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## A Brief Review On White Lesions Of Oral Cavity

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### ABSTRACT:

White lesions in the oral cavity encompass a range of conditions from benign to potentially malignant. Common causes include leukoplakia, lichen planus, oral candidiasis, frictional keratosis, oral submucous fibrosis, and hairy leukoplakia. These lesions may result from trauma, infections, autoimmune disorders, or neoplastic changes. Accurate diagnosis through clinical examination, patient history, and biopsy is essential, particularly for lesions with malignant potential. This review focuses on the clinical presentation, differential diagnosis, and management strategies for white oral lesions, emphasizing the importance of early detection and appropriate intervention to prevent progression and improve patient outcomes.

KEY WORDS : White lesions , Oral mucosa , Precancerous conditions

## INTRODUCTION :

The characteristic white appearance of oral mucosa lesions is due to light scattering on an altered mucosal surface. This alteration can result from hyperkeratosis, acanthosis, intracellular edema of epithelial cells, and reduced blood supply to underlying connective tissue. Furthermore, white or yellow-white lesions can also be caused by fibrin covering an ulcer, surface debris, or fungal infections. While clinical experience can help identify the cause of a lesion's white appearance, a definitive diagnosis ultimately relies on microscopic examination.<sup>[1]</sup>

Various reports stress the link between oral and overall health, noting that oral health extends beyond teeth. Physicians often encounter oral health issues, and diagnosing common oral lesions requires a thorough history, complete oral exam, and knowledge of clinical characteristics. Research has identified the most common oral lesions, including candidiasis, herpes, aphthous stomatitis, and others.<sup>[2]</sup>

## CLASSIFICATION OF WHITE LESIONS :<sup>[1]</sup>

The white lesions range from those that are genetically determined to those that are neoplastic.

### 1. Hereditary/Developmental :

- Leukoedema
- White spongy nevus
- Hereditary benign intraepithelial dyskeratosis
- Pachyonychia congenita
- Dyskeratosis congenital

**2. Reactive :**

- Frictional keratosis
- Morsicatio buccarum
- Nicotine stomatitis
- Tobacco pouch keratosis
- Chemical burn

**3. Immunologic :**

- Lichen planus
- Lichenoid mucositis
- Discoid lupus erythematosus
- Graft-versus- host disease

**4. Bacterial/Viral/Fungal :**

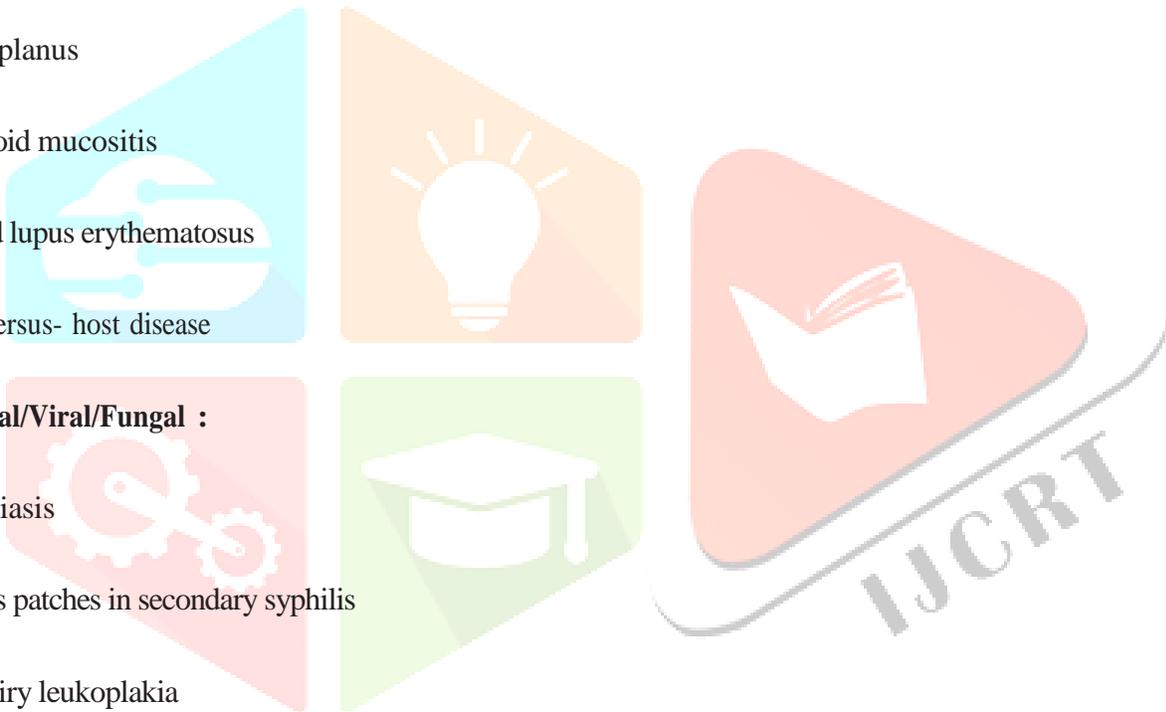
- Candidiasis
- Mucous patches in secondary syphilis
- Oral hairy leukoplakia

**5. Systemic disease :**

- Uremic stomatitis

**6. Potentially malignant disorders :**

- Leukoplakia
- Actinic cheilitis



## 7. Neoplastic :

- Squamous cell carcinoma

Some of the above mentioned white lesions are discussed below .

### **LEUKOEDEMA :**

Leukoedema is a benign, common variation of the oral mucosa, affecting up to 90% of blacks and 10-50% of whites, with no gender predilection. Its prevalence is higher among blacks due to increased mucosal pigmentation. Leukoedema is more noticeable in smokers but decreases after smoking cessation. Clinically, it appears as a diffuse, gray-white, non-scrapable, and veil-like condition, characterized by a milky, opalescent transformation of the oral mucosa. It often involves the buccal mucosa, lateral tongue borders, and occasionally the labial mucosa.

Leukoedema is asymptomatic, harmless, and has no potential for malignant transformation. No treatment is necessary, and it typically disappears temporarily with gentle mucosal stretching.<sup>[3]</sup>

### **WHITE SPONGY NEVOUS :**

White spongy nevus is a rare genetic disorder caused by mutations in the keratin 4 or 13 genes, inherited in an autosomal dominant pattern. The oral lesions may be widespread, often involved in the cheeks, palate, gingiva, floor of the mouth and portions of tongue. The mucosa appears thickened and folded or corrugated with a soft or spongy texture and a peculiar white opalescent hue. Ragged white area may also be present which can be removed sometimes by gentle rubbing without any ensuing bleeding. Histologically, the lesions show hyperkeratosis, acanthosis, and distinctive cellular changes. Electron microscopy reveals tangled keratin tonofilaments. This condition is asymptomatic and requires no treatment.<sup>[3]</sup>

### **FRICTIONAL KERATOSIS :**

Frictional keratosis refers to a white lesion with a rough surface that is related to an identifiable source of mechanical irritation and that are reversible on elimination of the irritant. This lesion is frequently associated with rough dentures or sharp cusps. Histologically such lesions exhibit varying degrees

of hyperkeratosis and acanthosis. 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.<sup>[4]</sup>

### **NICOTINE STOMATITIS :**

Nicotine stomatitis refers to a white lesion that develops on the palate in heavy cigarette, pipe and cigar smokers in response to heat rather than the chemicals in tobacco smoke. The palatal mucosa becomes diffusely gray or white. Numerous elevated white papules with punctuate red centers that represent inflamed ductal orifices are noted. Nicotine stomatitis is characterized histologically by hyperkeratosis and acanthosis of palatal epithelium and chronic inflammation of subepithelial connective tissue. Chronic inflammation of minor salivary glands and squamous metaplasia of excretory ducts are also noted. The condition is reversible, usually within 1 to 2 weeks of smoking cessation<sup>[4,5]</sup>

### **TOBACCO POUCH KERATOSIS :**

Tobacco pouch keratosis is a white mucosal lesion that develops in areas where tobacco is habitually chewed. Commonly affected areas include the lower jaw and cheek. The lesion appears as a thin, grayish-white, translucent plaque that's soft to the touch. Biopsy reveals characteristic changes, including parakeratin "chevrons" and acanthosis. In nearly all cases (98%), the lesion resolves and normal mucosal appearance returns within 2 weeks of quitting the habit.<sup>[5,6]</sup>

### **CHEMICAL INJURIES :**

Chemical burns from caustic substances like aspirin, hydrogen peroxide, or sodium hypochlorite can cause transient, non-keratotic white lesions on the oral mucosa. These lesions result from a superficial pseudomembrane forming over the affected area. They typically appear on the mucobuccal fold and gingiva, exhibiting a white, wrinkled appearance. Prolonged exposure can lead to necrosis and sloughing.

Microscopic examination reveals coagulative necrosis and inflammation. Superficial burns usually heal within 1-2 weeks, while deeper burns require debridement and antibiotic treatment. Prevention is key; using a rubber dam during endodontic procedures can minimize the risk of chemical burns.<sup>[5,6]</sup>

## ORAL LICHEN PLANUS :

Oral lichen planus affects up to 2% of individuals, impacting mucosa throughout the oral cavity. Those with hepatitis C have a sixfold higher incidence, and oral lichen planus may be the initial symptom. Half of individuals with cutaneous lichen planus also have oral lesions.

There are three main types: reticular (most common), erythematous, and erosive. The exact cause is unknown, but a T-cell immune response is suspected. Women are twice as likely to be affected as men, typically between 30-60 years old.

Symptoms vary: the classic reticular pattern has symmetric, white, lacy patterns on the buccal mucosa, often without symptoms. Erythematous and erosive varieties can cause pain and burning.

Oral lichen planus is chronic, with relapses and remissions. Localized oral lichenoid reactions may occur due to hypersensitivity to dental materials or allergens.

Diagnosis is clinical, with biopsy and dermal patch testing as needed. Asymptomatic cases require no treatment. Topical corticosteroids are the primary treatment, with systemic prednisone for severe cases

## ORAL CANDIDIASIS :

Between 30% to 60% of healthy individuals have Candida present in their mouths, usually *Candida albicans*. However, this fungus can cause opportunistic infections in people taking steroids, those with weakened immune systems, diabetes, malnutrition, or using antibiotics or dentures.

Oral candidiasis can manifest in different ways, including

- White plaques that can be wiped away, revealing red mucosa (pseudomembranous candidiasis)
- Generalized redness of the mucosa (atrophic candidiasis)
- Thick, non-wipeable plaques on the tongue and oral commissure (hyperplastic candidiasis)
- Redness and loss of papillae on the tongue (median rhomboid glossitis)

Symptoms may include oral burning, metallic taste, or no symptoms at all. Denture stomatitis, a type of atrophic candidiasis, can occur in people who wear dental prosthetics.

To manage oral candidiasis, it's essential to remove and clean oral appliances regularly. This can be done by soaking them in a solution of sodium hypochlorite or chlorhexidine. Avoid using toothpaste on dentures, as it can cause surface scratching.

If hyperplastic candidiasis is suspected, it's crucial to differentiate it from leukoplakia. If antifungal treatment doesn't resolve the lesions, a biopsy is necessary. Additionally, Candida and Staphylococcus aureus infections can cause angular cheilitis, a painful condition characterized by fissuring at the corners of the mouth.<sup>[7]</sup>

### **ORAL HAIRY LEUKOPLAKIA:**

Oral Hairy Leukoplakia (OHL) is a white lesion commonly seen in individuals with severe immunodeficiency, caused by the Epstein-Barr virus (EBV). Clinically, OHL appears as white streaks or thickened, furrowed areas on the tongue's lateral borders.

Histologically, OHL is characterized by a corrugated parakeratin layer, acanthosis, and distinctive "balloon cells" in the upper spinous layer. The epithelial cells exhibit nuclear changes, including clearing and chromatin margination, due to EBV replication.

A definitive diagnosis of OHL is made by detecting EBV within the lesion using techniques such as in situ hybridization, PCR, Southern blot, or electron microscopy.<sup>[7,8]</sup>

### **LEUKOPLAKIA :**

Leukoplakia is a clinical term describing a white patch or plaque on the oral mucosa that cannot be classified as any other disease. The World Health Organization (WHO) defines leukoplakia as a potentially precancerous lesion with an uncertain risk of malignancy.

Tobacco use is a significant risk factor for developing leukoplakia. Based on appearance, leukoplakia is classified into two types:

1. Homogeneous: slightly elevated, grayish-white, non-scrapable plaques with a fissured or wrinkled surface.
2. Non-homogeneous: includes speckled, nodular, verrucous, and proliferative verrucous leukoplakia (PVL) varieties.

A provisional diagnosis of leukoplakia is made when a predominantly white lesion cannot be diagnosed as another oral mucosa disease. A biopsy is essential for confirmation.

Microscopically, leukoplakia exhibits hyperkeratosis, with possible atrophy or acanthosis. Epithelial dysplasia is present in only 5-25% of cases.

Leukoplakia is a potentially malignant disorder with a malignancy transformation rate ranging from 4-47%. The presence of epithelial dysplasia is the strongest predictor of malignant transformation.

Management of leukoplakia depends on the presence and severity of dysplasia:

- No dysplasia or mild dysplasia: habit cessation and 6-month clinical evaluations.
- Moderate or severe dysplasia: surgical excision, cryosurgery, or laser ablation, followed by long-term monitoring. <sup>[8]</sup>

### **ACTINIC CHEILITIS :**

Actinic cheilitis is a potentially precancerous condition that develops on the lower lip's vermilion border, primarily due to prolonged sun exposure. It manifests as a small, white, oval or linear plaque, typically less than 1 cm in size. Histopathological examination reveals atrophic epithelium with varying degrees of dysplasia and altered collagen in the lamina propria. Surgical intervention is the preferred treatment for actinic cheilitis. <sup>[9]</sup>

## CONCLUSION :

White lesions in the oral cavity encompass a broad spectrum of conditions, from benign genetic disorders such as white spongy nevus to malignant neoplasms like oral squamous cell carcinoma (OSCC). Accurate histological diagnosis is crucial, as treatment options and prognoses vary significantly depending on the specific condition.

## REFERENCES

1. Jayanthi, Pa,\*; Ranganathan, Ka. Differential Diagnosis of White Lesions of Oral Mucosa. *Journal of Orofacial Sciences* 2(2):p 58-63, May–Aug 2010.
2. Gonsalves WC, Chi AC, Neville BW. Common oral lesions: Part I. Superficial mucosal lesions. *Am Fam Physician*. 2007 Feb 15;75(4):501-7. PMID: 17323710.
3. Mortazavi H, Safi Y, Baharvand M, Jafari S, Anbari F, Rahmani S. Oral White Lesions: An Updated Clinical Diagnostic Decision Tree. *Dentistry Journal*. 2019; 7(1):15. <https://doi.org/10.3390/dj7010015>
4. Regezi JA, Sciubba JJ, Jordan RCK. *Oral Pathology: Clinical Pathologic Correlations*. 4<sup>th</sup>ed. Saunders publishers; 2003:75- 90.
5. Neville BW, Damm DD, Allen CM, Bouquot JE. *Oral and Maxillofacial Pathology*. 2<sup>nd</sup> ed. Saunders publishers; 2003:253- 59.
6. Greenberg MS, Glick M. *Oral Medicine: Diagnosis and Treatment*. 10<sup>th</sup> ed. Elsevier publishers; 2003:85-120.
7. Randall DA, Wilson Westmark NL, Neville BW. Common Oral Lesions. *Am Fam Physician*. 2022 Apr 1;105(4):369-376. PMID: 35426641.
8. Warnakulasuriya S, Johnson NW, Van der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. *J Oral Pathol Med* 2007;36:575-80.
9. Batsakis JG, Suarez P, El-Naggar AK. Proliferative verrucous leukoplakia and its related lesions. *Oral Oncol* 1999;35:354- 59.

