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The Oral Mucosa's Lichenoid Tissue Reactions

Svetelina Velikova*

Department of Clinical Laboratory and Clinical Immunology, Medical University of Sofia, Bulgaria

Abstract

Lichenoid alterations in the oral mucosa can occur in a variety of lesions and have a variety of etiologies. Clinical and histologic similarities can occur in immune-mediated illnesses such as lichen planus, mucous membrane pemphigoid, discoid lupus erythematosus, and graft-versus-host disease. Lichenoid reactions to dental materials like amalgam, as well as several systemic medicines, are extensively documented. Oral dysplasia can also have a lichenoid histology, which might disguise the potentially malignant component. Proliferative verrucous leukoplakia is a unique clinical condition that clinically mimics oral lichen planus and necessitates careful correlation of clinical and pathologic features. The purpose of this study was to identify the tissue-derived extracellular vesicles immunogen implicated in the pathogenesis of oral lichen planus (OLP) and its variants oral lichen planus (OLP).

Lichen Planus is an autoimmune disease of the skin and mucous membranes, including the oral mucosa and sexual organs, with the potential to progress to cancer. Xerostomia and salivary gland hypofunction are common signs and complications of oral lichen planus (OLP), the origin of which is unknown. Aquaporins (AQP) are a type of membrane channel cell protein that is involved in intercellular water permeability. This study examines the expression of the aquaporin-3 (AQP3) gene in oral tissues of OLP patients and a control group for the first time. In this study, 30 OLP patients and 30 healthy people were chosen. The AQP3 gene expression was evaluated using the Real-Time PCR technique.

Keywords: Lichen planus; Lichenoid responses; Graft versus host disease (GVHD); Dysplasia of the mouth; Leukoplakia proliferative verrucous; Hypersensitivity; Metals; Contact lichenoid lesions of the mouth; Lichen Planus of the mouth; Patch test on the skin; Eruption of a chenoid medication

Introduction

At the 2006 World Workshop of Oral Medicine IV, oral lichenoid contact lesions (OLCLs) were defined as a sub-category of oral lichenoid lesions (OLLs). Contact hypersensitivity to dental materials may result in OLCLs. Allergic contact stomatitis (ACS) is an immunoinflammatory condition caused by a delayed hypersensitivity immunological response to allergens in direct contact with the oral mucosa mediated by an antigen specific T-cell. ACS-related OLCLs have been linked to dental amalgam. In contrast to Oral lichen planus (OLP), which has a clear anatomical link to the site of metallic restoration and/or prosthesis, the clinical presentation of OLCLs is frequently unilateral and asymmetrical. Because there are no confirmed histological diagnostic criteria, distinguishing between OLCLs and OLP remains difficult. However, the absence of basal cell liquefaction, the presence of an inflammatory infiltrate ranging from deep to superficial infiltrate in some or all areas (as opposed to a band-like distribution), a focal perivascular infiltrate, a high plasma cell count, and neutrophil infiltration into connective tissue may help distinguish OLCLs from OLP [1-2].

There have previously been reports of chenoid drug outbreaks. We present two examples of bilateral oral lichenoid lesions on the buccal mucosa that developed following rheumatoid arthritis treatment with low-dose methotrexate. The first case included a 71-year-old lady who was taking methotrexate for rheumatoid arthritis and developed bilateral mouth ulcers on the buccal mucosa. On examination, erythematous lesions with white plaques and striae were discovered. Clinically, oral lichen planus was suspected, although topical steroids were ineffective. Contact dermatitis of the oral mucous membrane (or contact stomatitis) is uncommon. In general, the mucosa is more resistant to primary irritants and does not become sensitive as easily as the skin does. Saliva constantly bathes the oral mucosa, washing sensitizers from the mucosal surface and preventing proper contact. Furthermore, the presence of extensive mucosal vasculature enables for the quick absorption and clearance of allergens. Nonetheless, contact responses on the oral mucous membrane can occur. Contact sensitivity has been implicated in recurrent oral ulceration43, 32 as well as oral lichenoid responses [3-4].

Oral lichen planus (OLP) is a prevalent chronic inflammatory mucocutaneous illness with a global frequency of 1.01 percent and significant geographic variances among study participants. The buccal mucosa, tongue, and gingiva are the most commonly impacted oral regions. Oral lichen planus primarily affects females between the ages of 30 and 60. Clinically, there are six kinds of this disease: reticular, popular, plaque-like, atrophic, ulcerative/erosive, and bullous. The key features are histopathological evidence of liquefaction degeneration in the basal cell layer, an inflammatory cell infiltrate composed primarily of lymphocytes, and the absence of epithelial dysplasia. These characteristics serve as the foundation for a clinical diagnosis of oral lichen planus. In this regard, the pathophysiology of oral lichen planus is yet unknown. It's thought to be a T-cell-mediated chronic inflammatory tissue with antigen-specific and nonspecific pathogenic processes [5].

Oral lichenoid lesions (OLL) are a chronic inflammatory condition with an unknown cause that has a similar prevalence to oral lichen

***Corresponding author:** Svetelina Velikova, Department of Clinical Laboratory and Clinical Immunology, Medical University of Sofia, Bulgaria, Tel; 3599559430775; E-mail: velikovatsvetelina@gmail.com

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planus. Most clinical and histological symptoms of oral lichen planus and oral lichenoid diseases are similar. Oral lichen planus (OLP) is a common chronic inflammatory condition of the mucous membranes with an unknown cause that has the potential to progress to cancer. The pathophysiology of OLP is linked to immune factors, infectious factors, genetic factors, and psychosocial issues. Immunomodulation is vital in the recurrence of OLP assaults and the maintenance of its inflammatory state. OLP is currently thought to be an autoimmune illness mediated by T cells. OLP was a prevalent oral mucosal disease with an unknown cause, and its pathophysiology was linked to immunological factors. Drugs, dental materials, allergies, mechanical damage, microbes, and other stimulating factors may enhance the modification of antigens on keratinocytes or the exposure of unknown antigens at the early stage of OLP lesion formation. These auto antigens, super antigens, or exogenous antigens can then activate the immune system [6-7].

Materials and Method

This study looked at 30 OLCL patients and 30 OLP patients who were age and gender matched. In addition, we assessed the efficacy of removing dental metal containing a positive metal allergy, as determined by a skin patch test and metal component analysis, in patients with OLCLs. All procedures in the studies were carried out in accordance with the ethical standards of the ethics committee board of the faculty of dentistry at Tokyo Medical and Dental University (D2015-575), as well as the 1964 Helsinki declaration and its subsequent amendments or comparable ethical standards. The ethics committee board waived the requirement for formal permission for this sort of investigation [8].

Hematoxylin and eosin (H&E) staining and optical microscopy were used to assess isological features. When all of the criteria were met, the diagnosis was verified as oral lichen planus; when some of the criteria were missing, the diagnosis was confirmed as oral lichenoid lesion. The current investigation included only classic and traditional cases of oral lichen planus and oral lichenoid lesions. Cases that were inconclusive were excluded. The oral inflammatory fibrous hyperplasia group was separated into two subgroups based on whether there was a lichenoid inflammatory infiltration (OIFHL) or not (OIFHNL). The samples were chosen at random for histopathologic and immunohistochemical staining [9].

Discussion

One of the most frequent oral mucosal illnesses is lichen planus. However, its aetiology and pathophysiology are still unknown. Because of the debate about malignant transformation, oral lichen planus is one of the most discussed disorders in oral and maxillofacial pathology. Furthermore, oral lichen planus and oral lichenoid lesions have lichenoid characteristics in clinical and histological aspects. This could explain the large differences in diagnosis between pathologists and physicians. As a result, studies that aim to fill this gap in the literature should be encouraged. In this context, the existence of interface mucositis with lymphocyte predominance in both diseases shows that Langerhans cells play a role in the pathophysiology of either disorder. According to the literature, these ailments are of many types. While oral lichen planus may be an immune-mediated process, oral lichenoid lesions appear to be caused by cell-mediated hypersensitivity.

We further propose that oral lichen planus has a CD207high pattern, whereas oral lichenoid lesions have a CD207low pattern. This could explain some of the differences in the development of both diseases. As a result, the frequency of distribution of Langerhans cell subsets can influence T-cell activation and, as a result, cytokine balance in the inflammatory milieu. The purpose of this study was to assess the distribution and concentration of LC in OLP and OLL, and it was discovered that there is a considerable increase in these cells when compared to noninflamed neighbouring tissue and normal mucosa. LC is essential for antigen processing and subsequent presentation to T lymphocytes. Their presence near the dermoepithelial junction, together with their increased abundance in OLP and OLL, suggests that they may play an important role in the adaptive immunological response to lichenoid conditions [10].

Conclusion

Finally, our findings corroborate Larsson and Warfringe's findings and support the concept that LCLs can occasionally transition into OSCC. Furthermore, the removal of amalgam and subsequent clinical and histological healing does not appear to serve a preventive function in the prevention of OSCC. Regular patient follow-up is also advised in cases of clinical and histological disease regression, as malignant transformation can occur several years after clinical remission.

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None

Conflict of Interest

None

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