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Review Article

Ocular manifestations of oral mucosal lesions

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ABSTRACT

Autoimmune blistering diseases refer to a diverse group of conditions that primarily target the skin and mucosa. However, in some cases, other organ systems can be involved, depending on the specific pathophysiology of the disease. Owing to the increased possibility of blindness associated with delayed diagnosis and treatment, it is essential to be aware of and detect ocular manifestations of these diseases at an early stage. The present article discusses the various oral mucosal lesions encountered in daily practice with emphasis on the ocular manifestations of each lesion.

1. Introduction

Specialized stratified epithelium derived from the ectoderm makes up the oral mucosa. An oral mucosal lesion (OML) is characterized by any unusual change influencing the oral mucosal surface coming about as a result of local pathology or auxiliary to systemic illness, including those of skin.[1] The etiology of OMLs is multifactorial and they frequently cause inconvenience or agony that impedes rumination, gulping, and speech, and additionally can lead to halitosis, xerostomia, or oral dysesthesia which interferes with normal agility and slows down day to day activities.[2]

OMLs are identified by their location, size, color, pattern, symptoms, and distribution. Due to similar symptoms and appearances, oral cavity lesions can be difficult to diagnose and treat. Typically, histopathological results and occasionally immunohistochemistry are used to confirm a diagnosis.[1] Although these lesions most frequently affect the oral cavity, they can also have peculiar ocular manifestations that often go unnoticed. It is important for any practitioner to be aware of these manifestations as they can offer important clues pertaining to the diagnosis and subsequent treatment planning of the condition.

2. Pemphigus Vulgaris

Loose sores accompanied by erosions seen on otherwise unaffected skin/mucosa represent hallmarks of an autoimmune condition, Pemphigus vulgaris (PV).[3] Oral and mucosal lesions may present with the first sign in as many as 80% to 90% of cases. The condition usually affects the oral mucosa, however, the larynx, esophagus, anus, cervix, and vulva have also shown to be affected. Rather than tense rankles evident in cases of bullous pemphigoid,

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patients with PV are seen to have flaccid rankles on an erythematous base.[4] The rankles may give the appearance of Nikolsky's sign (depicting the division of the epithelium with digressive strain on surface of the skin), offering a reasonably delicate however exceptionally unambiguous mark aiding in PV diagnosis.[5]

2.1. Ocular manifestations

Bean et al in 1975 first reported a case of ocular involvement in PV.[6] Visual association is usually restricted to certain areas namely the eyelids and conjunctiva without influencing visual acuity. The most frequently reported side effects include soreness, photophobia, agony, and irritation.[7] Bilateral conjunctivitis is an incessant kind of visual participation (Figure 1). Additional serious visual contribution showing up as palpebral conjunctival disintegration is additionally usually noticed and is more predominant. Bulbar conjunctivae disintegration is seldom noticed.[3] Medial half of the lower eyelid might represent with lid margin erosions, which may be pathognomonic of this condition.[8] A study by Akhyani M et al (2014) revealed that visual contribution was available in 16.5% PV patients with conjunctivitis being the most pervasive kind of visual inclusion (52.9%), trailed by disintegration of the palpebral conjunctiva (41.2%) and disintegration of the bulbar conjunctiva.[3]

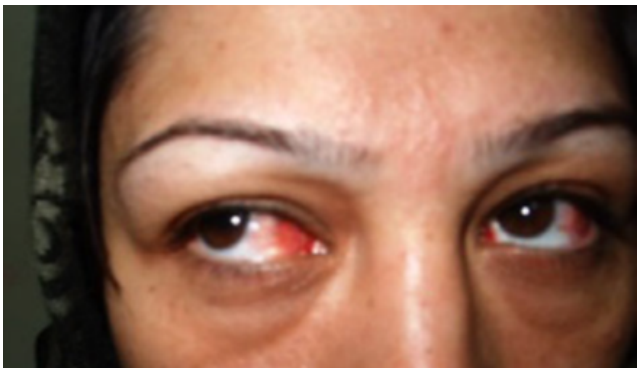

Figure 1: Bilateral mild conjunctivitis seen in PV[3]

Conjunctival involvement in PV has been shown to be effectively treated with a localized intralesional triamcinolone injection as well as systemic treatment.[9] Topical steroids like prednisolone acetate are typically employed for treating acute inflammation, on the other hand, cyclosporin suspension adjunct is required for chronic inflammation.[10]

3. Mucous Membrane Pemphigoid

Mucous membrane pemphigoid (MMP) refers to a persistent vesiculobullous lesion affecting the eyes principally by means of conjunctival scarring. Practically, every patient fosters oral association and individuals ranging from 61% to 80% report visual connection.[11] This disorder has been seen to be bound to the eye in roughly 20% of affected individuals.[12] Apart from the same, additionally affected mucous layers incorporate pharynx, nasal mucosa, larynx, genital mucosa, rectum, and esophagus.[13]

3.1. Ocular manifestations

MMP with visual contribution called as ocular cicatricial pemphigoid, refers to a sight-compromising sickness portraying as chronic conjunctivitis.[12] The condition seldom affects one-side and can happen in seclusion or occur alongside other mucous membranes.[14] Patients every now and again report with photophobia, burning, mucous release or unfamiliar sensations, while clinicians can see erythema or signs of scarring.[15] Lesion progression is consistently depicted by cicatrising conjunctivitis with subepithelial fibrosis eventuating in symblepharon advancement and fornix foreshortening, typically impacting the inferior fornix initially.[16] Average canthal scarring along with plica and caruncle loss has in like manner been advanced as an early indication.[17] Reports of the scarring framework annihilating goblet cells, lacrimal organ ductules, and meibomian gland orifices inciting dry eye ailment have similarly been recorded.[18] Advanced ailment contains lagophthalmos, trichiasis, ectropions, entropions, and corneal ulceration.[19] In case of not being dissected or treated early, development to extreme OSD leading to vision loss has been represented to occur in 33% of affected individuals.[14] To examine this condition in its starting stages, ophthalmologists should search for unpretentious inferior forniceal pressure and symblephara, which can be perceived when the lower eyelid is pulled down while the patient gazes upward (Figure 2). Ocular cicatricial MMP changes can be staged using a variety of scoring systems. Organizing system given by Foster has been used comprehensively as a disease development marker in visual MMP (Table 1).[20] In 1992, Tauber et al. cited these systems' individual insensitivity and suggested a combined format (Table 2).[21]

A multidisciplinary approach is much of the time essential in the administration of MMP. Triamcinolone acetonide and other effective corticosteroids can be utilized to treat some intense and visual injuries in the early phase.[22] Topical retinoids may help treat posterior lid margin conjunctival keratinization. The time when moderate conjunctival cicatrization is noticed, subconjunctival mitomycin C or steroid imbuement may be effective.[23] Visual surface reproduction, incorporating cautious correction of eyelid contortions or eyelash expulsion for trichiasis may be expected to achieve visual surface tranquility. Mechanical epilation, cryotherapy, bipolar electrolysis, radiofrequency removal, or laser

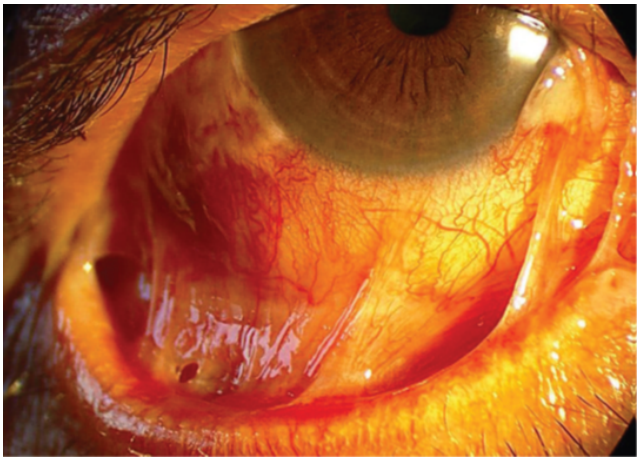

Figure 2: Inferior fornix shortening and bulbar and palpebral conjunctival fusion in mucous membrane pemphigoid

removal can be generally used to eliminate atypical lashes, which is important for forestalling corneal ulcers or disintegrations. Punctal hindrance can be helpful in overseeing dry eye associated with MMP.[4]

Table 1: Foster staging system

Stage	Characteristics
I	Subconjunctival scarring and fibrosis
II	Fornix foreshortening of any degree
III	Presence of symblepharon, any degree
IV	Ankyloblepharon, frozen globe

Table 2: Tauber staging system

Stage	Characteristics
I	Subconjunctival scarring and fibrosis
II	a. 0–25% loss of inferior fornix depth b. 25–50% loss of inferior fornix depth c. 50–75% loss of inferior fornix depth d. 75–100% loss of inferior fornix depth
III	a. 0–25% horizontal involvement of symblephara b. 25–50% horizontal involvement of symblephara c. 50–75% horizontal involvement of symblephara d. 75–100% horizontal involvement of symblephara n. Number of symblephara countable
IV	Ankyloblepharon, frozen globe

4. Paraneoplastic Pemphigus

Neoplasms either benign or malignant, are associated with paraneoplastic pemphigus (PNP). Non-Hodgkin lymphoma, chronic lymphocytic leukemia, Castleman's disease, and thymomas are the most frequently associated neoplasms.[19] Affected individuals report with erosive and rankling skin and mucosal sores. Mucous layer association happens in nearly every individual suffering from this condition. Mucosal involvement is seen in many body parts namely the lips, gingival, buccal, and lingual mucosa, nasopharynx, oropharynx, nasal septum, hypopharynx, larynx, tracheobronchial mucosa, esophagus, and epiglottis. Cutaneous lesions typically appear several days to months after mucous membrane lesions.[24]

4.1. Ocular manifestations

Ocular symptoms of PNP comprise of red discoloration, discomfort, irritation, mucus discharge and vision deterioration. Patients could report a reciprocal cicatrizing conjunctivitis alongside fornices reduction and symblepharon development. Apart from these, thickening of the eyelid edge, conjunctival deterioration, pseudomembranous conjunctivitis, and corneal epithelial deformations may also be seen. Decrease in baseline tear secretion may be seen in Schirmer tear production test.[25] In severe cases, two-sided corneal melting may also occur.[26]

When seeing cases of PNP, careful and prompt treatment can assist with forestalling vision misfortune and visual deficiency by monitoring for ocular surface complications. Notwithstanding fundamental immunosuppressive treatment and treatment of the basic harm, visual treatment might incorporate the utilization of surface corticosteroid drops, 10% N-acetylcysteine, sodium hyaluronate as well as successive additive-free artificial tears. Amniotic membrane grafting as a surgical treatment might be significant while examining instances with wide conjunctival scarring & fornice deficiency.[27]

5. Lichen Planus

Lichen planus (LP) refers to immune system disorder predominantly influencing the skin and mucous membranes.[28] LP comprises of different cutaneous, mucosal, & subungual appearances unaccompanied or related to each other. The assessed recurrence of mucosal affiliation ranges between 30-70%.[29] Oral mucosae is the most notable region of contribution seen in mucosal LP. This is followed by genitalia, esophageal and nasopharyngeal association.[30] Ear as well as the visual surface are various locales of association.[28] The course of the illness is an extreme disturbance of the skin or mucosal tissues giving rise to injuries and crumbling. The infection forms into a persistent harmless course set apart by fibrous bands and scarring after a self-restricting irritation.[30]

5.1. Ocular manifestations

In 1904, Gaucher and Druelle described the first case of ocular surface involvement in the form of white conjunctival streaks.[28] LP usually occurs in visual structures, yet just once in a blue moon, the visual contribution is for the most part joined with cutaneous or oral signs.[31] The disease can cause cicatricial conjunctivitis or eyelid and

corneal involvement. Conjunctival inclusion can prompt blepharitis, keratitis, symblepharon, corneal ulceration, and lingering visual debilitation.[32] LP can also cause epiphora and pathologies in the lacrimal canal.[33]

First-line medications incorporate topical corticosteroids and cyclosporin. Those patients not unresponsive to topical medicines might benefit with systemic treatment with corticosteroids and other immunosuppressive agents, for example cyclosporin, azathioprine, or mycophenolate mofetil.[34] Effective treatment of patients with detached conjunctival lichen planus utilizing skin cyclosporin A 2% in olive oil has likewise been reported.[35]

6. Linear Immunoglobulin A Disease

An uncommon condition known as linear immunoglobulin A disease(LAD) is described by the presence of circulating immune system antibodies that focus on the epithelial basement membrane zone.[4] Patients who are impacted normally present with a delayed prodrome of cutaneous burning or pruritus before lesion development. Commonly, the erythematous skin area is encircled by annular tense bullae that look like a "string of pearls" or "cluster of jewels". Wounds routinely incorporate the perioral region, perineum, and extremities. Moreover, mucous film affiliation is found in adults, with oral, genital, and conjunctival bruises that can bring about scarring.[36]

6.1. Ocular manifestations

Dry eye, the feeling of an unfamiliar body in the eye, conjunctival scarring with trichiasis, entropion, corneal opacification, neovascularization, and the possibility for visual impairment are the ophthalmologic signs.[37] This pathology can be hard to distinguish from other scarring infections like visual cicatricial pemphigoid. To confirm the diagnosis, a skin biopsy ought to show stores of IgA in an immediate dispersal over the intersection among dermis and epidermis. All LAD patients ought to have their eyes examined because ocular involvement is common and may not cause symptoms (about 50% of LAD patients).[38]

A short oral steroid course might be valuable in determined instances of medication prompted LAD. Drug-initiated cases ordinarily resolve rapidly after the culpable prescription is recognized and stopped. The mucosal and cutaneous lesions can be treated locally, but bullae don't usually require anything more than clean dressings, and infected lesions can be treated with topical antibacterials like mupirocin. The usage of dapsone or sulfapyridine is productive in a large portion of cases, bringing about observable reaction to treatment soon. Prednisone, dicloxacillin, sulfamethoxypyridazine, and colchicine are examples of adjunct therapies that may be beneficial for disease that has been treated for more than a few weeks.[4]

7. Epidermolysis Bullosa Acquisita

The most exceptional of the vesiculobullous immune system conditions is Epidermolysis bullosa acquisita (EBA). Around 9.6% of EBA cases have been related with different circumstances like rheumatoid joint inflammation, Crohn's ailment, ulcerative colitis, psoriasis, and thyroiditis. Rankles and bullae on the skin and mucous films are the signs of EBA. Visual, genital, esophageal, tracheal, and anal mucosae are the following most commonly impacted mucosal locales. Also oral sores are alluded to as inescapable, pain causing rankles, disintegrations, and scarring that can happen on any surface of the oral mucosa.[39]

7.1. Ocular manifestations

Corneal disintegrations or rankles, corneal scarring, symblepharon, blepharitis, ectropion, lacrimal duct obstruction, impeded vision, and visual impairment are among the visual intricacies of EBA that have been accounted for.[40] The frequency with which these complications occur varies between the various subtypes. The most generally perceived sort of epidermolysis bullosa related with visual ensnarements is the dystrophic type, which every now and again incorporates the conjunctiva, achieving scarring and symblepharon development.[41] Visual and tracheal contribution might prompt visual impairment and hazardous respiratory complications due to scarring.[39] The Epidermolysis Bullosa Sickness Action and Scarring Record (EBDASI), made in 2014, was the primary crucial gadget to consolidate a subsection scoring visual consideration with partition between illness 'action' and 'harm' (Table 3).[42]

Table 3: Ocular scoring in the Epidermolysis Bullosa Disease Activity and Scarring Index

Activity		**Damage**	
Erosions/ blisters/ erythema/ mucosal atrophy/ fissures/ stenosis	Score	Lesions	Score (0 = absent) (2 = present)
Absent	0	Ectropion	
1 lesion	1	Symblepharon	
2–3 lesions	2	Visible corneal opacity	
> 3 lesions or 2 lesions > 2 cm	5		
Entire area	10		
Total activity score	/10	Total damage score	/6

Systemic corticosteroids are used as the principal line of treatment for this condition. Amniotic layers can be utilized to heal harmed mucosal surfaces and remake corneal and conjunctival surfaces that are damaged in different conditions that disturb the visual surface.[43]

This tissue works with epithelialization and diminishes aggravation, vascularization, and scarring.[44] Symblepharon lysis and lamellar keratectomy are the suggested remedies for individuals with symblepharon and corneal obscurity. Rituximab and high-portion intravenous Immunoglobulin have demonstrated to be compelling in EBA therapy, especially in obstinate cases.[43]

8. Erythema Multiforme

Erythema multiforme (EM) is a class IV cytotoxic reaction generally focusing on keratinocytes, demonstrating commendable designated sores.[45] EM comprises of two varieties, erythema multiforme major (EMM) and erythema multiforme minor (EMm). EMM and EMm are both depicted by cutaneous wounds impacting <10% of the body surface. The wounds present with rankled or necrotic focuses, included by an erythematous ring. They can be intense and generally resolve with scarring. Oral mucosa (70%) is the most notable mucosal site included. Be that as it may, the eyes, genitalia, upper respiratory area, and pharyngeal mucosa could likewise be impacted.[46]

8.1. Ocular manifestations

Visual contribution in EM minor is ordinarily gentle and may appear as red conjunctivae, chemosis, and lacrimation (Figure 3).[47] As per study conducted by Chang YS, the underlying visual appearances of acute EM incorporate eyelid edema, erythema, crusting, and conjunctival hyperemia. These disclosures were tracked down in more than 90% patients with visual commitment. Residual 8.5 percent had more serious conjunctival aggravation (chemosis, development of the conjunctival layer, and pseudomembrane), and 18.8 percent had corneal contribution.[48]

Figure 3: Eye conjunctivitis seen in EM

Ointments hydrate the visual surface and flush away inflammatory substances. Effective anti-infection agents have displayed to diminish the risk of visual disease. Surgery is highlighted towards adjusting the essential imperfections or distortions. Lysis of symblepharon and conjunctival recession may be embraced to remake the conjunctival fornix. There are a number of ways to correct cicatricial entropion and ectropion, including using a skin or mucosal graft.[48]

9. Leukoplakia

Leukoplakia is a condition referring to a white patch/plaque that for the most part is seen on mucus membranes of the mouth.[49] The specific reason for leukoplakia is obscure, however smoking, drinking a lot of liquor, irritating food substances, and persevering friction — for example, because of improperly fitting dentures or spiked teeth — might be connected to it. The oral mucus membrane, vocal strings, digestive tract, or mucosal area of the urinary tract and private parts can be in every way impacted by plaque.[50]

9.1. Ocular manifestations

Leukoplakia can likewise happen on corneal surface, at times looking like a vernal keratoconjunctivitis-like calcium deposit or else mucous patch. This could be a direct result of entropion, which leads to corneal disturbance. Limbus region can also be affected by this condition and can extend to the cornea as well as bulbar conjunctiva (figure 4).[49] Leukoplakia of the cornea can likewise be seen as purported unique finger impression epithelial lines.[51]

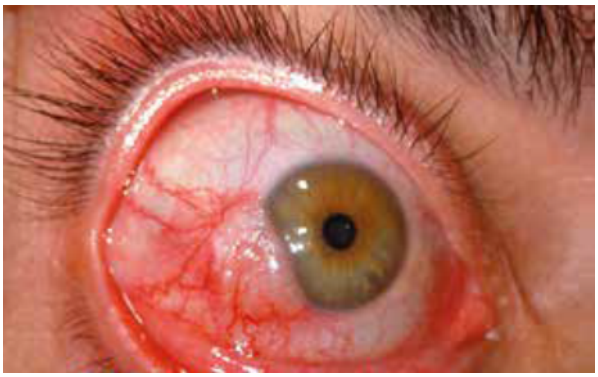

Figure 4: Leukoplakia on the bulbar conjunctiva

10. Candidiasis

Candida spp. are the microorganisms that cause the most invasive fungal infections.[52] Perhaps of the most widely recognized fungal infections that influence the oral mucosa is oral candidiasis. The yeast Candida albicans, which is one of the components of normal oral microflora, is responsible for these lesions.[53] Similar to the oral cavity, C. albicans colonizes the gastrointestinal and reproductive tracts asymptomatically of otherwise healthy individuals.[54] Furthermore, candida sp. represent one of the well-known

reasons for diseases in the circulatory system and have the potential to spread to any body part and can affect the heart, lungs, brain, kidneys, eyes, liver, and spleen.[55]

10.1. Ocular manifestations

Ocular candidiasis is a critical complication of candidemia. Endophthalmitis and chorioretinitis, both of which result in significant visual loss, can be brought on by Candidaemia. Candida endophthalmitis is brought about by hematogenous seeding of the choroid & retina by means of little vessels. Organic entities subsequently, at that point, multiply, causing both central aggravation and restricted abscess development. Resulting about spread to the vitreous body could lead to endophthalmitis and retinal separation. Subacute beginning of obscured vision accompanied along with low-rate torment, photophobia, and injection are typical side effects. Endophthalmitis is set apart by "cushion balls," or vitreous abscesses. Hypopyon, scleritis, alongside association of the third cranial nerve may also be noticed in endophthalmitis.[56]

Visual candidiasis can be dealt with the systemic and topical antifungal treatment, or with intravitreal imbuement of an antifungal agent, now and again alongside vitrectomy. Fluconazole and voriconazole, on the other hand, reach vitreous concentrations that range from 25% to 100% of their serum concentrations, while systemic amphotericin B and echinocandins don't infiltrate all the way into the vitreous humor.[57]

11. Conclusion

Ocular involvement in OMLs is well documented in the literature. Management of ocular complications might often require a collaborative effort among different specialists. Hence, a thorough knowledge regarding the ocular manifestations of oral mucosal lesions is a prerequisite for every specialist. The early diagnosis and treatment using established immunoregulatory agents, as well as the development of new ones, should be prioritized, as it aids in prevention of these diseases' morbidities.

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None.

13. Conflict of Interest

None.

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