Section: Dermatology

Original Research Article

A STUDY OF CLINICAL PRESENTATION OF LICHEN PLANUS AND ASSOCIATION BETWEEN LICHEN PLANUS AND HEPATITIS C INFECTION IN TERRITORY CARE HOSPITAL

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Abstract

Background: Lichen planus is a chronic inflammatory disease of skin and mucosa with a unique appearance and histology. Hepatitis C virus (HCV) infection is one of the numerous associations that have been reported. Many factors have been reported in the etiology of lichen planus. One of them is Hepatitis C virus infection. The link between lichen planus and Hepatitis C infection was discovered in 1991. The Aims and objective is to study the association between lichen planus and Hepatitis C virus infection in the patients attending Dermatology OPD, Government General Hospital, Kurnool to study various clinical presentations of lichen planus. **Materials and Methods:** All the patients who attended the DVL OPD, Government General Hospital, In the present study, most of the cases were in the age group 31-50 years Kurnool during December 2020 to May 2022, who met the inclusion and exclusion criteria and had a clinical diagnosis of lichen planus involving the skin, mucous membranes, or both were included in the study as the case group population. As a control group population, the voluntary blood donors who underwent screening at the Government General Hospital in Kurnool were chosen. **Result:** In the present study, most of the cases were in the age group 31-50 years, There is female preponderance (M:F = 1 : 3.7) in the present study, In this study, majority of the patients comes under the categories of house-wives (Female group), coolie & agricultural laborers / farmers (Elderly group) and students (Younger age group). Remaining cases are from different fields such as drivers, constables, teacher, nurse etc. In this study, classical lichen planus was the most common variant (35%) where other forms such as follicular, genital, actinic, lichen planus pigmentosus (2-3% each) were the least common. 3 patients were tested positive for anti-HCV antibodies in the study group with lichen planus (2 patients had generalized involvement and 1 patient had hypertrophic variant of lichen planus) where 5 positive cases of anti HCV antibodies were found in the voluntary blood donors (mean age group affected was 21-35 years). The odds ratio is 0.5 (<1) and the P value is 0.47 (>0.05). This indicates there is no association between the HCV and lichen planus and there is no statistically significant difference between the study and the control group. **Conclusion:** This study is done mainly to check the association between lichen planus and HCV infection in this region so that lichen planus may be employed, as a marker of HCV infection in asymptomatic people, allowing for earlier detection and treatment, as well as a better prognosis for the asymptomatic HCV infected patients by preventing severe hepatic damage in long term. The correlation of lichen planus with Hepatitis C virus infection differs in various geographic areas. Based on the present study and other studies that were conducted in India, there was no statistically significant difference between the study group and the control group, demonstrating that there is no association between HCV infection and lichen planus. Therefore, there is no need to evaluate anti HCV antibodies in the patients with lichen planus in this geographic area.

INTRODUCTION

Lichen planus is a chronic inflammatory disease of skin and mucosa with a unique appearance and histology. Skin, genitalia, hair follicles, nails, oral mucosa, nasal mucosa, esophagus, larynx, and eyes can all be affected. Hepatitis C virus (HCV) infection is one of the numerous associations that have been reported.

Hebra first described lichen planus, but Erasmus Wilson named it in 1869. Lichen is derived from the Greek word 'leichen,' which means ‘tree moss’. It refers to a distinct type of flowerless vegetation. Planus (Latin for "flat") means flat. Lichenplanusisa self- limiting condition that is frequent in middle-aged adults. It affects the skin, mucous membranes, hair, and nails.

The clinical manifestation of lichen planus in skin is itchy, violaceous, flat-topped, glistening, polygonal papules and plaques, most commonly occurring over the flexor aspect of extremities, particularly the wrist. Ridging, thinning, and pterygium are examples of nail changes. Scarring alopecia is seen in scalp involvement.

Hypertrophic, atrophic, actinic, follicular, bullous, annular, linear, and lichen planus pigmentosus are all the types of lichen planus.[1] When compared to cutaneous lichen planus, oral lichen planus has a lower tendency for spontaneous regression. Oral lichen planus can be of reticular, bullous, plaque, erosive, or atrophic variants.[2]

Many factors have been reported in the etiology of lichen planus. One of them is Hepatitis C virus infection. The link between lichen planus and Hepatitis C infection was discovered in 1991.[3] Many studies have since supported this viewpoint and suggested the importance of liver monitoring in patients with mucocutaneous lichen planus.

Because HCV infection is typically indolent,[4] patients may present only in the late stages of the disease, with serious complications such as cirrhosis and chronic liver disease. If the link between lichen planus and HCV infection is confirmed, screening patients with lichen planus may aid in early diagnosis, treatment, and decreases the transmission of HCV infection in asymptomatic individuals.[5]

The study will contribute to further supporting this fact, bringing us one step closer to developing guidelines for HCV screening in lichen planus patients.[6,7]

Lichen planus is mostly diagnosed clinically, but histopathology can be used to confirm the diagnosis in doubtful cases. Corticosteroids, retinoids, immunosuppressive agents, and other newer agents are among the treatment options.[8-10]

Treatment: Lichen planus is normally benign and self-limiting. Several different modalities of treatment include:

- **Topical:** Steroids, 1% pimecrolimus and tacrolimus
- **Systemic:** Steroids, dapsone, antimalarials, retinoids, immunosuppressants, photochemotherapy, anti-histamines.

Corticosteroids

The ideal drug for treating lichen planus is corticosteroid.

These can be included in formulations for topical, systemic, or intralesional usage.

Retinoids

- Topical: Isotretinoin gel (0.1%) and etretinate have been administered in cases of orallichenplanus.
- Systemic: Acitretin is administered in a dose of 30 mg/day for 8 weeks in cases of severe cutaneous lichen planus and 2 weeks in cases of LP-LE overlap syndrome.

Photochemotherapy: It is possible to treat generalized cutaneous and erosive oral lichen planus with PUVA therapy. The first dose is 0.5-2 J/cm2, and the maximum dose allowed in a single session is 7J/cm2, delivered 3 times/week.

Immunosuppressants: In severe cases of cutaneous, oral, and nail lichen planus, Cyclosporine is administered at doses of 3–10 mg/kg/day. LPP and generalized cutaneous involvement are also treated with Azathioprine. Mycophenolatemofetil (1.5g twice a day) is also effective in both oral and bullous variants. Both 1% pimecrolimus cream and 0.1% tacrolimus ointment are effective treatments for erosive oral lichen planus.

Miscellaneous:

- hydroxychloroquine (200-400 mg/day)- erosive &actinic lichen planus
- Thalidomide- erosive lichen planus
- Dapsone (200 mg/day), metronidazole (500 mg twice daily), phenytoin (100-200mg/day), and griseofulvin (1g/day) - erosive and bullous forms
- Low-molecular-weight heparin is injected (3 mg/week) for 6–10 weeks and has anti-proliferative and immunomodulatory effects.
- Although interferon-α2b has been linked to lichen planus as a cause or aggravating factor, it can also be used to treat generalized cutaneous involvement.
- Oral lichen planus can be treated with extracorporeal photopheresis and photodynamic treatment mediated by methylene blue (MB-PDT).
- Ulcerative lichen planus of the feet has been treated using split skin grafting.72

Lichen planus in HCV infection: The class II HLA-DR6 allele is mostly linked to lichen planus in HCV infection. Numerous researchers have hypothesized that concurrent HCV infection and the development of lichen planus may result from genetic, environmental, regional, or other variables74. The proposed mechanisms include local induction of an HCV epitope- specific immune response70, selective presentation of HCV-encoded peptides on the surface of monocytes by HLA-DR6 molecules to the CD4+ cells, extrahepatic HCV replication, antigenic alterations in keratinocytes that result in cell-

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mediated immune action and finally, the auto-immunity response. Lichen planus and HCV infection have been associated, according to reports with statistical significance in several studies from Japan, Spain, Europe, the USA, Italy, and Germany. However, a lot of researchers reported no associations.

Diagnosis: There are two types of tests available to detect HCV infection.

1. Serological assay like Enzyme Linked Immunosorbent Assay (ELISA) and Radio Immuno Blot Assay (RIBA) to detect HCV antibodies.
2. Molecular assays like Polymerase Chain Reaction (PCR) to detect HCV RNA.

Aims and Objectives

To study the association between lichen planus and Hepatitis C virus infection in the patients attending Dermatology OPD, Government General Hospital, Kurnool.

To study various clinical presentations of lichen planus.

MATERIALS AND METHODS

Study description: All the patients who attended the DVL OPD, Government General Hospital, Kurnool during December 2020 to May 2022, who met the inclusion and exclusion criteria and had a clinical diagnosis of lichen planus involving the skin, mucous membranes, or both were included in the study as the case group population. As a control group population, the voluntary blood donors who underwent screening at the Government General Hospital in Kurnool were chosen.

Study design: Clinical and prospective study

Sample size: 100 patients

Inclusion criteria

- Patients willing for the study and follow up.
- Male and female with lichen planus (> 18years and < 60 years).

Exclusion Criteria

- Patients not willing for the procedure and follow-up.
- Patients on drugs causing lichenoid eruptions like Beta blockers, Antimalarials, Diuretics, ACE inhibitors, Chlorpropamide, Simvastatin etc.
- Pregnant / lactating women
- Pre-existing liver disease
- Intravenous drug users
- Blood dyscrasias like Hemophilia.

Method of data collection

- Institutional Ethics Committee given Ethical clearance; form is obtained.
- Informed written consent taken as per the consent form attached in annexure.
- For each patient, demographic information and a thorough medical history and clinical examination (including the duration and location of lesions, the details of the involvement and any risk factors were recorded in a pre-designed proforma.
- Standardized photographs using a digital camera were taken
- Following investigations were done in all the patients:
  1. Complete blood picture (Hb, total WBC count, differential count, ESR, platelet count)
  2. Renal function tests like blood urea and serum creatinine
  3. Liver function tests like S. bilirubin, SGOT, SGPT, ALP, S. proteins
  4. Random blood sugar
  5. Hepatitis C antibody test (Immunochromatographic HCV test).
- Data collected was analyzed and tabulated using Microsoft Word and Microsoft Excel about clinical and epidemiological aspects (age, sex) and conclusions.

Immuno-Chromatographic HCV test (SD bioline HCV 02FK101)

A single step, quick immunochromatographic test for the detection of antibody against the hepatitis c virus iuman serum, plasma, or whole blood.

RESULTS

1. Age Distribution

In the present study, most of the cases were in the age group 31-50 years.

2. Sex Distribution

There is female preponderance (M:F = 1 : 3.7) in the present study.

3. Occupation

In this study, majority of the patients comes under the categories of house-wives (Female group), coolie & agricultural laborers / farmers (Elderly group) and students (Younger age group). Remaining cases are from different fields such as drivers, constables, teacher, nurse etc.

4. Duration of the Disease

Although maximum duration of the illness was 2 years, majority of the patients were having the disease since 7 months – 1 year (42%).

5. Symptoms

Only 6 of the patients in the study were asymptomatic. 82 people, however, experienced moderate to severe itching.

6. Risk Factors

Out of 100 patients, 17 were smokers/ tobacco chewers and 7 were alcoholics. Of these 24 cases, 6 were both alcoholic and smoker. 10 patients gave history of betelnut chewing. 6 patients disclosed a history of emotional stress, 3 patients had a history of surgery in the past.

7. Previous Medical History

In this study, 19 were diabetic and 10 were hypertensive. Of these 29 patients, 9 patients were both diabetic and hypertensive. 4 were on hypothyroid medication and 7 patients disclosed a history of jaundice in the past.

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8. Clinical Variants

In this study, classical lichen planus was the most common variant(35%) where other forms such as follicular, genital, actinic, lichen planus pigmentosus (2-3% each) were the least common.

9. Sites Of Involvement

In this study, upper limbs (47%) and lower limbs (50%) were predominantly affected, followed by oral cavity (36%) and trunk (29%) involvement. Genitalia was involved in 6 cases, face in 4 cases and nails in 3 cases. Least affected were scalp (1%) and neck (2%).

10. Association with HCV Antibodies

3 patients were tested positive for anti-HCV antibodies in the study group with lichen planus (2 patients had generalized involvement and 1 patient had hypertrophic variant of lichen planus) where 5 positive cases of anti HCV antibodies were found in the voluntary blood donors (mean age group affected was 21-35 years). The odds ratio is 0.5 (<1) and the P value is 0.47 (>0.05). This indicates there is no association between the HCV and lichen planus and there is no statistically significant difference between the study and the control group.

Table 1: ?

Variants	No. of cases	Percentage
Generalized	16	16
Hypertrophic	10	10
Classical	35	35
Linear	5	5
Oral	17	17
Vulvo-vaginal gingival syndrome	3	3
Actinic	4	4
Genital	3	3
Zosteriform	2	2
Follicular LP	2	2
LP Pigmentosus	3	3
Total	100	100

DISCUSSION

Lichen planus is a chronic, idiopathic, inflammatory disorder that affects skin, mucous membrane and appendages. Lichen planus is known for its 5P's - polygonal, plane, pruritic, purple, papules/ plaques predominantly involving the flexures with basal cell degeneration and interface dermatitis are the characteristic histopathological features.[11,12]

Prevalence: Out of 52,206 new patients who attended DVL OPD at Government General Hospital, Kurnool, 100 patients (between 18 to 60 years of age) were diagnosed to have lichen planus during this study period of one and half year (December 2020 to may 2022). Thus, the prevalence in the present study is approximately 0.19%. This is similar to the global prevalence of <1%.[6]

According to Anbar et al, the prevalence was 0.28% in Egypt.[13] The study by Bhattacharya had shown the prevalence of 0.38%in India.[14] Another report by Kachhwa et al. showing 0.8% prevalence in India.

Age distribution: In the current study, 52% of the cases fell within the age range of 31 to 50 years. This is similar to the study by Bhattacharya where the most common age group with disease was between 20-49 years.[14] Another comparable study is by Kachhwa et al. where 46.93% of the cases were between 20-39 years of age. Garg VK et al. reported that 30.6% of the cases were under the age category of 31-40 years.[15,16]

Sex distribution: In our analysis, there were noticeably more women than men (79%) with the ratio M:F= 1:3.7. This is analogous with the studies by Garg VK et al (M:F= 1:13)100, Wilson et al., Shandilya et al.[9] (M:F= 0.64:1) and Boyd et al,[6] showing female preponderance. Meanwhile, Bhattacharya et al,[14] have reported no sex predilection (M:F=1:1).

Occupation: In our study, housewives (43%), coolie (19%) and farmers/laborers (17%), students (14%) made up the majority of the patients, whereas research by Naldi et al (1979), manual laborers predominate (46%) and the Shandilya et al. survey revealed that 35.56% prevalence in students, 24.44% in housewives, 8.33% in farmers, 11.11% in laborers and 3.89% in other professions.[9,17,18]

Duration of the disease: In our study, the condition might last up to two years, and 42% of individuals had it for between seven months and one year, 23% between four and six months and 18% between one and three months. 5% had duration of <1 month where remaining 12% gave a history of > 1 year duration of illness.[19,20]

According to research by Sehgal et al., the majority of individuals had the condition for two to three months prior to the study's completion.[11]

Symptoms: Six percent of the cases were asymptomatic, 12% had slight itching, and 82% patients had moderate to severe itching in the present study.[21]

This is almost identical to the study by Kachhwa et al where 73% of the cases had moderate to severe itching and the report by Sehgal et al where 95% of the cases showed moderate to severe itching.[22]

Risk factors: In the ongoing study, 17 cases (17%) revealed the habit of smoking/tobacco chewing and 7 individuals (7%) were alcoholics. Six of these 24 cases involved in both alcohol intake and smoking. 10 patients (10%) had a history of betel nut chewing. Six patients (6%) disclosed a history of emotional

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distress, and three patients (3%) had previously had surgery.[23]

This is analogous to the study by Wilson et al. where 13.7% patients with history of smoking/ tobacco chewing. According to Shandilya et al. 18.33% cases were smokers, 9.44% patients were alcoholics and 22.78% patients had a habit of tobacco chewing, whereas alcohol and smoking were risk factors in 67% and 36% of the cases, respectively, according to Naldi et al.[24]

Previous medical history: In our study, there were 7individuals (7%) with a history of jaundice, 10 patients with hypertension (10%), and 19 patients with diabetes (19%). A study by Shandilya et al,[9] found diabetes in 2.78% of cases, hypertension in 5.56% of patients and hepatitis in 6.11% of cases. However, study by Anjana et al found no connection with diabetes or hypertension.

Variants: In our research, the most prevalent clinical form of the disease was classic lichen planus (35% of patients). Ten patients had (10%) hypertrophic LP, 16 patients had eruptive/generalized type (16%),17 patients were with pure oral involvement (17%), 5 cases had linear form (5%), 3 cases of vulvovaginal gingival syndrome (3%), 4 cases had actinic LP (4%), genital and LPP in 3 cases each, while the two least frequent variations were follicular and segmental/ zosteriform lichen planus, with two instances each.

As stated by Singh et al study, 74% of the patients had classical type, hypertrophic variant was seen in 13%, actinic type in 7.5%, follicular form in 2%, LPP in 1.5% and nail involvement in 1.5% of the cases. Bhattacharya et al stated that classical type is the most common (47.4%) according to their study.

In compliance with Shandilya et al. study, most common type is eruptive (38.89%), followed by hypertrophic (20.55%), annular (12.22%), lichen planus pemphigoides (10.56%), linear type (6.67%) and actinic type (3.89%).

Sites of involvement: The present study disclosed that the majority of lichen planus cases showed the involvement of upper (47%) and lower limbs (50%). 28% of cases impacted the trunk,6% the genitalia, 4% the face, 3% the nails, 2% the neck, 1% the scalp and 33% the oral mucosa.

This is in parallel with the study by Shandilya et al. in which the most affected site is upper limbs (57.22%), followed by lower limbs (48.33%), trunk in 44.44%, genitalia in 12.72%, face in 8.33% and scalp in 6.11% of the cases.[9] Bhattacharya et al. found that limbs were the most common site involved in 55.6% of the patients.[14]

In other investigations, skin, skin and mucosa, and mucosa alone were all involved in 98%, 68%, and 7% of cases (Stojanovic et al., 1981)81, and 71%, 20%, and 12% of cases (Sehgal et al., 2011).[11]

Koebner's phenomenon: The development of fresh skin lesions on previously uninvolved skin as a result of trauma is known as the Koebner phenomenon (KP), which was first named by Heinrich Koebner in 1876.

In the current investigation, 19% of the individuals exhibited Koebner's phenomena. This is in consistent with the findings of investigations by Boyd et al and Fine et al.

Nail changes: 3% of the participants in our study had abnormal nails. Nails were discovered to be implicated in 6.4% of cases by Kachhwa et al However, none of the cases in the study by Sehgal et al involved nails.

Tosti et al (1993), disclosed that out of 24 patients with nail lichen planus, Involvement of the nail matrix in 91.6% with pterygium (16.6%), koilonychia (8.3%), onychoschizia (8.3%), longitudinal ridging (70.8%), nail plate thinning (50%), onychorrhexis (33.3%), red lunula (25%), splinter hemorrhage (4.1%), and melanonychia striata (4.1%). Involvement of the nail bed in 58.3% with subungual hyperkeratosis (37.5%) and onycholysis (41.6%).

Wickham Striae: Louis Frederic Wickham first used the name Wickham striae (WS) in 1895 to describe the thin white or grey lines or dots that may be seen above the papules and oral mucosal lesions of Lichen planus.7,8 Darier (1909) published histological findings and suggested that an increase in the granular cell layer was responsible for the development of Wickham's striae.[1]

In the present study, 45% of the cases showed Wickham striae on both cutaneous and mucosal lesions.

Correlation with HCV infection: In this study, out of 100 cases in the study group with lichen planus, 3 patients had anti-HCV antibodies that were positive, and in 100 voluntary blood donors, there were 5 cases of anti-HCV antibodies that were positive. The test used was SD Bio-line immuno-chromatography. The P value is 0.47 (>0.05), and the odds ratio is 0.5 (<1). This demonstrates that there is no statistically significant difference between the study group and the control group and that there is no association between HCV and lichen planus.

On the report of the study by Tucker et al88 (England, 1999), the seropositivity to the hepatitis C virus was assessed in a total of 45 patients with classical and/or erosive mucosal membrane lichen planus and 32 controls. Only 1 of the control patients and none of the lichen planus patients had serological evidence of hepatitis C infection.

On the contrary, the study by Tanei et al (Japan, 1995) showed a strong correlation between lichen planus (LP) and chronic hepatitis C in a prospective clinical study. Of the 45 LP patients, anti-hepatitis C virus (HCV) antibodies were discovered in 17 (37.88%). Both male and female patients equally had this greater incidence of anti- HCV antibodies. The majority of individuals who tested positive for anti-HCV antibodies had elevated transaminase enzyme levels or had previously experienced chronic hepatitis. T lymphocyte infiltration with keratinocyte or hepatocyte damage served as the foundation for the histopathological findings of both LP and chronic hepatitis C. The levels of infiltrating cells that were

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positive for the human leukocyte antigen (HLA)-DR antibodies, UCHL-1, MX-pan B, Leu-7, and chronic hepatitis C seemed to be comparable to those in the LP lesions. These findings could imply a link between LP and chronic hepatitis C.

As stated by Zahra Ghodsi et al (Iran, 1998), there were 146 cases of lichen planus diagnosed, including 78 (53.1%) women and 69 (46.9%) men. It was shown that there is a statistically significant link between HCV infection and erosive lichen planus. Patients with and without HCV infection did not significantly differ on liver function.

In accord with the report by Lodi et al (Italy, 2004), nearly one in five (19.1%) in the LP group in the cross- sectional study tested positive for HCV, compared to a far lower prevalence of infection (3.2% in the control group). Six of the 25 relevant studies that came from the systematic review had a low risk of bias. Comparing patients with LP to controls, there was a statistically significant difference in the percentage of subjects who tested positive for HCV. Following meta-analysis, it appeared that geographical area, but not the age, influenced the diversity of HCV prevalence in LP patients.

In accord with the study by Zahra Rahnama et al. (Iran, 2005), Anti-HCV antibodies were detected using an enzyme-linked immunosorbent test (ELISA) in all participants in the study and control groups. A second-generation recombinant immunoblot assay (RIBA II) test was used to confirm positive diagnosis. Three of the controls (2.1%) and one patient with LP (1.5%) both tested positive for HCV-Ab. Between the two groups, there was no significant difference in the proportion of HCV-Ab positives (OR = 0.7; 95% CI = 0.1-6.9).

CONCLUSION

This study is done mainly to check the association between lichen planus and HCV infection in this region so that lichen planus may be employed, as a marker of HCV infection in asymptomatic people, allowing for earlier detection and treatment, as well as a better prognosis for the asymptomatic HCV infected patients by preventing severe hepatic damage in long term. The correlation of lichen planus with Hepatitis C virus infection differs in various geographic areas.

Based on the present study and other studies that were conducted in India, there was no statistically significant difference between the study group and the control group, demonstrating that there is no association between HCV infection and lichen planus.

Therefore, there is no need to evaluate anti HCV antibodies in the patients with lichen planus in this geographic area.

REFERENCES

1. Daoud MS, Pittelkow MR. Lichen planus. In: Irwin MF, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, editors. Fitzpatrick's Dermatology in general medicine. 6thedn. New York: McGrawHill;2003.p.463–77.
2. Sugerman PB, Savage NW, Walsh LJ, et al. The pathogenesis of oral lichen planus. Crit Rev Oral Biol Med 2002;13(4):350-65.
3. Mokni M, Rybojad M, Puppin D Jr, Catala S, Venezia F, Djian R, Morel P. Lichen planus and hepatitis C virus. J Am AcadDermatol 1991 May;24(5 Pt 1):792.
4. Chen SL, Morgan TR. The natural history of Hepatitis C virus (HCV) infection. Int J Med Sci 2006;3(2):47-52.
5. Hamid A, Aziz MA, et al. Lichen planus: Histopathological study of 57 cases. Indian J DermatolVenereol 1970;36:85-91.
6. Boyd AS, Neldner KH. Lichen planus. J Am AcadDermatol 1991;25:593-619.
7. Steffen C, Dupree ML. Louis-Frederic Wickham and the Wickham's striae of lichen planus. Skinmed.2004;3:287–9.
8. Rivers JK, Jackson R, Orizaga M. Who was Wickham and what are his striae? Int J Dermatol 1986;25:611–3.
9. N, Shandilya S, Ranjan A, Verma G. Lichen Planus: A Clinical and Epidemiological Study. Asian J. Med. Res. 2022;11(2):17-25.
10. Rogers RS III, Eisen D. Erosive oral lichen planus with genital lesions: the vulvovaginal-gingival syndrome and the peno-gingival syndrome. DermatolClin 2003 Jan;21(1):91-8.
11. Sehgal VN, Rege VL. Lichen planus- An appraisal of 147 cases. Int J Dermal 1976;15:752-756.
12. Jury CS, Munro CS. Linear lichen planus related to hepatitis Cinfection? Br J Dermatol 2000;142;836–7.
13. Mignogna MD, Lo Muzio L, Lo Russo L, et al. Oral lichen planus: different clinical features in HCV positive and HCV negative patients. Int J Dermatol 2000;39(2):134-9.
14. Bhattacharya M, Kaur I, Kumar B. Lichen planus- a clinical and epidemiological study. J Dermatol 2000;27:576-82.
15. Alabi GO, Akinsanya JB. Lichen planus in tropical Africa. Trop Geogr Med 1981;33:143–7.
16. Porter K, Konda P, Scully C, et al. Class I and II HLA antigens in British patients with oral lichen planus. Oral Surg Oral Med Oral Pathol 1993;75:176–80.
17. Halery S, Feuerman EJ. Abnormal glucose tolerance associated with lichen planus. ActaDermVenereol (Stockh) 1979;59:167–70.
18. Kim SG, Chae CH, et al. Apoptosis of oral epithelial cells in oral lichen planus caused by up regulation of BMP-4. J Oral Pathol Med 2006;35:37–45.
19. Reich HL, Nguyen JT, James WD. Annular lichen planus: a case series of 20 patients. J Am AcadDermatol 2004;50(4):595-9.
20. Breathnach SM, Black MM. Lichen planus and lichenoid disorders. In: Burns T, Breathnach S, Cox N, et al, editors. Rook's textbook of dermatology. 7thedn. Oxford: Blackwell Science;2004.p.42.1– 32.
21. Zhao ZZ, Sugerman PB, Zhou XJ, Walsh LJ, Savage NW. Mast cell degranulation and the role of T cell RANTES in oral lichen planus. Oral Dis 2001;7:246–51.
22. Turel A, Ozturckan S, Sahin MT, et al. Wolf's isotopic response: a case of zosteriform lichen planus. J Dermatol. 2002;29:339–42.
23. Gunning ST, Turiansky GW. Successive linear, generalized and oral lichen planus in a patient with chronic hepatitis C infection. J Am AcadDermatol 2003;49:1190–1.
24. Yesudian P, Rao R. Malignant transformation of hypertrophic lichen planus. Int J Dermatol 1985;24:177–8.

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