Oral Lichenoid Reaction-An Overview

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Abstract: Lichenoid reaction is a mucosal lesion characterized by linear whitish striae appearing on the mucosa and is triggered by external factors like restorative materials, betel nut, tobacco, graft versus host reaction and drugs. A detailed history will help in delineating the two lesions. The review provides an insight into the clinical, histological features and the malignant potential of Lichenoid reaction

Keywords: Lichenoid, drug-induced, malignant transformation, betel nut, tobacco

I. Introduction

The term Lichen refers to a group of skin diseases characterized by eruptive skin lesions which are grouped together as they resemble the algae lichen found growing on rocks. Lichen planus is a relatively common autoimmune disease that affects the skin and mucosa. A similar clinical and histological lesion is called Lichenoid reaction which is induced by external factors which could be either topical or systemic\textsuperscript{1}. This mucosal reaction is seen commonly in arecanut / tobacco products users also. A number of triggering factors such as restorative materials, graft versus host reaction and broad group of drugs are known to cause Lichenoid reaction.\textsuperscript{2,3} Lichenoid reactions have been classified based on their etiology into four types depending on the triggering factors-

- lichenoid reaction associated with amalgam restoration,
- drug related lichenoid reaction,
- lichenoid reaction associated with graft versus host reaction

Lichenoid reaction unclassified which cannot be categorized into any of these.\textsuperscript{4}

1.1 Lichenoid Reaction Associated With Amalgam Restoration

The diagnosis of oral lichenoid reaction (OLL) caused by Dental materials depends on the clinical presentation, histological confirmation of lichenoid reaction, a positive patch test to mercury, anatomical relationship of the lesion and amalgam restoration. A number of materials used in restorative dentistry have been identified as triggering agents, including silver amalgam, cobalt, palladium, chromium and even non-metals such as epoxy resins.

The erosive forms are commonly associated with this type than the reticular or striae. The mechanism behind this lichenoid appearance is that dental material or its corrosion product alter the antigenic properties of the basal keratinocyte and make it susceptible to cell mediated autoimmune damage\textsuperscript{5}. Therefore Lichenoid reaction may represent as a manifestation of chronic irritation in some or a delayed hypersensitivity reaction in others. The removal of restoration shows an improvement in 89% of these patients.\textsuperscript{6}

1.2 Drug Related Oll

Drug induced oral lichenoid reaction was first described in 1971 by Almeyda and Levantine A number of drugs have been implicated as causing Lichenoid reaction but the most common among them are Non steroidal anti-inflammatory drugs or NSAIDS and angiotensin converting enzyme inhibitors. NSAIDS drugs have been found to associated with erosive type of Lichenoid reaction\textsuperscript{6} Grinspan syndrome, a condition which was thought to be a syndrome complex is in fact a drug induced Lichenoid reaction emerging due to the drugs used to treat the diabetes and hypertension.\textsuperscript{7,8}

1.3 Lichenoid Reaction In Chronic Graft Versus Host Disease

Chronic graft versus host disease refers to disease occurring hundred days post bone marrow transplantation either as progression of acute GVHD (Graft Versus Host Disease) or arises de novo. Lichenoid reaction is frequently seen in these individuals with chronic GVHD and palatal involvement was more frequent. GVHD begins by donor cells recognizing a host peptide called Major Histocompatibility proteins. Though antigen specificity of Lichenoid reaction and GVHD are different entities they have a common immunological effector mechanism. This results in T-cell infiltration, basement membrane disruption and apoptosis of basal cell layer\textsuperscript{9}

1.4 Oll Unclassified
Flavouring agents added to food and dentrifices may trigger Lichenoid reaction. Such lesions can be identified after taking a proper history. These lesions associated with the use of a particular chewing gum or dentifrice or other flavouring substances disappears when the habit is discontinued.

II. Clinical Features

Lichenoid reaction resembles Lichen planus by presenting as a classic lace like white lesion which is present bilaterally on buccal mucosa. Lichenoid reaction can appear at sites like palate which are atypical for Lichen planus to occur. Labial involvement is seen in drug induced Lichenoid reactions. Patients presents with mild symptoms of sensitivity, pain and burning sensation.

III. Histopathological Features

Lichenoid reaction presents with similar histopathologic features of Lichen planus like hyperkeratosis, superficial band of infiltrate in the lamina propriae, areas of basal cell degeneration and saw tooth rete pegs. Certain variations like presence of a substantial number of plasma cell in the infiltrate along with some acute inflammatory cells like eosinophils and neutrophils differentiate Lichenoid reaction. The number of mast cells are comparatively lesser in Lichenoid reaction than in Lichen planus.

An indirect immunofluorescence has been used in the patients sera to identify drug induced Lichenoid reaction. The annular Fluorescence pattern to IgG has been termed “String of pearls” reaction.

In drug induced Lichenoid reaction the route of antigen presentation is unlikely across the epithelium for systemic drugs. For Lichenoid reaction due to topical agents the route may be across the epithelium. In Graft Versus Host Disease the antigen processing has essentially taken place while the Immunocompetant cells were still in the donor body. After grafting, those Immunocompetent cells react to antigens expressed in host cells.

IV. Pathogenesis

Cell mediated immunity plays an important role in the pathogenesis which is in tune with Lichen planus. Lichen planus is an auto immune disease where as in Lichenoid reaction there is alteration in immunological mechanism due to external agent. The preliminary step is the interaction of the antigen with the keratinocyte. The basal keratinocyte gets altered and stimulates the resident mast cells in the area. RANTES and other cytokines mediate this process. Mast cells in turn cause T cell activation which leads release of a number of cytokines. This also causes expression of CD54 expression by keratinocyte and leads to Basement membrane damage.

The number of Langerhans cells are more dendritic and shows decreased surface expression of class II major histocompatibility antigens in lichenoid reaction when compared with Lichen planus.

V. Diagnostic Criteria

According to Krutchkhoff diagnosis in Lichenoid lesions can range from Non-specific Lichenoid stomatitis, Atypical Lichenoid stomatitis and Lichenoid dysplasia. Atypical Lichenoid stomatitis and Lichenoid dysplasia represents lesions that show a epithelial maturation disturbance and so these lesions have a malignant potential. Lichenoid lesion it was observed that malignant transformation were more often associated with atrophic, erosive and ulcerative lesions.

Nowadays Patch testing is used detect allergies to specific substances. Patch testing results may be substantiated using the nature of the substance, their concentration, vehicle used and criteria for evaluating the patch test. Across the years there has been a steady increase in diagnosis of cases as Lichenoid reaction and a decline in cases diagnosed as Lichen planus. This immune mediated mechanism may be a sign of a disease process rather than pathognomonic for Lichen planus. Hence investigation into the underlying disease process is essential.

VI. Treatment

Amalgam related Lichenoid reaction are treated by replacing it with a alternative restorative material. Drug related Lichenoid are also commonly trated by using a substitute drug. Carbondioxide laser have been used in treatment of oral lichenoid reaction and has shown satisfactory healing. Tracolimus in orabase was found to be an alternative to topical steroids especially in patients at risk for oral candidiasis.

According to some studies Malignant transformation were found to be more likely in Lichenoid reaction than Lichen planus It was more often associated with atrophic, erosive and ulcerative lesions. All patients in the study had used Topical steroids for extended periods of time. The use of Corticosteroids therapy could not only hasten the development of malignancy but would also do so with reduced symptoms. Whether those therapies have indeed played a role in malignant transformation remains a matter of debate.
frequency of carcinoma arising from Lichenoid reaction due to Chronic GVHD is greater compared to typical Lichenoid reaction.19

VII. Conclusion
Thus lichenoid reaction are lesions similar to Lichen planus clinically and in pathogenesis, distinguishing between the two lesions is of prime importance.

References

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