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REVIEW ARTICLE

Oral lichen planus: clinical and histopathological considerations

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Summary

Oral lichen planus is one of the most common dermatological diseases presenting in the oral cavity; the prevalence in the general population is 1% to 2%. Although relatively frequent, oral lichen planus is the target of much controversy, especially in relation to its potential for malignancy. **Aim:** This study aimed to make clinical and histopathological considerations regarding oral lichen planus to increase the level of knowledge about this condition among health professionals, underlining the importance of long-term follow-up of these patients. **Conclusion:** The possibility of this lesion to turn malignant justifies the importance of long term follow up for patients with such disease.

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INTRODUCTION

Lichen planus is a chronic inflammatory disease that affects the skin and mucosa. It is one of the most common dermatological conditions involving the oral cavity; its prevalence is 1% to 2% in the general population. There is a strong preference for the female sex[1]

Sousa & Rosa[2] (2005) surveyed 79 oral lichen planus cases diagnosed between 1974 and 2003, and found that women are nearly four times more affected by this condition than men, and that white individuals are five and a half times more likely to develop this disease compared to other races.

These features, however, are among the few points of agreement about oral lichen planus; most of the remaining aspects are controversial, especially it's potential for malignant transformation. The aim of this paper, therefore, is to describe oral lichen planus clinically and histopathologically, and to help disseminate this information among health professionals; the importance of long-term follow-up of these patients is underlined.

ETIOPATHOGENESIS

Although oral lichen planus was initially described in 1869, little is known about mechanisms by which the disease develops.

Sugerman et al.[3] (2002) believe that specific and non-specific mechanisms may be involved in the etiopathogenesis of this condition. Specific mechanisms include antigen presentation by basement layer keratinocytes and cytotoxic T lymphocyte-caused death of antigen-specific keratinocytes, while non-specific mechanisms included mast cell degranulation and matrix metalloproteinase activation. These combined mechanisms appear to cause T lymphocytes accumulation in the lamina propria underlying the epithelium, as well as rupture of the basement membrane, intraepithelial T lymphocytes migration and keratinocyte apoptosis, all of which are characteristic of oral lichen planus. Furthermore, according to these authors, the chronic nature of this disease may be partly explained by deficient immunosuppression, mediated by the transforming growth factor-beta [1].

The factors that set this process in motion, however, have not been fully clarified. Still, stress, food such as tomatoes, citric fruit and seasoned dishes, dental procedures, systemic disease, alcohol abuse, and tobacco use in all its forms, have been associated with disease exacerbation periods.[4] Recently, systemic diseases, especially those resulting from hepatitis C virus infection, have come under the spotlight.

Lodi et al.[5] (2004) used the enzyme-linked immunosorbent assay (ELISA) test to investigate the presence of antibodies against the hepatitis C virus in 581 patients, 303 of which with a clinical and histopathological diagnosis of oral lichen planus, and 278 with no evidence of this disease (control group). Of 303 patients diagnosed with oral lichen planus, 58 (19.1%) were positive for the hepatitis C virus, compared to only nine (3.2%) in the control group. Furthermore, the authors reviewed the results of 24 similar studies done between 1994 and 2003, and found a statistically significant difference in the proportion of serum positive individuals for the hepatitis C virus among patients with oral lichen planus compared to controls.

The relation between oral lichen planus and the hepatitis C virus is not consistent; the prevalence of this virus in such patients varies widely from 0% to over 60%, depending on the country in which these studies were conducted. It is thought that such differences are mainly due to geographic discrepancies in the prevalence of this virus within the general population.[6]

Henderson et al.[7] (2001) have questioned this hypothesis. The author assessed the oral health and the availability of dental treatment for hepatitis C virus infected-patients in the United Kingdom, and found clinical evidence of lichen planus in 20% of these patients. This was a much higher percentage than that found in the general population, in which the prevalence was not more than 1%. This finding came to the author's attention, as the prevalence of the hepatitis C virus is low; the literature shows that there is a directly proportional relation between the prevalence of the hepatitis C virus and oral lichen planus.

Cunha et al.[8] (2005) noted similar findings in a study of 134 serum positive patients for the hepatitis C virus in Brazil. Although the prevalence of this virus is high in this country, the percentage of patients with lichen planus was 1.5%; there was, therefore, no statistically significant difference compared to the control group, in which 1.1% of patients presented signs of the disease.

A direct association between lichen planus and the hepatitis C virus cannot always be demonstrated. Mico-Llorens et al.[9] (2004), for instance, found no changes in the oral mucosa of 100 patients infected with the hepatitis B or C or both viruses that participated in a study done by the Digestive System Pathology Unit of the "Príncipes de España de Bellvitge" Hospital in September and October 2000.

Romero et al.[10] (2002), however, have underlined the need to investigate the presence of hepatitis C virus antibodies in all patients with oral lichen planus. These authors believe that the existence of clinical variants of the disease, in terms of its site and number of intra-oral lesions in hepatitis C virus-infected and non-infected patients, suggests that this virus has a significant role in the progression of lichen planus.