurs in gingival health or when there is gingival hyperplasia, the probe should be used to feel the CEJ subgingivally, and the measurement should be recorded as a negative number.

4. Clinical Attachment Loss

The CAL calculated using the numbers obtained after periodontal probing and after determining the distance from the gingival margin to the CEJ. The probing depth number is added to the GM-CEJ measurement. For example, if the probing depth is 2 mm and there is 2 mm of recession (GM-CEJ = +2), the CAL will be 4 mm. If the probing depth is 3 mm, and the gingival margin is 1 mm above the CEJ (GM-CEJ = −1), the CAL will be 2 mm. The values calculated for CAL are necessary for diagnosing periodontal disease.

5. Recession

Recession occurs when the gingival margin is below the CEJ. The Miller classification for recession defects may be used to categorize the recession, as follows:

Miller Class 1: Recession that does not extend to the mucogingival junction with no periodontal bone loss in the interdental areas.
Miller Class 2: Recession that extends to or beyond the mucogingival junction, with no interdental bone loss.
Miller Class 3: Recession that extends to or beyond the mucogingival junction, with some periodontal attachment loss in the interdental area or malpositioning of teeth.
Miller Class 4: Recession that extends to or beyond the mucogingival junction, with severe bone and/or soft tissue loss in the interdental area and/or severe malpositioning of teeth.

6. Furcation

Furcation classifications for maxillary molars, maxillary first premolars, and mandibular molars should be recorded. According to the Glickman classification for furcations, it should be recorded as grade 1, 2, 3, or 4.
Grade 1: There is incipient furcation involvement involving mostly soft tissue. Early bone loss may have occurred but is not visible radiographically.
Grade 2: There is a horizontal component to the bone loss in the furcation area resulting in an area that can be probed. Some bone does remain attached to the tooth.
Grade 3: There is no attached bone in the furcation area, resulting in a through-and-through tunnel. Soft tissue may still occlude the furcation.
Grade 4: There is a visible through-and-through tunnel that is easily probed.

7. Mobility

The mobility of each tooth should be tested using the blunt ends of two instruments. Depending on the mobility, the tooth should be classified using the Miller classification system as having class 1, 2, or 3 mobility.
Miller Class 1: Tooth can be moved <1 mm in the buccolingual or mesiodistal direction.
Miller Class 2: Tooth can be moved ≤1 mm in the buccolingual or mesiodistal direction but not in the apico-coronal direction.
Miller Class 3: Tooth can be moved ≤1 mm in the buccolingual or mesiodistal direction, and

movement in the apico-coronal direction is present as well.

8. Ridge Defects

In edentulous areas, the remaining ridge may be classified as a Siebert class 1, 2, or 3 ridge defect if there is bone loss in the area.

Seibert Class 1: Loss of bone in the buccolingual direction with normal adequate bone in the apico-coronal direction.

Seibert Class 2: Loss of bone in the apico-coronal direction with adequate bone in the buccolingual direction.

Seibert Class 3: Loss of bone in both the buccolingual and apico-coronal direction.

9. Plaque and Calculus

The presence of plaque and calculus should also be recorded on the periodontal chart as absent, mild, moderate, or abundant.

D. To obtain an accurate picture of a patient's periodontal health, radiographs should be taken and analyzed as a part of the periodontal examination. Typically a full mouth series of radiographs should be taken every 5 years and a set of bite-wing radiographs should be taken every 2 years [7]. Bite-wing radiographs provide a way to assess the crestal bone and observe bone loss in the posterior. If there is severe bone loss that cannot be seen in a horizontal bite wing, vertical bite-wing radiographs should be taken. Periapical radiographs are useful for viewing the bone level as well, but especially for determining the crown-to-root ratio, which helps in assigning a tooth's prognosis. Furcation involvement can also be seen in radiographs.

E. Diagnosis (American Academy of Periodontology [8]

If ≤30% of the sites are affected, the disease is localized.

If >30% of sites are affected, the disease is generalized

According to the AAP, patients are diagnosed based on their clinical attachment level.

If CAL = 0 mm, the patient is diagnosed with gingivitis.

If CAL = 1–2 mm, the patient is diagnosed with slight/mild periodontitis.

If CAL = 3–4 mm, the patient is diagnosed with moderate periodontitis.

If CAL = ≥5, the patient is diagnosed with severe periodontitis.

If the clinical attachment loss has occurred gradually for years, the disease is chronic.

If the clinical attachment loss has occurred rapidly, the disease is aggressive.

Figure 9–14 further describe the classification of periodontal diseases and conditions [9,10].

Table 1: Classification of periodontal diseases and conditions

I. Gingival Diseases
- A. Dental plaque-induced gingival diseases
  - 1. Gingivitis associated with dental plaque only
    - a. without other local contributing factors
    - b. with local contributing factors
  - 2. Gingival diseases modified by systemic factors
    - a. associated with the endocrine system
      - 1) puberty-associated gingivitis
      - 2) menstrual cycle-associated gingivitis
      - 3) pregnancy associated
        - a) gingivitis
        - b) pyogenic granuloma
      - 4) diabetes mellitus-associated gingivitis
    - b. associated with blood-dyscrasias
      - 1) leukemia-associated gingivitis
      - 2) other
  - 3. Gingival diseases modified by medications
    - a. drug-influenced gingival diseases
      - 1) drug-influenced gingival enlargements
      - 2) drug-influenced gingivitis
        - a) oral contraceptive-associated gingivitis
        - b) other
  - 4. Gingival diseases modified by malnutrition
    - a. ascorbic acid-deficiency gingivitis
    - b. other
- B. Non-plaque-induced gingival lesions
  - 1. Gingival lesions of specific bacterial origin
    - a. *Neisseria gonorrhea*-associated lesions
    - b. *Treponema pallidum*-associated lesions
    - c. streptococcal species-associated lesions
    - d. other
  - 2. Gingival diseases of viral origin
    - a. herpesvirus infections
      - 1) primary herpetic gingivostomatitis
      - 2) recurrent oral herpes
      - 3) varicella-zoster infections
    - b. other
  - 3. Gingival diseases of fungal origin
    - a. *Candida*-species infection
      - 1) generalized gingival candidosis
    - b. linear gingival erythema
    - c. histoplasmosis
    - d. other
  - 4. Gingival lesions of genetic origin
    - a. hereditary gingival fibromatosis
    - b. other
  - 5. Gingival manifestations of systemic conditions
    - a. mucocutaneous disorders
      - 1) lichen planus
      - 2) pemphigoid
      - 3) pemphigus vulgaris
      - 4) erythema multiforme
      - 5) lupus erythematosus
      - 6) drug-induced
      - 7) other
    - b. allergic reactions
      - 1) dental restorative materials
        - a) mercury
        - b) nickel
        - c) acrylic
        - d) other
      - 2) reactions attributable to
        - a) toothpastes/dentifrices
        - b) mouthrinses/mouthwashes
        - c) chewing gum additives
        - d) foods and additives
      - 3) other
  - 6. Traumatic lesions (factitious, iatrogenic, accidental)
    - a. chemical injury
    - b. physical injury
    - c. thermal injury
  - 7. Foreign body reactions
  - 8. Not otherwise specified (NOS)

II. Chronic Periodontitis
- A. Localized
- B. Generalized

III. Aggressive Periodontitis
- A. Localized
- B. Generalized

IV. Periodontitis as a Manifestation of Systemic Diseases
- A. Associated with hematological disorders
  - 1. Acquired neutropenia
  - 2. Leukemias
  - 3. Other
- B. Associated with genetic disorders
  - 1. Familial and cyclic neutropenia
  - 2. Down Syndrome
  - 3. Leukocyte adhesion deficiency syndromes
  - 4. Papillon-Lefèvre syndrome
  - 5. Chediak-Higashi syndrome
  - 6. Histiocytosis syndromes
  - 7. Glycogen storage disease
  - 8. Infantile genetic agranulocytosis
  - 9. Cohen syndrome
  - 10. Ehlers-Danlos syndrome (Types IV and VIII)
  - 11. Hypophosphatasia
  - 12. Other
- C. Not otherwise specified (NOS)

V. Necrotizing Periodontal Diseases
- A. Necrotizing ulcerative gingivitis (NUG)
- B. Necrotizing ulcerative periodontitis (NUP)

VI. Abscesses of the Periodontium
- A. Gingival abscess
- B. Periodontal abscess
- C. Pericoronal abscess

VII. Periodontitis Associated With Endodontic Lesions
- A. Combined periodontic-endodontic lesions

(*Continued*)

Table 1: (*Continued*)

VIII. Developmental or Acquired Deformities and Conditions
- A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/ periodontitis
  1. Tooth anatomic factors
  2. Dental restorations/appliances
  3. Root fractures
  4. Cervical root resorption and cemental tears
- B. Mucogingival deformities and conditions around teeth
  1. Gingival/soft tissue recession
     a. facial or lingual surfaces
     b. interproximal (papillary)
  2. Lack of keratinized gingiva
  3. Decreased vestibular depth
  4. Aberrant frenum/muscle position
  5. Gingival excess
     a. pseudopocket
     b. inconsistent gingival margin
     c. excessive gingival display
     d. gingival enlargement
  6. Abnormal color
- C. Mucogingival deformities and conditions on edentulous ridges
  1. Vertical and/or horizontal ridge deficiency
  2. Lack of gingiva/keratinized tissue
  3. Gingival/soft tissue enlargement
  4. Aberrant frenum/muscle position
  5. Decreased vestibular depth
  6. Abnormal color
- D. Occlusal trauma
  1. Primary occlusal trauma
  2. Secondary occlusal trauma

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Table 2: Main clinical features and characteristics of chronic periodontics (1999 Classification)

* Most prevalent in adults, but can occur in children and adolescents
* Amount of destruction is consistent with the presence of local factors
* Subgingival calculus is a frequent finding
* Associated with a variable microbial pattern
* Slow to moderate rate of progression, but may have periods of rapid progression
* Can be associated with local predisposing factors (e.g., tooth–related or iatrogenic factors)
* May be modified by and/or associated with systemic diseases (e.g., diabetes mellitus)
* Can be modified by factors other than systemic disease such as cigarette smoking and emotional stress

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Table 3: Features of aggressive periodontitis that are common to both the localized and generalized forms of the disease (1999 Classification)

Primary features
- Except for the presence of periodontitis, patients are otherwise clinically healthy
- Rapid attachment loss and bone destruction
- Familial aggregation

Secondary features (often present)
- Amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction
- Elevated proportions of *Actinobacillus actinomycetemcomitans* and, in some populations, *Porphyromonas gingivalis* may be elevated
- Phagocyte abnormalities
- Hyperresponsive macrophage phenotype, including elevated levels of prostaglandin E2 (PGE2) and interleukin-1β (IL-1β)
- Progression of attachment loss and bone loss may be self-arresting

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Table 4: Specific features of localized and generalized aggressive periodontitis (1999 Classification)

Localized aggressive periodontitis
- Circumpubertal onset
- Robust serum antibody to infecting agents
- Localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors

Generalized aggressive periodontitis
- Usually affecting persons under 30 years of age, but patients may be older
- Poor serum antibody response to infecting agents
- Pronounced episodic nature of the destruction of attachment and alveolar bone
- Generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors.

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Table 5: Comparison of the main clinical characteristics of chronic periodontitis, localized aggressive periodontitis, and generalized aggressive periodontitis.

Chronic periodontitis
- Most prevalent in adults, but can occur in children
- Slow to moderate rates of progression
- Amount of microbial deposits consistent with severity of destruction
- Variable distribution of periodontal destruction; no discernible pattern
- No marked familial aggregation
- Frequent presence of subgingival calculus

Localized aggressive periodontitis
- Usually occurs in adolescents (circumpubertal onset)
- Rapid rate of progression
- Amount of microbial deposits not consistent with severity of destruction
- Periodontal destruction localized to permanent first molars and incisors
- Marked familial aggregation
- Subgingival calculus usually absent

Generalized aggressive periodontitis
- Usually affects people under 30 years of age, but patients may be older
- Rapid rate of progression (pronounced episodic periods of progression).
- Amount of microbial deposits sometimes consistent with severity of destruction
- Periodontal destruction affects many teeth in addition to permanent molars and incisors
- Marked familial aggregation
- Subgingival calculus may or may not be present

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Case 2

Plaque-Induced Gingivitis

CASE STORY

A 27-year-old Caucasian male presented with the chief complaint of: "My gums bleed." The patient noticed blood in the gingiva whenever he brushed (A). He also noted that his gums bled when he flossed (A). There had never been any swelling or pain associated with his gums, and the patient had never had an episode like this before. The patient claimed to brush his teeth once daily, and he flossed two to three times a week (B).

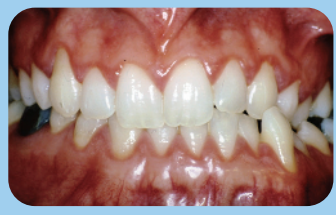


Figure 1: Preoperative presentation.

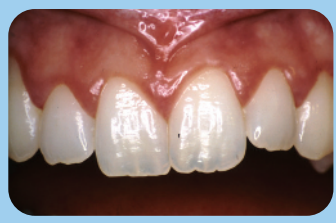


Figure 2: Preoperative maxillary anteriors.

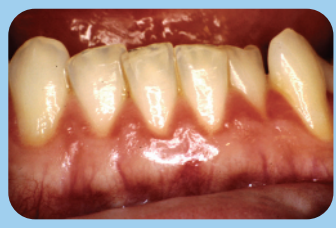


Figure 3: Preoperative mandibular anteriors.

LEARNING GOALS AND OBJECTIVES

- To be able to diagnose gingivitis
- To identify the possible etiology for the same condition and to address them
- To understand the importance of oral hygiene in preventing gingivitis

Medical History

There were no significant medical problems. The patient had no known allergies or medical illnesses. On questioning the patient stated he was taking no medications and he had no allergies.

Review of Systems

- Vital signs
  - Blood pressure: 120/65 mm Hg
  - Pulse rate: 72 beats/minute (regular)
  - Respiration: 15 breaths/minute

Social History

The patient did not drink alcohol. He did smoke (started at age 23 and currently smoked half a pack of cigarettes daily).

Extraoral Examination

No significant findings. The patient had no masses or swelling, and the temporomandibular joint was within normal limits.

Intraoral Examination

- The soft tissues of the mouth (except gingiva) including the tongue appeared normal.
- A gingival examination revealed a mild marginal erythema, with rolled margins and swollen papillae (Figures 1–3).

- A hard tissue and soft tissue examination were completed (Figure 4) (F).

Occlusion

There were no occlusal discrepancies or interferences.

Radiographic Examination

A full mouth set of radiographs was ordered (G). (See Figure 5 for the patient's bite-wing radiographs.)

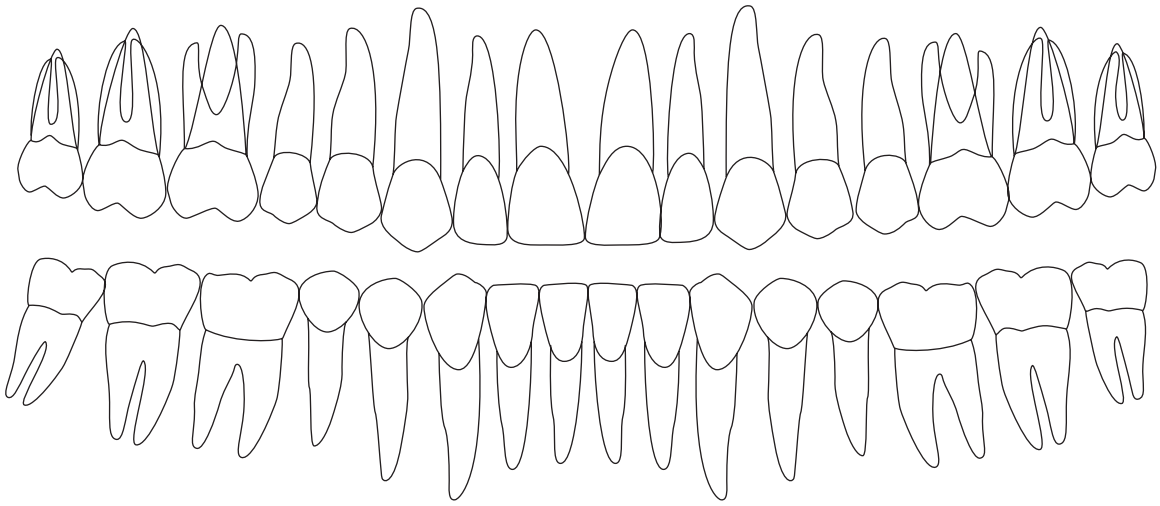
Diagnosis

After reviewing the history and both the clinical and radiographic examinations, a differential diagnosis was generated (H).

Treatment Plan

The treatment plan of the periodontal problems for this patient includes an initial phase of scaling with polishing and a 6-week reevaluation.

Buccal	323	323	333	333	333	323	212	212	212	222	323	213	323	323	323	323
Palatal	333	323	333	323	323	213	212	212	212	212	223	212	313	323	333	333



Buccal	323	323	333	322	333	323	222	212	222	212	323	213	323	323	323	323
Lingual	323	323	333	322	323	333	212	222	213	213	333	212	223	323	333	333

Figure 4: Probing pocket depth measurements.

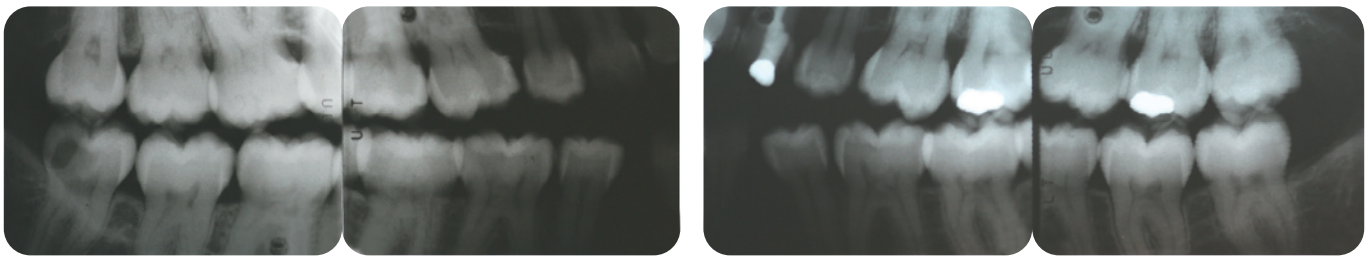


Figure 5: Bite-wing radiographs depicting the interproximal bone levels.

Treatment

The patient received a scaling and polishing. At the 6-week reevaluation, the clinical signs and symptoms had not improved, even though the patient claimed to be practicing excellent oral hygiene as per your instructions (I).

Discussion

Most patients who present with these symptoms (bleeding gums) have a plaque-induced gingivitis. A thorough history and periodontal examination must be completed to arrive at a diagnosis. Other characteristic features associated with plaque-induced gingivitis include the presence of plaque at the gingival margin, sulcular temperature change, increased gingival exudate, bleeding on probing. With good plaque control, the condition should resolve [1]. If there is a medical concern, it is typically identified by obtaining a thorough medical history. Conditions such as diabetes and leukemia have a profound effect on gingival health, and therefore the patient must be evaluated accordingly. In women, hormonal changes such as those that occur during the onset of puberty, pregnancy, or menstruation have a transient effect on the inflammatory status of these patients [2], which when combined with poor plaque control will lead to severe gingivitis [3,4]. After a diagnosis is reached, the treatment plan will include oral hygiene instructions, an initial phase of treatment (scaling or scaling and root planing) with a 4- to 6-week reevaluation. If the symptoms persist at this visit, the patient should be referred to a physician to rule out any systemic conditions that might cause bleeding.

Self-Study Questions

A. List the systemic/medical reasons the patient might present with bleeding gums when he flosses. What questions in a dental history might help you start to form a differential diagnosis?

B. What are the "ideal" brushing/flossing habits and techniques for a patient?

C. What effects can smoking have on the periodontium? On the oral cavity?

D. How would you perform an oral cancer examination?

E. What are the components of a periodontal examination?

F. What information should be recorded on a periodontal or soft tissue charting?

G. What kind of radiographs should be ordered for a periodontal examination?

H. What are the components that make one diagnose a case as gingivitis versus periodontitis?

I. What should a practitioner do in the case where gingival/periodontal symptoms have not resolved despite the prescribed dental care?

Answers located at the end of the chapter.

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TAKE-HOME POINTS

A

a. Bleeding disorders
   Idiopathic thrombocytopenic purpura
b. Medications
   Use of blood thinners
   Use of oral contraceptives
c. Systemic conditions
   Hormonal changes during pregnancy and puberty
   Gingivitis associated with diabetes mellitus
   Leukemia
   Vitamins C and/or K deficiency
d. Others
   Ill-fitting dental appliances or from its components (e.g., clasps)

Questions to help develop a differential diagnosis include the following:

- How often do you brush or floss?
- Do you bruise easily?
- When you wake up do you notice any blood in your mouth?
- When you cut yourself, do you tend to clot within a normal amount of time?
- What medicines are you taking currently?
- Are you pregnant (for female patients)?
- Are you a mouth breather?/Do you have difficulty breathing through your nose?

B. A patient should ideally brush twice daily and floss once daily. Evidence indicates that the use of rotary brushes is better than manual brushes for interproximal plaque removal and stain removal [5,6]. A toothbrush with soft bristles is strongly recommended. The bristles should be positioned at a 45-degree angle to the junction of the tooth and marginal gingiva, and then the brushing should be initiated using short circular gentle motions (Bass method of brushing). The same technique should be repeated for the rest of the mouth. If a patient has gingival recession, coronal sweeping motion of bristles from the gingiva to the teeth is recommended to prevent the progression of recession (modified Stillman's technique).

C. Smoking has been identified as a risk factor for periodontitis [7]. The number of cigarettes an individual smokes per day and the number of years an individual has been smoking are two important parameters strongly associated with the degree of attachment loss [8]. It is well established that smoking affects the host immune response, causes local tissue ischemia, and also alters the bacterial profile, shifting the plaque ecology and increasing the periodontal pathogens in the host [9]. This risk factor is "behavioral" and can be modified.

Smoking causes an increased risk for oral and throat cancers [10]. Oral cancer is the sixth most common cancer in males and the twelfth most common cancer in women in the United States [11].

D. A thorough extraoral examination should be conducted. Visualization and palpation of the soft tissues of the head and neck should be completed including palpation of the muscles and lymph nodes.

The intraoral examination should consist of visualization and palpation of the tongue. The tongue represents the most common site (50%) for oral cancer and ventral and lateral surfaces (20%), in particular, have a higher predilection for cancer than the dorsal surface of the tongue (4%) [11]. The floor of the mouth is the second most common site for oral cancer, and therefore careful examination of this area of the mouth should be a part of cancer screening. Other areas that should be examined specifically for oral cancer include the soft palate, gingiva, and buccal and labial mucosa [11].

E. A periodontal examination includes looking at and describing the gingival color, contour, consistency, texture, presence or absence of exudates from sulcus, and bleeding on probing. Six probing depths (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual) per tooth should be recorded. Areas of recession, mobility, and furcation involvement are also recorded and graded according to the established classifications for each of these conditions. Radiographs and study models are also important because they offer valuable information that is not obtained from the clinical oral examination.

F. The following are essential components of a periodontal chart:

- Name of the patient and the date of recording
- Missing teeth should be recorded
- Probing pocket depth: measured on six surfaces of each tooth in the mouth using a periodontal probe
- Degree of recession: measured using periodontal probe
- Mobility: measured using two flat ends of dental instruments such as dental mirror and/or periodontal probe and pushing the teeth with one instrument against the second instrument.
- Fremitus: measured by placing the inner pad of the fingers on the gingiva of the teeth in question and asking the patient to tap teeth three or four times. In traumatic occlusion, fremitus is usually felt by the examiner's fingers, which is then recorded.
- Degree of furcation involvement: examined using Naber's probe
- Mucogingival complex: the width of the mucogingival complex should be measured from the gingival margin to the apical-most part of the attached gingiva in every tooth, using a periodontal probe, and recorded.

G. Radiographs form an essential component of a periodontal examination. Apart from giving information about the periodontium of the tooth in question, other valuable information such as root length, root form, periapical lesions, and root proximity can be ascertained. The American Dental Association recommends a full mouth set of radiographs should be taken for a full diagnosis (typically every 5 years). A set of four bite wings should be exposed every 2 years. The diagnosis of periodontal disease can be made using periapical radiographs and bite-wing radiographs. Bite-wing radiographs are the most diagnostic for reading bone height because the head of the x-ray tube is perpendicular to the film. Vertical bite wings are recommended for areas with extensive bone loss. In general, paralleling technique is recommended over bisecting angle technique because it reduces the errors associated with film angulations.

H. Clinical attachment loss (CAL) (distance from the cementoenamel junction to the base of a periodontal pocket) and bone loss as seen on a radiograph are the gold standards used to help distinguish a patient with periodontitis versus gingivitis. Patients with gingivitis do not exhibit CAL and bone loss (radiographically), whereas if the diseases progresses to periodontitis, CAL and bone loss are characteristically observed.

I. A referral to physician should be made to rule out any systemic conditions.

Case 3

Non–Plaque-Induced Gingivitis

CASE STORY

A 41-year-old Latin American female presented with a chief complaint of: "My gums and teeth are sensitive." She had been referred by her general dentist for periodontal treatment. She reported a 5-year history of gingival sensitivity and progressive gingival recession. She experienced lingering pain after drinking hot and cold liquids and also noted sensitivity when brushing.

LEARNING GOALS AND OBJECTIVES

- To be able to distinguish desquamative gingivitis from plaque-induced gingivitis
- To formulate a differential diagnosis for common causes of desquamative gingivitis
- To be able to arrive at a definitive diagnosis and properly manage a patient with desquamative gingivitis

Medical History

There were no significant findings found in the patient's medical history. The patient saw her physician for an annual physical. She did not currently take any medications and she had no known drug allergies.

Review of Systems

- Vital signs
  - Blood pressure: 120/70 mm Hg
  - Heart rate: 78 beats/minute
  - Respiration: 15 breaths/minute

Dental History

The patient had received sporadic general dental care and orthodontics in Brazil and was unsure if the city water she consumed in her childhood was fluoridated. She had received dental care more regularly since moving to the United States 13 years ago. She had her teeth cleaned twice yearly. She reported that her teeth and gums were very sensitive and that local anesthesia was often needed during her cleanings.

Social History

The patient had been born and raised in Brazil and had moved to the United States when she was 28 years old. She was married and had two daughters. She worked as a housecleaner. The patient consumed one to three alcohol drinks per week and denied the use of tobacco products.

Family History

Both of the patient's parents currently resided in Brazil and were in good health. She had one brother who lived in the United States and was also in good health. The patient was unaware of any dental problems in the members of her immediate family.

Extraoral Examination

The patient had no detectable lesions, masses or swelling. The temporomandibular joint was within normal limits.

Intraoral Examination

- The buccal mucosa adjacent to the mandibular third molars demonstrated diffuse white reticulations.
- Generalized gingival erythema was present with desquamation of the gingiva in the maxillary and mandibular anterior regions (Figure 1).

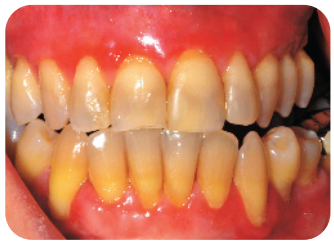

Figure 1: Frontal view.

Hard Tissue Examination

- The patient had had several restorations in her posterior dentition. All of the restorations appeared clinically and radiographically sound.
- No carious lesions were detected.

Periodontal Examination

- The periodontal examination revealed probing depths of 1–4 mm with generalized bleeding on probing.
- There was diffuse gingival erythema with varying degrees of mucosal sloughing and erosion.
- There was generalized mild to moderate gingival recession on the buccal and lingual/palatal surfaces of her teeth.
- The patient had mild plaque accumulations on her posterior teeth.

Occlusion

- The patient had class III occlusion with an open bite on the posterior left side.

Radiographic Examination

- A full mouth set of radiographs was taken.
- There were no carious lesions present.
- There was generalized mild horizontal bone loss.
- There were no other pathologic findings noted.

Diagnosis

Following review of the history and clinical evaluation, a clinical diagnosis of desquamative gingivitis was rendered.

Treatment Plan

Desquamative gingivitis is not a definitive diagnosis but a nonspecific clinical term associated with one of a variety of underlying conditions. To arrive at a definitive diagnosis, a tissue biopsy is required. The patient will receive a dental scaling with oral hygiene instructions. The treatment of her desquamative gingivitis will be determined after a definitive diagnosis is established.

Discussion

Once a clinical diagnosis of desquamative gingivitis has been rendered, a definitive diagnosis must be made. In this case, punch biopsies of the gingiva and buccal mucosa were performed and submitted for pathologic evaluation. One sample was placed in 10% formalin for hematoxylin and eosin staining and the second in Michel's medium for direct immunofluorescence. The pathology report was signed out as oral lichen planus (LP). After the diagnosis was established, the patient was prescribed a topical corticosteroid (fluocinonide gel 0.05%) and instructed to apply it two to three times daily. The patient returned after 2 weeks of treatment with a reduction in gingival erythema. The patient was informed that because there is no cure for her condition, the medication would need to be reapplied whenever she became symptomatic.

Self-Study Questions

A. How are non–plaque-induced gingival lesions classified?

B. What is desquamative gingivitis, and how does it differ from plaque-induced gingivitis?

C. What is the differential diagnosis for desquamative gingivitis?

D. How is desquamative diagnosis managed?

E. What is the presentation and prevalence of oral LP?

F. How is a diagnosis of oral LP rendered?

G. What is the etiology of oral LP?

H. What are the histopathologic features of oral LP?

I. How is oral lichen planus managed?

J. What is the long-term prognosis for oral LP?

Answers located at the end of the chapter.

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TAKE-HOME POINTS

A. According to the American Academy of Periodontology 1999 World Workshop on the classification of periodontal disease, the etiology of the non–plaque-induced gingival lesions can be divided into several categories [1]. It should be emphasized that even though the direct cause of the lesions in these cases is not plaque, the severity of the inflammation often depends on the interaction with the bacterial plaque present.

- Gingival lesions of infectious origin (e.g., herpes simplex, candidiasis)
- Gingival lesions of genetic origin (e.g., hereditary gingival fibromatosis)
- Gingival manifestations of systemic conditions (LP, mucous membrane pemphigoid, pemphigus vulgaris)
- Traumatic lesions (e.g., factitious, iatrogenic, accidental)

Arriving at a specific diagnosis may be a complex process and requires taking a detailed history along with the specific clinical presentation of a particular patient.

B. Desquamative gingivitis (DG) is a clinical term used to describe a condition characterized by intense erythema, desquamation, and ulceration of the gingiva [2]. A variety of different conditions can manifest as desquamative gingivitis, but it is most often associated with one of the vesiculoerosive diseases (see answer to question C).

Plaque-induced gingivitis is a response to inadequate oral hygiene practices and generally presents with inflammation at the gingival margin. This inflammation often causes the tissue to become erythematous and edematous. There is commonly bleeding on probing and an increase in gingival crevicular fluid or exudate. The clinical signs and symptoms of plaque-induced gingivitis are usually reversed after removing the primary etiology of bacteria-laden plaque. Refer to Chapter 1, Case 2, for a detailed description on plaque-induced gingivitis.

C. Desquamative gingivitis can be a manifestation of any of a number of dermatologic conditions, most commonly oral LP, benign mucous membrane pemphigoid, or pemphigus vulgaris [3]. Other conditions that can present as desquamative gingivitis include chronic ulcerative stomatitis, bullous pemphigoid, linear immunoglobulin A disease, dermatitis herpetiformis, or lupus erythematosus [4]. Once a clinical diagnosis of desquamative gingivitis is rendered, a definitive diagnosis must be established.

D. Regardless of the underlying cause of DG, it has been shown that improved oral hygiene can decrease the severity of the lesions [5]. However, this will not bring about complete resolution and, more importantly, does not address the underlying cause. A biopsy of the lesion must be taken to establish a definitive diagnosis. The biopsy should include perilesional tissue because the center of an ulceration histologically reveals only nonspecific granulation tissue. A second specimen submitted for immunofluorescence studies may aid in the diagnosis.

The symptoms of desquamative gingivitis are managed based on the underlying cause of the condition. In most cases, the oral lesions themselves may be managed with topical corticosteroids. A common first line of treatment is 0.05% fluocinonide gel, which may be applied to the lesions three times daily. This may be delivered directly to the gingiva or, in the case of widespread lesions, placed in a custom-fabricated tray analogous to a bleaching tray typically used for tooth whitening. Alternatively, a dexamethasone elixir may be prescribed for patients to swish and expectorate three times daily. It is important to monitor the patients for signs of oral candidiasis that may develop in the setting of steroid use.

In the case of a diagnosis of mucous membrane pemphigoid, the patient should be referred to an ophthalmologist who is familiar with the ocular lesions of this condition to guard against vision loss. Although corticosteroid therapy has helped to reduce the mortality rate associated with pemphigus vulgaris to less than 10% [6], patients with this diagnosis should be evaluated by their

primary care physicians or dermatologists for the evaluation of cutaneous lesions.

E. Oral LP is one of the most common mucocutaneous diseases manifesting on the gingiva. Oral involvement alone is common; concomitant skin lesions in patients with oral lesions have been found in 5–44% of cases [7]. Most patients who present with oral LP are middle aged. Children are rarely affected [8]. A predilection for women is shown in most series of cases by a ratio of 3:2 over men. The prevalence of oral LP in various populations has been found to be 0.1–4% [9].

Oral LP has traditionally been divided into reticular and erosive forms. Simultaneous presence of more than one type of lesion is common [10]. The reticular form is much more common than the erosive and often goes unnoticed by the patient. It generally involves the posterior buccal mucosa bilaterally, but any area of the oral mucosa may be affected. Reticular LP is named because of its characteristic pattern of interlacing white lines (Wickham striae). Erosive/erythematous LP is less common but more significant for the patient because the lesions are usually symptomatic. Clinically there are erythematous areas with or without ulcerations. The periphery is usually bordered by the fine white radiating striae of reticular LP. Erythema and/or ulceration involving the gingiva produce desquamative gingivitis.

F. The most characteristic clinical manifestations of oral LP are white interlacing white striae appearing bilaterally on the posterior buccal mucosa [11]. A diagnosis of reticular LP can often be made based on the clinical presentation alone of the lesions. Erosive LP can be more challenging to diagnose based on clinical features alone. Unilateral lesions or presentations lacking typical radiating white striae may be difficult to distinguish from other ulcerative or erosive diseases. If the diagnosis is in question after clinical examination, a biopsy is necessary to confirm a diagnosis.

G. The etiology of most oral LP cases is idiopathic. Oral lichenoid lesions can also be associated with various types of medications including nonsteroidal anti-inflammatory drugs, antihypertensive agents, antimalarials, gold salts, and penicillamine [12]. Unilateral oral lichenoid lesions are rare and may be secondary to contact with amalgam dental restorations.

H. Oral LP presents histologically with varying degrees of orthokeratosis and/or parakeratosis. There is disruption of the basal cells and transmigration of T lymphocytes into the basal and parabasal cell layers of the epithelium. Degenerating keratinocytes termed *Civatte bodies* (colloid bodies) are often found at the junction of the epithelium and connective tissue. There is characteristically a subepithelial bandlike accumulation of T lymphocytes and macrophages characteristic of a type IV hypersensitivity reaction [13] (Figure 2). These features are characteristic but not specific to oral LP. Other interface processes including lupus erythematous and chronic ulcerative stomatitis have similar histopathologic presentations.

I. Reticular LP often causes no symptoms and need not be treated. The first line of treatment for symptomatic erosive LP is topical corticosteroids. Fluocinonide gel applied to the most symptomatic

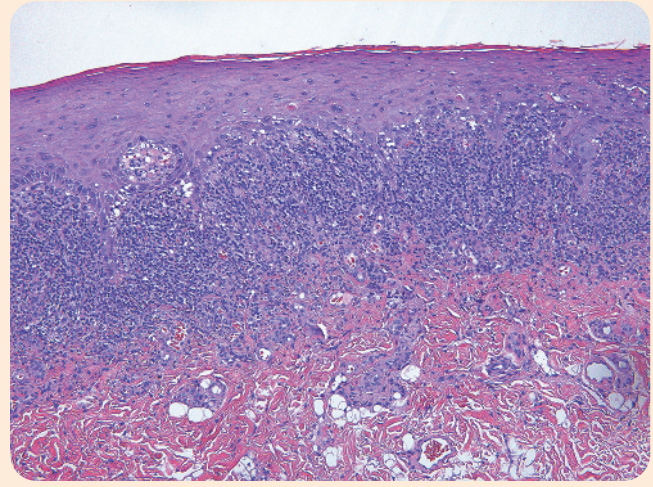


Figure 2: Characteristic histopathology of lichen planus demonstrates parakeratinized and/or orthokeratinized stratified squamous epithelium with sawtooth-shaped rete ridges, squamatization of basal cells, and a bandlike infiltrate of lymphocytes in the superficial connective tissue.

areas or dexamethasone elixir used as a mouthrinse three to four times per day is often sufficient to induce healing within 1–2 weeks. Patients should be informed that the lesions will likely return and the corticosteroids should be reapplied. Patients should be monitored for their response and for the possibility of candidiasis induced by the use of the steroids. Another important part of the therapeutic regimen in patients with desquamative gingivitis is meticulous plaque control, which results in significant improvement in many patients [14].

J. LP is a chronic condition with lesions that wax and wane over time. The erosive form should be monitored and treated as necessary to improve patient comfort. Some investigators suggest that patients with erosive LP be evaluated every 3 months, particularly if the lesions are not typical. Cases of malignant transformation of LP have been reported, but a clear association is lacking in evidence. If the potential for malignant transformation does exist, it appears to be small and generally confined to patients with the erosive form of LP [15].

Case 4

Gingival Enlargement

CASE STORY

A 35-year-old Caucasian female presented with a chief complaint of: "My gums are swollen and bleed." The patient noticed swelling of the gingiva 2 months after she started taking phenytoin (Dilantin) for epilepsy, which was first diagnosed 13 years ago. The patient did not brush or floss her teeth consistently.

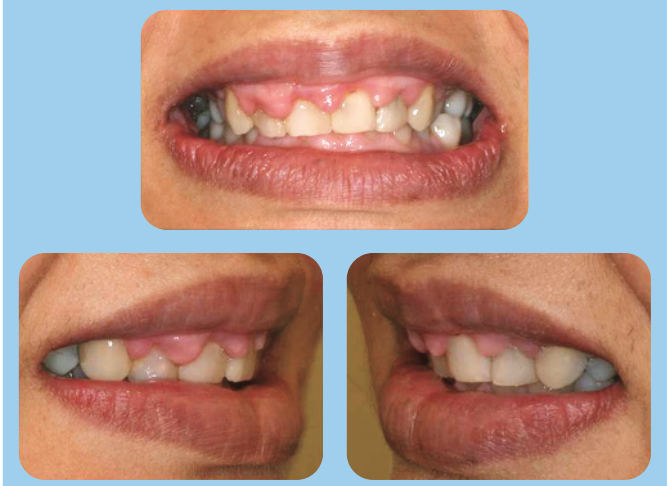


Figure 1: Initial presentation of a patient with phenytoin-induced gingival enlargement: smile frontal, right, and left views.

LEARNING GOALS AND OBJECTIVES

- To be able to diagnose gingival overgrowth
- To identify the etiology and to address it in the treatment plan
- To understand the difference between true pockets and pseudo-pockets

Medical History

The patient had been diagnosed with epilepsy about 13 years ago. Since that time she had been taking Dilantin 500 mg daily. Additionally, the patient currently took 2000 mg Depakote daily (1000 mg bid) and 10 mg Zyprexa at bedtime. There were no other significant medical problems and the patient had no known allergies.

Social History

The patient did not smoke or drink alcohol.

Extraoral and Intraoral Examinations

- There were no significant findings on extraoral examination. The patient had no masses or swelling and the temporomandibular joint was within normal limits.
- With the exception of the gingiva, the soft tissues of the mouth including the tongue appeared normal.
- Examination of the gingiva revealed generalized marginal erythema, edema, rolled margins, enlarged papillae, and bleeding on probing (Figures 1 and 2). Probing depths ranged from 2 to 5 mm (pseudo-pockets due to gingival overgrowth; Figure 3).
- The hard tissue examination found multiple restorations.

Occlusion

Angle class II, division 2 occlusion with tooth #12 in cross-bite due to arch incongruence.

Radiographic Examination

A full mouth set of radiographs revealed normal levels of alveolar bone throughout the mouth including the maxillary and mandibular anterior where clinical gingival enlargement was present (Figure 4).

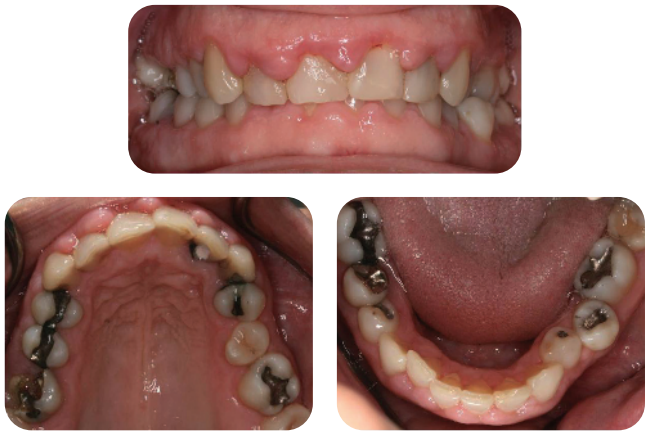


Figure 2: Initial presentation of a patient with phenytoin-induced gingival enlargement: buccal view in occlusion, maxilla and mandible occlusal views. Note that gingival enlargement is localized to anterior and facial segments on both maxilla and mandible.

Diagnosis

After reviewing the history and the clinical and radiographic examinations, the patient was diagnosed with phenytoin-associated gingival overgrowth and a differential diagnosis was generated (A).

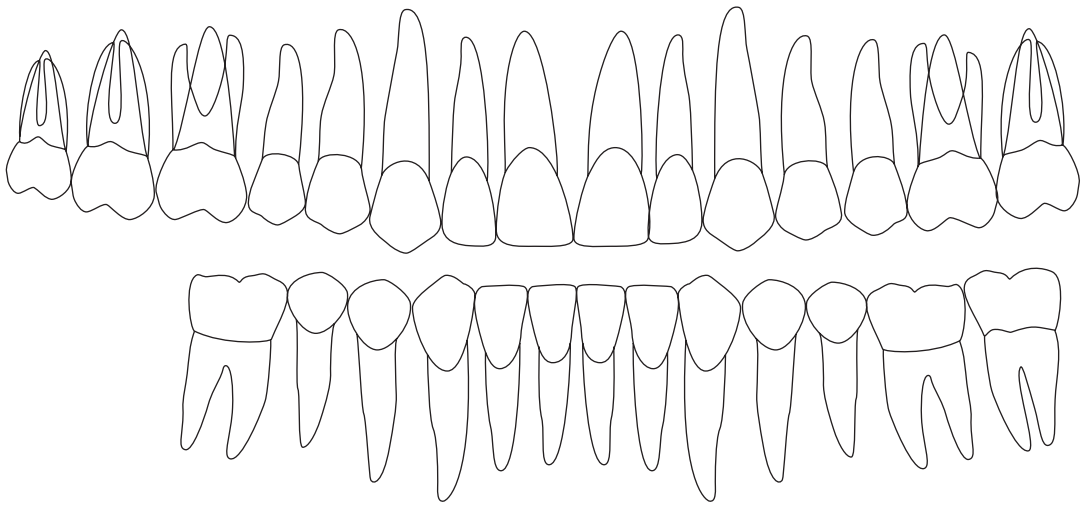
Treatment Plan

The treatment plan for the phenytoin-associated gingival overgrowth includes interdisciplinary consultation (to include the primary care physician regarding alternative medication for treatment of epilepsy), oral hygiene instructions, initial phase therapy consisting of supra- and subgingival scaling with polishing, reevaluation at 6 weeks, and surgical phase (gingivectomy) if gingival overgrowth persists. Routine maintenance therapy should be performed every 3 months following resolution of the gingival overgrowth (G).

Treatment

The patient received full-mouth scaling and polishing. The patient was referred to a restorative dentist for

Buccal	-	423	323	433	423	434	435	534	434	524	314	424	324	423	323	-
Palatal	-	433	322	323	333	322	222	322	322	223	323	323	334	323	333	-



Buccal	-	-	323	333	323	424	434	334	434	424	323	333	323	333	333	-
Lingual	-	-	333	333	323	333	333	323	323	223	223	222	323	323	333	-

Figure 3: Probing pocket depth measurements.

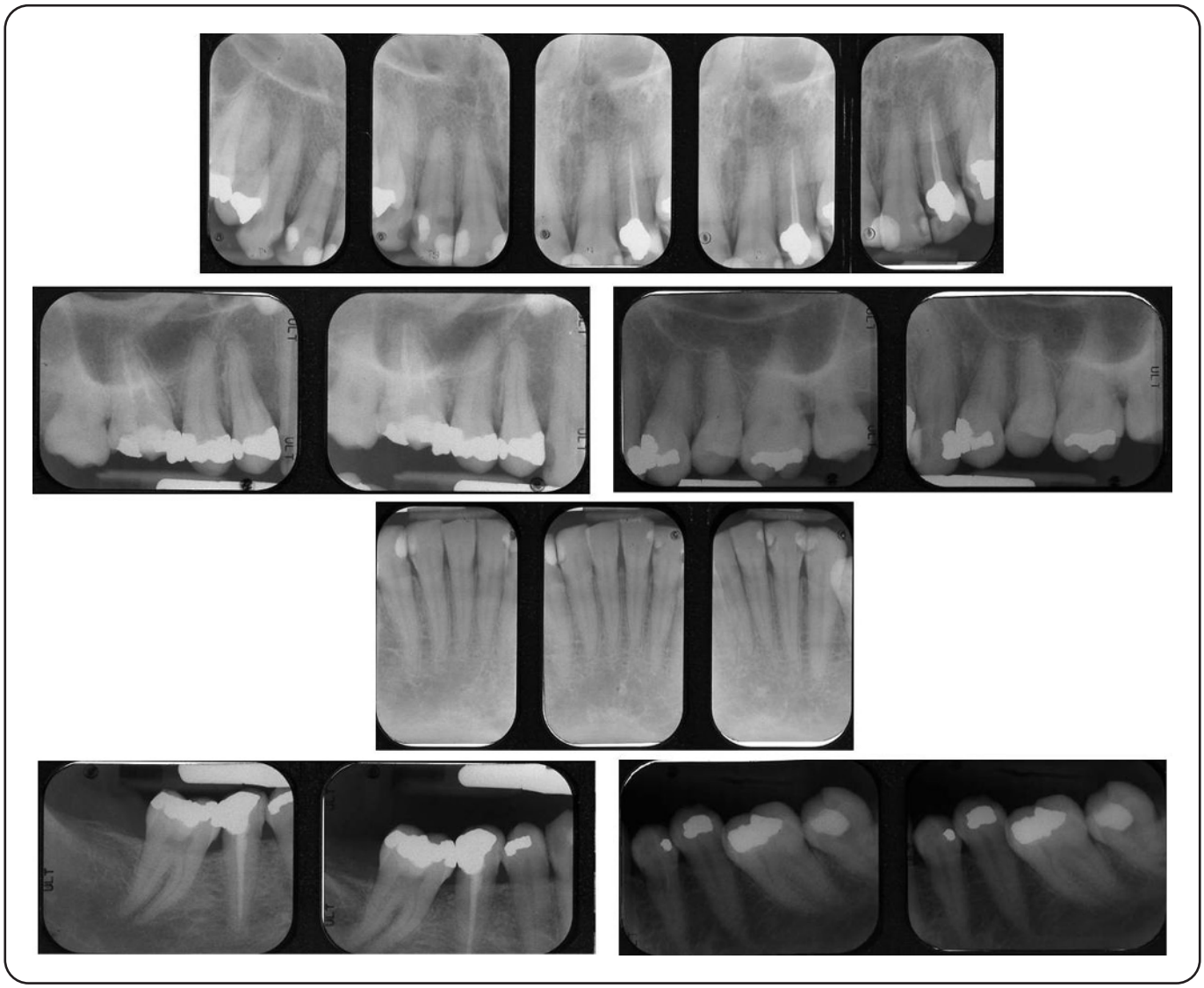


Figure 4: Peri-apical radiographs depicting the interproximal bone levels.

detailed hard tissue examination and treatment of active decay. At 6-week reevaluation, the patient demonstrated excellent oral hygiene, but the clinical signs and symptoms had improved only slightly. Therefore, gingivectomy and gingivoplasty were performed to restore gingival contours (G; Chapter 3, Case 1). The patient is currently on 3-month recall.

Discussion

Most patients who have gingival enlargement present with the chief complaint of an unaesthetic smile. Comprehensive medical and dental histories (A) as well as a complete periodontal examination are needed to come to the appropriate diagnosis. Most commonly, gingival overgrowth is a dental plaque–induced gingival disease modified by medications, such as phenytoin, cyclosporine A, or calcium channel blockers. In some rare cases, a non–plaque-induced gingival enlargement called hereditary gingival fibromatosis (A) may be seen.

Once the diagnosis is determined, the treatment plan includes oral hygiene instructions, an initial phase of treatment (scaling and polishing), and communication with the primary care physician for potential alternative medication to address the systemic condition (G). Phase 1 therapy is followed by periodontal reevaluation at 4–6 weeks. If the gingival enlargement persists at this visit, surgical excision of excessive gingiva is recommended with subsequent reinforcement of home care oral hygiene and a periodontal maintenance every 3 months is instituted (G).

Self-Study Questions

A. What is the etiology for gingival overgrowth? What questions in a dental history might help you begin to form a differential diagnosis?

B. What are the characteristics of drug-induced gingival enlargement?

C. How would you differentiate between a "true" periodontal pocket and a "pseudo-pocket"?

D. What are the clinical characteristics that distinguish gingival enlargement versus periodontitis?

E. What is the pathogenesis of gingival enlargement?

F. What is another reason for a short clinical crown?

G. What is the current treatment for patients with drug-induced gingival enlargement? What is the long-term prognosis with treatment for these patients?

Answers located at the end of the chapter.

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TAKE-HOME POINTS

A. Gingival overgrowth or enlargement is a common side effect and unwanted outcome of certain systemic medication. Drug-influenced gingival enlargement refers to an abnormal growth of the gingiva secondary to use of systemic medication and is classified by the America Academy of Periodontology as a form of dental plaque–induced gingival disease modified by medications [1]. Currently three pharmaceutical categories of medication (anticonvulsants, immunosuppressants, and calcium channel blockers) are associated with gingival enlargement. However, a strong association has been noted only with phenytoin (when used in a chronic regimen to control epileptic seizures), cyclosporine A (powerful immunoregulator drug primarily used in the prevention of organ transplant rejection), and nifedipine (commonly prescribed as antihypertensive, antiarrhythmic, and antianginal agent). The prevalence of the gingival overgrowth varies widely: the prevalence related to use of phenytoin is approximately 50%, whereas cyclosporine and nifedipine produce significant gingival changes in about 25% of the patients treated.

Among the non–plaque-induced gingival lesions, gingival fibromatosis of genetic origin has been also described as associated with gingival overgrowth [2]. Hereditary gingival fibromatosis (HGF) is an uncommon disorder that can occur as an isolated finding or as part of a genetic syndrome. HGF is most frequently reported to be transmitted as an autosomal dominant trait, but autosomal recessive inheritance has also been reported [3]. The clinical presentation of HGF is variable, both in the distribution (number of teeth involved) and in the degree (severity) of expression [3]. Affected individuals have a benign slowly progressive, nonhemorrhagic, fibrous enlargement of the oral masticatory mucosa [4]. A mutation in the Son of Sevenless-1 (*SOS1*) gene was reported to cause hereditary gingival fibromatosis type 1 [4]. HGF usually develops before the person reaches 10 years of age, often at or about the time of eruption of the permanent incisors. However, cases have been reported to occur during the eruption of the deciduous dentition and even to appear at birth [3].

Gingival enlargement has been found to be one of the oral manifestations associated with acute leukemias [5], in addition to cervical adenopathy, petechiae, mucosal ulcers and gingival inflammation. Gingival bleeding is a common and usually the initial oral sign and/or symptom in 17.7% and 4.4% of patients with acute and chronic leukemias, respectively [5]. Gingival inflammation in leukemic patients presents as swollen, glazed, and spongy tissues that are red to deep purple in appearance [6]. Gingival enlargement has been associated with leukemia beginning at the interdental papilla and extending to the marginal and attached gingiva [6].

Questions important in the development of a differential diagnosis include:

- When did your gingiva start to swell?
- Did anybody in your family describe a similar pattern of gingival enlargement?
- Are you taking any medication?
- How long have you been taking the specific medication?
- Do your gums bleed easily?

B. As detailed in the Annals of Periodontology 1999 [1], the common clinical characteristics of drug-related gingival enlargement include a variation in interpatient and intrapatient pattern (such as genetic predisposition), predilection for anterior and facial segments, higher prevalence in children (due to phenytoin most often used in young patients and having the highest prevalence of all medication-induced gingival enlargement), onset within 1–3 months of drug use, change in gingival contour leading to modification of gingival size, enlargement starting at the interdental papilla, change in gingival color, pronounced inflammatory response of gingiva in association with bacterial plaque and reduction in severity with decrease in dental plaque, bleeding upon provocation, increased gingival exudate, and found in gingiva

with or without bone loss but is not associated with attachment loss. Patients with this diagnosis are usually taking one of the following: phenytoin, cyclosporine A, or certain channel blocker drugs.

C. The probing depth is the distance from the gingival margin to the bottom of the gingival sulcus. The normal sulcus, measuring between 1 and 3 mm is normally measured to the nearest millimeter by means of a graduated periodontal probe with a standardized tip diameter of approximately 0.4–0.5 mm. The measurements recorded clinically with the periodontal probe have generally been considered a reasonably accurate estimate of sulcus or pocket depth. A probing of the sulcus depth (PPD) of ≥4 mm suggests a diseased state and represents a true periodontal pocket. A "true" periodontal pocket is the measurement from the gingival margin to the bottom of the pocket, recording an increased value (PPD ≥4 mm) beyond that found in the normal gingival sulcus. This depth increase is the result of apical migration of the junctional epithelium subsequent to alveolar bone resorption in patients with periodontitis.

Pocket depths >3–4 mm may also be caused by the swelling of the gingiva without a concomitant apical migration of dentogingival epithelium from the cementumenamel junction (CEJ), as the case of gingival enlargement. This increase in pocket depth is called a "pseudo-pocket" because it is not associated with bone loss or apical migration of the junctional epithelium.

Probing depth, in fact, is a histologic term expressing the distance from the gingival margin to the most coronal level of the junctional epithelium. Clinical probing depth measured from the gingival margin seldom corresponds to sulcus or pocket depth. The discrepancy is least in the absence of inflammation and increases with increasing degrees of inflammation [7]. In the presence of periodontitis the probe tip passes through the inflamed tissues to stop at the level of the most coronal intact dentogingival fibers, approximately 0.3–0.5 mm apical to the apical termination of the junctional epithelium. Decreased probing depth measurements following periodontal therapy may be in part due to decreased penetrability of the gingival tissues by the probe. Therefore, a distinction should be made between the histologic and the clinical PPD to differentiate between the actual depth of anatomic defect and the measurement recorded by the periodontal probe [7].

D. Gingival enlargement is usually associated with certain medications (i.e., Dilantin, nifedipine, cyclosporine A), and the clinical presentation is typically characteristic: papillary and free marginal gingiva is enlarged, mostly localized in anterior facial segments, increased probing depth with normal bone levels generally; possibly there are signs of inflammation.

Periodontitis is characterized by plaque-induced inflammation localized at the marginal gingiva, with bleeding on probing, increased probing depth with loss of periodontal tissues – cementum, periodontal ligament along with crestal bone resorption – therefore "true" periodontal pockets. Depending on the degree of clinical attachment loss, periodontitis can be mild, moderate, or severe.

Diagnosis of each disease type is critical due to distinct treatment that is needed to restore form and function and/or stabilize the periodontal disease progression, as mainly in the case of periodontitis.

E. The biologic origins for gingival overgrowth are complex. Recent studies indicate that molecular markers and clinical features of gingival overgrowth differ in their response to medication and that multiple genetic loci are linked to the inherited forms of gingival overgrowth [8].

Multiple hypotheses have been suggested and tested to better understand the molecular mechanisms underlying the clinical features of drug-induced gingival overgrowth. One leading theory is that substances that cause gingival overgrowth may do so by altering the normal balance of cytokines in gingival tissues because abnormally high levels of specific cytokines were found in enlarged gingival tissues. Among the cytokines and growth factors found to be at elevated levels in human drug-induced gingival

overgrowth are interleukin (IL)-6, IL-1β, platelet-derived growth factor-B, fibroblast growth factor-2, transforming growth factor-β, and connective tissue growth factor [8].

Connective tissue growth factor (CTGF, or CCN2), is a 38-kDa secreted protein belonging to the CCN family of growth factors. It has been shown to promote the synthesis of various components of the extracellular matrix, and its overexpression is associated with the onset and progression of fibrosis in many organs including human gingiva [9]. Moreover, fibrotic human gingival tissues express CTGF/CCN2 in both epithelium [10] and connective tissues [11], suggesting that interactions between epithelial and connective tissues could contribute to gingival fibrosis.

It has also been suggested that variations in the balance between cell proliferation and apoptosis contribute to the etiology of gingival overgrowth. Increased fibroblast proliferation and a simultaneous decrease in apoptosis were found to contribute to gingival overgrowth [12].

F. Short clinical crowns associated with healthy-appearing gingiva can be due to gingival tissue located more incisally or occlusally on the anatomic crown. Volchansky and Cleaton-Jones described this condition as delayed passive eruption [13,14]. They reported an incidence of 12% of patients examined demonstrating delayed passive eruption. Goldman and Cohen also described this condition where the gingival margin fails to recede to the CEJ during tooth eruption as altered (retarded) passive eruption [15].

G. The treatment of patients with drug-induced gingival enlargement consists of oral hygiene instructions, supra- and subgingival scaling and polishing, and referral to the primary care physician for possible substitution of one medication for another (i.e., phenytoin can be replaced with carbamazepine or valproic acid, cyclosporine with tacrolimus, and nifedipine with one of many dihydropteridines) not as strongly associated with gingival overgrowth. If the previously described treatment does not result in significant resolution of gingival enlargement, surgical excision of the excessive gingiva is performed using a classic external bevel gingivectomy or an internal bevel gingivectomy approach [16] (Chapter 3, Case 1). The internal bevel approach provides primary closure and reduction of postoperative bleeding, discomfort, and infection. More recently, a carbon dioxide laser has been used for surgical excision and provides rapid hemostasis and compatibility with a host with underlying medical conditions. It also has been reported to reduce surgical time [16].

Having the patient in a rigorous home plaque control regimen as well as regular 3-month periodontal maintenance are strongly recommended [17] and may considerably reduce the risk of recurrence. In a study of 38 individuals, 18 months after surgical therapy, the recurrence rate of gingival overgrowth in patients taking cyclosporin A or nifedipine was 34%. Age, gingival inflammation, and attendance at periodontal maintenance visits were all significantly related to recurrence, and they suggest that regular re-motivation and professional care at frequent recall appointments are of great importance in patients with a history of drug-induced gingival overgrowth [18]. To prevent postsurgical recurrence, a chlorhexidine rinse twice daily is recommended [19].

Several medications have been shown to ameliorate gingival enlargement such as systemic or topical folic acid [20] or a short course of metronidazole or azithromycin. The latter drugs work particularly well for significant resolution of cyclosporine-induced gingival overgrowth [21]. The mechanism of action for these antibiotics is not clear, but it is suggested they may contribute to inhibition of collagen fiber proliferation in addition to their antimicrobial action.

Case 5

Aggressive Periodontitis

CASE STORY

A 23-year-old African American female presented with a chief compliant of: "Bleeding gums on brushing and swollen gingiva in specific areas of the mouth." The patient's dentist had observed 7- to 10-mm probing depths in several teeth in all four quadrants of the mouth and referred her to a periodontist for a periodontal consultation.

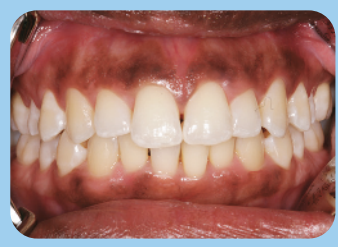

Figure 1: Preoperative frontal view.

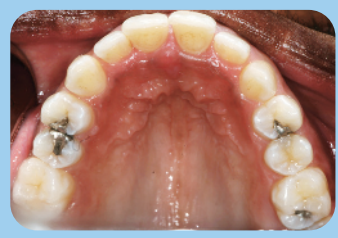

Figure 2: Preoperative maxillary dentition.

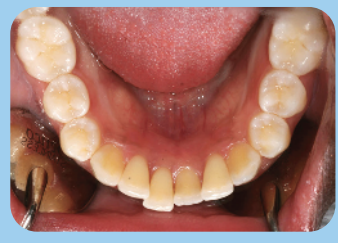

Figure 3: Preoperative mandibular dentition.

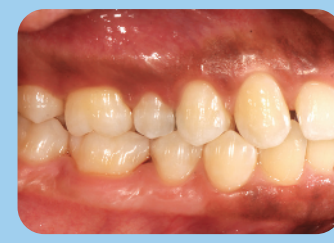

Figure 4: Preoperative left occlusal view.

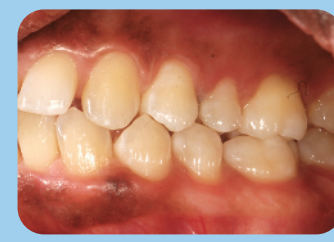

Figure 5: Preoperative right occlusal view.

LEARNING GOALS AND OBJECTIVES

- To be able to understand the definition and diagnostic criteria of aggressive periodontitis
- To understand the various treatment options available for this condition
- To understand the prognosis of periodontal and implant treatment in these patients

Medical History

No relevant medical history was noted, and the patient did not report any allergies to food or to drugs. The patient was not taking any medications.

Review of Systems

- Vital signs
  - Blood pressure: 120/80 mm Hg
  - Pulse rate: 73 beats/minute (regular)
  - Respiration: 15 breaths/minute

Social History

The patient was a nonsmoker and reported that she did not consume alcohol.

Extraoral Examination

There were no significant findings. The patient had no masses or swelling, and the temporomandibular joint was within normal limits.

Intraoral Examination

- No abnormal findings with respect to tongue, floor of the mouth, palate, and buccal mucosa were observed.
- A gingival examination revealed mild marginal erythema with areas of rolled margins and swollen papillae in relation to all first molars and mandibular incisors (Figures 1 and 3).
- A periodontal charting was completed (Figures 4 and 5). Tooth #3, 14, 19, and 30 exhibited probing depths >7 mm, especially in the interproximal areas. Mandibular incisors also exhibited probing depths in the range of 6–7 mm (Figures 6 and 7).
- Grade 3 mobility was observed in teeth #23 to 26.
- The teeth other than incisors and molars exhibited probing depths in the range of 2–4 mm.
- Grade II furcation involvements were recorded for all the affected molars.
- The patient's oral hygiene was good.

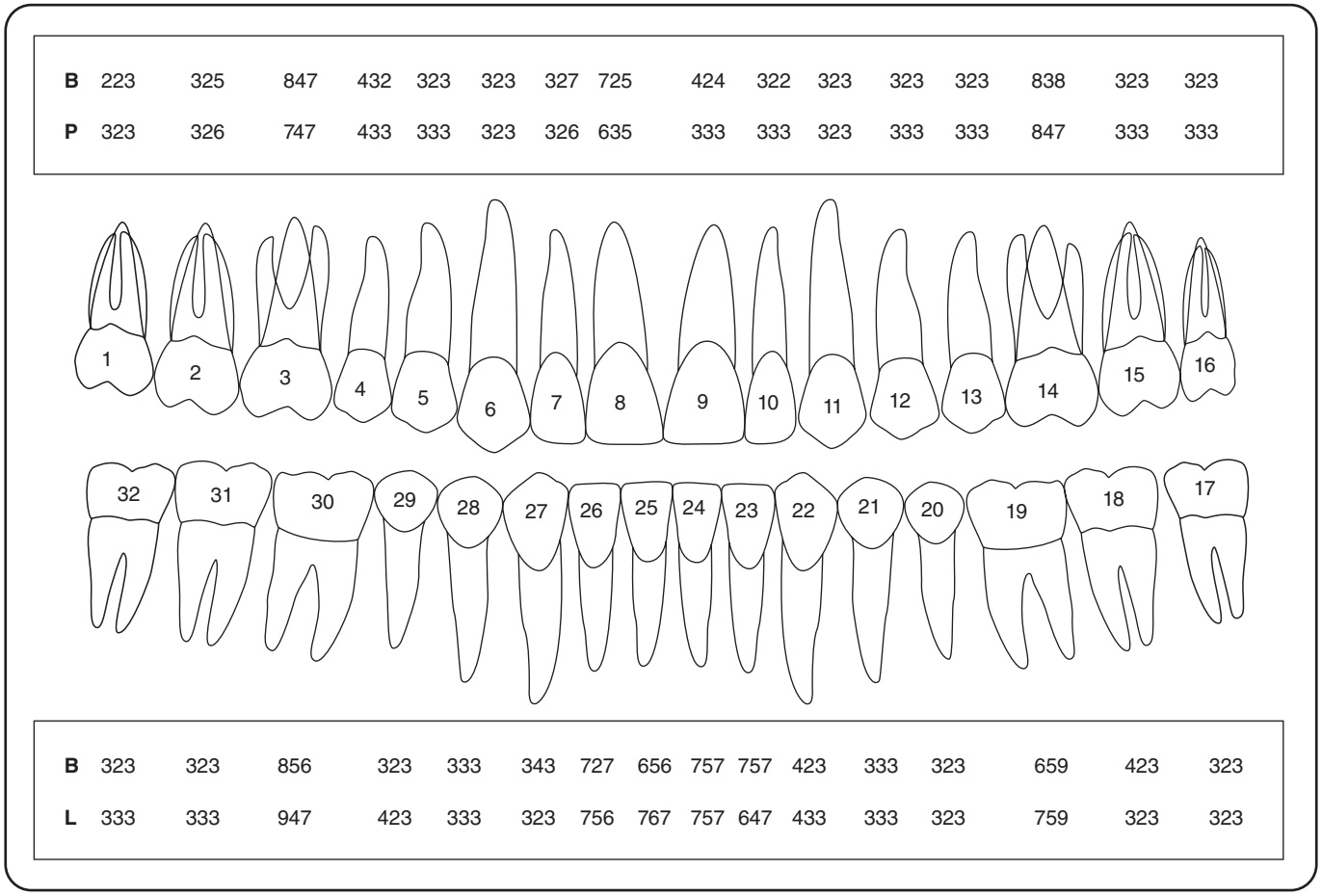

Figure 6: Probing pocket depth measurements during phase I revaluation. B, buccal; P, palatal; L, lingual.

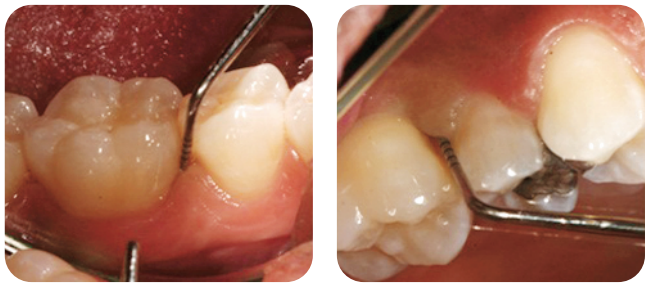

Figure 7: Intraoral clinical photographs depicting deeper probing depth associated with maxillary and mandibular molars.

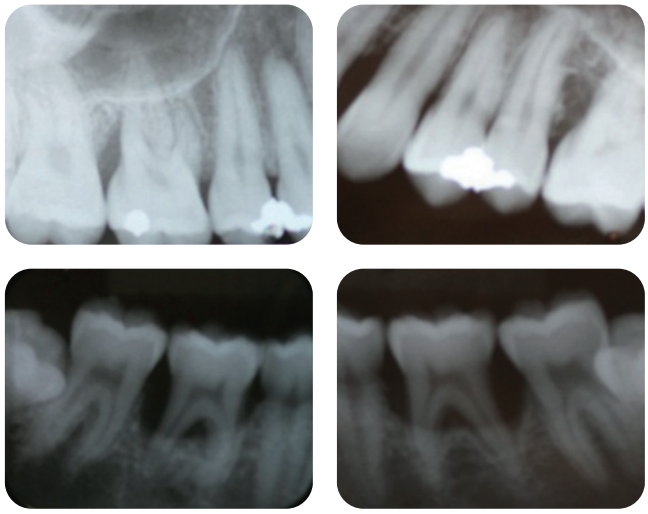

Figure 8: Periapical radiographs demonstrating the intrabony defects surrounding all four molars and the relatively normal premolars and second molars.

Occlusion

There were no occlusal discrepancies or interferences.

Radiographic Examination

A full-mouth set of radiographs was ordered. Periapical radiographs of the affected molars are shown in Figure 8. The radiographs clearly demonstrate the involvement of all first molars, the classical presentation of localized aggressive periodontitis (LAP). All the defects were intrabony and confined to interproximal areas of the maxillary molars, involving the proximal furcations, and are circumferential in the mandibular molars involving the buccal or lingual furcation areas.

Diagnosis and Prognosis

The patient's age, ethnicity, history, and clinical and radiographic examinations led to the diagnosis of LAP. Molars are not mobile, and the defects surrounding them are intrabony, which are highly amenable to regeneration by bone grafting or by guided tissue regeneration. Moreover, the patient had very good oral hygiene and was highly compliant. Therefore, these molars will have a good prognosis after treatment. With respect to mandibular incisors, the long-term prognosis will be questionable because the sites exhibit severe destruction of the periodontium with bone loss almost up to the apex of the teeth.

Treatment Plan

The treatment plan for a typical case of LAP consists of the following phases:

- The diagnostic phase consists of a comprehensive periodontal examination, radiographs, and study models. In some cases, microbial testing and genetic testing are performed.
- The disease control phase includes oral hygiene instruction, splinting of the mandibular incisors, and scaling/root planing of all the affected areas with adjunctive antibiotics (amoxicillin 500 mg and metronidazole 250 mg tid for 14 days). Extractions of all the third molars can be included in this phase.
- The reevaluation phase consist of revisiting her probing pocket depths and her overall periodontal condition plus treatment planning for sites that did not improve after the initial phase of therapy or those that warrant further treatment.
- The sites that need further treatment will be treated surgically.
- Bone grafting or guided tissue regeneration is commonly used to treat intrabony defects associated with molars.
- After the surgical phase, the sites will again be evaluated for improvement in the periodontal condition.
- Once the periodontal condition is stabilized, the patient will be placed on a 3- to 4-month maintenance protocol.

Discussion

Aggressive periodontitis, categorized as a separate class of periodontal disease in the American Academy of Periodontology classification [1] is highly unique and distinct from the most common form of periodontitis (i.e., chronic periodontitis). The characteristic clinical and radiographic features associated with aggressive periodontitis allow the oral health care provider to diagnose the condition without much difficulty. The

case presented here exhibits the classical clinical and radiographic features of LAP. Therefore a clear-cut diagnosis of LAP was made. In some cases, microbiologic and immunologic tests can be used as an adjunct to diagnose this disease. Increased levels of *Actinobacillus actinomycetemcomitans* (especially serotype b) microbes and a robust antibiotic response to the same microorganism are expected in such testings. After completing phase I therapy consisting of scaling and root planing, a drastic improvement in probing depth reduction and clinical attachment gain are expected in deeper pockets. Residual pockets (>6 mm) remaining after phase I therapy are usually treated with surgical periodontal therapy. Adequate oral hygiene is critical in the successful outcome of any periodontal therapy. Usually patients with LAP tend to exhibit insignificant amount of local factors such as plaque and calculus and tend to have good oral hygiene. Even in the case described in this chapter, the patient had insignificant amounts of local factors (Figures 1–5). With respect to outcomes of surgeries performed in patients with aggressive periodontitis, long-term stability after regenerative therapy has been shown but mostly in the form of case reports [2,3]. The success rates of dental implants in patients with aggressive periodontitis in not conclusive. Considering the defect in host response in these patients, it is reasonable to expect lower implant survival rates in these patients. Some studies have indicated that the success rates in these patients are slightly lower (10%) than in patients with chronic periodontitis [4–6].

Self-Study Questions

A. How do you define aggressive periodontitis and how do you classify it?

B. What are the other terms used to describe aggressive periodontitis?

C. What are the characteristic clinical presentations common to LAP and generalized aggressive periodontitis (GAP)?

D. What are the features that distinguish localized from generalized periodontitis?

E. How common is aggressive periodontitis, and which sector of the population is more susceptible to this disease?

F. What are the etiologic agents responsible for aggressive periodontitis?

G. How do you treat patients with aggressive periodontitis?

H. In aggressive periodontitis patients, what will be the prognosis after treatment?

Answers located at the end of the chapter.

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TAKE-HOME POINTS

A. Aggressive periodontitis is a rapidly progressing form of periodontitis, characterized by early onset and familial aggregation; affected individuals are otherwise clinically healthy [1,7].

Aggressive periodontitis is subclassified into localized (LAP) and generalized (GAP) forms. LAP is defined as the interproximal loss of attachment localized to at least two permanent first molars/incisors, one of which is a first molar, and involving no more than two teeth other than first molars and incisors. When the interproximal loss of attachment extends to at least three permanent teeth other than first incisors and molars, the condition is called generalized aggressive periodontitis. If left untreated, 35% of originally classified LAP may progress to GAP [8].

B. Aggressive periodontitis was formerly called "Periodontosis" [9,10] and later called "early-onset periodontitis" (EOP). EOP includes prepubertal, juvenile, and rapidly progressive periodontitis. Because aggressive periodontitis generally affects young patients, there was a tendency to consider it a childhood disease. The current diagnosis is recommended to be based on clinical, radiographic, historical, and/or laboratory findings, rather than the age of the patient [1].

LAP and GAP replace the former terms *localized prepubertal/juvenile periodontitis* and *generalized prepubertal/juvenile periodontitis*, respectively. The patients who were classified with rapidly progressive periodontitis (RPP) are now be assigned to either GAP or "chronic severe periodontitis" based on the clinical presentation.

Most of the patients diagnosed with "generalized prepubertal periodontitis" in the 1989 American Academy of Periodontics (AAP) classification actually had been found to be associated with systemic conditions and should now be placed under the category of "periodontitis as a manifestation of systemic diseases."

C. The Following are primary features common to both LAP and GAP [11]:

- Rapid attachment loss accompanied with severe bone destruction. The progression rate of aggressive periodontitis is about three to four times faster than that of chronic periodontitis. The rapidly progressive vertical bone loss is often half-moon shaped and symmetric to the contralateral tooth [12].
- Patients are usually medically healthy.
- Familial aggregation is a common to both LAP and GAP.

Secondary features that are frequently but not always present in LAP and GAP include the following:

- Inconsistency in the relationship between the amount of microbial deposits and the severity of periodontal destruction.
- Elevated levels of *A. actinomycetemcomitans* and/or *Porphyromonas gingivalis*.
- Patients usually exhibit phagocyte abnormalities (e.g., abnormal polymorphonuclear neutrophil in adherence, chemotaxis, phagocytosis, superoxide (O_2^-) production and bactericidal activity [13–16].
- Elevated levels of inflammatory cytokines (e.g., prostaglandin E2, interleukin [IL]-1α, and IL-1β) from primed macrophages.
- Progression of attachment loss and bone loss may be self-arresting and remain stationary for years.

D.

Table 1: Features distinguishing localized aggressive periodontitis from generalized aggressive periodontitis

Features	LAP	GAP
Age of onset	Circumpubertal	<30 years of age but may be older
Clinical manifestation	Involves no more than two teeth other than incisors and first molars	Involves at least three teeth other than incisors and first molars
Serum antibody response to infecting agents [17]	Robust response	Poor response

LAP, localized aggressive periodontitis; GAP, generalized localized aggressive periodontitis.

E. The prevalence of aggressive periodontitis varies among racial and geographic groups and ranges from 0.1% to 35% [18]. The prevalence is about 0.2% in white populations and about 2.6% for people of African descent [19]. Early studies showed that aggressive periodontitis was more common in females than in males. However, the high incidence in females is believed due to the high number of females in the study populations. The proportion of affected males and females is now believed to be similar [20,21].

F. Nonmotile gram-negative anaerobic rods such as *A. actinomycetemcomitans, P. gingivalis* [22,23], and red and some orange complex species [24] are the most numerous and prevalent periodontal pathogens in aggressive periodontitis and present in most of the diseased sites compared with healthy sites. *A. actinomycetemcomitans* (especially serotype b) was found in higher numbers and frequency when compared with other pathogens in aggressive periodontitis [25,26].

G. The general treatment methods should be similar to those used for chronic periodontitis, including oral hygiene instruction/reinforcement, plaque control, scaling and root planing, and occlusal adjustment (if necessary).

Additional treatments that may be required in certain patients include:

- General medical evaluation to determine the presence of any systemic diseases. Consultation with the physician may be indicated.
- Counseling of family members.
- Adjunctive antimicrobial therapy combined with scaling and root planing. Tetracycline is contraindicated in young patients due to the problem of tooth staining. Systemic administration of amoxicillin 500 mg plus metronidazole 250 mg/500 mg tid or metronidazole 500 mg alone tid for 7 days resulted in significant clinical improvement and reduction of the levels of key periodontal pathogens for up to 6 months in deep pockets of GAP patients [27,28].
- Periodontal maintenance with short intervals may be needed.

Teeth with poor prognosis are usually extracted mostly in phase 1 or sometimes in phase 2 of periodontal therapy. Most of the intrabony defects that result from aggressive periodontitis and that are amenable to regeneration are surgically treated using either guided tissue regeneration (GTR) or using bone grafts (Figure 9). See the appropriate chapters in this textbook for more details on these surgical techniques.

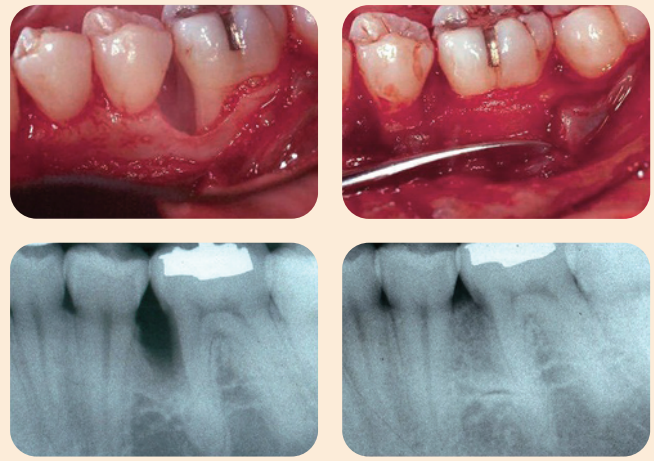

Figure 9: Classical intrabony defect affecting a mandibular first molar in another patient with LAP (top left). Guided tissue regeneration (GTR) was performed to regenerate the periodontal defect using bone grating and membrane (top right). Periapical radiographs depicting the vertical bony defect before (lower left) and after (lower right) GTR therapy. Significant radiographic bone fill was obtained after GTR therapy.

H. Scaling and root planing in combination with amoxicillin 375 mg and metronidazole 250 mg (tid for 7 days) in patients with *Actinobacillus actinomycetemcomitans*–associated periodontitis or aggressive periodontitis improved clinical parameters and suppressed *A. actinomycetemcomitans* below cultivable levels in most of the patients for up to 2 years with supportive periodontal therapy once every 3–6 months [29,30]. Long-term stabilization of periodontal health also has been reported after administering amoxicillin 500 mg and metronidazole 250 mg along with periodontal surgeries with a small percentage (5–10%) of recurrence in 5 years [31,32].

Treated LAP patients, when followed for 15 years, have been shown to have limited recurrence rate even in the absence of maintenance [33].

Successful treatment outcomes also have been shown following GTR [2,3,34] or enamel matrix derivative [35] in LAP.

The success rates in patients with aggressive periodontitis is not conclusive. Considering the defect in host response in these patients, it is reasonable to expect lower survival rates of the teeth in these patients, compared with chronic periodontitis patients. Some studies have indicated that the success rates in these patients are slightly lower (10%) than in patients with chronic periodontitis [4–6].

Case 6

Chronic Periodontitis

CASE STORY

The patient had been referred by his general dentist for periodontal treatment. Although he had a long history of dental treatment, he had never been diagnosed with periodontal disease. At the time of his first visit he had no chief complaint. He did report occasional gingival bleeding during toothbrushing.

LEARNING GOALS AND OBJECTIVES

- To be able to identify the clinical features and overall characteristics of chronic periodontitis
- To be able to list difficulties in the proper diagnosis of early chronic periodontitis
- To understand possible overlaps with the diagnosis of aggressive periodontitis
- To know what clinical changes can be anticipated in the response of chronic periodontitis to anti-infective therapy

Medical History

There were no significant medical problems, and the patient had no known allergies. He had been previously hospitalized for a day surgery to remove polyps from his vocal cords. At the time of his first appointment he was not taking any medication.

Review of Systems

- Vital signs
  - Blood pressure: 120/75 mm Hg
  - Pulse rate: 70 beats/minute (regular)

Social History

The patient was a 43-year-old white man, originally from Texas, but had been living in Massachusetts for the past 10 years. He drank alcohol socially, quit smoking 10 years ago, and reported not consuming recreational drugs. He was a musician, divorced, and has no children. His mother had heart disease, and his father died of lung cancer.

Extraoral Examination

His extra-oral examination was unremarkable: skin, head, neck, temporomandibular joint, and muscles were all within normal limits.

Intraoral Examination

The oral cancer screen was negative. His gingiva was pink, firm, with pointed papillae on the buccal aspect (Figure 1). The lingual and palatal aspects, though, presented with signs of inflammation, with erythematous and edematous gingival margins. Adequate amounts of attached tissue were present around most teeth. Gingival recession was present at several sites (see periodontal chart for details). Supra- and subgingival calculus could be detected on several tooth surfaces, particularly on the buccal surface of upper molars and lingual surfaces of lower incisors. There was generalized plaque accumulation. Saliva was of normal flow and consistency.

The periodontal chart presented in Figure 2 includes the following periodontal parameters: (1) probing pocket depth (PD) in millimeters; (2) measurement from the cementoenamel junction (CEJ) to the gingival margin (GM) in millimeters (gingival recession was recorded as a negative value); (3) clinical attachment level (CAL), which was calculated by subtracting the CEJ-GM distance from the PD; and (4) presence (1) or absence (0) of bleeding on probing (BOP). Each clinical

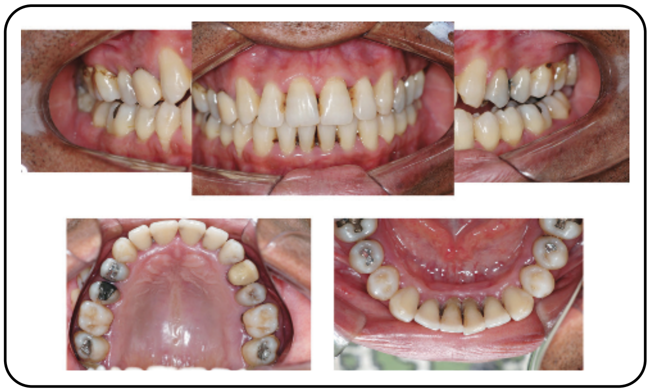

Figure 1: Clinical presentation of the case at initial visit. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

parameter was measured at six sites per tooth excluding third molars. Probing values were colored in blue, green, or red to highlight shallow (<4 mm), intermediate (4–6 mm), and deep pockets (>6 mm), respectively. Bleeding on probing was detected in 68% of sites, and the mean values for PD and CAL were 3.2 mm and 2.7 mm, respectively.

Occlusion

There were no signs of trauma from occlusion, no major occlusal discrepancies and interferences, and no significant mobility.

Radiographic Examination

A full mouth set of radiographs (Figure 3) was exposed. There was generalized moderate to severe

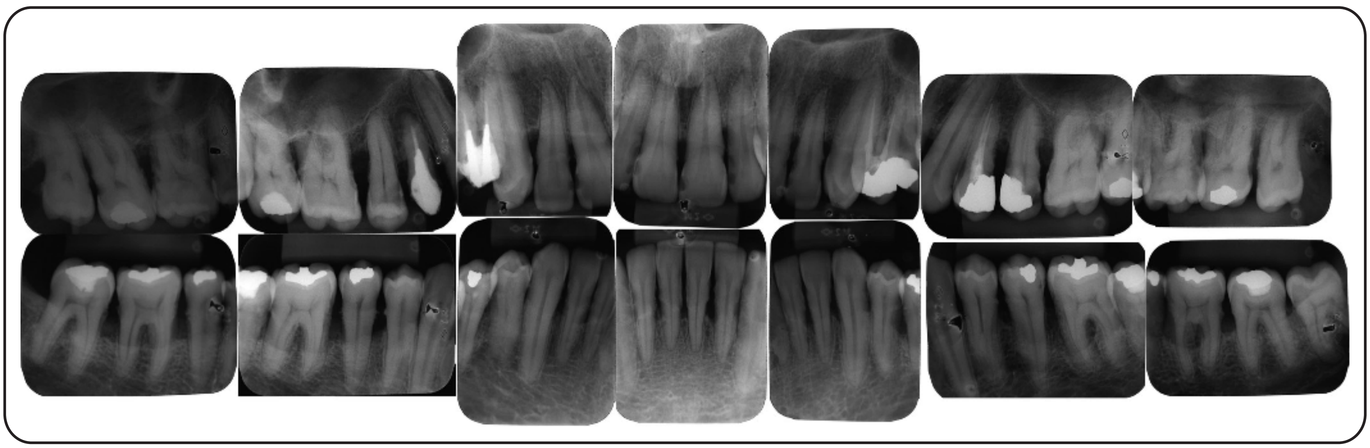

Figure 2: Periodontal chart, initial visit. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

Visit:	Initial Exam																				
Surface	Buccal																				
Tooth	2			3			4			5			6			7			8		
PD	8	3	4	5	3	4	7	2	5	4	2	4	4	2	3	5	1	3	3	1	4
CEJ-GM	2	-2	-1	-1	-2	-1	1	-1	1	1	-1	1	2	0	0	1	0	0	0	0	1
CAL	6	5	5	6	5	5	6	3	4	3	3	3	2	2	3	4	1	3	3	1	3
BOP	1	1	1	1	1	1	1	0	0	1	0	1	1	0	0	1	0	0	0	0	1
Surface	Palatal																				
Tooth	2			3			4			5			6			7			8		
PD	8	2	7	7	2	6	7	2	5	5	2	5	4	3	4	5	4	4	3	2	3
CEJ-GM	2	0	2	2	-1	2	2	0	2	2	0	2	2	1	1	1	1	1	1	0	1
CAL	6	2	5	5	3	4	5	2	3	3	2	3	2	2	3	4	3	3	2	2	2
BOP	1	1	1	1	1	1	1	1	1	1	1	1	0	0	1	1	1	1	1	1	1
Surface	Buccal																				
Tooth	31			30			29			28			27			26			25		
PD	3	2	3	5	1	1	3	1	3	1	1	2	2	1	1	1	1	1	1	1	2
CEJ-GM	3	0	2	2	-1	0	1	0	1	0	-2	1	0	0	-1	-1	0	-2	-2	0	-2
CAL	0	2	1	3	2	1	2	1	2	1	3	1	2	1	2	2	1	3	3	1	4
BOP	1	0	1	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Surface	Lingual																				
Tooth	31			30			29			28			27			26			25		
PD	4	2	6	6	4	5	5	4	4	4	1	2	3	1	3	3	2	2	2	1	2
CEJ-GM	4	2	2	2	1	2	2	2	2	2	0	2	2	0	1	1	-1	-2	-2	-3	-3
CAL	0	0	4	4	3	3	3	2	2	2	1	0	1	1	2	2	3	4	4	4	5
BOP	1	1	1	1	1	1	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1

Visit:																					
Surface																					
Tooth	9			10			11			12			13			14			15		
PD	4	1	2	3	1	3	4	1	4	4	1	6	5	2	6	6	2	6	6	2	7
CEJ-GM	2	-1	-1	0	0	0	1	0	2	2	-1	2	1	-1	2	2	-2	1	1	-1	2
CAL	2	2	3	3	1	3	3	1	2	2	2	4	4	3	4	4	4	5	5	3	5
BOP	1	0	0	0	0	0	0	0	1	1	0	1	1	1	1	1	1	1	1	1	1
Surface																					
Tooth	9			10			11			12			13			14			15		
PD	3	3	3	4	3	4	3	3	3	3	2	4	5	2	7	6	3	7	7	3	8
CEJ-GM	1	0	0	0	0	1	1	0	1	1	1	1	1	0	2	2	0	2	2	1	2
CAL	2	3	3	4	3	3	2	3	2	2	1	3	4	2	5	4	3	5	5	2	6
BOP	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Surface																					
Tooth	24			23			22			21			20			19			18		
PD	2	1	2	1	1	1	1	1	1	1	1	1	2	1	2	2	1	3	3	1	3
CEJ-GM	-2	0	-2	-1	0	0	1	0	0	0	-1	1	1	-1	1	1	0	1	1	-1	2
CAL	4	1	4	2	1	1	0	1	1	1	2	0	1	2	1	1	1	2	2	2	1
BOP	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	1	0	1	1	0	1
Surface																					
Tooth	24			23			22			21			20			19			18		
PD	2	3	4	4	3	3	4	1	3	4	2	5	6	3	5	4	2	6	6	3	2
CEJ-GM	-3	-3	-2	-2	-1	0	2	1	2	1	0	2	2	2	2	2	2	2	2	2	2
CAL	5	6	6	6	4	3	2	0	1	3	2	3	4	1	3	2	0	4	4	1	0
BOP	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1

Figure 3: Full-mouth periapical radiographs of the case at initial visit. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

horizontal bone loss. There was furcation involvement seen in teeth #1, 2, 3, 14, 15, 16, 18, 19, and 30. There was a periapical radiolucency noted on tooth #5. There were root canal treatments seen on teeth #5/12. There were several amalgam restorations and recurrent decay noted on tooth #13.

Treatment Plan

The treatment plan for this case consisted primarily of four to six sessions of scaling and root planning accompanied by oral hygiene instructions. A reassessment of the case was planned for 3 months after the completion of this initial phase when the need for additional therapy would be decided.

Treatment

After the patient's initial examination, radiographs, and charting (Figures 2–4), a comprehensive treatment planning was presented and agreed upon. The first session involved full-mouth gross scaling and oral hygiene instructions on the proper toothbrushing technique and use of dental floss. Due to the presence of large amounts of subgingival calculus, the patient required six sessions of subgingival scaling and root planing (SRP) under local anesthesia (one sextant of the mouth was instrumented per session). During this active phase of anti-infective therapy, previously instrumented sextants were constantly reexamined for the detection of residual supra- and subgingival calculus, and whenever detected, residual calculus was removed. Every SRP session was accompanied by reinforcement of the oral hygiene instructions.

Three months after the last SRP session, the subject was reexamined (Figures 4 and 5), residual pockets ≥4 mm received additional SRP, a full-mouth prophylaxis was performed, and oral hygiene instructions reinforced. Mean PD and CAL were reduced to 2.3 mm and 2.4 mm, respectively, and there was a reduction in percentage of BOP to 13%. At that time no additional periodontal therapy was deemed necessary. The patient was placed in a recall system for supportive periodontal therapy every 3 months.

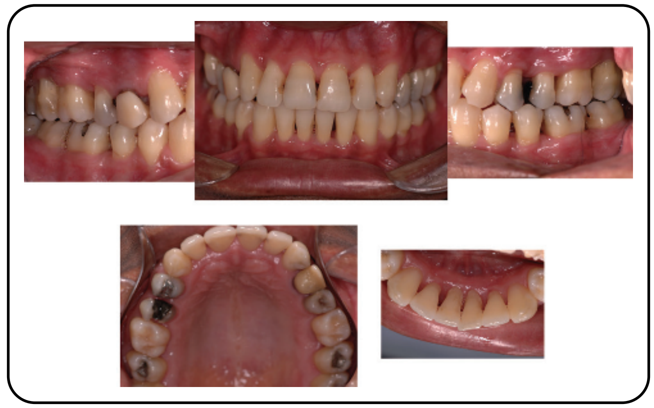

Figure 4: Clinical presentation of the case 3 months after therapy. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

Visit: 3 months post-therapy

Surface	Buccal																				
Tooth	2			3			4			5			6			7			8		
PD	6	2	3	4	3	3	4	1	3	2	2	2	2	2	3	3	1	2	2	1	2
CEJ-GM	0	-3	-3	-2	-2	-1	-1	-2	-1	-1	-1	-1	1	0	0	-1	-1	-2	-1	-1	0
CAL	6	5	6	6	5	4	5	3	4	3	3	3	1	2	3	4	2	4	3	2	2
BOP	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Surface	Palatal																				
Tooth	2			3			4			5			6			7			8		
PD	5	2	4	3	2	4	4	2	4	3	2	3	2	3	2	3	2	2	3	2	3
CEJ-GM	1	0	1	2	-1	1	1	0	1	2	0	2	1	1	1	1	1	1	1	0	1
CAL	4	2	3	1	3	3	3	2	3	1	2	1	1	2	1	2	1	1	2	2	2
BOP	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Surface	Buccal																				
Tooth	31			30			29			28			27			26			25		
PD	3	2	3	4	1	1	2	1	2	1	1	2	2	1	1	1	1	1	1	1	2
CEJ-GM	2	0	1	0	-1	0	-1	-1	0	0	-2	1	0	0	-1	-1	0	-2	-2	0	-2
CAL	1	2	2	4	2	1	3	2	2	1	3	1	2	1	2	2	1	3	3	1	4
BOP	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Surface	Lingual																				
Tooth	31			30			29			28			27			26			25		
PD	3	2	4	4	3	4	3	2	3	3	1	2	3	1	3	2	2	2	2	1	2
CEJ-GM	3	2	1	1	0	1	2	3	2	2	0	2	2	0	0	-1	-2	-2	-2	-3	-3
CAL	0	0	3	3	3	3	1	-1	1	1	1	0	1	1	3	3	4	4	4	4	5
BOP	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0

Surface	Buccal																				
Tooth	9			10			11			12			13			14			15		
PD	2	1	2	2	1	2	2	1	2	2	1	3	3	2	4	4	2	4	4	2	4
CEJ-GM	-1	-1	-1	-1	-1	-1	0	0	0	0	-1	-2	-1	-1	-1	0	-2	-1	-2	-2	0
CAL	3	2	3	3	2	3	2	1	2	2	2	5	4	3	5	4	4	5	6	4	4
BOP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	1	0	1
Surface	Palatal																				
Tooth	9			10			11			12			13			14			15		
PD	2	2	3	3	2	3	3	2	3	3	2	3	4	2	4	4	2	4	4	2	5
CEJ-GM	1	0	0	0	0	1	1	0	1	1	1	1	1	0	1	1	0	1	2	1	1
CAL	1	2	3	3	2	2	2	2	2	2	1	2	3	2	3	3	2	3	2	1	4
BOP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	1
Surface	Buccal																				
Tooth	24			23			22			21			20			19			18		
PD	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	2	2	1	2
CEJ-GM	-2	0	-2	-1	0	0	0	0	0	0	-1	0	-1	-2	-1	0	0	0	0	-1	0
CAL	4	1	4	2	1	1	1	1	1	1	2	1	2	3	2	2	1	2	2	2	2
BOP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	1
Surface	Lingual																				
Tooth	24			23			22			21			20			19			18		
PD	2	1	2	2	1	2	2	1	2	2	2	3	4	2	3	3	2	4	4	2	2
CEJ-GM	-3	-3	-2	-2	-2	0	0	0	1	1	0	1	1	1	1	2	2	2	1	2	2
CAL	5	4	4	4	3	2	2	1	1	1	2	2	3	1	2	1	0	2	3	0	0
BOP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	1

Figure 5: Periodontal chart, 3 months after therapy. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

Figures 6 and 7 illustrate the clinical presentation and the periodontal parameters 1 year after completion of periodontal therapy.

Discussion

Chronic periodontitis is diagnosed based on the clinical signs of inflammation and clinical evidence of periodontal tissue destruction. Radiographs also help to determine the extent of bone loss. Chronic periodontitis is distinguished from gingivitis primarily on the basis of the presence of loss of attachment and resorption of alveolar bone. Therefore, in addition to signs such as redness, swelling, bleeding tendency, and suppuration, the diagnosis of periodontitis requires the presence of periodontal pockets associated with clinical loss of attachment. Alveolar bone loss is also a hallmark of the pathology and can be detected radiographically. It is not uncommon to detect extensive accumulation of dental plaque and calculus, although this can also be found in gingivitis cases [1]. According to the American Academy of Periodontology, the severity of the condition can be categorized into slight, moderate, and severe. Strict criteria for these distinctions are not provided, but guidelines suggest a loss of clinical attachment of 1–2 mm, 3–4 mm, and ≥5 mm for each clinical category of severity, respectively [2]. The disease can also be described in terms of its extent as localized (≤30% of sites present involved) or generalized (>30% of sites present affected) [2]. Based on these criteria, the present case would be diagnosed as chronic, severe, generalized periodontitis.

Although the diagnosis of chronic periodontitis for cases such as the one presented here is straightforward, the determination of cases at the beginning of the disease process and the distinction between advanced generalized cases and aggressive forms of periodontitis is not always easy. The distinction between early periodontitis and advanced gingivitis is complicated by difficulties in determining early clinical attachment loss in the absence of radiographic evidence of alveolar bone loss, mainly in

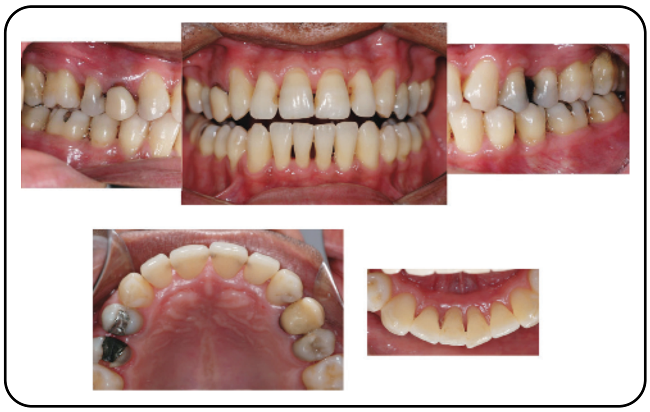

Figure 6: Clinical presentation of the case 1 year after therapy. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

Visit: 1 year post-therapy

Surface	Buccal																				
Tooth	2			3			4			5			6			7			8		
PD	6	2	3	4	3	3	5	1	3	2	2	2	2	2	3	3	1	2	2	1	2
CEJ-GM	0	-3	-3	-2	-2	-1	0	-2	-1	-1	-1	-1	1	0	0	-1	-1	-2	-1	-1	0
CAL	6	5	6	6	5	4	5	3	4	3	3	3	1	2	3	4	2	4	3	2	2
BOP	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Surface	Palatal																				
Tooth	2			3			4			5			6			7			8		
PD	5	2	5	4	2	4	4	2	4	3	2	3	2	3	2	3	2	2	3	2	3
CEJ-GM	1	0	2	2	-1	1	1	0	1	2	0	2	1	1	1	1	1	1	1	0	1
CAL	4	2	3	2	3	3	3	2	3	1	2	1	1	2	1	2	1	1	2	2	2
BOP	1	0	1	0	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0
Surface	Buccal																				
Tooth	31			30			29			28			27			26			25		
PD	3	2	3	4	1	1	2	1	2	1	1	2	2	1	1	1	1	1	1	1	2
CEJ-GM	2	0	1	0	-1	0	-1	-1	0	0	-2	1	0	0	-1	-1	0	-2	-2	0	-2
CAL	1	2	2	4	2	1	3	2	2	1	3	1	2	1	2	2	1	3	3	1	4
BOP	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Surface	Lingual																				
Tooth	31			30			29			28			27			26			25		
PD	3	2	5	5	3	4	3	2	3	3	1	2	3	1	3	2	2	2	2	1	2
CEJ-GM	3	2	2	1	0	1	2	3	2	2	0	2	2	0	0	-1	-2	-2	-2	-3	-3
CAL	0	0	3	4	3	3	1	-1	1	1	1	0	1	1	3	3	4	4	4	4	5
BOP	1	0	1	1	0	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0

Surface	Buccal																				
Tooth	9			10			11			12			13			14			15		
PD	2	1	2	2	1	2	2	1	2	2	1	3	3	2	4	4	2	5	5	3	4
CEJ-GM	-1	-1	-1	-1	-1	-1	0	0	0	0	-1	-2	-1	-1	-1	0	-2	0	-1	-1	0
CAL	3	2	3	3	2	3	2	1	2	2	2	5	4	3	5	4	4	5	6	4	4
BOP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	1	1	0	1
Surface	Palatal																				
Tooth	9			10			11			12			13			14			15		
PD	2	2	3	3	2	3	3	2	3	3	2	3	4	2	4	4	2	5	5	2	5
CEJ-GM	1	0	0	0	0	1	1	0	1	1	1	1	1	0	1	1	0	1	2	1	1
CAL	1	2	3	3	2	2	2	2	2	2	1	2	3	2	3	3	2	4	3	1	4
BOP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	1	0	0
Surface	Buccal																				
Tooth	24			23			22			21			20			19			18		
PD	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	2	2	1	2
CEJ-GM	-2	0	-2	-1	0	0	0	0	0	0	-1	0	-1	-2	-1	0	0	0	0	-1	0
CAL	4	1	4	2	1	1	1	1	1	1	2	1	2	3	2	2	1	2	2	2	2
BOP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1
Surface	Lingual																				
Tooth	24			23			22			21			20			19			18		
PD	2	1	2	2	1	2	2	1	2	2	2	3	4	2	3	3	2	4	4	2	2
CEJ-GM	-3	-3	-2	-2	-2	0	0	0	1	1	0	1	1	1	1	2	2	2	1	2	2
CAL	5	4	4	4	3	2	2	1	1	1	2	2	3	1	2	1	0	2	3	0	0
BOP	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	1	0	1	1	0	1

Figure 7: Periodontal chart, 1 year after periodontal therapy. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

areas where severe gingival inflammation causes hyperplasia of the gingival margin.

The issue is further complicated by the existence of areas of incidental attachment loss [3] not caused by the bacterial-induced inflammation characteristic of periodontitis. For instance, isolated sites of gingival recession caused by toothbrush trauma should not be confused as a sign of chronic periodontitis. These lesions are easily distinguishable from recession of the gingival margin as a consequence of chronic periodontitis on the basis of their clinical features. They involve primarily the buccal surface of teeth, with no loss of adjacent interproximal tissue; they are primarily associated with teeth with thin buccal soft tissues such as maxillary canines and premolars. The presence of these isolated lesions is not sufficient for the diagnosis of chronic periodontitis, even though they are associated with attachment and alveolar bone loss.

Other common examples of incidental attachment loss lesions are the bone loss associated with restorations invading the biologic width and the defects on the distal aspect of second molars caused by the malposition of unerupted or partially erupted third molars. The mesial tipping of teeth can also lead to a clinically deepened sulcus and a radiographic image suggestive of a vertical bone loss. This appearance is the consequence of the apical displacement of the mesial CEJ and should not lead to the erroneous diagnosis of chronic periodontitis. As one can see, there are several circumstances where the diagnosis of early chronic periodontitis can be complicated.

The distinction between chronic and aggressive periodontitis can also be difficult. The two conditions are differentiated based on rates of progression and the age of onset [4]. Chronic forms of periodontitis are characterized by a relatively slow progression of attachment and bone loss starting around 30 years of age, whereas, as the term implies, aggressive periodontitis shows a faster rate of tissue loss. However, clinicians rarely have the opportunity to measure rates of disease progression. Therefore, although both conditions can affect individuals of any age, chronological age remains an essential component of the differential diagnosis. If a younger individual presents with advanced attachment and bone loss, it is concluded that these subjects are presenting a faster rate of progression.

The classic form of localized aggressive periodontitis presents several clinical features that make it easily distinguishable from chronic periodontitis, such as age of onset, only firsts molars and incisors are affected by the disease, and an overall lack of clinical signs of inflammation and minimal amounts of gross plaque and calculus accumulation despite severe bone loss around the affected teeth [3]. The lesions associated with the first molars also tend to manifest as infrabony angular defects.

The treatment of chronic periodontitis as discussed in later chapters depends on the ability of the clinician to remove plaque and calculus from the root surfaces allowing for proper healing of the gingival tissues and on the capacity of the patient to perform proper plaque control. These are the cornerstones of periodontal therapy. Although the focus of this chapter is the diagnosis and not treatment of chronic periodontitis, the results obtained with anti-infective periodontal therapy will determine the long-term prognosis of the case and the need for additional treatment. Therefore, it is essential that clinicians examine the outcome of the initial therapy before any additional decisions regarding the case can be made, and this reevaluation could be considered part of the diagnostic process.

Studies examining the prognostic ability of periodontal clinical parameters have demonstrated that the presence of plaque, BOP, and suppuration have very low positive predictive values but very high negative predictive values [5,6]. This indicates that sites without clinical signs of inflammation are at very low risk for disease progression and might not require additional therapy. In addition, the accumulation of information regarding the clinical parameters for a given site over time increases the prognostic value of these parameters. Sites with constant BOP have a much higher chance of progression than sites that bleed sporadically [6]. It is also well established that the prognosis of chronic periodontitis directly depends on the patient's ability to control plaque accumulation. A longer time of follow-up will afford the clinician a better assessment of the patient's oral hygiene skills.

The presence of residual pockets after initial therapy, rather than the presence of deep pockets at the initial examination, is associated with an increased risk of future attachment loss. This information is readily available to periodontists and can add great insights into the long-term prognosis of the case. When clinicians are trying to assess the outcome of their initial periodontal therapy, another key piece of information is how much improvement one can

anticipate. In other words, what should be the realistic expectation of a therapist regarding the treatment outcome? This clearly will depend on the severity and extent of the periodontal condition at the beginning of the treatment. Several longitudinal studies have been conducted, and guidelines regarding the amount of pocket depth reduction and clinical attachment gains for each initial pocket depth are available and should be used to keep the outcome of treatment in perspective [7,8]. It is unrealistic, for instance, to expect a 9-mm pocket to convert to a 3-mm sulcus after SRP.

Self-Study Questions

A. What are the clinical features that differentiate incidental loss of attachment resulting from mechanical trauma from periodontitis-induced loss of attachment?

B. If a clinician cannot decide on the final diagnosis for the case (i.e., generalized (severe) chronic or generalized aggressive periodontitis), how should he or she proceed?

C. What are the possible therapeutic consequences of a differential diagnosis between generalized severe chronic periodontitis and generalized aggressive periodontitis?

D. If the initial outcome of a periodontal anti-infective therapy is below standards indicated by the literature, what should the therapist suspect and how should he or she proceed?

E. How should the periodontist proceed if, by the reexamination, the case has several residual pockets?

Answers located at the end of the chapter.

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TAKE-HOME POINTS

A. Incidental loss of attachment associated with mechanical trauma has distinct clinical features that make it clearly distinguishable from infection/inflammation-induced attachment loss. It tends to be circumscribed to buccal surfaces, particularly in areas of a very thin soft tissue, and the neighboring papilla present normal height. The tissue surrounding the recession has a very healthy appearance, and it is not uncommon to have toothbrush abrasion associated with the gingival recession. The collapse of gingival tissues on buccal surfaces can also be associated with marginal gingival inflammation, but in this case, the presence of plaque and calculus is not an uncommon finding and the tissues surrounding the gingival recession tend to shown clinical signs of inflammation.

B. As discussed earlier, the distinction between the two diagnoses can be difficult at times. The differences are typically determined by the age of onset, rate of progression, and the patterns of bone loss. In general, chronic periodontitis starts at age 30–35 and progresses in random bursts in different sites throughout one's life. If the disease presents in younger patients or can be documented to have occurred in a short period, it tends to be classified as aggressive. Both diseases can occur in a localized or generalized pattern. Familial aggregation is also one of the features of aggressive forms of periodontitis; therefore, if this diagnosis is suspected, the clinician should inquire about the periodontal status of close relatives or suggest they receive an oral examination. Finally, if the clinician cannot decide on a diagnosis, the safe assumption is to define the case as chronic periodontitis, due to the much higher prevalence of this condition compared with aggressive forms of the disease [9].

C. Although it is not the intent of this chapter to address therapy, which is discussed extensively in other chapters of the book, the relevance of the distinction between chronic and aggressive forms of periodontitis depends on the assumption that these two clinical forms of the disease would require specific types of treatment. However, recent literature has indicated that subjects with generalized aggressive periodontitis and generalized chronic periodontitis with comparable extent and severity of the conditions tend to respond similarly to periodontal therapy [10]. This would imply that the prognosis of the case would depend more on the extent and severity of the periodontal disease than on its precise diagnosis as chronic or aggressive.

D. In these circumstances, the clinician should rule out any systemic causes for the disease (risk factors such as smoking or uncontrolled diabetes, immunocompromise, endocrine disorders, or syndromes). The next thing to check is the efficiency of the patient's plaque control. This can be easily ascertained by the gingival and plaque indices. In the presence of large amounts of plaque, reinfection of pockets occurs fast and healing is compromised. Once the level of oral hygiene of the patient has been assessed, provided it was not the problem, the next steps will depend greatly on the level of periodontal training of the therapist. If the clinician is an experienced periodontist, the chances are that therapy was properly executed and there is very little room for improvement. The clinician might be dealing with a rare case of refractory periodontitis, where the subject does not respond to conventional therapy. The next step would be to consider adjunctive or complementary approaches such as systemic antibiotics and/or periodontal surgery. If we are dealing with a less experienced clinician, the fair assumption is there was failure in calculus removal to a level compatible with resolution of the periodontal condition. Particularly for severe generalized forms of the disease this should not be a surprise because subgingival debridement of deep pockets is not an easy task and requires highly skilled and well-trained individuals. Residual pockets should be carefully reinstrumented, and this step should involve as many sessions as needed. A new period of 3 months of healing should be given before an

assessment of the outcome of this new cycle of SRPs is conducted. Clinicians should keep in mind that there is very little risk to the patient in delaying a possible surgical phase of treatment and make every effort to guarantee an optimal outcome of the anti-infective therapy.

E. Although the presence of residual pockets has been demonstrated as a good predictor of future attachment loss [11], clinicians should interpret this information with caution. Several other clinical aspects will impact the prognosis of the case such as the level of plaque control by the subject, the presence of BOP, and the presence of furcation defects. Further, one must keep in mind that periodontal surgical procedures involve nonaffected periodontally diseased sites adjacent to residual pockets. If residual pockets are isolated nonbleeding lesions, they present a very low risk of progression and can be easily addressed with SRP during supportive periodontal therapy. Conversely, if residual pockets cluster around a few adjacent teeth and BOP is a recurrent finding over several sessions of maintenance, a surgical approach seems adequate. Periodontal surgery in the absence of proper plaque control exposes the periodontal patient to the risk of accelerated attachment loss [12] and should be avoided at all costs.

Case 7

Local Anatomic Factors Contributing to Periodontal Disease

CASE STORY

A 47-year-old female presented with a chief complaint of: "My gums around one of the lower right teeth hurts." The patient reported soreness and discomfort around tooth #30 from time to time, especially on the buccal side. On occasion, the patient experienced bleeding when brushing her teeth (Figures 1 and 2).

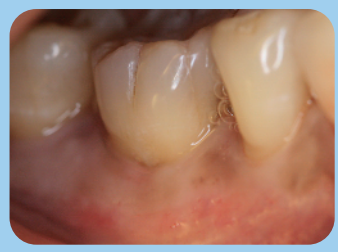

Figure 1: Clinical presentation of tooth #30.

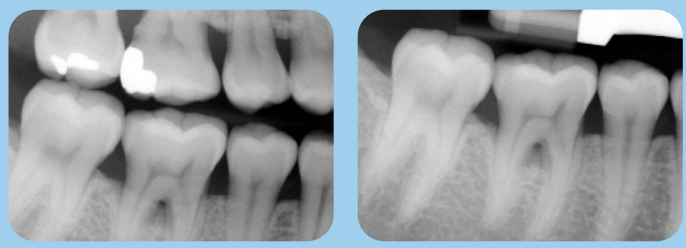

Figure 2: Radiographic presentation of tooth #30.

LEARNING GOALS AND OBJECTIVES

- To be able to identify local anatomic factors that may contribute to periodontal disease
- To understand the anatomy of the furcation and root
- To be able to diagnose a furcation invasion using a furcation classification system

Medical History

The patient's medical history was not significant, except for hypercholesterolemia that was controlled with simvastatin. The patient reported no allergies to any medication, metal, or food.

Review of Systems

- Vital signs
  - Blood pressure: 132/82 mm Hg
  - Pulse rate: 72 beats/minute
  - Respiration: 15 breaths/minute

Social History

The patient denied ever smoking, occasionally drank alcohol during social events, and denied the use of recreational drugs. The patient was happily married with a son and a daughter.

Oral Hygiene Status

The patient brushed twice a day and sometimes flossed.

Extraoral Examination

No significant findings were present.

Intraoral Examination

- Soft tissues including buccal mucosa, hard and soft palate, floor of the mouth, and tongue were all within normal limits.
- There was an adequate amount of attached keratinized gingiva present in general but with mild marginal erythema.
- Refer to Figure 3 for the periodontal charting.
- Tooth #30 exhibited a probing depth of 5 mm on the midbuccal aspect and clinically had grade II furcation invasion. No significant mobility was detected.
- A cervical enamel projection (CEP) was detected at the buccal furcation area below the gingival margin on #30.

Occlusion

No occlusal interferences were detected.

Radiographic Examination

A full-mouth radiographic series was taken. Periapical and bite-wing radiographs of tooth #30 showed evidence of furcation invasion (Figure 2).

Diagnosis

A diagnosis of localized severe chronic periodontitis was made based on the clinical and radiographic examinations. The attachment loss and grade II furcation invasion on tooth #30 required definitive periodontal treatment. The prognosis of #30 was questionable because the grade II furcation made the patient's daily oral hygiene maintenance in this area very difficult [1].

Treatment Plan

The treatment plan and sequence were as follows.

- Diagnostic phase: comprehensive dental and periodontal examination, radiographic examination

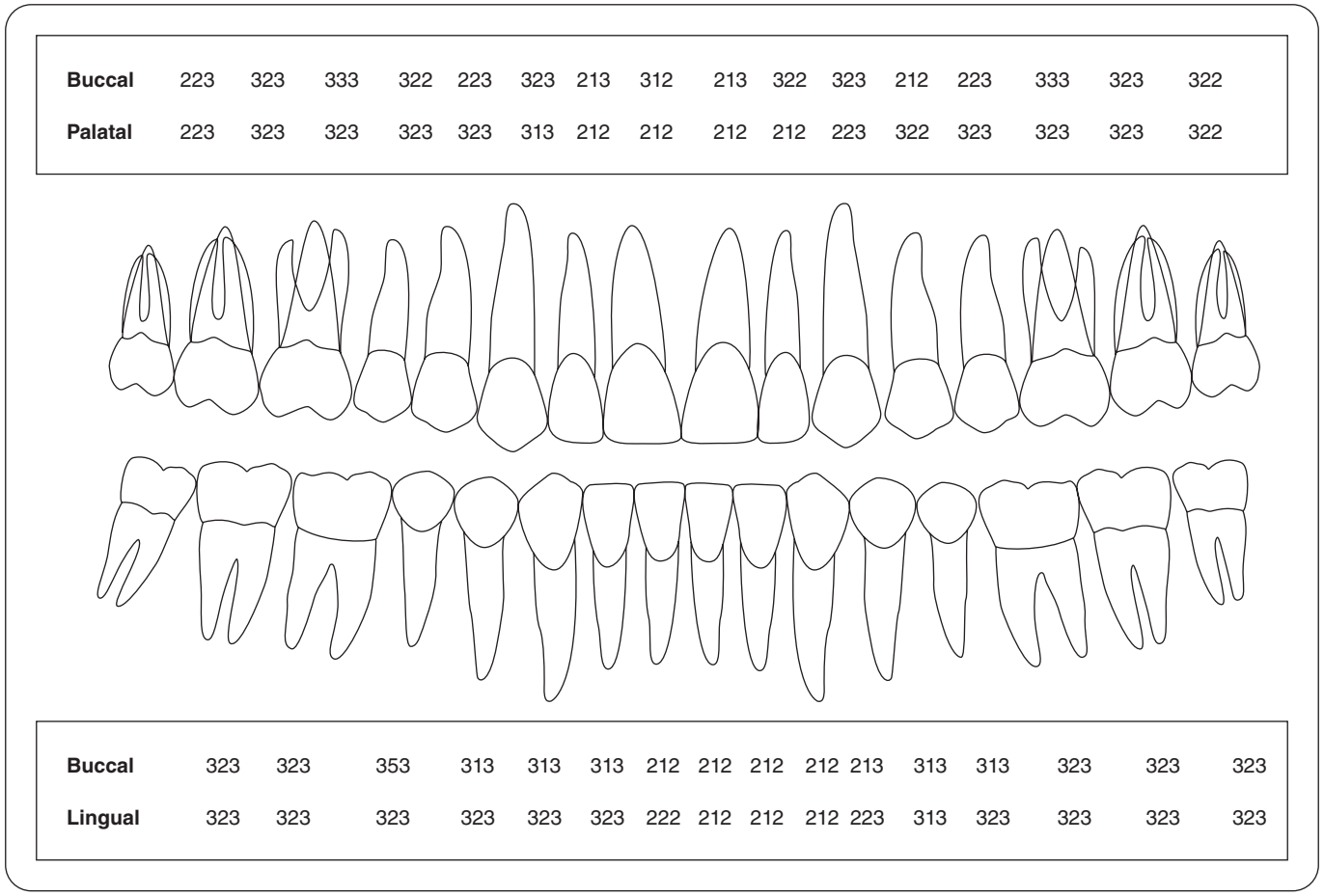

Figure 3: Periodontal probing depth measurements during initial visit.

- Disease control phase: oral hygiene instruction, adult prophylaxis, localized scaling, and root planing of the buccal furcation of tooth #30
- Reevaluation phase: periodontal reevaluation of tooth #30, oral hygiene evaluation and reinforcement
- Surgical phase: open flap debridement and removal of the CEP on the buccal aspect of tooth #30
- Maintenance phase: regular 3-month recall visits

Treatment

Localized scaling and root planing of tooth #30 was performed using a Cavitron and hand instruments. After a healing period of 6 weeks, periodontal reevaluation was done revealing a probing depth of 5 mm with bleeding on probing on the midbuccal of #30. The treatment plan at this point included surgical treatment to remove the CEP. An intrasulcular incision was made from the mesial line angle of #29 to distal line angle of #31 using a #15 blade. Full-thickness buccal and lingual flaps were raised to expose the furcation area of #30 to allow adequate visualization and to give access to remove the CEP. As shown in Figure 4, the CEP extended apically almost to the level of bone crest. A diamond burr was then used to remove the CEP completely (Figure 4), and the furca was debrided using a Cavitron and hand instruments. The flap was eventually sutured back to its original position. Postoperative instructions were given and the patient was seen 2 weeks afterward for a follow-up. Six months later, localized probing at tooth #30 showed a probing depth of 3 mm without bleeding on probing.

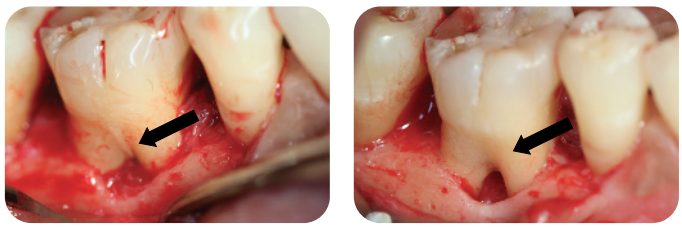

Figure 4: Cervical enamel projection (CEP) on buccal of #30 (left); CEP removed (right).

Discussion

The patient presented with a probing depth of 5 mm on the buccal furcation area of tooth #30 possibly due to the presence of a CEP that extended deep into the furcation. The presence of the CEP prevented proper soft tissue attachment at the furcal area, leading to the formation of a deep periodontal pocket. Bone loss at the furcal area is most likely due to the prolonged plaque accumulation in this periodontal pocket that subsequently led to chronic inflammation and hence attachment loss. By removing the CEP, enamel at furcation was eliminated to expose the underlying dentin, thereby allowing soft tissue attachment to occur over this area. In so doing, a periodontal pocket was eliminated. Note that a grade II furcation can also be treated with guided tissue regeneration or a bone graft (see Chapter 4, Case 3).

It is critical to identify all local etiologic factors because they may accelerate periodontal disease progression and affect the diagnosis, prognosis, and the treatment of the disease.

Self-Study Questions

A. What are some anatomic factors that may contribute to periodontal disease?

B. Describe the anatomy of a furcation and define furcation invasion.

C. Name different classification systems of furcation invasion.

D. How should you diagnose a furcation invasion?

Answers located at the end of the chapter.

ACKNOWLEDGMENT
We would like to thank Dr. David Yu for providing Figure 8.

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TAKE-HOME POINTS

A.

Proximal Contact Relation

Open interproximal contacts or uneven marginal ridge relations may encourage food impaction between the teeth. If proper oral hygiene is absent, food impaction can lead to inflammation, thereby potentially resulting in attachment loss in the interproximal area (Figure 5).

Root Proximity

Close root proximity between the two adjacent teeth will render oral hygiene difficult to maintain for both the patient and the dental professionals. Hence without good oral hygiene there can be loss of attachment between the two teeth (Figure 6).

Cervical Enamel Projections and Enamel Pearls

Cervical enamel projections (CEPs) refer to the extension of enamel to the furcal area of the root surface. CEPs may potentially predispose a furcation to attachment loss because they prevent connective tissue attachment at furcation. As such, a periodontal pocket may form, leading to plaque accumulation and possibly furcation invasion.

Most clinicians agree there is a correlation between CEPs and the incidence of furcation invasion. Masters and Hoskins reported that 90% of mandibular furcation invasions have CEPs [2]. Bissada and Abdelmalek reported a 50% correlation between CEPs and furcation invasion [3]. Swan and Hurt observed a statistically significant association between CEPs and furcation invasion [4].

In descending order of occurrence, CEPs are most commonly seen in mandibular second molars, maxillary second molars, mandibular first molars, and maxillary first molars. When CEPs are observed, they are usually seen on buccal aspects of molars [2] (Figure 7).

Enamel pearls are ectopic globules of enamel and sometimes pulpal tissue that often adhere to the cementoenamel junction (CEJ). They are present in roughly 2.7% of the molars and are mostly found on maxillary third and second molars [5]. Moskow and Canut suggested that enamel pearls may also predispose a furcation to attachment loss [5] (Figure 8).

Root Concavity

The furcal aspects of the roots frequently have concavities with a certain amount of depth (see Question B for details) that will encourage plaque

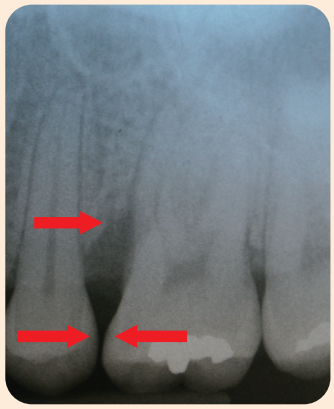

Figure 5: Interproximal open contact between #13 and #14 (indicated by the red arrows) and vertical bone loss on #14 mesial.

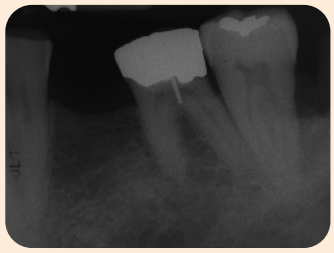

Figure 6: Close root proximity between #18 and #19.

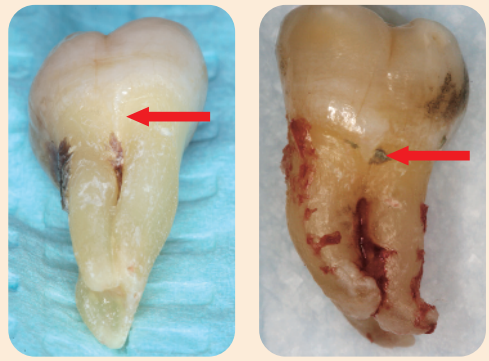

Figure 7: Cervical enamel projection (indicated by the red arrow).

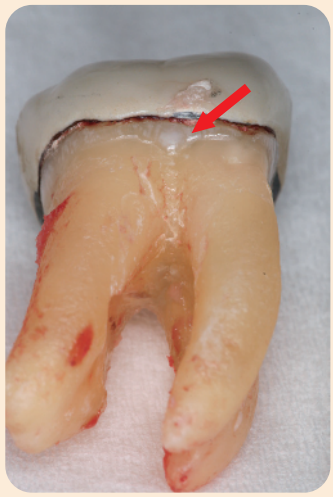

Figure 8: Enamel pearl (indicated by the red arrow).

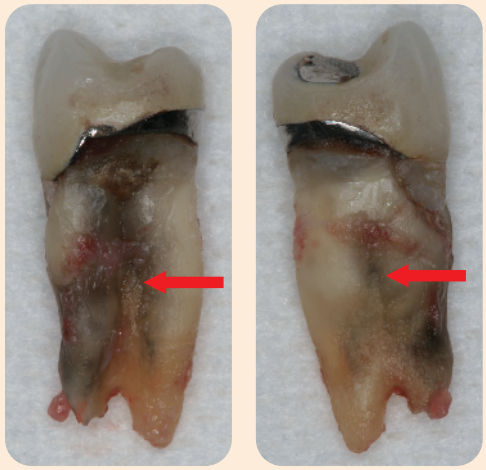

Figure 9: Mesial and distal root concavities of maxillary first premolar.

accumulation and prevent proper instrumentation of furcation. Hence a root concavity may predispose the furcation to attachment loss (Figure 9).

Size of Furcation Entrance

Approximately 80% of all furcation entrances are <1.0 mm in diameter with about 60% of them <0.75 mm [6]. Because frequently used curettes and scalers have a face width of 0.75–1.10 mm, it is unlikely that effective removal of accretions at furcation can be achieved by using these instruments alone. Hence a small furcation entrance may predispose a furcation to attachment loss (Figure 10).

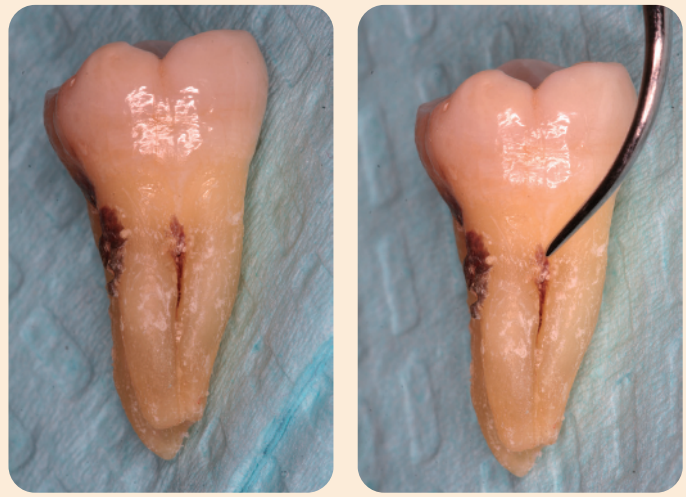

Figure 10: The size of the Cavitron tip is too big to enter the furcated area, rendering scaling and root planing in this area very difficult.

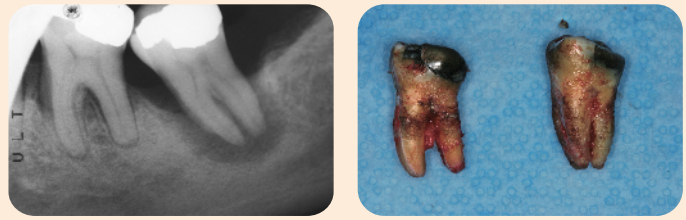

Figure 11: The root divergence of #19 is more prominent than #17.

Root Divergence and Root Fusion

The degree of root divergence in a multirooted tooth will influence the ability of the patient and dental professionals to control plaque level. Diverging roots allow easier instrumentation to the furcation area, whereas converging roots (e.g., root fusion) render the access to the furcation area very difficult, resulting in poor plaque control and possible attachment loss (Figure 11).

Root Trunk Length

The length of root trunk affects attachment loss. The longer a given root trunk, the less likely a furcation will be predisposed to attachment loss.

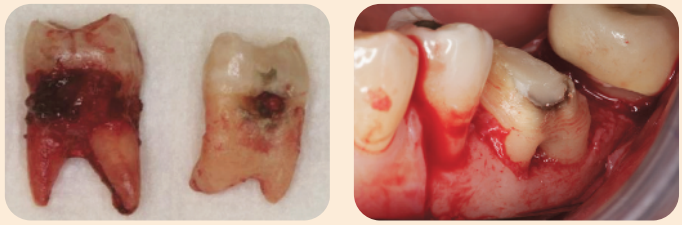

Figure 12: Long root trunk length (left) and short root trunk at #19 (right).

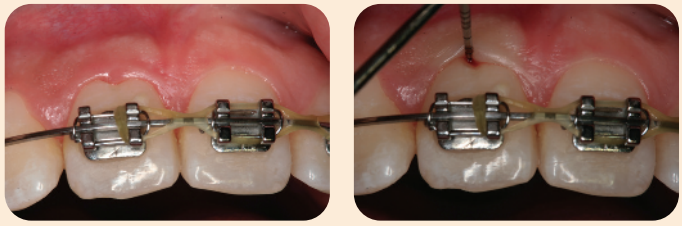

Figure 13: Buccal radicular groove present on #8 as indicated by the probe tip.

Teeth with Taurodontism usually have apically displaced furcation and longer root trunk length [7] (Figure 12).

Intermediate Bifurcation Ridge

Intermediate bifurcation ridges are ridges spanning across the bifurcation of mandibular molars in the mesiodistal direction. These ridges are present in 70–77% of the mandibular molars [8,9]. Just like other anatomic structures, the presence of an intermediate bifurcation ridge may hinder effective plaque control and root preparation by both the patient and dentist.

Buccal Radicular Groove and Palato-Gingival Groove

Buccal radicular grooves and palato-gingival grooves are developmental phenomena that affect mainly the maxillary anterior teeth [10,11]. These grooves run on the roots in the coronal-apical direction. Due to their anatomy, the grooves frequently provide a plaque-retentive area that is very difficult to instrument, making teeth with these developmental grooves more prone to attachment loss (Figure 13).

Accessory Pulpal Canals

Accessory pulpal canals are small endodontic canals branching off from the main root canal that

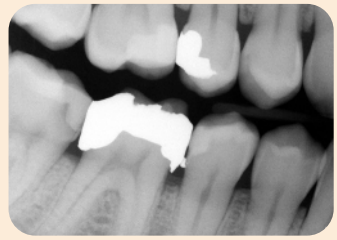

Figure 14: Overhangs on the mesial and distal of #30 that may eventually lead to bone loss on the mesial and distal of #30.

may furnish a communication between the pulpal chamber and the periodontal ligament. These accessory canals are usually located near the root apex; however, they can also be found anywhere along the root, including the furcation area. There is a theory that some periodontal infections can originate from endodontic sources, traveling through accessory/lateral canals located in the furcation areas. In these cases there is periodontal involvement in the furcation, but the infection originated in the pulp. Although still controversial, it has been proposed that periodontal disease can result from pulpal infection. An endodontic infection may be present at the furcation area when the infection travels through accessory canals that end at the furca. Vertucci and Williams reported that accessory canals at furcations are present in 46% of human lower first molars [12]. Burch and Hulen observed accessory canals in 76% of maxillary and mandibular molars [9].

Restorative Considerations

Dental restorations with overhangs or open margins are plaque-retentive areas that may result in gingival inflammation and attachment loss. Restorative margins are most compatible with the periodontium when located either supragingivally or at the level of gingival margin. Should the restorative margin violate the biologic width, the resulting inflammatory process may lead to gingival recession, bone loss, and the exposure of the restorative margin. The restorative contour (e.g., crown contour) should follow the root surface contour rather than accentuating the cervical bulge to support periodontal health. In the case of bridges, the design of the pontic can affect its ability to be cleaned and hence the periodontal health of the teeth (Figure 14).

B. A furcation is an anatomic area where the roots of a multirooted tooth start to diverge. Mandibular molars and maxillary first premolars are bifurcated because they each have two roots. Maxillary molars are trifurcated because they each have three roots.

A furcation consists of two parts: (1) root separation area: the area where alveolar bone begins to separate the roots and (2) fluting area: the part of the root that is directly coronal to the root separation area.

There are often concavities in the furcal side of the roots. In mandibular molars, all the mesial roots have concavities on the furcal side with each concavity averaging 0.7 mm in depth [13]. Likewise, 99% of the distal roots of mandibular molars have concavities on the furcal side with an average depth of 0.5 mm [13]. The root trunk, which is the distance from the CEJ to the level of root separation, is about 4.0 ± 0.7 mm in mandibular first molars [13,14].

In maxillary molars, 94% of the mesio-buccal roots have concavities on the furcal side, with each concavity averaging 0.3 mm in depth [15]. Roughly a third (31%) of the mesio-distal roots and a quarter (17%) of the palatal roots have concavities, and each concavity is about 0.1 mm in depth [15]. The length of root trunks of maxillary molars is 3.6 mm, 4.2 mm, and 4.8 mm on the mesial, buccal, and distal surfaces, respectively [16,17].

All bifurcated maxillary first premolars have a mesial and distal root trunk of about 8 mm. In addition, almost all the buccal roots have "developmental depressions" also known as "buccal furcation groove" present at the 9.4-mm level on the furcal side [18,19].

Furcation invasion is defined as a loss of attachment within a furcation. When there is a loss of clinical attachment, the presence of concavities on these roots at furcation will hinder effective plaque control at these areas.

C. There are a number of different classification systems of furcation invasion. The three most commonly used systems are as follows.

1. The Glickman classification [20] describes both the vertical and horizontal components of the furcation invasion:
   - Grade I: pocket formation into the fluting area but with intact interradicular bone
   - Grade II: pocket formation into the root separation area with interradicular bone loss that is not completely through to the opposite side of the furcation
   - Grade III: same as grade II but with a through-and-through interradicular bone loss (the soft tissue still covers part of the entrance of the furcation)
   - Grade IV: same as grade III but with gingival recession making furcation clinically visible
2. The Hamp et al classification [21] describes the horizontal component of the furcation invasion.
   - Degree I: horizontal bone loss going into the furcation <3 mm
   - Degree II: horizontal bone loss going into the furcation >3 mm but not to the opposite side
   - Degree III: a through-and-through horizontal bone loss in the furcation
3. The Tarnow and Fletcher classification [22] describes the vertical component of the furcation invasion.
   - Subclass A: vertical attachment loss 0–3 mm in furcation
   - Subclass B: vertical attachment loss of 4–6 mm in furcation
   - Subclass A: vertical attachment loss of >7 mm

D. The most effective way to diagnose a furcation invasion is to use a combination of clinical examination and radiographic evaluation. The clinical examination involves using periodontal and furcation probes to detect the furcation invasion.

Radiographs must be taken with a paralleling technique to minimize the distortion of the images. Note that radiographically the palatal root of maxillary molars may leave a grade III furcation invasion undetected due to the overlapping of the palatal root with mesio-buccal and distobuccal roots. In addition, the presence of a furcation arrow (a triangular show seen either at the mesial or distal roots in the interproximal area on maxillary molars) may possibly suggest the presence of

grade II–III furcation invasion on maxillary molars [23] (Figure 15). The more extensive a given furcation invasion, the higher the likelihood of observing the furcation arrow. However, it must be noted that the absence of furcation arrow does not necessarily suggest the absence of a furcation invasion.

Generally interproximal surfaces of the maxillary molars are more prone to furcation invasion than buccal surfaces [24].

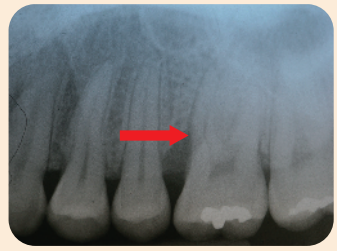

Figure 15: Furcation arrow as indicated by the red arrow is showing furcal involvement on the mesial of #14 radiographically.