RED AND WHITE LESIONS

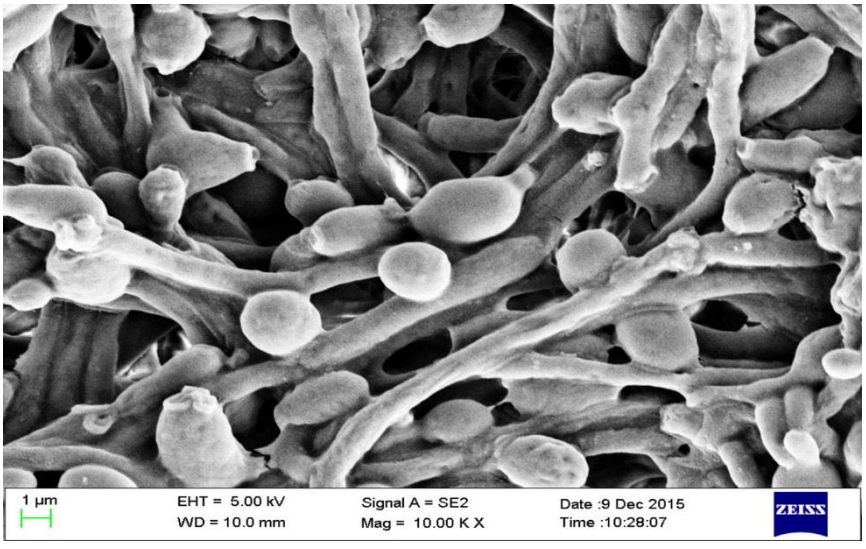
A white appearance of the oral mucosa may be caused by a variety of factors:

1- The oral epithelium may be stimulated to an increased production of keratin (hyperkeratosis).
2- an abnormal but benign thickening of stratum spinosum (acanthosis).
3- Intra- and extracellular accumulation of fluid in the epithelium may also result in clinical whitening.
4- Microbes, particularly fungi, can produce whitish pseudomembranes consisting of sloughed epithelial cells, fungal mycelium, and neutrophils, which are loosely attached to the oral mucosa.

While red lesions of the oral mucosa may develop as the result of atrophic epithelium, characterized by a reduction in the number of epithelial cells or increased vascularization that is dilatation of vessels and/ or proliferation of vessels.

INFECTIOUS DISEASES

ORAL CANDIDIASIS



1- Oral candidiasis is the most prevalent opportunistic infection affecting the oral mucosa.

2- In the vast majority of cases, the lesions are caused by Candida albicans.

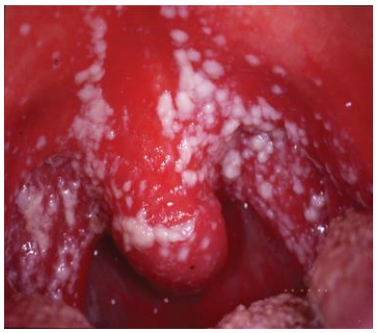
3- A number of predisposing factors have been shown to convert C. albicans from the normal commensal flora (saprophytic stage) to a pathogenic organism (parasitic stage). C. albicans is usually a weak pathogen, and candidiasis is said to affect the very young, the very old, and the very sick.

TABLE 5-2 Predisposing Factors for Oral Candidiasis and Candida-Associated Lesions

Local
Denture wearing
Smoking
Atopic constitution
Inhalation steroids
Topical steroids
Hyperkeratosis
Imbalance of the oral microflora
Quality and quantity of saliva
General
Immunosuppressive diseases
Impaired health status
Immunosuppressive drugs
Chemotherapy
Endocrine disorders
Hematinic deficiencies

4- Most candidal infections only affect mucosal linings, but rare systemic manifestations may have a fatal course.

5- Oral candidiasis is divided into primary and secondary infections . The primary infections are restricted to the oral and perioral sites, whereas secondary infections are accompanied by systemic mucocutaneous manifestations.



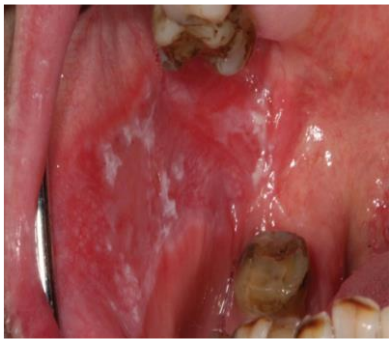


TABLE 5-1 Classification of Oral Candidiasis

Primary Oral Candidiasis	Secondary Oral Candidiasis
Acute	Familial chronic mucocutaneous candidiasis
Pseudomembranous	Diffuse chronic mucocutaneous candidiasis
Erythematous	Candidiasis endocrinopathy syndrome
	Familial mucocutaneous candidiasis
Chronic	Severe combined immunodeficiency
Pseudomembranous	DiGeorge syndrome
Erythematous	Chronic granulomatous disease
Plaque-like	Acquired immune deficiency syndrome (AIDS)
Nodular	
Candida-associated lesions	
Denture stomatitis Angular cheilitis Median rhomboid glossitis	

1-Pseudomembranous Candidiasis (thrush)

1- It is acute predominantly affects patients taking antibiotics, immunosuppressant drugs, or having a disease that suppresses the immune system.

2- typically presents with loosely attached membranes comprising fungal organisms and cellular debris, which leaves an inflamed, sometimes bleeding area if the pseudomembrane is removed.

3- The chronic form may emerge as the result of human immunodeficiency virus (HIV) infections as patients with this disease may be affected by a pseudomembranous candidal infection for a long period of time. Patients treated with steroid inhalers may also show pseudomembranous lesions of a chronic nature. Patients infrequently report symptoms from their lesions,

although some discomfort may be experienced from the presence of the pseudomembranes.

2-Erythematous Candidiasis

1- The erythematous form of candidiasis was previously referred to as atrophic oral candidiasis. However, an erythematous surface may not just reflect atrophy but can also be explained by increased vascularization.

2- The lesion has a diffuse border , which helps distinguish it from erythroplakia, which usually has a sharper demarcation and often appears as a slightly submerged lesion

3- The infection is predominantly seen in the palate and the dorsum of the tongue of patients who are using inhalation steroids. Other predisposing factors that can cause erythematous candidiasis are smoking and treatment with broad-spectrum antibiotics. The acute and chronic forms present with identical clinical features.

3-Chronic Plaque-Type and Nodular Candidiasis

1- The chronic plaque type of oral candidiasis replaces the older term, candidal leukoplakia.

2- A white irremovable plaque characterizes the typical clinical presentation, which may be indistinguishable from oral leukoplakia.

3- A positive correlation between oral candidiasis and moderate to severe epithelial dysplasia has been observed, and both the chronic plaque-type and the nodular type of oral candidiasis have been associated with malignant transformation, but the possible role of yeasts in oral carcinogenesis is unclear.

4-Denture Stomatitis

1- The most prevalent site for denture stomatitis is the denture- bearing palatal mucosa It is unusual for the mandibular mucosa to be involved.

2- Denture stomatitis is classified into three different types. **Type I** is limited to minor erythematous sites caused by trauma from the denture. **Type II** affects a major part of the denture-covered mucosa. In addition to the features of type II, **type III** has a granular mucosa. The denture serves as a vehicle that accumulates sloughed epithelial cells and protects the microorganisms from physical influences such as salivary flow.

3- The microflora is complex and may, in addition to *C. albicans* contain bacteria from several genera, such as *Streptococcus*-, *Veillonella*-, *Lactobacillus*-, *Prevotella*- (formerly *Bacteroides*), and *Actinomyces strains*. It is not known to what extent these bacteria participate in the pathogenesis of denture stomatitis.

5-Angular Cheilitis

1- Angular cheilitis presents as infected fissures of the commissures of the mouth, often surrounded by erythema The lesions are frequently coinfected with both Candida albicans and Staphylococcus aureus.

2- VitaminB12 deficiency, iron deficiencies, and loss of vertical dimension have been associated with this disorder. Dry skin may promote the development of fissures in the commissures, allowing invasion by the microorganisms.

3- Thirty percent of patients with denture stomatitis also have angular cheilitis, but this infection is only seen in 10% of denture-wearing patients without denture stomatitis.

6-Median Rhomboid Glossitis

1- Median rhomboid glossitis is clinically characterized by an erythematous lesion in the center of the posterior part of the dorsum of the tongue. As the name indicates, the lesion has an oval configuration. This area of erythema results from atrophy of the filiform papillae and the surface may be lobulated.

2- The etiology is not fully clarified, but the lesion frequently shows a mixed bacterial/fungal microflora. Biopsies yield candidal hyphae in more than 85% of the lesions. Smokers and denture-wearers have an increased risk of developing median rhomboid glossitis as well as patients using inhalation steroids.

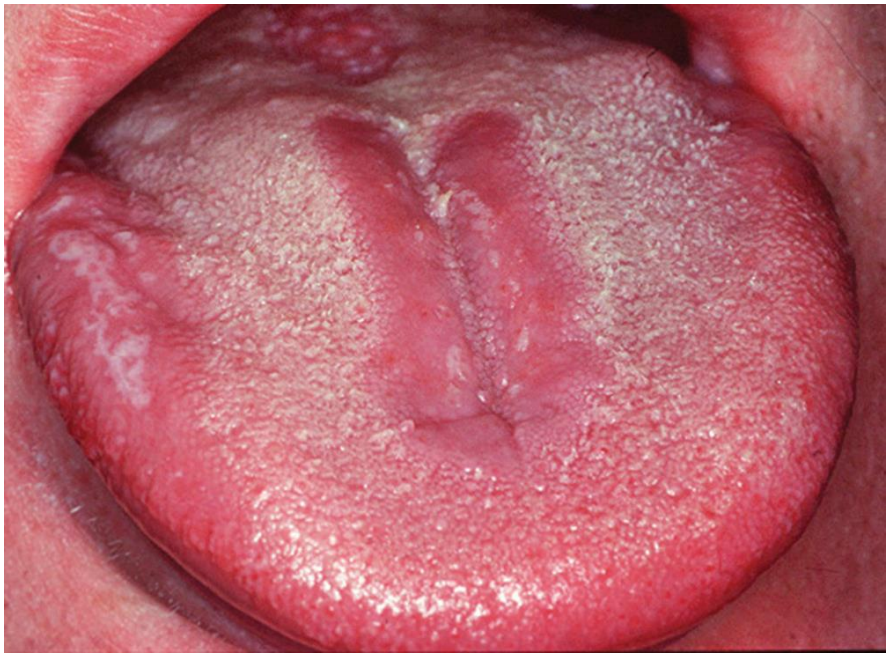
3- Median rhomboid glossitis is asymptomatic, and management is restricted to a reduction of predisposing factors. The lesion does not entail any increased risk for malignant transformation.

7-Oral Candidiasis Associated with HIV

1- More than 90% of acquired immune deficiency syndrome (AIDS) patients have had oral candidiasis during the course of their HIV infection, and the infection is considered a portent of AIDS development .

2- The most common types of oral candidiasis in conjunction with HIV are pseudomembranous candidiasis, erythematous candidiasis, angular cheilitis, and chronic plaque-like candidiasis.

3- As a result of the **highly active antiretroviral therapy (HAART)**, the prevalence of oral candidiasis has decreased substantially.



Erythematous candidiasis at the central part of the tongue in an AIDS patient. Hairy leukoplakia can be seen at the right lateral border.

Diagnosis and Laboratory Findings for candida infection

1- Smear from the infected area comprising epithelial cells, creates opportunities for detection of the yeasts. The material is fixed in isopropyl alcohol and air-dried before staining with periodic acid–Schiff (PAS). The detection of yeast organisms in the form of hyphae- or pseudohyphae-like structures is usually considered a sign of infection although these structures have also been identified in normal oral mucosa.

2- This technique is particularly useful when pseudomembranous oral candidiasis and angular cheilitis are suspected.

3- In chronic plaque-type and nodular candidiasis, cultivation techniques have to be supplemented by a histopathologic

examination. This examination is primarily performed to identify the presence of epithelial dysplasia and to identify invading candidal organisms by PAS staining. However, for the latter, there is a definitive risk of false-negative results.

Management of candida infection

1- All the predisposing factors that may cause occurrence and recurrence of candida infection should be addressed and managed along with any pharmaceutical therapy, Local factors are often easy to identify but sometimes not possible to reduce or eradicate.

2- In smokers, cessation of the habit may result in disappearance of the infection even without antifungal treatment.

3- nystatin and amphotericin B are usually the first choices in treatment of primary oral candidiasis and are both well tolerated.

4- Whenever possible, elimination or reduction of predisposing factors should always be the first goal for treatment of denture stomatitis as well as other opportunistic infections. This involves improved denture hygiene and a recommendation not to use the denture while sleeping. the denture should be stored in antimicrobial solutions during the night. Different solutions, including alkaline peroxides, alkaline hypochlorites, acids, disinfectants, and enzymes, have been suggested. The latter seems to be most effective against candidal strains. Chlorhexidine may also be used but can discolor the denture and also counteracts the effect of nystatin.

5- Type III denture stomatitis may be treated with surgical excision in an attempt to eradicate microorganisms present in the deeper fissures of the granular tissue. If this is not sufficient, continuous treatment with topical antifungal drugs should be considered.

6- Topical treatment with azoles such as miconazole is the treatment of choice for angular cheilitis often infected by both *S. aureus* and candidal strains. This drug has a biostatic effect on *S. aureus* in addition to the fungistatic effect. If angular cheilitis comprises an erythema surrounding the fissure, a mild steroid ointment (Hydrocortisone ointment) may be required to suppress the inflammation. If the antifungal treatment failed , a potent topical antibacterial cream (fusicid acid) must be used with the miconazole ointment .To prevent recurrences, patients have to apply a moisturizing cream, which may prevent new fissure formation

7- Systemic azoles may be used for deeply seated primary candidiasis, such as chronic hyperplastic candidiasis, denture stomatitis, and median rhomboid glossitis with a granular appearance, and for therapy-resistant infections, mostly related to compliance failure.

8- Persistent chronic plaque-type and nodular candidiasis have been suggested to be associated with an increased risk for malignant transformation compared with leukoplakia, not infected by candidal strains.

9- Although not strictly an antifungal drug, iodoquinol has antifungal and antibacterial properties. When compounded in a cream base with a corticosteroid, this material is very effective as topical therapy for angular cheilitis.

Drug	Form	Dosage	Comments
Amphotericin B	Lozenge, 10 mg	Slowly dissolved in mouth 3–4 × /d after meals for 2 wk minimum	Negligible absorption from gastrointestinal tract. When given IV for deep mycoses may cause thrombophlebitis, anorexia, nausea, vomiting, fever, headache, weight loss, anemia, hypokalemia, nephrotoxicity, hypotension, arrhythmias, etc.
	Oral suspension, 100 mg/mL	Placed in the mouth after food and retained near lesions 4 × /d for 2 wk	
Nystatin	Cream	Apply to affected area 3–4 × /d	Negligible absorption from gastrointestinal tract. Nausea and vomiting with high doses.
	Pastille, 100,000 U	Dissolve 1 pastille slowly after meals 4 × /d, usually for 7 d	
	Oral suspension, 100,000 U	Apply after meals 4 × /d, usually for 7 d, and continue use for several days after postclinical healing	
Clotrimazole	Cream	Apply to the affected area 2–3 times daily for 3–4 wk	Mild local effects. Also has antistaphylococcal activity.
	Solution	5 mL 3–4 times daily for 2 wk minimum	
Miconazole	Oral gel	Apply to the affected area 3–4 times daily	Occasional mild local reactions. Also has antibacterial activity. Theoretically the best antifungal to treat angular cheilitis. Interacts with anticoagulants (warfarin), terfenadine, cisapride, and astemizole. Avoid in pregnancy and liver disease.
	Cream	Apply twice per day and continue for 10–14 d after the lesion heals	

Topical antifungal therapy

Ketoconazole	Tablets	200–400-mg tablets taken once or twice daily with food for 2 wk	May cause nausea, vomiting, rashes, pruritus, and liver damage. Interacts with anticoagulants, terfenadine, cisapride, and astemizole. Contraindicated in pregnancy and liver disease.
Fluconazole	Capsules	50–100 mg capsules once daily for 2–3 wk	Interacts with anticoagulants, terfenadine, cisapride, and astemizole. Contraindicated in pregnancy and liver and renal disease. May cause nausea, diarrhea, headache, rash, liver dysfunction.
Itraconazole	Capsules	100 mg capsules daily taken immediately after meals for 2 wk	Interacts with terfenadine, cisapride, and astemizole. Contraindicated in pregnancy and liver disease. May cause nausea, neuropathy, rash.

Systemic Azole antifungal therapy

ORAL HAIRY LEUKOPLAKIA

1- Oral hairy leukoplakia (OHL) is the second most common HIV-associated oral mucosal lesion. HL has been used as a marker of disease activity since the lesion is associated with low CD4+ T-lymphocyte counts. The lesion is not pathognomonic for HIV disease since other states of immune deficiencies, such as caused by immunosuppressive drugs and cancer chemotherapy, have also been associated with OHL.

2- Rarely, individuals with a normal immune system may present with OHL Oral hairy leukoplakia is strongly associated with Epstein-Barr virus (EBV) and with low levels of CD4+ T lymphocytes.

3- The condition is more frequently encountered in men, but the reason for this predisposition is not known. A correlation between smoking and OHL has also been observed.

4- Oral hairy leukoplakia is frequently encountered on the lateral borders of the tongue but may also be observed on the dorsum and in the buccal mucosa. The typical clinical appearance is vertical white folds oriented as a palisade along the borders of the tongue. The lesions may also be seen as white and somewhat elevated plaque, which cannot be scraped off.

5- Oral hairy leukoplakia is asymptomatic, although symptoms may be present when the lesion is superinfected with candidal strains.

6- A diagnosis of OHL is usually based on clinical characteristics, but histopathologic examination and detection of EBV can be performed to confirm the clinical diagnosis. It may most easily be confused with chronic trauma to the lateral borders of the tongue.

7- Oral hairy leukoplakia can be treated successfully with antiviral medication, but this is not often indicated as this disorder is not

associated with adverse symptoms. In addition, the disorder has also been reported to show spontaneous regression. HL is not related to increased risk of malignant transformation. Medication with HAART has reduced the number of HL to a few percent in HIV-infected patients.

PREMALIGNANT DISORDERS

leukoplakia and erythroplakia

1- The development of oral leukoplakia and erythroplakia as premalignant lesions involves different genetic events. This notion is supported by the fact that markers of genetic defects are differently expressed in different leukoplakias and erythroplakias.
2- Most oral leukoplakias are seen in patients beyond the age of 50 and infrequently encountered below the age of 30. In population studies, leukoplakias are more common in men, but a slight majority for women has been found in some studies . A comprehensive global review points at a prevalence of 2.6%.found in some studies.
3- Oral erythroplakia is not as common as oral leukoplakia, and the prevalence has been estimated to be in the range of 0.02%–0.1%.

Clinical feature

1- Oral leukoplakia is defined as a white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer. This disorder can be further divided into a homogeneous and a nonhomogeneous type.

2- The typical homogeneous leukoplakia is clinically characterized as a white, often well-demarcated plaque with an identical reaction pattern throughout the entire lesion.
3- The surface texture can vary from a smooth and thin to a leathery appearance with surface fissures sometimes referred to as “cracked mud.” The demarcation is usually distinct which is different from an OLP lesion.
4- The lesions are asymptomatic in most patients. The nonhomogeneous type of oral leukoplakia may have white patches or plaques intermingled with red elements . Due to the combined appearance of white and red areas, the nonhomogeneous oral leukoplakia has also been called erythroleukoplakia and speckled leukoplakia.
5- The clinical manifestation of the white component may vary from large white verrucous areas to small nodular structures. If the surface texture is homogeneous but contains verrucous, papillary (nodular), or exophytic components, the leukoplakia is also regarded as nonhomogeneous Oral leukoplakias, where the white component is dominated by papillary projections, similar to oral papillomas, are referred to as verrucous or verruciform leukoplakias.
6- Oral leukoplakias with a more aggressive proliferation pattern and high recurrence rate are designated as proliferative verrucous leukoplakia (PVL).The malignant potential is very high, and verrucous carcinoma or squamous cell carcinoma may be present at the primary examination. .
7- The floor of the mouth and the lateral borders of the tongue have been considered high-risk sites for malignant transformation.
8- Oral erythroplakia has not been studied as extensively as oral leukoplakia, presumably because it is less common. Erythroplakia is

defined as a red lesion of the oral mucosa that excludes other known pathologies . The lesion comprises an eroded somewhat submerged red lesion that is frequently observed with a distinct demarcation against the normal-appearing mucosa.

9- Clinically, erythroplakia is different from erythematous OLP as the latter has a more diffuse border and is surrounded by white reticular or papular structures.

10- Erythroplakia is usually asymptomatic, although some patients may experience a burning sensation in conjunction with food intake.

Diagnosis

The diagnostic procedure of oral leukoplakia and erythroplakia is identical. The provisional diagnosis is based on the clinical observation of a white or red patch that is not explained by a definable cause, such as trauma. If trauma is suspected, the cause, such as a sharp tooth or restoration, should be eliminated. If healing does not occur in two weeks, a tissue biopsy is essential to rule out malignancy.

Treatment

1- Cold-knife surgical excision, as well as laser surgery, is widely used to eradicate leukoplakias and erythroplakias but will not prevent all premalignant lesions from malignant development.

2- On the contrary, surgery has been strongly questioned as squamous cell carcinomas are almost equally prevalent in patients subjected and not subjected to surgery However, in the absence of evidenced-based treatment strategies for oral

leukoplakias, surgery will remain the treatment of choice for oral leukoplakias and erythroplakias.

3- Such a treatment regimen is supported by the fact that serial sections of the total lesion after surgical removal has shown that as much as 7% of the lesions contained frank squamous cell carcinomas, which is not revealed by an incisional biopsy Malignant transformation of oral leukoplakias has been reported in the range of 1%–20% over 1 to 30 years.

Oral Submucous Fibrosis

1. Oral submucous fibrosis is a chronic disease affecting the oral mucosa, as well as the pharynx and the upper two-thirds of the esophagus. There is substantial evidence that lends support to a critical role of areca nuts in the etiology behind oral submucous fibrosis.
2. There is also evidence of a genetic predisposition of importance for the etiology behind oral submucous fibrosis.
3. The first signs of oral submucous fibrosis are erythematous lesions, sometimes in conjunction with petechiae, pigmentations, and vesicles. These initial lesions are followed by a paler mucosa, which may comprise white marbling .
4. The most prominent clinical characteristics will appear later in the course of the disease and include fibrotic bands located beneath an atrophic epithelium. Increased fibrosis eventually leads to loss of resilience, which interferes with speech, tongue mobility, and a decreased ability to open the mouth.
5. The atrophic epithelium may cause a smarting sensation and inability to eat hot and spicy food. More than 25% of the patients

also exhibit oral leukoplakias An international consensus has been reached where at least one of the following characteristics should be present:

•• Palpable fibrous bands

•• Mucosal texture feels tough and leathery

•• Blanching of mucosa together with histopathologic features consistent with oral submucous fibrosis (atrophic epithelium with loss of rete ridges and juxta-epithelial hyalinization of lamina propria).

6. Treatment :cessation of areca nuts chewing habits.If this is successfully implemented, early lesions have a good prognosis as they may regress. A plethora of treatment strategies have been tried, such as topical and systemic steroids, supplement of vitamins and nutrients, repeated dilatation with physical devices, and surgery. None of these treatments have reached general acceptance and the long-term results are dubious.
7. Malignant transformation of oral submucous fibrosis has been estimated in the range of 7%–13% and the incidence over a 10-year period at approximately 8%

TOXIC REACTIONS

Smoker's Palate

1- The most common effects of smoking are presented clinically as dark brown pigmentations of the oral mucosa (smoker's melanosis) and as white leathered lesions of the palate, usually referred to as nicotine stomatitis or smoker's palate.

2- In smoker's palate, an erythematous irritation is initially seen, and this lesion is followed by a whitish palatal mucosa reflecting a hyperkeratosis .

3- As part of this lesion, red dots can be observed representing orifices of accessory salivary glands, which can be enlarged and display metaplasia.
4- Smoker's palate is more prevalent in men and is a common clinical feature in high consumers of pipe tobacco and cigarettes and among individuals who practice inverse smoking. The etiology is probably more related to the high temperature rather that the chemical composition of the smoke, although there is a synergistic effect of the two.

REACTIONS TO MECHANICAL TRAUMA

Morsicatio

1- Morsicatio is instigated by habitual chewing .This parafunctional behavior is done unconsciously and is therefore difficult to bring to an end.
2- Morsicatio is most frequently seen in the buccal and lip mucosa and never encountered in areas that are not possible to traumatize by habitual chewing.
3- Morsicatio is three times more common among women. Morsicatio has a very typical clinical appearance, and the diagnosis is relatively easy to establish, with one exception. If the lesion affects the borders of the tongue, it may mimic hairy leukoplakia.
4- The management is limited to assurance, and the patient should be informed about the parafunctional behavior.

Frictional Hyperkeratosis

Oral frictional hyperkeratosis is typically clinically characterized by a white lesion without any red elements. The lesion is observed in areas of the oral mucosa subjected to increased friction caused by, for example, food intake.

Clinical Findings

Frictional hyperkeratosis is often seen in edentulous areas of the alveolar ridge but may also be observed in other parts of the oral mucosa exposed to increased friction or trauma.

OTHER RED AND WHITE LESIONS

Benign Migratory Glossitis (Geographic Tongue)

Geographic tongue is an annular lesion affecting the dorsum and margin of the tongue. The lesion is also known as erythema migrans.

Etiology and Pathogenesis

Although geographic tongue is one of the most prevalent oral mucosal lesions, there are virtually no studies available with the objective to elucidate the etiology behind this disorder. Heredity has been reported, suggesting the involvement of genetic factors in the etiology.

Clinical Findings

1- Geographic tongue is circumferentially migrating and leaves an erythematous area behind, reflecting atrophy of the filiform papillae. The peripheral zone disappears after some time, and healing of the depapillated and erythematous area starts.

2- Geographic tongue is characterized by periods of exacerbation and remission with different durations over time. The disorder is usually non-symptomatic, but some patients are experiencing a smarting sensation. In these cases, a parafunctional habit, revealed by indentations at the lateral border of the tongue, may be a contributing factor to the symptoms.

3- Patients often report that their lesions are aggravating during periods of stress. Geographic tongue and fissured tongue may be observed simultaneously. Most likely, fissured tongue should be interpreted as an end stage of geographic tongue in some patients .

4- A geographic appearance can be rarely observed at other sites of the oral mucosa than on the dorsum of the tongue and then is denoted geographic stomatitis.

5- An increased prevalence of geographic tongue has been observed in patients with generalized pustular psoriasis. But no studies have demonstrated that patients with geographic tongue are at increased risk of acquiring psoriasis. smoking was revealed.

Management

1- As the etiology is unknown, no causal treatment strategy is available. Symptoms are rarely present, and the management is confined to proper information about the disorder’s benign character.

2- When symptoms are reported, topical anesthetics may be used to obtain temporary relief. Other suggested treatment strategies include antihistamines, anxiolytic drugs, or steroids, but none of these has been systematically evaluated.

Leukoedema

Etiology and Pathogenesis

The etiology of leukoedema is not clear.

Clinical Findings

1- Leukoedema is a white and veil-like alteration of the oral mucosa that is merely considered a normal variant. The condition is often encountered bilaterally in the buccal mucosa and sometimes at the borders of the tongue.
2- Leukoedema is less clinically evident after stretching the mucosa but reappears after this manipulation is discontinued. In more pronounced cases, leukoedema is accompanied by mucosal folds. The condition is asymptomatic and has no malignant potential.

Management

There is no demand for treatment as the condition is nonsymptomatic and has no complications, including premalignant features.

White Sponge Nevus

Epidemiology

White sponge nevus has been listed as a rare disorder by the National Institutes of Health, which implicates a prevalence below 1 in 200,000. In a population study of 181,338 males between 18 and 22 years of age, two cases of white spongenevus were identified. The clinical appearance usually commences during adolescence, and the gender distribution has been reported to be equal.

Clinical Findings

1- White sponge nevus is an autosomal dominant disorder with high penetrans. The typical clinical appearance is a white lesion with an elevated and irregular surface comprising fissures or plaque formations.
2- The most affected sites are the buccal mucosa, but the lesion may also be encountered in other areas of the oral cavity covered by parakeratinized or non-keratinized epithelium. The disorder may also involve extraoral sites, such as the esophagus and anogenital mucosa.
3- Although the lesion does not entail any symptoms, it may cause dysphagia when the esophagus is involved.

Diagnosis

1- White sponge nevus may constitute a differential diagnostic problem as this disorder may be taken for other oral dyskeratoses, for example, oral leukoplakia and plaque-type candidiasis.
2- The hallmark microscopic feature of this disorder is pronounced intracellular edema of the superficial epithelial cells, predominantly located within the stratus spinosum. Cells with pyknotic nuclei are present, and these cells may imitate koilocytosis observed in viral infections. No or just mild infiltrations may be seen in the subepithelial tissue.

Management

White sponge nevus does not entail any symptoms, and no treatment is therefore required.

Hairy Tongue

Etiology and Pathogenesis

The etiology of hairy tongue is unknown in most cases. A number of predisposing factors have been related to this disorder, such as:

1- neglected oral hygiene
2- a shift in the microflora
3- antibiotics and immunosuppressive drugs
4- oral candidiasis
5- excessive alcohol consumption
6- oral inactivity
7- therapeutic radiation.

The impact of ignored oral hygiene and oral inactivity is supported by the high prevalence of hairy tongue in hospitalized patients, who are not able to carry out their own oral hygiene. Hairy tongue is also associated with smoking habits.

Clinical Findings

1- Hairy tongue is characterized by an impaired desquamation of the filiform papilla, which leads to the hairy-like clinical appearance.
2- The elongated papillae have to reach lengths in excess of 3 mm to be classified as "hairy," although lengths of more than just 15 mm have been reported in hairy tongue.
3- The lesion is commonly found in the posterior one-third of the tongue but may involve the entire dorsum. Hairy tongue may adopt colors from white to black depending on food constituents and the composition of the oral microflora. Patients with this disorder may experience both physical discomfort and esthetic embarrassment related to the lengths of the filiform papillae.

Management

The treatment of hairy tongue is focused on reduction or elimination of predisposing factors and removal of the elongated filiform papillae. The patients should be instructed on how to use devices developed to scrape the tongue.