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A Comprehensive Guide to Diagnosing Oral Mucosal Lesions: Part III. Noninfectious Processes

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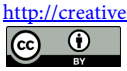

Abstract

Numerous noninfectious conditions that affect the oral mucosa often present similarly as ulcerations, erythema, or plaques in addition to infectious causes. These include immune-mediated diseases like pemphigus vulgaris, mucous membrane pemphigoid, Behçet disease, and systemic lupus erythematosus, as well as potentially malignant conditions like leukoplakia, erythroplakia, and oral lichen planus. Actinic cheilitis, oral submucous fibrosis, and graft-versus-host disease are among the other conditions that exhibit distinct oral features with systemic implications. Since many lesions may indicate malignancy or systemic disease, accurate clinical and histopathological evaluation is necessary for diagnosis. Improved patient outcomes are made possible by prompt intervention and early recognition.

Subject Areas

Dentistry

Keywords

Oral Lesions, Potentially Malignant Disorder, Mouth Diseases, Autoimmune Disease

1. Introduction

The oral cavity can also be the site of a wide variety of non-infectious disorders, many of which present with similar clinical features like ulceration, erythema, or mucosal plaques, in addition to infectious causes. These conditions may be immune-mediated or associated with systemic diseases, and they demand a thorough clinical and histopathological evaluation to ensure accurate diagnosis and

appropriate management. The most important noninfectious processes that affect the oral mucosa are examined in this section, with an emphasis on their clinical manifestations, diagnostic criteria, and potential for malignant transformation or systemic consequences. In cases where oral lesions may be the first or only signs of a broader pathology, understanding these entities is essential for prompt detection and treatment for dental practitioners.

2. Oral Potentially Malignant Disorders

2.1. Leukoplakia (LK)

Description:

LK, as defined by the World Health Organization (WHO), is a "white plaque of uncertain risk, having ruled out (other) known diseases or disorders that do not increase the risk of cancer" [1]. Risk factors are equivalent to those for oral cancer. They include smoking, excessive alcohol consumption, betel nut chewing, advanced age and exposure to UV rays [2].

Clinical manifestations:

LK includes 2 main clinical forms:

- **Homogeneous LK**: A thin, flat, uniform white lesion with a smooth surface that may show slight fissures.
- **Non-homogenous nodular LK**: Characterized by small polypoid or rounded outgrowths, which may be red or white.
- **Non-homogenous verrucous LK:** Has a raised, exophytic surface with a wrinkled or wavy appearance [3] [4] (**Figure 1**).
- **Erythroleukoplakia:** is defined as a predominantly leukoplakic lesion, presenting areas of erythema in the form of small punctate spots or large irregular patches [2].

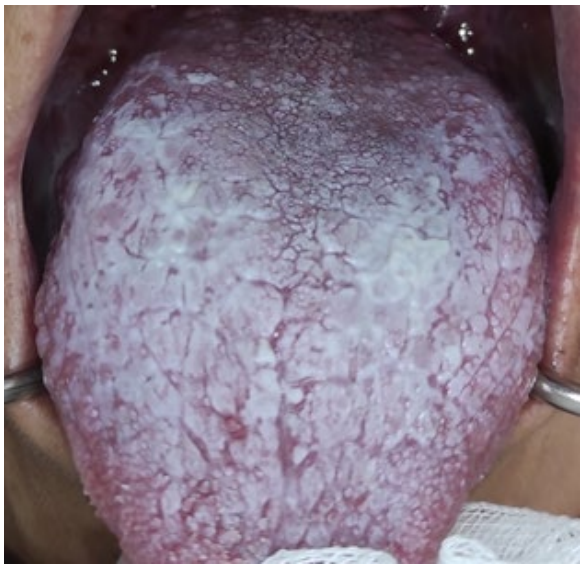

Figure 1. Non homogenous leukoplakia manifesting on the tongue of a heavy smoker patient.

The most frequently affected areas by LK are the lateral edge of the tongue, the floor of the mouth, followed by the buccal mucosa, the palate, and the gingiva.

Homogenous LK has a lower risk of transformation (0.6% - 5%) compared to the non-homogeneous cases (20% - 25%). Verrucous LK is the most aggressive entity with a malignant transformation rate of 61.0% over an average follow-up period of 7.4 years [1].

Diagnosis:

The diagnosis of LK is based on an expert clinical and pathological examination of the oral cavity [1].

The differential diagnosis of leukoplakic lesions can be grouped into the following main categories: congenital anomalies (leukoedema, white naevus), infections (pseudomembranous candidiasis), inflammations (lichen planus, lichenoid mucositis, oral lesions of systemic lupus erythematosus), as well as mucosal lesions (chemical or thermal burns, linea alba and friction keratoses) [2].

Management:

Dental practitioners should recognize PK as a potentially malignant disorder. Any persistent white lesion that cannot be rubbed off or clinically identified must be biopsied. Management strategies for oral leukoplakia include surgical excision, observation, and medical therapy. Surgery, including CO_2 laser and photodynamic therapy, shows variable recurrence rates (0% - 35%). Due to frequent recurrences, topical agents like retinoids, bleomycin, and phytochemicals have been explored. While complete response rates ranged from 25% to 40%, recurrence remained common across treatments [1].

2.2. Erythroplakia (EPK)

Description:

EPK appears as a red patch on the oral mucosa, which cannot be attributed to any other identifiable lesion. It must not be due to trauma, vascular lesions or inflammation. EPK generally affects middle-aged and elderly patients, appearing most often between the sixth and seventh decades of life [4].

Although rare, EPK has a much higher rate of malignant transformation than other precancerous conditions such as LK or submucosal fibrosis. Reported rates of malignant transformation range from 14% to 50%, four times higher than those of leukoplakic lesions [2].

Clinical manifestations:

EPK appears as a smooth solitary erythematous plaque accompanied by a burning sensation. Patients may experience discomfort, tingling and sensitivity to touch, hot drinks or spicy foods. Erosive, granular or nodular changes may appear in long-standing lesions [4].

The soft palate is the most common site for EPK, then comes the ventral tongue, floor of mouth, and tonsillar pillars [5].

Diagnosis:

The definitive diagnosis of EPK is established after identification and, if

possible, elimination of presumed etiological factors. In the case of persistent lesions, histopathological examination is necessary to confirm the diagnosis [6].

Differential diagnosis includes erythematous candidiasis, denture-associated stomatitis, erosive, inflammatory/infective disorders, desquamative gingivitis, lupus erythematous, erosive lichen planus and Pemphigoid [4] [7].

Management:

Due to its high malignant potential, any erythematous oral patch unlinked to trauma or inflammation requires urgent biopsy. Dentists must refer the patient promptly and ensure close monitoring after diagnosis. Risk factor counseling (e.g., smoking cessation) and ongoing clinical surveillance are vital even after treatment [6].

2.3. Oral Lichen Planus (OLP)

Description:

OLP is an inflammatory disease of immune origin with characteristic relapses and remissions, that causes specific lesions on the mucosa that are usually multiple and have a bilateral distribution [8]-[10]. It affects around 2% of the adult population, with a predominance of women, and occurs mainly in middle age [8] [11].

The etiology of oral lichen planus remains uncertain, but it is assumed that external factors such as certain viruses (hepatitis C virus), as well as various medications like non-steroidal anti-inflammatory drugs, antihypertensives, antirheumatics, antimalarials, gold salts, penicillamine and retroviral treatments. Dental metallic materials and trauma may also play a role [8].

Clinical manifestations:

OLP is usually bilateral, symmetric or asymmetric, located on buccal mucosa, tongue, lips and/or gingiva. The clinical presentation of OLP is characterized by the presence of slender white lines forming a lace-like network (Wickham's striae) in all its forms [8] [12].

There are mainly 6 clinical forms of OLP [13]:

- **Reticular OLP:** Usually asymptomatic with the presence of Wickham's striae encircled by distinctive erythematous borders (**Figure 2**).
- **Papular OLP:** As small asymptomatic papules on the mucous membranes, particularly the buccal mucosa, and is often associated with the reticular form of oral lichen planus [9] [11].
- **Plaque-like OLP:** is clinically similar to LK, presenting as large, homogeneous white plaques [13].
- **Erosive (ulcerated) OLP:** presents as irregular erosions or ulcerations covered by fibrinous plaques or pseudomembranes, with a varnished bright red appearance, variable size and slight protrusion (**Figure 3**) [4].
- **Atrophic OLP:** manifests as atrophic, erythematous lesions with fine, white radiating striae at the periphery [11].
- **Bullous OLP:** is the rarest clinical manifestation, that mainly affects the ducts of the salivary glands and is characterized by the formation of vesicles, which

rupture, causing painful ulcerations [9].

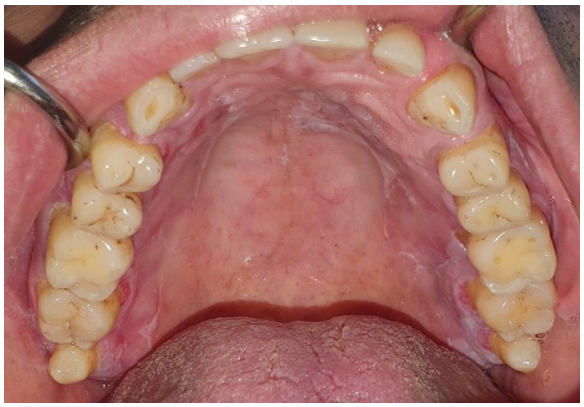

Figure 2. Asymptomatic reticular lichen planus on the hard palate.

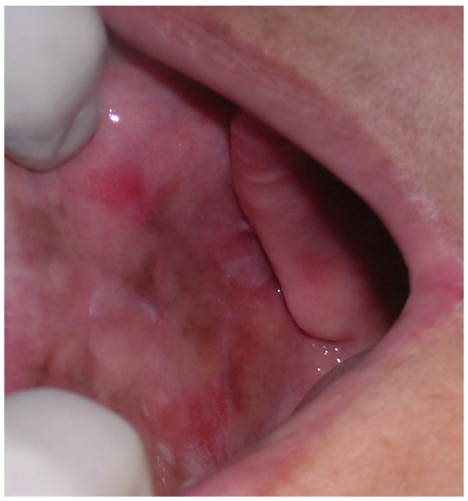

Figure 3. Erosive lichen planus on the jugal mucosa.

Diagnosis:

Clinical features alone may be sufficient to make a diagnosis, especially in the "classic" reticular form. Histopathological examination can be indicated if malignancy is suspected [13] [14].

Oral lichenoid lesions (OLL) are striated intraoral lesions similar to LP, but with an identifiable underlying cause. Certain distinguishing features, such as unilateral presentation or changes due to dental amalgams and medications, aid in differential diagnosis. OLL are divided into four categories: Atypical OLL, contact lesions due to dental materials, lichenoid drug reactions and lesions associated with graft-versus-host disease [5] [9].

Management:

OLP management involves identifying and eliminating local irritants (e.g., sharp restorations, allergens). Symptomatic cases benefit from topical corticosteroids (e.g., clobetasol). Dentists should monitor lesions regularly due to the risk of malignant transformation, particularly in erosive forms. Referral may be necessary

for biopsy or systemic therapy [14].

2.4. Oral Submucous Fibrosis (OSF)

OSF is a chronic disease characterized by reduced elasticity of the oral cavity, oropharynx and upper esophagus. The condition is associated with the use of betel quid and areca nut chewing as the main risk factors [2] [15] [16].

Early symptoms include a burning sensation of the mucosa in response to spicy foods, accompanied by atrophy and whitening of oral cavity structures. In more advanced stages, palpable fibrous bands and a leathery texture of the mucosa appear. These changes cause rigidity of the tongue, reducing its mobility and progressively limiting mouth opening [2] [15] [17].

2.5. Actinic Cheilitis (AC)

AC is a precancerous lesion of the lower lip, characterized by inflammation due to excessive sun exposure; and that is seen in middle-aged, light-skinned males [2]. Manifestations include isolated or diffuse lesions in the form of crumbly or scaly white patches, interspersed with red areas. More rarely, patients may also experience dry lips [18]. Approximately 6% - 8% of lesions show dysplasia on biopsy. Depending on clinical signs and histopathological findings, treatment is generally based on superficial surgical excision [17].

2.6. Palatal Lesion in Reverse Smokers

Reverse smoking is the habit of placing the lit end of a cigarette inside the oral cavity [2]. Clinically, it manifests as thick white patches on the palate, mucous nodules, growths around the orifices of the small palatine mucous glands, as well as yellowish-brown discoloration, erythema and ulcerations [18]. Lesions may appear as red, white or mixed (red and white) spots on a background marked by tobacco stains [7].

3. Immune-Induced Diseases

3.1. Pemphigus Vulgaris (PV)

Description:

PV is the most common form of the pemphigus group. It is a potentially fatal cutaneous-mucosal autoimmune disease characterized by the formation of bullae and ulcers. The oral mucosa is often affected before skin lesions appear [19] [20]. The disease affects both men and women, generally between the ages of 40 and 60 [21].

Clinical manifestations:

Painful and delicate superficial ulcerations (of 0.5 to 2 cm), bullae and erosions appear on the oral mucosa, palate and tongue as first manifestations of the disease; frequently extending into the posterior oropharynx. This condition is also often associated with desquamative gingivitis, causing chronic bleeding of gum tissue [21]-[23].

Diagnosis:

PV must be distinguished from other chronic mucosal and skin diseases, such as oral OLP, mucosal pemphigoid and lupus oral lesions, due to their clinical similarities. Diagnosis of pemphigus is based on systematic biopsy of injured tissue, supplemented by immunological tests on the patient's tissue and serum [19].

Management:

Dental practitioners are often the first to identify painful oral ulcers in PV. They should suspect the disease when lesions are chronic, fragile, and involve desquamative gingivitis. Immediate referral for biopsy and immunofluorescence testing is needed. Avoiding trauma during dental care and managing oral hygiene with non-irritating rinses are important supportive measures [24].

3.2. Mucous Membrane Pemphigoid (MMP)

Description:

MMP is a chronic subepithelial autoimmune disorder marked by the development of bullae within the oral cavity. These bullae eventually rupture, leading to ulceration. The disease predominantly affects the oral mucosa but may also involve the genital, conjunctival, and skin mucosa in individuals over 50 years [19] [20] [25].

MMP has the potential to cause significant scarring of the mucous membranes. In advanced cases, ocular involvement is common and may result in blindness due to scarring fibrosis [20] [25].

Clinical manifestations:

The most common manifestation of MMP is desquamative gingivitis.

In less cases, erythema, erosions covered with a pseudomembrane, ulcers, and sometimes intact vesicles or bullae can affect the palatal, gingival, labial and buccal mucosa, as well as the tongue [22].

These conditions manifest themselves through a variety of symptoms, from burning and bleeding to masticatory difficulties [20].

Diagnosis:

Diagnosing MMP through oral manifestations involves a combination of clinical evaluation (lesion appearance, symptom inquiry, chronicity and presence of other ocular, genital, or skin lesions), histopathological analysis, and immunological testing.

Histological findings typically show epithelial detachment from the connective tissue. Direct immunofluorescence is crucial for confirming diagnosis in doubtful cases, demonstrating linear involvement at the basal membrane. It is particularly valuable for differentiating pemphigoid from pemphigus, lichen planus, periodontal disease, and systemic lupus erythematosus (SLE) [19] [20].

Management:

Dental practitioners should consider mucous membrane pemphigoid (MMP) in cases of persistent gingival erosions or desquamation and refer promptly for biopsy and immunological testing. Mild forms can often be managed with topical

corticosteroids, sometimes combined with dapsone. Severe cases typically require systemic treatment and may respond slowly. Ocular involvement must be closely monitored due to the risk of serious complications. Effective management also includes gentle oral hygiene, avoidance of trauma, and coordination with specialists for systemic care [20].

3.3. Behçet Disease (BD)

Description:

BD is a chronic, multisystemic inflammatory pathology with unclear etiology, manifesting itself mainly as oral aphthous ulcers, uveitis, skin lesions and genital ulcerations. Involvement of the intestines, blood vessels or central nervous system (CNS) is rarer [26].

Clinical manifestations:

The clinical presentation begins with oral aphthous ulceration and progresses to systemic involvement.

Oral aphthous stomatitis is the most frequent symptom of BD, affecting 98.1% of patients. These ulcers resemble those in other diseases and 25% of healthy individuals, appearing as well-defined lesions with a necrotic base and a red rim, often clustering on various oral sites (most frequently on the lips and cheeks) [26] [27].

Diagnosis:

The diagnosis of BD is primarily based on clinical examination, as there are no established pathognomonic symptoms or definitive laboratory tests. While multiple diagnostic criteria exist, the most widely recognized are those developed by the International Study Group for BD. These criteria mandate that recurrent oral ulcerations are present, plus at least 2 of the following: recurrent genital ulceration, eye lesions, skin lesions [27].

Management:

BD is treated with local or systemic corticosteroids, often in combination with immunosuppressants to reduce relapse risk. Early management of oral and mucocutaneous lesions is critical to prevent disease progression and irreversible organ damage, particularly during active phases. The condition can be life-threatening, especially with vascular complications such as aneurysm rupture or thrombosis [20].

3.4. Crohn's Disease

Description:

Crohn's disease falls into the group of chronic inflammatory bowel diseases, which involves both genetic and environmental factors. It can affect any part of the gastrointestinal tract, but most frequently manifests itself in the ileocecal region, causing thickening and ulceration of the mucosa [28].

Clinical manifestations:

The main symptoms include abdominal pain, constipation or diarrhea,

obstructions, and malabsorption, often accompanied by systemic issues like fever, fatigue, anemia, appetite loss, and weight loss [27].

As for oral lesions, frequent signs involve aphthous ulcers, persisting or recurrent lip swelling, "cobblestone" mucosa, angular stomatitis, gingival hypertrophy, and erythema. Other findings include mucosal macules, linear ulcers with nodular swelling on the tongue, indurated lower lip fissures, and occasionally orocutaneous fistulas [27] [28] (**Figure 4**).

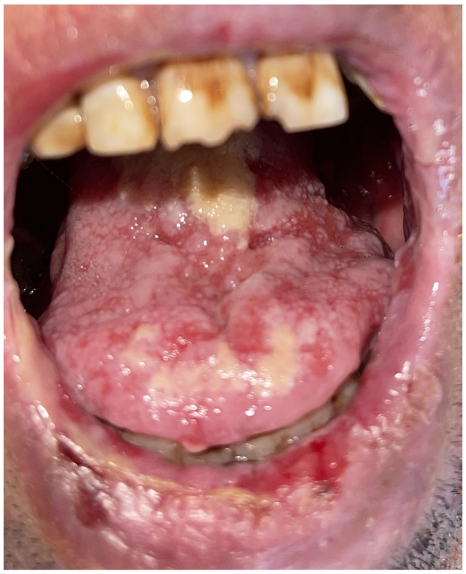

Figure 4. Oral manifestations of Crohn's Disease.

Diagnosis:

There are no gold standard criteria for diagnosing CD. Diagnosis is based on a combination of clinical data, including patient history, physical examination, blood tests (e.g., for anemia) and biopsy of oral lesions to determine the presence of non-caseating granulomas to support the diagnosis. Diagnostic tools include colonoscopy with biopsies for histological analysis, as well as imaging studies such as barium studies and small bowel imaging. Autoimmune antibody testing and genotyping are not currently recommended by the American College of Gastroenterology [27].

Management:

Nutritional deficiencies should be addressed with appropriate supplementation, and dietary modifications are often advised. Maintaining good oral hygiene is essential, and corticosteroids—whether systemic, topical, or intralesional—are commonly used in management. Fluoride supplementation may also offer additional benefits [28].

3.5. Erythema Multiform (EM)

Description:

EM is a mucocutaneous condition marked by ulcerative, vesicular, and target-

shaped or iris lesions, typically appearing symmetrically on the extremities and trunk. EM presents in a range of clinical forms, most often triggered by hypersensitivity reactions, and is known for its pronounced tendency to recur [29].

It is primarily triggered by infections, with herpes simplex virus responsible for 70% - 80% of cases. Medications such as sulfonamides, NSAIDs, penicillin, and anticonvulsants can also act as triggers [30].

Clinical manifestations:

EM typically follows an episodic course lasting 1 to 4 weeks and is most observed in young, otherwise healthy individuals.

Oral lesions in EM can vary significantly in appearance, ranging from diffuse erythema to multiple shallow ulcers. Vesicles or bullae may also appear initially. The buccal mucosa, palate, and tongue are most affected, along with lip lesions showing hemorrhagic crusting. Oral and perioral pain can range from mild to severe, affecting speech, eating, and fluid intake. However, lip and oral lesions generally heal without scarring [30].

Diagnosis:

The diagnosis primarily relies on patient history and clinical presentation, as histopathological and laboratory findings are nonspecific. Other conditions in the differential diagnosis include primary HSV gingivostomatitis, autoimmune vesiculobullous diseases (e.g., pemphigus vulgaris, bullous pemphigoid, paraneoplastic pemphigus), urticaria, Stevens-Johnson syndrome (SJS), and fixed drug eruptions [30].

Management:

EM treatment depends on severity. Mild cases are managed with topical corticosteroids (e.g., fluocinonide 0.05% applied 2 - 3×/day) and anesthetic mouthwash (lidocaine 2%, diphenhydramine, and Maalox, up to 4×/day). Severe cases may require prednisone 40 - 60 mg/day tapered over 2 - 4 weeks. Recurrent HSV-associated EM can be prevented with antivirals: acyclovir 400 mg BID, valacyclovir 500 mg BID, or famciclovir 250 mg BID; refractory cases may need azathioprine (100 - 150 mg/day), mycophenolate mofetil (1000 mg BID), or dapsone (100 - 200 mg/day) [30].

3.6. Stevens Johnson Syndrome (SJS)

Description:

SJS is a severe, potentially life-threatening hypersensitivity reaction that primarily affects the skin and mucous membranes.

The major causes of SJS have been attributed to drugs. The most common are sulfa drugs, (sulfonamides and co-trimoxazole); anticonvulsive drugs, such as phenytoin, carbamazepine, and phenobarbital; and nonsteroidal anti-inflammatory drugs (butazone and oxicam) [29].

Clinical manifestations:

In SJS, skin lesions include small blisters on purple macules or target lesions, mainly on the torso. Nikolsky's sign is positive, with limited epidermal detachment.

Lesions last 2 to 6 weeks. Mucosal involvement, particularly of the lips, and scarring are common, with oral, ocular, nasal, and genital mucosa often affected [22].

Diagnosis:

The history and clinical appearance are the most important for making a diagnosis. The histology is non-specific [22].

Management:

There is no definitive treatment for SJS. Prompt withdrawal of the causative drug and management of infections are critical. Severe cases require hospitalization, supportive care, and possibly treatments like IV immunoglobulin, plasmapheresis, or cyclophosphamide. Prophylactic antibiotics, nutritional support, fluid balance, and ophthalmologic care are essential to reduce complications and mortality [29].

3.7. Systemic Lupus Erythematous (SLE)

Description:

SLE is a chronic autoimmune disease marked by diverse clinical symptoms and systemic involvement and often fluctuating between periods of flares and remission. Autoantibodies target nuclear and cytoplasmic antigens, leading to widespread effects [27] [31].

While it can impact any organ, it most commonly affects the joints, skin, lungs, heart, nervous system, blood vessels, and liver [31].

Clinical manifestations:

SLE predominantly affects women and is most diagnosed between the ages of 20 and [32].

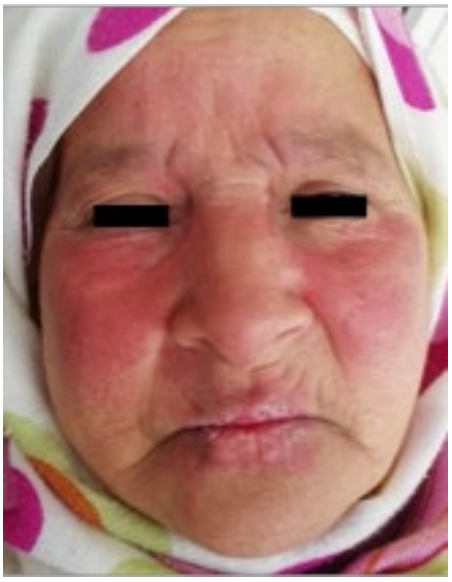

Figure 5. Malar rash in a female patient with SLE.

SLE symptoms often begin with non-specific general signs, such as fever,

fatigue and weight loss. Subsequently, various organs may be affected, including mucocutaneous (such as malar rashes), renal, neurological/psychiatric, arthritic, cardiovascular and hematological manifestations [31] (**Figure 5**).

Oral manifestations are among the most frequent, and can sometimes be the only lesions present, enabling early diagnosis and intervention. The classical oral lupus lesion is a whitish plaque with erythema in the center and keratotic striae in the periphery at times with telangiectasia. Other oral symptoms include ulcers, particularly in young people, hyposalivation in elderly patients, hyperkeratosis, pigmentation, glossodynia, fissured tongue, cheilitis and secondary Sjögren's syndrome [27] [31].

Diagnosis:

SLE is often difficult to diagnose because presentations vary and mimic other diseases. Diagnosis requires a biopsy, with intraoral biopsy typically preferred over skin biopsy when possible to minimize scarring [27].

Management:

SLE management focuses on preventing flares, controlling symptoms, and reducing inflammation. Mild cases may respond to salicylates or non-steroidal anti-inflammatory drugs, while more severe forms require hydroxychloroquine, corticosteroids, or immunosuppressants like azathioprine and cyclophosphamide. Topical corticosteroids and calcineurin inhibitors treat skin lesions, alongside sun protection. Prognosis is generally favorable, but severe renal involvement can be life-threatening [20].

3.8. Graft vs Host Disease (GVHD)

Description:

Graft-versus-host disease (GVHD) is a major complication in patients receiving allogeneic hematopoietic stem cell or bone marrow transplants. Its exact cause is not fully understood, but it is thought to result from donor T-lymphocytes reacting to minor histocompatibility antigens expressed by recipient cells [13].

Clinical manifestations:

GVHD is divided into acute, that is occurring within 100 days after transplantation, or chronic, appearing more than 100 days after transplantation. Acute oral GVHD is painful, characterized by erythema, ulceration, or significant desquamation [7]. Chronic Oral GVHD involves mucosal changes (lichenoid lesions, erythema, ulcers), salivary gland dysfunction, and sclerotic changes in the mouth and surrounding tissues. It can mimic OLP, Sjögren's syndrome, and scleroderma, with symptoms like dry mouth, limited mouth opening, and fibrosis [27].

Diagnosis:

The diagnosis of OLL-GVHD is based on clinical evaluation, patient history, and histopathological tests.

Biopsy is recommended in two situations: (1) when other organ systems show no signs of involvement or tests return negative or nonspecific results, and (2) in cases of atypical clinical presentation to rule out dysplasia or malignancy,

particularly in patients with long-standing chronic GVHD [13].

Management:

In GVHD patients, oral signs like lichenoid lesions, dryness, and mucosal sclerosis may be seen. Dentists must ensure optimal oral hygiene, recommend salivary substitutes, and prescribe topical steroids if needed. Coordination with the transplant team is crucial, as is regular monitoring for dysplasia or malignancy [13].

4. Conclusion

Non-infectious processes of the oral mucosa encompass a diverse array of disorders, ranging from precancerous lesions like leukoplakia and erythroplakia to complex autoimmune and systemic conditions such as pemphigus vulgaris, Behçet's disease, and graft-versus-host disease. While some are primarily localized, others reflect or herald multisystem involvement. Due to their often-overlapping clinical presentations, a careful diagnostic approach—integrating clinical examination, patient history, histopathological evaluation, and when needed, immunological testing—is critical. Early recognition and differentiation from infectious conditions not only prevent mismanagement but also play a pivotal role in improving patient outcomes, especially in potentially malignant or life-threatening disorders.

Conflicts of Interest

The authors declare no conflicts of interest.

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