RED & WHITE LESIONS

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HEREDITARY WHITE LESIONS

- Leukoedema
- White Sponge Nevus
- Hereditary Benign Intraepithelial Dyskeratosis
- Dyskeratosis Congenita

Burkett’s classification 2008
REACTIVE/INFLAMMATORY WHITE LESIONS

- Linea Alba (White Line)
- Frictional (Traumatic) Keratosis
- Cheek Chewing
- Chemical Injuries of the Oral Mucosa
- Actinic Keratosis (Cheilitis)
- Smokeless Tobacco–Induced Keratosis
- Nicotine Stomatitis
- Sanguinaria-Induced Leukoplakia

Burkett’s classification 2008
INFECTIONOUS WHITE LESIONS AND WHITE AND RED LESIONS

- Oral Hairy Leukoplakia
- Candidiasis
- Mucous Patches
- Parulis

Burkett’s classification 2008
HEREDITARY WHITE LESIONS
LEUKOEDEMA

- Diffuse grayish-white milky appearance of the buccal mucosa.
- Appearance will disappear when cheek is everted and stretched.
TREATMENT

- No treatment is indicated for leukoedema since it is a variation of the normal condition.
- No malignant change has been reported.
White spongy nevus

- White sponge nevus (WSN) is a rare autosomal dominant disorder.
- With a high degree of penetrance and variable expressivity.
- It predominantly affects noncornified stratified squamous epithelium.
Clinical features of white spongy nevus

- Presents as bilateral symmetric white, soft, “spongy,” or velvety thick plaques of the buccal mucosa.
- Other sites in the oral cavity may be involved, including the ventral tongue, floor of the mouth, labial mucosa, soft palate, and alveolar mucosa.
TREATMENT

- No treatment is indicated for this benign and asymptomatic condition.
- If the condition is symptomatic, patients may require palliative treatment.
Hereditary Benign Intraepithelial Dyskeratosis

- Also called as Witkop-Von Sallmann Syndrome
- Autosomal dominant/common in children.
- White, spongy, macerated lesions seen on buccal mucosa. In some oral lesions, candidal infection may superimpose.
- Eye lesions include gelatinous plaques on the bulbar conjunctiva. There may be photophobia & blindness caused by involvement of cornea by plaque formation & scarring.
- Eye lesions appear or increase in severity in the springs & disappear.

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Management

- Ophthalmologist consultation for eye lesions.
- Antifungal medication in case if the lesion is superimposed with candidiasis
Dyskeratosis Congenita

- Well recognized rare genokeratosis, which is probably inherited as a recessive characteristic.
- Mutation in DKC 1 gene will cause disruption in maintenance of telomerase which is critical in determining the normal cellular longevity.
- Disease manifested has 3 typical signs: oral leukoplakia, dystrophy of nails, & pigmentation of skin.
- C/F: It is evident during first 10 yrs of life, almost exclusively seen in males.
- Nail dystrophy & shedding sometimes after the age of 5 yrs.

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Grayish brown Pigmentation usually seen on trunk, neck & thigh, skin may become atrophic, telangiectatic & face appears red.

- Thrombocytopenia develops during 2nd decade of life.
- Other features include: frail skeleton, mental retardation, dysphagia, deafness, hyperhydrosis of palms & soles.
- Oral involvement common sites are tongue & buccal mucosa. Vesicles & ulcerations followed by white patches of necrotic epithelium.
- Atrophy of tongue
• Recurrent ulcerations are common in the age group of 14-20yrs & frequently develop erythroplasia or Red mucosal lesion.
• Finally between the age of 20-30yrs, there is development of erosive leukoplakia & carcinoma.
• OPG may show severe periodontal bone loss.
• It’s a clinical diagnosis including Leukoplakic type lesions, nail changes, skin pigmentation & so periodic check up should be done for evidence of malignant transformation.

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Clinical characteristics of dyskeratosis congenita

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REACTIVE AND INFLAMMATORY WHITE LESIONS
Linea Alba (White Line)

- Is a horizontal streak on the buccal mucosa at the level of the occlusal plane.
- It is a very common finding most likely associated with pressure, frictional irritation, or sucking trauma from the facial surfaces of the teeth.

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Frictional (Traumatic) Keratosis

- Is defined as a white plaque with a rough and frayed surface that is clearly related to an identifiable source of mechanical irritation.
- Usually resolve on elimination of the irritant.
TREATMENT

- Upon removal of the offending agent, the lesion should resolve.
- Within 2 weeks. Biopsies should be performed on lesions that do not heal to rule out a dysplastic lesion.
Cheek biting

- Ragged, irregular white tissue of the buccal mucosa in the line of occlusion.
- May be ulcerated.
- Due to chewing or biting the cheeks.
- May also be seen on labial mucosa.
TREATMENT AND PROGNOSIS

- Since the lesions result from an unconscious and/or nervous habit, no treatment is indicated.
- For those desiring treatment and unable to stop the chewing habit, a plastic occlusal night guard may be fabricated.
Chemical Injuries of the Oral Mucosa

- Transient nonkeratotic white lesions of the oral mucosa.
- Are often a result of chemical injuries caused by a variety of caustic agents retained in the mouth for long periods of time.
- such as aspirin, silver nitrate, formocresol, sodium hypochlorite, paraformaldehyde, dental cavity varnishes, acid etching materials, and hydrogen peroxide.
Chemical Injuries of the Oral Mucosa

- The white lesions are attributable to the formation of a superficial pseudomembrane composed of a necrotic surface tissue and an inflammatory exudates.

Aspirin burn, creating a pseudomembranous necrotic white area.
Chemical Injuries of the Oral Mucosa

Extensive tissue necrosis caused by injudicious use of silver nitrate

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Chemical Injuries of the Oral Mucosa

Severe ulceration and sloughing of mucosa, caused by use of a cinnamon-containing dentifrice

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Actinic Keratosis (Cheilitis)

- Actinic (or solar) keratosis is a premalignant epithelial lesion directly related to long-term sun exposure.
- Classically found on the vermilion border of the lower lip as well as on other sun-exposed areas of the skin.
- A small percentage of these lesions will transform into squamous cell carcinoma.
ACTINIC CHEILITIS

Distinctive raised white plaque, representing actinic cheilitis.
Actinic keratosis

- Premalignant
- Due to long term sun exposure
- Vermillion border of lower lip
- Treatment: surgery
Smokless tobacco induced keratosis

- In the area of tobacco contact
- Precancerous
- May be wrinkled or folded
- May be accompanied by gingival recession & perio-destruction
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Nicotine Stomatitis

- Palate initially becomes diffusely erythematous and eventually turns grayish white secondary to hyperkeratosis
- Multiple keratotic papules with depressed red centers correspond to dilated and inflamed excretory duct openings of the minor salivary glands
Stomatitis nicotina
Nicotinic stomatitis

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Nicotina Stomatitis

**Histologic** appearance of nicotine stomatitis, showing hyperkeratosis and acanthosis with squamous metaplasia of the dilated salivary duct. (Hematoxylin and eosin, ×40 original magnification)
TREATMENT AND PROGNOSIS

- Nicotine stomatitis is completely reversible once the habit is discontinued.
- The lesions usually resolve within 2 weeks of cessation of smoking.
- Biopsy of nicotine stomatitis is rarely indicated except to reassure the patient.
- Biopsy should be performed on any white lesion of the palatal mucosa that persists after month of discontinuation of smoking habit.
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INFECTIONOUS WHITE LESIONS AND WHITE AND RED LESIONS

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Oral Hairy Leukoplakia

- Corrugated leukoplakic lesions occurring on lateral & ventral surface of tongue in persons with AIDS or ARC.
- However it is also seen in severely immunocompromised individuals (bone marrow, kidney & liver transplants).
- No known premalignant potential.
- Epstein-Barr virus (EBV) have been associated with hairy leukoplakia on the basis of electron microscopy.
- Management includes antivirals such as zidovudine, ganciclovir, however aciclovir is most effective drug (1-4gm/day).
- Topical application of vitamin A is believed to regress the lesions of hairy leukoplakia.

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Oral Candidiasis

- Occurs in persons with poorly controlled diabetes, pregnancy, hormone imbalance, those receiving broad spectrum antibiotics, long term steroid treatment, cancer therapy and other immunocompromised individuals
- Oral lesions may be erythematous, pseudomembranous, hyperplastic or angular cheilitis
Classification of Oral Candidiasis

Acute
- Pseudomembranous
- Atrophic (erythematous)
- Antibiotic stomatitis

Chronic
Atrophic
- Denture sore mouth
- Angular cheilitis
- Median rhomboid glossitis
- Hypertrophic/hyperplastic
- Candidal leukoplakia
- Papillary hyperplasia of the palate (see denture sore mouth)
- Median rhomboid glossitis (nodular)
- Multifocal
- Mucocutaneous
- Syndrome associated
- Familial +/- endocrine candidiasis syndrome
- Myositis (thymoma associated)
- Localized
- Generalized (diffuse)
- Immunocompromise (HIV) associated
Clinical features

- Diffuse, patchy, or globular white thickened plaques on the tongue, soft palate & buccal mucosa.
- Can be wiped off erythematous, atrophic, or ulcerated mucosa.
- Mild burning pain severe when coagulum scraped.
1-Pseudomembranous Candidiasis

- Acute superficial mucosal infection.
- Infants & immune compromised.
- Systemic corticosteroid therapy, chemotherapy, AIDS, or acute debilitating illness.
Oral Candidiasis/Acute

Pseudomembranous candidiasis on the palate.

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Oral Candidiasis / Acute

A patient with a history of chronic iron deficiency anemia developed red, raw, and painful areas of the mucosa, diagnosed as acute atrophic candidiasis.
More-extensive pseudomembranous lesions associated with an erythematous base in an adult with severe thrush.

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Oral Candidiasis / Chronic

Chronic mucocutaneous candidiasis: multiple lesions on the tongue

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Chronic candidiasis

Atrophic

- Denture sore mouth.
- Angular cheilitis.
- Median rhomboid glossitis.
Denture sore mouth

- Denture stomatitis is a common form of oral candidiasis.
- Manifests as a diffuse inflammation of the maxillary denture-bearing areas.
- Often associated with angular cheilitis.
progressive stages of denture sore mouth.
• Allergic reaction due to denture base:- it is a very rare condition. If there is failure to response anti-fungal therapy then one should suspect allergic reaction.

• Management include Troches contating Clotrimazole and Nystatin 4 to 5 times applied on the denture after meal and bed time.
Angular cheilitis

- Angular cheilitis is the term used for an infection involving the lip commissures.
- The majority of cases are \textit{Candida} associated and respond promptly to antifungal therapy.
- There is frequently a coexistent denture stomatitis.
Angular cheilitis

Other possible etiologic cofactors include:

- reduced vertical dimension.
- nutritional deficiency (iron deficiency anemia and vitamin B or folic acid deficiency) sometimes referred to as perlèche.
- diabetes, neutropenia, and AIDS.
- co-infection with Staphylococcus and beta-hemolytic streptococcus.
Angular cheilitis
Median Rhomboid glossitis

- Erythematous patches of atrophic papillae located in the central area of the dorsum of the tongue.
- Considered a form of chronic atrophic candidiasis.
- These lesions were originally thought to be developmental in nature but are now considered to be a manifestation of chronic candidiasis.
Median Rhomboid glossitis
Chronic hyperplastic candidiasis

- Candidal leukoplakia.
- Papillary hyperplasia of the palate (denture sore mouth).
- Median rhomboid glossitis (nodular).
Hyperplastic candidiasis

• Superficial infection of the oral mucosa by the fungus Candida albicans and less common species of the same genus.
Hyperplastic candidiasis

Predisposing factors:
- poor oral hygiene
- xerostomia,
- recent antibiotic treatment
- dental appliance
- Compromised Immune system.
- early infancy
- AIDS
- Corticosteroid
- anemia,
- diabetes mellitus
Chronic mucocutaneous candidiasis

- Syndrome associated
  - Familial +/- endocrine candidiasis syndrome
  - Myositis (thymoma associated)
- Localized.
- Generalized (diffuse).
Chronic mucocutaneous candidiasis
Lab. Investigation

Confirmation:
- Gm stained smear shows candidal hyphae
- Biopsy: hyperplastic epithelium, inflammatory edema & cells
- Staining with PAS shows candidal hyphae
Candidiasis- Treatment:

Mild to Moderate- Topical Therapies
Nystatin (suspension 100KU/mL, or 1% cream), Clotrimazole (troche, 10mg).

Moderate to Sever- Systemic Therapies
Fluconazole (100mg/day), Itraconzole (oral suspension 10mg/mL).
Candidiasis Treatment:

- Topical therapy with nystatin or clotrimazole is effective. Treatment length is usually 10-14 days, follow up.

  Clotrimazole 10mg, 1 tab 5x/day, dissolve slowly and swallow, 10 day treatment

- Systemic treatment with fluconazole 100 mg/day for 10 days for oropharyngeal/esophageal disease, follow up.
Other Anti-fungal Agents

- Amphotericin B (5-10 ml oral solu. is used as rinse and then expectorated 3-4 times daily).
- Elixir containing both Tetracycline and Amphotericin B may also prove to be beneficial in acute atrophic candidiasis.
- Mycostatin cream 1 lac unit or Lactose containing vaginal tablet kept under the tongue.
- Mycostatin can be used as rinse for 7-10 days, 3-4 times a day.

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Systemic Treatment

- **Nystatin:** 250 mg TID for 2 weeks followed by 1 Troche per day for third week.

- **Ketoconazole:** 200 mg once daily but liver monitoring is necessary for long term use because of side effect like hepatotoxicity.

- **Fluconazole:** 100 mg once daily for 2 weeks.

- **Itraconazole:** 100 mg or 200 mg cap or oral suspension is also available.

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- Systemic candidaasis requires systemic administration of high dose of Amphotericin-B 10 mg QID, miconazole and 5-flucytosine.
- Mycostatin 50,000 units 8 hourly.
- Daily use of 200 mg ketoconazole for 2 weeks.
- Side-effects are increased liver enzymes and abdominal pain.
Mucous Patches

Secondary Syphilis
Gum Boil / Parulis

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Thank You

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