

SERUM IMMUNOGLOBULIN LEVELS IN LICHEN PLANUS

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Serum immunoglobulin levels were studied in 20 controls and 30 patients of lichen planus diagnosed clinically and confirmed by histopathology. The serum level of all immunoglobulins was raised as compared to normal controls but it reached statistically significant for IgA only ($P < 0.01$). Our results suggest that this may be due to some chronic infections and humoral immunological mechanism, possibly does not play any role in the aetiology of lichen planus.

Key Words : Lichen planus, Immunoglobulin

Introduction

Lichen planus is usually a self limiting condition of unknown etiology. Several hypothesis were put forward, still no satisfactory answers were found for its etiology. An immune mechanism was suggested by various workers where they have reported its association with several autoimmune disorders.¹⁻⁶

In various immunological studies of lichen planus patients contradictory observations have been reported; increased serum IgG^{7,8}, IgA^{8,9}, IgM⁸ and decreased serum IgG¹⁰, IgA^{7,11,13} and IgM¹¹⁻¹³ have been documented. Normal values have also been recorded for IgG^{5,12,14}, IgA^{5,6,14} and IgM^{5,7,14}. Mahood¹⁰ observed clear rise in IgM and IgA levels after lesions healed.

Hence the present study was undertaken to find out the serum immunoglobulins levels in lichen planus patients from tropics.

Materials and Methods

In this study 30 patients of lichen planus diagnosed clinically and confirmed by

histopathology were included. None of the patients had taken any drug known to cause lichenoid eruptions prior to the onset of disease. A thorough general physical and systemic examination was done. Haemoglobin, total and differential leucocyte count, erythrocyte sedimentation rate and urine for sugar were done. Oral glucose tolerance test was carried out only in urine sugar positive patients. Serum immunoglobulin profile (IgG, IgM and IgA) was done by single radial immuno-diffusion technique¹⁵ in 30 patients and 20 controls. Students 't' test was used for statistical analysis.

Results

Out of the 30 patients, 25 (83.33%) were males and 5 (16.67%) females. The mean age was 35.6 years (range 10 years to 70 years) for patients and 32.2 (range 10 to 52 years) for controls.

Complete blood examination was normal. Frank diabetes mellitus was detected in 3 (10%) patients by oral glucose tolerance test in whom urine examination showed presence of sugar.

Serum immunoglobulin levels of patients and controls are compared in Table 1. The serum level of all immunoglobulins was raised in the patients but it was statistically significant for IgA only ($P < 0.01$).

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Table I. Comparison of serum immunoglobulin levels

	Mean (\pm S. D.) mg/dl		
	IgA	IgM	IgG
Patients	282.13 (\pm 87.85)	255.96 (\pm 82.64)	1646.47 (\pm 400.03)
Controls	195.86 (\pm 62.32)	234.8 (\pm 65.70)	1471.97 (\pm 238.78)
t Value	3.91*	0.384	1.805

* $P < 0.01$ (highly significant)

Comments

In our study serum levels of all immunoglobulin (IgG, IgM, IgA) were raised but were statistically significant for IgA only ($p < 0.01$). This is in accordance with others.^{8,9} However this contradicts the hypothesis of immunodeficiency^{7,10-13} in causation of lichen planus.

This is a well known fact that persons with low immunoglobulin levels are more susceptible to infection, atopy and autoimmune diseases and they usually present with diarrhoea, malabsorption or infestation with *Giardia lamblia*.¹⁶ There was no evidence of such diseases in our study except diabetes mellitus. The association with autoimmune diseases¹⁻⁶ used to lend support to an autoimmune etiology of lichen planus. But these were not the result of any systemic study to determine the incidence of autoimmune diseases in an unselected group of patients of lichen planus. Furthermore, if there was an autoimmune background to lichen planus, it might be anticipated that autoantibodies would be found with increased frequency when compared with control group. Shuttleworth et al,³ did not find any increased prevalence of autoimmune diseases or autoantibodies in the lichen planus group as compared to controls.

The raised level of IgA in Indian population may likely be due to some chronic infection by bacteria, virus and/or parasitic

infestation like worm infestation or by malaria due to *P. falciparum*. Thus in our opinion humoral immunological mechanism possibly does not play any role in the aetiology of lichen planus.

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