Chapter

Diagnosis and Treatment Plan for Gingival Diseases and Conditions

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Abstract

The prevalence of gingival and periodontal disease is manifold and has not been highlighted much due to its asymptomatic and milder symptoms. It is usually given its due importance when the gingival disease progresses to advanced periodontal disease, displays symptoms of dull pain and tooth mobility, and is associated with pus discharge. The starting point of periodontal disease is usually gingival disease which is a reversible condition. It is therefore necessary to diagnose gingival diseases at an early stage to prevent its progression to irreversible periodontal disease. The diagnosis of gingival disease becomes cumbersome due to its similarity in the presentation of signs and symptoms. Gingival diseases can occur due to microbial attack from the plaque biofilm which is usually bacterial in nature. There are other viral, fungal, and immune-mediated mechanisms which can result in gingival diseases. Some systemic conditions also influence the gingiva which allows for diagnosing systemic diseases and treating these conditions appropriately. It is said that oral cavity is the mirror of the body, and in that sense the gingiva is the biggest surface where any changes or manifestations could be observed.

Keywords: gingivitis, gingival disease, diagnosis, treatment

1. Introduction

The gingiva or commonly referred to as gums surround and protect the teeth (Figure 1). Gingival diseases by namesake denote to the diseases affecting the gingival tissues. These diseases have burdened the human race since the early civilization, and this is proof enough to gauge the importance of diagnosing gingival diseases and treating them. Gingival disease if left untreated can progress to periodontal tissues and result in periodontal disease which is easier to diagnose probably due to its chronic and severe nature as compared to gingival disease. No wonder periodontal disease has been mentioned in the literature of ancient Egypt and a step toward preventing it by means of oral hygiene practices deserves its mention in the ancient scriptures [1].

2. Gingival disease terminology

The gingival disease terminology and classification has undergone many changes, and the current classification given at the World Workshop in 2017
classifies gingival condition in health and disease under three broad categories of health, dental biofilm-induced gingivitis, and non-dental biofilm-induced gingival disease [3] (Table 1).

| Periodontal health and gingival health | Dental biofilm-induced gingivitis | Non-dental biofilm-induced gingival disease |
|---------------------------------------|----------------------------------|-------------------------------------------|
| Clinical gingival health on an intact periodontium | Clinical gingival health on a reduced periodontium | Associated only with dental biofilm | Mediated by systemic or local risk factors | Drug-influenced gingival enlargement | Genetic/development disorders |
| Stable periodontitis | Non-periodontitis | Specific infections and inflammatory and immune conditions | | | |
| | | Reactive processes | | | |
| | | Neoplasms | | | |
| | | Endocrine, nutritional, and metabolic diseases | | | |
| | | Traumatic lesions | | | |
| | | Gingival pigmentation | | | |

Table 1. Classification of periodontal health, gingival disease, and condition [3].
2.1 Diagnosis of plaque-induced gingivitis

Gingivitis per se refers to the inflammation of the gingival tissues and is labeled with different diagnostic terms based on the etiology and clinical presentation to aid in formulation of the best-suited treatment. As mentioned above, the broad etiologic factors which result in gingival disease is the dental biofilm, which contain microbes, causing a microbial attack on the gingiva resulting in a dysbiosis amounting to a host response manifested in the form of the inflammatory disease called plaque-induced gingivitis. The plaque microbes have an influence on the gingiva depending upon its quantity and quality of pathogens present. Although the increased plaque burden is almost always associated with gingivitis, there are instances where paucity of plaque can again result in gingivitis due to the effect of modifying factors which make the host response more accentuated and exaggerated as they tend to have a more systemic affect than a local one [2, 4]. These modifying factors include few systemic conditions, factors which increase plaque accumulation and influence of drugs on gingiva. How these factors can affect gingivitis is summarized in Table 2.

| Factor | Effect on gingiva | Signs and symptoms for diagnosis | Diagnosis | Treatment [5] |
|--------|-------------------|---------------------------------|-----------|---------------|
| Bacterial dental biofilm only | Microbial attack mounts a host response in the form of inflammation | Mild redness with or without broken line of bleeding | Incipient gingivitis | OHI |
| | | | Mild changes in color and texture of the gingiva | Mild gingivitis | OHI+/OP |
| | | Glazing redness, edema, enlargement, bleeding on probing | Moderate gingivitis | OHI+OP |
| | | | Overt redness and edema and bleeding on palpation rather on probing | Severe gingivitis |

Potential modifying factors of plaque-induced gingivitis

**Systemic conditions**

| Sex steroid hormones (estrogen and progesterone) | Exaggerate the host inflammatory response in the presence of minimal plaque | Bleeding on probing or bleeding with toothbrushing, mild to moderate redness | Diagnostic term not given as not seen frequently in population and if present can be diagnosed as gingivitis associated with puberty | OHI + OP |

(1) Puberty

(2) Menstrual cycle

Mild redness, edema based on severity of inflammation seen during the menstrual cycle | Diagnostic term not given as not seen frequently in population and if present can be diagnosed as gingivitis | OHI + OP |
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| Factor             | Effect on gingiva                                                                 | Signs and symptoms for diagnosis                                      | Diagnosis                              | Treatment [5]                               |
|--------------------|----------------------------------------------------------------------------------|-----------------------------------------------------------------------|----------------------------------------|--------------------------------------------|
| (3) Pregnancy      | The hormones exaggerate the host inflammatory response in the presence of minimal plaque | Deep gingival probing depths, bleeding on probing or bleeding with toothbrushing, and elevated gingival crevicular fluid flow in pregnancy | Pregnancy-associated gingivitis       |                                             |
| (4) Oral contraceptives | The high-dose hormones in the pills exaggerate the host inflammatory response in the presence of minimal plaque; low dose does not have much effect | Mild redness, edema based on severity of inflammation seen after 1 to 3 months of use | Currently the dose of oral contraceptives is low; hence diagnostic terms have been removed | OHI + OP + reduction of high-dose oral contraceptive Low-dose contraceptive does not require any change |
| Hyperglycemia      | High blood glucose levels increase the pathogenic bacteria and also form more AGE which affect collagen turnover and healing | Signs of inflammation of gingivitis + high blood glucose levels | Gingivitis associated with diabetes mellitus | OHI + OP + maintenance of blood glucose levels by diet restriction/exercise/medication |
| Leukemia           | Increases number of WBCs which accumulate in the gingival tissues and decreases number of platelets which causes bleeding | Cervical lymphadenopathy, petechiae, ulcers seen in the mucosa, bleeding on slight provocation, swollen, glazed, spongy gingiva, red to deep purple color of gingival lesions | Gingivitis associated with acute/chronic leukemia | Treat leukemia + symptomatic treatment for gingivitis with careful OHI and OP to prevent excessive bleeding |
| Smoking            | Direct smoking can cause vasoconstriction of gingival vasculature                 | No redness, edema, or swelling present. Color may change to blue and pale pink. No gingival changes and pocket depths increase when lesions progress to periodontitis | No gingivitis                          | Smoking cessation                          |
| Malnutrition       | Deficiency of vitamin C affects crosslinking of collagen                          | Bleeding on probing, mobility, and swollen gums in severe cases with minimal plaque | Scurvy                                 | Vitamin C supplementation + OHI + OP      |

Oral factors enhancing plaque accumulation
2.2 Tools used for gingival diagnosis

The crude tools used are a questionnaire/interview to collect important aspects of the patient demographics, medical history, current medications, and habits. The next step involves patient examination starting from extraoral structures to any abnormal intraoral findings to specific examination of the gingiva. The gingival disease is visually examined for clinical signs and symptoms using a mouth mirror under ambient lighting of the dental chair, cotton/gauze to dry the tissues, and sometimes the use of three-way air water syringe to wash way the debris for better inspection. Changes in color, contour, consistency, texture, size, position, etc. are

| Factor | Effect on gingiva | Signs and symptoms for diagnosis | Diagnosis | Treatment [5] |
|--------|------------------|-----------------------------------|-----------|---------------|
| Prominent subgingival restoration margins | Roughness and closeness of these restorations to gingival tissue cause accumulation of plaque bacteria and irritation | Localized mild redness, bleeding on probing, slight edema in area of restoration | Gingivitis due to faulty restoration | Correction of restoration + OHI + SRP |
| Hyposalivation | Decreased saliva causes sticking of bacteria on tooth surfaces | Dental caries, taste changes, halitosis, mucosal and gingival dryness, and gingival inflammation | Gingivitis associated with hyposalivation | OHI + OP + salivary substitutes |

**Drug-influenced gingival enlargements**

- **Phenytoin, sodium valproate**
  - Drugs and plaque cause fibroblasts to increase production of collagen and extracellular connective tissue
  - Onset after 3 months of drug intake, common in anterior gingiva, gingival size increases which starts from interdental papilla and may extend to the margin and attached gingiva in severe cases. The enlarged areas are firm to soft depending upon the presence of gingival inflammation
  - Drug-influenced mild gingival enlargement (if only papilla is involved)
  - Drug-influenced mild gingival enlargement (if papilla and margin is involved)
  - Drug-influenced mild gingival enlargement (if papilla, margin, and attached gingiva is involved)
  - OHI + OP + drug substitution if required, followed by gingivectomy to correct enlarged gingival tissues

- **Nifedipine, amlodipine, verapamil, diltiazem, felodipine**

- **Cyclosporine**

_OHI, oral hygiene instruction, OP, oral prophylaxis._

**Table 2.**

*Diagnosis based on etiology, modifying factors, and clinical features [2, 4].*
| Advanced diagnostic aid for gingival disease | Mechanism/working | Inference |
|-------------------------------------------|------------------|-----------|
| Periotemp probe                           | Detects the difference in subgingival temperature which is reflected by red or green light | Red light indicates future periodontal breakdown and increase in periopathogens |
| New generation of periodontal probes      | First-generation | Detects pocket depth using traditional probes |
|                                           | Second-generation | Pressure-sensitive probe with uniform pressure |
|                                           | Third-generation | Pressure-sensitive and captures data on computer |
|                                           | Fourth-generation | Uses 3D technology to detect pocket |
|                                           | Fifth-generation | Uses 3D technology and ultrasound to detect pocket |

**Advances in radiography**

- Use of charged-coupled device, complementary metal oxide semiconductor, and cone beam-computed tomography allow digital recording
- These are used to detect bone loss and bone defects in 2D and 3D for periodontal defects rather than gingival diseases

**Advances in microbial culturing**

- High-performance liquid chromatography
- Can detect bacterial cell wall components
- Flow cytometry
- Can detect various bacteria
- Latex agglutination test
- Can detect pathogenic antigen, proteins, and antibody by agglutination reaction
- Direct and indirect immunofluorescence
- Can detect pathogenic antigen, proteins, and antibody by agglutination and adding fluorescent dyes
- Enzyme-linked immunosorbent assay
- Evalusite can detect *P. gingivalis*, *P. intermedia*, and *A. actinomycetemcomitans*
- Nucleic acid and DNA checkerboard hybridization techniques
- Detects microbes based on matching of unknown sample with known hybridization technique of nuclei acid/DNA
- DNA probe
- Omnigene can detect *P. gingivalis*, *P. intermedia*, *A. actinomycetemcomitans*, *E. corrodens*, *C. rectus*, *F. nucleatum*
- Perioscan uses BANA (N-benzoyl-DL arginine naphthylamide) hydrolysis carried out by trypsin-like protease
- Detects trypsin-like protease releasing bacteria, such as *P. gingivalis*, *T. denticola*, and *T. forsythus*
- IAI Pado Test 4.5 RNA probe test kit uses oligonucleotide probes complementary to conserve fragments of the 16S rRNA gene that encodes the rRNA
- Detects *A. actinomycetemcomitans*, *P. gingivalis*, *Tannerella forsythia*, and *T. denticola*
- MyPerioPath is a DNA test and uses saliva samples
- To identify the type and concentration of periodontal bacteria
- Perio-Check
- Detects neutral proteases like collagenases in GCF (gingival crevicular fluid)
noted. This is followed by palpation of the gingiva for any spontaneous bleeding, pain, discharge, blanching, consistency (by checking the resiliency of tissues on applying pressure), and pitting edema. The UNC-15 or the Michigan O periodontal probe with William’s marking is used to check for bleeding on probing, subgingival faulty restorative margins, and the presence of deeper than 5-mm pockets which is the critical probing depth to differentiate between gingivitis and periodontitis. Apart from these traditional tools used, advanced diagnostic aids have been introduced to further confirm the presence of gingival disease (Table 3) [5, 6].

2.3 Diagnosis of non-plaque-induced gingival diseases

Apart from plaque-induced gingivitis, it is imperative to diagnose and differentiate the non-plaque-induced gingival diseases and conditions to provide appropriate treatment and to avoid overtreatment. The etiology of non-plaque-associated gingival disease is usually related to some genetic defect or systemic disorder. In many instances the oral lesions precede the extraoral findings and can help in diagnosing a disease which could affect the full body. Therefore, while diagnosing these conditions, we need to look for other associated conditions to arrive at a correct diagnosis. Table 4 attempts to highlight the clinical features to help arrive at a diagnosis [7–11].
| C       | Cr     | Cs        | T             | S       | P       | L                      | Lab & H/P                                                                 | Add Sym                                         | D                      | Rx                        |
|---------|--------|-----------|---------------|---------|---------|------------------------|---------------------------------------------------------------------------|------------------------------------------------|------------------------|--------------------------|
| G       | Flat or rounded | Firm and resilient | Loss of stippling + | ++      | Coronal to CEJ | Gingival enlargement | Excisional biopsy shows fibrous connective tissue | Hereditary gingival fibromatosis | Gingivectomy to contour the topography + OHI |
| P-R/B-Br | Blunted | Soft and friable | Ulcerative + | ---     | Varies from papillary destruction to beyond mucogingival junction | Gingival ulceration | Bacterial culture for various bacteria types such as *Treponema*, *Selenomonas*, *Fusobacterium*, and *Prevotella intermedia*. | Loss of taste, woody sensation in teeth and feeling of extruded teeth accompanied with underlying risk factors such as poor oral hygiene and systemic conditions | Necrotizing periodontal disease | Debridement of local factors + CHX+ amoxicillin and metronidazole |
| FR/W    | No change | Soft and edematous | Ulcerative/white pseudomembranous + | No change | Erythematous | Bacterial culture for *Neisseria gonorrhoeae* | Pharyngitis and lymphadenopathy. Other sites: urethra, anus, cervix, oral mucosa | Gonorrhea | Systemic antibiotic therapy |
| FR      | No change | Edematous | Loss of stippling and ulceration with whitish membrane + | No change | Chancre (rare) | Bacterial culture for *Treponema pallidum*, followed by serologic reaction tests | Genital and skin lesions | Syphilis | Systemic antibiotic therapy |
| R-Gy patches | No change | Firm | Nodular/papillary proliferation + | No change | Nodular/papillary proliferation | Positive delayed hypersensitivity (tuberculin) skin reaction to purified protein derivative (ppd), isolation of mycobacterial antigen from bacterial cultures, and demonstration of acid-fast mycobacteria in clinical specimens. H/P: characteristic multinucleated giant cells | Commonly associated with lung infections. Involves floor of the mouth, extraction sites, and lymph nodes | Tuberculosis | Regimens of multiple antibiotics like isoniazid, rifampicin, pyrazinamide, or ethambutol |
| Dim | C | Cr | Cs | T | S | P | L | Lab & H/P | Add Sym | D | Rx |
|-----|---|----|----|---|---|---|---|----------|---------|---|----|
| RP  | Rounded | Soft | Erythematous patch | – | Blunted papilla sometimes | Painful ulcers after vesicle rupture | Culture for streptococcal strains. Biopsy | Upper respiratory infections | Skin lesions, low-grade fever | Hand, foot and mouth disease | Supportive treatment to correct fever and pain |
| RP  | No change | Soft and ulcerative | Small vesicles/fibrinous coated ulcer | + | Coronal or apical to CEJ | Lymphadenitis, fever, malaise | Primary herpetic gingivostomatitis | Acyclovir and aspirin/paracetamol, fluids. Dyclonine hydrochloride 0.5% for anesthesia |
| RP  | Flat and rounded | Soft and edematous | Ulcerated, loss of stippling | + | Attached gingival and hard palate | Rarely required. If needed fluorescent staining is more sensitive. HSV isolation of a virus in tissue. Culture is the most positive method of identification. Scraping made from the base of the lesion and stained with giemsa. H/P: Wright’s or Papanicolaou stain and shows syncytium and ballooning. Degeneration of the nucleus | Fever | Recurrent intraoral herpes simplex | Acyclovir and aspirin/paracetamol, fluids. Dyclonine hydrochloride 0.5% for anesthesia |
| BR  | No change | Soft | Vesicular | +/− | Diffuse erythema and isolated small vesicles that rupture quickly leaving ulcerations | Lesions on skin and mucosa | Fluorescent-antibody staining of smears using fluorescein-conjugated monoclonal antibodies is more reliable than routine cytology | Fever, malaise, and skin rash | Chicken pox (Varicella) | Acyclovir/valacyclovir for healing and reducing acute pain. Systemic corticosteroids to prevent postherpetic neuralgia, combination of |

- **RP** Rounded
- **Cr** Culture
- **Cs** Clinical
- **T** Test
- **S** Stains
- **P** Physical
- **L** Lab
- **Lab & H/P** Laboratory & Histology
- **Add Sym** Additional Symptoms
- **D** Diagnosis
- **Rx** Treatment
| C | Cr | Cs | T | S | P | L | Lab & H/P | Add Sym | D | Rx |
|---|----|----|---|---|---|---|-----------|---------|---|----|
| R patches +W halo | Blunt or rounded | Soft and friable | Ulcerated | -- | Unilateral vesicles which rupture | Necrosis of periodontium and alveolar bone | Culture | Skin lesion | Shingles (herpes zoster) | Oral acyclovir 800 mg five times a day, famciclovir 500 mg three times a day, or valacyclovir 500 mg three times a day |
| Pi | No change | Soft | Papules | ++ | Raised nodular or popular lesions | Mucosal lesions are rare | Discrete papules on skin of face and trunk and in genital areas | Molluscum contagiosum virus | Cryotherapy/laser |
| G | No change | Firm | Exophytic and verrucous | ++ | Exophytic papillomatous, verrucous or flat lesions | Squamous cell papilloma, condyloma acuminatum, verruca vulgaris, focal epithelial hyperplasia | Surgical removal, laser ablation, cryotherapy, and topical application of keratolytic agents. For smaller lesions, topical application of 25% podophyllum resin to reduce the size. Intralesional injection of interferon-α 1,000,000 iu/cm² once weekly and subcutaneous injections 3,000,000 iu/cm² twice weekly |
| C     | Cr | Cs | T                        | S          | P                   | L                          | Lab & H/P                                                                 | Add Sym                                                                 | D                          | Rx                                |
|-------|----|----|--------------------------|------------|---------------------|-----------------------------|--------------------------------------------------------------------------|-------------------------------------------------------------------------|-----------------------------|-----------------------------------|
| W-R   | No change | Soft and resilient | Scrapable lesion | +/-        |                     | Pseudomembrane/ erythematous/plaque-like/ nodular                        | H/P: culture of infected tissues or exudates on Sabouraud’s dextrose agar or other appropriate media | Oral involvement is secondary to serious systemic infection | Candidiasis               | Topical antifungal medications, nystatin, and amphotericin b |
| BR    | Rounded | Soft and friable | Chronic vegetating painful ulcer | ++        |                     | Nodular, papillary, or granulomatous lesions                           | Biopsy of infected tissue shows small oval yeasts within macrophages and reticuloendothelial cells as well as chronic granulomas, epitheloid cells, giant cells, and occasionally caseation necrosis | Caviation of the lung and dissemination of the organism to the liver, spleen, adrenal glands, and meninges | Histoplasmosis | Ketoconazole or itraconazole for 6–12 months |
| RP    | Violaceous marginal gingiva in early stage | Soft and friable | Necrosis and covered with pseudomembrane in advanced cases | -- | Lesions are necrotic and covered by pseudomembrane | | | Systemic involvement is present. Late stage involves destruction of alveolar bone and facial muscles | Aspergillosis | Systemic antifungals |
| R+ W  | Normal | Soft | Lichenoid reaction | No change | | Lichenoid-like reaction | Patch test by placing aluminum disk with known allergens for 48 hours on hairless skin and wait for any inflammation as a positive test. H/P: chronic inflammatory reaction with lichenoid infiltration of lymphocytes | Contact allergy | Topical corticosteroids |
| C     | Cr   | Cs   | T                        | S     | P                             | L                                    | Lab & H/P                                           | Add Sym                                      | D                                   | Rx                          |
|-------|------|------|--------------------------|-------|-------------------------------|---------------------------------------|---------------------------------------------------|---------------------------------------------|---------------------------------------------|----------------------------------------------|
| R     |      |      | Velvety texture          | +     | Seen in anterior maxillary gingiva | Plasma cells in lamina propria         | Plasma cell gingivitis                         | Topical corticosteroids                    |                               |                               |
| R-W   | Soft and friable | Smooth or disrupted | — |                          | Round lesion with central red area or pale pink surrounded by red periphery | Biopsy an epidermal pattern characterized by lichenoid vasculitis and intraepidermal vesicles and a dermal pattern characterized by lymphocytic vasculitis and subepidermal vesiculation | Skin lesions symmetrically present on distal extremities and moving proximally Hand, face, elbow and knees | Erythema multiforme                      | Anesthetic mouthwash, corticosteroids in severe cases, and acyclovir if associated with HSV |
| RP-W  | Normal | Soft and friable | Smooth and loss of stippling | No change | Lesions on free and attached gingiva | Desquamative gingivitis with vesiculobullous lesions which rupture | ELISA to detect circulating antibody to desmoglein in 1 and 3. Histopathology suprabasilar acantholysis may be observed | Bullous lesions on skin                      | Pemphigus vulgaris                      | Prednisolone usually given in dosages of 1–2 mg/kg/d and later – – |
| R area | Normal | Soft | Smooth and loss of stippling | — | Positive Nikolsky sign: rubbing the gingiva forms bulla | Desquamative lesions with bulla formation | Histopathology: circulating antibodies not always found by indirect immunofluorescence | Scarring in ocular lesions                   | Pemphigoid                              | Systemic corticosteroids                  |
| R-W streaks | Normal | Soft and resilient | Smooth and ulcerative | No change | Papular, reticular, plaque type or bullous lesions | Hyperkeratosis and saw tooth-shaped rete pegs | Skin lesions                                  | Lichen planus                            | Topical corticosteroids or intrasional steroids like 0.05% fluocinonide (Lidex) and 0.05% clobetasol (temovate) |
| R and W striae | Smooth and ulcerative | —/+ |                            |       | Central atrophic area with small white dots surrounded by white striae | Hyperorthokeratosis with keratotic plugs, atrophy of the rete ridges, and liquefactive | Red butterfly-shaped photosensitive, scaly, macules on | Lupus erythematosus                      | Systemic immunosuppressant and protection from sunlight |
| C    | Cr | Cs | T    | S   | P | L                     | Lab & H/P                                                                 | Add Sym                                                                 | D                      | Rx                        |
|------|----|----|------|-----|---|-----------------------|--------------------------------------------------------------------------|------------------------------------------------------------------------|------------------------|---------------------------|
| Pl   | Normal | Soft | ++   |      |    |                       | degeneration of the basal cell layer                                    | the nose bridge and cheeks                                          |                        |                          |
| Pl   | Normal | Soft | ++   |      |    | Cobblestone appearance of mucosa and linear ulceration              | Histopathology                                                          | Intestinal pain, anal fissures, diarrhea, and labial enlargement       | Crohn’s disease         | Steroids and immunosuppressants to decrease progression |
| RP   | Soft and friable | Loss of stippling | ++   |      |    | Gingival recession, Nodules and ulceration, Loosening of teeth      | Hyperglobulinemia, an elevated level of serum angiotensin-converting enzyme, evidence of depressed cellular immunity. H/P: noncaseating epithelioid granulomas in more than one organ system | Swelling of salivary glands                                      | Sarcoidosis             | Systemic steroids and anti-inflammatory agents       |
| Pi   | Normal | Fibrous | Smooth | +   |    | Exophytic smooth masses                                            | H/P: bundles of collagen covered with the epithelium                      | Fibrous epulis                                                       | Excision and curettage  |                          |
| RP   | Normal | Fibrous | Smooth | ++  |    | Start from interdental papilla, Pedunculated to sessile masses      | H/P: cellular fibroblastic tissue containing rounded or lobulated masses of calcified cementum-like tissue | Calcifying fibroblastic granuloma                                    | Excision of lesion      |                          |
| RP   | +    |    | Ukerated, smooth, and pedunculated mass | H/P: discontinuous hyperplastic parakeratinized stratified squamous epithelium and endothelial cells in the connective tissue | Pyogenic granuloma                                                        | Excision of lesion                                                    |                          |                          |
| C       | Cr       | Ca       | Add. Sym | Lab & HP | Diag. Sym | Rx       | Histopathology                                                                 |
|---------|----------|----------|----------|----------|-----------|----------|--------------------------------------------------------------------------------|
| C-Br    | Br-       | C-       |          |          |           | Rx       | Peripheral giant cell granuloma                                               |
| C-       | Br-       | C-       |          |          |           | Rx       | Soft ++ Sessile or pedunculated tumor-like process                            |
| C-       | Br-       | C-       |          |          |           | Rx       | H/P: multinucleated giant cell forming granuloma                              |
| C-       | Br-       | C-       |          |          |           | Rx       | Surgical excision/chemotherapy and laser ablation                           |
| C-       | Br-       | C-       |          |          |           | Rx       | Corrugated or verrucous surface                                              |
| C-       | Br-       | C-       |          |          |           | Rx       | Non-removable white spot                                                      |
| C-       | Br-       | C-       |          |          |           | Rx       | Peripheral giant cell granuloma                                               |
| C-       | Br-       | C-       |          |          |           | Rx       | Tissue biopsy, Yal stained with toluidine blue and cytochrome techniques.     |
| C-       | Br-       | C-       |          |          |           | Rx       | H/P: dysplastic cells with nuclear pleomorphism and loss of cellular polarity and orientation |
| C-       | Br-       | C-       |          |          |           | Rx       | Histological sections of the tissue biopsy                                  |
| C-       | Br-       | C-       |          |          |           | Rx       | Vital staining with toluidine blue and cytochrome techniques.                |
| C-       | Br-       | C-       |          |          |           | Rx       | H/P: dysplastic cells with nuclear pleomorphism and loss of cellular polarity and orientation |
| C-       | Br-       | C-       |          |          |           | Rx       | Tissue biopsy, Yal stained with toluidine blue and cytochrome techniques.     |
| C-       | Br-       | C-       |          |          |           | Rx       | H/P: dysplastic cells with nuclear pleomorphism and loss of cellular polarity and orientation |
| C-       | Br-       | C-       |          |          |           | Rx       | Histological sections of the tissue biopsy                                  |
| C-       | Br-       | C-       |          |          |           | Rx       | Vital staining with toluidine blue and cytochrome techniques.                |
| C-       | Br-       | C-       |          |          |           | Rx       | H/P: dysplastic cells with nuclear pleomorphism and loss of cellular polarity and orientation |
| C-       | Br-       | C-       |          |          |           | Rx       | Histological sections of the tissue biopsy                                  |
| C-       | Br-       | C-       |          |          |           | Rx       | Vital staining with toluidine blue and cytochrome techniques.                |
| C-       | Br-       | C-       |          |          |           | Rx       | H/P: dysplastic cells with nuclear pleomorphism and loss of cellular polarity and orientation |
| C-       | Br-       | C-       |          |          |           | Rx       | Histological sections of the tissue biopsy                                  |
| C-       | Br-       | C-       |          |          |           | Rx       | Vital staining with toluidine blue and cytochrome techniques.                |
| C-       | Br-       | C-       |          |          |           | Rx       | H/P: dysplastic cells with nuclear pleomorphism and loss of cellular polarity and orientation |
| C-       | Br-       | C-       |          |          |           | Rx       | Histological sections of the tissue biopsy                                  |
| C-       | Br-       | C-       |          |          |           | Rx       | Vital staining with toluidine blue and cytochrome techniques.                |

### Oral Diseases

- **Leukoplakia**
  - Surgical excision:
  - W: Corrugated or verrucous surface
  - R: Shallowly demarcated from surrounding mucosa
  - P: Smooth
  - S: Soft
- **Erythroplakia**
  - Surgical excision:
  - W: Corrugated or verrucous surface
  - R: Shallowly demarcated from surrounding mucosa
  - P: Smooth
  - S: Soft
- **Squamous cell carcinoma**
  - Surgical excision:
  - W: Corrugated or verrucous surface
  - R: Shallowly demarcated from surrounding mucosa
  - P: Smooth
  - S: Soft

### History of Tobacco/Alcohol Intake

- Squamous cell carcinoma
  - History of tobacco/alcohol intake
  - Surgical excision:
  - W: Corrugated or verrucous surface
  - R: Shallowly demarcated from surrounding mucosa
  - P: Smooth
  - S: Soft

### Soft and Edematous Tissue

- **Squamous cell carcinoma**
  - Surgical excision:
  - W: Corrugated or verrucous surface
  - R: Shallowly demarcated from surrounding mucosa
  - P: Smooth
  - S: Soft

### Monitoring of the Patient

- **Squamous cell carcinoma**
  - Monitoring of the patient for infection during neutropenic periods and early management of infection.
  - Corticosteroids, adrenocorticotropin, or testosterone modulates the sharp reduction in marrow function.
  - Granulocyte colony...
| C | Cr | Ca | T | S | P | L | Lab & H/P | Add Sym | D | Rx |
|---|---|---|---|---|---|---|----------|---------|---|----|
| P | Rounded | Soft | Smooth | ++ | | | Histopathology will show Reed-Sternberg cells | Swollen lymph nodes | Lymphoma | Radiation and chemotherapy plus doxorubicin, bleomycin, vincristine, and dacarbazine for Hodgkin’s lymphoma and cyclophosphamide, vincristine, and prednisone for non-Hodgkin’s |

| W plaques | No change | Soft | Loss of stippling | + | Seen on facial attached gingiva | Leukoplakia-like asymptomatic plaque | H/P: dense fibrous connective tissue | Frictional keratosis | Prevention of deleterious habits |
|---|---|---|---|---|---|---|---|---|---|
| RP | No change | Soft and friable | — | Gingival recession | Superficial and horizontal gingival laceration | Not much significant | Toothbrushing-induced gingival ulceration | Changing the brushing technique |
| R-W | — | | | | Surface slough or ulceration | Not much significant | Chemical insult due to etching, chlorhexidine, hydrogen peroxide, acetylsalicylic acid, dentifrice, detergent, calcium hydroxide, etc. | Removal of offending irritant |
| R | — | | | | Erythematous lesion that slough a coagulated surface, vesicles and ulceration may be present | Not of much significance | Burns of mucosa | Supportive care and hydration |
| C  | Cr | Cs | T  | S  | P  | L                  | Lab & H/P                                | Add Sym                              | D                        | Rx                        |
|----|----|----|----|----|----|--------------------|-----------------------------------------|---------------------------------------|--------------------------|--------------------------|
| Br-Bl | No change | No change | No change | = | | Pigmented deposits in the epithelium and connective tissue | Addison's disease, Albright syndrome, Peutz-Jeghers syndrome | Gingival pigmentation | Not required |
| Br | No change | Firm | No change | = | | Mandibular facial gingiva | H/P: pigmented macules seen in section | Smoker's melanosis | Smoking cessation for 2 weeks |
| Bl-Gy-Br-Bl | No change | No change | No change | = | | Diffuse pigmentation | Drug-induced pigmentation (antimalarial, minocycline) | Cessation of drug if required |
| Bl-Gy-Br-Bl | No change | No change | No change | = | | H/P: discrete granules in connective tissue | Amalgam tattoo | Removal of amalgam debris and replacement of amalgam if required |

C, color; Cr, contour; Cs, consistency; T, texture; S, size; P, position; L, lesion; lab and H/P, laboratory procedures and histopathology; add sym, additional symptoms; D, diagnosis; Rx, treatment; FR, fiery red; G, same as surrounding gingiva; W, white; PR, pink to reddish; B-Br, black to brown; R-Gy, red to gray; RP, reddish pink; BR, bright red; Pi, pink; Pl, pale pink; Pr, purple; B, blue; OHI, oral hygiene instruction; CHX, chlorhexidine; +, slightly increased; ++, increased; --, slightly decreased; --/--, decreased; --/-++, may increase or decrease; =, remains the same.

Table 4. Clinical features for diagnosis and treatment of non-plaque-induced gingival diseases.
3. Treatment of gingival disease

The treatment of gingival disease is based on resolving the etiologic factors and maintaining the systemic status of the individual. In the case of plaque-induced gingivitis, the main treatment plan involves removal of plaque and calculus by scaling and root planning, followed by oral hygiene instruction which includes modified bass method of brushing and the use of chemical plaque control agents like 0.2% or 0.02% chlorhexidine gluconate or essential oil mouthwash. In cases of gingival enlargement, initial therapy is focused on removing plaque and calculus, followed by a review on the gingival condition; only if the condition does not improve the drug substitution may be considered, followed by gingivectomy to remove the enlarged gingival tissue. Plaque-induced gingival disease influenced by modifying factors is controlled by reducing the exposure of the modifying factor in addition to removal of plaque and calculus to maintain oral hygiene. The details of the treatment have been mentioned in Table 2. Non-plaque-induced gingival diseases are treated depending on the etiology of the gingival disease. For example, viral lesions are treated by providing antiviral medications in addition to oral hygiene instruction. The details of treatment in brief are mentioned in Table 4. Diagnosis is essential for providing the proper treatment plan and updating recent research which might help prevent undue treatment [8].

4. Conclusions

Gingival diseases are an initial starting point of the advanced periodontal disease and in some cases depict the manifestation of an underlying undiagnosed systemic condition. Therefore, the early diagnosis of gingival disease and its treatment are warranted.

Conflict of interest

The authors declare no conflict of interest.

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