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Original Research Article

Comparison of salivary and serum IgG and IgA levels with total protein in oral lichen planus

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ABSTRACT

Background: Lichen planus is the common dermatologic disease with oral manifestations. It is an immunologically based, chronic, inflammatory, mucocutaneous disorder with undetermined etiology. The role of salivary immunoglobulins in the immunopathogenesis and the clinical course of oral mucosal diseases, have not been yet studied comprehensively. IgG and IgA being the predominant immunoglobulins in normal serum. Secretory IgA constitutes the predominant immunoglobulin isotype in secretions, including saliva.
Materials: Followed by informed consent 20 patients diagnosed with Lichen planus are enrolled in the study from the Department of Oral and Maxillofacial Pathology, MNR Dental College and Hospital, Sangareddy. A new particle-enhanced turbidimetric immunoassay using mono-specific sheep antibodies on the SPAPLUS® analyzer is used to determine the soluble antigen concentration (IgG, IgA).
Results: The results showed that the mean serum IgG and IgA levels in lichen planus patients is higher when compared with controls. There is a significant difference in the mean IgG and IgA levels levels in both the study groups (<0.001; sig). The current study results showed that the mean salivary IgG and IgA levels in lichen planus patients is elevated compared to controls. A significant difference is found in the mean IgG and IgA levels in patients and controls (<0.001; sig).
Conclusion: The results might conclude that elevated levels of serum and salivary IgG and IgA show that humoral immunity is implicated in the pathogenesis of oral lichen planus. Further studies with more number of cases could throw light on the etiology and pathogenesis of the disease.

Keywords: Oral lichen planus, Immunoglobulins, Serum and salivary IgG and IgA

INTRODUCTION

Lichen planus is the most common dermatologic disease with oral manifestations. It is an immunologically based, chronic, inflammatory, mucocutaneous disorder with undetermined etioiogy. However, the immunological ground is one of the priority mechanisms in the multicausal pathogenesis of oral lichen planus. The oral subgroup of the disease, affects up to 1-2% of the population, occurring most often in middle age and with a female predominance.[1] There are seven recognized oral presentations of lichen planus reticular, papular, plaque-form, atrophic, ulcerative (erosive), vesiculo-bullous and desquamative gingivitis, this latter term is a clinical descriptor, used to describe inflammation, with a mix of erythema, erosion or ulceration of the gingival tissues.[2]

Clinical presentations range from symptom-free to a burning sensation or severe pain interfering with phonation, mastication, and deglutition. Oral lichen planus (OLP) is classified as a potentially malignant disorder (PMD) of the oral mucosa with a transforming rate of 0–6.25%.

Histologically, lesions are characterized by hyperkeratosis, basal layer liquefaction of the oral epithelium, and a dense infiltration of a band of lymphocytes.[3] The etiology and pathogenesis of OLP is still not well established and has been the focus of research. Cell mediated immunity is the most accepted mechanism involved in the pathogenesis of the disease. It is a T-cell mediated immune response which involves, MHC class I- and MHC class II-restricted antigen presentation by lesional keratinocytes, followed by activation of antigen- specific CD4+ helper T-cells and CD8+ cytotoxic T-cells, leading to keratinocyte apoptosis.[2] Nonspecific inflammatory mechanisms, autoimmunity and role of humoral immunity have also been put forward to explain the pathogenesis.[3] Immunoglobulins are glycoproteins, which are expressed as membrane receptors on the surface of B-cells or as soluble molecules secreting B-cells and are carriers of humoral immunity. Five distinct classes of immunoglobulin molecules namely IgG, IgA, IgM, IgD and IgE are recognized in humans.[4] The role of salivary immunoglobulins in the immunopathogenesis and the clinical course of oral mucosal diseases, have not been yet studied comprehensively. IgG and IgA being the predominant immunoglobulins in normal serum. Secretory IgA constitutes the predominant immunoglobulin isotype in secretions, including saliva.[5] Serum and salivary IgG and IgA can be quantified using turbidometric immune assay.[6] The determination of soluble antigen concentration by turbidometric method involves the reaction with specific antiserum to form insoluble complexes. Although many studies are reported on the serum immunoglobulins, the role of salivary immunoglobulins in the pathogenesis of lichen planus are very less explored. The immunological ground being one of the priority mechanisms in the multicausal pathogenesis of oral lichen planus, immunoglobulins and autoantibodies play the key roles.

Literature reports differ to confirm and justify because many studies on the humoral response of the disease, have observed increased, reduced or even normal immunoglobulin fractions. Total protein and haemoglobin can be considered as broad indicators of anemia and status of nutrition. The relation between the nutritional status and lichen planus is insignificant is yet to be established.6 The present study is done to estimate and evaluate the role of immunoglobulins IgG and IgA in etiopathogenesis of the disease, to assess the clinical utility of salivary and serum IgG and IgA levels as prognostic markers of the disease and to establish any correlation between the nutritional status and the disease progression.

METHODS

The present study is carried out in the Department of Oral and Maxillofacial Pathology, MNR Dental College and Hospital and Care Hospital and Research Centre, Banjarahills, Hyderabad. Approval of the institutional ethical committee board is taken before pursuing the research project.

Subjects

Followed by informed consent 20 patients diagnosed with Lichen planus are enrolled in the study from the Department of Oral and Maxillofacial Pathology, MNR Dental College and Hospital, Sangareddy. The study is conducted from June 2023. Enrolled 20 controls are matched with the age and sex of the patients.

Inclusion criteria

Patients, physically healthy and well oriented in time space and as a person, clinically and histopathologically diagnosed with oral lichen planus, had symptoms i.e. pain and/or burning sensation secondary to oral lichen planus, not on any treatment for the same (in case they are, receiving such treatment is stopped and a washout period of two weeks is given), who agreed to give saliva and blood samples were included.

Exclusion criteria

Patients, suffering from any systemic disease/s like diabetes, hypertension, cardiovascular system disease, renal dysfunction, and liver disorders, with any other mucosal disease, recent infections or any other skin disease which may be associated with oral lesions, on any drug therapy which may cause lichen planus like lesions, with severe caries and periodontitis, with findings of any physical or mental abnormality, which would interfere with or be affected by the study procedure were excluded.

Sample collection

5 ml of blood is collected by venepuncture using 24-gauge needle 2 ml is used for hemoglobin routine and total protein estimation, serum is extracted from 3 ml of whole blood using centrifugation at 2500 rpm for 10-15 min. Saliva is collected from the patients after they rinsed their mouth with distilled water. Later, the patient is asked to incline in head forward position. 2 ml of mixed unstimulated saliva pooled in the mouth is collected and centrifuged at 2500 rpm for 10 min. The supernatant is used for estimation.

Experimental methods

A new particle-enhanced turbidimetric immunoassay using mono-specific sheep antibodies on the SPAPLUS® analyzer is used to determine the soluble antigen concentration. (IgG, IgA). The procedure is followed

according to the manufactures protocol for measuring IgG and IgA on the two analyzers. The total protein in the samples is estimated using biuret test.

Statistical analysis

All the analysis is done using statistical package for the social sciences (SPSS) version 18. A p value of <0.05 is considered statistically significant. Comparison of mean values between the groups is done independent sample t-test. Correlations are done using Pearson correlation coefficient.

RESULTS

This study is conducted on total of 20 patients who are divided into two groups of 20 each. Group A includes patients with oral lichen planus. Group B include healthy controls. Age and sex matched individuals are taken for the study.

Table 1 shows the comparison of mean, standard deviation and p value of protein, haemoglobin, IgG and IgA concentration levels in the serum samples of controls and tests (patients). The table also compares the protein, IgG and IgA concentration levels in saliva samples.

Table 1: Comparison table of salivary and serum IgG and IgA, total protein and Hb levels, between test and controls.

Groups	Group A (control)		Group B (test)		P value
	Mean	SD	Mean	SD	
Protein serum	6.93	0.67	6.55	0.79	<0.001; NS
HB serum	11.06	1.51	11.08	1.54	<0.001; NS
IgG serum	1096.21	167.83	1455.25	183.98	<0.001; Sig
IgA serum	147.68	50.85	397.85	150.14	<0.001; Sig
Protein saliva	1.34	0.24	1.43	0.23	0.207; NS
IgG saliva	2.04	0.67	2.73	1.11	0.025; Sig
IgA saliva	14.34	2.69	20.38	3.48	<0.001; Sig

As illustrated in Figure 1 the mean serum IgG levels in lichen planus patients is 1455.25 mg/dl and in that of controls it is 1096.21mg/dl. Figure 2 shows the mean salivary IgA levels in lichen planus patients is 20.38 mg/dl and in that of controls it is 14.34 mg/dl. In the present study mean serum protein is 6.93 mg/dl in test samples when compared to controls which is 6.55 mg/dl.

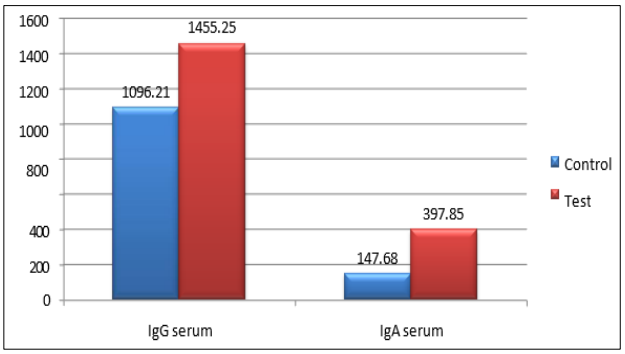

Figure 1: Comparison between serum IgG and IgA levels.

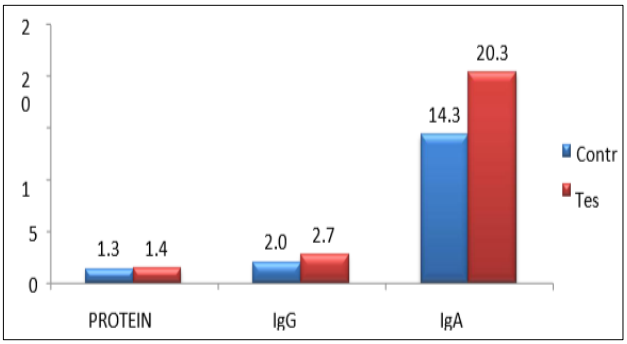

Figure 2: Comparison between salivary IgG, IgA, Protein levels.

Table 2 shows the comparison of mean, standard deviation and p values of IgG and IgA concentration levels in the serum and saliva samples of erosive and reticular lichen planus. Figure 3 shows the mean value of IgG serum is 1676.50 mg/dl in erosive lichen planus which is significantly higher when compared to reticular lichen planus 1372.77 mg/dl (<0.001; sig).

Table 2: Comparison between Erosive and Reticular lichen planus.

Groups	Erosive		Reticular		P value
	Mean	SD	Mean	SD	
IgG serum	1676.50	66.58	1372.77	120.31	<0.001; sig
IgA serum	572.17	77.03	309.62	93.49	<0.001; Sig
IgG saliva	3.25	1.30	2.54	1.02	0.21; NS
IgA saliva	22.98	2.78	19.45	3.25	0.035; Sig

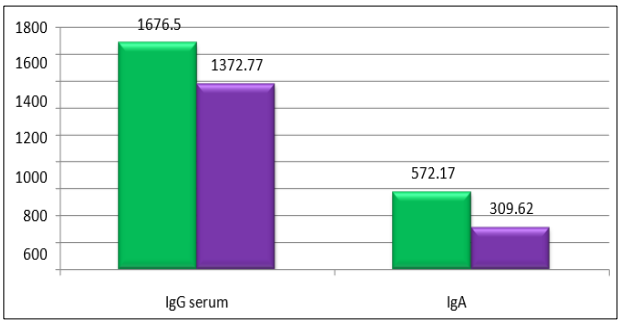

Figure 3: Comparison between serum IgG and IgA levels of erosive and reticular lichen planus.

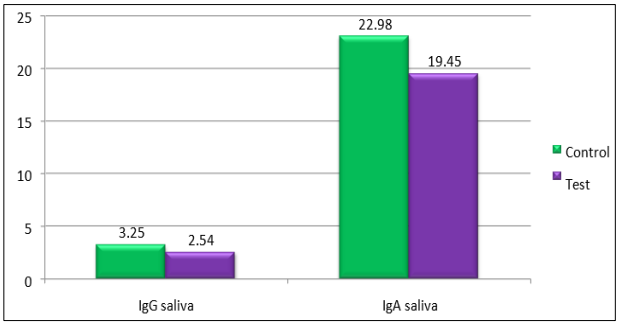

Figure 4: Comparison between salivary IgG and IgA levels of erosive and reticular lichen planus.

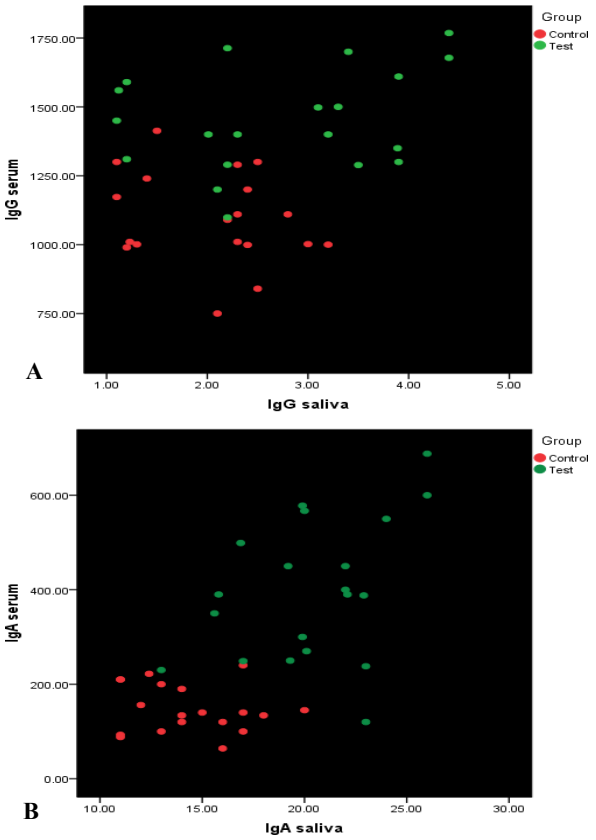

Figure 5 (A and B): Correlation between salivary and serum IgG and IgA levels.

Figure 4 illustrates the mean value of IgG saliva is 3.25 mg/dl in erosive lichen planus which is not significant when compared to reticular lichen planus 2.54 mg/dl (<0.21; NS). Mean salivary IgA values in erosive lichen planus 22.98 mg/dl are found significant when compared to reticular lichen planus 309.62 mg/dl (0.035; sig). Figure 5 shows the correlation between salivary and serum IgG and IgA levels.

DISCUSSION

In the current study 40 subjects are enrolled; 20 patients are diagnosed with lichen planus and 20 controls to evaluate IgG and IgA in serum and saliva in patients and controls and compare with total protein and haemoglobin levels. In normal serum immunoglobulin G is found in 70-75%, followed by IgA accounting for 15-20%.[6] Where as in saliva, the level of immunoglobulin G is reduced and reaches a maximum 2-3 mg/dl, and the IgA level is increased and is around 10-20 mg/dl.[7] The serum immunoglobulins are assayed in various cancers such as cervical cancer, prostate cancer and nasopharyngeal carcinoma.[8-10]

From the results it is found that the mean serum IgG and IgA levels in lichen planus patients is higher when compared with controls. There is a significant difference in the mean IgG and IgA levels levels in both the study groups (<0.001; sig).

Gandolfo et al suggested that elevated serum level of IgG may represent a secondary oral infection during mucosal erosion, while Lundstrom et al suggested that it might represent a continous autogenous production of soluble antigens.[11,12] Rabinovich et al concluded that the role of humoral immunity in the pathogenesis of OLP is probably secondary to the cell mediated reaction against basal keratinocytes.[13] Similarly, study conducted by Sun et al showed a significant increase in the level of serum IgG in patients with oral lichen planus.[14] A study by Albanidou-Farmaki et al attempted to estimate the participation of humoral immunity in the pathogenesis of the disease, comparing the levels of serum immunoglobulins IgG and IgA levels.[15] Lukenda et al in their study reported elevated levels of serum IgA and IgM show that humoral immunity is implicated in the pathogenesis of OLR, where significantly elevated serum IgA and IgM indicate emergence of defense mechanisms starting from the local level and suggest that this immunological mechanism is attempt to eliminate bacterial antigen as possible etiological factor in the development of Oral Lichen planus.[16] It is concluded that this could be a result of changed humoral immune reaction as a consequence of possible immunodeficiency associated with hypogamma-globulinemia. Obtained differences between studies might reflect the fact that the samples were taken in the different stages of the disease.

Another study conducted by of Bharadwaj et al estimated IgA levels in blood of normal healthy volunteers and

patients with lichen planus. It was evident that in all patients of Lichen planus, the serum IgA level ranged between 0.51-1.88 gm. which was significantly high, when compared to controls.[17] Another study by Popovska et al revealed a significant decrease of IgA in serum during exacerbation.[18] Present study results are in coordance with the findings of Rajalalitha et al, Remani et al, and Stankler et al.[19-21] Regarding this, Sklavounou et al pointed out a significant reduction of all immunoglobulin fractions, including IgA in serum.[22] Examining a quite small number of cases, Stankler et al concluded that there was a significant lowering in the levels of both IgA and IgM.[21]

The current study results also showed that the mean salivary IgG and IgA levels in lichen planus patients is elevated compared to controls. A significant difference is found in the mean IgG and IgA levels in patients and controls (<0.001; sig).

The results are in accordance with Ghaleyani et al confirmed the level of salivary IgA and IgG in oral lichen planus and oral lichenoid reaction patients is higher than healthy controls.[23]

The role of salivary immunoglobulins in the immunopathogenesis and the clinical course of oral mucosal diseases, have not been yet studied comprehensively. Femiano et al as well as others have proposed that the key event in the pathogenesis of OLP is a surface antigen change on the basal keratinocytes, which is not recognized by one's own immune system and consequently starts a chain reaction which finishes in the destruction of the keratinocytes themselves. Serum immunoglobulin studies and tissue analyses utilizing immunofluorescent techniques, monoclonal antibodies, and immunochemistry have implicated immunological dysfunction in the pathogenesis of lichen planus.[24] Serum IgG levels might be significantly elevated in the HCV which elucidates a local humoral immune response. However, simultaneous determination of IgG in saliva and sera was found to be helpful. IgG found in saliva has been reported to originate mainly from serum and is transported by passive transmucosal diffusion.[25] At this point we can assume that a systemic immune response conducted mainly through IgG is triggered in patients with OLP. Some authors also postulated that changes in IgG might reflect disease activity, such as acute stage versus remission period as it was seen in bullous pemphigoid patients.[25] However, increase in salivary IgA levels in our present study suggested that changes in IgA might result from different antigenous stimulation which reflect microbial stimulation seen in acute phase in comparison to the remission period.

Guptha et al estimated salivary IgA levels are increased in precancerous lesions, when compared to controls.[26] Popovska, et al estimated values of IgA in serum in oral erosive lichen planus during exacerbation showed decreased levels and during remission increased levels in comparison with the control group, resulting in both cases in highly significant differences. Circulating immune complexes values in exacerbation and remission were increased. C3 values were slightly increased in both examined phases in our groups of examinees and controls. Salivary IgA levels in oral erosive lichen planus during exacerbation and remission phases varied identically as in serum. Changes in the IgA levels on all examined media, as well as of CIC and component C3, may correlate with the changes of oral mucosa emphasizing the role of humoral immune response in the pathogenesis of oral lichen planus.[27] In our study there is significant increase in salivary IgG and IgA levels in the patients when compared to healthy controls which implicate the important role of salivary immunoglobulins in the disease pathogenesis.

In the present study mean serum protein is slightly higher in patients than controls which is 6.55 and is found to be insignificant statistically (<0.001; NS).

In the current study the inter group comparison is done on salivary samples of reticular and erosive lichen planus. It is found that the mean values of IgG are elevated in erosive lichen planus and found to insignificant when compared to reticular lichen planus (<0.21; NS). It is also noted that the mean of salivary IgA values in erosive lichen planus is less when compared to reticular lichen planus and it is significant (0.035; sig).

The results were found consistent with that of Gandolfo et al who estimated Humoral immunological parameters in Italian patients with oral lichen planus erosive forms and with that of reticular forms. increased levels of serum IgG approaching the statistical significance are found.[15] Sun et al confirmed concentration In erosive lichen planus, serum IgM were elevated as compared with healthy subjects when compared to patients with the non- erosive type, which are consistent with our study results.[14] Lucak et al also confirmed that the differences in the serum of desmoglein autoantibodies suggested that pathological mechanisms in erosive and reticular forms of oral lichen planus might not be the same.[28]

All the studies along with our present study indicate that erosive type of lichen planus shows increased levels of salivary and serum IgG and IgA levels. This indicates that salivary and serum IgG and IgA can be used as prognostic markers of disease progression. The concept that humoral immunity is of crucial importance in the pathogenic sense has been supported by the latest investigations and knowledge that speak in favour of the fact that B-cells are found in infiltrates and these are precursors of cell creating antibodies. However, Yanossy et al identified only a minimum synthesis of the immunoglobulins in the biopsy specimens compared to other diseases, but, on the other hand, lymphocytes and histiocytes were predominant.[29]

As the pathogenesis of oral lichen planus is still unknown. During recent years, it has become more evident that the immune system has the primary role in the development of this disease. The incidence of malignant transformation of

lichen planus is low and presumably does not exceed an incidence of 0.2% per year. The atypical forms atrophic, erosive, "in plaque" had higher risk for malignant transformation than the reticular form.

Rai et al detected high levels of circulating immune complexes in leukoplakia followed by lichen planus and then oral submucous fibrosis. Thus, the high value of mean CIC levels in leukoplakia and lichen planus may point to the risk of their malignant potential.[30] Studies by Kohli et al have shown significant elevation of IgG in pretreatment cancer stages.[31] The elevation of IgG levels in oral cancer patients may be due to dependencies of IgG on the intensity of the antigenic stimulation and functional capacity of the antibody producing mechanism. Rai et al also stated that the pretreatment levels of circulatory immune complexes can be used as a prognostic indicator. A high circulatory immune complexes level could probably indicate a poor prognosis.[30]

The present study is conducted on a limited study group and it is single-centered. In future the study can include a larger sample size and a multi-centered study for validation of the obtained results.

CONCLUSION

In conclusion, the present study proved to be consistent in not only augmenting the already existing hypothesis but also imparting new concepts of hypothesis. The results might conclude that elevated levels of serum and salivary IgG and IgA show that humoral immunity is implicated in the pathogenesis of oral lichen planus. Further studies with more number of cases could throw light on the etiology and pathogenesis of the disease. Possibly, understanding the role of humoral immunity and circulatory immunoglobulin levels can be helpful in early detection of a lesion. More research will be required to determine their role.

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