PREVALENCE OF ORAL LICHEN PLANUS IN BRAZILIAN PATIENTS WITH CUTANEOUS LICHEN PLANUS

PREVALÊNCIA DO LÍQUEN PLANO BUCAL EM PACIENTES BRASILEIROS PORTADORES DE LÍQUEN PLANO CUTÂNEO

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ichen planus (LP) is a mucocutaneous disease affecting 0.5% to 2.0% of the population. We made this study to determine the prevalence of oral mucous involvement in Brazilian patients with cutaneous lichen planus (CLP). Forty-five patients with CLP and 100 patients carriers of other diseases were clinically examined. Of the 45 patients with CLP, 28 were men (62.2%) and 17 were women (37.8%), with an average age of 37.2 years and an age range of 4 to 74 years. In the control group, of the 100 patients with other diseases, 26 were men (26%) and 74 were women (74%), with an average age of 36.1 years and an age range of 5 to 79 years. The patients were submitted to a full general clinical, dermatological and intraoral examination and there was histopathological confirmation of the clinical diagnosis. Eighty six point seven percent (86.7%) of patients reported emotional stress during onset of disease. We noted highly significant increase in CLP about past diseases (CLP-88.9%,CG-48%), tobacco consumption (CLP-33.3%, CG-17%) and alcohol consumption (CLP-62.2%, CG-14%). Family history for LP was found in 11.3%. Only 22.2% presented oral LP (reticular - 50%; papular - 30%; plaque-like - 20%). In our study, specific oral involvement was lower than found in literature. The importance of this study has been the fact that the patients who sought a physician for cutaneous involvement didn't know whether they had oral involvement simultaneously or not.

UNITERMS: Lichen planus; Mucocutaneous disease.

INTRODUCTION

Lichen planus (LP) is a mucocutaneous disease reportedly affecting 0.5% to 2.0% of the population²². This is a disease of unknown aetiology first described in 1869 by Wilson. The possibility of autoimmunity has been raised in many studies, especially in cases where LP is associated with one or more autoimmune diseases⁸. Many authors suggest as etiologic factors emotional stress and enzymatic alterations¹⁴.

Cutaneous lichen planus is characterized by shiny flat papules. The papules retain the skin lines and are described as polygonal. Individual papules vary in size from pinpoint to a centimetre or more across and may be closely aggregated or widely dispersed. Although the size of papule is often fairly uniform in each patient, this is not necessarily so, and minute and large papules are intermingled in some cases. On the surface there may often be seen white lines, known as Wickham's striae. The overall colour is also often characteristic, and is described as violaceous¹.

The review of literature shows that about 50% of the patients with skin lesions have oral lesions, whereas about 25% of all LP patients have only oral lesions⁸. Clinically, these can appear in at least six forms: reticular, papular, plaque-like, atrophic, erosive, and bullous lesions that can occur separately or simultaneously²².

The objective of this study was to determine the presence of oral lichen planus in Brazilian patients with cutaneous lichen planus.

MATERIAL AND METHODS

Forty-five patients with cutaneous lichen planus (CLP) and 100 patients carriers of other diseases were clinically examined. Of the 45 patients with CLP, 28 were men (62.2%) and 17 were women (37.8%), with an average age of 37.2 years and an age range from 4 to 74 years. In the control group, of the 100 patients with other diseases, 26 were men (26%) and 74 were women (74%), with an average age of 36.1 years and an age range from 5 to 79 years. The patients were submitted to a full general clinical, dermatological and intraoral examination. All the general alterations, the dermatological and the oral lesions found were recorded and there was histopathological confirmation of the clinical diagnosis.

Before the physical examination, all patients were submitted to a full history: their history of present illness, other present diseases, past medical history, tobacco and alcohol consumption, atopy and familiar history as to lichen planus.

The results of CLP were compared to control group by c^2 statistic test through Woolf's methods (1955).

RESULTS

The results are shown in the following tables:

TABLE 1 – Prevalence rate of cutaneous lichen planus and control group according to gender

GENDER	CUTANEOUS LICHEN PLANUS N(%)	CONTROL N(%)
MALE	28 (62.2)	26 (26)
FEMALE	17 (37.8)	74 (74)
TOTAL	45 (100.0)	100 (100,0)

χ²= 16.48 DF=1 p<0.01

TABLE 2 - Prevalence rate of cutaneous lichen planus and control group according to race

RACE	CUTANEOUS LICHEN PLANUS N(%)	CONTROL N(%)
WHITE	35 (77.8)	89 (89)
NOT WHITE	10 (22.2)	11 (11)
TOTAL	45 (100.0)	100 (100)
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 χ^2 = 3.26 DF=1 n.s.

TABLE 3 - Prevalence rate of cutaneous lichen planus and control group according to presence of emotional stress

EMOTIONAL STRESS	CUTANEOUS LICHEN PLANUS N(%)	CONTROL N(%)
POSITIVE	39 (86.7)	71 (71)
NEGATIVE	6 (13.3)	29 (29)
TOTAL	45 (100.0)	100 (100)

 χ^2 = 3.92 DF=1 p<0.05

 TABLE 4 - Distribution of patients by types of emotional stress that triggered CLP

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TYPES OF EMOTIONAL	CUTANEOUS LICHE
STRESS	PLANUS N (%)
IRRITABILITY DEPRESSION ANXIETY DEATH OF FAMILY MEMBER MISERY FEAR INTROVERSION EMOTIONAL FITS ADULTERY DISCOVERED BY SPOUSE	9 (20.0) 8 (17.8) 7 (15.6) 7 (15.6) 6 (13.3) 4 (8.9) 3 (6.7) 2 (4.4) 1 (2.2)
ADULTERY OF SPOUSE	1 (2.2)
JEALOUSY	1 (2.2)
FEELING OF GUILT	1 (2.2)

PAST DISEASES	CUTANEOUS LICHEN PLANUS N(%)	CONTROL N(%)
NEGATIVE	5 (11.1)	52 (52)
POSITIVE	40 (88.9)	48 (48)
TOTAL	45 (100.0)	100 (100)

TABLE 5 - Distribution of patients according to presence of history of past diseases

χ²= 18.71 DF=1 p<0.01

TABLE 6 - Distribution of patients according to tobacco consumption

TOBACCO CONSUMPTION	CUTANEOUS LICHEN PLANUS N(%)	CONTROL N(%)
POSITIVE	15 (33.3)	17 (17)
NEGATIVE	30 (66.7)	83 (83)
TOTAL	45 (100.0)	100 (100)
TOTAL	45 (100.0)	100 (100)

χ²= 4.84 DF=1 p<0.05

 TABLE 7 - Distribution of patients according to alcohol consumption

ALCOHOL CONSUMPTION	CUTANEOUS LICHEN PLANUS N(%)	CONTROL N(%)
POSITIVE	28 (62.2)	14 (14)
NEGATIVE	17 (37.8)	86 (86)
TOTAL	45 (100.0)	100 (100)

χ²= 30.74 DF=1 p<0.01

TABLE 8 - Distribution of patients according to presence of history of atopy

ΑΤΟΡΥ	CUTANEOUS LICHEN PLANUS N(%)	CONTROL N(%)
NEGATIVE	31 (68.9)	82 (82)
POSITIVE	14 (31.1)	18 (18)
TOTAL	45 (100.0)	100 (100)

 χ^2 = 3.18 DF=1 p<0.05

TABLE 9 - Distribution of patients according to the presence of family history for lichen planus

FAMILY HISTORY	CUTANEOUS LICHEN PLANUS
POSITIVE	5 (11.1)
NEGATIVE	40 (88.9)
TOTAL	45 (100.0)

TABLE 10 - Prevalence	of oral	lichen	planus	in	patients
with cutaneo	ous liche	n plan	us		

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ORAL LICHEN PLANUS	CUTANEOUS LICHEN PLANUS N (%)
POSITIVE	10 (22.2)
NEGATIVE	35 (77.8)
TOTAL	45 (100.0)

TABLE 11 - Distribu	ution of patients according to type of oral
lichen p	blanus

ORAL LICHEN PLANUS	CUTANEOUS LICHEN PLANUS N (%)
RETICULAR	5 (50.0)
PAPULAR	3 (30.0)
PLAQUE-LIKE	2 (20.0)
TOTAL	10 (100.0)

DISCUSSION

About the involvement of sex incidence, we saw 62.2% of CLP in male sex and 37.8% in female sex (Table 1). Women are said to be affected rather more often than men, although an opposite ratio or equal involvement of sex incidence has been found¹. In our study, the results showed a highly significant association of CLP with male sex.

We found increased prevalence of CLP in white race (CLP-77.8% and C-89%) over not white race (CLP-22.2% and C-11%) (Table 2). We believe that with this work, we may not link the disease with race, because in control group, we found higher prevalence of the white race too.

Eighty six point seven percent (86.7%) of patients reported emotional stress during onset of disease. We noted statistically significant increase in CLP about emotional stress when compared to the control group (Table 3). The literature relates emotional stress as associated with the disease^{3, 14, 16}. We found many different situations which were probable causes of stress and we have listed them below: depression, misery, irritability, anxiety, death of family member, fear, introversion, emotional fits, adultery discovered by spouse, adultery of spouse, feeling of guilt, jealousy (Table 4).

We noted highly significant increase in CLP about past diseases, tobacco consumption, alcohol consumption when compared to the control group (Tables 5, 6, 7). We believe these situations to be aggressions that could trigger the disease.

Many authors suggest immunologic mechanism as the cause of lichen planus. Factor XIIIa+ "dendrocytes", normal residents of the submucosa and dermis are a morphologically and phenotypically distinctive subset of

the monocyte-macrophage system. Believing these cells participate in the regulation of immune responses, REGEZI et al¹³ (1994) postulated that they may play a role in the pathogenesis of lichen planus. They found XIIIa+ dendrocytes significantly increased in number and size in lichen planus. The coexistence of lichen planus, primary biliary cirrhosis, and sicca syndrome manifest in the same patient substantiates a theory of pathogenesis based on a cell-mediated immune response analogous to that in allogeneic bone marrow transplant recipients with chronic graft-versus-host disease⁶. SASAKI et al¹⁵(1996) described three cases of linear lichen planus on the lower extremities unaccompanied by mucous lesions. Dental metal compounds were thought to be the precipitating factor in all cases. We didn't find association with contact dermatitis by dental metal. The history of atopy didn't have significant differences compared to the control group (Table 8). The atopy has immunologic mechanism in its ethiopathology^{18, 21}.

A wide variety of drugs have been implicated in its cause. Sufficient evidence exists that beta-blockers, methyldopa, penicillamine, quinidine, and qinine play a role in this disorder. Evidence is insufficient for angiotensin-converting enzyme inhibitors, sulfonylurea agents, carbamazepine, gold, lithium, and a host of miscellaneous drugs. Given available epidemiological evidence, nonsteroidal antiinflammatory agents probably should also be considered causative²⁰. We however have not found any relationship between specific drugs and CLP so far.

Family history for lichen planus was found in 11.3% (Table 9). Increased prevalence of family history is related. Associations with HLA has been shown^{1,10}. The keratinocytes has been shown to express HLA-DR antigens only⁸.

Only 22.2% presented oral lichen planus (Table 10). In our study, specific involvement was lower than found in literature, which refers about 50% of the patients with skin lesions have oral lesions⁸. HYRAILLES et al.⁷ (1995) refer both mucous and cutaneous lesions in 30% of cases.

Various forms of oral lichen planus appear clinically: reticular, papular, plaque-like, atrophic, erosive and bullous lesions that can occur separately or simultaneously². We found these types of oral lichen planus: reticular, papular, plaque-like (Table 11).

Lichen planus has recently been described in some patients with hepatitis (virus-related liver disease)⁷. Ten patients were reported to suffer from oral lichen planus associated with chronic liver diseases linked to HCV. All patients were affected by varieties of erosive oral lichen planus. GANDOLFO et al.⁵ (1994) suggested the possible existence of erosive relationship between oral erosive lichen planus and HCV infection. In our study, we didn't find erosive oral lichen planus, nor HCV infection in history of past diseases.

The diagnosis of oral lichen planus is very important because many authors say that this may be a precancerous condition of the mouth mucous, without any other concomitant condition¹¹. For EISENBERG⁴ (1992), evidence for LP's reputed precancerous nature is unconvincing. Moreover, it is possible that biopsy-proven LP occurs far less frequently than believed previously. Diagnosis of LP has too often been made without strictly adhering to strict diagnostic criteria, on data that fail to satisfy accepted diagnostic principles. As to malignant transformation of the skin lesions, SIGURGEIRSSON; LINDELÖF¹⁷ (1991) made an epidemiological study of 2071 patients that indicated that patients with cutaneous lichen planus do not carry an increased risk of malignant transformation. However, recents studies show that 1.2% to 3.2% of patients who suffer from oral lichen planus develop malignant transformation^{9, 11, 19}.

CONCLUSION

In our study, only 22.2% of the patients with cutaneous lichen planus presented oral lichen planus. The importance of this study, which evaluated oral mucous in patients with CLP, has been the fact that the patients who sought a physician for cutaneous involvement didn't know whether they had oral involvement simultaneously or not. A situation which clearly differs from other authors in literature, where patients sough help specially for oral complaints.

RESUMO

O líquen plano (LP) é uma doença mucocutânea de etiologia desconhecida, que afeta 0,5 a 2% da população. Este estudo foi realizado para se determinar a prevalência de comprometimento bucal específico em pacientes brasileiros portadores de líquen plano cutâneo (LPC). Quarenta e cinco pacientes com LPC e 100 pacientes portadores de outras doenças (Grupo controle - GC) foram clinicamente examinados. Dos 45 pacientes portadores de LPC, 28 eram do sexo masculino (62,2%) e 17, do sexo feminino (37,8%). As idades do grupo LPC variaram de 4 a 74 anos, com média de 37,2 anos. No grupo controle, dos 100 pacientes portadores de outras doenças, 26 eram do sexo masculino (26%) e 74 do sexo feminino (74%). As idades do grupo controle (GC) variaram de 5 a 79 anos, com média de 36,1 anos. Os pacientes foram submetidos à exame clínico geral completo, exame dermatológico e bucal. Realizou-se exame histopatológico para confirmar os diagnósticos clínicos. Oitenta e seis, sete por cento (86,7%) dos pacientes relataram estresse emocional como desencadeante da doença. Observou-se um aumento significativo no grupo LPC de doenças anteriores (LPC-88,9%, GC-48%), tabagismo (LPC-33,3%, GC-17%), etilismo (LPC-62,2%, GC-14%). História familiar para LP foi relatada em 11,3%. Somente 22,2% apresentavam LP bucal (reticular - 50%; papular - 30%; em placa -

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20%). Neste estudo, o comprometimento bucal específico foi menor do que o relatado na literatura. A importância deste estudo está no fato de que os pacientes procuraram o médico, devido ao comprometimento cutâneo, não sabendo previamente se apresentavam comprometimento bucal simultâneo.

Unitermos: Líquen plano; Doença mucocutânea.

REFERENCES

- BLACK, M.M. Lichen planus and lichenoid eruptions. In: ROOK, A. et al. Textbook of dermatology. 4.ed. Blackwell Scientific Publications, 1986. p.1665-85.
- BUAJEEB, W. et al. Efficacy of topical retinoic acid compared with topical fluocinolone acetonide in the treatment of oral lichen planus. Oral Surg., v.83, p.21-5, 1997.
- 3. CRAN, J.A. Lichen planus associated with psoriasis: case report. Aust. dent. J., v.11, p.429-30, 1966.
- EISENBERG, E. Lichen planus and oral cancer: is there a connection between the two? J. Amer. dent. Ass., v.123, p.104-8, 1992.
- GANDOLFO, S. et al. Oral lichen planus and hepatitis C virus (HCV) infection: is there a relationship? A report of 10 cases. J. oral pathol. Med., v.23, n.3, p.119-22, 1994.
- GART, G.S.; CAMISA, C. Ulcerative and oral lichen planus associated with sicca syndrome and primary biliary cirrhosis. Cutis, v.53, n.5, p.249-50, 1994.
- HYRAILLES, V. et al. Lichen plan et virus de l'hepatite C. A propos de 5 nouveaux cas. Gastroent. Clin. Biol., v.19, n.10, p.833-6, 1995.
- JUNGELL, P. Lichen planus. Int. J. Oral Maxillofac. Surg., v.20, p.129-35, 1990.
- LOZADA-NUR, F.; MIRANDA, C. Oral lichen planus: epidemiology, clinical characteristics, and associated diseases. Semin. Cutan. Med. Surg., v.16, n.4, p.273-7, 1997.
- McKUSICK, VA Mendelian inheritance in man. Catalogs of autosomal dominant, autosomal recessive and X-linked phenotypes. The Johns Hopkins University Press, 1986. p.461.
- MARKOPOULOS, A.K. et al. Malignant potential of oral lichen planus; a folow-up study of 326 patients. Oral Oncol., v.33, n.4, p. 263-9, 1997.
- 12. MOHADJER, C. et al. Prakanzerose der Mundschleimhaut:: Lichen ruber planus. **HNO**, v.43, n.3, p.191-2, 1995.
- REGEZI, J.A. et al. Increased submucosal factor XIIIa-positive dendrocytes in oral lichen planus. J. oral pathol. Med., v.23, n.3, p.114-8, 1994.
- 14. SAMPAIO, S.A.P. et al. **Dermatologia básica.** 3. ed. São Paulo, Artes Médicas, 1985. p.145-6.

- SASAKI, G. et al. Three cases of linear lichen planus caused by dental metal compounds. J. Dermat., v.23, n.12, p.890-2, 1996.
- SHAFER, W.G. et al. Tratado de patologia bucal. 4.ed. Guanabara, 1987. p.748-53.
- 17. SIGURGEIRSSON, B.; LINDELÖF, B. Lichen planus and malignancy. Arch. Dermat., v.127, p.1684-8, 1991.
- SIHRA, B.S. et al. Expression of high-affinity IgE receptors (Fc epsilon RI) on peripheral blood basophils, monocytes, and eosinophils in atopic and nonatopic subjects: relationship to total serum IgE concentrations. J. Allergy Clin. Immun., v.99, n.5, p.699-706, 1997.
- SILVERMAN, S. JR; BAHL, S. Oral lichen planus update: clinical characteristics, treatment responses, and malignant transformation. Amer. J. Dent., v.10, n.6, p. 259-63, 1997.
- THOMPSON, D.F.; SKAEHILL, P.A. Drug-induced lichen planus. Pharmacotherapy, v.14, n. 5, p. 561-71, 1994.
- ULBRECHT, M. et al. High serum IgE concentrations: association with HLA-DR and markers on chromosome 5q31 and chromosome 11q13. J. Allergy Clin. Immun., v.99, n.6, pt.1, p. 828-36, 1997.
- VICENT, S.D. et al. Oral lichen planus: the clinical, historical, and therapeutic features of 100 cases. Oral Surg., v.70, p.165-71, 1990.