# First semester-classification of periodontal and peri implant conditions (2017)
# Lec.4.part2

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# Content



- **Non plaque induce gingival disease**

1 **GENETIC/DEVELOPMENTAL ABNORMALITIES**

**2.SPECIFIC INFECTIONS**

**3 INFLAMMATORY AND IMMUNE CONDITIONS AND LESIONS.**

4 **REACTIVE PROCESSES.**

**5. NEOPLASMS.**

**6. ENDOCRINE, NUTRITIONAL, AND METABOLIC DISEASES.**

**7. TRAUMATIC LESIONS.**

8. **GINGIVAL PIGMENTATION/melanoplakia**

**1 Genetic/developmental disorders**
Hereditary gingival fibromatosis

**2 Specific infections**
a. Bacterial origin
Neisseria gonorrhoeae (gonorrhea)
Treponema pallidum (syphilis)
Mycobacterium tuberculosis (tuberculosis)
Streptococcal gingivitis (strains of streptococcus)
b. Viral origin
Coxsackie virus (hand-foot-and-mouth disease)
Herpes simplex 1/2 (primary or recurrent)
Varicella-zoster virus (chicken pox or shingles affecting V nerve)
c. Fungal
Candidosis

**3 Inflammatory and immune conditions and lesions**
a. Hypersensitivity reactions
Contact allergy
Plasma cell gingivitis
Erythema multiforme
b.Autoimmune diseases of skin and mucous membranes
Pemphigus vulgaris
Pemphigoid
Lichen planus
Lupus erythematosus
c.Granulomatous inflammatory conditions (orofacial granulomatosis)
Crohn's disease
Sarcoidosis

**4 Reactive processes**
Epulides
Fibrous epulis

**5 Neoplasms**
a. Premalignant
Leukoplakia
Erythroplakia
b. Malignant
Squamous cell carcinoma
Leukemia
Lymphoma

**6 Endocrine, nutritional, and metabolic diseases**
Vitamin deficiencies

**7 Traumatic lesions**
a. Physical/mechanical insults
Toothbrushing-induced gingival ulceration
Factitious injury (self-harm)
b. Chemical (toxic) insults
Etching
Chlorhexidine
Acetylsalicylic acid
c. Thermal insults
Burns of mucosa

**8 Gingival pigmentation**
Smoker's melanosis
Amalgam tattoo

## B. Non-plaque induce gingival lesion

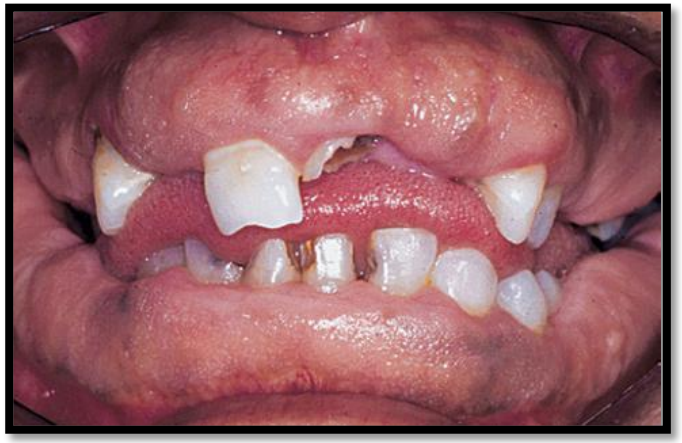


The origin of gingival inflammation in this group is different from that of the routine plaque-associated gingivitis. Its not cause by plaque and usually dose not disappear after plaque removal.

# 1 GENETIC/DEVELOPMENTAL ABNORMALITIES

- **Hereditary gingival fibromatosis (HGF)**

Clinically, gingival fibromatosis may present gingival overgrowth in various degrees., hereditary gingival fibromatosis is a rare disease which may occur as an isolated disease or as part of a syndrome.

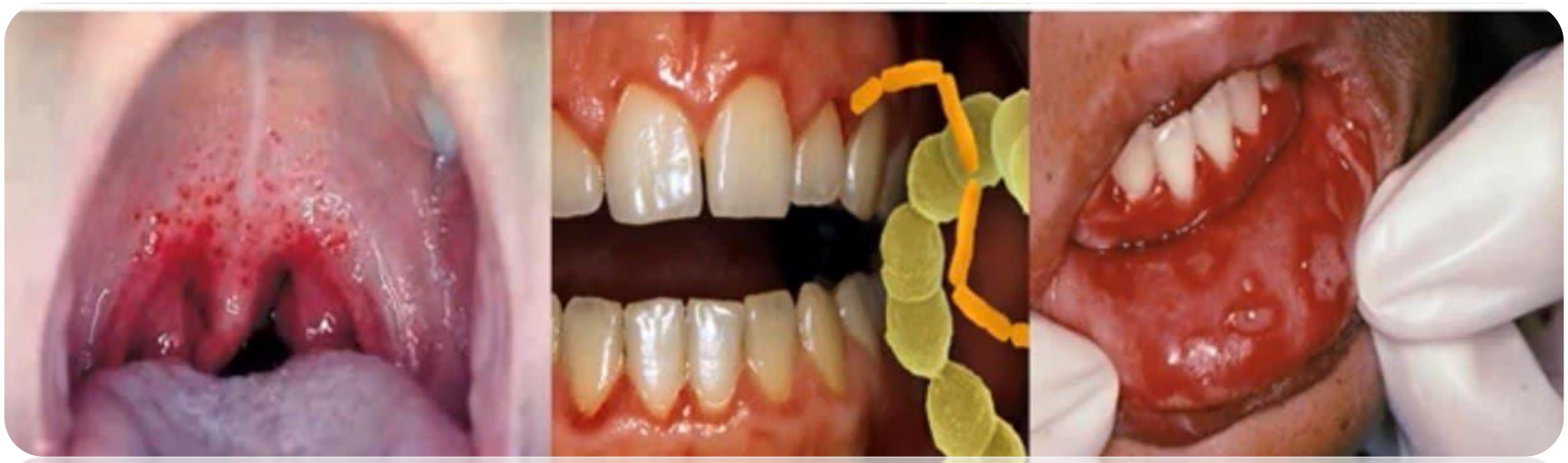


# 2.SPECIFIC INFECTIONS

## 2.1 Bacterial origin

## 2.2 Viral origin

## 2.3 Fungal origin



# 2.1 Bacterial origin

Gingivitis caused by a specific bacterial infection may, however, arise due to a loss of homeostasis between non–plaque-related pathogens and innate host resistance.

- ✓ Neisseria gonorrhoeae
- ✓ Treponema pallidum
- ✓ Mycobacterium tuberculosis

## 2.2 Viral origin

The most important viruses to cause gingival manifestations are Coxsackie viruses and the herpes viruses including herpes simplex virus types 1 (HSV-1) and 2 (HSV-2) and varicella-zoster virus. These infections may be accompanied by fever, malaise, and regional lymphadenopathy.

a) Herpes virus infections

1) Primary herpetic gingivostomatitis

2) Recurrent oral herpes

b) Oral Epstein- Barr virus lesions

c) Varicella- Zoster infections

d) Other

## Herpetic gingivostomatitis

- occurs in infants
- an incubation period of 1 week.
- an asymptomatic course in early childhood, but it may also give rise to gingivostomatitis with severe manifestations.

- **A characteristic feature is the formation of few or many vesicles, which rupture, coalesce, and leave fibrin-coated ulcers often of irregular extension.**

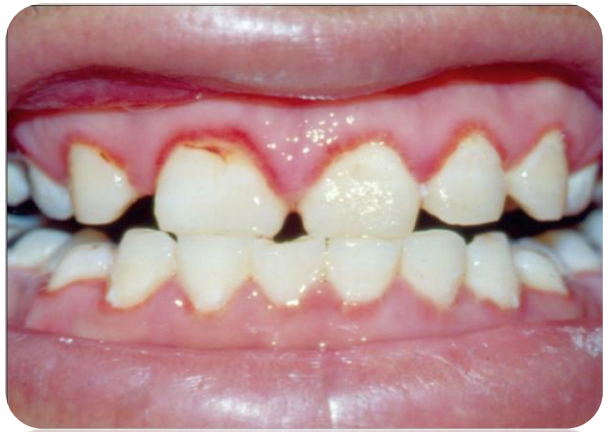
# 2.3 Fungal origin

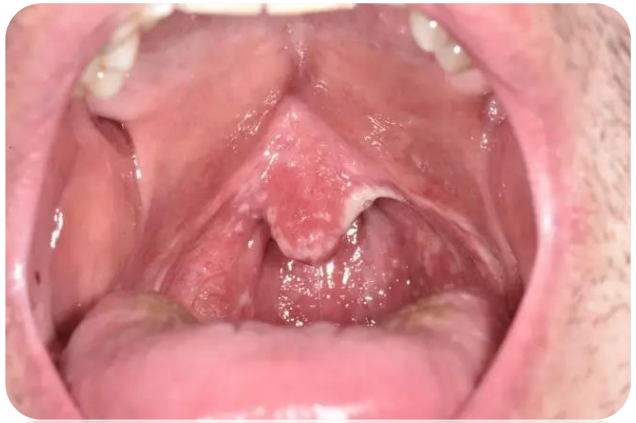
- **Candidiasis**

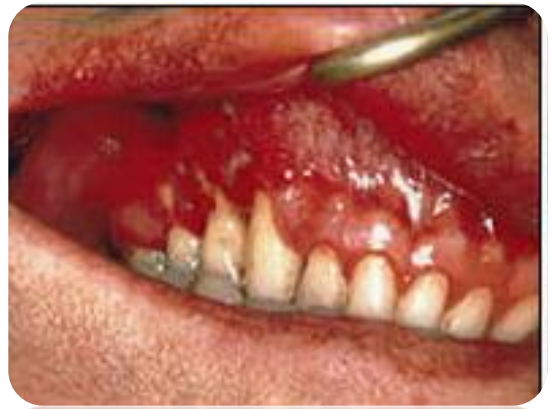
- The most common fungal infection of the oral mucosa is candidosis mainly caused by C. albicans.

- C. albicans is a normal commensal organism of the oral cavity but also an opportunistic pathogen.

- The most common clinical characteristic of gingival candidal infection is redness of the attached gingiva, often with a granular surface





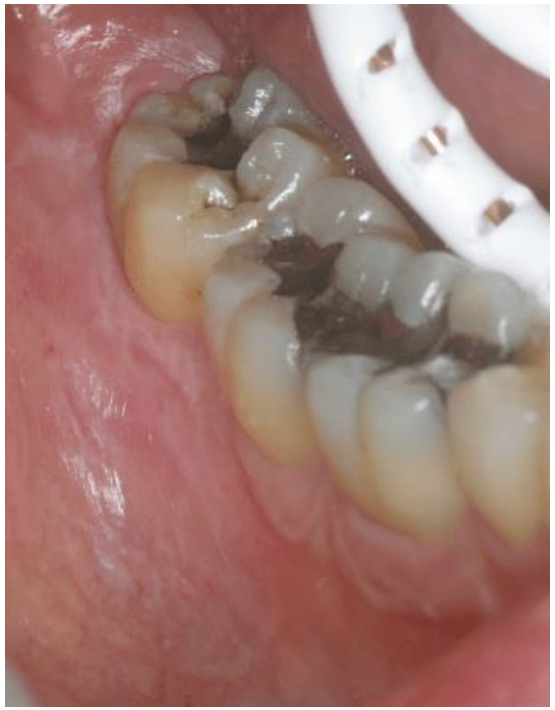


# 3 INFLAMMATORY AND IMMUNE CONDITIONS AND LESIONS.

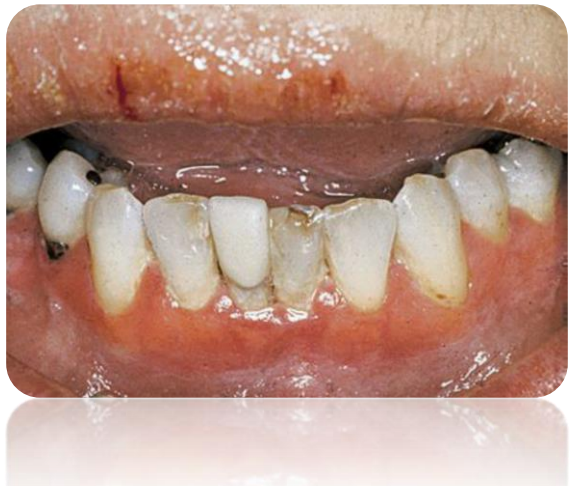
## 3.1 Hypersensitivity reactions

- **Contact allergy**

Oral mucosal manifestations of hypersensitivity (allergy) are very uncommon. such reactions may be due to dental restorative materials, dentifrices, mouthwashes, and foods and are most often type IV hypersensitivity reactions (contact allergy).



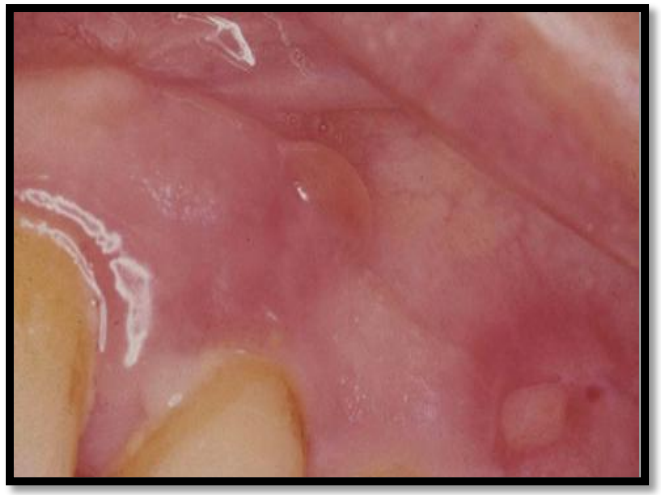
- **Plasma cell gingivitis.**



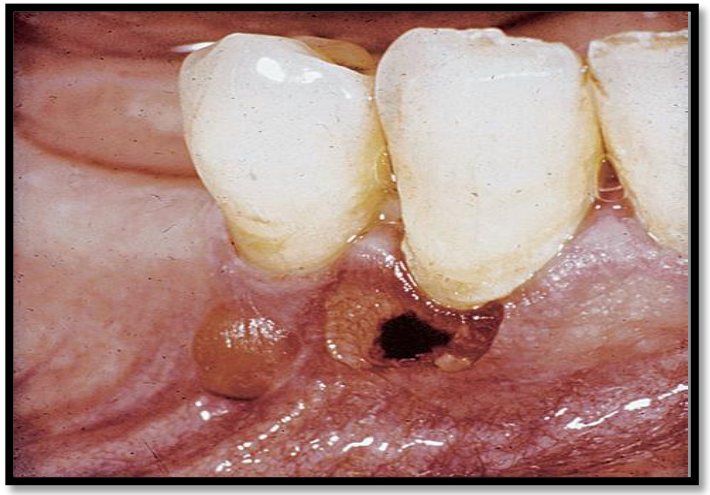
- **Erythema multiforme (EM).**

## 3.2 Autoimmune diseases of skin and mucous membranes

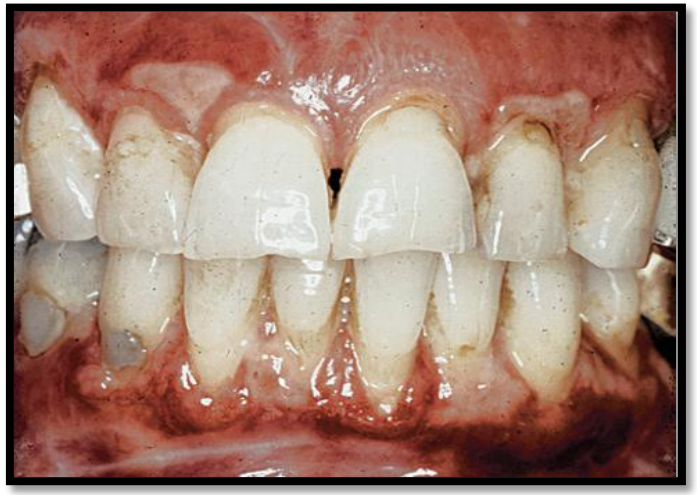
**Pemphigus vulgaris (PV)**



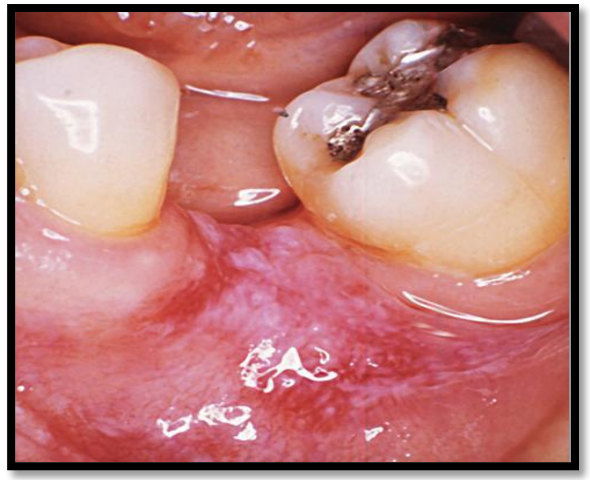
**Pemphigoid**



**Lichen planus**



**Lupus erythematosus (LE)**



# 4 REACTIVE PROCESSES

## 4.1 Epulides

Epulis is a term often applied to exophytic processes originating from the gingiva. usually there are no symptoms, although the reactive processes are thought to represent an exaggerated tissue response to limited local irritation or trauma, and they are classified according to their histology. True epulides include:

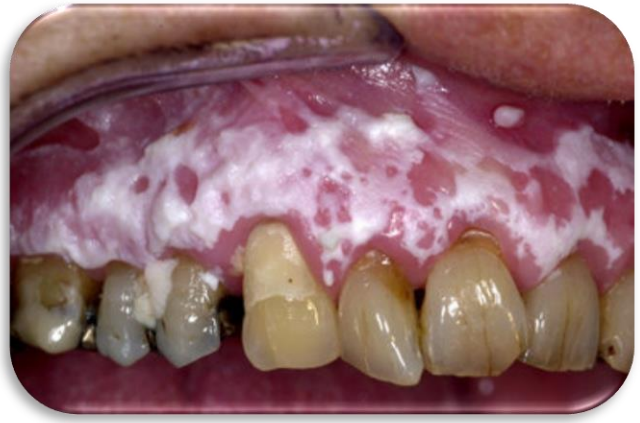
- Fibrous epulis.
- Calcifying fibroblastic granuloma.
- Pyogenic granuloma (vascular epulis).
- Peripheral giant cell granuloma (or central).

# 5. NEOPLASMS

## 5.1 Premalignant

- Leukoplakia

The term “leukoplakia” refers to a white lesion of the oral mucosa that cannot be characterized as any other definable lesion.



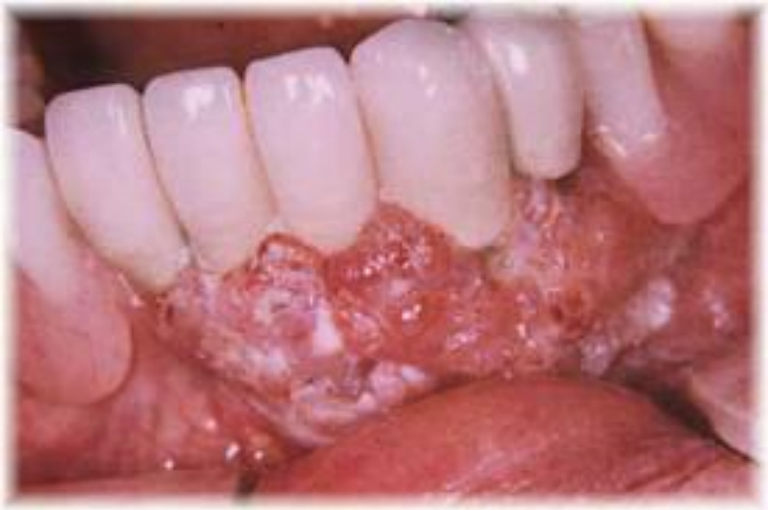
- Erythroplakia

Erythroplakia is the red counterpart of leukoplakia in the sense that it is a red lesion, which cannot be diagnosed as any other disease. Erythroplakia usually has a higher premalignant potential.

# 5.2 Malignant

- **Squamous cell carcinoma**

Squamous cell carcinoma of the gingiva represents about 20% of intraoral carcinomas and occurs most frequently in the mandibular premolar and molar regions. Mobility of adjacent teeth is common, and invasion of the underlying alveolar bone is apparent in approximately 50% of cases.



S.C.C.

- **Leukemia**

- **Lymphoma**

# 6. ENDOCRINE, NUTRITIONAL, AND METABOLIC DISEASES

## 6.1 Vitamin deficiencies

Vitamin C deficiency (scurvy)

Ascorbic acid (vitamin C) is necessary for various metabolic processes in the connective tissue Clinically, scurvy is characterized by gingival bleeding and soreness.

# 7. TRAUMATIC LESIONS

## 7.1 Physical/mechanical insults

These traumatic lesions may be accidental or result from inappropriate oral hygiene procedures, inadequate dental restorations, poorly designed dental appliances, and orthodontic bands and devices

## 7.2 Chemical (toxic) insults

Toxic chemical products may result in mucosal surface erosions, including reactions of the gingiva. Surface sloughing or ulceration may be related to the use of chlorhexidine, acetylsalicylic acid, cocaine, hydrogen peroxide, or to dentifrice detergents.

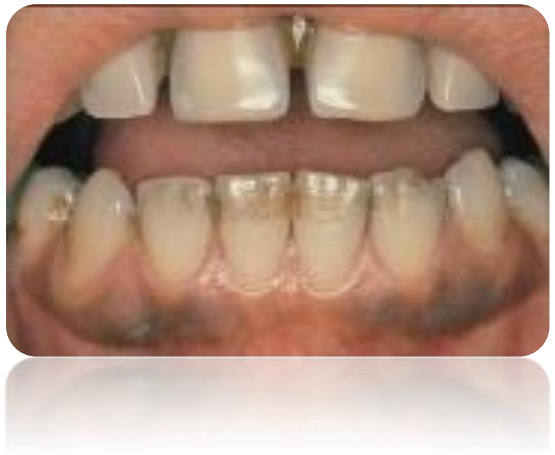
## 7.3 Thermal insults

Thermal burns of the gingiva may be prevalent due to a hurried lifestyle with intake of microwave-heated foods and drive-through coffee shops.

# 8. GINGIVAL PIGMENTATION/melanoplakia

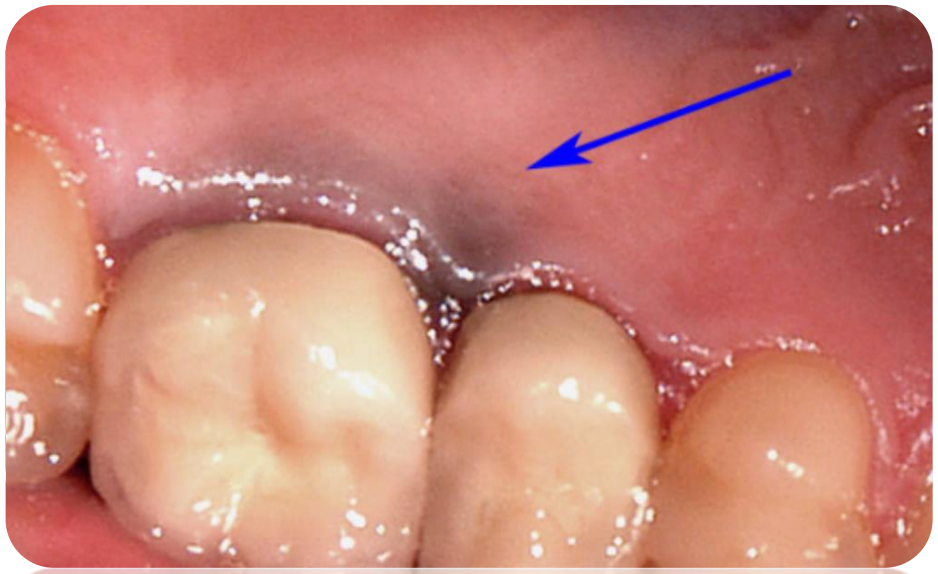
- **Smoker's melanosis**

A primary etiologic factor in melanocytic pigmentation of the oral mucosa is cigarette smoking. Smoker's melanosis occurs most frequently on the mandibular anterior facial gingiva. Melanosis gradually improves or may completely resolve upon cessation of smoking.



- **Amalgam tattoo**

Pigmentation of the oral mucosa due to amalgam is frequently seen in the gingiva and alveolar mucosa. The lesion is a well-de- fined bluish, blackish, or greyish discoloration, which is not elevated. Radiographic imaging may demonstrate underlying amalgam debris



- **Drug-induced pigmentation (DIP)**

# Reference


- HOLMSTRUP, P., PLEMONS, J. & MEYLE, J. J. J. O. C. P. 2018. Non–plaque-induced gingival diseases. 45, S28-S43.


Thank You
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