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Original Research Article

A cross sectional study of lichen planus: It's epidemiological, clinico-histopathogical and serological perspective

Palakurthi Sri Sneha[1], K. Seetharamanjaneyulu[1,*], G Venkata Ramana[1], Satya Saya[1]

[1]*Dept. of Dermatology & Venereology and leprosy, GSL Medical College & General Hospital, Rajahmundry, Andhra Pradesh, India*

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ABSTRACT

Introduction : Lichen planus (LP) is a common papulosquamous skin disease with a prevalence 1-2% globally and 0.1 - 1.5% in Indian studies with many morphological presentations. LP is strongly associated with chronic HCV infection (3.1- 18.3% in different studies), while the association with other viral infections (Hepatitis B and HIV) is not that strong. There are only a few studies in Indian patients and the association reported is not uniform. Further studies will help to consolidate the association.
Aims and Objectives : 1) To study the epidemiological and clinico- histopathological pattern in LP.
2) To determine the serology and assess the possible association with HCV, HBsAg and HIV.
Materials and Methods : 140 patients of LP after histopathological confirmation were tested for detection of HCV antibodies, Hbs antigen and HIV antibodies by HCV-TRIDOT, HEPACARD and HIV –TRIDOT respectively. Statistical analysis was performed by SPSS software 20.0.
Results : Most common age group involved was 21-30 years. 75.6% of the patients presented with in 6 months of onset. Extremities and trunk were commonly involved. The most common clinical variants are classical LP(35%) and hypertrophic LP(31%). Mucosal involvement alone was observed in 9.3% patients . HBs Ag was positive in 4 cases , Anti HCV antibodies were positive in 3 cases and HIV was reactive in 3 cases.
Conclusion : There is no significant relationship between LP and Hepatitis B, C and HIV virus. It may be suggested that viral serology (HBV, HCV, HIV) may not be necessary in routine screening for LP.

1. Introduction

Lichen planus (LP) is a common papulosquamous skin disease with a prevalence of 1-2% globally and 0.1% to 1.5% in Indian studies.[1] The disease was mostly reported in middle-aged patients 30-60 years of ageand is less common in extremes of age.[1]Few studies show female preponderance.[2,3] LP most commonly involves the flexor surfaces of extremities with pruritic, purple, polygonal, planar, papules, plaques. The lesions show Wickham's striae and positive Kobners phenomenon.[4] The lesions are typically bilateral and relatively symmetrical. Strong association has been reported with HCV infection, while the association with other viral infections like EBV, CMV, Hepatitis B, HIV and Human herpes simplex virus is not that strong.[5,6]

The association of HCV infection and lichen planus varied from the different studies. Positive association was seen in 3.1% to as high as 18.3%. More significant association was seen in Oral lichen planus.[7–12] Few studies have found no association between HCV infection and lichenplanus.[13–18] Though there were several reports showing a higher prevalence of HBV with LP, a possible association between HBV infection and LP remains unclear.[19,20] Regarding association of HIV and LP, very few reports were there from India and the relation was not consistent.[21] We conducted a prospective study to assess the association of HCV, HBV, HIV infections with LP.

* *Corresponding author*.
E-mail address: seetharamka@gmail.com (K. Seetharamanjaneyulu).

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2. Materials and Methods

All patients with LP attending to our OPD, were recruited during the study period of 18 months (Jan 2017-Jun 2018). The demographic data and morphological patterns were documented. All were biopsied and histopathological features were recorded. All were tested for detection of HCV antibodies, Hepatitis B antigen and HIV antibodies by HCV-TRIDOT, HEPACARD and HIV –TRIDOT respectively.

3. Results

140 Lichen planus patients were enrolled. Lichen planus was seen in all age groups, however it was more common during 3^{rd} and 4^{th} decade. Most common age group involved was 21-30 years with mean age ± SD = 34.23 ± 12.99 years. There were 73 (52.1%)males and 67 (47.9%)females. 75.6% of the patients sought treatment with in 6 months of onset. Maximum duration of disease was 18 months and the mean duration was 4.15 ± 2.99 months. 22.9% of the patients were housemakers, 22.1% were labourers and 20.7% were students. Family history was noted in 2.9% patients. Pruritus was seen in 74% of the patients. Koebner's phenomenon was observed in 43% of the patients. Of the total patients 5% of the patients were smokers, 4% were alcoholics, 5% were both smokers and alcoholics. Extremities were commonly involved followed by trunk. The most common clinical variants were classical LP(35%) ,followed by hypertrophic LP(31%)(Table 1). Lichen planus transforming into Keratoacanthoma was observed in one case.(Figure 1). Oral involvement was seen in 23 (16.4%) patients. The most common site involved was buccal mucosa and reticulate pattern was seen in 79% of the oral LP. Nail involvement was seen in 3.57%. Longitudinal ridging was the commonest followed by pitting, pterygium and nail dystrophy. Cutaneous involvement alone was seen in 80% patients, Mucosal involvement alone was observed in 9.3% patients whereas both cutaneous and mucosal involvement was seen in 7.2% patients. On histopathology, hypergranulosis was most common, seen in 78% cases, followed by hyperkeratosis, basal cell degeneration and band like infiltrate in 76% cases each. Acanthosis was seen in 75% cases. and saw tooth rete ridges were seen in 57% cases. HBs Ag was positive in 4 cases, Anti HCV was positive in 3 cases, and HIV was positive in 3 cases.

Table 1: Sex wise distribution

Sex	Frequency	Percent
Female	67	47.9
Male	73	52.1
Total	140	100.0

Table 2: Clinical variants of lichen planus

Clinical variant	Frequency	Percent
Actinic	4	2.9
Bullous	3	2.1
Classical	45	32.1
Classical and oral	4	2.9
Follicular	6	4.3
Genital	4	2.9
Hypertrophic	37	26.4
Hypertrophic and oral	6	4.3
Linear	4	2.9
LP Pigmentosus	11	7.9
Nail	1	.7
Nail and oral	4	2.9
Oral	9	6.4
Palmoplantar	2	1.4
Total	140	100.0

Table 3: Histopathological features in patients with lichen planus

Histopathological finding	Percentage
Hyperkeratosis	76%
Hypergranulosis	78%
Acanthosis	75%
Saw toothed rete ridges	57%
Basal cell degeneration	76%
Band like infiltrate	76%
Melanin incontinence	70%
Civette bodies	12%

Table 4: Viral serologies in patients with lichen planus

HBsAg		Anti – HCV		HIV	
Positive	Negative	Positive	Negative	Positive	Negative
4	136	3	137	3	137

4. Discussion

Lichen Planus is a chronic inflammatory and immune mediated disease which affects skin, hair, nails, mucous membranes and appendages. Cell-mediated immunity plays a major role & humoral immunity plays a secondary role in the pathogenesis of LP. The major steps involved in the pathogenesis of LP are

1. LP- specific antigen recognition by CD4+Tcells and NK cells
2. Cytotoxic lymphocyte activation
3. Keratinocyte apoptosis

Lichen planus was reported commonly in 3^{rd} and 4^{th} decade. In our study most of the patients (55.7%) were between 21-40 yrs, similar to other studies.[22] The mean age involved in our study was 34.23 years, similar to Srivani etal in which the mean age was 37.1years.[23] LP in children was observed in 10% of the patients in our study(<18years), but it was varied from 5.43%[24] to 18%[25]in earlier studies. The low incidence reported by

Shankar et al, could be due to the inclusion of children less than 12 years,[24] Whereas the other studies included up to 18 years, which could be the reason for the higher prevalence in their studies. The childhood Lichen planus in the tropics was more, may be due to early exposure to infectious agents and other environmental triggers like trauma.[25] The elder population was effected rarely. Only 2.1% of the patients were affected in our study above the age of 60 years. LP was slightly more frequently reported in males. It was seen in 52.14% of males in our study, similar to other Indian studies.[22,26–28]The shortest duration of the disease was 15 days and the longest duration was 18months in our study. Mean duration of the disease was 4.1months and 47% of the patients were having the disease for 1-3 months, similar to the study by Shankar et al.[24]Most of the patients in our study were housewives(22.9%) followed by labourers (22.1%) and students(20.7%). Naldi etal, reported more frequently in labourers.[29] Family history was observed in 2.9% of the patients in the present study, similar to the study by Kachawa et al,[30] (2.13%). Pruritus was an important complaint in 74% of the patients in our study. Similarly Bhattacharya et al, Ireddy et al, Kachhawa et al and Abdallat et al reported pruritus in 79.3% to 82.6% of their cases.[24,30–32] Thus pruritus is a hallmark feature of Lichen planus. In our study 15% of the patients were diabetics, and 11% of the patients were hypertensive. Of these 3.6% of these patients were both diabetic and hypertensive. Urvashi et al had found Diabetes and hypertension in 10% and 7% of their 100 cases.[28] Increased association of hypertension(22.3%) was observed in a study by Sina et al among 134 oral lichen planus patients. The most commonly involved sites in our study were lower limb (62.1%) followed by upper limb(61.4%), trunk(25.7%), and oral cavity(16.42%). Face(4.9%), neck(2.1%), scalp(3.57%), nail(3.57%), genitalia(2.9%), palms and soles(2.8%) were less involved. Urvashi et al, Salah et al, and Bhutani et al also documented that the lower limbs were the common site for LP.[28,32,33]Venous stasis has been implicated as a likely pathogenic mechanism for common involvement of legs. . Palms and soles (14.7%) and Nails (17.9%) were involved more in the study by Urvasi et al,[28] whereas the involvement of palms (1.4%), soles (1.4%), and nails (3.6%) was less in our study. Oral cavity was involved in 23 patients (16.5%) in our study. Reticulate, erosive pattern was observed in 79%, 21% of patients with Oral LP respectively. Shinde et al , Urvashi et al and Sreedevi et al had reported higher percentage of, 40%, 42%, 56.4%, oral involvement respectively.[27,28,34] The lower incidence of oral lichen planus could be attributed to the fact that the majority of the patients with oral lesions chiefly present to Dental OPD or ENT OPD.[28] The most common clinical variant observed in our study was classical papular LP followed by hypertrophic LP. In our study classical LP and hypertrophic LP were present in 35% and 30.7% respectively but in study done by Urvashi et al , classical LP and hypertrophic LP were present in 58.9 % and 28.4% respectively.[28] Palmoplantar LP and Bullous LP were the least common variants found in our study constituting 1.4% and 2.9% respectively. The palmoplantar involvement may be due to isomorphic response to trauma on palms and soles.[25]

HCV and Hepatitis B viruses have been implicated in the pathogenesis of LP. Though the exact association between Hepatitis B and LP has not been established, hepatitis B vaccines are known to trigger LP, especially after second injection.[35]

In our study, 2.9% of the patients were positive for Hepatitis B infection. Nayaf et al, and Daramola et al had reported Hepatitis B infection in 6% and 15% of their LP patients respectively.[17,36] However studies from Uttarpradesh (India) and Nepal, had not shown any association with hepatitis B infection.[10,13] LP could be a cytotoxic reaction to keratinocytes expressing HBsAg and not epitopes shared by hepatocytes damaged by the virus.[13]

Many studies have suggested a role of HCV infection in LP. It was more commonly reported from Japan, USA, Italy and Spain. However studies from England, France and India had not shown any significant association between HCV and LP.[37] HCV infection was reported about 1.72 -3.3% of their LP patients.[7,10,12,15] HCV was more frequently associated with oral LP from Thailand, Pakistan and Saudi Arabia.[8,9,38] In India, studies conducted at Calicut, Kolkata, New Delhi, have failed to demonstrate a statistically significant association whereas studies conducted at Hyderabad and Bangalore have shown a significant association.[16] Pavani et al from Telangana had reported HCV infection in 12% of their Oral LP patients,[18] but a study from Puducherry did not show any association[16] The association was not consistent. In our study, 2.1% of the patients were positive for Hepatitis C virus infection.

In our study, 2.1% of the patients were positive for HIV infection. The mechanism of development of LP in HIV infected individual may be due to suppression of CD4 positive cells, differences in antigen presentation and altered immune response to antigenic stimuli.[39]

Keratoacanthoma arising from hypertrophic lichen planus was observed in one elderly male in our study with risk factors like smoking, alcohol and diabetes. (Figure 1) The site of involvement was lower limb and duration of the disease was 18 months. Biopsy showed pseudocarcinomatous hyperplasia with interface dermatitis in this patient. Pseudoepitheliomatous hyperplasia can be present in hypertrophic lichen planus and keratoacanthoma which can be confused with squamous cell carcinoma.[40,41] Malignant transformation in Lichen planus is a rare phenomenon. In oral lesions, it was reported with a frequency of 1-10%, but long-standing hypertrophic or ulcerative variants of cutaneous lichen planus have 0.4% risk of malignant transformation.[40,41] exposure, trauma, therapeutic agents

and chronic inflammation may predispose to malignancy.[42]

FIGURE 1 : Lichen planus transforming to keratoacanthoma

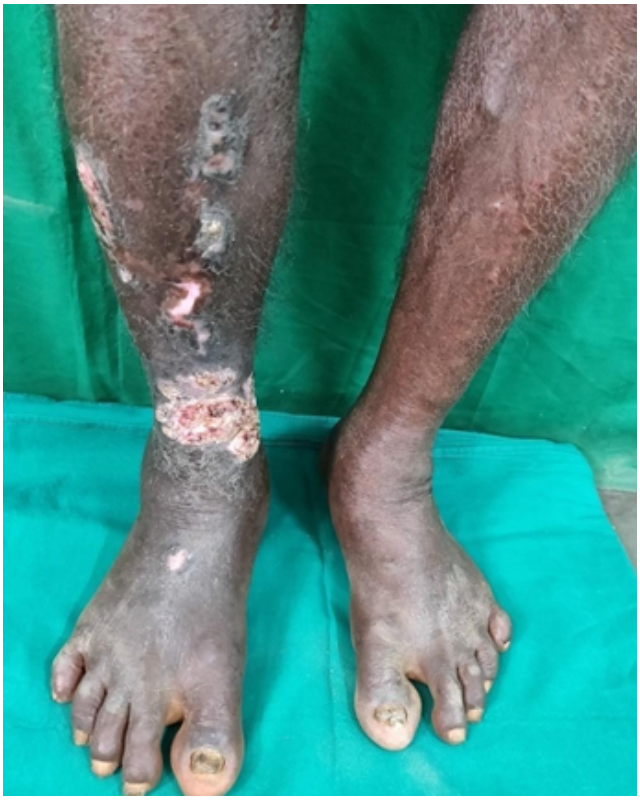

Fig. 1: Lichen planus transforming to keratoacanthoma

5. Conclusion

Lichen planus is a common papulosquamous disease commonly seen in middle aged adults usually on the extremities. Diabetes and Hypertension are associated with LP. Classical Papular LP was the commonest form, followed by Hypertrophic form. Malignant transformation can occur, so long term follow up is needed. Clinicopathological correlation has a pivotal role in providing optimal patient care. There is no relationship between LP and Hepatitis B, C and HIV virus. Hence we suggest that viral serology (HBV, HCV, HIV) for LP may not be done as a routine screening process.

6. Source of funding

None.

7. Conflict of interest

None.

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Author biography

Palakurthi Sri Sneha Senior Resident

K. Seetharamanjaneyulu Professor

G Venkata Ramana Professor

Satya Saya Assistant Professor

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