# Acute

#### **Gingival and Periodontal Conditions**

#### Sam Malkinson

DMD, Cert Perio, FRCD(C), Diplomate of the American Board of Periodontology

**Okanagan Periodontal Study Club** 

November 4, 2017

#### Objectives

#### By the end of this lecture you will:

- know what constitutes an "acute" periodontal problem
- know how to identify and manage the <u>most common</u> acute periodontal problems
- know how to behave when confronted with an acute periodontal problem (or any acute problem, for that matter)

#### Overview

- I. Introduction
- 2. Periodontal abscesses
- 3. Necrotizing periodontal diseases
- 4. Acute gingival conditions of viral and fungal origin
- 5. Miscellaneous acute situations
- 6. A final word of caution



#### I. Introduction

#### Introduction

- The overwhelming majority of disease entities we deal with regarding the periodontium are chronic in nature
- A chronic condition is a human health condition or disease that is persistent or otherwise long-lasting in its effects (3 months is the cut-off)<sup>1</sup>

#### Chronic vs. ?????

- When diagnosing periodontitis, the "opposite" of chronic is usually considered to be aggressive
- Aggressive ≠ acute

#### "Acute"

- Refers to an entity "with a rapid onset and/or a short course"<sup>2</sup>
- Acute ≠ severe

#### Armitage Classification

- This classification comprises every currently recognized periodontal diagnosis in existence
- Some are chronic in nature, while others are

## ACUTE

#### Armitage Classification

#### Development of a Classification System for Periodontal Diseases and Conditions

Volume 4 • Number 1 • December 1999 Ann Periodontol

vise specified (NOS)

- I. Gingival Diseases A. Dental plaque-induced gingival diseases\*
  - I. Gingivitis associated with dental plaque only
    - a. without other local contributing factors
  - b. with local contributing factors (See VIII A)
  - 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 Non-plaque-induced gingival lesions

- 1. Gingival diseases 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) vario
- b. other

Figure 1.

lla-zoster intections	<ol> <li>Foreign bo</li> <li>Not other</li> </ol>
tal diseases and conditions.	

<ol> <li>Gingival diseases of fungal origin         <ol> <li>Candida-species infections</li> <li>generalized gingival candidosis</li> </ol> </li> </ol>	II. Chronic Periodontitis <sup>†</sup> A. Localized B. Generalized
<ul> <li>b. linear gingwal erythema</li> <li>c. histoplasmosis</li> <li>d. ethan</li> </ul>	III. Aggressive Periodontitis <sup>†</sup> A. Localized
<ol> <li>Other</li> <li>Ciacial lations of constitution</li> </ol>	B. Generalized
<ul> <li>a. hereditary gingival fibromatosis</li> </ul>	IV. Periodontitis as a Manifestation of Systemic Diseases
<ul> <li>b. other</li> <li>5. Gingval manifestations of systemic conditions <ul> <li>a. mucocutaneous disorders</li> <li>I) lichen planus</li> <li>2) pemphigoid</li> </ul> </li> </ul>	A. Associated with hematological disorders     I. Acquired neutropenia     Z. Leukemias     Other     B. Associated with genetic disorders
<ul> <li>3) pemphigus vulgaris</li> <li>4) erythema multiforme</li> <li>5) lupus erythematosus</li> <li>6) drug-induced</li> <li>7) other</li> <li>b. allergic reactions</li> <li>1) dental restorative materials <ul> <li>a) mercury</li> <li>b) nickel</li> <li>c) acrylic</li> <li>d) other</li> </ul> </li> <li>2) reactions attributable to <ul> <li>a) toothpastes/dentifices</li> </ul> </li> </ul>	
<ul> <li>b) mouthrinses/mouthwashes</li> <li>c) chewing gum additives</li> <li>d) foods and additives</li> <li>3) other</li> <li>6. Traumatic lesions (factitious, iatrogenic, accidental)</li> <li>a. chemical injury</li> </ul>	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
b. physical injury c. thermal injury	C. Pericoronal abscess
7. Foreign body reactions	

#### Figure 1. (Continued)

† Can be further classified on the basis of extent and severity. As a general guide, extent can be characterized as Localized = <30% of sites involved and Generalized =>30% of sites involved. Severity can be characterized on the basis of the amount of clinical attachment loss (CAL) as follows: Slight = 1 or 2. mm CAL, Moderate = 3 or 4 mm CAL, and Severe = ≥5 mm CAL.

- VII. Periodontitis Associated With Endodontic Lesions A. Combined periodontic-endodontic lesions VIII. Developmental or Acquired Deformities and Conditions
  - A. Localized tooth-related factors that modify or predispose

Armitage

- 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
  - I. 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 (See I.A.3. and I.B.4.)
- 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

Classification of periodon \* Can occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is not progressing.

#### Armitage Classification

 This list is very long, so this lecture is going to focus mainly on the clinical entities you will see <u>most commonly</u>

#### Overview

- I. Introduction
- 2. Periodontal abscesses

#### Abscesses

- The general process of abscess formation involves neutrophil activation and migration toward the site of an infectious pathogen or foreign body
- The concomitant cytokine release recruits other WBCs and increases regional blood flow
- The area is encapsulated by healthy cells

#### **Gingival Abscess**

- This implies that the process is confined to the marginal gingiva or interdental papilla, without involvement of the supporting structures of the tooth
- Usually presents as a localized, painful, rapidly expanding papule with a red swollen periphery and shiny surface
- Filled with seropurulent exudate



#### Periodontal Abscess

 This now implies that the supporting structures of the tooth have become involved, with some level of associated destruction of bone/pdl

#### Case I

 53 y.o. Q healthy, non-smoking patient presented for emergency exam relating to 4-day history of severe pain in sextant 2, fever, and malaise. All teeth in sextant 2 were vital except 13, which had been endo-treated

many years before







### Signs and Symptoms

- PAIN is the most common presenting symptom
- Swelling, mobility, extrusion, draining sinus tract, radiographic bone loss, regional lymphadenopathy and body temperature increase are all secondary features which may be present

#### Environmental Etiology

- Frequently occurs in a pre-existing periodontal pocket
- Basically, the offending pathogens or foreign bodies get into a situation where they are encapsulated and have nowhere to go

#### \*Environmental Etiology\*

 One common scenario is when a deep pocket is not scaled adequately, and the coronal portion heals by repair, leaving bacteria in the apical portion



## Microbial Etiology

- A mixed infection, highly gram-negative (66.2%) and highly anaerobic (65.2%)<sup>3</sup>
- Some of the microorganisms isolated from abscesses include:
  - B. melaninogenicus subspecies<sup>3</sup>
  - F. nucleatum<sup>3</sup>
  - Vibrios<sup>3</sup>
  - Capnocytophagae<sup>3</sup>
  - Peptococci<sup>3</sup>
  - Peptostreptococci<sup>3</sup>
  - P. gingivalis<sup>4,6</sup>
  - P. intermedia<sup>4,6</sup>
  - C. albicans<sup>5</sup>
  - A. actinomycetemcomitans<sup>6</sup>

#### Very similar to chronic periodontitis

#### **Differential Diagnosis**

- Develop a mental algorithm for eliminating the following possibilities:
  - Periapical abscess (vitality of the tooth)
  - Acute pulpitis (radiographic appearance)
  - Vertical root fracture (pain profile/radiographic appearance/probing profile/inflammation)
  - Pericoronitis (presence of an overlying flap of tissue)
  - Periodontal Cyst (pain profile)
  - Osteomyelitis (radiographic appearance/patient profile)

#### **Treatment Options**

- (Incision and drainage)
- Thorough scaling and root planing ± systemic antibiotics
- Surgical debridement ± systemic antibiotics

   Use of systemic chemotherapy should be based on systemic symptoms

#### Periodontal Abscesses – Diabetes Mellitus

• Multiple periodontal abscesses are nearly pathognomonic of an uncontrolled diabetic

state



#### Case I

• So what ended up happening to our friend?





#### Case I

• After Sc/RP with a course of amoxicillin....



#### Overview

- I. Introduction
- 2. Periodontal abscesse
- 3. Necrotizing periodontal diseases

#### Necrotizing Periodontal Diseases

- Necrosis implies some form of cellular injury which leads to premature cell death<sup>7</sup>
- A continuum of necrotizing diseases exist in the current periodontal diagnostic classification system which begins with the gingiva, progresses to the periodontium, and may eventually lead to involvement of the basal bone and overlying tissues of the oral cavity

#### The Continuum

- NUG = necrotizing ulcerative gingivitis, implies involvement restricted to the gingiva
- 2. NUP = necrotizing ulcerative periodontitis, implies the supporting structures of the tooth have become involved, with some level of associated destruction of bone/pdl<sup>8</sup>

 38 y.o. A HEAVY smoker presents for emergency exam. 6 month hx of progressively worsening pain and oral malodour with "something funny on my gums." Pt. complains of feeling "stressed."

![](_page_29_Picture_2.jpeg)

![](_page_30_Picture_1.jpeg)

 29 y.o. A malnourished immigrant presents for emergency exam. I year hx of progressively worsening pain and oral malodour. Pt. has not

seen a physician in over 20 years.

![](_page_31_Picture_3.jpeg)

![](_page_32_Picture_1.jpeg)

![](_page_32_Picture_2.jpeg)

![](_page_33_Picture_0.jpeg)

![](_page_34_Picture_0.jpeg)

![](_page_34_Picture_2.jpeg)

### Signs and Symptoms

- PAIN
- Ulcerated/eroded/punched-out interproximal papillae
- Gingival tissue covered by a pseudomembrane (yellowish or gray-white film of desquamative tissue), covering erythematous tissue
- Gingival hemorrhage
- Pronounced foul odour
## The Continuum

- Necrotizing stomatitis implies that the destruction has gone beyond the mucogingival junction<sup>9</sup>
- 4. In principle, if left unchecked, this can progress to noma/cancrum oris

#### Necrotizing Stomatitis/Noma







#### **Environmental Etiology**

- Poor oral hygiene<sup>10,11</sup>
- Pre-existing gingivitis/periodontitis<sup>10,11</sup>
- Previous NUG/NUP<sup>12,13</sup>





## Systemic Etiology

- Stress<sup>14,15</sup>
- Smoking<sup>10,15</sup>
- Malnutrition<sup>16</sup>
- AIDS, or other immunocompromise<sup>17</sup>





# Microbial Etiology

- Some of the bacteria associated with necrotizing periodontal diseases include<sup>18</sup>:
  - Treponemae
  - Selenomae
  - B. melaninogenicus subspecies intermedius
  - Fusobacteria

## Histological Appearance

- Famous histological study characterized 4 distinct zones, starting with the most superficial<sup>19</sup>:
  - I. Bacterial zone
  - 2. Neutrophil-rich zone
  - 3. Necrotic zone
  - 4. Zone of spirochetal infiltration



1. Bacterial zone

2. Neutrophilrich zone

3. Necrotic zone

4. Zone of spirochetal infiltration



#### Treatment

- Scaling and root planing of all affected areas ± systemic antibiotics
  - Use of systemic chemotherapy should be based on systemic symptoms (metronidazole, tetraycline or penicillin)
- Antibacterial mouthrinses (chlorhexidine 0.12% or hydrogen peroxide 3%)
- Close follow-up (1-2 days)

# Prognosis

- With appropriate treatment, patients' clinical symptoms should regress within 4-6 days
- The decision then becomes, "Is there anything that requires surgical therapy to correct?"

#### Case 2

#### • After Sc/RP, metronidazole and chlorhexidine....



#### Overview

- I. Introduction
- 2. Periodontal abscesse
- 3. Necrotizing period on tal diseases
- 4. Acute gingival conditions of viral and fungal origin

# Primary Herpetic Gingivostomatitis

- Diffuse intraoral ulcerative gingivitis and mucositis
- Pain
- Fever
- Lymphadenopathy

#### Primary Herpetic Gingivostomatitis

https://www.dermquest.com/image-library/image/5044bfd1c97267166cd6711d





#### Recurrent Intra-oral Herpes

 May have similar systemic symptoms to primary herpetic gingivostomatitis, but lesions appear on keratinized tissue only, such as gingiva and palate<sup>20</sup>



https://www.researchgate.net/figure/45089093\_fig5\_Figure-4-Recurrent-intraoral-herpes-Ulcers-started-as-small-individual-vesicles-that

# Etiology

- HSV-1 or HSV-2 infection
- Most infections are subclinical, but clinical signs and symptoms may be precipitated by:
  - Trauma<sup>21,22</sup>
  - Illness<sup>21,22</sup>
  - Emotional stress<sup>21,22</sup>
  - Ultraviolet radiation<sup>21,22</sup>

#### Treatment

 Treatment of intra-oral herpetic infections is essentially palliative in nature, though there is some suggestion that if initiated within first 3 days of onset of symptoms, antiviral meds may help<sup>23</sup>:

*B*:Acyclovir 200mg caps Disp: 33 caps Sig: take 3 caps stat, then 2 caps tid x 5 days or

R: Famciclovir 125mg tab Disp: 10 tabs Sig: take 1 tab bid x5 days until all finished or R:Valacyclovir 500mg tabs Disp: 10 tabs Sig: take 1 tab bid x 5 days and/or

**R**:Acyclovir 5% ointment Disp: 15g tube Sig:Apply to affected area 6x/day

## Varicella Zoster

- Primary infection with VZV manifests as chickenpox, but then the virus remains latent in neural ganglia
- Reactivation is termed shingles
- It can affect the trigeminal ganglion, and cause intra-oral shingles

#### Varicella Zoster

- Typical intra-oral presentation is of vesicles or ulcerations, surrounded by an erythematous halo, unilaterally on the palate<sup>24</sup>, though can present on other tissues
- Circumscribed dermatomally<sup>24</sup>
- Painful, but heal in a few days
- Can result in post-herpetic neuralgia (not fun)



# Etiology

- VZV reactivation occurs more often in the:
  - Immunocompromised
  - Elderly (confounded by immune status?)
- Stress and trauma may also precipitate an outbreak

#### Treatment

- Again, treatment is essentially palliative, including a soft diet, hydration, rest, gentle oral hygiene (supplemented by chlorhexidine)
- All the previously described systemic antiviral prescriptions have been used, and all have been shown to reduce the severity of the symptoms and to prevent post-herpetic neuralgia<sup>25</sup>

## Human Papilloma Virus

- Can occur on multiple oral sites, including the gingiva<sup>26,27,28</sup>
- HPV types 6, 11 associated with squamous papilloma<sup>29</sup>
- HPV types 2, 4, 40 associated with verruca vulgaris<sup>29</sup>
- HPV types 2, 6, 11, 53, 54 associated with condyloma acuminatum<sup>30</sup>

## Human Papilloma Virus



Squamous Papilloma



Verruca Vulgaris

Condyloma Acuminatum



Rose & Mealey

#### Treatment

- Complete sexual history is indicated<sup>31</sup>
- Treatment of choice is excisional biopsy
- Recurrence is rare
- REMEMBER that some types of HPV (16, 18) are associated with malignant transformation

# Linear Gingival Erythema

- Most often seen in HIV+ patients
- Really a mixed bacterial/fungal infection<sup>32</sup>
- Needs scaling to remove plaque, but if no response then systemic antifungal therapy is indicated<sup>33</sup>:

R: Ketoconazole 200 mg tabs Disp: 8 tabs Sig: take 1 tab w/ breakfast



#### Overview

- I. Introduction
- 2. Periodontal abscess
- 3. Necrotizing periodontal diseases
- 4. Acute gingival contributions of viral and fungal origin
- 5. Miscellaneous acute situations

#### Pericoronitis

- Implies an inflammation of gingival tissue overlying the crown of a tooth
- Most often happens with 3<sup>rd</sup> molars, but can in theory happen when any tooth erupts into the mouth

#### Pericoronitis



# Signs and Symptoms

- PAIN
- Inflamed operculum
- Trismus/soreness upon opening
- Foul taste
- Fever
- Lymphadenopathy
- Malaise

## Negative Sequelae

- Untreated periocoronitis can lead to:
  - Peritonsillar abscess
  - Cellulitis
  - Dysphagia
  - Ludwig's Angina



# Etiology

 Occurs when plaque, calculus, food, or any other debris becomes lodged under the operculum



#### Treatment

 Irrigation/debridement under the operculum to remove the local irritant ± systemic antibiotics

Then, once acute phase has healed....

- Operculectomy?
- Extraction of the offending tooth?

#### **Reactive Nodular Lesions**

- The Big 4:
  - Fibroma
  - Pyogenic Granuloma
  - Peripheral Ossifying Fibroma
  - Peripheral Giant Cell Granuloma

#### **Reactive Nodular Lesions**



Pyogenic Granuloma

> Peripheral Ossifying Fibroma

#### Fibroma

Peripheral Giant Cell Granuloma







#### Treatment

- Excisional biopsy with localized Sc/RP
- Why Sc/RP? They're called reactive because they REACT to local factors
- In the case of a pregnant patient, if suspecting pyogenic granuloma, have the option to monitor until after baby is born...lesion may spontaneously regress

## Desquamative Gingivitis

- Generally a manifestation of a mucocutaneous disorder:
  - Lichen Planus
  - Mucous Membrane Pemphigoid
  - Pemphigus Vulgaris
  - Systemic Lupus Erythematosus
  - Epidermolysis Bullosa
  - Erythema Multiforme

#### Desquamative Gingivitis


# Diagnosis

- Take a thorough medical history
- Check Nikolsky's sign!
- Biopsy is indicated to identify the exact disease process
- Request direct and indirect immunofluorescence, in addition to routine pathology

#### Treatment

- Referral to an oral pathologist, oral medicine specialist, dermatologist, or ophthalmologist may be indicated
- Depending on the diagnosis, a variety of topical/systemic medications may be indicated

# Malignancies

- Though some malignancies may be insidious and therefore best considered more as chronic problems, their prognosis is usually better the earlier they are identified
- Other malignancies are acute in nature

## Malignancies



Squamous Cell Carcinoma

Melanoma



## Malignancies



Acute Myelogenous Leukemia

Metastatic Adeno-carcinoma

## **Diagnosis and Treatment**

- A thorough history and time course is essential
- A comprehensive exam is a MUST
- Referral to oral pathology/OMFS, or in the case of a suspected hematological malignancy, to a hematologist

## Overview

- I. Introduction
- 2. Periodontal abscess
- 3. Necrotizing period tal diseases
- 4. Acute gingival conditions of viral and fungal origin
- 5. Miscellaneous agate situation
- 6. A final word of caution

# Wisdom

- <u>The House of God</u> by Samuel Shem, pub. 1978, semi-fictional account of the experiences of medical interns
- I3 laws about how to survive residency:
  - Law III: At a cardiac arrest, the first procedure is to take your own pulse.
  - Law X: If you don't take a temperature, you can't find a fever.



## Case 3

 44 y.o. 3 presents FRIGHTENED OUT OF HIS #\$%&ING MIND, with a 1 month hx of a slightly tender 2x2mm papule on the crestal gingiva overlying the edentulous area of #46



# Why so scared?

- Because the GP who had referred the case had taken one look at the papule, and said, "Oh my G-d you have to go and see the specialist right away!"
- Aside from scaring the patient, she also omitted a very important step

# Why did I relate this story?

 To remind you all that you're expected to do what you're supposed to do, and that not doing so can have negative consequences for the patient

# "You don't want no drama."

#### -Stacy Ann "Fergie" Ferguson Singer, as heard in the song "My Humps"



## References

- 1. Chronic diseases, World Health Organization, retrieved 2012-11-26.
- 2. Mosby's medical dictionary: illustrated in full color throughout. (5. ed. ed.). St. Louis: Mosby. 1998.
- 3. Newman MG, Sims TN. The predominant cultivable microbiota of the periodontal abscess. J Periodontol. 1979 Jul;50(7):350-4.
- 4. van Winkelhoff AJ, Carlee AW, de Graaff J. Bacteroides endodontalis and other black-pigmented Bacteroides species in odontogenic abscesses. Infect Immun. 1985 Sep;49(3):494-7.
- 5. DeWitt GV, Cobb CM, Killoy WJ. The acute periodontal abscess: microbial penetration of the soft tissue wall. Int J Periodontics Restorative Dent. 1985;5(1):38-51.
- 6. Slots J, Listgarten MA. Bacteroides gingivalis, Bacteroides intermedius and Actinobacillus actinomycetemcomitans in human periodontal diseases. J Clin Periodontol. 1988 Feb;15(2):85-93.
- 7. Proskuryakov SY, Konoplyannikov AG, Gabai VL. Necrosis: a specific form of programmed cell death? Experimental Cell Research 283, 1-16.
- 8. MacCarthy D, Claffey N. Acute necrotizing ulcerative gingivitis is associated with attachment loss. J Clin Periodontol. 1991 Nov;18(10):776-9.
- 9. Williams CA, Winkler JR, Grassi M, Murray PA. HIV-associated periodontitis complicated by necrotizing stomatitis. Oral Surg Oral Med Oral Pathol. 1990 Mar;69(3):351-5.
- 10. Melnick SL, Roseman JM, Engel D, Cogen RB. Epidemiology of acute necrotizing ulcerative gingivitis. Epidemiol Rev. 1988;10:191-211.
- 11. Shields WD. Acute necrotizing ulcerative gingivitis. A study of some of the contributing factors and their validity in an Army population. J Periodontol. 1977 Jun;48(6):346-9.
- 12. Jiménez M, Baer PN. Necrotizing ulcerative gingivitis in children: a 9 year clinical study. J Periodontol. 1975 Dec;46(12):715-20.
- 13. Giddon DB, Zackin SJ, Goldhaber P. Acute necrotizing ulcerative gingivitis in college students. J Am Dent Assoc. 1964 Mar;68:380-6.
- 14. Maupin CC, Bell WB. The relationship of 17-hydroxycorticosteroid to acute necrotizing ulcerative gingivitis. J Periodontol. 1975 Dec;46(12):721-2.
- 15. Clarke NG, Shephard BC, Hirsch RS. The effects of intra-arterial epinephrine and nicotine on gingival circulation. Oral Surg Oral Med Oral Pathol. 1981 Dec;52(6):577-82.

## References

- 16. Pindborg JJ. Gingivitis in military personnel with special reference to ulceromembranous gingivitis. Odontol Tidskr. 1951;59(6):403-99.
- 17. Cogen RB, Stevens AW Jr, Cohen-Cole S, Kirk K, Freeman A. Leukocyte function in the etiology of acute necrotizing ulcerative gingivitis. J Periodontol. 1983 Jul;54(7):402-7.
- 18. Loesche WJ, Syed SA, Laughon BE, Stoll J. The bacteriology of acute necrotizing ulcerative gingivitis. J Periodontol. 1982 Apr;53(4):223-30.
- 19. Listgarten MA. Electron microscopic observations on the bacterial flora of acute necrotizing ulcerative gingivitis. J Periodontol. 1965 Jul-Aug;36:328-39.
- 20. Eisen D. The clinical characteristics of intraoral herpes simplex virus infection in 52 immunocompetent patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998 Oct;86(4):432-7.
- 21. Scully C. Orofacial herpes simplex virus infections: current concepts in the epidemiology, pathogenesis, and treatment, and disorders in which the virus may be implicated. Oral Surg Oral Med Oral Pathol. 1989 Dec;68(6):701-10.
- 22. Bergström T, Lycke E. Neuroinvasion by herpes simplex virus. An in vitro model for characterization of neurovirulent strains. J Gen Virol. 1990 Feb;71 (Pt 2):405-10.
- 23. Woo SB, Challacombe SJ. Management of recurrent oral herpes simplex infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007 Mar;103 Suppl:S12.e1-18.
- 24. Eisenberg E. Intraoral isolated herpes zoster. Oral Surg Oral Med Oral Pathol. 1978 Feb;45(2):214-9.
- 25. Mustafa MB, Arduino PG, Porter SR. Varicella zoster virus: review of its management. J Oral Pathol Med. 2009 Oct;38(9):673-88.
- 26. Abbey LM, Page DG, Sawyer DR. The clinical and histopathologic features of a series of 464 oral squamous cell papillomas. Oral Surg Oral Med Oral Pathol. 1980 May;49(5):419-28.
- 27. Green TL, Eversole LR, Leider AS. Oral and labial verruca vulgaris: clinical, histologic and immunohistochemical evaluation. Oral Surg Oral Med Oral Pathol. 1986 Oct;62(4):410-6.

## References

- 28. Zunt SL, Tomich CE. Oral condyloma acuminatum. J Dermatol Surg Oncol. 1989 Jun;15(6):591-4.
- 29. Ward KA, Napier SS, Winter PC, Maw RD, Dinsmore WW. Detection of human papilloma virus DNA sequences in oral squamous cell papillomas by the polymerase chain reaction. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1995 Jul;80(1):63-6.
- 30. Sykes NL Jr. Condyloma acuminatum. Int J Dermatol. 1995 May;34(5):297-302.
- 31. Nelke KH, Lysenko L, Leszczyszyn J, Gerber H. Human papillomavirus and its influence on head and neck cancer predisposition. Postepy Hig Med Dosw (Online). 2013 Jul 15;67:610-6.
- 32. Velegraki A, Nicolatou O, Theodoridou M, Mostrou G, Legakis NJ. Paediatric AIDS-related linear gingival erythema: a form of erythematous candidiasis? J Oral Pathol Med. 1999 Apr;28(4):178-82.
- 33. Ryder MI, Nittayananta W, Coogan M, Greenspan D, Greenspan JS. Periodontal disease in HIV/AIDS.Periodontol 2000. 2012 Oct;60(1):78-97.