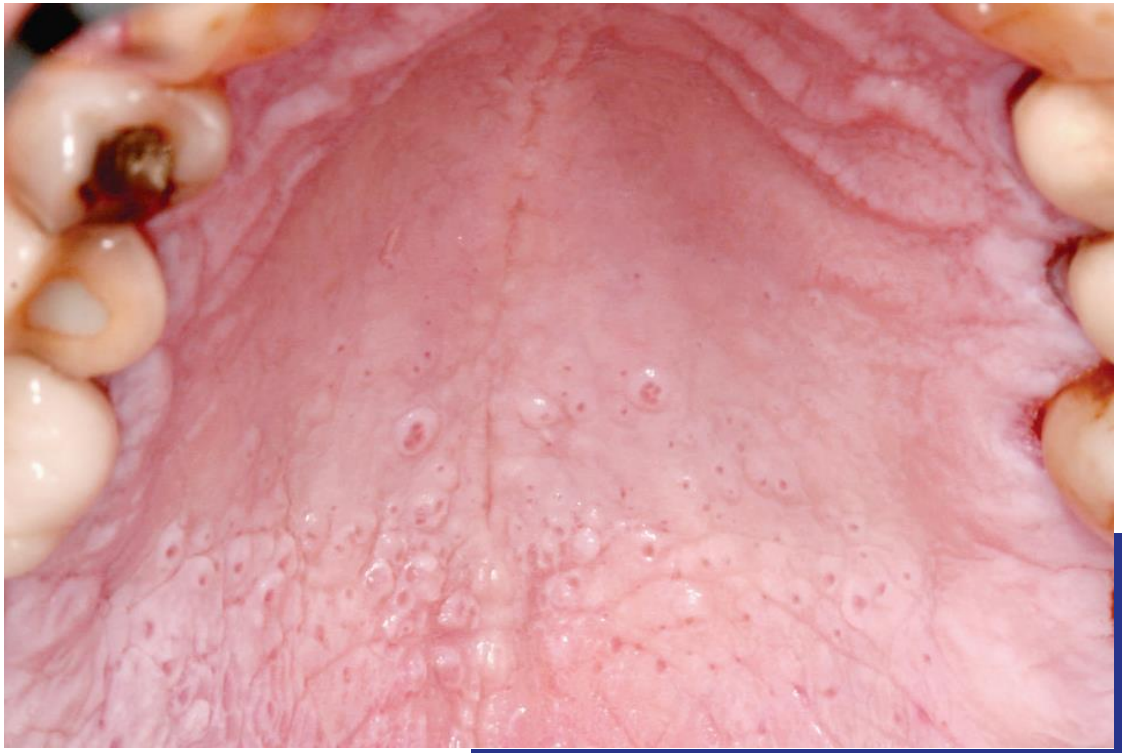

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Oral Pathology module

Physical and Chemical Injuries of the Oral Cavity

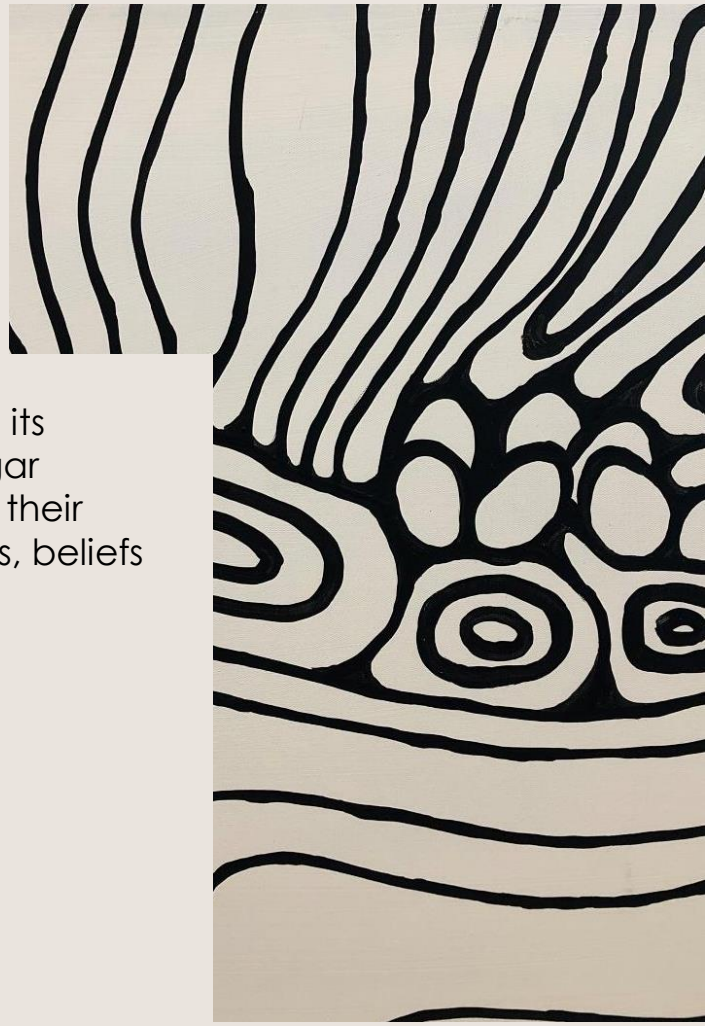
A/Prof Omar Kujan

BDS DipOPath MDSc MFDS RCPS FHEA FRCPath PhD

DENT4217

Acknowledgement of country

The University of Western Australia acknowledges that its campus is situated on Noongar land, and that Noongar people remain the spiritual and cultural custodians of their land, and continue to practise their values, languages, beliefs and knowledge.



Artist: Dr Richard Barry Walley OAM

Learning outcomes

1. Describe common physical and chemical lesions of the oral mucosa.
2. Identify the aetiology of the oral mucosa's associated physical and chemical lesions.

Cell injuries

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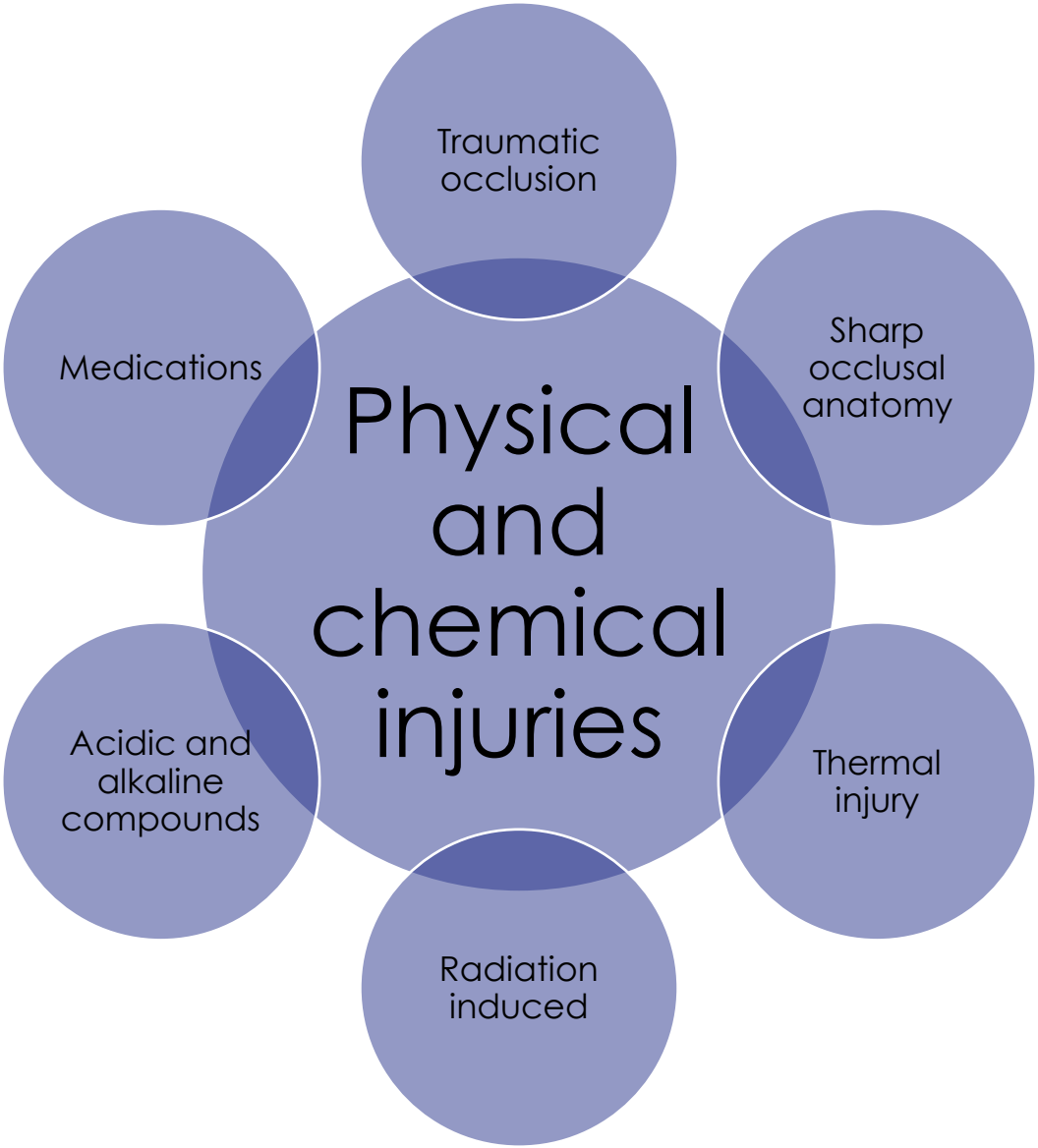
Types of Injuries (aetiology)

- Physical (e.g., surgical trauma)
- Chemical (e.g., exposure to caustic substances)
- Thermal (e.g., burns)
- Infectious (e.g., microorganisms)

- Clinically, the patient's chief complaint includes a painful area or a "lump".

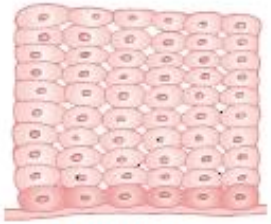
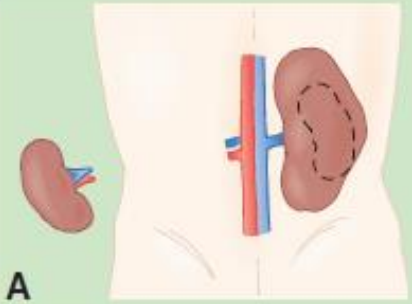
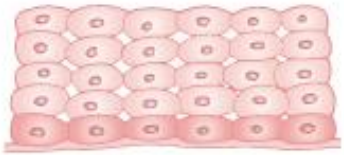

Traumatic occlusion
Sharp occlusal anatomy
Thermal injury
Radiation induced
Acidic and alkaline compounds
Medications
Physical and chemical injuries

Cell injuries

Types of cell injuries (biological response)	
Reversible	**Irreversible**
Hyperplasia	Necrosis
Hypertrophy	Ischemia
Atrophy	Apoptosis
Metaplasia	Free radical damage
Dysplasia	Pathologic calcification
Intracellular pigments	Cell ageing

Definitions

- **Hyperplasia** is an increase in the size of an organ or tissue due to an increase in the number of cells (physiologic or pathologic)
- **Hypertrophy** is an increase in size of a tissue or body part due to the increase in the size of individual cells (physiologic or pathologic).
- **Atrophy** refers to decrease in size of cells, organs, tissues, or body parts because of disease, hormonal alteration, injury, or lack of use (physiologic or pathologic).
- **Metaplasia** is caused by a stimulus that changes one cell type into another.
- **Dysplasia** is defined as lack of proper maturation of a tissue. When cells are unable to mature, the tissue cannot properly develop.

	Physiologic	Pathologic
 Normal cells		
 Hyperplasia	 A	 B
 Hypertrophy	 C	 D
 Atrophy	 E	 F

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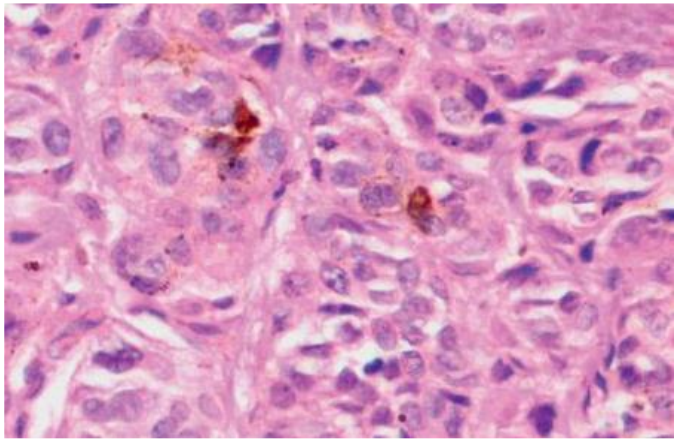
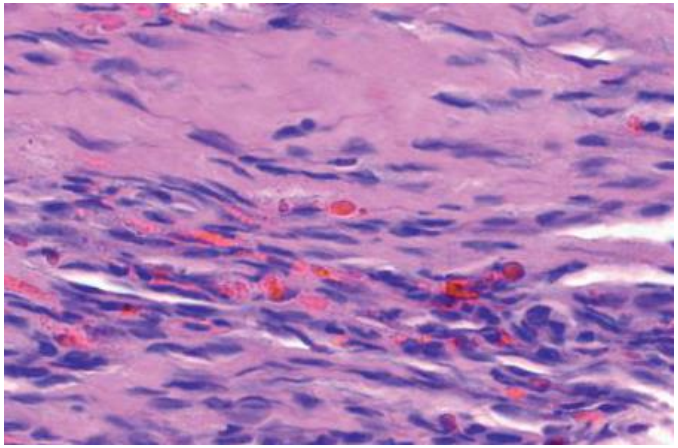
Endogenous Intracellular Pigments

- **Melanin** is produced by melanocytes located in the basal layer of the epithelium. It can be seen in the skin following lacerations and chronic inflammatory disorders as oral lichen planus.



- **Hemosiderin** is a pigment derived from hemoglobin of red blood cells. It is composed of ferric oxide. A localized deposit of hemosiderin is seen after bruising when the red blood cells break down and the hemoglobin is released into the surrounding tissues



Definitions

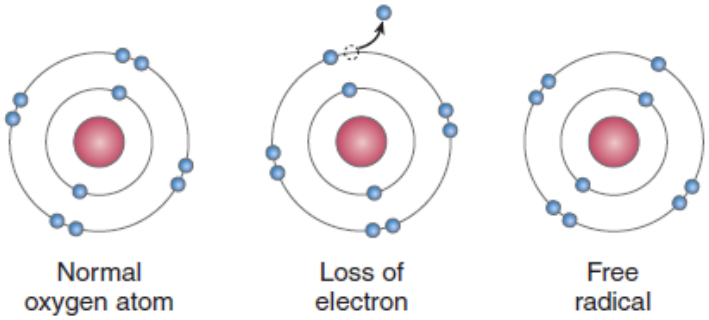
- **Ischemia** is a restriction in blood supply generally due to damaged blood vessels. The result is damage, dysfunction, or death of tissue supplied by that blood vessel, called **infarction**.

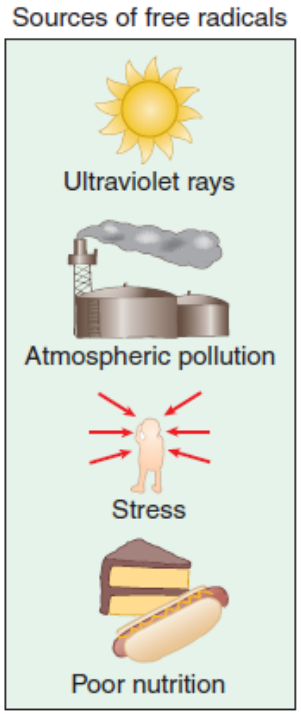
- **Necrosis** is the death of cells and tissues that fail to adapt to environmental changes. Pathologic necrosis occurs when normal cell functions cannot be sustained due to infection, toxins, trauma, or lack of oxygen.

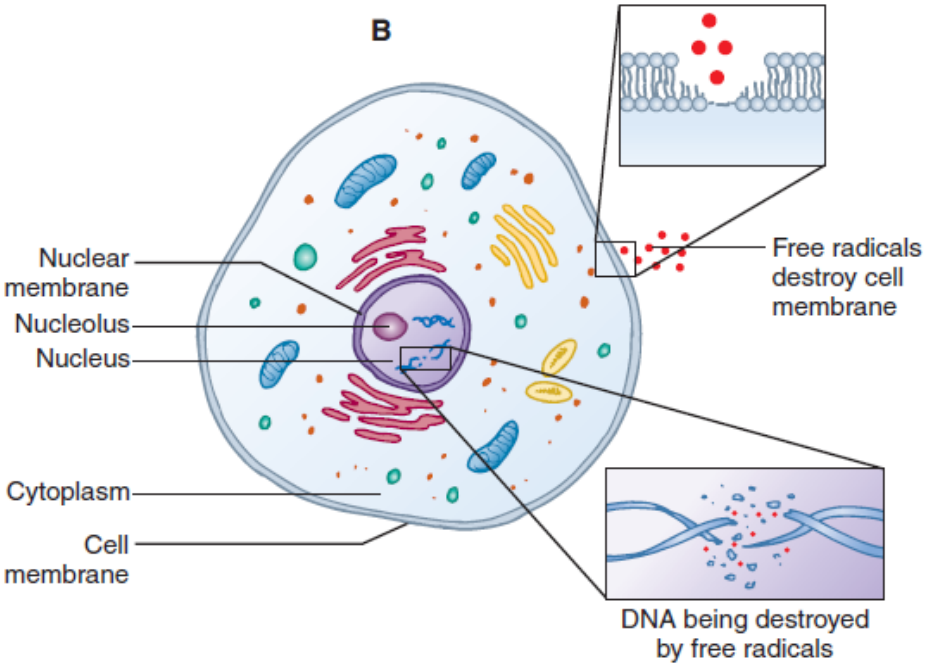
- **Apoptosis** is the process of programmed or planned cell death (tumours and inflammatory disorders). This contrasts with cell death due to injury.

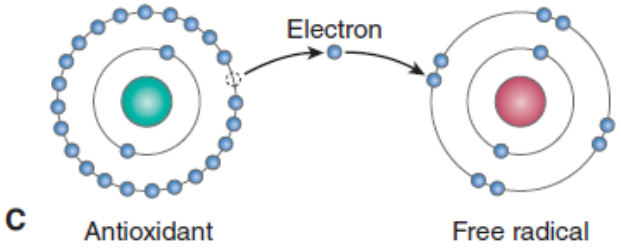
- **Free radical damage:** Free radicals inflict damage when they react with cell membranes or cellular DNA via peroxidation of membrane lipids, a process that has been implicated in some diseases and ageing.

- **Pathologic calcification:** abnormal deposition of calcium and may be dystrophic or metastatic**. Dystrophic calcification** can be observed in both vital and necrotic pulp tissues. Causes include irritation, injury, and reaction to the caries process.


A
Normal oxygen atom
Loss of electron
Free radical


Sources of free radicals
Ultraviolet rays
Atmospheric pollution
Stress
Poor nutrition


B
Nuclear membrane
Nucleolus
Nucleus
Cytoplasm
Cell membrane
Free radicals destroy cell membrane
DNA being destroyed by free radicals


Electron
C
Antioxidant
Free radical

Inflammation

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Table 2.2 Types of Inflammation

	Acute	Chronic	Granulomatous
Onset	Sudden	Gradual	Gradual
Duration	Short	Long-standing	Long-standing
Predominant cell type(s)	Neutrophils, macrophages	Lymphocytes, plasma cells, macrophages	Epithelioid histiocytes, multinucleated giant cells, lymphocytes
Clinical characteristics	Pain, erythema, and edema; fever	Loss of function	Nonpainful enlargement; nodules
Outcome	Abscess; resolution; progression to chronic inflammation	Fibrosis; tissue destruction	Medical or surgical intervention

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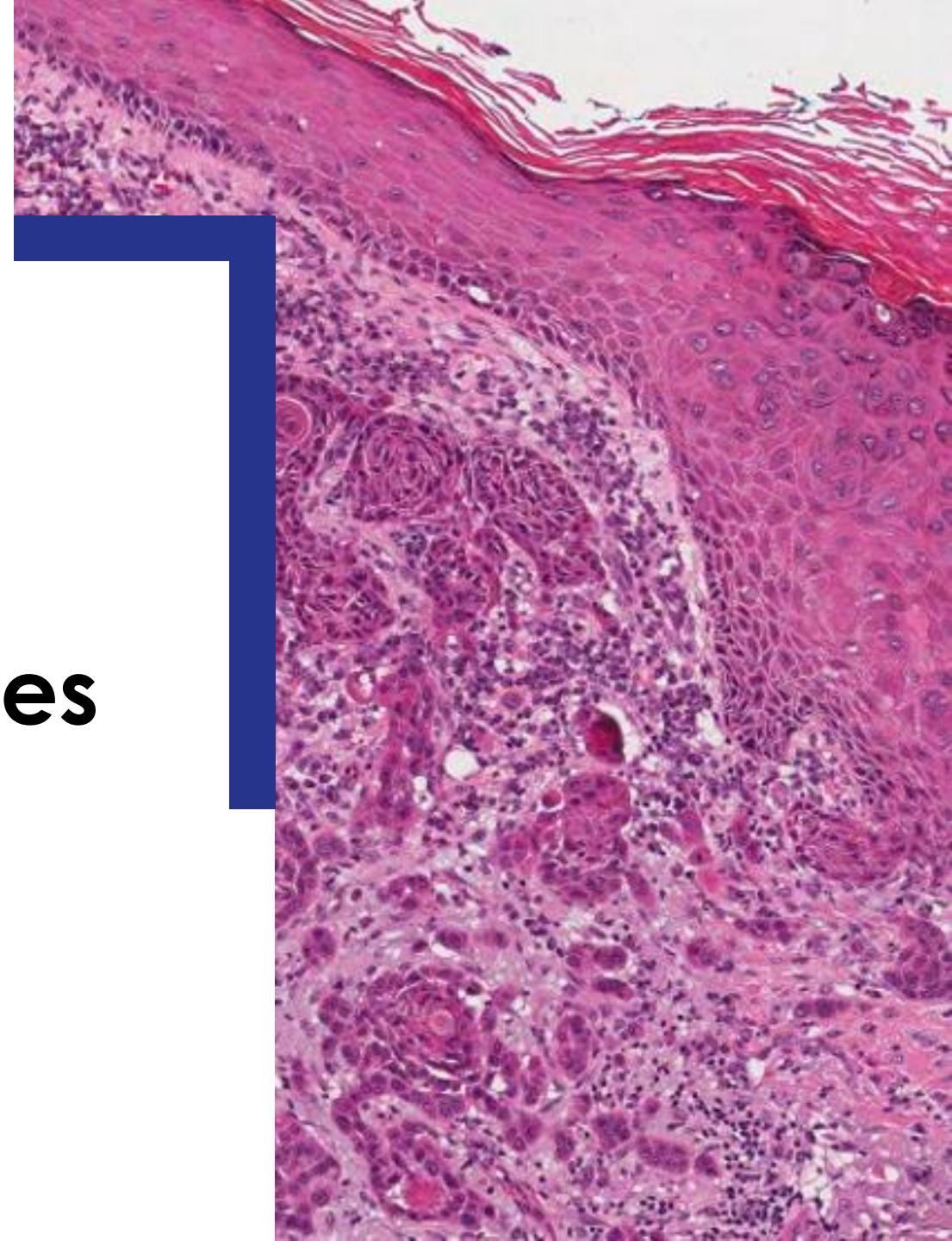
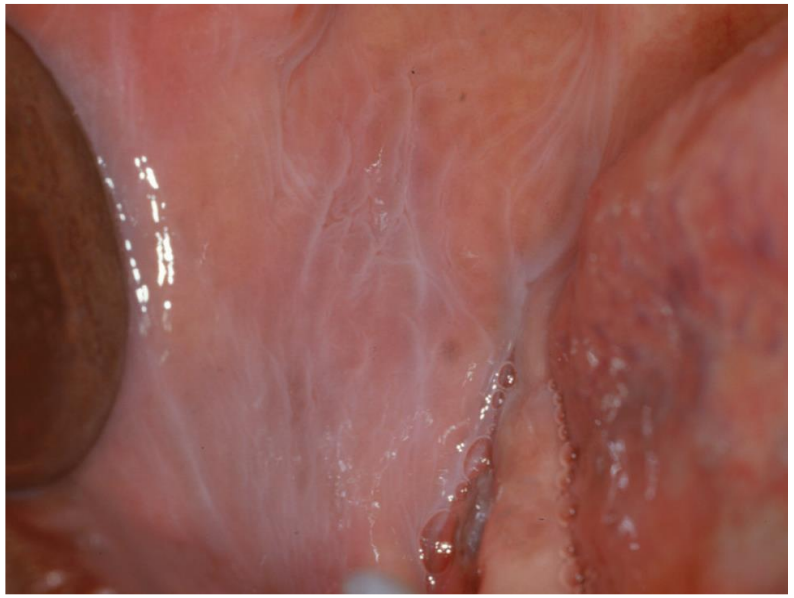
Physical and chemical injuries

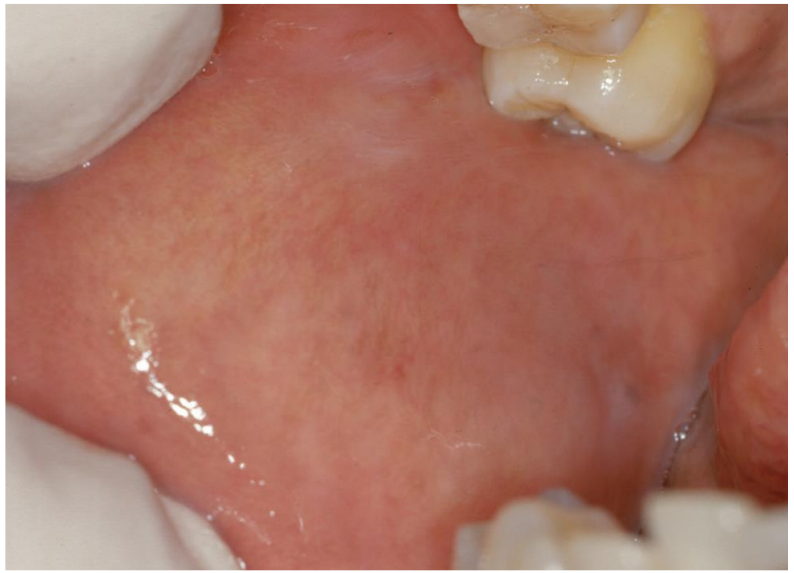


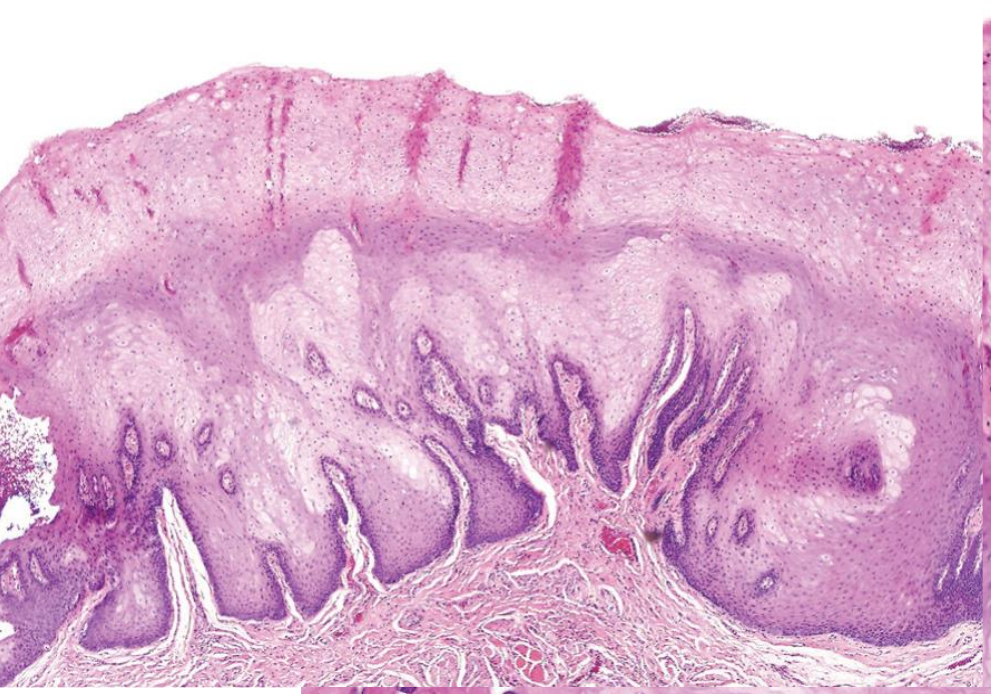
Leukoedema

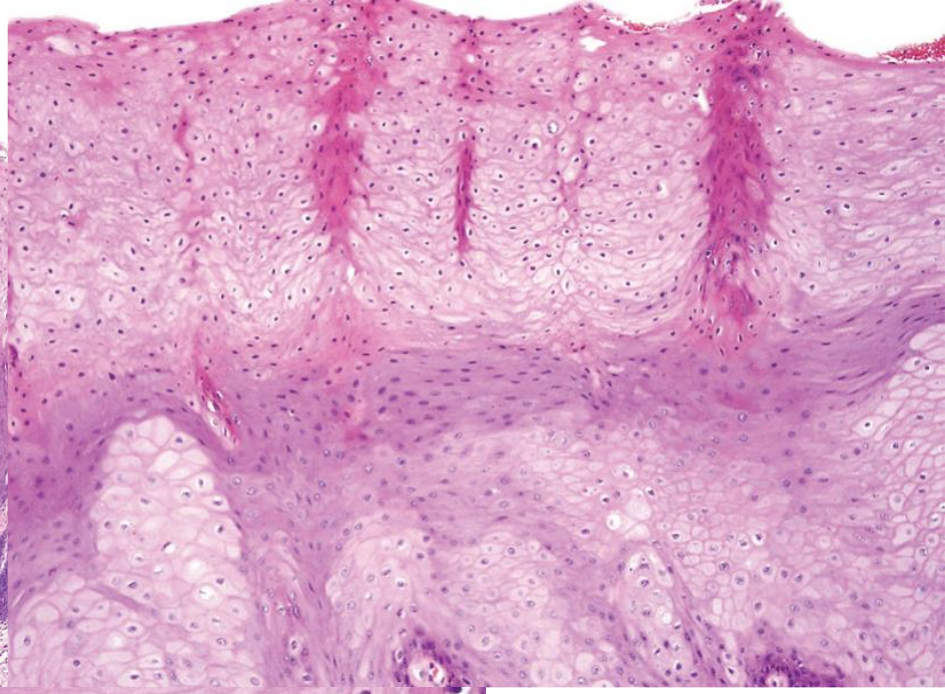
This term should only be reserved for clinical presentation; the histopathologic feature is keratinocyte edema.

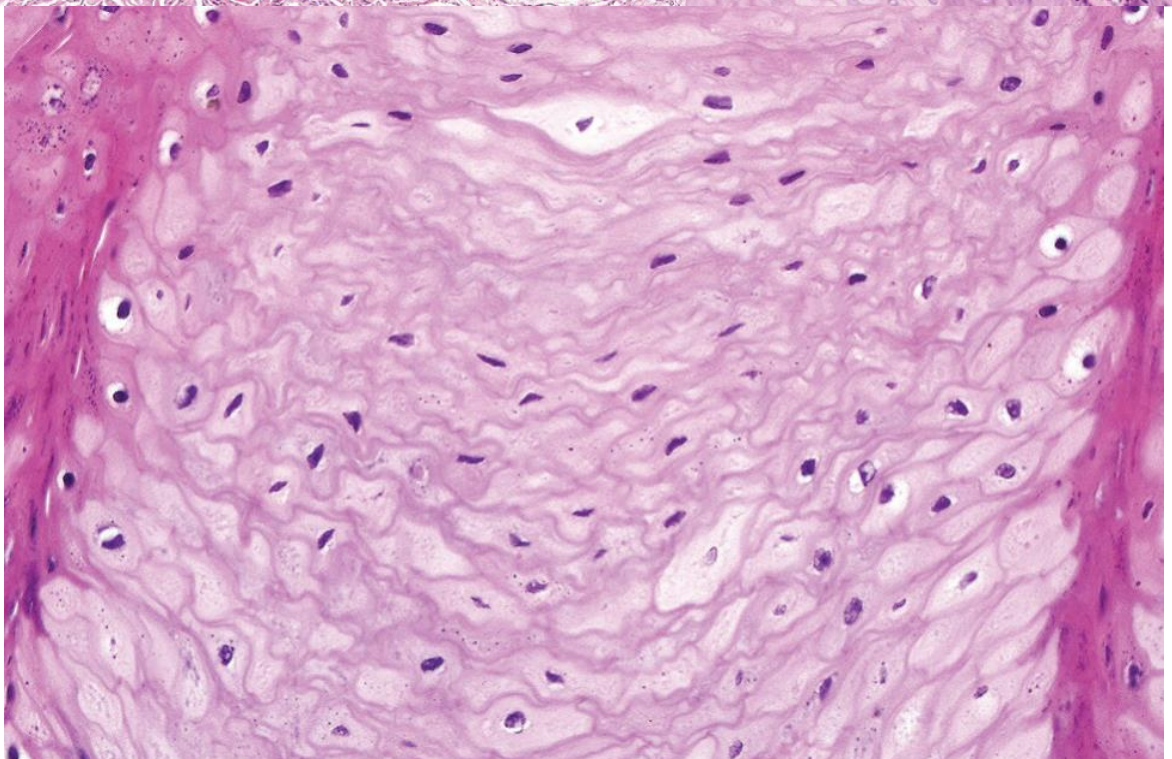
- **Clinical Findings**
  - This is present in up to 90% of the population and is more readily discerned in dark-skinned individuals.
  - Delicate lacy, grey-white lines are present on the buccal mucosa or tongue (nonkeratinized sites) that disappear with stretching







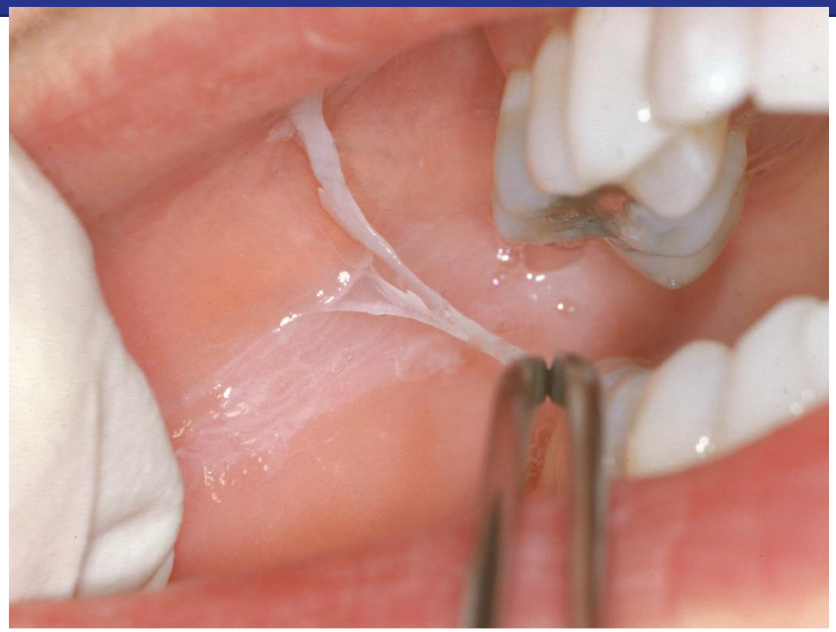


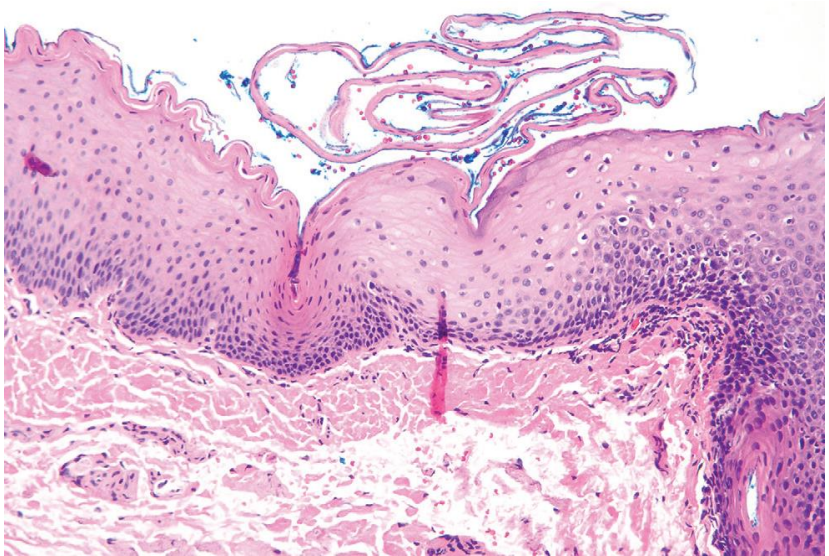


Contact Desquamation

- superficial injury to the mucosa caused by direct contact with a mild-to-moderately irritating or caustic toothpaste or mouthwash

- Strips of desquamated keratinocytes appear eosinophilic and degenerated or coagulated, and generally lie on the surface of the otherwise intact mucosa

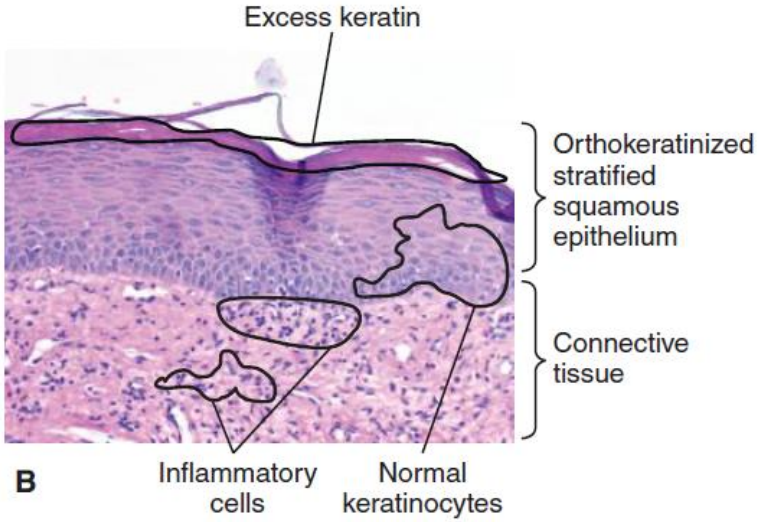


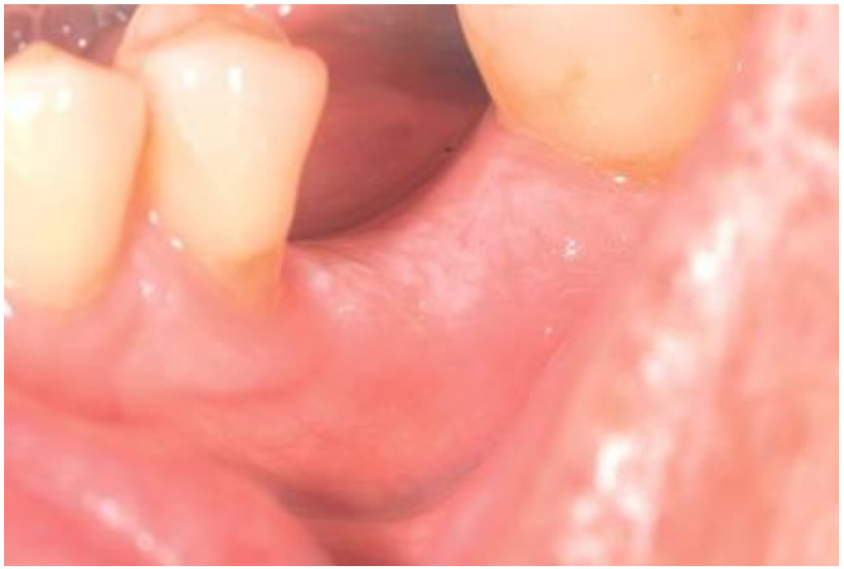


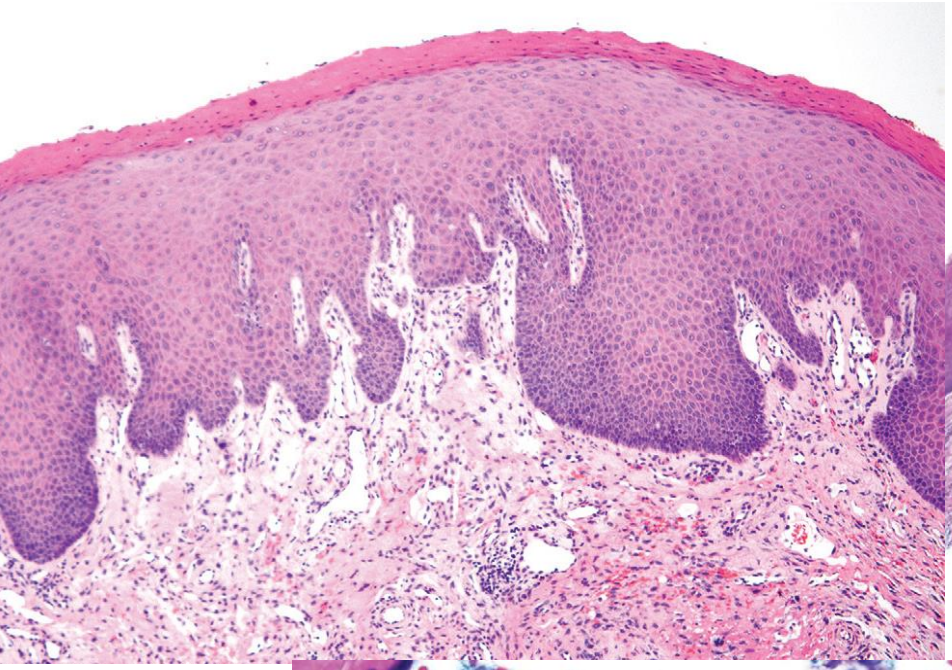
Frictional Keratosis

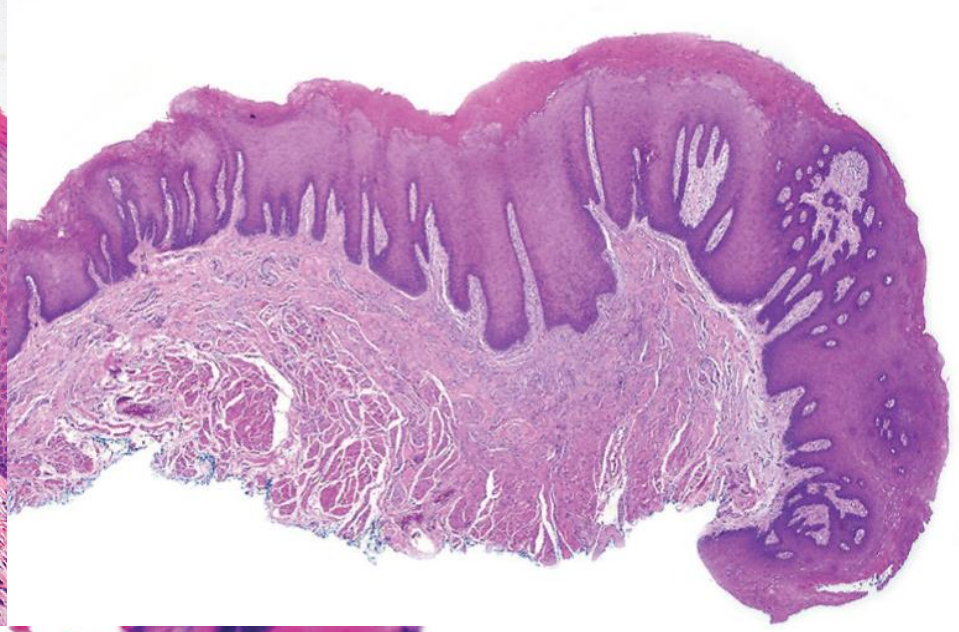
- **Frictional Keratosis**: benign reactive phenomenon occurs when the mucous membranes are repeatedly irritated over a prolonged period.

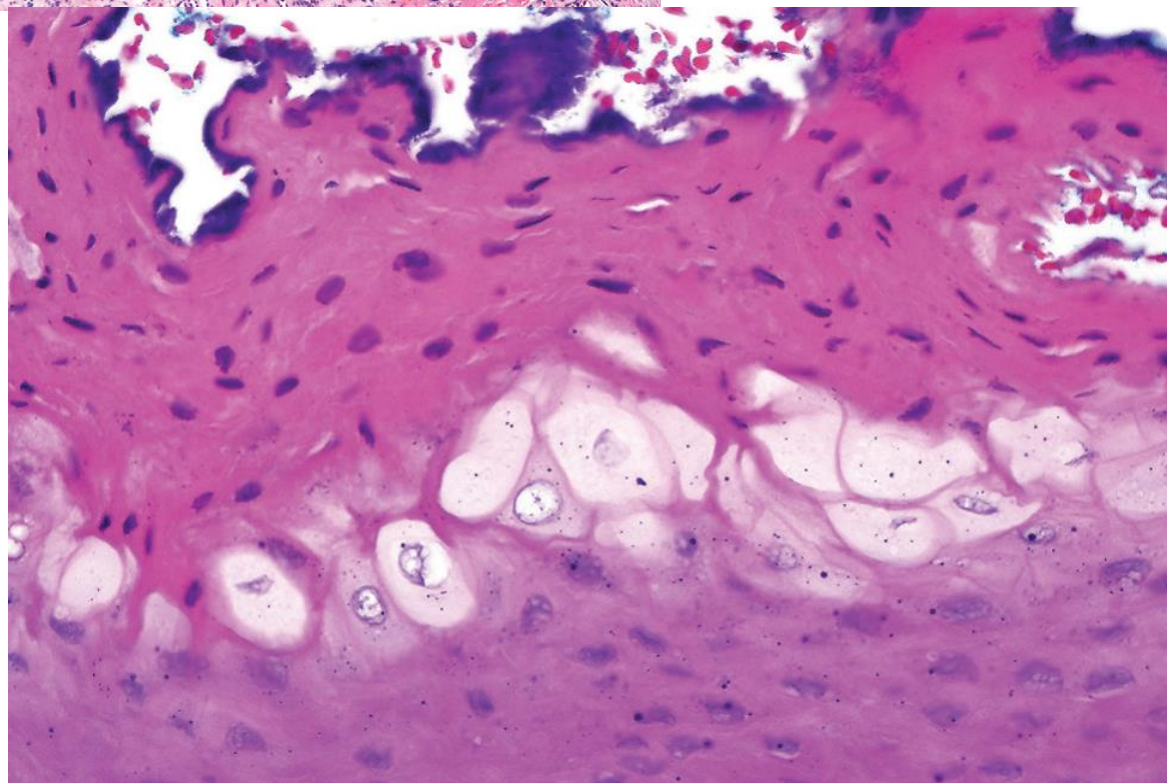
- It represents a protective mechanism that increases the thickness of the surface epithelium to protect the underlying tissues from persistent irritation.







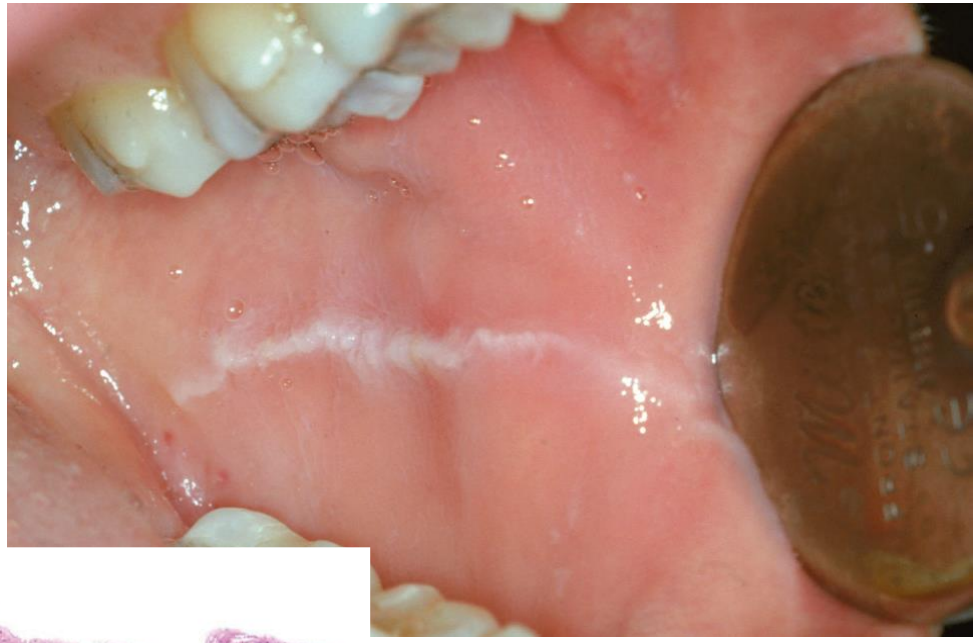


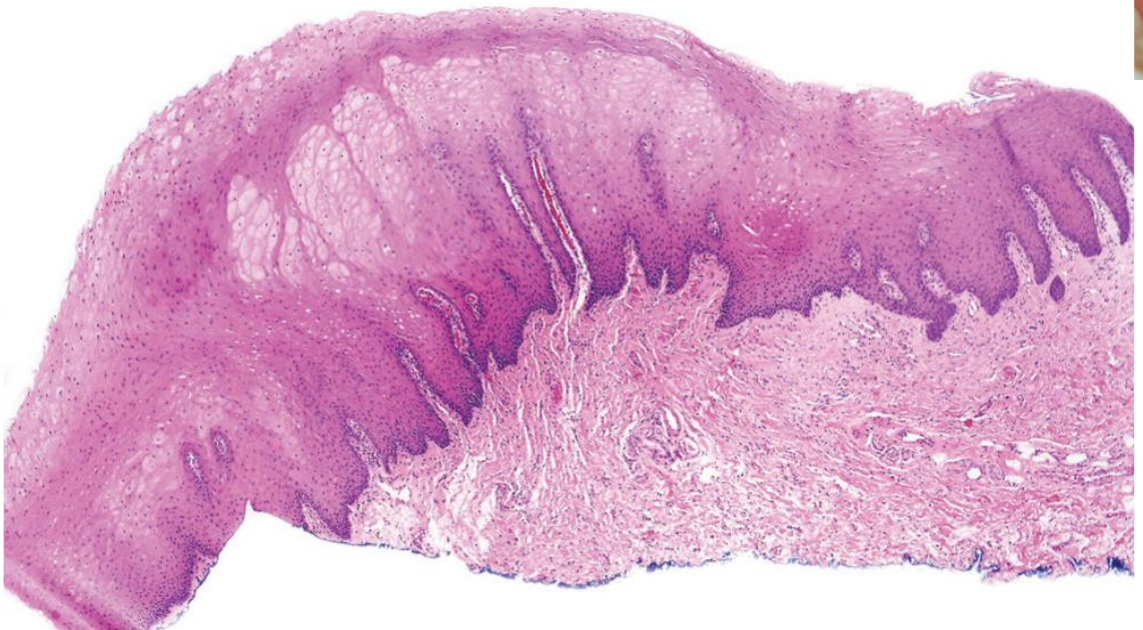


Linea alba

- Linea alba (“white line”) is a specific form of frictional keratosis seen as a thin, slightly raised white keratotic line along the occlusal plane on the buccal mucosa, and it is often bilateral.

- Linea alba is a variation of normal anatomy but may become prominent in some individuals.

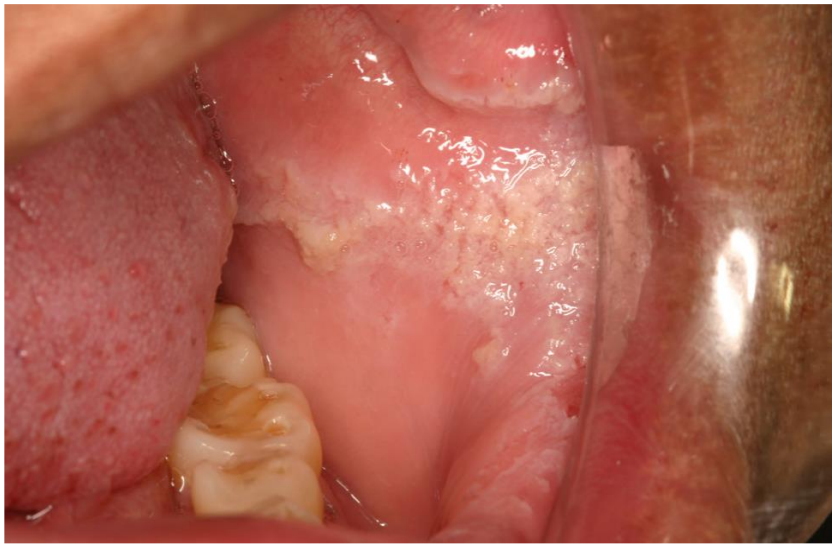


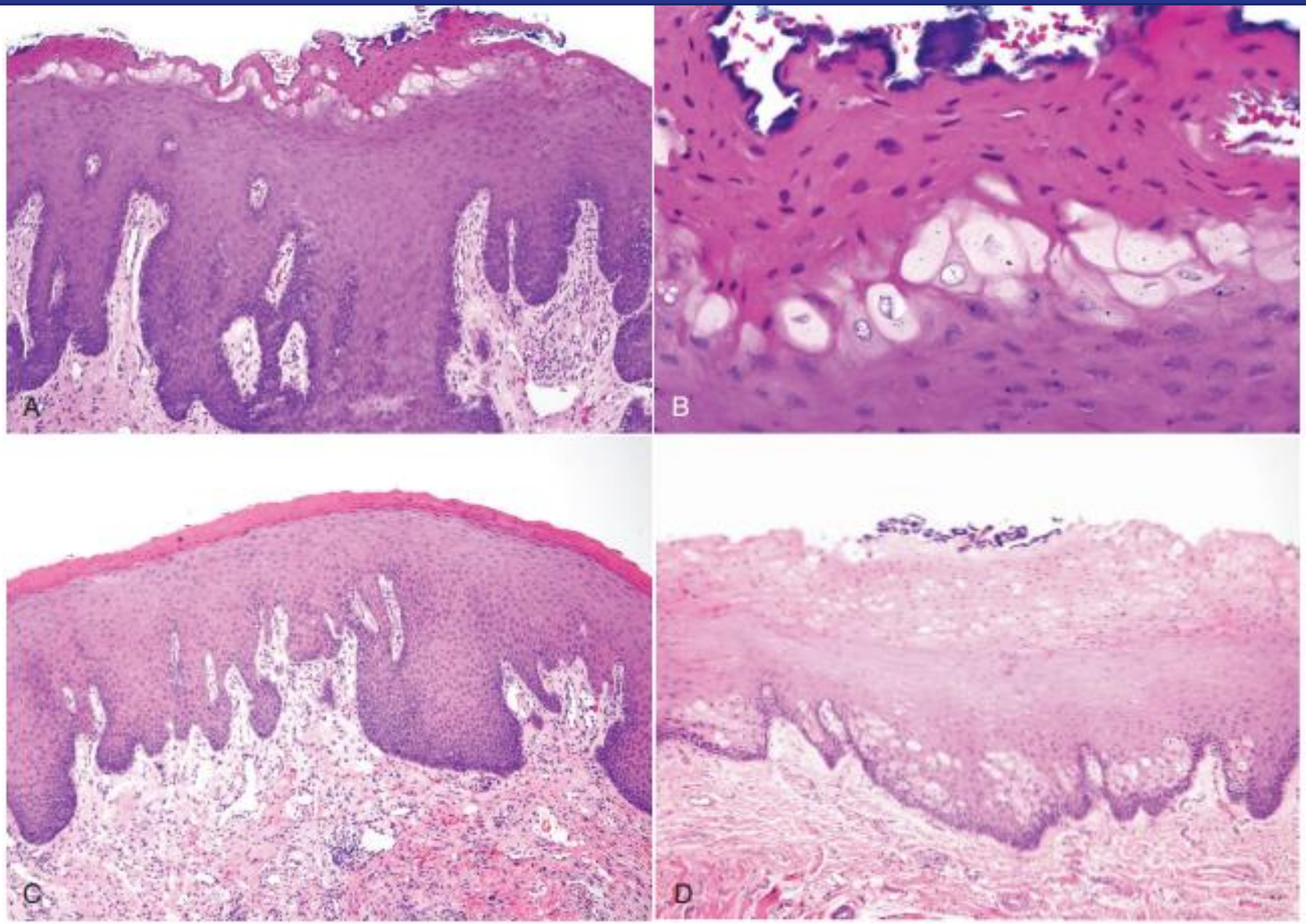


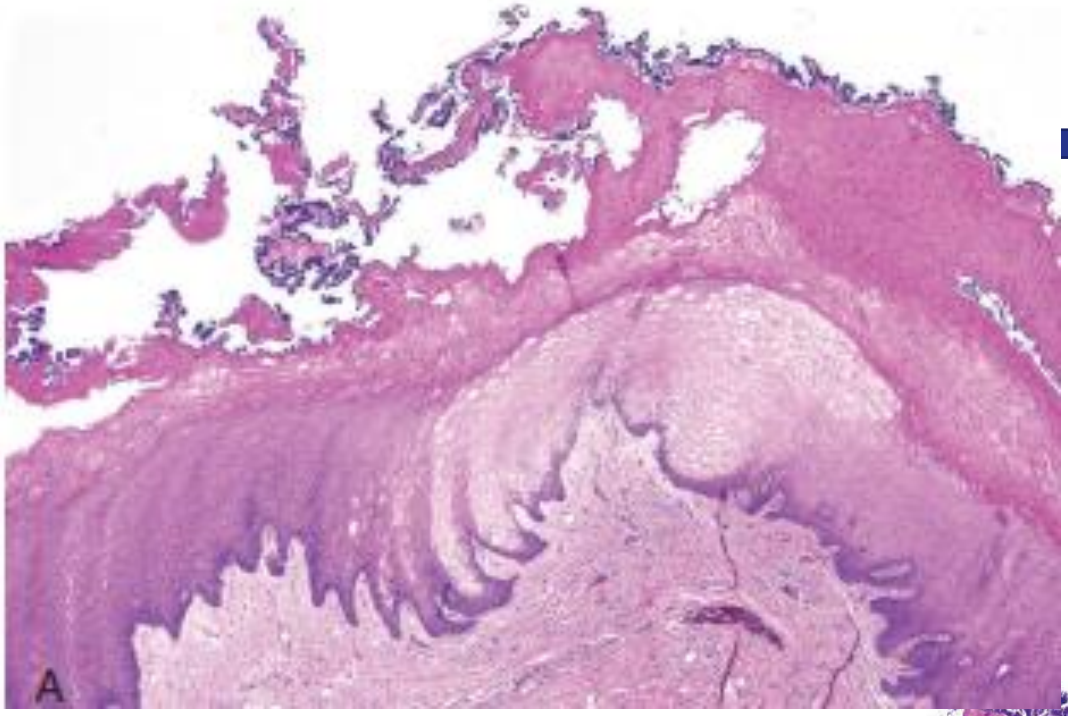
Morsicatio buccarum

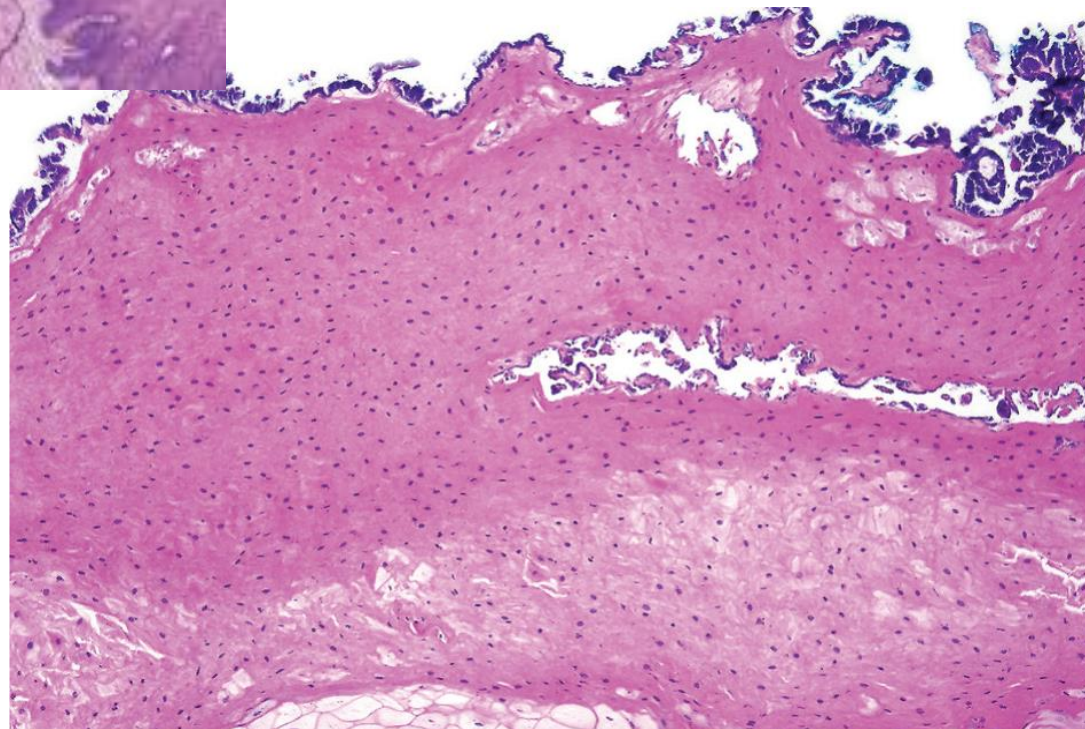
- Morsicatio buccarum, linguarum, and labiorum are forms of frictional keratosis caused by habitual chewing or nibbling of the cheek (morsicatio buccarum), tongue (morsicatio linguarum), and lip (morsicatio labiorum).

- Extra surface keratin builds up (hyperkeratosis) to minimise damage to the deeper tissues. The surface appears white and somewhat shredded. Cessation of the habit usually leads to regression of the lesion.




A
B
C
D

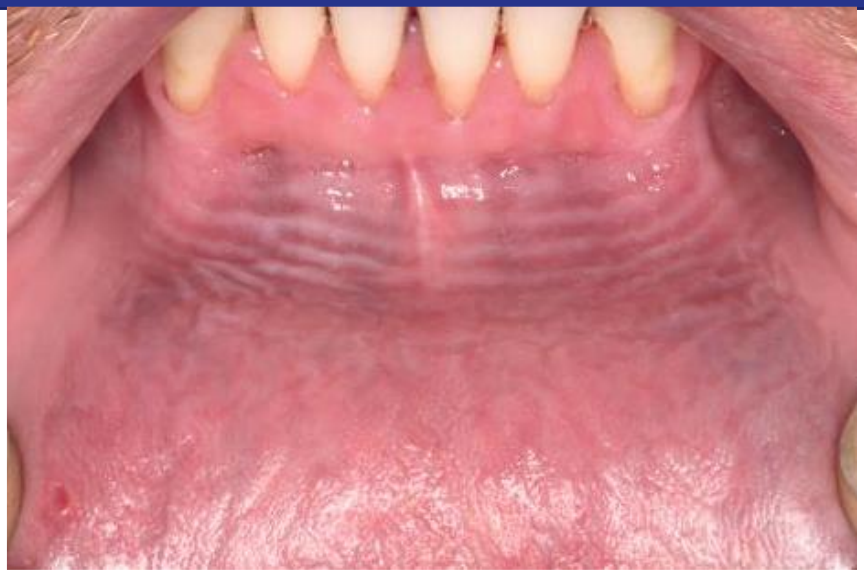

A

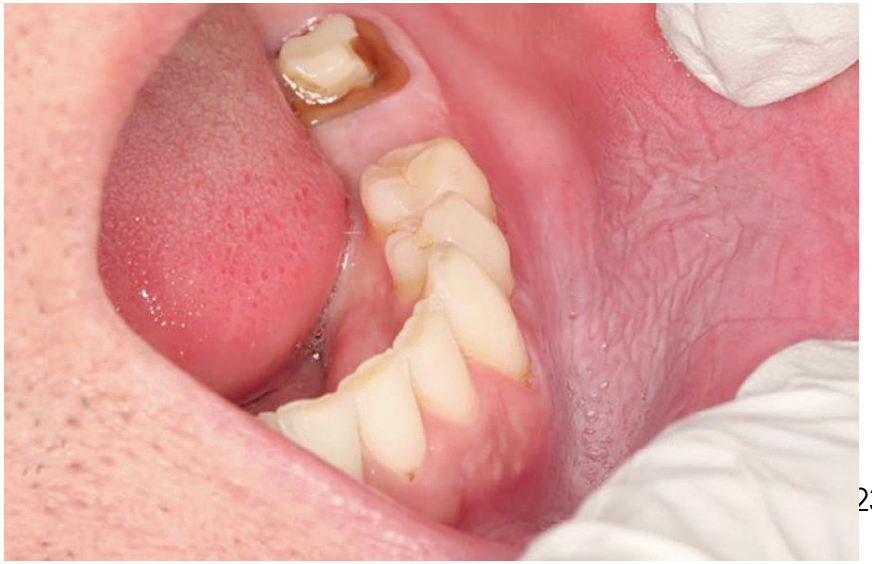


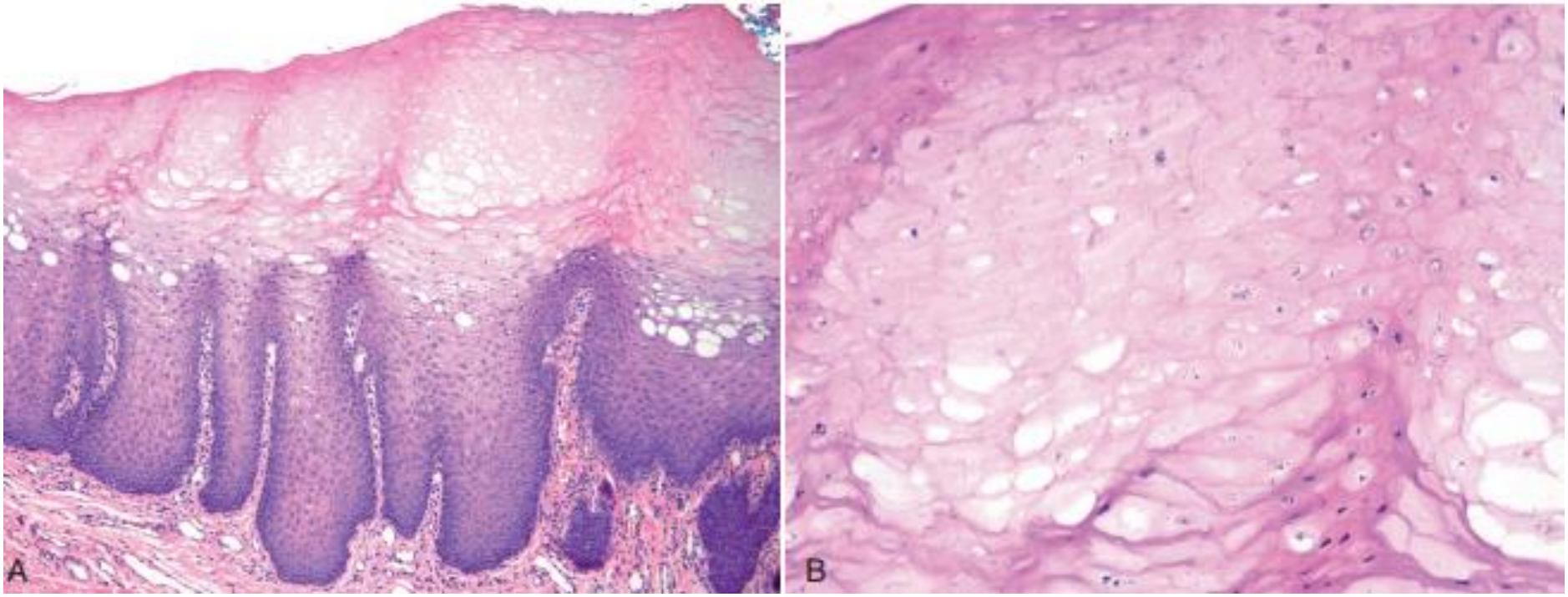
Snuff dippers keratosis

- **Snuff dippers keratosis** is seen in users of smokeless tobacco. White plaques develop in the vestibule where the tobacco directly touches the tissues. These lesions are characterised by thickened white mucosa that is typically wrinkled or corrugated.

- The clinical presentation may vary from an ill-defined area of white wrinkled thickening to deeply folded tissue with red patches. In addition to mucosal changes, gingival recession around the teeth may be pronounced.





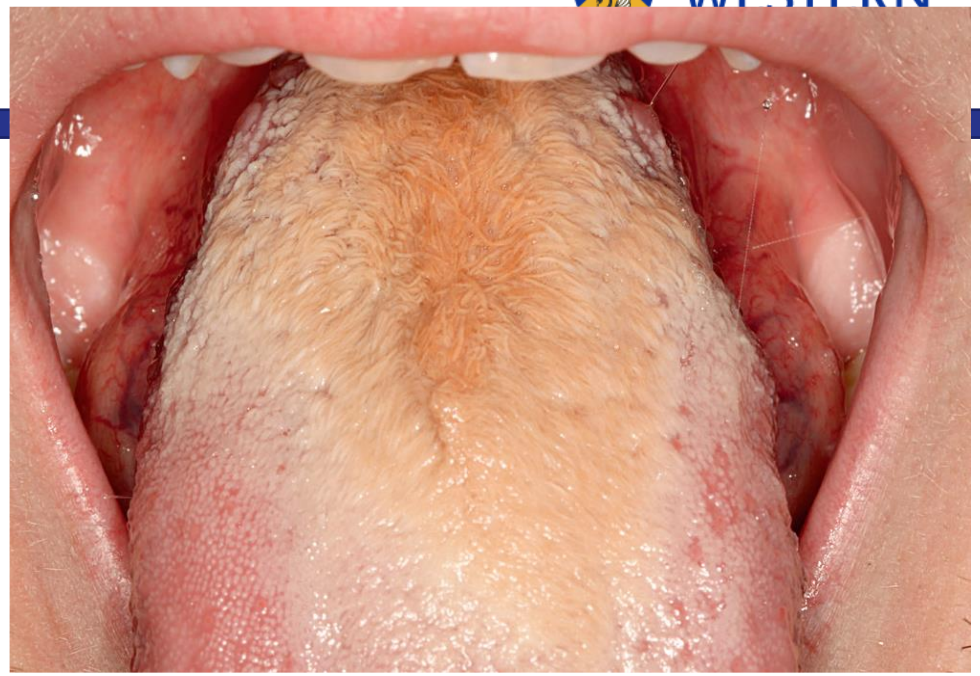

A
B

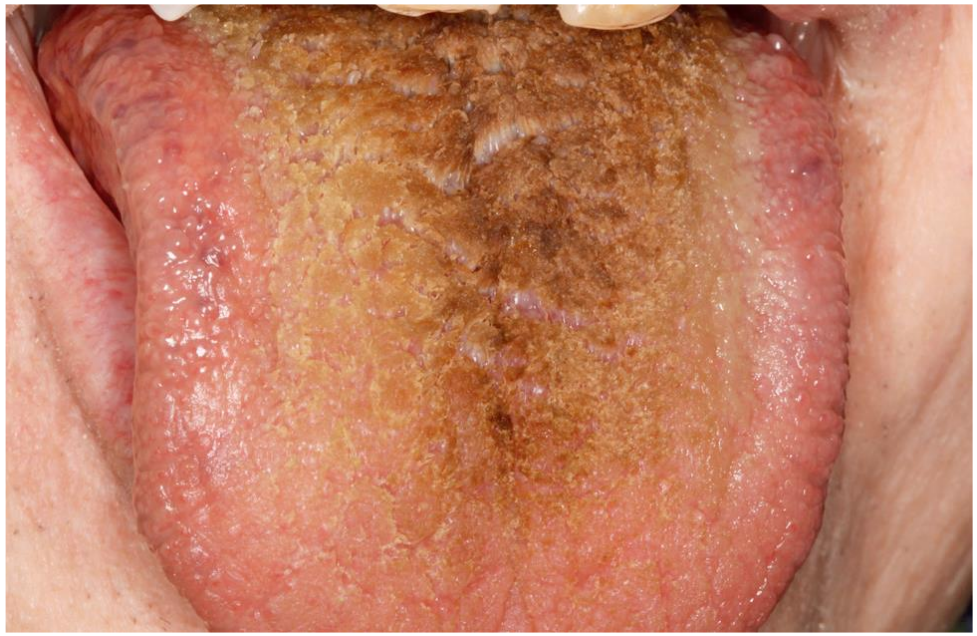
Black hairy tongue

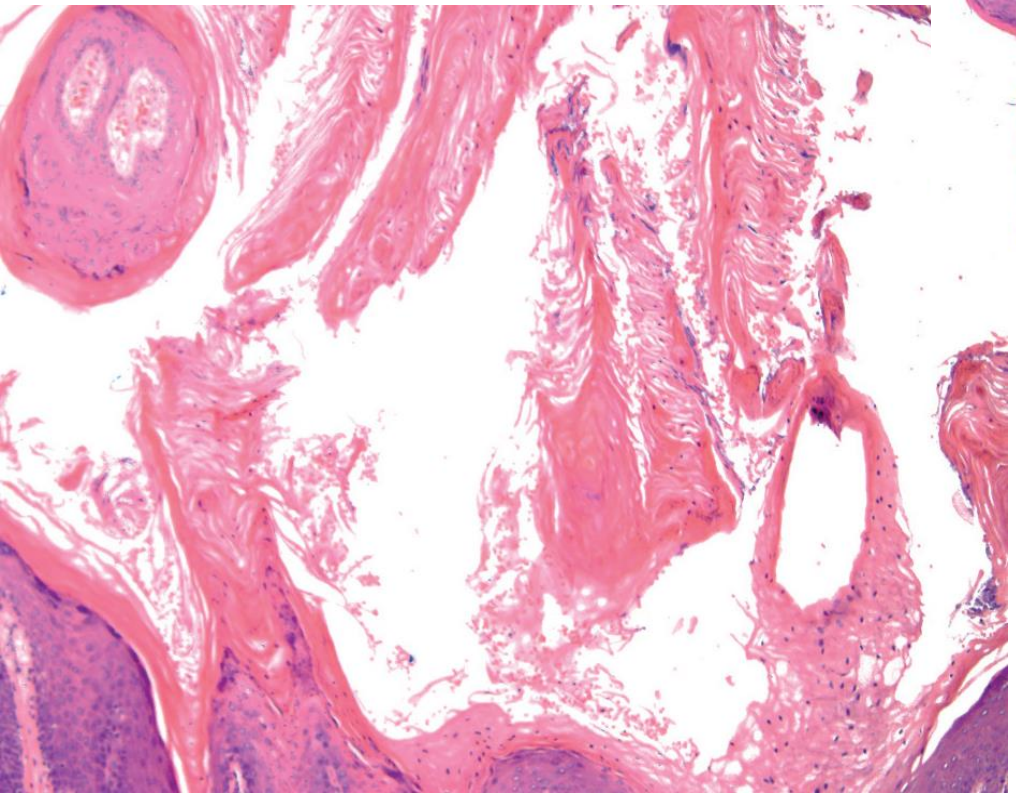
- Coated tongue is retention keratosis caused by dehydration with reduced watery and increased mucous saliva, and poor diet low in fresh fruits and vegetables, leading to retention of keratin rather than normal shedding.

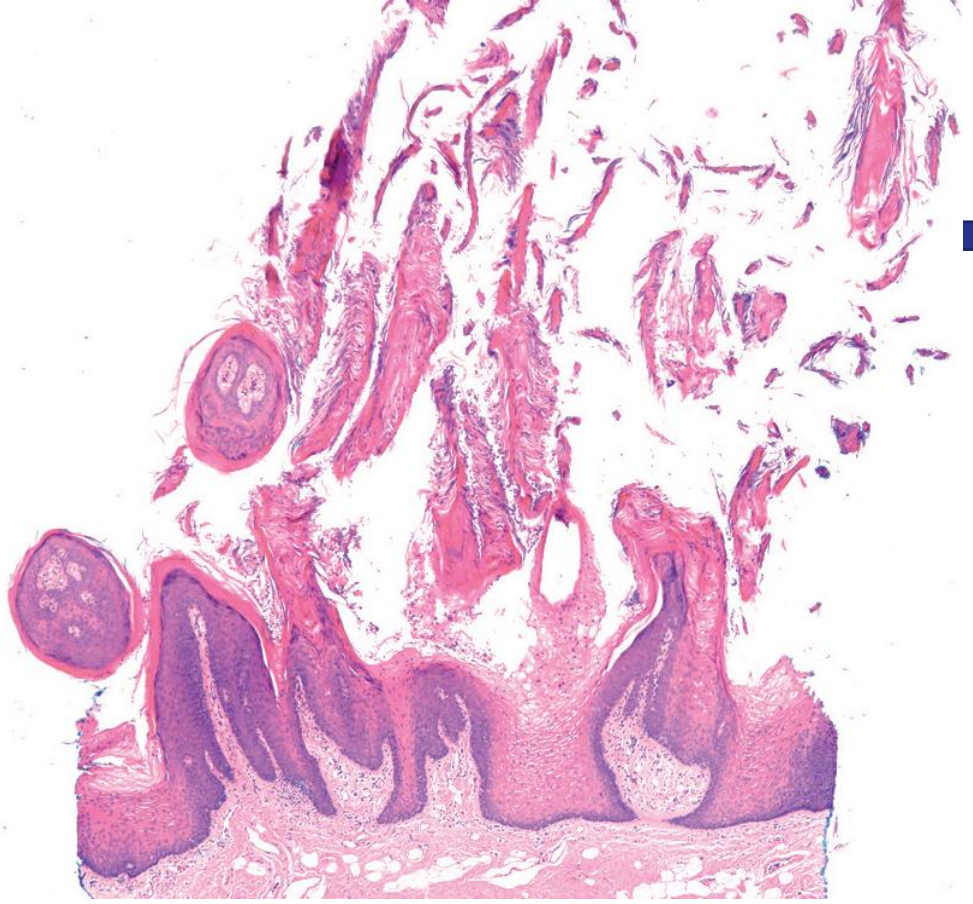
- It is very commonly seen in inpatients in the hospital.

- Antibiotics have been associated with black hairy tongue but they are not a direct effect of the antibiotic. The patient is ill and required antibiotic therapy and is likely dehydrated and not eating normally.









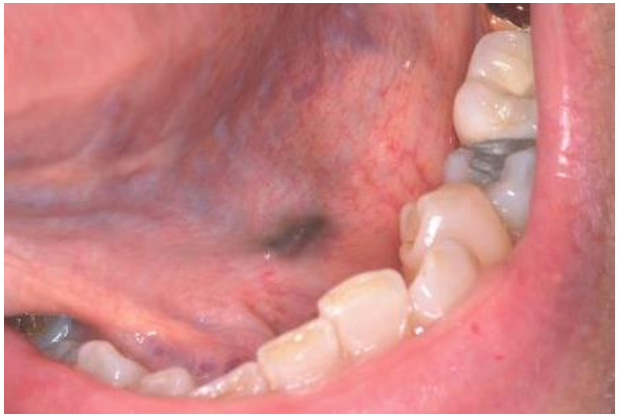
Amalgam tattoo

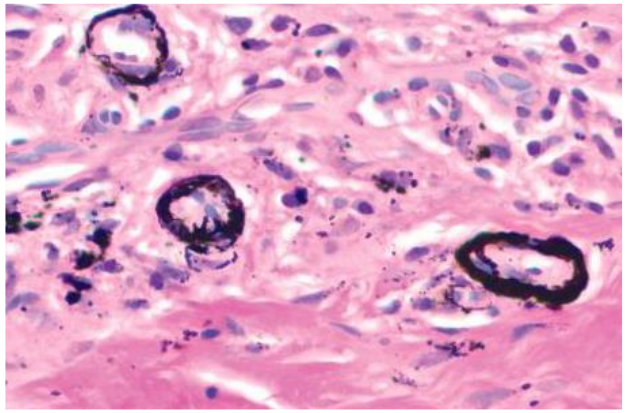
Amalgam tattoos result from accidental implantation of dental amalgam within the oral tissues.

Amalgam can be introduced into oral mucosa in several ways:

- During placement or removal of amalgam restorations or during crown preparation.
- Fracture of dental amalgams via trauma or tooth extraction may also leave residual amalgam within soft tissues.
- Endodontic treatment that involved placing amalgam at the apex of the tooth

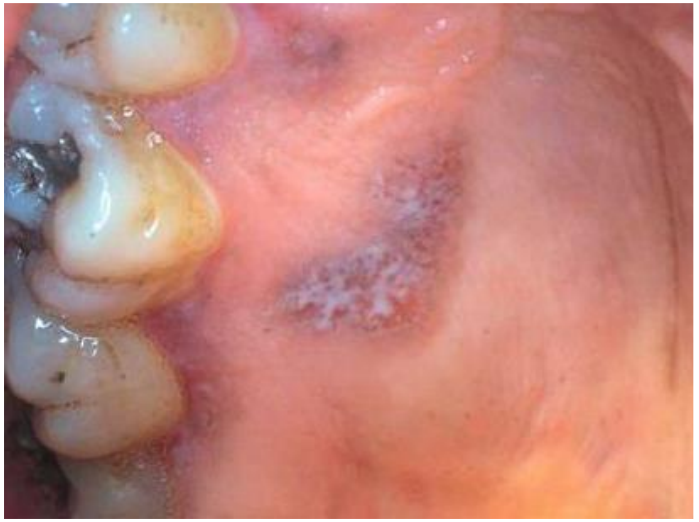
Diagnosis is confirmed by x-ray.

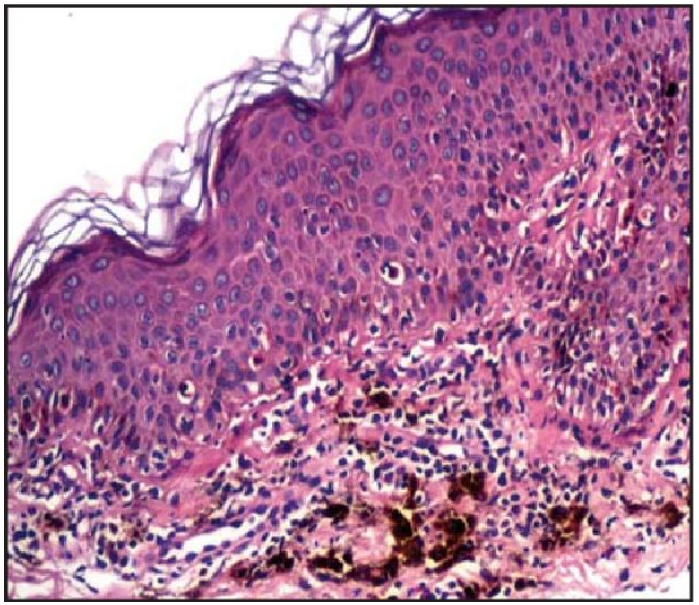




Post-inflammatory pigmentation

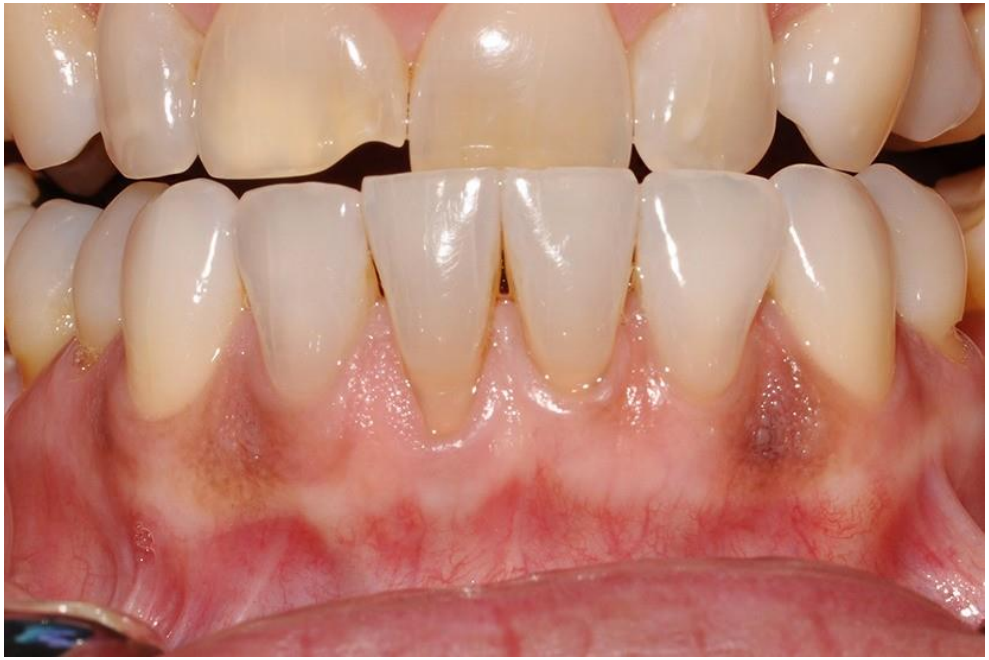
- Inflammation releases prostaglandins and other inflammatory products that stimulate melanocytes to increase synthesis of melanin.
- Melanin becomes trapped by macrophages, called *melanophages.*
- More common in individuals with darker skin.
- Lesions disappear shortly after the inflammatory process resolves.
- Hypermelanosis may present for many years
- These lesions cause concern because they may resemble early melanoma.





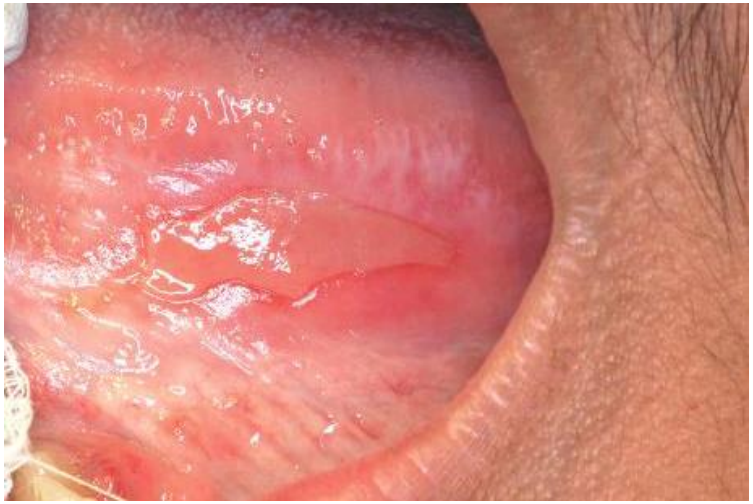
Smoker melanosis

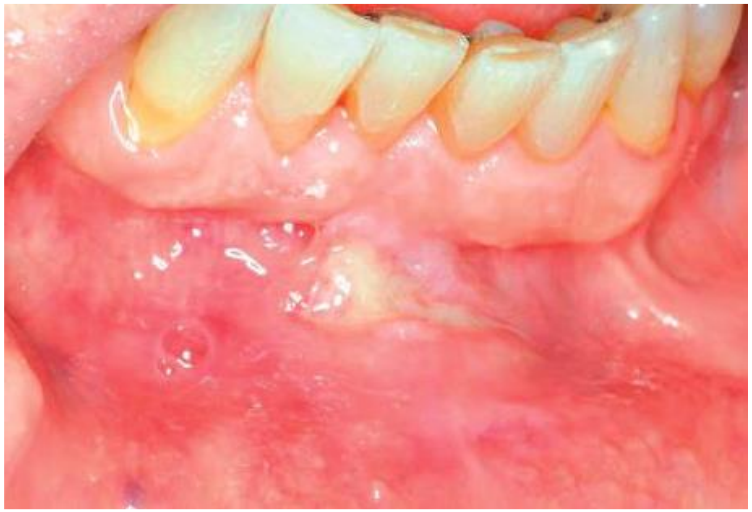
- *Smoker's melanosis* is a condition of excessive melanin pigmentation found in the gingiva of about 20% of smokers and seen more frequently in females.
- Melanin deposition is considered a protective mechanism.
- Melanosis is thought to be an attempt by the body to protect itself from toxins and heat produced by smoking tobacco.
- Any mucosal surface can be affected, but melanosis is most commonly seen in the facial anterior gingiva and rarely in the molar areas



Traumatic ulcer

- **Ulcer:** the loss of surface epithelium with exposure of the underlying connective tissues.
- Ulcers are one of the most common injuries to the human body.
- Traumatic ulcers are almost always of acute onset, immediately following the traumatic event.
- The lateral border of the tongue and labial mucosa are the most common locations.
- Lesions will appear as round to ovoid depressions with yellow necrotic centers and erythematous irregular borders.
- Patients complain of pain that slowly subsides as the lesion heals over 7 to 10 days.





Traumatic ulcer

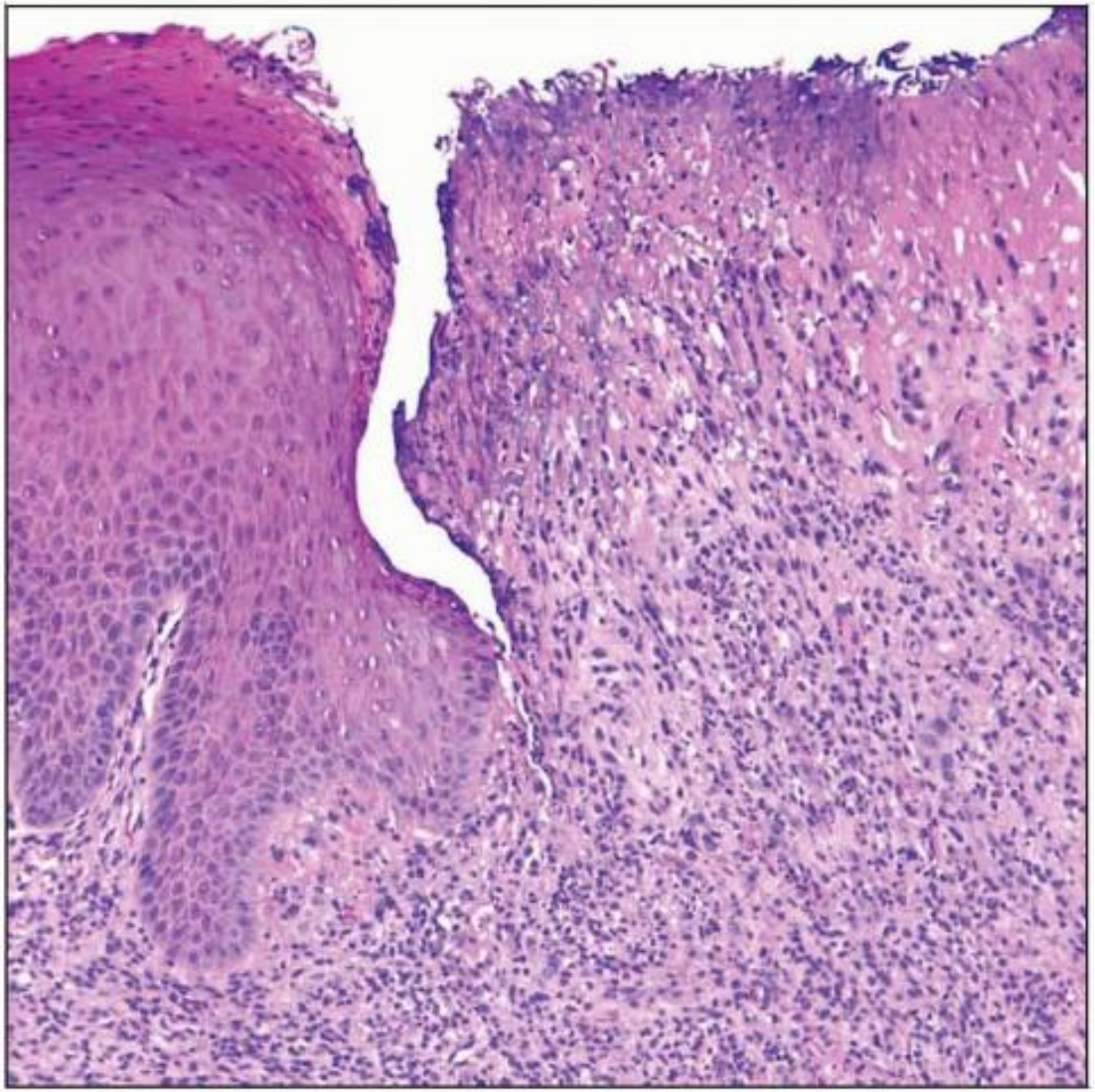


Table 2.8 Traumatic Ulcerations of the Oral Mucosa

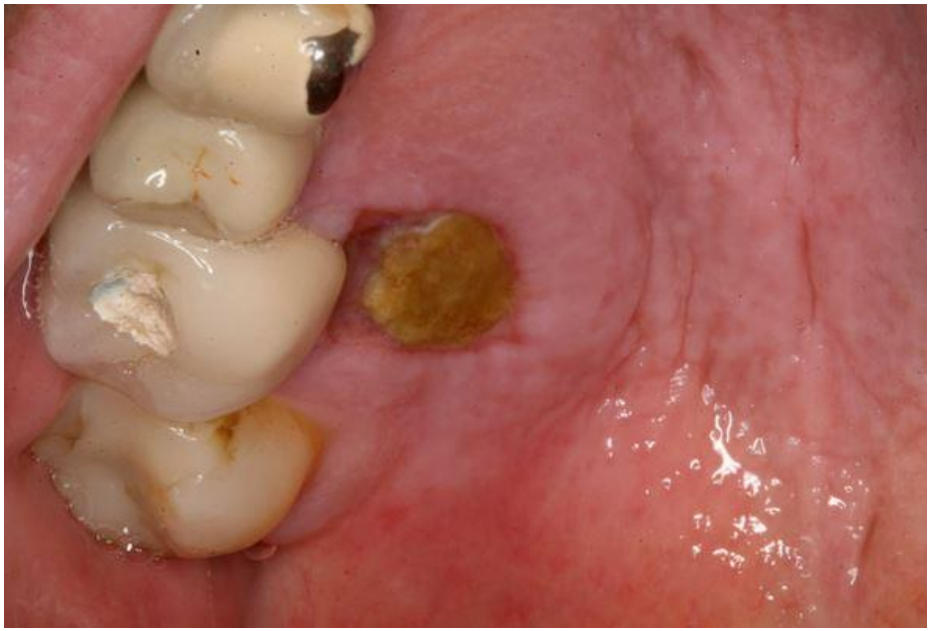
	Etiology	Clinical Features
Traumatic ulcer	Physical injury such as biting the tongue or contact with sharp object	Usually single, well-defined area of erythema surrounding yellow fibrinopurulent membrane; slightly raised border; tender/painful; resolves in 7 to 10 days with removal of etiology
Traumatic ulcerative granuloma	Traumatic ulcer that receives persistent mild chronic trauma	Long duration; raised rolled border; crater with yellow fibrinopurulent membrane; nonpainful or mildly tender; slow to resolve with removal of etiology; often requires surgical excision and healing by primary intention
Denture ulcer	New denture with ill-adapted flange; prolonged denture wearing	Ovoid erythematous area with yellow necrotic center; contacts irregular area of denture; resolves with denture adjustment
Anesthetic necrosis	Ischemia from epinephrine in local anesthetic or trauma during injection	Usually on hard palate at injection site; painful well-defined intense red area with central necrosis; heals without treatment in 10 to 14 days
Thermal burn	Contact with hot foods such as pizza	Painful yellow to white zone of necrosis of surface mucosa of palate or buccal mucosa; tissue sloughs; patient reports etiologic event; resolve with no treatment
Electrical burn	Contact with live electrical cord or extension cord	Yellow to black painless area that gradually becomes edematous; sloughs and bleeds; usually on lips of children
Chemical burn	Contact with caustic medications, dental materials, improper use of analgesics, mouth rinses	Superficial white corrugated or "cracked" appearance; epithelium sloughs leaving red painful surface; resolves without treatment in 10 to 14 days

Anesthetic necrosis

- Anesthetic necrosis is an unusual traumatic ulceration typically seen in patients who receive an injection in the greater palatine foramen for dental procedures on the maxillary teeth. It is believed that the epinephrine in the local anesthetic causes mild ischemia, leading to localized necrosis.

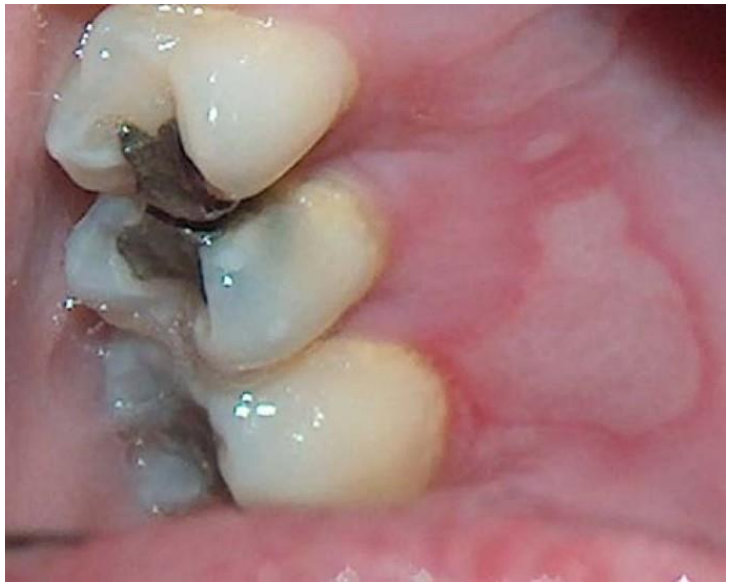
- Anesthetic necrosis appears as a well-defined tender-to-painful lesion at the injection site.

- The lesion heals without treatment in 7 to 10 days.



Burns

- ***Burns*** to the oral mucous membranes destroy surface epithelium and are usually very painful.



- Burns can be thermal or chemical and are of acute onset following exposure to the source.

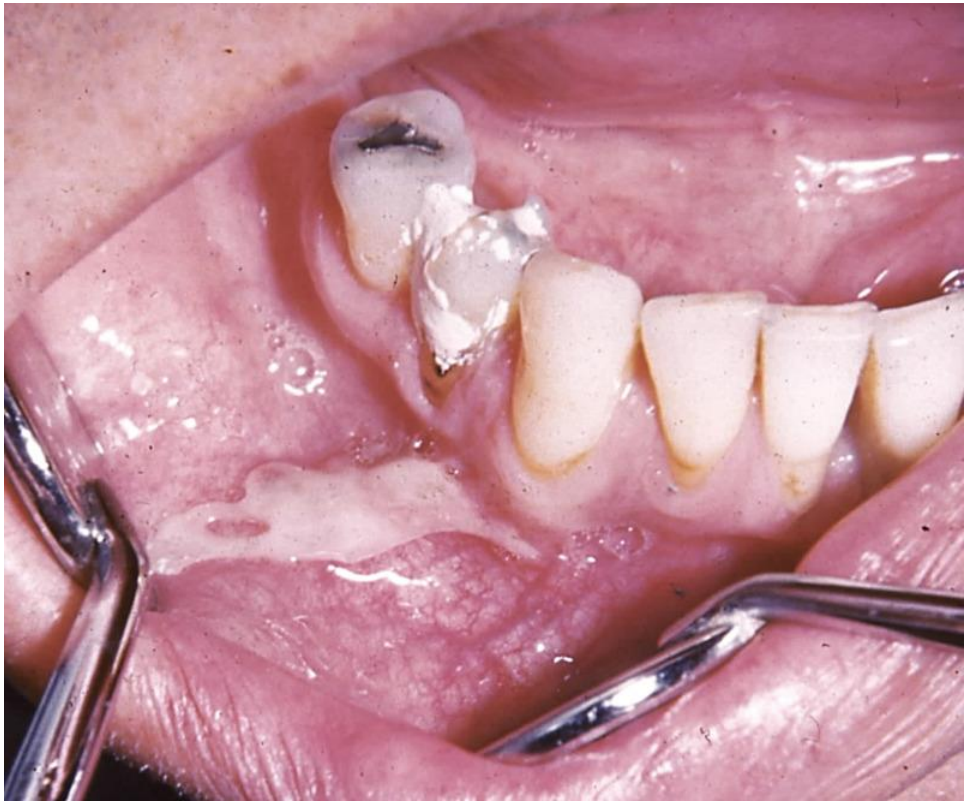
- ***Thermal burns*** can be induced by contact with hot foods or liquids. Hot foods that contact the lips or oral cavity mucosa may result in acute tissue destruction.

- Palatal burns from eating hot pizza are a common example. Occasionally, accidental contact with live electrical wires can cause deep burns to the face and/or oral cavity.

Electrical burns of the lips and commissures can be seen in young children who accidentally chew or bite into electrical cords. Electrical burns to the lips are often severe, involving extensive tissue damage and requiring special reconstruction by a plastic surgeon.

Burns

- *Chemical burns* can result when caustic agents contact the oral mucosa. Chemical burns may appear as thick, rough hyperkeratotic plaques with a corrugated or cratered surface and/or areas of ulceration.
- Patients may misuse acidic medications, such as aspirin tablets or powders that contain acetylsalicylic acid.
- Aspirin may cause a significant burn if placed adjacent to or over a painful tooth, rather than being swallowed.
- Patients may use strong agents, such as hypochlorite (bleach), to clean or disinfect



CLINICAL REPORT

Australian Dental Journal 2000;45:(4):257-269

THE UNIVERSITY OF WESTERN AUSTRALIA

Safety issues relating to the use of hydrogen peroxide in dentistry

Laurence J. Walsh*

Abstract

Hydrogen peroxide is used widely in professionally and self-administered products. Hydrogen peroxide is a highly reactive substance which can damage oral soft tissues and hard tissues when present in high concentrations and with exposures of prolonged duration. This report provides an overview of health issues relating to the use of hydrogen peroxide, with an emphasis on safety with prolonged exposure to low concentrations of peroxide products. There is good evidence for the safety of hydrogen peroxide when used at low concentrations on a daily basis over extended periods of time, in self-administered oral health care products such as dentifrices and mouthrinses. These low concentrations neither damage oral hard or soft tissues, nor do they pose a significant risk of adverse long-term effects. Caution should be exercised with the increasingly higher concentration peroxide products used for 'walking' or 'power' bleaching due to the possibility of chemical irritation of oral soft tissues with injudicious use. The volumes of material and application times should be controlled carefully. Thorough education of patients is particularly important with self-applied gels because of the lack of professional supervision with such products. Such education is part of the duty of care of the dentist who supplies bleaching gels for at-home use.

Key Words: Dentifrice, bleaching, peroxide, toxicity.

applied bleaching agents which either contain or generate hydrogen peroxide, by both the dental profession and the general public. The most common ingredient used is carbamide peroxide, which when present at a concentration of 10 per cent releases 3.5 per cent hydrogen peroxide.[1]

While hydrogen peroxide can be toxic in high concentrations and with exposures of prolonged duration, concentrated (30-35 per cent) hydrogen peroxide solutions have been used for in-office bleaching treatments with no serious adverse soft and hard tissue effects observed clinically, other than the relatively common but self-limiting post-treatment sensitivity.[2] Nevertheless, there have been occasions when accidental ingestion of hydrogen peroxide products in the home has led to hospitalization and adverse health outcomes, and this should be kept in mind when assessing the relative safety of hydrogen peroxide-containing products for home (unsupervised) use.[3]

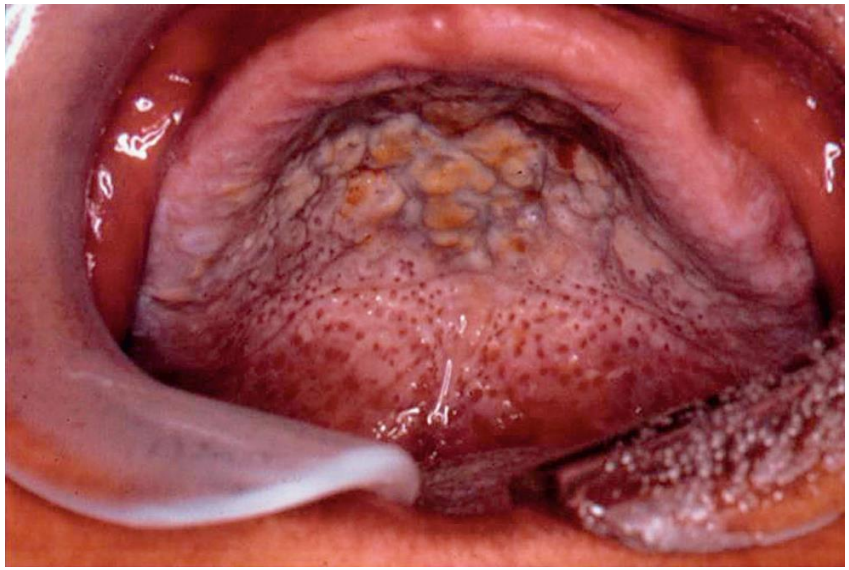
Much of the recent dental literature on the safety of hydrogen peroxide has focused on dentist-prescribed home bleaching, in which the teeth and oral soft tissues can be in contact with peroxide-type agents for extended periods of time. This provides a very different situation from either in-office bleaching

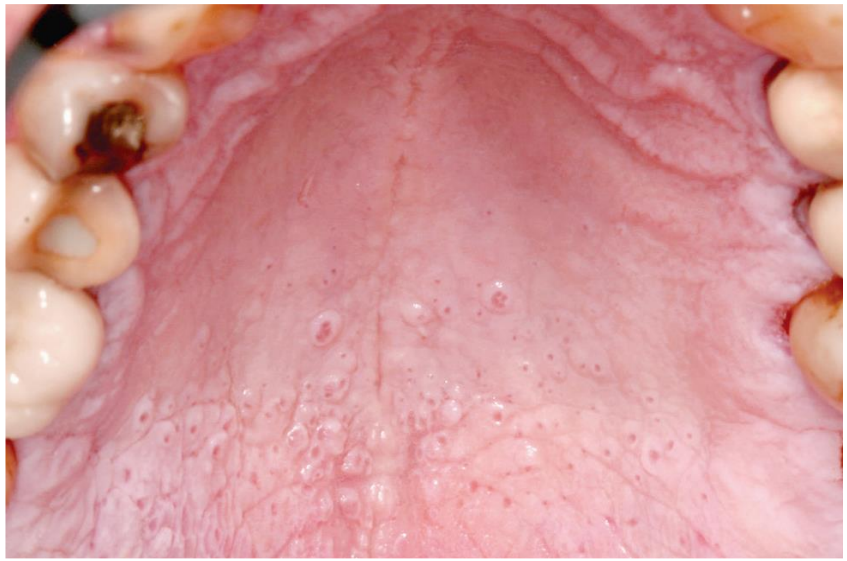
Nicotinic stomatitis

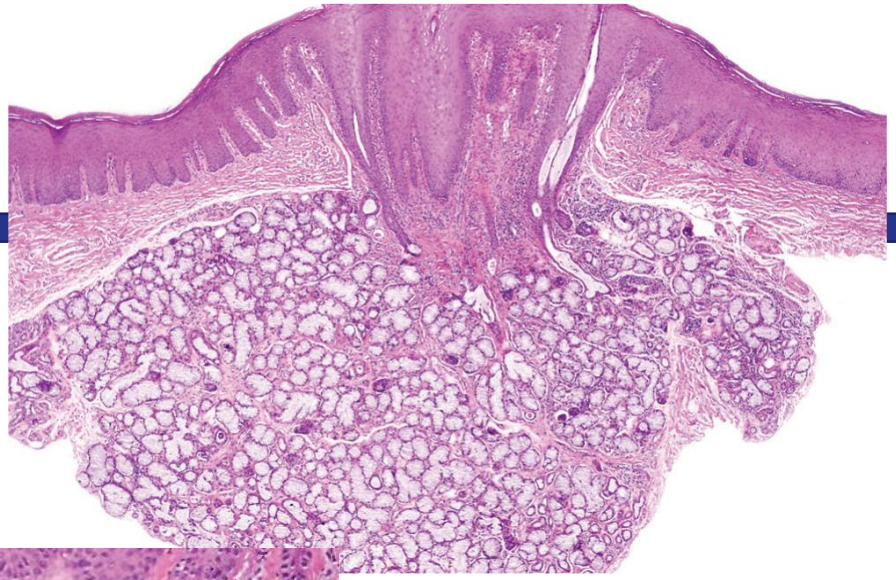
- *Nicotine stomatitis* occurs in smokers and results from exposure of the palate to the smoke and heat of burning tobacco products.

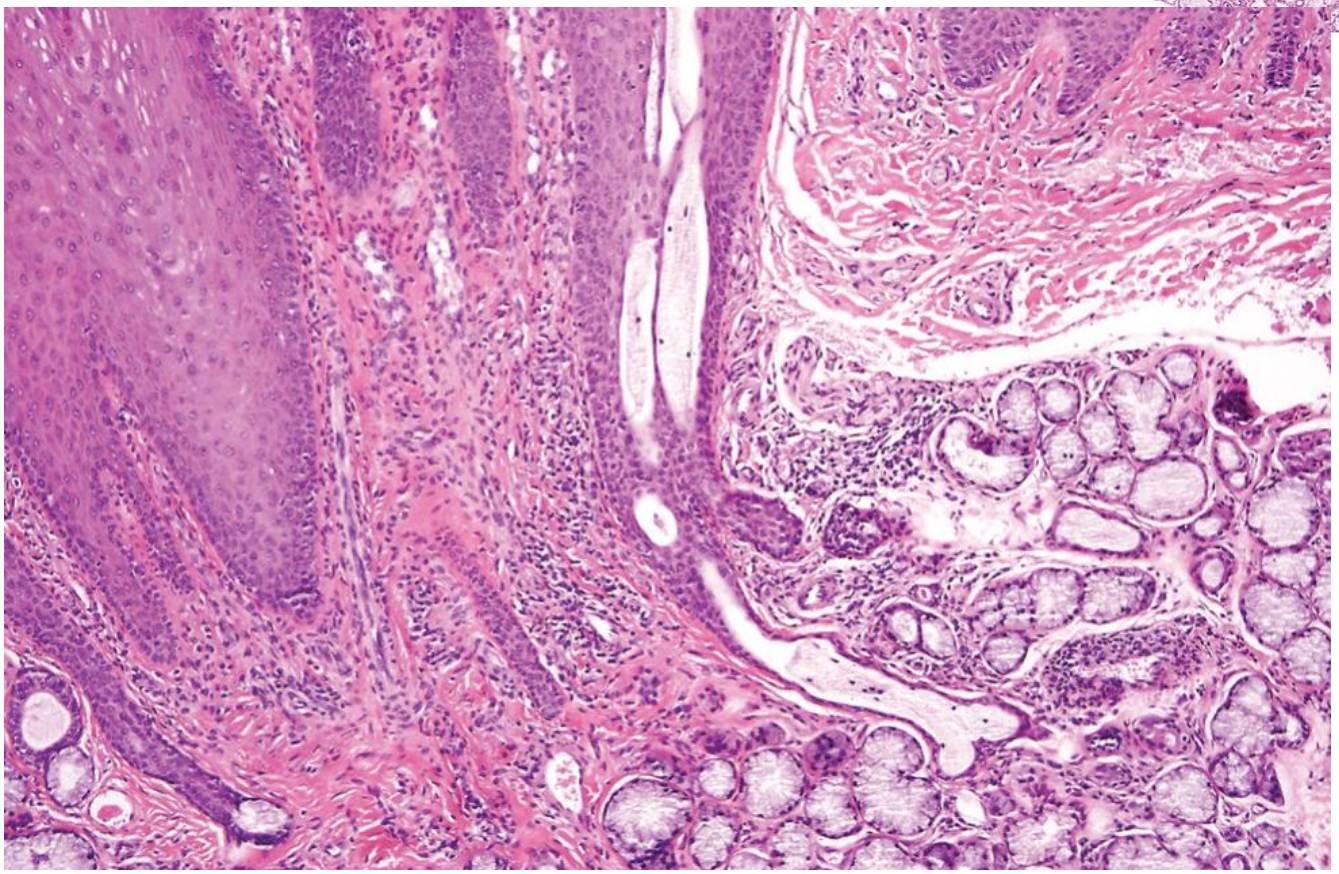
- It generally appears as a thick, white plaque of the hard and soft palate, containing scattered, tiny, raised red (erythematous) dots. The erythematous dots represent irritated minor salivary gland ducts.

- Nicotine stomatitis persists as long as the individual continues smoking. The lesion itself is benign and will regress upon smoking cessation.





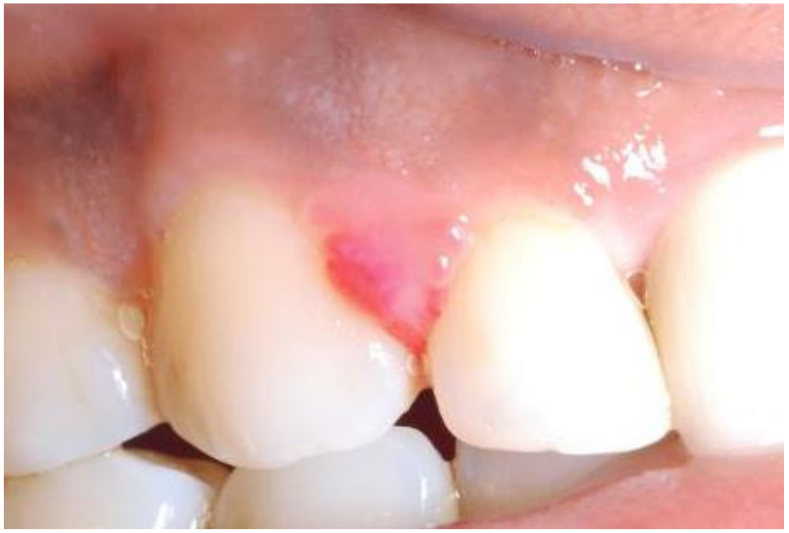


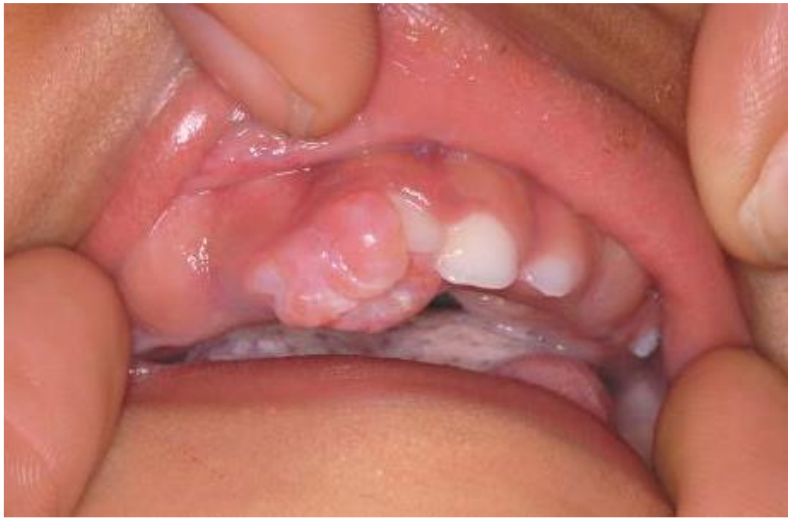


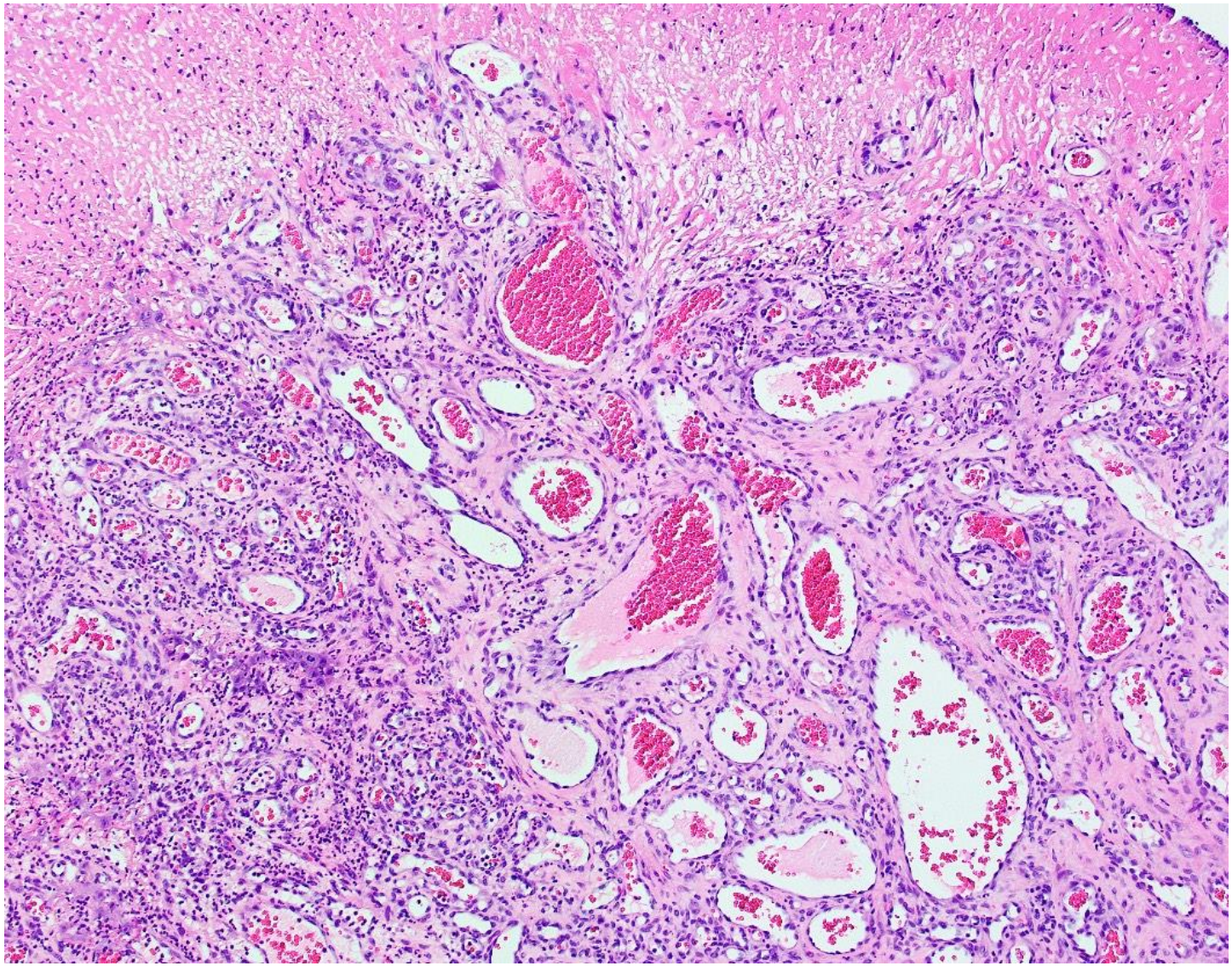
Pyogenic granuloma

- *Pyogenic granulomas* are exophytic lesions composed of an exuberant overgrowth of granulation tissue in response to minor chronic irritation.

- The name is inaccurate because it is not due to pyogenic (pus-producing) bacteria and is not granulomas.

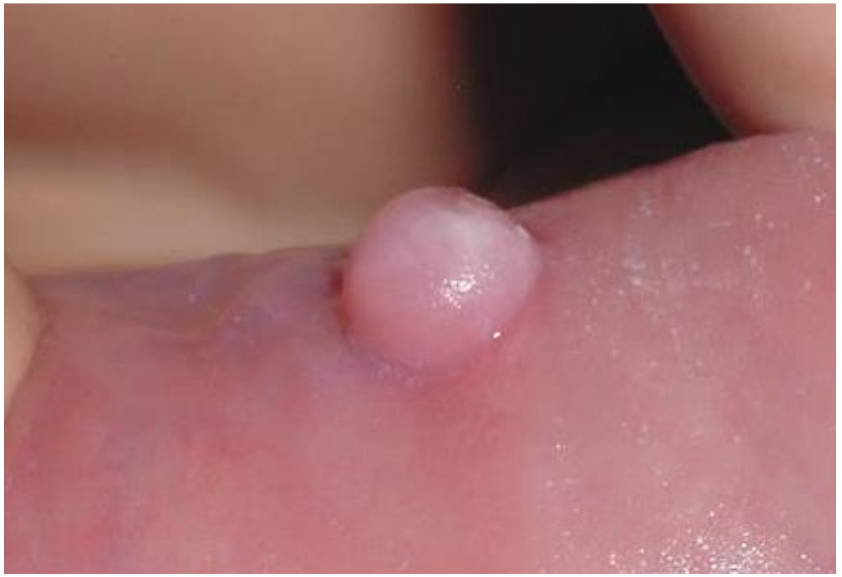


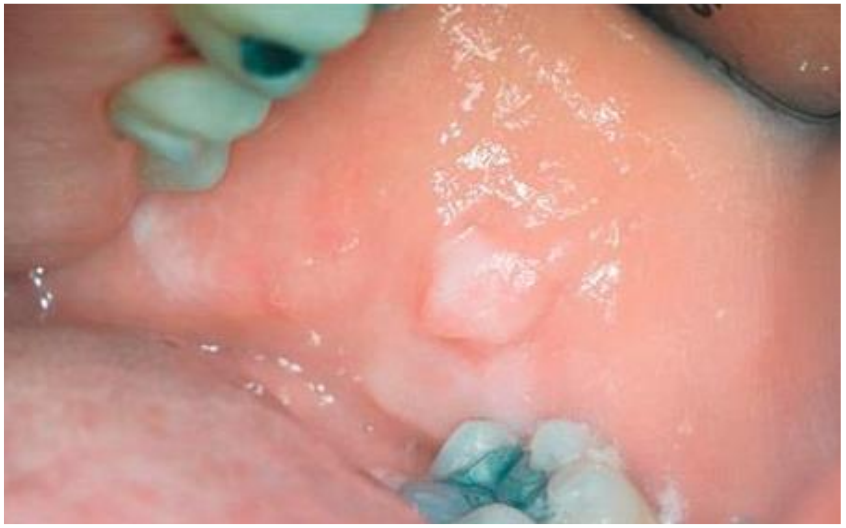




Traumatic fibroma

- Traumatic (irritation) fibromas are benign lesions with dense, highly fibrous connective tissue, like a scar.
- They are more appropriately called *focal fibrous hyperplasia* to denote a reactive rather than neoplastic origin.
- They occur anywhere that persistent chronic tissue irritation occurs, most often along the line of occlusion, where repetitive trauma from biting may occur.
- They may be sessile or pedunculated with a pink smooth surface



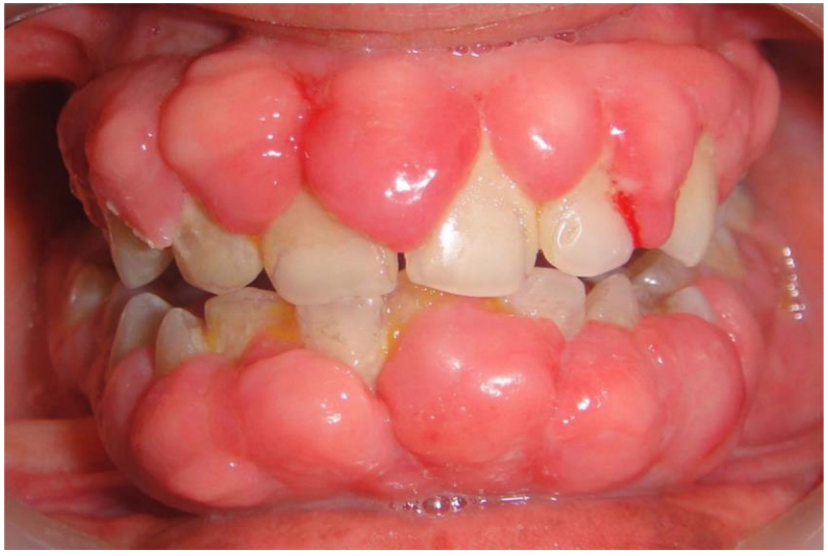


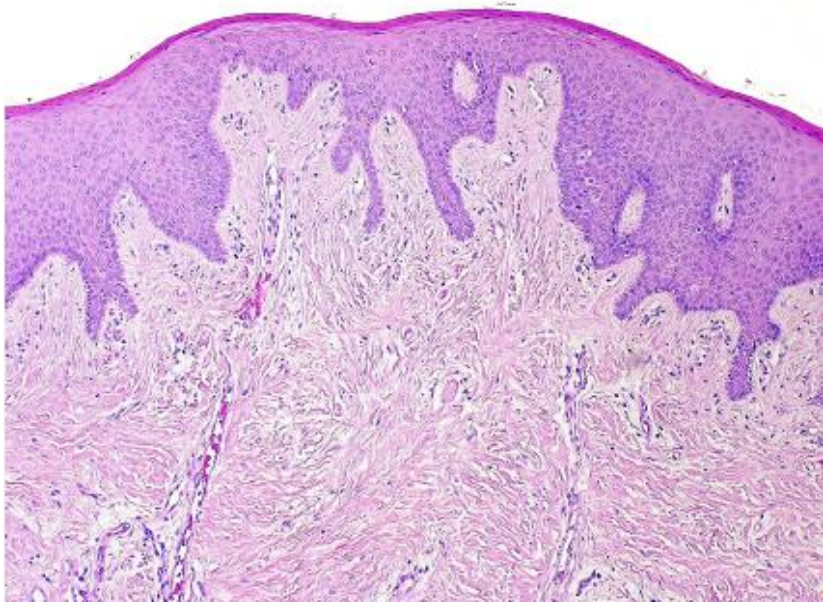
Drug-induced gingival hyperplasia

- *Drug-induced gingival overgrowth* is considered a reactive phenomenon seen in patients taking medications that stimulate collagen growth or prevent its breakdown.

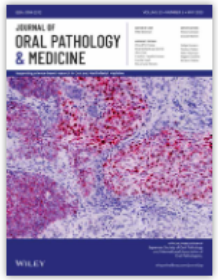
- It is well documented that phenytoin (Dilantin), cyclosporine, nifedipine, and other calcium channel blockers may lead to gingival overgrowth.

- Clinically, the lesions are similar and do not differ based on type of drug.





JOURNAL OF ORAL PATHOLOGY & MEDICINE



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Full Access

On the mechanism of drug-induced gingival hyperplasia

Ronald S. Brown, William T. Beaver, William K. Bottomley

First published: May 1991 | https://doi.org/10.1111/j.1600-0714.1991.tb00419.x | Citations: 83

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Abstract

Proposed mechanisms of the side effect of drug-induced gingival hyperplasia are reviewed. Hypotheses with regard to inflammation from bacterial plaque, increased sulfated glycosaminoglycans, immunoglobulins, gingival fibroblast phenotype population differences, epithelial growth factor, pharmacokinetics and tissue binding, collagenase activation, disruption of fibroblast cellular sodium/calcium flux, folic acid and a combination hypothesis are evaluated.

Recommended

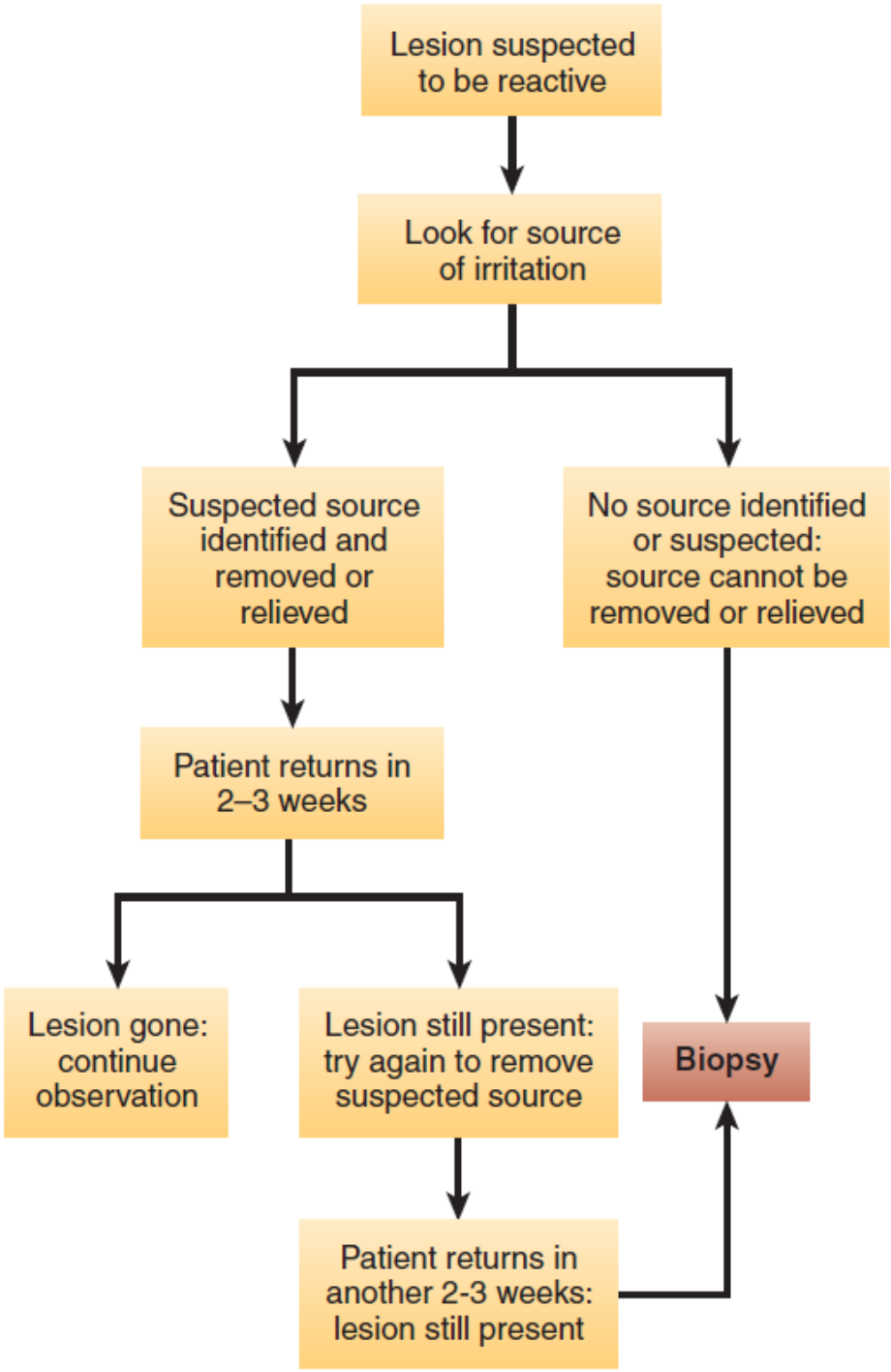
The effect of a plaque control program on the development of phenytoin-induced gingival overgrowth

Göran Dahllöt, Thomas Modéer

Journal of Clinical Periodontology

Prevalence and risk of gingival overgrowth in patients treated with diltiazem or verapamil

Jaume Miranda, Lluís Brunet, Pere Roset, Leonardo Berini, Magí Farré


Lesion suspected to be reactive
Look for source of irritation
Suspected source identified and removed or relieved
No source identified or suspected: source cannot be removed or relieved
Patient returns in 2–3 weeks
Lesion gone: continue observation
Lesion still present: try again to remove suspected source
Biopsy
Patient returns in another 2-3 weeks: lesion still present

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Quiz

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Questions