بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات
لم ترد بالاصل

IMMUNOHISTOCHEMICAL DEMONSTRATION OF T-CELL SUBSETS AFTER DIFFERENT TREATMENT MODALITIES OF CORTICOSTEROIDS IN PATIENTS SUFFERING FROM EROSIVE ORAL LICHEN PLANUS

Thesis

Submitted in partial fulfillment for the requirement of the Master Degree in Oral Medicine and Periodontology

By

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بسم الله الرحمن الرحيم

﴿ قالوا سبحانك لا علم لنا إلا ما علمتنا
إنك أنت العليم الحكيم ﴾

صدق الله العظيم

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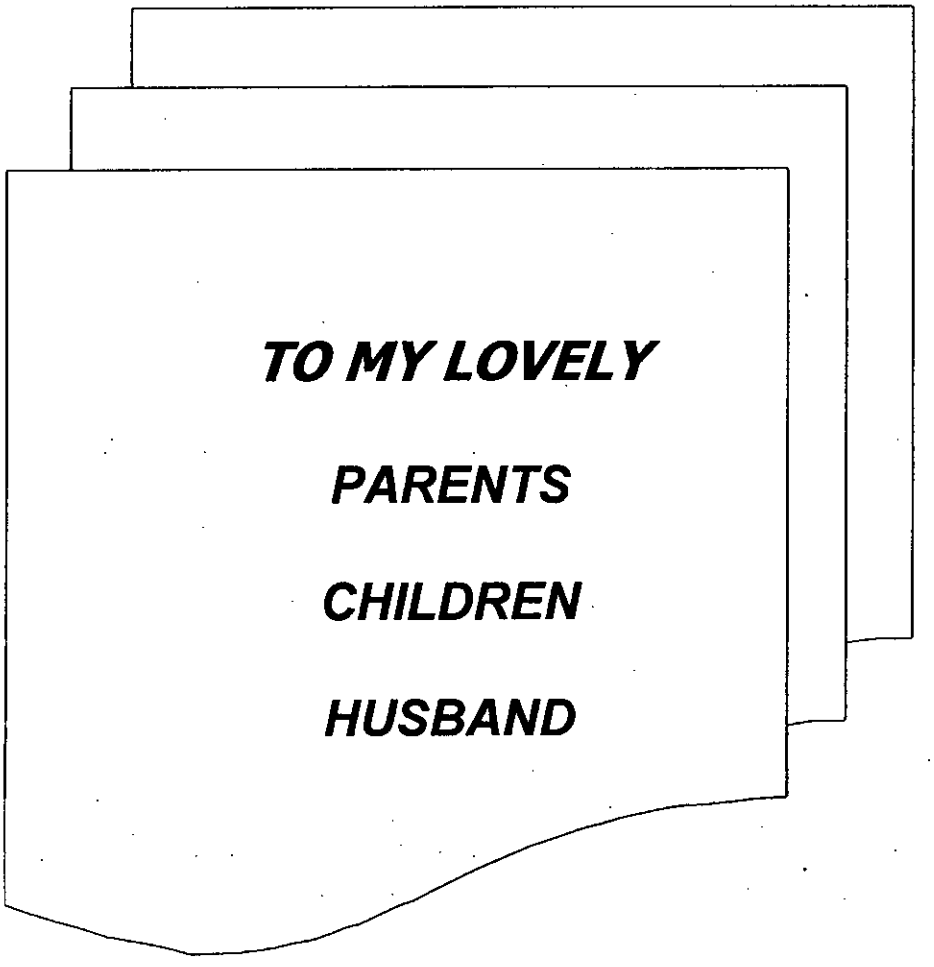

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INTRODUCTION & REVIEW OF LITERATURE

INTRODUCTION AND REVIEW OF LITERATURE

Lichen planus is a lymphocyte mediated immunologic disorder in which the basal cells appear to be the target of T-lymphocytes (***Alexandra et al., 1999***). The disease was first described by (***Wilson 1869***).

The prevalence of lichen planus in the adult population is in the order of 0.9 to 1.2%, but the prevalence rates may differ among races and geographic areas (***Vincent et al., 1991***). Racial groups that tend to have strong emotional responses are predisposed to lichen planus (***McCartan et al., 1995***). The disease tends to develop in individuals who hold positions of responsibility and who exist in an environment of tension and stress (***Warylow, 1997***), with an age incidence between 30 to 60 years (***Silverman et al., 1989***).

Lichen planus may appear as skin lesions, oral lesions or a combination of both. Lesions affecting mucous membranes tend to follow a prolonged natural history and are more painful and refractile to treatment than those affecting the skin ***(Walton et al., 1998)***. Healing of the skin lesions occur within one or two years leaving areas of hyperpigmentation (***Vincent et al., 1991***).

Many factors have been suggested in the **etiology** of lichen planus including genetic predisposition (***Jontell et al., 1987***). Systemic medications (***Firth et al., 1989***). Contact sensitivity (***Bolewska et al., 1990***). Mechanical trauma (***Katz et al., 1988***). Bacterial, viral infection,

vitamin deficiencies (***Strauss et al., 1988***) and psycogenic disturbance (***Hampf et al., 1989***). It was observed that stressfull events frequently preceede episodes of symptoms (***Lavental et al., 1984).*** Stress has been suggested as a modifying event in the disease with physical bases. Disease association with lichen planus has been recognized (***Scully and ELkom., 1985***). Since (***1978) Grinspan et al.*** suggested an association of lichen planus with diabetes and hypertension, this triad is referred to as Grinspan syndrome. This syndrome is likely to be a drug-related phenomena. Any such relation might be partially explained by the capacity of the drugs used in the treatment of those disorders to produce lichenoid reactions (***Yammomoto et al., 1990***). The prevalence of diabetes mellitus among oral lichen planus patients varies between 1.6% and 37.7% according to different investigators (***Albrect et al., 1992***).

In the last two decades an association has been proposed between lichen planus and chronic hepatic disease (***Bagan et al., 1995***). Recently, it has been noticed that HCV plays an important role not only in liver disease but also in establishment of extrahepatic manifestations and immune abnormalities (***Misiani et al., 1992; Parlotsky et al., and Koike et al., 1997***). Lichen planus lesions that appear in the oral cavity in patients infected with HCV have been considered to be extrahepatic lesion induced by HCV (***Gandolfo et al., 1994; Jubert et al., 1994 and Nagao et al., 1995***). However, the direct relationship among oral lichen planus and HCV infection has been elucidated through the presence of HCV-RNA in oral lichen planus lesions. Moreover, the reported frequency of anti-HCV antibodies in groups of cutaneous lichen planus (CLP) and oral lichen planus (OLP) varied from 4% to 6.5% (***Vanderwaal, 1999***). The performance of liver function tests has been strongly recommended in patients with lichen planus especially when

strongly recommended in patients with lichen planus especially when oral erosive lesions are present. They should be screened systemically for the presence of HCV infection (***Silvestri et al., 1996***).

A number of drugs have been implicated in the etiology of lichenoid lesions that may involve the skin and/or the mucous membrane including antirheumatic agents, non-steroidal anti-inflammatory analgesics and hypoglycemics, anti-hypertensives and antibiotics (***Scully and Elkom., 1985***). Drug induced lichenoid reaction are frequently erosive rather than reticular. The exact mechanism where by drugs induce this type of lesion is unclear although it has been suggested that it may be a type IV hypersensitivity reaction (***Warylow et al., 1997***). To implicate a drug as responsible for lichenoid reaction is difficult as there is no specific test, and one must depend on resolution of the lesion, on cessation of drug used, and recurrence of lesion on exposure to the drug (***Dale et al., 1996***).

Recently oral lichen planus (OLP) and lichenoid contact lesions (CL) have become regarded as different immunopathologic entities of the oral mucosa (***Bratel et al., 1998***). Although the (CL) are confined to areas in contact with amalgam fillings and (OLP) involves larger areas of oral mucosa, the reaction patterns that characterize the two lesions are clinically and histopathologically identical (***Holmstrup, 1991***). However reactivity to mercury demonstrated by epicutaneous patch testing, was more frequently found among patients with (CL) than in patients with (OLP) (***Bolewska et al., 1990***). (CL) improve or heal following replacement of the amalgam filling while most (OLP) lesions resist healing following replacement (***Henriksson et al., 1995 and Bratel et al., 1996***). These observations suggested that different antigenic