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Evaluation of multifactorial nature of oral lichen planus by assessment of biochemical profiles: A Case control study

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Abstract

Background: Oral Lichen planus (OLP) is a common chronic mucocutaneous and autoimmune disease.The etiology of OLP is considered to be multifactorial and complicated. Since, many researchers in earlier studies have evaluated one or very few parameters associated with OLP.Hence, this study is intended to make an attempt to inquire into the possible correlation of OLP with alterations in various serum profile patterns and other parameters associated with OLP along with critical analysis of the results.

Aim: To evaluate the association of profiles related to Hematology, Blood glucose , Lipids, Liver & Renal function , Thyroid , Ultra sound abdomen,blood groups, viral infections like hepatitis B, C and HIV in OLP.

Materials and Methods: In this randomized comparative case control study, 80 patients comprising 40 cases of clinically and histopathologically diagnosed OLP and 40 age- and sex-matched healthy controls in the age group of 21-60 yrs that reported to the department of Oral Medicine and Radiology; PMVIDS & RC, Hyderabad were enrolled for the study and then subjected for laboratory evaluations**.** The obtained data were compared with standard values to assess any alterations using statistical analysis Chi square test, Independent sample t test and Fisher's test to measure association between two nominal variables. A p- value ≤ 0.05 was considered statistically significant.

Results: The mean value of total WBC count,ESR, Monocytes,Basophils ,TG, SGOT, SGPT, ALP indicated

statistically significant elevation in cases compared to controls. No significant relationship was found with blood grouping and Rh typing,viral infections, blood glucose, serum proteins, complete urine examination & Ultrasound abdomen findings.

Conclusions: The current study revealed few evident significant and insignificant correlation of parameters in our population in accordance with previous studies.We emphasize OLP is basically an autoimmune disorder with minimal systemic effects. The results may vary when done at a larger scale and longer duration due to genetic and demographic variability. We recommend imminent studies on a larger population to additionally substantiate & confirm its causal association.

Keywords: Blood Glucose; Blood Groups; Hematology; Hepatitis B & C; HIV; Oral Lichen Planus; Lipids; Liver &Renal Function; Thyroid; Ultra Sound Abdomen.

Introduction

Lichen Planus (LP) is a relatively common chronic inflammatory mucocutaneous disorder which may affect the skin and/or the oral mucosa. [1] OLP is clinically classified as Reticular, Atrophic, Erosive and Bullous.[2] Although the exact cause of OLP is ambiguous, experimental evidence suggests that it is an inflammatory T-cell mediated immune response.[3] The prevalence rate may differ among races and geographic areas.[4] OLP has multifactorial pathogenesis, previous studies in the literature showed association and no association of single systemic biochemical parameter with OLP. A number of studies have assessed the association of LP with liver complaints and with known etiological factors of liver diseases. [5] The hypothesis of viral etiological agent has gained association of the hepatotrophic viruses namely Hepatitis B and C viruses with LP in the past one decade. Only one study in the mediterranean population showed the absence of OLP in HIV-co infected patients suggesting immunosuppression secondary to defective CD4 functions.[6] There are controversial studies on association of the ABO blood grouping with OLP which are inconclusive.[7] A pathogenetic link may exist between dyslipidemia , cardiovascular (CV) risk due to chronic systemic inflammation and OLP.[8]Furthermore, studies have demonstrated the prevalence of hematological abnormalities in the erosive OLP group which was greater than in the non-erosive group .[9] It has been found that diabetes patients are associated with dermal and oral lesions of LP.The association between OLP and thyroid diseases/ thyroid medication, in particular hypothyroidism, have been reported in some studies.[10]

Till date to the best of my knowledge, no study was conducted which showed multiple systemic biochemical parameters correlation with OLP in the Hyderabad population. So, keeping in view the different controversies surrounding OLP and systemic diseases, we carried out investigations on a group of patients and compared the biochemical alterations with controls to investigate its association. Hence, this work presents novelty in its depth of analysis of the epidemiology of this disease to evaluate the systemic biochemical alterations with OLP.

Materials and Methods

The study was carried out at a single institution & laboratory diagnostic centre to compare the association of altered serum profiles related to Hematology, Lipid, Liver and Renal, Glucose levels, Thyroid , blood groups, Viral infections like Hepatitis B, C and HIV patterns along with Urine & US abdomen analysis.The Institutional Ethical Committee approved the study. The criteria for the case and control group selection were as follows.

Inclusion criteria

- Age group of 21-60 yrs

- Clinically and histopathologically diagnosed OLP (cases)
- Apparently healthy oral mucosa (controls)
- Signed informed consent

Exclusion criteria

- Pregnant or lactating females
- Systemic disease
- Oral lesions in resemblance with OLP such as contact allergy, lichenoid reaction
- Use of steroid, immunomodulators and on therapy for OLP.

Screening and Diagnostic examinations

A total of 80 patients were screened out in the age group of 21-60 yrs were enrolled in the study and classified into two groups by a randomized simple approach:

1. Cases - 40 individuals with clinically and histopathologically diagnosed OLP

2. Controls - 40 age- and sex-matched individuals with apparently healthy oral mucosa.

The demographic details, clinical findings and complete medical history was recorded using a specially designed proforma and then advised for serological investigations and ultrasound abdomen examination following which symptomatic cases were managed by conventional therapy. Figures 1 and 2 were the clinical photographs of the cases selected.

Venous blood samples were collected from the patients in the case and control group for the assessment of individual serum profile levels. The values were subsequently recorded in the respective pro forma. The obtained data were compared to standard values as follows:[11]

Complete blood picture Total RBC Count: 3.5-5.5 mill/cumm; Total WBC Count: 4,000-11000cells/cumm; Platelet count: 1.5-4 lakhs/cumm; Polymorphs: 40-70%; Lymphocytes: 20-40%; Eosinophils: 1-6%; Monocytes: 2-10%; Basophils: 0-1%, **Hemoglobin percentage:** Male- 12-16 GM%, Female- 11-15 GM%.**ESR** Male: 0-13MM, Female: 0-20MM**,ABO Blood Grouping and RH Typing :**A Positive/Negative, B Positive/ Negative, O Positive/ Negative, **Random Blood Glucose :**70 - 140 mg/dl, **Renalparameters:**SerumCreatinine(CRE)0.6–1.5MG/DL,

Serum Urea (BUN) 12.6 – 42.6 MG/DL, Serum Uric acid in Male – 3.4 – 7.0 mg/dl; Female – 2.5- 5.7 mg/dl, **Hepatic parameters:** Serum glutamic oxaloacetic transaminase (SGOT) 10 – 40 IU/L,Serum Glutamate Pyruvate Transaminase (SGPT) 10 – 40 IU/L, Serum total bilirubin content upto 1 MG/DL,Direct and indirect bilirubin upto 0.5 mg/dl, ALP in Male : 50 - 126 IU/L; Female : 37-103 IU/L,Total Protein 6.0 – 8.0 gm%,Albumin3.7 – 5.3 gm%, **Lipid parameters**:Triglycerides Less than 160 MG/DL **,**Total Cholesterol upto 200 MG/DL,Heavy Density Lipoproteins-Cholesterol (HDL- C) 30 - 60 MG/DL**,** Low Density Lipoproteins-Cholesterol (LDL-C) Less than 130 MG/DL, **Thyroid profile (T3 , T4, TSH)** in (µIU/ml) Low normal range 0.42; High normal range 5.45; Normal range 0.35-5.5 ; T3 : Normal value- 40-181ng/dl;T4:Normal value-5.5-11ug/dl,**Viral infections** Human immune deficiency virus (HIV-1 & HIV-2) & Hepatitis C virus by TRI- DOT & Rapid Visual Test, Hepatitis B virus (HBsAg) by One Step Rapid Visual Test HEPACARD,**Complete Urine Examination** by Urine test strip or Dipstick test for the presence of proteins, glucose, ketones, acetone,hemoglobin, bilirubin, urobilinogen, nitrite and leucocytes as well as testing of pH and specific gravity,**Ultrasound Abdomen** was done using the Philips HD11 XE Ultrasound System, Abdominal 2- 8 Mhz curved array 3D/ volume transducer /probes, High Resolution non-interlaced flat CRT (HD11XE employs an LCD monitor), 4 active probe

ports. Tissues that do not produce signals, such as fluid-filled cysts, are said to be anechoic and appear black. Tissues that produce a weak signal are hypoechoic, whereas tissues that produce intense signals such as are hyperechoic and appear bright.

Statistical analysis

The data were tabulated in Microsoft Excel and subjected to statistical analysis using SPSS Statistics (version 22.0, SPSS Inc.,). Descriptive statistical procedures such as means, standard deviations, medians, minimum, maximum, and percentages were used to summarize all variables. Chi square test, Independent sample t test and Fisher's test was procured to measure the association between two nominal variables. $P <$ 0.001* was considered statistically significant.

Results

In the study group of 80 participants; the statistical comparison between study and control group showed equal gender distribution with equal frequency of 20 (Males=Females) with Chi square value=0.0, p value=1.0 was found to be non significant [Table I].The mean age and standard deviation of 43.78 +/- 9.8 in study group and 43.50 yrs +/- 11.7 in control group with no significant difference (t=0.00, p value=1.0)[Table II & Graph I].

Complete blood picture revealed the mean value in the study group compared to control group in relation to total WBC count & Monocytes (p value <0.001*) , ESR & Basophils (p value <0.05*) showed statistically significant elevation where as Heamoglobin, total RBC count, Platelet count, Polymorphs, Eosinophils, Lymphocytes count did not show any significant difference (Table III & Graph II, III, IV, V, VI, VII).**Blood grouping and Rh typing** showed that a total of 31.3% were O positive, 2.5% were O negative, 23.8% were A positive **,** 3.8% were A negative and AB positive, 35 % were B positive with Fisher's exact value= 5.83, p= 0.31 found no statistically significant difference (Table IV & Graph VIII, IX).**Viral infections (HIV1&2, HBSAG & HCV)** found no association of viral infections. Only one patient was HBsAg positive (Table V).**Liver function tests** showed the mean value in the study group compared to control group in relation to SGOT (p value 0.02*), SGPT (p value 0.01*), Alkaline Phosphatase (p value <0.001*) indicating statistically significant elevation where as Total bilirubin, Direct and Indirect bilirubin were statistically insignificant (Table VI & GraphX, XI).**Random blood Glucose** showed no statistically significant difference between both the groups (p value 0.46) (Table VII & Graph XII). The total proteins (p value 0.33), Serum albumin (p value 0.90), Serum globulin (p value 0.90) and A.G. ratio (p value 0.11) showed no statistically significant difference between both the groups (Table VIII & Graph XIII, XIV).**Serum lipid profile** showed that the mean value in the study group compared to control group with Trigylcerides (p value 0.01*), Total Cholesterol (p value 0.009*), HDL Cholesterol (p value <0.001*), LDL Cholesterol(p value 0.004*) indicated statistically significant elevation where as VLDL Cholesterol & CHOL/HDL ratio showed no statistically significant difference (Table IX & Graph XV, XVI).**Thyroid function tests (T3, T4, TSH)** showed that the mean value in relation to T3 (p value 0.004*) was statistically significant elevation in the control group when compared to study group where as T4 and TSH showed no significance (Table X & Graphs XVII, XVIII, XIX).**Renal function tests** showed the mean value of blood urea (p value 0.01*) was significantly elevated in the control group when compared to study group where as Serum Creatinine & Uric acid showed no statistical significance (Table XI & Graph XX).**Complete urine analysis** revealed straw colored urine in only 1 patient, 2 patients showed turbid urine appearance in both groups and 1

patient showed positive urine Albumin and 7 showed trace amounts in the study group compared to control group. Urine pus cells and Epithelial cells were analysed by Fisher's test where 95% showed urine pus cells less than 10 and 5% showed greater than 10 (p value 0.12 NS).97.5% Urine Epithelial cells were less than 20 and 2.5% were greater than 20 (p value 1.00 NS) and Urine specific gravity (p value 0.44 NS) found no statistically significant difference between both the groups (Table XII, XIII, XIV).**Ultrasound scanning of whole abdomen findings** revealed out of 40 patients in the study group, 2.5% showed Anterior wall uterine fibroid, 2.5% showed Bilateral small renal calculus, 2.5% showed Bulky uterus with small endometrium & simple small left ovarian cyst, 2.5 % showed renal calculi, 2.5 % showed small right renal cortical cysts, 7.5% showed Bulky uterus with too small anterior wall fibroids,30% showed Grade I fatty liver. In comparison with the control group, 73.75% patients showed no abnormality where as 26.25% patients showed abnormality indicating statistically insignificant findings (Table XV,XVI & Graph XXI).

Discussion

OLP is a T-cell-mediated, chronic inflammatory oral mucosal disease of unknown etiology. Several factors have been proposed contributing to etiology including genetic background, dental materials, drugs, infectious agents, autoimmunity, immunodeficiency, food allergies, Anxiety & stress, habits, trauma, diabetes, hypertension, malignant neoplasm, and bowel disease.[12] Antigen-specific and nonspecific mechanisms play a role in its pathogenesis, leading to T-cell accumulation in superficial lamina propria, intraepithelial T-cell migration, and keratinocyte apoptosis of the basal cells of the oral epithelium in OLP. [13] Very few studies have shown this premalignant disorder association with blood groups. It is important to look for any impairment in liver function tests in cases of OLP as elevation of transaminase levels was reported in many studies conducted earlier.[14] Few studies have shown a pathogenetic link with dyslipidemia, hypothyroidism and hematological alterations in OLP.[15] Gupta *et al* [13] reported that the exact incidence and prevalence of LP is unknown which is most often been reported in middle aged patients with 30 to 60 years of age and is more common in females than in males. Similarly, the present study showed that the occurrence of OLP was more in 30-50 years age group with women in their fifties are most common reporters of the disease. Abhishek J *et al* [14] conducted a cross-sectional and observational study to evaluate altered hematological profile in a total of 22,252, of them 147 were suffering from OLP in South Indian (Kerala) population. They found that total WBC count, neutrophil and lymphocyte counts are significantly greater in the OLP patients and the difference in the ESR values of the female OLP patients with the normal females is highly significant ($p<0.0001$). The increase in eosinophil count in the adult males aged 30-60 years is significantly high compared to such non-diseased males and the decrease in hemoglobin level in the OLP patients do not vary to a significant level.High WBC count, Monocytes, Basophils are accounted by the immune system disorder or inflammatory disorders or acute stress or infectious diseases, involved in the etiopathogenesis of OLP. Elevated ESR can prevail in autoimmune disorders. Comparatively our study showed that the total WBC,ESR, Monocytes, Basophils indicating statistically significant elevation in the study group compared to control group where as Hemoglobin, total RBC count, Platelet count, Polymorphs, Eosinophils, Lymphocytes count were insignificant. Arshiya S *et a l*[15] reported that the incidence of diabetes mellitus was 10% (5 of 50) which is far less compared to that of 62% (13 of 21) of Powell S.M. *et al*[15] , 42% (17 of 40) of Lowe N.J. *et al*[15] in OLP.

Nosratzehi *et al*[16] reported that diabetes mellitus does not have a direct role in the OLP etiology but could be contributing to oral lichen planus like lesions in oral cavity as a result of various medications . In accordance the present study suggested no association of Diabetes mellitus in our population.

Not many studies have been carried out to test the association between OLP and thyroid dysfunction. Manzoor *et al* [17]reported that thyroid function tests were deranged in 7 (14%); 4 (8%) females and 3 (6%) males while in the control group, thyroid function tests were deranged in 1(2%) female and significant percentage of lichen planus patients have deranged thyroid function especially hypothyroidism (T3↓,T4↓, TSH↑). The authors suggested that the association of OLP and hypothyroidism could be linked to a similar, but still unknown, immune-mediated mechanism. In contrast our study showed that the mean value of T3 levels was statistically significant elevation in the control group when compared to study group indicating no deranged thyroid function status in OLP cases. Kumar *et al* [7] reported that blood group A had 1.28 times higher risk of developing OLP with significant female predilection in third and fourth decade of life followed by AB, B &O. Thus, ABO blood grouping can be used as an adjunct in the diagnosis of OLP. They explained the fact that blood group antigens, in addition to being present on red blood cell membranes, are also found on epithelial cells of various other tissues, including the oral mucosa. H antigen is a blood group antigen present in all the individuals irrespective of blood group types. It is the precursor for the formation of A and B antigens. In people belonging to A and B blood groups, the precursor H antigen is converted to A and B antigen, respectively, whereas in O blood group individuals, it remains in the original forms. People with O blood group have the highest amount of H antigen,which affords protection against OLP. Maryam *et al* [18] revealed that there is no statistically significant relationship between ABO blood groups, Rh system and oral lichen planus disease, and hence are not risk factor for oral lichen planus. In accordance, the present study showed no statistically significant difference and relationship between ABO blood groups, Rh system and OLP in comparison with control group. The possible explanation for this could be that expression of histo-blood-group antigens in normal human tissues is dependent on the type of differentiation of the epithelium and are expressed in a highly regulated way that correlates with the pattern of epithelial differentiation and with cell maturation. Hence their expression may vary in different geographic locations and racial groups.

Chakraborti G *et al* [19] reported that Serum Uric Acid levels were significantly decreased in patients with respect to controls in skin LP cases which was the only study reported in the literature. According to previous studies it was hypothesized that serum uric acid has an antioxidant defense mechanism and scavenger of reactive oxygen species. Auto immune disorders can affect many organs and tissues in the body among which majorly involved organs are heart, liver, kidney, lung and skin. Since, there was no previously reported literature showing association, the present study was an attempt to rule out correlation of renal function abnormalities with OLP where the mean value of blood urea ,Serum Creatinine and Uric acid showed insignificant association.

Dreiher *et al*[20] reported that the prevalence of dyslipidemia was significantly higher in patients with LP. Amer *et al*[21] reported no association of dyslipidemia in LP patients. It has been hypothesized that the association between OLP and cardiovascular (CV) risk is due to chronic systemic inflammation has a important role in dyslipidemia which also constitutes a risk factor for

atherosclerosis. Cytokines such as TNF alpha, IL 6,10,4 involved in LP pathogenesis could explain the association of dyslipidemia as chronic inflammation has been suggested.[9] In accordance, the present study showed mild dyslipidemia. Konidena A *et al* [22] reported that their results did not differ significantly in liver function tests. Bhattacharya *et al* [23] reported that levels of serum bilirubin, SGOT and SGPT were not significantly elevated in either the patients or controls. Statistically insignificant difference found with altered levels of serum proteins, albumin and globulin levels as compared to controls concluding negative association between LP and chronic liver disease. Similarly, the present study, showed that the total proteins, Serum albumin, Serum A.G. ratio,Total bilirubin, Direct and Indirect bilirubin indicated no statistically significant difference whereas SGOT, SGPT, Alkaline Phosphatase indicated statistically significant elevation in the study group when compared to control group. Although the pathogenesis of LP still remains unknown, abnormalities in both humoral and cellular immune mechanisms may play an important role. Elevated transaminases and alkaline phosphatases could be attributed to immunological responses in OLP.

Gerayli S *et al* [24] reported non-significant relationship exists between OLP and hepatitis C. Shengyuan *et al* [25] reported that hepatitis C virus infection is associated with a statistically significant risk for development of LP and may be used as a predictive marker in certain geographical regions. Viral infections are usually indolent so that patients may present only in late stages of the disease with serious complications like cirrhosis and chronic liver disease. Hypothesis is that the virus is capable of duplication, development and proliferation in oral epithelium which in turn raises autoimmune reactions and contributes to emergence of OLP in oral cavity.The putative pathogenetic link between OLP and HCV still remains controversial and needs a lot of prospective and interventional studies for a better understanding. Similar to the many previous studies the present study found non-significant relationship exists between OLP and Hepatitis B, C, HIV and only one patient was HBsAg positive. The negative association of viral infections with OLP could be attributed to endemicity in varied geographical regions. Furthermore, studies in patients with viral infections are required to confirm its association.

Though complete urine analysis were done in few studies as routine screening but no correlation was made with OLP. Hence, the present study was carried out to check even biochemical changes in urine in OLP patients and it showed insignificant association. Thus could be attributed to the cell mediated immunopathogenesis of OLP contributing to expression of unknown antigens.No studies were reported in the literature showing correlation of ultrasound abnormalities with OLP. Hence, the present study was an attempt to rule out any organic abnormalities apart from biochemical changes of liver, kidney, bladder, pancreas, uterus, prostrate which could have an association.Early diagnosis and education of the complications associated with such abnormalities can prevent major comorbidities. It is hypothesized that OLP is an auto immune disorder and mucosal LP can affect any organ through mucosal surfaces and lining of gastro intestinal tract, peritoneum, genitals and bladder.[26]With respect to ultrasound abdomen scanning, the present study found that there were greater percentage of patients with Grade I fatty liver, all the ultrasound abdominal findings were statiscally insignificant in comparison with control group.

Conclusions

At the end of the study, the results showed that there was significant elevation of few hematological parameters such as total WBC count, ESR, monocytes and basophils,

lipid parameter such as only triglyceride levels of lipid and few parameters of liver function tests such as SGOT, SGPT and Alkaline phosphatase. There was insignificant association of viral infections, blood groups & Rh typing, blood glucose levels, serum proteins, thyroid function tests, renal function tests, complete urine examination and Ultra sound abdomen findings in our population. Nevertheless, our study does not show an increased incidence of clinical or biochemical evidence of systemic abnormalities in patients with OLP. Hence, the etiology of OLP is obscure and is not significantly associated with systemic biochemical alterations unlike other autoimmune disorders which have varied systemic effects. The controversy surrounding the association between OLP and systemic biochemical abnormalities is nevertheless fascinating. However, further research, recruitment of a large cohort of patients from wider background and for longer duration may substantiate to confirm its causal association.

Abbreviations

CRE : Creatinine; CHOD : Cholesterol Oxidase; ESR : Erythrocyte Sedimentation Rate; GLDH : Glutamate Dehydrogenase; HCV : Hepatitis C Virus; HIV : Human Immunodeficiency Virus; OLP : Oral Lichen Planus; Hb : Haemoglobin; HDL-C : High Density Lipoproteins Cholesterol; LDL-C : Low Density Lipoproteins Cholesterol; RBC : Red Blood Cells; Rh : Rhesus; SGOT : Serum Glutamic-Oxaloacetic Transaminase; SGPT : Serum Glutamic-Pyruvic Transaminase; PAP : Phenol+Aminophenazone; TGL : Triglycerides; T3 : Tri Iodo Thyronine; T4: Thyroxin; TSH : Thyroid Stimulating Hormone; WBC : White Blood Cells; VLDL : Very Low Density Lipoproteins

References

1. Amerikanou CP, Markopoulos AK, Belazi M, Karamitsos D, Papanayotou P. Prevalence of oral lichen planus in diabetes mellitus according to the type of diabetes. Oral Disease.1998;4:37-40.
2. Burket's. Oral medicine text book; Eleventh edition, 2008; BC Decker Inc; Hamilton.
3. Roopashree MR, Gondhalekar RV, Shashikanth MC, George J, Thippeswamy SH, Shukla A. Pathogenesis of oral lichen planus – a review. J Oral Pathol Med. 2010 Nov;39(10):729-34.
4. Vas dis ML, Parks ET. Prevalence of oral lichen planus in patients with diabetes mellitus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:700.
5. Anbar TE, Barakat M, Ghannam SF. A clinical and epidemiological study of lichen planus among Egyptians of al-Minya province. Dermatol Online J 2005; 11: 4.
6. Carrozzo M, Gandolfo S, Carbone M et al. Hepatitis C virus infection in Italian patients with oral lichen planus: a prospective case–control study. J Oral Pathol Med 1996; 25: 527–533.
7. Kumar T, Puri G, Laller S, Bansal T, Malik M. Association of ABO blood grouping with Oral lichen planus. Univ Res J Dent 2014;4: 93-6.
8. Lopez-Jornet P, Camacho-Alonso F and Rodríguez-Martínes MA. Alterations in Serum Lipid Profile Patterns in Oral Lichen Planus: A Cross-Sectional Study. Am J Clin Dermatol.2012 ;13 (6): 399-404.
9. Narayan V, Gnanasundaram N, Arvind M. Prevalence of Oral Lichen Planus in Patients with Diabetes Mellitus. J Indian Acad Oral Med Radiol 2013; 25(4):261-264.
10. Dusek JJ, Frick WG. Lichen Planus: Oral manifestations and suggested treatments. J Oral Maxillofac Surg 1982; 240-243.

11. Sugerman PB, Savage NW. Oral lichen planus: Causes, diagnosis and management. Aust Dent J 2002;47:290-7.
12. Roopashree MR, Gondhalekar RV, Shashikanth MC, George J,Thippeswamy SH, Shukla A, *et al.* Pathogenesis of oral lichen planus – A review. J Oral Pathol Med 2010;39:729-34.
13. Gupta S and Jawand MK Oral Lichen Planus: An Update on Etiology, Pathogenesis, Clinical Presentation, Diagnosis and Management. Indian J Dermatol 2015; 60(3).
14. Abhishek J, Pratiti G. Altered Hematological Profile of Oral Lichen Planus Patients. ResJj Pharm Biol Chem Sci 2014; 5(5):1271-76.
15. Arshiya Ara S, Mamatha GP, Balaji Rao B. Incidence of diabetes mellitus in patients with lichenplanus. International journal of dental clinics.2011;3(1):29–33.
16. Nosratzehi T, Arbabi Kalati F, Arefpoor Z. Lack of association between diabetes mellitus and oral lichen planus in Zahedan (South- East of Iran).Caspian J Dent Res 2015; 4: 8-12.
17. Manzoor S, Qayoom S, Sultan J and Yasmeen J. Thyroid profile in lichen planus patients from Kashmir valley. Egypt Dermatol Onl J. 2013 ; 9 (1):1-5.
18. Maryam M , Fahimeh R, Fateme D, Sarah M , Seyed S S. World J. Med.Sci. 2014; 10 (2): 103-105.
19. Chakraborti G, Biswas R, Chakraborti S, Sen P. K. Altered Serum Uric Acid Level in Lichen Planus Patients. Indian J Dermatol 2014 Nov;59(6):558-61.
20. Dreiher J, Shapiro J, Cohen AD. Lichen planus and dyslipidaemia: a case control study. Br J Dermatol 2009; 161: 626–629.
21. Amer M, Galal A, Amer A. Lichen Planus Not Associated With Hyperlipidemia; Pyrx.J.Med.Med.Sci. May 2015; 2 (2) :031-036.
22. Konidena A, Pavani B V. Hepatitis C virus infection in patients with oral lichen planus. Niger J Clin Pract 2011;14:228-31.
23. Bhattacharya A, Kaur I, Kumar B. Lichen planus and liver disease. Indian J Dermatol Venereol Leprol 1999; 65: 273-6.
24. Gerayli S., Meshkat Z., Pasdar A., Mozafari M.P, Banihashemi E., Khajavi M.A.et al. The Association Between Oral Lichen Planus and Hepatitis C Virus Infection; A Report From Northeast of Iran .Jundishapur J Microbiol. 2015;8(4):167-74.
25. Shengyuan L, Songpo Y, Wen W, Wenjing T, Haitao Z, Binyou W. Hepatitis C virus and lichen planus: a reciprocal association determined by a meta-analysis. Arch Dermatol 2009;145: 1040–7.
26. Yamada T, Alpers DH; et al. (2009). Textbook of gastroenterology (5th ed.). Chichester, West Sussex: Blackwell Pub. p. 3304.

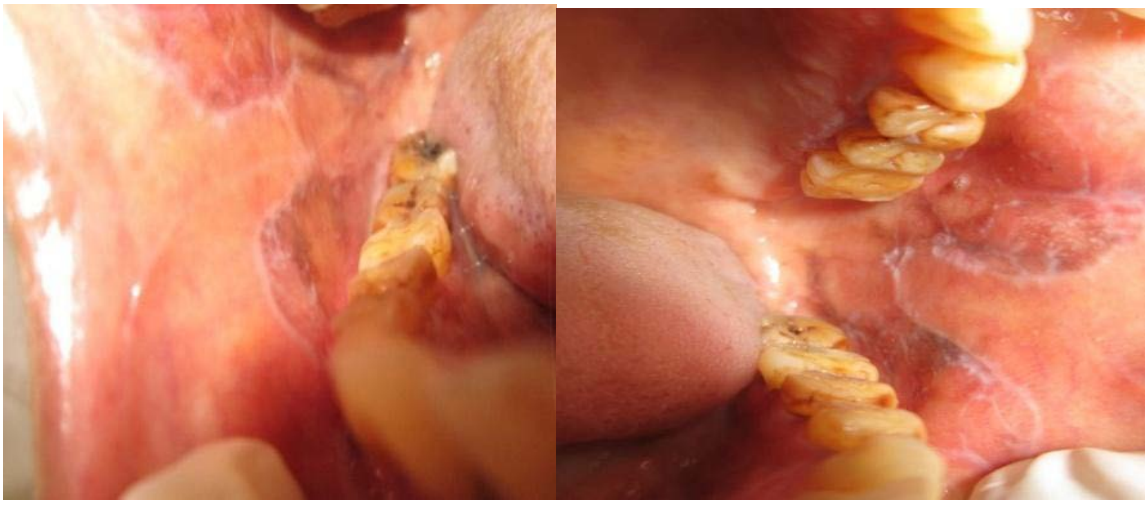

Fig-1: A 52 year old female patient showing Reticular OLP on right and left buccal mucosa.

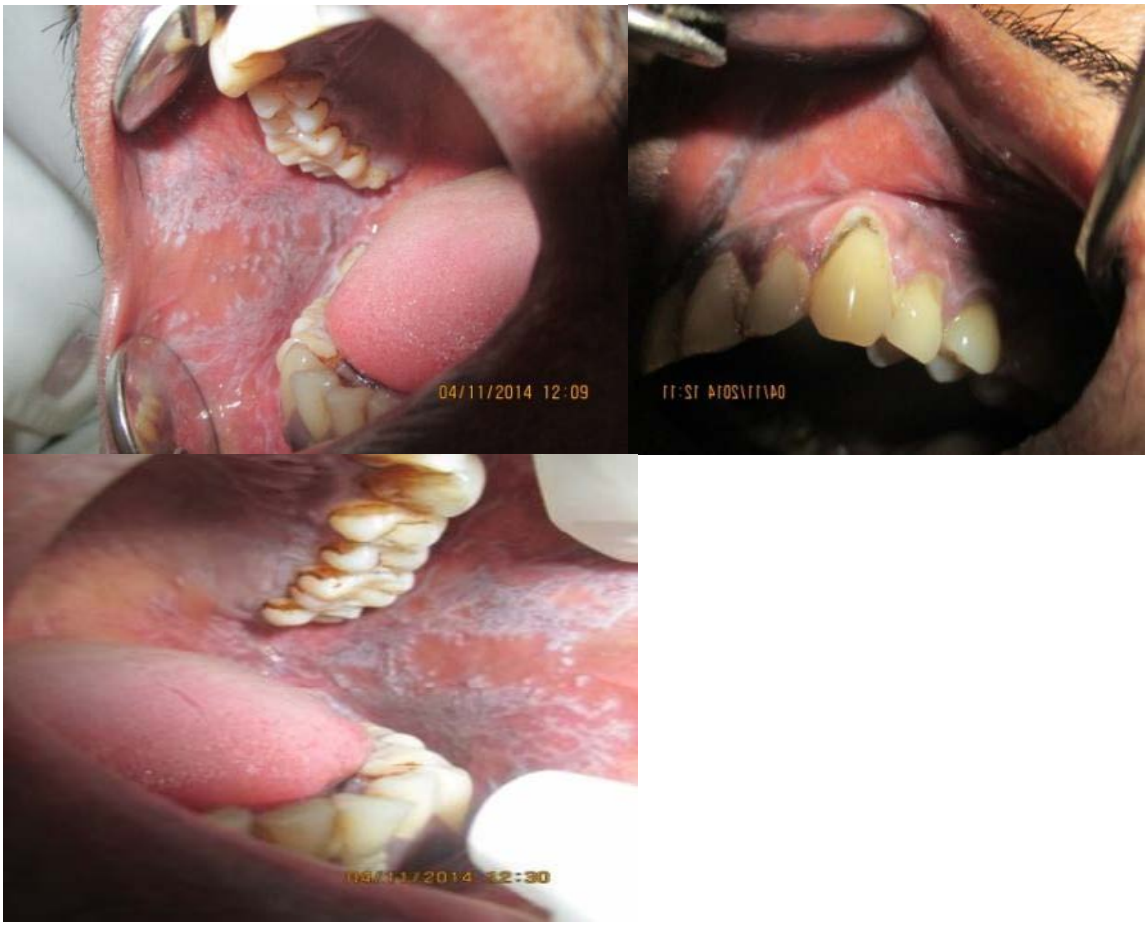

Fig-2: A 30 year old male patient showing Papular and Reticular OLP on right and left buccal mucosae , labial mucosae and gingivae.

TABLE-I : Distribution of study and control subjects according to Gender

	Gender	Frequency	Percentage
Study group	**Males**	20	50
	Females	20	50
	Total	40	100
Control group	**Males**	20	50
	Females	20	50
	Total	40	100
Chi square value=0.0 p value=1.0(NS)			

TABLE II: Distribution of study and control subjects according to Age

Group	N	Age	Standard Deviation(±)
Study group	40	43.78	9.8
Control group	40	43.50	11.7
t=0.00 p value =1.0(NS)			

TABLE-III: Comparison of Complete blood picture (Hemogram) between study and control group

	Group	Mean(SD)	T	p-value
Hemoglobin	**Study group**	12.39(2.76)	0.004	0.99(NS)
	Control group	12.39(2.25)		
Total RBC Count	**Study group**	4.14(.793)	0.01	0.99(NS)
	Control group	4.14(.768)		
Total WBC Count	**Study group**	8897.5(2493.42)	4.13	<0.001*
	Control group	7042.50(1357.20)		
Platelet count	**Study group**	2.65(.61)	-0.44	0.65(NS)
	Control group	3.31(9.20)		
Polymorphs	**Study group**	70.60(5.17)	1.96	0.05(NS)
	Control group	67.93(6.86)		

Lymphocytes	**Study group**	26.20(5.34)	-. 40	0.68(NS)
	Control group	26.73(6.12)		
Eosinophils	**Study group**	2.28(1.15)	0.24	0.81(NS)
	Control group	2.23(0.62)		
Monocytes	**Study group**	2.55(1.10)	-6.32	<0.001*
	Control group	1.18(0.81)		
Basophils	**Study group**	0.18(0.38)	-2.87	<0.05*
	Control group	0.00(0.00)		
ESR	**Study group**	18.13(9.62)	2.95	<0.05*
	Control group	13.05(5.03)		

TABLE-IV : Comparison of Blood grouping and Rh typing between study and control group

	Study Group	**Control Group**	
O+	16(40.0%)	9(22.5%)	25(31.3%)
A-	1(2.5%)	2(5.0%)	3(3.8%)
A+	10(25.0%)	9(22.5%)	19(23.8%)
AB+	0	3(7.5%)	3(3.8%)
B+	12(30.0%)	16(40.0%)	28(35.0%)
O-	1(2.5%)	1(2.5%)	2(2.5%)
Fisher's exact value= 5.83, p= 0.31(NS)			

TABLE-V : Comparison of Viral infections (HIV1&2, HBs Ag & HCV) between study and control group

Group	**HIV1&2**		**HBSAG**		**HCV**	
	Positive	Negative	Positive	Negative	Positive	Negative
Study group	0	40	1	39	0	40
Control group	0	40	0	40	0	40

TABLE-VI : Comparison of Liver function tests between study and control group

	Group	Mean(SD)	T	p-value
Total bilirubin	**Study group**	0.81(0.26)	0.71	0.47(NS)
	Control group	0.77(0.23)		
Direct bilirubin	**Study group**	0.21(0.11)	-0.43	0.66(NS)
	Control group	0.22(0.14)		
Indirect bilirubin	**Study group**	0.60(0.16)	1.08	0.28(NS)
	Control group	0.56(0.14)		
SGOT	**Study group**	24.15(9.79)	2.32	0.02*
	Control group	19.65(7.31)		
SGPT	**Study group**	23.75(11.18)	2.64	0.01*
	Control group	18.00(8.03)		
Alkaline Phosphatase	**Study group**	105.43(35.75)	3.98	<0.001*
	Control group	80.38(17.47)		

TABLE- VII : Comparison of Random blood sugar between study and control group

	Group	Mean(SD)	T	p-value
Random blood sugar	**Study group**	103.35(25.53)	0.728	0.46(NS)
	Control group	98.08(28.89)		

TABLE-VIII : Comparison and Estimation of Total proteins, serum albumin and A.G. ratio between study and control group

	Group	Mean(SD)	t	p-value
Total proteins	**Study group**	6.87(0.27)	-0.97	0.33(NS)
	Control group	8.33(9.51)		
Albumin	**Study group**	3.65(0.21)	0.12	0.90(NS)
	Control group	3.64(0.33)		
Globulin	**Study group**	3.21(0.22)	0.12	0.90(NS)
	Control group	3.210(0.13)		
A.G. ratio	**Study group**	1.13(0.18)	1.59	0.11(NS)
	Control group	1.08(0.09)		

TABLE-IX : Comparison of serum Lipid profile for checking levels of HDL, LDL, VLDL cholesterol, triglycerides between study and control group

	Group	Mean(SD)	T	p-value
Total Cholesterol	**Study group**	156.63(28.88)	-2.66	0.009*
	Control group	177.60(40.62)		
HDL Cholesterol	**Study group**	41.48(2.55)	-4.95	<0.001*
	Control group	44.23(2.41)		
LDL Cholesterol	**Study group**	85.63(33.38)	-2.93	0.004*
	Control group	108.23(35.49)		
VLDL Cholesterol	**Study group**	30.08(11.94)	0.57	0.57(NS)
	Control group	27.98(19.95)		
Trigylcerides	**Study group**	153.95(58.59)	2.54	0.01*
	Control group	122.77(50.58)		
CHOL/HDL ratio	**Study group**	3.74(0.66)	-0.64	0.52(NS)
	Control group	3.85(0.91)		

TABLE-X: Comparison of Thyroid function tests (T3, T4, TSH) between study and control group

	Group	Mean(SD)	T	p-value
T3	**Study group**	1.15(0.22)	-2.99	0.004*
	Control group	1.32(0.28)		
T4	**Study group**	8.96(1.72)	-0.76	0.44(NS)
	Control group	9.26(1.76)		
TSH	**Study group**	3.22(3.72)	1.72	0.08(NS)
	Control group	2.13(1.38)		

TABLE-XI : Comparison of Renal function tests (RFTs) between study and control group

	Group	Mean(SD)	T	p-value
Blood urea	**Study group**	26.98(5.91)	-2.54	0.01*
	Control group	29.85(3.99)		
Serum creatinine	**Study group**	1.14(0.49)	1.11	0.26(NS)
	Control group	1.04(0.17)		
Serum uric acid	**Study group**	5.87(1.29)	-0.82	0.41(NS)
	Control group	6.07(0.74)		

TABLE XII, XIII, XIV: Comparison of Complete urine examination between study and control group

TABLE XII

	Group	Mean(SD)	T	p-value
Urine specific gravity	**Study group**	1.017(0.008)	-0.77	0.44(NS)
	Control group	1.018(0.008))		

TABLE-XIII

Group	Urine colour		Urine appearance		Urine reaction	Urine albumin		
	Pale yellow	Straw	Clear	Turbid	Acidic	Nil	Positive	Trace
Study group	39	1	38	2	40	32	1	7
Control group	40	0	38	2	40	40	0	0

TABLE XIV

	URINE PUS CELLS			URINE EPITHELIAL CELLS		
	<10	**>10**	**Total**	**<20**	**>20**	**Total**
Group 1	36(90.0%)	4(10.0%)	40	39(97.5%)	1(2.5%)	40
Group 2	40(100.0%)	0	40	39(97.5%)	1(2.5%)	40
Total	76(95.0%)	4(5.0%)	80	78(97.5%)	2(2.5%)	80
Fisher's exact test	p-value = 0.12(NS)			p-value = 1.00(NS)		

Table XV, XVI : Comparison of Ultrasound abdomen findings between study and control group

Table XV

Ultrasound abdomen	Group		Total
	1	2	
Anterior wall uterine fibroid	1(2.5%)	0	1
Bilateral small renal calculus	1(2.5%)	0	1
Bulky uterus with small endometrium & simple small left ovarian cyst	0	1(2.5%)	1
Bulky uterus with too small anterior wall fibroids	1(2.5%)	0	1
Cholelithiasis & grade i fatty liver	1(2.5%)	0	1
Grade I fatty liver	6(15%)	2(5%)	8
Grade I fatty liver & grade I prostamegaly	1(2.5%)	0	1
Grade I fatty liver; bulky uterus with small posterior wall of fibroid	1(2.5%)	0	1
Grade I prostamegaly	1(2.5%)	1(2.5%)	2
Left renal calculus & mild degree of fatty changes in liver	1(2.5%)	0	1
Mild bulky uterus with small uterine fibroids	1(2.5%)	0	1
Mild hepatomegaly with fatty liver	1(2.5%)	0	1
NAD(No abnormality detected)	23	36	59
Small right renal cortical cysts	1(2.5%)	0	1
Total	40	40	80

Table XVI

Ultrasound abdomen	**Group**		**Total**
	Study	**Control**	
NAD	**23(57.5%)**	**36(90.0%)**	**59(73.75%)**
Present	**17(42.5%)**	**4(10.0%)**	**21(26.25%)**
Total	**40(50.0%)**	**40(50.0%)**	**80(100.0%)**
	Chi square value(df)= 10.91(1), p<0.001*		