Symposium - Pediatric Dermatoses

Lichen planus in children

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ABSTRACT

Lichen planus in children is considered to be rare overall, though it does not appear to be so in Indian subcontinent. Most of the large studies on lichen planus in children have been undertaken in India. We review here the epidemiology, pathogenesis, clinical features, diagnosis, management and prognosis pertaining to lichen planus in children with emphasis on studies published from India.

Key words: Lichen planus, children, epidemiology, pathogenesis, clinical features, diagnosis, management, prognosis

INTRODUCTION

The term 'lichen' is probably derived from the Greek verb 'to lick'. However, the use of the term is adapted to a noun in both Greek and Latin for a symbiotic form of plant life. The dermatosis, lichen planus (LP) was first described by Erasmus Wilson in 1869 and is characterized by purple, polygonal, pruritic, papular eruption of unknown etiology affecting skin that can also involve the mucous membranes and the nails.

EPIDEMIOLOGY

Lichen planus is considered to be rare in children.[1] However, it does not appear to be uncommon in Indian subcontinent. Childhood LP may be common in Middle Eastern countries as well, with a study reporting an incidence of 7.5% among all registered LP cases in a clinic.[2] Most of the large studies have been reported from India, the largest one by the authors in 2009 involving 100 children below 18 years of age seen over 6.5 years.[3] Even in those studies which are published from the European countries, a proportion of patients were Indians. The largest study from outside the Indian subcontinent is by Balasubramaniam *et al*.[4] Of their 26 patients, 21 (80.8%) were from the Indian subcontinent while in the population where the patients hailed from, 58% were whites. We are yet to know the cause of rarity of LP in children and relative abundance in Indian children.

It has been hypothesized that the rarity of associated autoimmune conditions, exposure to drugs and dental restorative materials, infective agents and other environmental triggers that have been known to initiate lichen planus may be responsible for the overall rarity of LP in children. The scarcity of reports may further be due to overall rarity of LP in children, 2-3% of total LP occurring in children below 20 years of age.[5] Under-reporting may also influence the apparent rarity of childhood LP, as a study from India reported an incidence of 11.2% among all LP cases.[6]

LP is extremely uncommon in infants with the youngest documented case being a three-month-old child[7] while the reported earliest age at onset has been two weeks.[3] The mean age of onset in the larger studies has been 7.1-8.4 years.[3,5,8-10] Maximum proportion of patients had disease onset between 5 and 9 years in the study by Handa and Sahoo.[5] They observed that the lesions appeared earlier in boys than in girls.

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In the majority of the studies, either the sexes were equally affected or there was marginal male preponderance. Sharma and Maheshwari, however, observed that boys outnumbered girls by a ratio of almost 2:1.[8] This finding may be due to reporting bias. Girl children being less privileged in many Indian societies are not taken to the clinicians for apparently minor ailments.

Although LP is usually sporadic, there is a familial form of LP, comprising 1-2% of all cases of childhood LP.[11,12] Familial LP differs from the classical form clinically, with earlier age at onset, more generalized involvement, and more common mucosal involvement.[13] There is an increased tendency for erosive, ulcerative and linear forms, with prolonged course and frequent relapses.[13] In most of the studies, no familial LP was observed,[5,8,14] while Nanda *et al*[9] reported one girl with classic LP whose father had also similar complaints. The present authors observed familial LP in 2% of their patients.[3] Familial oral lichen planus (OLP) has also been described. Bermejo- Fenoll has described 13 clinical cases of OLP in six families, out of 249 cases seen by them. Thus, the incidence of familial OLP was 5.2% in their study.[15]

PATHOGENESIS

The exact etiology of lichen planus is unknown. Genetic linkage studies have been undertaken to establish HLA- association for lichen planus. Copeman *et al* found an association between familial LP and HLA-B7.[16] In a subsequent study by White and Rostom in Arab people, an association of HLA- DR1 and DR10 with lichen planus was observed.[17] The occurrence of LP in monozygotic twins indicates toward genetic linkage of LP. However, majority of the studies could not prove the genetic association for LP.

No precipitating factors were observed in the study by Kanwar *et al*[14] as well as by Handa and Sahoo.[5] Similarly, no history of previous drug intake was elicited in any of their patients by Sharma and Maheshwari[8] and Luis- Montoya *et al*.[18] On the contrary, Nanda *et al* found a precipitating factors in 22% of their patients in form of upper respiratory tract infection in four and viral exanthem in one patient before the onset of LP.[9]

Vaccination, viral infection and childhood lichen planus: In 1990, first case of LP occurring after hepatitis B vaccination was reported.[19] Many reports followed. The pathogenesis of such occurrence is not exactly known. An autoimmune reaction similar to chronic graft versus host disease has been suggested. Cross reactivity between hepatitis B antigen used in the vaccine and shared epitopes in the keratinocytes may be causative. LP may appear after any dose of vaccination and the latent period from the latest vaccination varies from many days to three months. Fifteen percent of patients in the study by Kanwar and De developed LP after vaccination, the mean interval between vaccination and LP onset was three years, ranging between three months and 11 years.[3] In those patients with shorter interval, hepatitis B vaccination could have been causally related. However, the association could have been weak because the rate of uptake of hepatitis B vaccination is much higher in general in children of the area from which the patients were drawn than that in the patient cohort.

Similarly, LP appearing in a child following combined measles- mumps- rubella and diphtheria- pertussis-tetanus- polio vaccine has been reported.[20] A viral cause, hepatitis C or human herpesvirus 7 infecting skin cells has also been suggested.[21,22] Similarly, infection with HCV has been speculated to be a risk factor for the development of lichen planus. Positive associations have been indicated in studies from Japan, Italy and even from India.[23] However, majority of the studies could not prove such an association.

Different mechanisms have been postulated for virus or vaccination induced lichen planus: cytopathic changes in the keratinocytes, autoimmunity directed against antigens expressed on keratinocytes, or triggering of autoimmune processes.[23] However, these associations seem fortuitous given the fact that prevalence of lichen planus is much lower than the prevalence of such infection or vaccination in the community.

CLINICAL FEATURES

The primary lesion of lichen planus is a violaceous, flat topped, polygonal, pruritic, papule, and represents commonest among all the morphologies of lichen planus in all age groups [Figures 1-5]. Classical (bilateral symmetrical papules and plaques on lower back, wrist and neck) LP was the most common variant observed in all the reported studies. The frequency was 42-76%.[3,5,8,9,14,18] The second most common variant differed between studies: lichen planus hypertrophicus (LPH,12%),[14] actinic LP

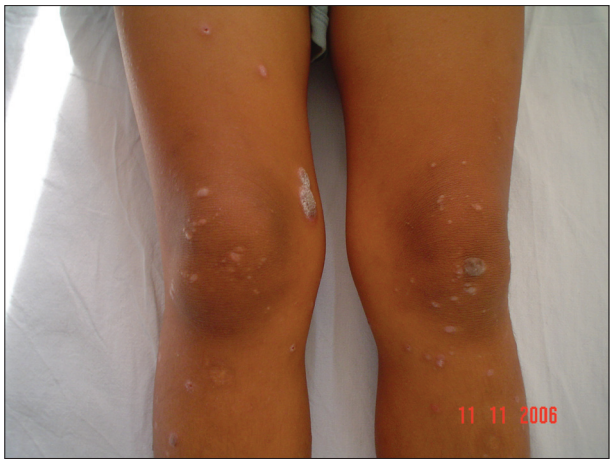

Figure 1: Papules and hypertrophic plaques of LP

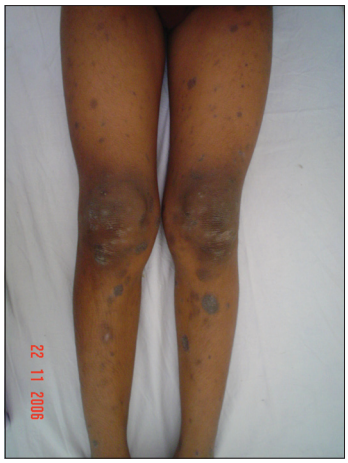

Figure 2: Papules and postinflammatory hyperpigmentation of LP

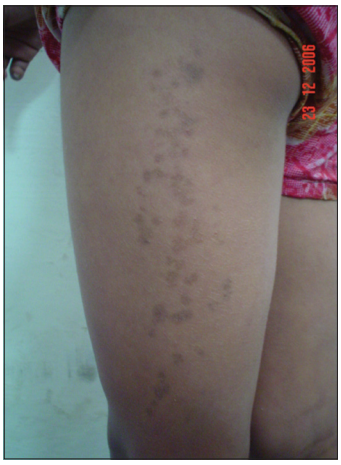

Figure 3: Linear lesions

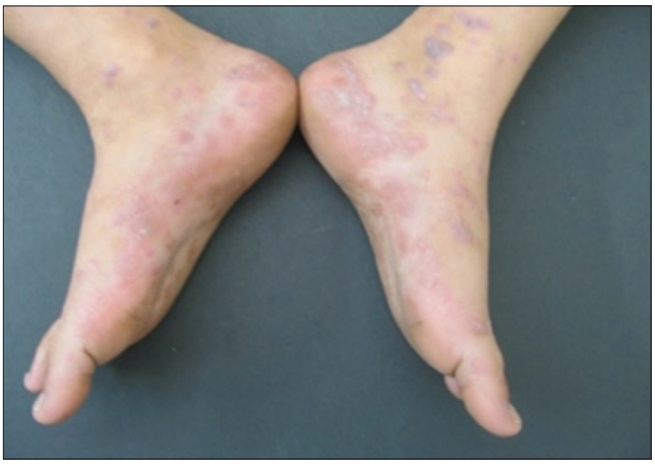

Figure 4: Papular and plaque lesions. Note involvement of the soles

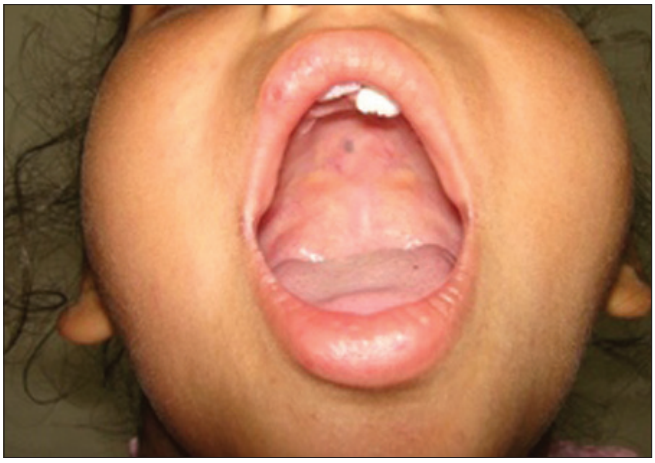

Figure 5: Single violaceous papule on hard palate

(11.5%),[5] eruptive LP (13%),[9] and LPH in (26%).[8] Postinflammatory hyperpigmentation is considered to be more intense in childhood LP. The summary of clinical features of childhood LP derived from large published studies is listed in Table 1.

Linear LP, LPH, and annular LP are known to be common variants while mucosal involvement is rare in children. Actinic LP is common in tropical and sub-tropical countries including India. Koebner's phenomenon is considered to be common in children with LP, varying between 24 and 28%.[5,8,14] However, it was observed in only 6% patients in the study by Kanwar and De.[3]

Initially linear LP was thought to be more common in children as compared to adults, but recent studies have shown results on the contrary. Linear lichen planus has been observed in 8-30.4% patients.[5,8,9,14] Both the patients in the study by Nanda *et al* had lesions along the lines of Blaschko. The high incidence of linear lesions in children may be due to increased

Table 1: Published studies on lichen planus in children (arranged chronologically according to the year of publication, adapted from Reference No. 3)

No	Author (year of publication)	No. of patients	M:F	Age (in years)	Age at onset (in years)	Site of onset	Morphology of lesions	Mucosal involvement	Nail involvement
1	Kanwar *et al*,[14] 1991	17	1.1:1	-	8 months-12	Limbs (41%) Trunk (30%) Scalp (6%)	Classical (76%) LPH (12%) Nail (6%) Eruptive (6%)	6%	-
2	Raybojad *et al*,[10] 1998	12	1.4:1	-	7.3 (2-13)	-	Eruptive (7/12) Bullous (2/12) Linear (1/12) Isolated nail involvement (2/12)	1/12	2/12
3	Sharma and Maheswari,[8] 1999	50	2:1	8.9 (7 months to 14)	8.4 (5 months-13)	Limbs (70%) Back (22%) Face (6%) Oral (2%)	Classical (60%) LPH (26%) Linear (8%) Eruptive (4%) Annular (2%) Actinic (2%)	30%	Nil
4	Nanda *et al*,[9] 2001	23	1.1:1	8.28 (2.5- 12)	7.14 (1.5- 11.8)	Extensor lower leg and ankle (52%)	Classical (70%) Eruptive (13%) Linear (9%) LPP (4%) Actinic (4%)	39%	Nil
5	Handa and Sahoo,[5] 2002	87	1.1:1	-	7.1 (8 months-12)	Lower limbs (51.7%) Limb overall (69%) Lower back (16.1%) Face (11.5%)	Classical (60.9%) Actinic (11.5%) LPH (9.2%) Linear (9.2%) Eruptive (6.9%)	13.7%	2.6%
6	Luis- Montoya,[18] 2005	24	1:1.2	-	-	-	Classical (43.5%) Linear (30.4%) LPP (13%) Actinic (4.3%)	4.3%	-
7	Nnoruka *et al*,[34] 2007	13	1.5:1	11.3		Limbs (69.3%) Trunk (23.1%) Genitalia (7.7%) Nail (7.7%) Mucosal 23.1%	Classic (61.5%) Linear (30.8%) LPH (23.1%) Eruptive (15.4%) Annular (15.4%)	23.1%	7.7%
8	Kanwar and De,[3] 2009	100	1.5:1	8.76 (2-18)	7.6 (15 days to 18)	Lower limbs (54%) Trunk (14%) Nails (12%)	Classical (42%) Eruptive (19%) Linear (12%) Nail (11%) LPH (8%) Actinic (5%) LPP (2%) Bullous LP (1%)	17%	19%

LPH: Lichen planus hypertrophicus, LPP: Lichen planus pigmentosus

tendency of children to traumatize themselves leading to Koebnerization. In general, lesions in linear lichen planus are disposed along solitary strips or segments of skin and are more extensive than those observed with Koebner's phenomenon. Multiple linear lesions resembling a zosteriform distribution may occur.

LPH is thought to be common in children. Sharma *et al* reported an incidence of 26% in children, lesions being present mainly on the extensor aspect of the legs.[8] Other authors have reported a lower incidence (8-10%).[3,9,14]

Actinic lichen planus is considered to be the disease of middle aged people (third decade) and has been reported commonly from Middle East. It occurs uncommonly in children. In the study by Sharma *et al*,[8] this variant of LP comprised 2% of cases while Handa and Sahoo[5] reported an incidence of 11.5%. The most common morphology of actinic LP was annular plaque with variable pigmentation at the center. It was observed that patients with actinic LP attended the clinic earlier (3.9 months) due to acute onset of the lesions and cosmetic reasons as compared to other variants of LP.[5] Five percent of the patients by

Kanwar and De had actinic LP, in three of whom it was difficult clinically to differentiate from polymorphic light eruption, and the diagnosis was established by histopathology.[3]

Nail involvement is rare in children while it occurs in 1-10% of adults. In different studies, nail involvement has been found in 0-8.7% of patients.[5,8,9,14,18] On the contrary, Kanwar and De have observed nail involvement in 19% of their patients.[3] Longitudinal ridging was the most common finding in 17%, followed by pitting in 15%, thinning of nail plate in 9% patients, trachyonychia, discoloration, nail dystrophy, subungual hyperkeratosis, onycholysis, nail splitting, thickening of nail plate and leukonychia in decreasing order of frequency. Nail changes of multiple types were seen in most of the patients (17/19, 86%). This corroborated the view of Tosti *et al* who considers nail lichen planus (NLP) in children to be under- estimated due to lack of skin and mucosal lesions which makes clinical diagnosis difficult.[24] The other reason may be general reluctance to perform nail biopsies in children. We however feel that nail biopsy is not required in every child with NLP where clinical manifestation is characteristic like nail plate thinning with longitudinal ridging and fissuring with or without pterygium. The presence of skin lesions along with nail changes suggestive of NLP such as trachyonychia or idiopathic atrophy of the nails does not require nail biopsy.

Scalp involvement is rare in children. None to nine percent of children with lichen planus have scalp involvement.[8,9] Five percent of Kanwar and De's patients had scalp involvement with papular lesions classical of LP. None had scarring alopecia (personal observation, unpublished data).

The most common site of onset has been the limbs, more commonly the lower limbs. Limbs have been the site of onset in 41-70% patients across studies.[3,5,8,9,14] Significant proportion of Kanwar and De's patients had disease onset in the nails..[3] This is an important finding as nail involvement as a whole is rare in children, a commonly perceived notion among dermatologists.

Different disease associations, some of them may be coincidental, have been described in different studies. Two of Nanda and colleagues' 23 patients had atopic dermatitis, and one patient each had hemophilia and bronchial asthma.[9] Cottoni *et al*'s one patient had associated active hepatitis.[25] In the study by Luis Montaya *et al*, one patient each had atopic dermatitis and vitiligo.[18] Five percent of Kanwar and De's patients had associated lichen nitidus.[3] Lichen planus appearing after generalized lichen nitidus has been described.[26] The possibility of lichen nitidus being micropapular variant of LP has not been convincingly excluded. Lichen nitidus can accompany clinical variants of LP and both conditions can occur together in the same patient.[27]

DIAGNOSIS OF LICHEN PLANUS IN CHILDREN

Diagnosis is essentially clinical. Histopathology, which can be done in diagnostic difficulties, reveals essentially similar findings as in adult lichen planus with hyperkeratosis, hypergranulosis, basal cell degeneration, pigmentary incontinence, effacement of the rete ridges and eosinophilic colloid bodies in the lower epidermis and superficial dermis. The issue about biopsy in nail lichen planus has already been discussed. Direct immunofluorescence of biopsy specimen from either skin or mucous membrane reveals shaggy fibrin deposit at the dermo-epidermal junction and colloid bodies.[28]

DIFFERENTIAL DIAGNOSIS

LP in children may have to be differentiated from lichenoid drug eruption, pigmented plane warts, lichen simplex chronicus (LSC), lichen amyloidosus, etc occasionally. The differential diagnoses of lichen planus in children according to morphology and site of involvement are listed in Table 2.

TREATMENT AND PROGNOSIS

There is no consensus regarding the treatment of childhood LP. Topical corticosteroids and oral antihistamines remain the treatment of choice in most patients with localized classic disease. For mucosal LP, the presence of dental amalgam should be looked for and its removal can be considered, if the lesions do not improve with commonly used medication. Topical treatment options for oral lichen planus include corticosteroids in orabase, topical tretinoin or isotretinoin gel, and topical tacrolimus or pimecrolimus. Oral agents that can be used for mucosal LP are systemic glucocorticoids, griseofulvin, hydroxychloroquine, azathioprine and mycophenolate mofetil. Intraleisonal triamcinolone may be used

Table 2: Differential diagnoses of lichen planus in children according to morphology and site of involvement

Type of lichen planus (LP)	Differential diagnoses
Lichen planus hypertrophicus	Lichen simplex chronicus (LSC)
	Lichen amyloidosus
	Lichenoid psoriasis
Follicular LP	Darier's disease
	Keratosis pilaris
	Lichen scrofulosorum
Linear LP	Lichen striatus
	Linear psoriasis
	Inflammatory linear verrucous epidermal nevus
Actinic LP	Lichenoid polymorphous light eruption
	Melasma
Annular LP	Annular psoriasis
	Granuloma annulare
Atrophic LP	Lichen sclerosus et atrophicus
Guttate LP	Guttate psoriasis
LP of oral mucosa	Contact dermatitis to dental amalgam
	Healing oral erosions of pemphigus vulgaris
LP of palms and soles	Psoriasis
	Focal palmoplantar keratoderma
	LSC

for both oral and cutaneous LP (hypertrophic) if the child can be convinced about the procedure. Otherwise, superpotent topical corticosteroids under occlusion can be used for LPH. Other options for cutaneous lichen planus are oral acitretin, dapsone, antimalarials, thalidomide, cyclosporine, azathioprine, mycophenolate mofetil etc. Topical tacrolimus has been used successfully in a six-year-old boy with scaly papuloplaque lesions.[29] Short courses of systemic steroids have been found to be effective in widespread eruptive disease, given at a dose of 1-2 mg/kg/day for one-two weeks and then weaned.[30,31] Nanda *et al* has observed that UVB phototherapy is safe and effective in children with acute widespread LP.[9] They found dapsone as a useful treatment for patients with chronic, recurrent LP. Ultraviolet B, both broadband and narrowband, has been used in generalized cutaneous lichen planus by Pavlotsky *et al*.[32] Complete response was achieved in 70%, and 85% of those were still in remission after a median of 34.7 months. The complete response rate and the need for higher cumulative exposure doses were not influenced by sex, age, skin type, presence of additional diseases, failure of previous treatment or disease duration. This result is encouraging; the carcinogenicity associated with phototherapy should not be of much concern as relapses in lichen planus is much less frequent compared to other childhood dermatoses such as atopic dermatitis. Oral acitretin has been used in a dose of 0.5 mg/kg/day for 12 weeks in a nine-year-old boy for acute extensive eruptive lichen planus.[33] The risk of premature closure of epiphyses has to be kept in mind if long-term use of acitretin is foreseen in a particular patient.

According to our departmental protocol, oral steroids namely prednisolone are given to those patients who have extensive/eruptive lesions. Subsequently, it is gradually tapered off with clinical improvement and replaced with oral dapsone (1.5 mg/kg/day) if required. Topical corticosteroids remain the treatment of choice in most patients with localized lesions. Triamcinolone acetonide is used for patients with symptomatic oral lesions and tazarotene gel (0.05%) is used topically on the periungual folds if few nails are involved. If multiple nails are involved, oral dexamethasone (2.5 mg/day) is used in two consecutive days per week (oral mini pulse).

Overall, we consider LP in children respond to appropriate treatment contrary to the belief that it has a protracted course and responds poorly to treatment. The long-term prognosis of childhood LP is not defined.

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