Episode 62 – Oral Manifestations of Autoimmune Disorders & Drug-induced Oral Conditions

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Oral manifestations of autoimmune disorders[1]

Autoimmune disorders occur when the immune system mistakenly targets healthy cells as pathogens or faulty cells (i.e., the immune system cannot distinguish self from nonself and produces antibodies to an endogenous antigen or autoantigen). There are more than 80 types of autoimmune disorders. [1] [2] [3]

Symptoms of autoimmune disorders depend on the type of disorder and the part of the body affected. Common signs and symptoms include fatigue, fever, myalgia, arthralgia, skin problems, abdominal pain, digestive problems, and lymphadenopathy. Symptoms may come and go and can be mild to severe.

An autoimmune disorder may affect more than one organ or tissue type. Areas often affected by autoimmune disorders include connective tissues, blood vessels, joints, muscles, endocrine and exocrine glands, skin, and mucous membranes.

An individual may have more than one autoimmune disorder at the same time. Examples of autoimmune disorders include celiac disease,[2] Sjögren's syndrome, systemic lupus erythematosus, systemic sclerosis, pemphigus vulgaris, mucous membrane pemphigoid, and lichen planus. The exact cause of autoimmune disorders is not known. Many autoimmune disorders are more common among females. [1] [2]

Oral manifestations are frequently the first sign of autoimmune conditions. The disorders themselves, as well as their pharmacologic management, can adversely affect the oral cavity and significantly impact quality of life. Oral manifestations vary depending on the type of disorder and may include oral ulcerations, plaque-like lesions, changes in salivary flow, and desquamative gingivitis.

Oral healthcare professionals play an important role in the early detection of emerging autoimmune disorders. Timely referral for diagnosis and treatment is essential to decrease morbidity, disability, and mortality caused by autoimmune disorders. [4]

[1] Refer to Episode 45 for additional information on autoimmune disorders.
[2] Refer to Episode 60 for oral manifestations of celiac disease.

Autoimmune disorders

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
Sjögren's syndrome	Relatively common chronic, systemic, inflammatory disorder affecting various exocrine glands or other organs.	Etiology is unknown. More common among middle-aged females. Some individuals also have concurrent autoimmune disorders (e.g., rheumatoid arthritis, systemic lupus erythematosus, Hashimoto thyroiditis).	Treatment is usually symptomatic.	Dryness of mouth, eyes, & other mucous membranes due to lymphocytic infiltration of exocrine glands & secondary gland dysfunction.	• Xerostomia is a significant oral symptom. • Saliva can be thick, ropey, & mucinous, or absent (i.e., lack of saliva pooling in the floor of the mouth). Decreased saliva can lead to odynophagia (painful swallowing), dysphagia (difficulty swallowing), dysgeusia (taste disturbances), difficulty speaking & an increase risk of salivary gland stones, dental caries, & periodontal disease. • Oral mucosal changes typical of xerostomia include dry, red, wrinkled mucosa & smooth red cobblestone appearance of the tongue, due to papillae atrophy. Tongue fissuring & angular cheilitis are common. • Oral candidiasis[3] is common & may cause burning sensation. • Parotid glands may enlarge & are usually firm, smooth, mildly tender. Enlargement can be asymmetric; however, highly disproportionate, persistent enlargement of one gland may indicate a tumour requiring evaluation. Chronic salivary gland enlargement is rarely painful unless obstructed or infected. [4] [5] [6]
Systemic lupus erythematosus (SLE)	Chronic inflammatory connective tissue disorder that can	Exact cause is not known, multiple genetic predispositions &	Depends on signs & symptoms, may include antimalarials (e.g.,	Depends on which body systems are affected. Signs & symptoms may	• Malar rash (butterfly-shaped rash across nose & cheeks). • Photosensitive rashes typically occur on the face or other sun exposed areas.

[3] Refer to Episode 61 for additional information on candidiasis.

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
	involve joints, heart, lungs, brain, kidneys, skin, mucous membranes, & blood vessel walls. Difficult to diagnose because presentation mimics many other conditions & varies greatly between individuals.	gene-environment interactions have been identified. Mainly occurs in females (usually of child-bearing age) but can affect any age, including neonates.	hydroxychloroquine), nonsteroidal anti-inflammatory drugs [NSAIDs], corticosteroids, & immunosuppressants.	develop suddenly or slowly; be mild or severe; temporary or permanent. Discoid lupus erythematosus (DLE) (chronic cutaneous lupus erythematosus) is a form of lupus limited to skin & mucous membranes. Skin lesions begin as erythematous plaques[4] progressing to atrophic scars,	• Lichenoid lesions are common, although lesions with a granulomatous appearance have been reported. Lesions may be located on palate, gingiva, or buccal mucosa. • Cheilitis involving the lower lip vermilion (i.e., lupus cheilitis). • Xerostomia increases risk for candidiasis, periodontal disease, caries, etc. • Use of corticosteroids to treat SLE can lead to oral candidiasis & other oral infections due to immunosuppression. • Use of antimalarial medication (e.g., chloroquine [Aralen], hydroxychloroquine [Plaquenil], or quinacrine) to treat SLE may induce pigmentation of lips & oral mucosa. • Lesions affect mucous membranes, especially in oral cavity including the gingiva.[5] • Sometimes lesions are hypertrophic & may mimic oral lichen planus. • Oral lesions appear as well-demarcated ulcerations or erythematous plaques with atrophy encircled by white, radiating striae. Sunburst pattern is usually unilateral as compared to reticulated red & white plaques of oral lichen planus. [4] [7] [8] [9] [10] [11]

[4] Refer to Episode 61 for glossary of terms.
[5] If DLE presents with gingival manifestations, the gingival condition is classified as a non-plaque-induced gingival disease of autoimmune origin. [9]

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
				with hair loss in affected areas. Rash clusters on light-exposed areas of skin (e.g., face, scalp, ears).	
Systemic sclerosis (scleroderma)	Rare autoimmune connective tissue disorder characterized by degenerative changes & scarring in skin, joints, internal organs (especially esophagus, lower gastrointestinal tract, lungs, heart, kidneys) & by blood vessel abnormalities. Systemic sclerosis causes an overproduction of collagen & other proteins in various tissues.	Etiology is not known. More common among females, more frequent among individuals aged 20-50 years, rare among children.	No treatment to cure or prevent overproduction of collagen. Emphasis on controlling symptoms & preventing complications, & may include corticosteroids, immunosuppressants, calcium channel blockers (for Raynaud phenomenon), proton pump inhibitors (for acid reflux).	Dependent on parts of the body affected. Common symptoms include Raynaud phenomenon;[6] polyarthralgia (pain in multiple joints); dysphagia; acid reflux; painful skin tightening & contractures of fingers.	• Microstomia (limited mouth opening) & inability to close lips due to perioral collagen deposition. Makes oral selfcare challenging; aggravated by reduced manual dexterity. • Xerostomia due to progressive fibrosis of salivary glands, which often mirrors progression of microstomia. • Intraoral & perioral telangiectasias.[7] • Oral mucosa atrophy & blanching. • Fibrosis at the hard & soft palate. • Dysphagia. • Tongue rigidity due to fibrosis, making speech & swallowing difficult. • Increased risk of periodontal disease & caries. • Loss of attached gingiva & gingival recession. • Perimolysis (dental erosion due to acid reflux). • Mandibular bone resorption, possibly linked to facial skin tightening & taut musculature compressing mandible.

[6] Raynaud phenomenon is a vascular disorder characterized by intermittent loss of blood to various body parts, especially fingers, toes, nose, and/or ears, typically occurring after exposure to cold and causes tingling sensations, numbness, and/or pain.
[7] Telangiectasias are small, widened blood vessels on the skin.

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
					• Trigeminal neuralgia possibly due to excess collagen accumulation in the perineurium[8] & reduced vascularity to the nerve itself. • Fibrosis & shortening of lingual frenum may affect speech, saliva control, & bolus manipulation. • Widening of periodontal ligament space may be seen radiographically, especially around posterior teeth. • Temporomandibular (TMJ) problems due to tissue constriction (e.g., TMJ pseudoankylosis characterized by limited mouth opening & mandibular movements). • Use of calcium channel blockers to treat Reynaud phenomenon may result in gingival hyperplasia. • Use of corticosteroids & immunosuppressants to treat systemic sclerosis can lead to oral candidiasis & other oral infections. [4] [12] [13] [14] [15] [16] [17] [18] [19]
Pemphigus vulgaris (PV)[9]	Rare autoimmune disorder characterized by painful intraepidermal blisters & extensive erosions on skin &	Exact cause is not known. Circulating IgG[10] autoantibodies are directed against keratinocyte cell surfaces, which causes loss of cell	Life-threatening condition without proper treatment. Treatment aimed at reducing symptoms & preventing complications such as	Primary lesion of PV is a flaccid bulla (elevated, fluid-filled blister ≥10 mm in diameter) that arises on healthy skin or on an erythematous	• Oral blisters are often the first sign, followed by skin blisters that may come & go. Blisters may occur anywhere on oral mucosa (buccal mucosa most common site followed by palatal, lingual, & labial mucosa).

[8] Perineurium is a protective sheath of connective tissue surrounding a bundle of nerve fibres within a nerve.
[9] Refer to Episode 30 for additional information on pemphigus and use of low-level laser therapy for management of PV lesions.
[10] Refer to Episode 44 for discussion on immunoglobulins and Episode 45 for additional information on autoantibodies.

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
	mucous membranes.	adhesion (acantholysis) resulting in formation of blisters that easily rupture. Usually occurs in middle-aged or older individuals, affects males & females equally, rarely occurs in children.	serious infections of ruptured blisters as well as loss of body fluids & protein. Treatment may include corticosteroids, immunosuppressive drugs, & immunotherapy.	base. Blisters are fragile & may rupture, producing painful erosions (most common skin presentation) that can limit an individual's daily activities.	• Gingiva least commonly affected site. When gingiva is involved, it may present as desquamative gingivitis (i.e., erythema, desquamation, erosion, & blistering of attached & marginal gingiva) &/or as vesiculobullous lesions of the free & attached gingiva. Desquamative gingivitis is the most common manifestation of the disorder.[11] • Intact oral bullae are rare due to lesion fragility. Bullae easily rupture leading to painful erosions, which are ill-defined, irregularly shaped, & slow to heal making eating difficult. May lead to dehydration, malnutrition, & weight loss. • Other mucosal surfaces involved may include esophagus, which can further cause odynophagia &/or dysphagia; & larynx, resulting in hoarseness. • Pharmacologic therapy may cause adverse oral effects as previously described. [20] [21] [22] [23] [24] [25]
Mucous membrane pemphigoid (MMP)	Group of rare chronic autoimmune blistering disorders characterized by subepithelial lesions primarily affecting mucous	Exact cause is not known. Circulating IgG & IgA autoantibodies are directed against antigens of the basement membrane, resulting	Goal of treatment is to suppress extensive blister formation, promote healing, & prevent scarring. Treatment depends on disease severity & sites involved.	Typically presents with persistent, painful erosions on mucous membranes. Clinical signs are dependent on sites involved. For example:	• >90% of individuals have oral mucosal lesions consisting of recurrent, painful erosions. • Gingiva most commonly affected, followed by palate & buccal mucosa; however, any oral mucosal site may blister (e.g., lips, pharynx, tongue, floor of mouth). Lesions manifest as erythema, erosions, & pseudomembrane. Intact

[11] If pemphigus presents with gingival manifestations, the gingival condition is classified as a non-plaque-induced gingival disease of autoimmune origin. [9]

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
	membranes, with mouth & eyes most often affected, but other mucosal sites (e.g., nasal, pharynx, larynx, esophagus, anogenital area) & skin (usually head & upper trunk) may be involved.	in detachment of epithelium from connective tissue, causing tense blisters (e.g., bullae) that do not rupture easily. Older individuals are most often affected. Slightly higher prevalence among females.	Topical corticosteroids may be used for mild or moderate oral MMP. Intralesional corticosteroids may be used to treat recalcitrant lesions or as an adjunct to topical steroids. Topical corticosteroids plus systemic therapy (e.g., corticosteroids, immunosuppressants), may be used to treat moderate-to-severe oral MMP.	Ocular involvement may present with pain, eye grittiness, conjunctivitis, & scarring may lead to blindness. Nasal involvement may present as epistaxis (nose bleeds), bleeding after nose blowing, nasal crusting, & discomfort. Skin lesions may manifest as tense vesicles or bullae that may be hemorrhagic. Lesions occurring at any site may heal with scarring.	blisters rarely seen & may appear flaccid or tense. • Mucosal bleeding. • Oral erosions often begin on gingiva,[12] particularly marginal gingiva, described as desquamative gingivitis, which is the most common manifestation. • Spontaneous gingival bleeding. • Involvement of the oropharynx may cause hoarseness or dysphagia. • Pharyngeal scarring may lead to esophageal stenosis requiring dilatation procedures. • Supraglottic involvement may lead to airway compromise requiring tracheostomy. • Severe symptoms may prevent proper dietary intake resulting in severe malnutrition. • Pain may prevent good oral selfcare increasing risk of plaque accumulation, periodontal disease, & caries. • Pharmacologic therapy may cause adverse oral effects as previously described. [9] [22] [25] [26] [27] [28] [29] [30] [31]
Lichen planus (LP)	Rare, chronic, inflammatory autoimmune skin & mucous membrane disease.	Etiology is not known. It is thought to be caused by a T cell-mediated autoimmune	In mild cases, symptoms may be minimal or absent, requiring no therapy.	Skin lesions present as pruritic, violaceous (purple) flat-topped papules & plaques. Lesions are	• Oral mucosa is involved in ~50% of cases. • Lesions, usually bilateral, often involve the gingiva[13] & present as desquamative gingivitis causing pain & discomfort during eating & oral selfcare.

[12] If MMP presents with gingival manifestations, the gingival condition is classified as a non-plaque-induced gingival disease of autoimmune origin. [9]
[13] If OLP presents with gingival manifestations, the gingival is classified as a non-plaque-induced gingival disease of autoimmune origin. [9]

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
	May occur with other autoimmune disorders (e.g., ulcerative colitis, alopecia areata, vitiligo, myasthenia gravis).	response against basal epithelial keratinocytes in individuals with genetic predisposition. Certain medications, metal fillings (causing oral lichen planus [OLP]), stress, injury, or infection (e.g., hepatitis C infection) may trigger an autoimmune reaction. LP mainly occurs in individuals aged 30-60 years, but can occur at any age. Slightly higher prevalence among females; children are rarely affected.	Treatment is aimed at reducing length & severity of symptomatic outbreaks. Treatment usually requires topical or intralesional corticosteroids. Severe cases may require phototherapy or systemic corticosteroids, retinoids, or immunosuppressants.	initially 2-4 mm in diameter & may coalesce into rough scaly plaques, often accompanied by oral &/or genital lesions. Lesions are usually symmetrically distributed, most commonly on the flexor surfaces of wrists, forearms & legs, trunk, genitalia, & oral & vaginal mucosae, but can be widespread. Facial skin usually spared. Onset may be abrupt or gradual.	• Buccal mucosa & tongue also commonly involved. • Periods of remission occur where symptoms & lesions appear & regress at intervals. • OLP can presents clinically in various forms, such as reticular, plaque-like, papular, erosive, atrophic, or bullous types. • Reticular OLP, most common form, presents as a lacy pattern of interconnecting keratotic white lines (Wickham striae); typically located bilaterally on buccal mucosa, mucobuccal fold, gingiva, & less commonly, on tongue, palate & lips; usually asymptomatic • Plaque-like OLP (clinically resembles leukoplakia) ranges in presentation from smooth, flat areas to irregular, elevated areas. Commonly found on dorsum of the tongue & buccal mucosa; usually asymptomatic. Seen with higher frequency among tobacco smokers. • Papular OLP appears as papules on the oral mucosa, especially the buccal mucosa, often occurs concurrently with reticular OLP. • Erosive OLP, (ulcerative) second most common type, presents as mix of erythematous & ulcerated areas surrounded by finely radiating keratotic striae. Lesions migrate over time & tend to be multifocal. Gingival lesions produce desquamative gingivitis. Symptoms of erosive form range from episodic pain to severe

Disorder	Description	Etiology & prevalence	Treatment	General symptoms	Oral manifestations
					discomfort that can interfere with eating & oral selfcare. • Atrophic OLP (erythematous) appears as diffuse, erythematous patches surrounded by fine white striae. Can cause significant discomfort. • Bullous OLP usually presents as bullae on buccal mucosa & lateral borders of the tongue; bullae rupture soon after they appear, resulting in classic appearance of erosive OLP. • Oral lesions may present alone or with skin lesions or other LP mucous lesions. • Excellent oral hygiene is thought to reduce severity of symptoms, but can be difficult to achieve during periods of active disease. • Malignant transformation occurs in ~1% of cases but is more prevalent in atrophic & erosive lesions, emphasizing the need for long-term follow-up of the condition. • Pharmacologic therapy may cause adverse oral effects as previously described. [9] [32] [33] [34] [35] [36] [37] [38] [39] [40] [41] [42] [43]

Drug-induced conditions

Many lesions and disorders of the oral cavity are caused or are associated with pharmacologic therapies. Therefore, it is essential to thoroughly review medication history for clients presenting with oral pathology.

Drug-induced oral conditions

Oral condition	Clinical features	Pharmacologic agent
Aphthous stomatitis (Recurrent aphthous ulcers)	Aphthous ulcers[14] are round, crateriform, white-yellow depressions surrounded by halo of erythema, painful, & self-limiting. Usually form on unattached oral mucous membranes. Outbreaks of multiple ulcerations may occur. Aphthous minor: 1-3 mm, resolve in 7-10 days, occur in ~80-85% of cases. Aphthous major: >1 cm, resolve in 14-21 days, occur in ~15% of cases.	• Nonsteroidal anti-inflammatory drugs (NSAIDs) • ACE inhibitors • Bisphosphonates • Potentially any drug can produce aphthous-like reaction [4] [44]
Xerostomia[15]	Xerostomia has been associated with >500 medications. It can lead to dysgeusia, halitosis, oral dysesthesia (burning feeling in the mouth), cracked dry lips, dry or sore throat, lobulated/deeply fissured tongue with atrophy of filiform papillae, & increased thirst. Increased risk of oral candidiasis, caries, & periodontal disease. Increased prevalence of traumatic ulcerations & increased difficulty in speech, eating, swallowing & retaining dentures due to lack of lubrication, which can result in decreased food intake & poor nutrition.	• Alpha-receptor antagonists • Anticholinergics • Antidepressants • Antiemetics • Antiepileptics • Antihistamines • Antihypertensives • Antimigraine medications • Antiparkinson drugs • Antipsychotics • Antivirals • Anxiolytics • Appetite suppressants • Benzodiazepines • Bronchodilators • Decongestants • Diuretics • Muscarinic agonists • Muscle relaxants • Proton-pump inhibitors • Opioids • Retinoids • Caffeinated & alcohol beverages/products • Cannabis[16] [4] [45] [46] [47]

[14] Refer to Episode 61 for additional information on aphthous ulcers.
[15] Refer to Episode 55 for additional information on xerostomia.
[16] Refer to Episode 58 for additional information on cannabis & oral health

Oral condition	Clinical features	Pharmacologic agent
Lichen planus	>200 drugs have been implicated in lichen planus-like or oral lichenoid drug reactions. Drug-associated oral lichen planus generally presents as reticular form; however, ulcerated & eroded forms have been reported. A thorough medication history may help identify the causative agent. However, average time span between drug intake & an oral lichenoid drug reaction is 2-3 months, & delayed onset after 12 months has occurred. If the reaction becomes very extensive or painful, the causative drug may have to be changed.	• ACE inhibitors • Beta-blockers • NSAIDs • Diuretics (e.g., thiazides) • Hydroxychloroquine • Zidovudine (AZT) • Sulfonylureas (type 2 diabetes) • Penicillamine [4] [32] [36] [48]
Gingival hyperplasia	Gingival enlargement typically begins 1-3 months after starting therapy. Overgrowth may be localized or diffuse, often presenting as nodular enlargement of interdental papillae. Anterior gingiva most often affected. Growth may progress until gingiva covers entire crowns of teeth. Overgrowth makes oral hygiene difficult & increases risk of oral infections. Diligent home care is necessary to reduce hyperplasia severity. Discontinuing the drug or changing to an alternative medication may reduce overgrowth. Spontaneous remission is rare. Conservative surgical excision of excess tissue helps with esthetics & plaque control. However, gingival tissue usually regrows necessitating repeated surgeries while taking the causative drug.	• Anticonvulsants (e.g., phenytoin, phenobarbital) • Calcium channel blockers (e.g., nifedipine, verapamil, amlodipine) • Immunosuppressants (e.g., cyclosporine) [4] [49] [50] [51]
Candidiasis[17]	Antibiotics can destroy beneficial bacteria disturbing the natural balance of oral microflora & allowing overgrowth of *Candida*. Steroids & chemotherapy alter normal immune function, altering the oral equilibrium in favour of candidal overgrowth. May manifest as pseudomembranous form (white plaques overlying erythematous mucosa, especially buccal mucosa & palate); angular cheilitis; chronic atrophic candidiasis (i.e., denture stomatitis, chronic erythema & edema of palate under denture base); or erythematous form (areas of erythema & atrophy, commonly on palate or dorsum of tongue).	• Antibiotics • Corticosteroids (inhaled & oral) • Chemotherapy [4] [52]

[17] Refer to Episode 61 for additional information on candidiasis.

Oral condition	Clinical features	Pharmacologic agent
Pigmentation	Drug-induced pigmentation of oral mucosa may be due to direct melanocytic stimulation, deposition of pigmented drug metabolites, or both. Melanocytic lesions appear due to an increase in melanin production &, less frequently, from an increase in the number of melanocytes. Lesions are usually macular (flat) & can either be localized or diffuse. Lesions may appear immediately after drug administration or after several days or years, & usually disappear when drug administration is stopped. Most pigment-inducing drugs present a dose-related relationship with intensity & extension of pigmentation. Pigmentation appears most frequently on the hard palate, gingiva, & buccal mucosa. Colour can vary from brown, grey, blue, & black. Biopsy may be indicated to confirm diagnosis, since some melanotic lesions can be malignant.	• Antimalarial agents (e.g., chloroquine, hydroxychloroquine, quinacrine, quinidine), diffuse blue, grey, or black areas commonly reported on hard palate & buccal mucosa • Tranquilizers (e.g., chlorpromazine) • Minocycline, diffuse bluish-gray discolouration any site of oral cavity • Amiodarone (antiarrhythmic drug) • Imatinib (oral chemotherapeutic agent) causes well-defined bluish to greyish pigmentation, usually in hard palate • Hydroxyurea (antineoplastic agent), mainly buccal mucosa & tongue • Zidovudine (antiretroviral treatment), brownish macules in buccal mucosa & lips [4] [11] [48] [53] [54] [55]
Burning mouth syndrome	Burning mouth syndrome is a chronic orofacial pain syndrome, without evidence of mucosal lesions, other clinical signs of disease, or laboratory abnormalities. Symptoms include burning mouth pain, xerostomia, taste disturbances. Discomfort usually worse at the end of the day. More common among females, mainly affecting post-menopausal individuals.	• ACE inhibitors • Anticoagulants • Antipsychotics • Antiretrovirals • Benzodiazepines [56]
Medication-related osteonecrosis of the jaw (MRONJ)	MRONJ[18] is a rare condition typically presenting as an area of exposed bone in the maxilla &/or mandible that does not heal after >8 weeks. Usually painful with purulent discharge, but may be asymptomatic. Other signs & symptoms may include loosening of teeth unrelated to periodontal disease; periapical or periodontal fistula not associated with pulpal necrosis due to caries; irritation of soft tissues from sharp exposed bone; pain, swelling, difficulty chewing, bad taste & fetid odour due to secondary infection; paresthesia.	• Bisphosphonates, especially administered intravenously • Receptor activator for nuclear factor κ B (RANK) ligand inhibitors (e.g., denosumab) • Sclerostin inhibitor (e.g., romosozumab) [57] [58] [59]

[18] Refer to Episodes 41 & 42 for additional information on osteoporosis & MRONJ.

Take home messages

- Oral manifestations are often the first sign of autoimmune conditions. Thus, oral healthcare professionals can play a pivotal role in the early detection, referral, and the multidisciplinary care after diagnosis. Early diagnosis can enhance effectiveness of therapeutic interventions, helping to improve health outcomes and quality of life.
- Awareness of possible oral conditions induced by pharmacologic agents is important to help determine etiology and management.

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