"A CROSS SECTIONAL CLINICAL STUDY OF PAPULOSQUAMOUS DISORDERS IN CHILDREN"

Submitted by

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M.D.

In

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Under the guidance of

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Date

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LIST OF ABBREVIATIONS

Ps –	Psoriasis
LP –	Lichen planus
LN –	Lichen nitidus
LS –	Lichen striatus
PR -	Pityriasis rosea
PRP –	Pityriasis rubra pilaris
PLC –	Pityriasis lichenoides chronica
PLEVA –	Pityriasis lichenoides et varioliformis acuta
LPP –	Lichen planopilaris
GPP –	Generalized pustular psoriasis
OPD –	Out patient department
HHV –	Human herpes virus
HIV –	Human immunodeficiency virus
UVB –	Ultraviolet - B

ABSTRACT

Background - Papulosquamous disorders include chronic recurring skin diseases such as psoriasis, pityriasis rubra pilaris, pityriasis rosea and lichenoid group of disorders like lichen planus, lichen nitidus, lichen striatus, pityriasis lichenoides chronica. These disorders are inflammatory, noninfectious with unknown etiology, which have distinct clinical and histopathological features.

Objectives - To study epidemiology and clinical pattern of papulosquamous disorders in children.

Method - It is a hospital based, cross-sectional, analytical study. One hundred and twenty five children, up to 18 years of age, suffering from papulosquamous disorders attending the Dermatology, Venereology and Leprosy out patient department of a tertiary care hospital were included in this study. Detailed history of illness, regarding age, duration, onset, symptoms, recurrence, family history of the diseases, pre-existing medical conditions and any history of drug intake were recorded. Each patient was subjected to a complete systemic and cutaneous examination. Relevant information about preceding history of fever, cough, sore throat was noted.

Complete hemogram and urine analysis was done. In children with psoriasis swab for culture from pharynx and perianal area was taken. C-reactive protein, anti-streptolysin-o titers and in patients with lichen planus hepatitis B surface antigen, hepatitis C antibodies were done, wherever necessary. Skin biopsy for histopathological examination was taken from the lesions.

Results - A total of 125 children with papulosquamous disorders were examined during the study period. Out of them 39 (31.2%) children were affected with psoriasis, 35 (28%)

with lichen planus, 11(8.8%) with lichen nitidus, 15(12%) with lichen striatus, 20 (16%) with pityriasis rosea, 4(3.2%) with pityriasis rubra pilaris and 1 (0.8%) child had pityriasis lichenoidis chronica . Incidence of papulosquamous disorders was highest among 11-15 years of age group, There was male preponderance as compared to females(M=71, F=54).

Conclusion - Papulosquamous disorders may impose both social and economic burden upon affected children and their family affecting quality of life. Hence, early diagnosis and management helps in improving quality of life of these children.

Keywords - Papulosquamous disorders, psoriasis, lichen planus, lichen nitidus, lichen striatus, pityriasis rosea, pityriasis rubra pilaris, pityriasis lichenoides chronica.

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INTRODUCTION

'Papulosquamous' is a term used for the skin lesions which are papular and located in the superficial skin layer (squamous layer). Children affected with papulosquamous disorders have skin lesions characterized by erythematous macules that progress to papules, develop scales, and is associated with itching. This group of diseases are chronic, persisting from weeks to months or even upto years.[1] Papulosquamous disorders include chronic recurring skin diseases such as psoriasis (Ps), pityriasis rubra pilaris (PRP), pityriasis rosea (PR) and lichenoid group of disorders like lichen planus (LP), lichen nitidus (LN) and lichen striatus (LS).

The childhood papulosquamous disorders differ in epidemiology, clinical features, treatment options, and long-term clinical and psychological outcome.[2] These diseases have a strong genetic background and is characterized by complex alteration in epidermal proliferation and differentiation. In childhood it is likely to have profound emotional, physical, social and psychological effects that ultimately influence the quality of life of children. The social development domain, which is one of the developmental milestones in a child, is particularly impaired.[3]

Depression probably plays a role in the impairment of quality of life among children affected with papulosquamous disorders.[4] Studies using scales from the "Impact of Chronic Skin Disease on Daily Life questionnaire" (that assesses stigmatization, severity of skin lesions, and physical symptoms), showed higher scores, which indicate that juvenile psoriasis and LP have a negative effect on the physical, emotional, and social functioning of children.[5]

Hence, children with papulosquamous disorders and their family members require special attention. It may disrupt family and social relationships, interfere with day to day life, and affect normal development.[6]

Very few Indian studies have been conducted to know the epidemiology of papulosquamous disorders amongst pediatric patients. So far, the available clinical studies on papulosquamous disorders are mostly of individual diseases like psoriasis, lichen planus or pityriasis rosea. This study includes all major papulosquamous disorders, like psoriasis, LP, PRP, PR, LN and LS.

The present study is conducted to know the epidemiologic characters and clinical pattern of papulosquamous disorders in children from north Karnataka.

OBJECTIVE OF STUDY

1. To study epidemiology and clinical pattern of papulosquamous disorders in children.

REVIEW OF LITERATURE

Papulosquamous disorders comprise of a group of skin diseases characterized by the presence of papules and scales. These conditions are not very frequent among pediatric patients attending dermatology out patient department (OPD).[6]

The exact prevalence of this group of diseases is unknown. Commonly observed papulosquamous disorders are psoriasis, pityriasis rubra pilaris, pityriasis rosea, pityriasis lichenoides, lichen planus, lichen nitidus and lichen striatus.[2] Among these, psoriasis, pityriasis rosea and lichen planus are commonly seen. These disorders are inflammatory, non-infectious and of unknown etiology, having distinct clinical and histopathological features.

PSORIASIS

Psoriasis is a papulosquamous disorder with a variable clinical spectrum and unpredictable clinical course, characterized by remissions and relapses.[7]

Epidemiology:

Only few epidemiologic studies provide estimates on the prevalence of psoriasis in children. In an UK based study, prevalence of psoriasis was 55/10,000 children aged 0 to 9 years and 137/10,000 children and adolescents aged 10 to 19 years, with a higher prevalence in girls than in boys.[8] In Germany, 71/10,000 children were affected by psoriasis.[9] In Netherlands, a study conducted by dermatologists and general practitioners suggested a prevalence of 37/10,000 children aged 0 to 10 years and 109/10,000 children aged 11 to 19 years.[10] In an epidemiological study of various dermatoses in school children aged 6-14 years from North India, the point prevalence

of psoriasis was found to be 0.02%. Psoriasis was the underlying etiology in 15% of all cases of erythroderma in children (< 12 years) from Delhi, India.[2]

The incidence of psoriasis may be increasing over time as suggested by a more recent study of both adults and children in the UK, that demonstrated an incidence of 140/100,000 population.[11] Most of the children were found to have chronic plaque psoriasis, followed by guttate psoriasis, sebo-psoriasis and least commonly found was pustular psoriasis.[12] In a study conducted in Ahmedabad, India, out of the 700 children aged < 14 years, who were examined in the dermatology OPD, psoriasis was observed in 0.99%.[13]

In a study conducted in JIPMER, Pondicherry, India, psoriasis was observed in 1.44% out of the 2100 children aged < 14 years, who were examined in the dermatology OPD.[14] In a study of 419 patients of childhood psoriasis from a tertiary care hospital in north India, psoriasis constituted 0.3% of all the dermatology outpatients and 12.5% of the total psoriasis patients.[2]

Onset of psoriasis in childhood has been reported in as many as 40% of adult patients, with at least one-third of the patients demonstrating features of psoriasis before the age of 16 years.[7] Onset of disease before the age of 20 years was reported in 35% of adult patients by Farber and Nall.[15] Another study by Watson et al[15] have reported that, out of 2144 patients 12% of adult patients had onset of psoriasis before the age of 10 years, and in 25% between the age of 2 and 19 years. A Scandinavian study reported that 45% with patients of psoriasis had disease onset before the age of 16 years.

In only 2% patients psoriasis is diagnosed prior to the age of 2 years and in 10% before 10 years of age. Establishing the diagnosis in infancy may be challenging because of limited involvement or an atypical appearance.[16] In an Australian study

by de Jager et al,[10] 16% of pediatric patients with psoriasis were < than 1 year of age, 27% < 2 years, 7% were < 5 years and 45% < 12 years of age.

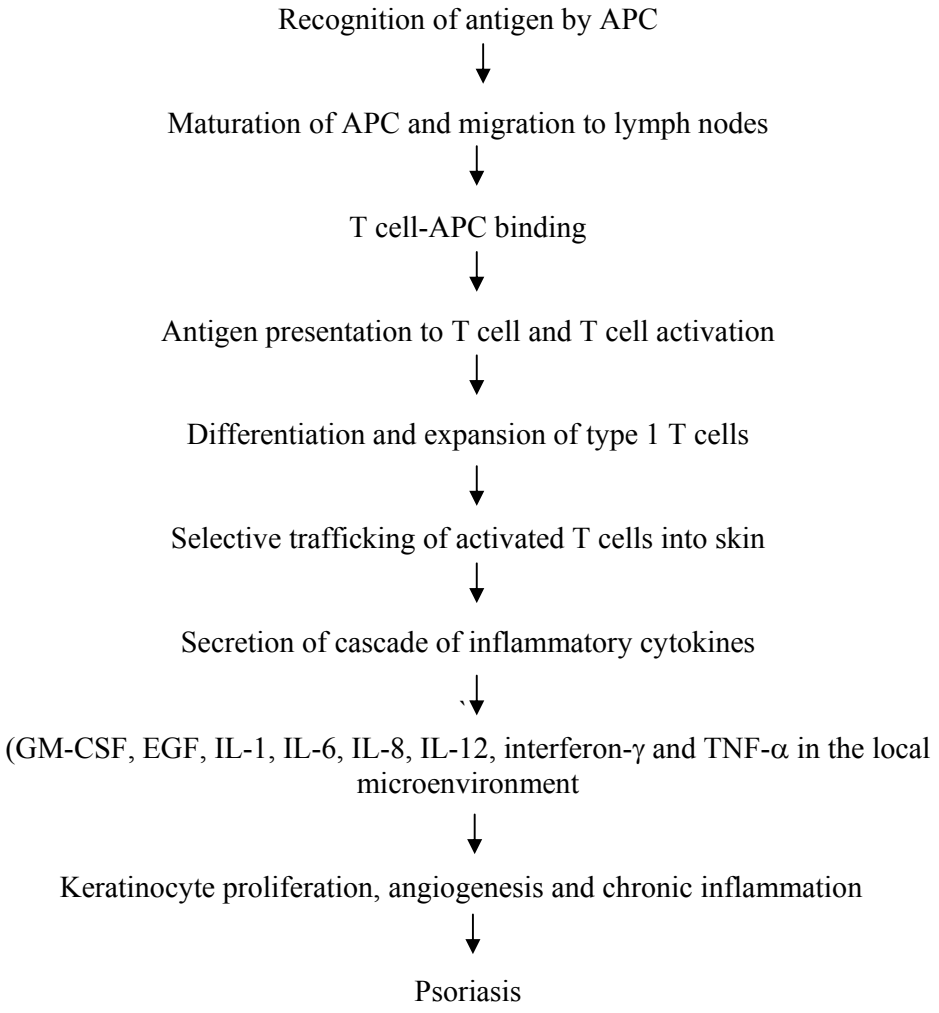
In several studies, it has been reported that girls are more commonly affected with psoriasis as compared to boys.[17] Family history of psoriasis is important when one considers the diagnosis of psoriasis in a child. It has been seen that earlier the onset of psoriasis, greater is the probability of a positive family history. There exists compelling evidence of genetic predisposition in psoriasis, with lifetime risks of 4%, 28% and 65%, if neither, one or both parents, respectively, are affected.[18] Familial prevalence is greater in childhood psoriasis (58%) than in adult-onset psoriasis (10-30%).[15] Among patients who develop psoriasis in childhood, 49% have first degree relatives affected with it as compared to 37% in adult psoriasis.[19]

Etiopathogenesis

The etiology and pathogenesis of psoriasis remains puzzling. Like all forms of auto-immunity, there exists an interplay between genetic and environmental factors. Susceptibility is likely genetic, but environmental triggers are required to initiate disease activity.[7] T-cells play a pivotal role in the pathogenesis of psoriasis. Cytokine pattern is skewed towards Thl response.[16]

Group A β-hemolytic streptococcal antigen is a well accepted inciting factor in the development or a flare of guttate or plaque-type psoriasis.[20] Shelley et al[20] elicited a local pustular inflammatory reaction upon intradermal injection of the *Streptococcus pyogenes* antigen. Baker et al[20] demonstrated that recurrent tonsillitis was a provoking factor in the occurrence of generalized pustular psoriasis (GPP).

Immunopathogenesis of psoriasis:[21]



(APC-Antigen presenting cell, GMCSF-granulocyte macrophage colony stimulating factor, EGF-epithelial growth factor, IL- interleukin, TNF - tumour necrosis factor)

Clinical Features of psoriasis in children:

Pediatric psoriasis has been divided into three main types:[7]

- Infantile psoriasis
- Early onset psoriasis
- Pediatric psoriasis with psoriatic arthritis

The various clinical presentations of psoriasis in childhood include plaque-type, guttate, erythrodermic, napkin and nail-based disease though different forms may occur in the same patient at different times.[1] Childhood psoriasis has been reported to differ from psoriasis in adults in that it is more frequently pruritic with a preponderance in girls, and the lesions are relatively thinner, softer, and less scaly.[2]

Plaque type is the most common form of the disease (Fig 1). Psoriasis in children is more frequently precipitated by infections and manifests as acute guttate psoriasis.[22] However, Indian studies have shown that children present with established plaque type of the disease more often. Facial involvement is a frequent (Fig 2) observation in majority of the reports, which varies from 18 to 46%, whereas mucosal involvement has been rare in Indian children.[7]

A study from north India reported that extensors of the legs were the most common initial site affected, followed by scalp. Classical plaque psoriasis was the most frequent clinical presentation, followed by plantar psoriasis. Nail involvement was observed in 130 cases. Pitting was the most common nail change, followed by ridging and discoloration. Five children had psoriatic arthropathy. Koebnerization was observed in 27.9% of patients.[7]

In plaque psoriasis lesions are 5-10 cm in diameter, located symmetrically over the extensor surfaces of the limbs.[8] The involvement of flexural surfaces is particularly common in young children. Napkin area is frequently the first site affected (Fig 3), with well-defined erythema devoid of scales.[16]

Guttate psoriasis develops abruptly, often in response to a streptococcal infection of the throat or skin. Infection may be confirmed by culturing a throat swab and by measuring the serum antistreptolysin-O titre.[23]

The possibility that congenital psoriasis may be more common in the presence of "cradle cap" in infants that may represent an early sign of psoriasis. A hypothesis that remains untested is the possibility that congenital psoriasis may result from the transmission of a blood-borne factor across the placenta.[24] Children may present with localized, and or disseminated pustular psoriasis, sometimes with an annular or circinate appearance, which may disappear during puberty and is replaced by the vulgaris type.[25]

GPP is a rare type of psoriasis first described in 1910 by von Zumbusch and is perhaps considered the most severe form of psoriasis. In a study of 112 children with psoriasis by Nanda et al,[20]only 0.9% presented with GPP. A review of 1,262 cases of childhood psoriasis found that only 0.6% children were affected with pustular psoriasis.[26]Several trigger factors have been proposed in GPP, including medications, bacterial infections, sun burn, use of coal tar, emotional stress, vaccination, hypocalcemia and withdrawal of corticosteroids. The onset of childhood GPP is generally abrupt and is associated with toxic features.[26]

GPP can be divided in two groups: patients with a past history of psoriasis vulgaris (pso + GPP) and patients without a history of psoriasis vulgaris (pso - GPP). The groups differ in several aspects. In the "pso + GPP" group corticosteroid withdrawal is the most common precipitating factor, whereas in the "pso - GPP" group the disorder is frequently precipitated by infections. Age of onset of pustular outbreaks is earlier in the "pso - GPP " group. The "pso + GPP"group is significantly related with HLA-A1, HLA-B37, and HLA-DRw10, which is closely related to psoriasis vulgaris in the studied populations, whereas this correlation was not detected in "pso – GPP."[26]

In children four clinical patterns of pustular psoriasis have been described: generalized pustular psoriasis (von Zumbusch), annular pustular psoriasis (APP), exanthematic pustular psoriasis and localized pustular psoriasis.[27] Classically it presents as widespread sheets or pools of sterile pustules on brightly erythematous skin that resolves within 3-4 days. Pustulation is typically associated with fever and toxicity.[26]

The incidence of nail lesions as the only clinical feature of psoriasis is rare in children. Manchanda et al [28] found the prevalence of nail involvement to be 37.81% (boys > girls) among children who had psoriasis.[28] Psoriatic nail disease has many clinical signs like oil drop sign, pitting, subungual hyperkeratosis, onycomadesis and onycolysis.[29]

Rare presentations:

Certain clinical variants like erythroderma, arthropathy, and localized or generalized pustular psoriasis are rare in children. Many types of psoriasis seen in adults are described rarely in children, like linear and follicular forms [30] (Fig 4)**.**

Histopathology:

Histopathological features show epidermal proliferation. The epidermis is thickened, with elongation of rete ridges, acanthosis, parakeratosis and thinning of granular layer. Immediately below the parakeratotic stratum corneum, there is collection of neutrophils (spongiform pustules of Kogoj). Munro's microabscesses are commonly seen in the early lesion, located within the parakeratotic stratum corneum (Fig 5). In dermis, the dermal papillae are elongated and edematous. There is vasodilatation of dermal blood vessels with an infiltration of lymphocytes. [22]

Treatment

Treatment goals for pediatric patients with psoriasis include improving both physical and psychological symptoms and minimizing the adverse effects of psoriasis or its therapy on future health and psychosocial development.[31]

Table 1: Treatment options available for pediatric psoriasis

Topical Agents	Systemic agents	Phototherapy
Moisturizers	Methotrexate	Narrow-band UVB
Topical Steroids	Retinoids	
Tar	Cyclosporine A	
Salicylic acid	Biological agents	
Anthralin		
Vitamin D analogues		
Retinoids		
Calcineurin inhibitors		

Tacrolimus 0.1% ointment results in excellent improvement to complete clearance in pediatric patients with facial or inverse psoriasis. Response in pediatric patients may even be superior to those of adults.[32]

LICHEN PLANUS

Lichen planus (LP), was first described by Erasmus Wilson in 1869 as a pruritic papulosquamous disease of unknown etiology, affecting skin, mucous membranes, hair and nails. It is characterized by purple, polygonal, pruritic, papular eruptions that has a typical histopathological picture.[33]

Earlier, LP used to be considered rare in children. Patients < 20 years of age accounted for 2% to 3% of the total number of reported cases. However, Luis-Montoya et al [34] have, reported in their study on LP that 16% of patient were < 20 years of age, and 10.2% were in the pediatric age group.[34]

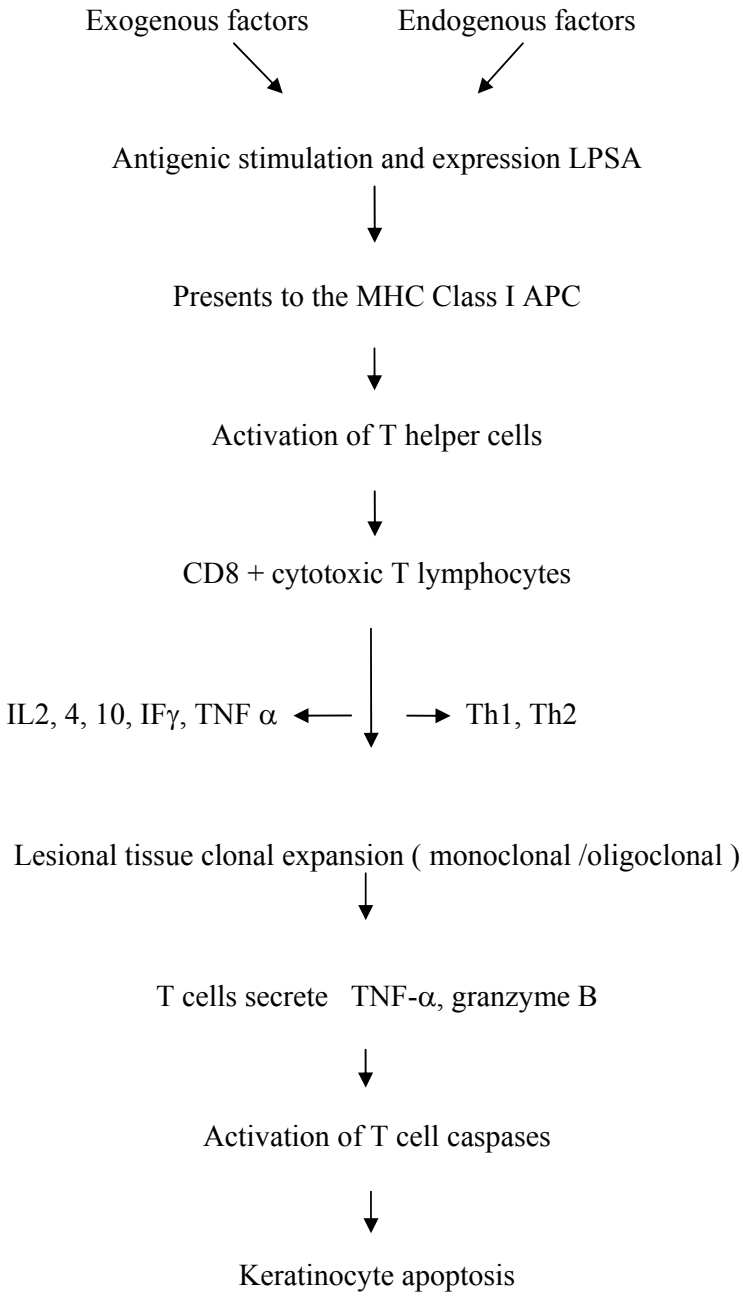
Pediatric LP does not appear to be uncommon in the Indian subcontinent. Childhood LP may be common in the middle east as well, with a study reporting an incidence of 7.5% in children, among all registered cases of LP in a clinic.[35] Most of the large studies on childhood LP have been reported from India, the largest one was in 2009, involving 100 children < 18 years seen over a period of 6.5 years. Majority proportion of the patients had disease onset between 5 to 9 years. A study by Handa et al [35] had recorded earlier appearance of skin lesions in boys than in girls.

Pathogenesis

LP represents a cell-mediated immune response to an induced antigenic change in the epidermal cells in a genetically predisposed individual. There are 3 sequential stages[36].

- LP specific antigen recognition
- Cytotoxic lymphocyte activation
- Keratinocyte apoptosis

Immunopathogenesis of lichen planus:



(LPSA: Lichen Planus Specific Antigen, MHC –Major histocompatibility complex

APC: Antigen Presenting Cell, IL- interleukin, TNF - Tumour necrosis factor)

Clinical features:

The primary lesion of lichen planus is a violaceous, flat topped, polygonal, pruritic papule, and represents the commonest of all the morphologies of lichen planus in all age groups. Classical (bilateral symmetrical papules and plaques on hand, wrist, neck, chest, lower back) LP was the most common variant observed in all the reported studies (Fig 6 and 7). The frequency was 42-76%.[33] The high incidence of linear lesions in children is probably due to their increased tendency to traumatize themselves leading to Koebnerization. Sharma et al[33] have reported occurrence of LP in 26% of the children in their study, lesions distributed mainly over the extensor aspects of the legs.

Oral LP can be divided into hyperkeratotic, reticular papular, plaque, atrophic, erosive/ulcerative types.[37]

Nail involvement occurs in 1-10% of adults, but it is rare in children. In different studies, nail involvement has been found in 0-8.7% of patients.[33] Common changes are exaggeration of the longitudinal lines and linear depressions, pterygium unguis, atrophy of the nails, twenty-nail dystrophy, longitudinal melanonychia, hyperpigmentation, subungual hyperkeratosis and onycholysis.[38]

Koebner effect are frequently found in LP (Fig 8). Linear LP lesions are few centimeters in length, but long, narrow linear lesions extending the whole length of a limb may occur (Fig 9). Such cases may overlap with epidermal nevi.[38]

Actinic or subtropical LP generally occurs in children with dark skin living in tropical countries. Lesions occur on exposed skin (usually face and hands) as well defined annular or discoid patches, which have a deeply hyperpigmented centre surrounded by a striking hypopigmented zone.[38]

Lichen planopilaris (LPP), a term coined by Pringle in 1895, and follicular lichen planus, a name proposed by Silver et al,[35]are clear terms describing the clinical syndrome of lichen planus associated with cicatricial alopecia of scalp.

LP affects the scalp in a distinctive clinical and histopathologic pattern that selectively involves hair follicles and eventually causes cicatricial alopecia[39] (Fig 10). Lesions typical of LP are not commonly seen on the scalp but at other sites. "Follicular lichen planus of the scalp," "lichen planopilaris," "folliculitis decalvans et atrophicans," and "Graham Little–Piccardi–Lassueur syndrome" are all clinical syndromes of LP. These involve the scalp and appear as keratotic follicular papules with an evolving, often scarring alopecia.[40]

Histopathology:

This consists of hyperkeratosis, wedge-shaped hypergranulosis, irregular acanthosis, vacuolar degeneration of basal layer with the presence of intraepidermal or subepidermal colloid bodies, saw-toothing of the rete ridges and papillary dermal band-like infiltrate[41] (Fig 11).

Direct immunofluorescence (DIF) of lesional skin shows ragged fibrin band at the basement membrane zone (BMZ) and clusters of colloid bodies (with IgM and C_3; to a lesser extent with other classes of immunoglobulin) in almost all patients.[42]

In 1983, Olsen et al [42] demonstrated LPSA by indirect immunofluorescence technique using autologous lesional skin. LPSA is unique to lichen planus and is expressed in the stratum granulosum and stratum spinosum of patients with LP. It can be demonstrated by indirect immunofluorescence using the patient's serum and

autologous lesional skin that helps to differentiate atypical cases of lichen planus from other dermatoses.[42]

In actinic LP there is foci of spongiosis, parakeratosis and pigmentary incontinence.[38] In LPP, there is absence of arrector pili muscles and sebaceous glands. A perivascular and perifollicular lymphocytic infiltrate is present in the reticular dermis. Superficial perifollicular scarring, fibrosis and loss of hair follicles are seen , which are replaced by linear tracts of fibrosis[43] (Fig 12).

Differential diagnosis

LP in children has to be differentiated from lichenoid drug eruption, pigmented plane warts, lichen simplex chronicus (LSC) and lichen amyloidosis.[33]

Treatment

Topical corticosteroids and oral antihistamines remain the treatment of choice in most patients with localized disease. Other topical treatment options for LP include tretinoin or isotretinoin gel, and tacrolimus or pimecrolimus.

Oral agents are systemic glucocorticoids, azathioprine and mycophenolate mofetil. Intraleisonal triamcinolone may be used for both oral and cutaneous LP (hypertrophic). [33]

LICHEN NITIDUS

Lichen nitidus was first described in 1907 by Pinkus. It is characterized by multiple, discrete, 1 to 2 mm, flesh-colored, shiny papules (Fig 13). It most commonly involves the penis, arms, forearms, chest, and abdomen.[44] The lesions are usually asymptomatic, or sometimes mildly pruritic. The disorder is more prevalent in children and young adults. All races and both sexes are equally affected. Clinical

variants of lichen nitidus include confluent, vesicular, hemorrhagic, palmar and plantar, spinous, follicular, perforating, linear, and generalized forms.[38]

Histopathology:

The epidermis is thinned out with occasional basal cell degeneration. There are focal areas of a well-circumscribed, intense inflammatory infiltrate situated in the upper dermis. It is composed of lymphohistiocytes, a few plasma cells and a few Langhans giant cells. The rete ridges are elongated in a claw-like fashion to encircle each focus of infiltrate ('claw clutching a ball').[45]

LICHEN STRIATUS

Lichen striatus occurs in children from 5-15 years of age. It manifests as unilateral eruptions along Blaschko's line on the extremeties, trunk and neck, as either a continuous or interrupted bands composed of minute, slightly raised erythematous papules, which may have a scaly surface[46] (Fig 14).

Histopathology:

Epidermis shows focal parakeratosis, acanthosis and spongiosis. There is vacuolar alteration of the basal layer and necrotic keratinocytes. Superficial perivascular lymphohistiocytic infiltrate (Fig 15) is seen in the dermis.

In the papillary dermis, the infiltrate may have a band like distribution with extension into the lower portion of the epidermis. A very distinctive feature is the presence of inflammatory infiltrate in the reticular dermis around the hair follicles and eccrine glands.[45]

PITYRIASIS ROSEA

Pityriasis rosea (PR) is an acute or sub-acute, symptomatic or asymptomatic condition affecting mainly young adults and apparently healthy children.[47] The characteristic skin lesions typically last from 2 to 10 weeks.[48]

Etiology:

Epidemiologic data indicating an increased incidence of PR during the fall, winter and spring suggests an infectious cause in the etiology of PR.[49] Other evidence suggesting viral reactivation includes occasional relapses and occurrence of PR during altered immune status. There is slightly increased prevalence in patients with decreased immunity, such as pregnant women and bone marrow transplant recipients.[47]

The raised anti-streptolysin O titers suggests a possible involvement of streptococcus in PR. *Chlamydia* and *Mycoplasma* are both highly infectious and known to cause upper respiratory tract infection that may precipitate PR.[48] Relapses are rare, suggesting immunity following the first attack. Clustering of cases has been frequently reported, supporting an infectious etiology.[50]

Many drugs have been reported to cause pityriasis rosea or PR-like rashes (allopurinol, arsenic, bismuth, barbiturate, captopril, gold, hydrochlorothiazide, metronidazole, nimesulide, d-penicillamine, isotretinoin, ketotifen and omeprazole).[36] Ampicillin and systemic corticosteroids have been found to exacerbate PR.[36] Association of PR with new, unwashed garments or old ones that have been in storage for long period, have been reported.[51] Recent studies have concentrated on finding an association between PR and HHV-6/HHV-7.[52]

Pathogenesis

It has been postulated that autoimmune mechanism may play a role in the pathogenesis of PR. According to one study, patients with PR have been demonstrated to have T-lymphocytotoxic antibodies (28%) and are also significantly more likely to have antinuclear antibodies.[53]

Clinical features:

The first sign in PR is classically a solitary erythematous or salmon coloured, oval or round lesion known as 'herald patch' or 'mother patch' which usually appears on the trunk, sometimes on the neck or extremities, rarely on face or penis (Fig 16). The centre of the herald patch is wrinkled with a darker red peripheral zone separated by a collarette of fine scales. Incidence of herald patch varies from 40%-76% in various studies.[51,53] In an Indian study the incidence of herald patch was found to be 70%, the commonest site being trunk (40%).[38] This is followed by secondary eruptions. The interval between primary and secondary eruptions is variable (2 days to 2 months), but usually 7-14 days. The lesions are symmetrical and localized mainly over the trunk, adjacent areas of neck and extremities[36] (Fig 17).

Clinical types:[38]

- Inverse PR
- Pustular PR
- Vesicular PR
- Purpuric PR
- PR urticata.
- Gigantean PR
- Erythema multiforme like PR

Histopathology:

Epidermis shows patchy parakeratosis, acanthosis, spongiosis and mild exocytosis. Vascular dilatation and extravasation of RBCs is common in the upper dermis and may extend into the lower layers of the epidermis. Superficial lymphohistiocytic dermal infiltration is seen[54] (Fig 18).

Treatment

PR is a self resolving condition most of the time. Asymptomatic and self-limiting cases require no treatment. If itch is troublesome, or the appearance is distressing, a topical steroid and broadband ultraviolet irradiation (UVB) can be used. Oral erythromycin 250mg qid / day for 2 weeks has been used in PR with clearance of lesions.[38] If HHV-6 or HHV-7 is the cause of the eruption, antiviral drug like acyclovir 800 mg five times daily for 1 week should be used.[52]

PITYRIASIS RUBRA PILARIS

Pityriasis rubra apilaris (PRP) is a chronic papulosquamous disorder of unknown etiology characterized by keratotic follicular papules, reddish orange scaly plaques and palmoplantar keratoderma.[55] It was first described by Tarral in 1828 and was named by Besnier in 1889. In adults both sexes are equally affected but in children males are more commonly affected (M : F= 3:2).[56]

Grifiths divided PRP into 5 categories, presented in table 2:[56]

Table -2: Classification of PRP [56]

Type 1 : Classic adult type:	Most common form of PRP, accounting for more than 50% of all cases. It has the best prognosis.
Type II : Atypical adult type:	This form accounts for about 5% of all cases of PRP.
Type III : Classic juvenile type:	Accounts for about 10% of all cases of PRP. Onset is within the first 2 years of life.
Type IV : Circumscribed juvenile type	This form accounts for about 25%.Occurs in prepubertal children.
Type V : Atypical juvenile type:	This form accounts for about 5% of all. Early onset and runs a chronic course.
Type VI : HIV-associated PRP.	Patients with HIV infecton may have nodulocystic and pustular acneiform lesions. These patients tend to be resistant to standard treatments, but they may respond to antiretroviral therapies.[38]

Clinical features:

PRP is characterized by small follicular papules with a central keratotic plug, surrounding salmon colored erythema (Fig 19). There are few disseminated yellowish pink scaly plaques surrounding islands of normal skin. Hyperkeratosis of the palms and soles are presenting features[55] (Fig 20). Keratoderma of palms and soles develops in the majority of affected children. This has been referred to as a "keratodermic sandal". Keratoderma shows a sharply demarcated border. The nails are dystrophic in 13% of patient with thickened, transverse striations, and subungual debris.[56]

Clinical diagnostic criteria for PRP proposed by Gelmetti et al,[56] is as follows:

Primary diagnostic criteria:[56]

- Follicular papules (knees, ankles, hands)
- Keratoderma
- Cephalic rash
- Salmon colour

Secondary Diagnostic criteria:

- Edema (nonpitting)
- Nail changes
- Ectropion
- Koebner phenomenon
- Pruritus
- Mucosal changes
- Characteristic histopathology
- Resistance to therapy

Histopathology:

PRP demonstrates psoriasiform hyperplasia with alternating orthokeratosis and parakeratosis in a check board pattern (Fig 21). There is shoulder parakeratosis with thick suprapapillary plates, broad rete ridges and narrow dermal papillae. Acantholytic dyskeratosis, follicular plugging and hypergranulosis are most predictive of PRP. An inflammatory dermal infiltrate comprised of eosinophils and plasma cells can also be observed.[55]

Treatment

The treatment option for localized PRP include topical glucocorticoids, topical vitamin D analogues, topical and systemic retinoids.[57] Various therapeutic option for PRP has been presented in table 3.

Table – 3: Treatment options for PRP

Topical Treatment	Systemic Treatment
Emollients	Retinoids
Calcipotriol	Methotrexate
Retinoic Acid(0.05%)	Ciclosporin
Topical Steroids	Psoralens/Ultravoilet A
Keratolytic Agents • Salicylic Acid (2-5%) • Propylene glycol (20%) • Urea (10%)	Miscellaneous agents • Stanozolol • Zidovudine • γ interferon • Azathioprine
Hydrating Agents [56]	

PITYRIASIS LICHENOIDES:

Pityriasis lichenoides is a spectrum of cutaneous eruptions in children and young adults considered within the group of disorders called parapsoriasis.[57]

It has been subdivided into

1) An acute form (acute guttate parapsoriasis): parapsoriasis varioliformis / pityriasis lichenoides et varioliformis acuta (PLEVA) / Mucha-Habermann disease.
2) A chronic form : pityriasis lichenoides chronica / guttate parapsoriasis.[58]

PLEVA is a polymorphous eruption that begins as asymptomic to pruritic symmetrical 2-3mm oval or round, reddish brown macules and papules. The papules occur in crops and rapidly evolve into vesicular, necrotic and sometimes pruritic lesions. Crusts develop and gradually resolve with or without a varioliform scar.

Pityriasis lichenoides chronica (PLC) is characterized by a small, firm lichenoid papule 3–10 mm in diameter, and reddish brown in colour. An adherent'mica-like' scale can be detached by gentle scraping to reveal a shiny brown surface. Post-inflammatory hypopigmentation may occur, and is occasionally persistent, but scarring is unusual in PLC.[38]

Histopathology:

Epidermal changes shows mild spongiosis, confluent parakeratosis. There is vacuolar alteration of the basal layer and few necrotic keratinocytes.

In dermis superficial perivascular and lichenoid infiltrate is seen, composed of lymphocytes which extend into the epidermis. Melanophages and small number of extravasated erythrocytes are seen in the papillary dermis.[58]

Treatment:

Topical steroids and tacrolimus have been used in the treatment of PLC. Systemic treatment is comprised of erythromycin or tetracyclines, azithromycin, systemic steroids, methotrexate, γ-globulins, cyclosporine, oral retinoids, dapsone, biologicals and narrow band UVB photo therapy.[59]

Papulosquamous disorders are not an uncommon entity among children. There is a considerable difference in the etiological factors, clinical patterns and treatment options in children as compared to that of adults. Hence, further studies in this aspect are needed so, as to fill the gaps in the existing literature and further enrich our knowledge regarding papulosquamous disorders in children.

METHODOLOGY

SOURCE OF DATA:

A hospital based, cross-sectional, analytical study on clinico- epidemiological pattern of papulosquamous disorders in children was conducted in the department of Dermatology Venereology and Leprosy of B.L.D.E.U's Shri. B.M. Patil Medical College Hospital and Research Centre, Bijapur. One hundred twenty five children suffering from papulosquamous disorders were recruited from the out patient section of the department. Informed consents were taken from their parents. The study was conducted between the period of October 2010 to September 2012.

METHOD OF COLLECTION OF DATA:

Inclusion Criteria:

Pediatric population up to 18 years of age, irrespective of gender, suffering from papulosquamous disorders like psoriasis, pityriasis rubra pilaris, pityriasis rosea and lichenoid disorders like lichen planus, lichen nitidus and lichen striatus, attending the Dermatology, Venereology and Leprosy out patient department were included in this study.

PROCEDURE:

Children up to 18 years of age were included and detailed history of illness, regarding age, duration, onset, symptoms, recurrence, family history of the diseases, preexisting medical conditions and any history of drug intake were recorded.

Each patient was subjected to a complete systemic and cutaneous examination. Relevant information about preceding history of fever, cough, sore throat was noted. Clinical diagnosis of various papulosquamous disorders was done based on typical morphology and distribution of lesions at presentation

Complete hemogram and urine analysis was done. In psoriasis swab for culture from pharynx and perianal area, C-reactive Protein, ASO titers and in lichen planus Hepatitis B surface antigen, hepatitis C antibodies were done wherever necessary. Skin biopsy for histopathological examination from the skin lesion was performed with parents' consent

STATISTICAL ANALYSIS:

The observations pertaining to the parameters under study among the pediatric age group was expressed in percentage

Diagrammatic representation of all the parameters recorded (age and sex related), was done.

Mean was calculated.

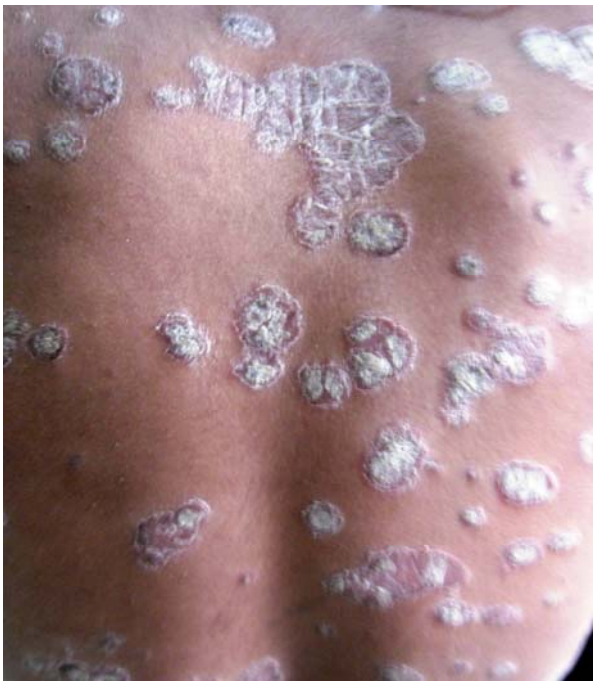


Fig. 1 Erythematous scaly plaques of psoriasis over trunk in a child.

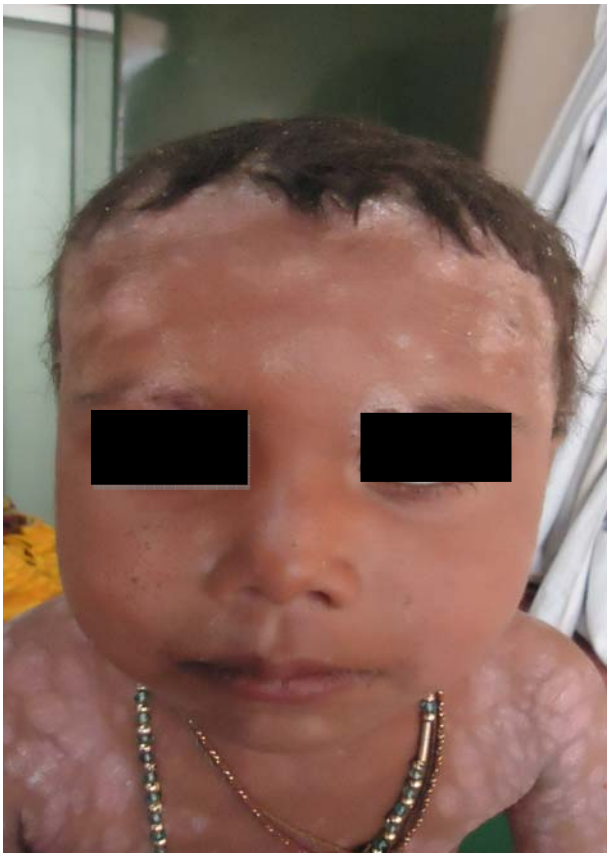


Fig.2 Scaly plaques of psoriasis over face and scalp.

Fig. 3 Erythematous glazy plaque of psoriasis over genitalia of a girl.

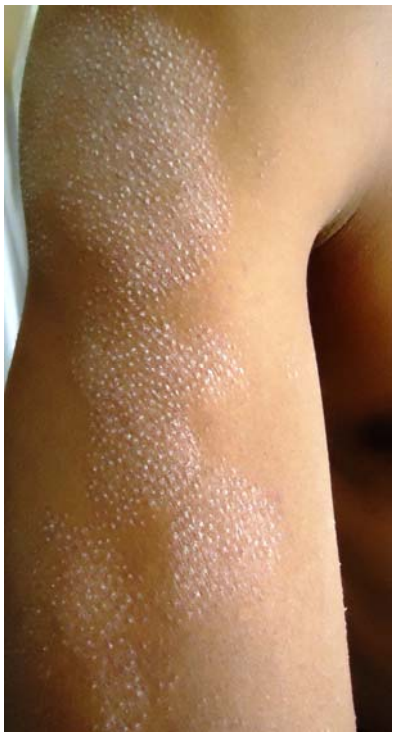


Fig. 4 Follicular psoriasis.

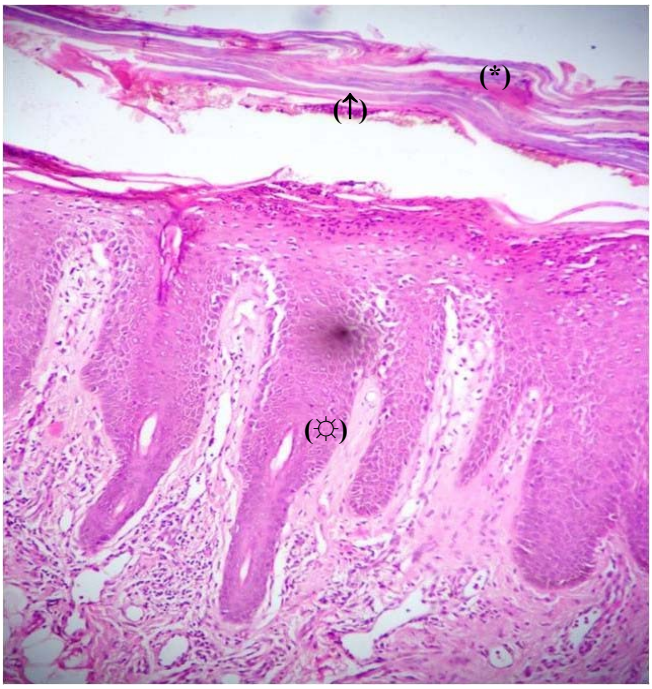


Fig. 5 Photomicrograph of psoriasis showing parakeratosis (↑), micro Munro abscesses (*) and elongation of rete (☼) ridges.(H&E,10X)

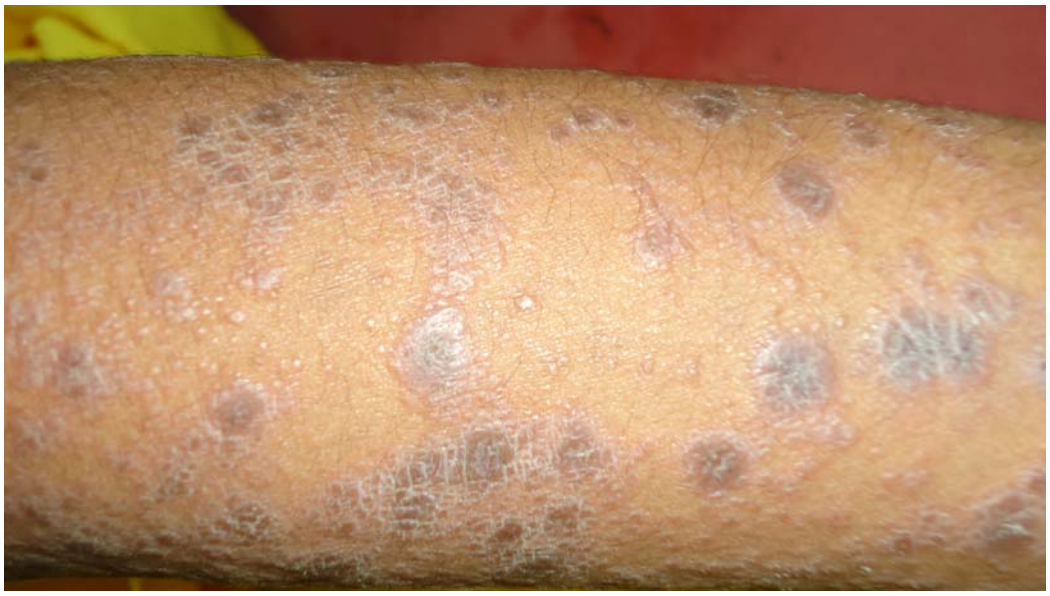


Fig. 6 Violaceous flat-topped papules of LP over forearm of a child.

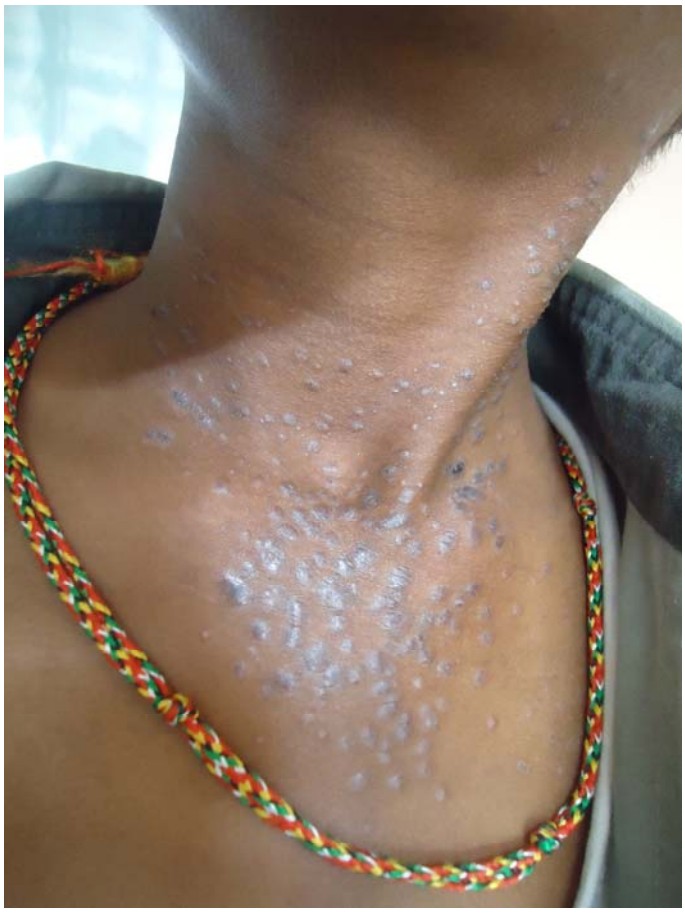


Fig. 7 Violaceous papules of LP over V area of the neck.

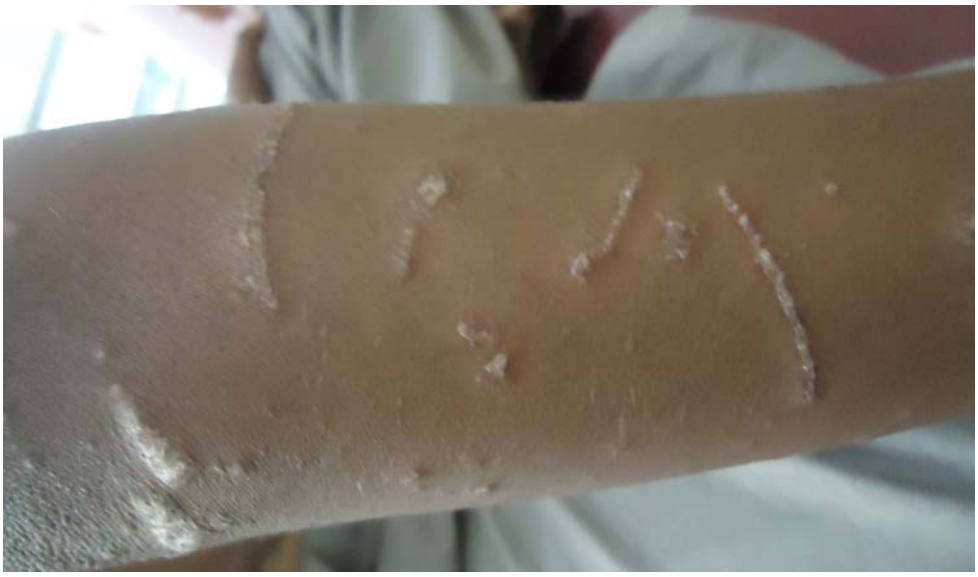


Fig. 8 Koebner's phenomenon in LP.

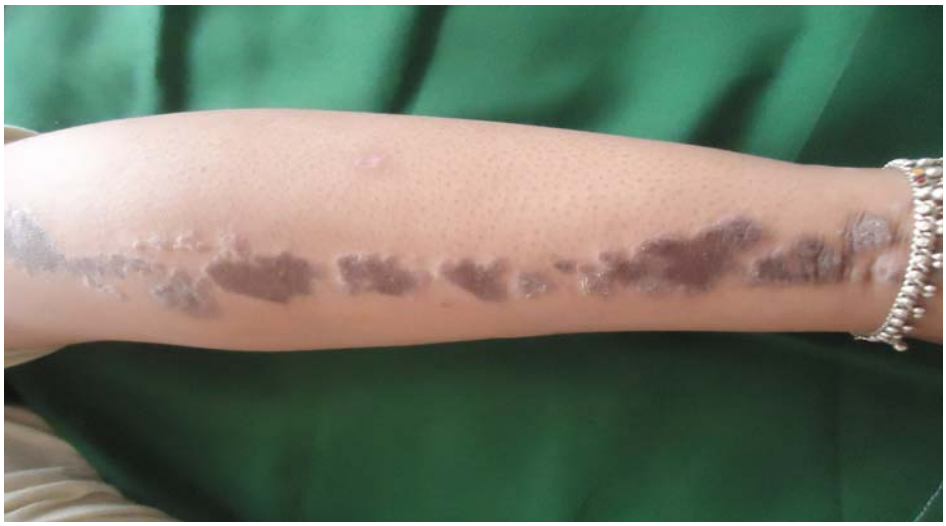


Fig. 9 Linear lichen planus in a child.

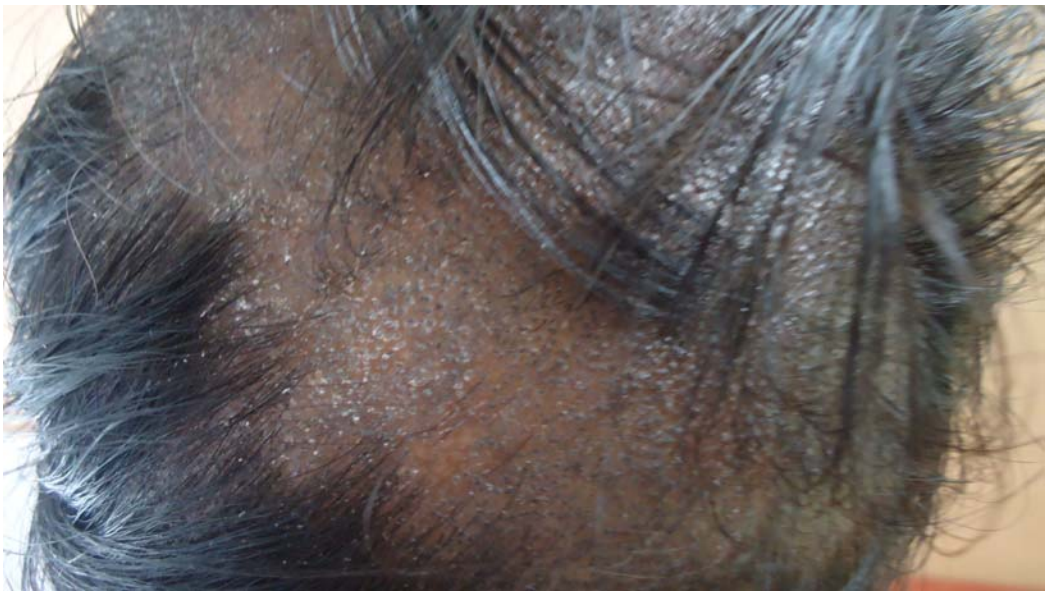


Fig. 10 Keratotic follicular papules of lichen plano pilaris with scarring alopecia in a child.

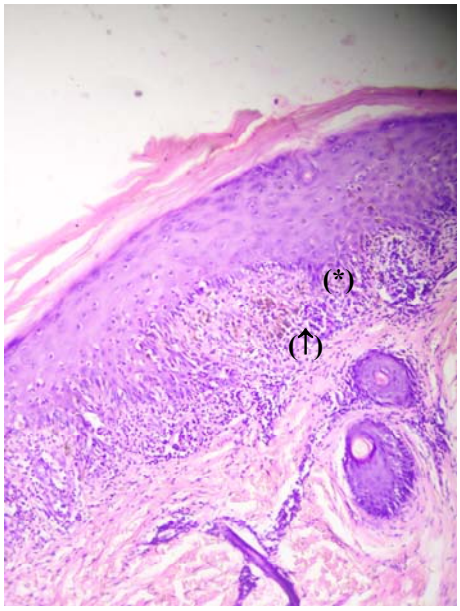


Fig. 11 Photomicrograph showing band like lymphocytic infiltration (↑) and basal cell vacuolization (*) in LP (H&E,10X)

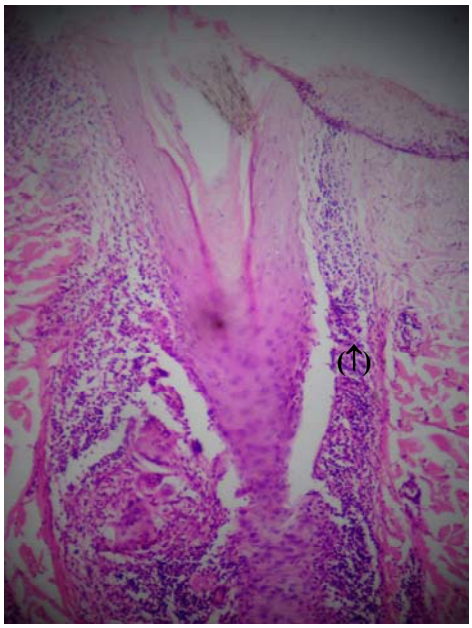


Fig. 12 Photomicrograph showing perifollicular lymphocytic infiltrate (↑) seen in the reticular dermis in LPP (H&E,10X)

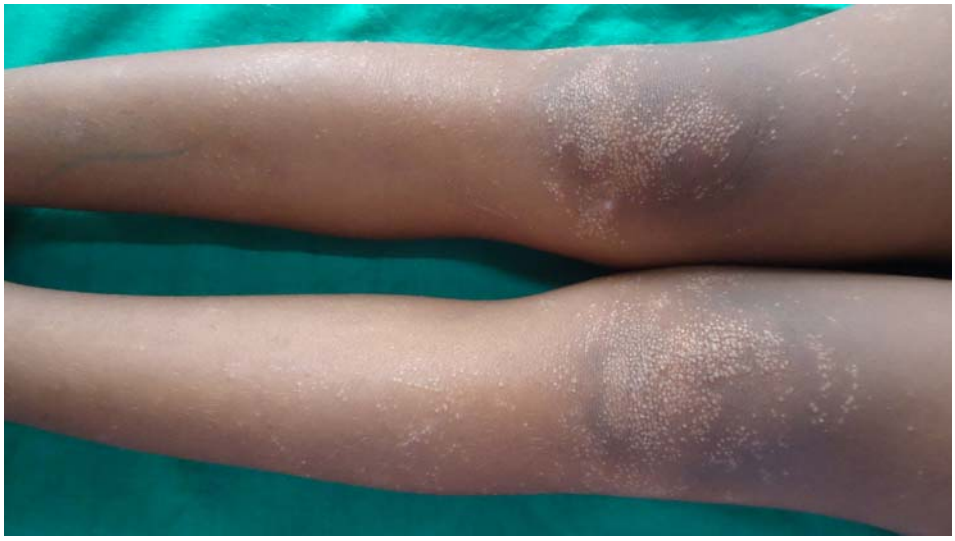


Fig. 13 Grouped, shiny papules of lichen nitidus on knee joints; Koebnerization is seen over legs.

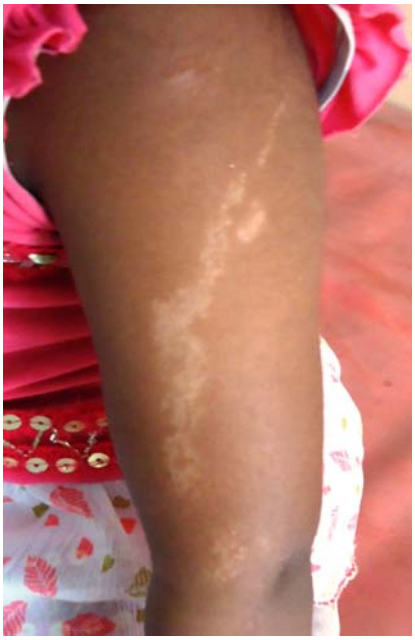


Fig. 14 Linear, hypopigmented lesion of lichen striatus over the arm of a child.

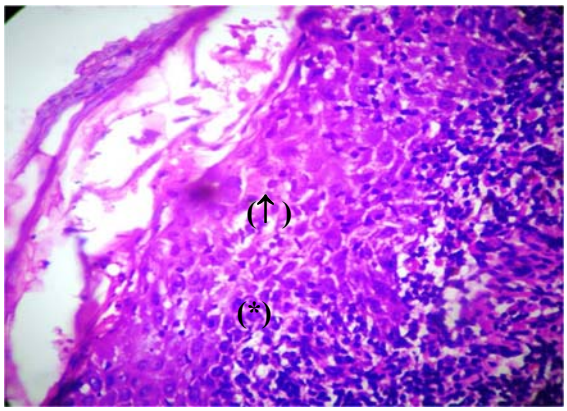


Fig. 15 Photomicrograph showing spongiosis (↑) and lymphohistiocytic (*) infiltration in lichen striatus (H&E,40X)

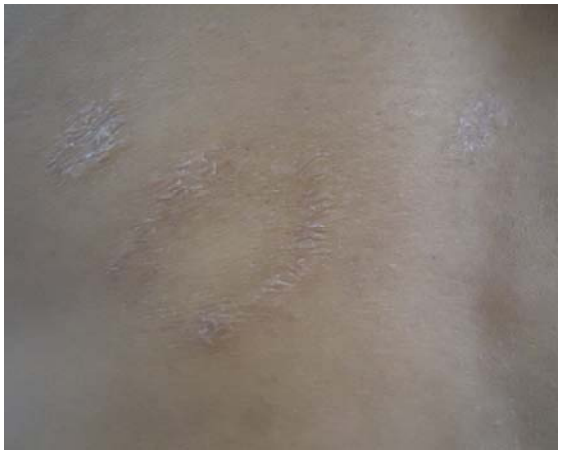


Fig. 16 Herald patch of Pityriasis rosea present over the back of a child.

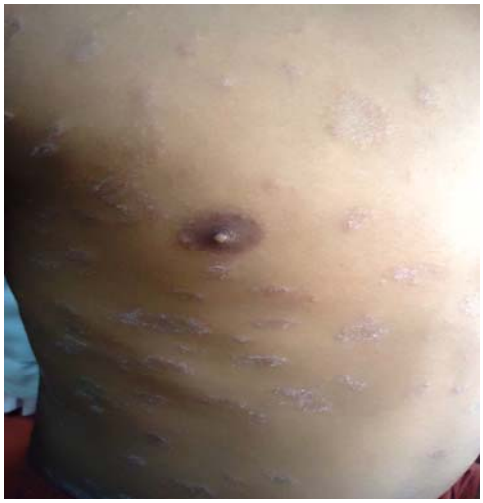


Fig. 17 Secondary eruptions of Pityriasis rosea over the trunk of a child.

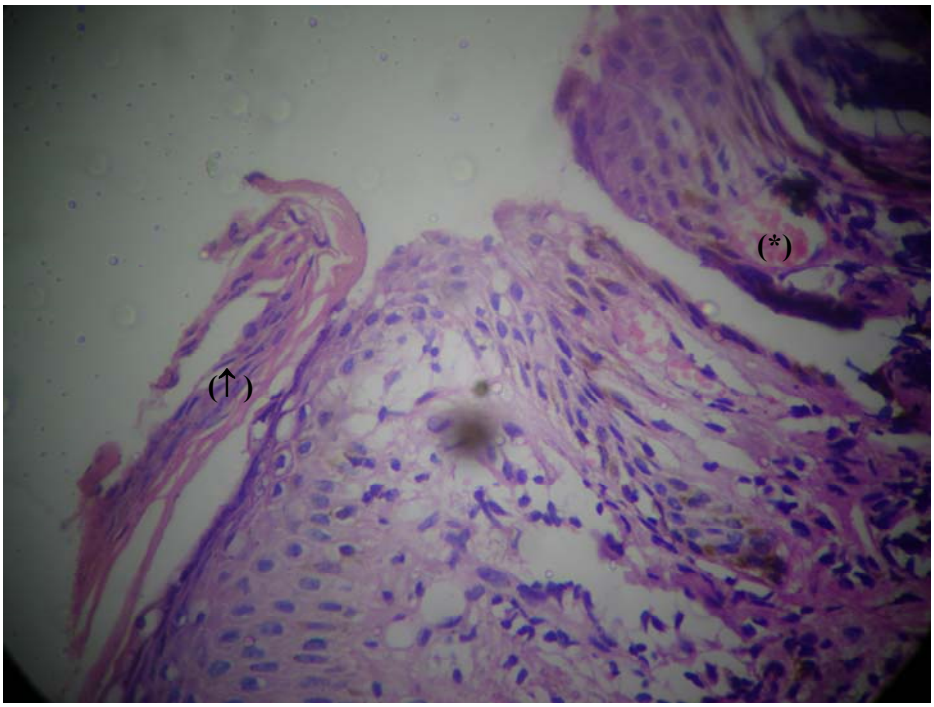


Fig. 18 Photomicrograph showing parakeratosis (↑) and extravasation of RBCs (*) in Pityriasis rosea (H&E, 10X)

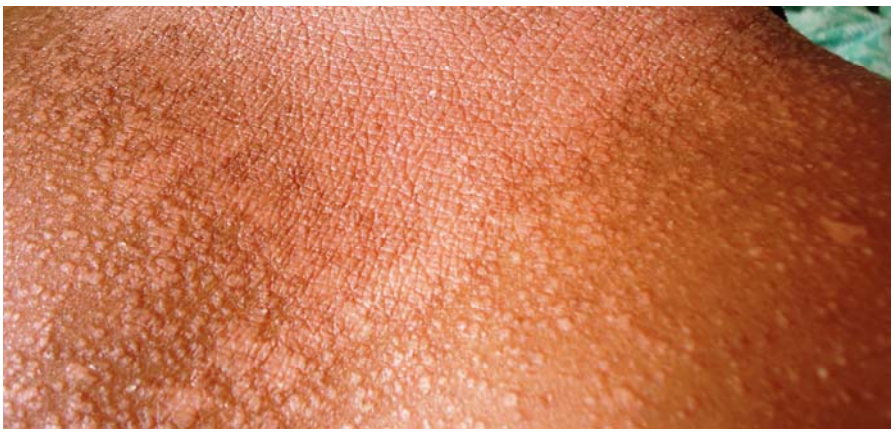


Fig. 19 Follicular papules of PRP over back.

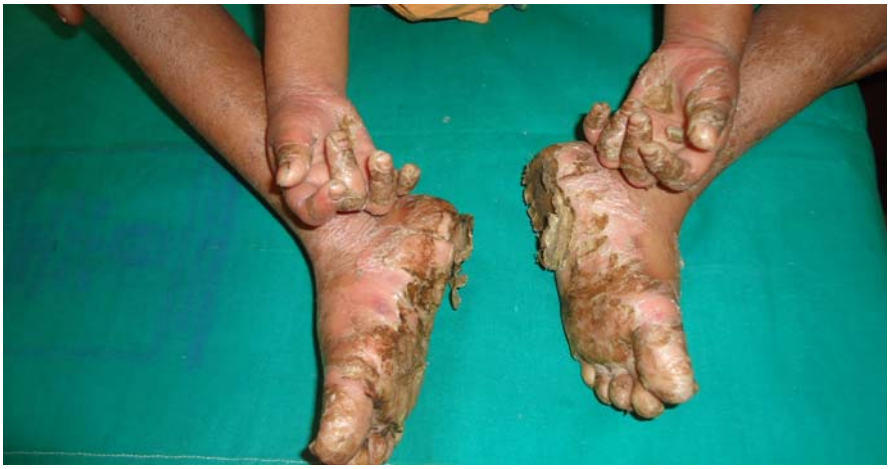


Fig. 20 Palmo plantar keratoderma in a child with PRP

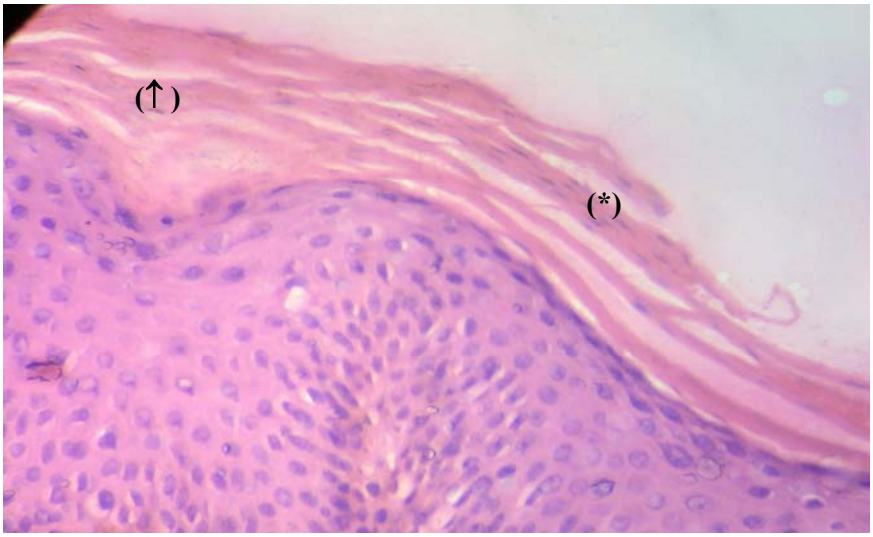


Fig. 21 Photomicrograph showing alternate ortho (↑) and parakeratosis (*) (H& E, 40 X)

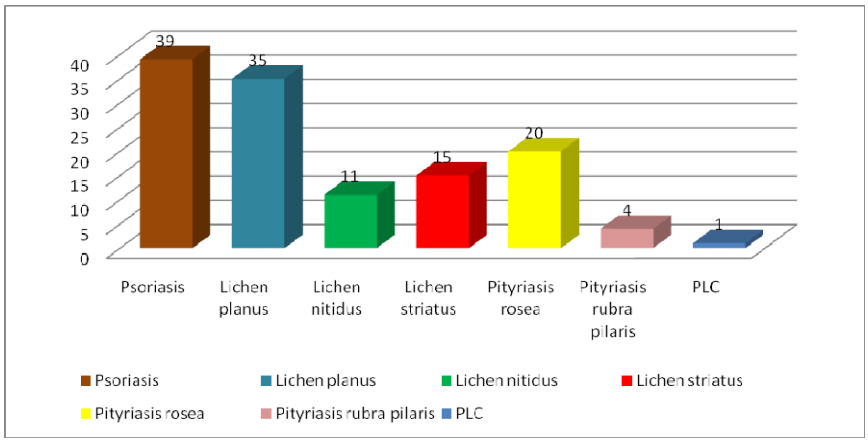
RESULTS

A total of 125 children with papulosquamous disorders were examined during the study period. Out of them 39 (31.2%) children were affected with psoriasis, 35 (28%) with lichen planus, 11(8.8%) with lichen nitidus, 15(12%) with lichen striatus, 20 (16%) with pityriasis rosea, 4 (3.2%) with pityriasis rubra pilaris and one (0.8%) child had pityriasis lichenoides chronica (PLC). Clinical types of papulosquamous disorders have been presented in table 4 and figure 22

Table 4: Clinical types of papulosquamous disorders

Clinical type	No.	%
Psoriasis	39	31.2
Lichen planus	35	28
Lichen nitidus	11	8.8
Lichen striatus	15	12
Pityriasis rosea	20	16
Pityriasis rubra pilaris	04	3.2
PLC	01	0.8

Figure 22: Clinical types of papulosquamous disorders



Age incidence and sex distribution:

The age of the children ranged from 10 months to 18 years (mean 9.4years). Incidence of papulosquamous disorders was highest among 11-15 years of age group followed by 6-10 years. There was male preponderance as compared to females (M=71, F=54). Age distribution of the children included in this study has been presented in table 5.

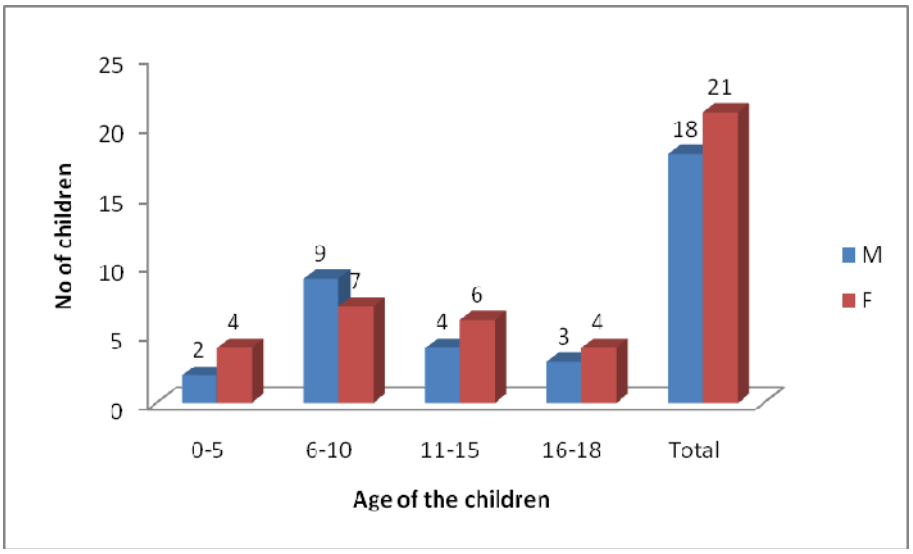
Table 5: Age distribution of the children included in the study.

Clinical types	Age-groups in years				
	0-5	**6-10**	**11-15**	**16-18**	**Total**
Ps	6	16	10	7	39
LP	3	10	16	6	35
LN	2	3	5	1	11
LS	5	4	5	1	15
PR	2	8	7	3	20
PRP	2	0	1	1	04
PLC	-	-	-	1	01
Total	**20**	**41**	**44**	**20**	**125**

Psoriasis:

Total 39(31.2%) children were suffering from psoriasis. The age of disease onset in these children ranged from 10 months to 18 years (mean 9.4years). Incidence of psoriasis was highest among children of 6-10 years of age. There was female preponderance (F=21,M=18), the ratio being 1.17:1. Gender-wise age distribution of children with psoriasis has been presented in figure 23.

Figure 23:Gender-wise age distribution of the children with psoriasis



Clinical types:

Twenty six children (66.7%) had chronic plaque psoriasis, 5 had guttate psoriasis and 5 had scalp psoriasis. One case each had pustular, follicular and inverse psoriasis. Clinical types of psoriasis has been presented in table 6.

Table 6: Clinical types psoriasis

S.No	Types	Male	Female	Total
1	Chronic plaque psoriasis	13	13	26
2	Scalp psoriasis	1	4	5
3	Guttate psoriasis	3	2	5
4	Inverse psoriasis	1	0	1
5	Pustular psoriasis	1	0	1
6	Follicullar psoriasis	1	0	1
	Total	**20**	**19**	**39**

Distribution of psoriatic lesions over various body sites have been presented in table 7.

Table 7: Distribution of psoriatic lesions

Sex	Scalp	Extremities	Trunk	Genitalia
M	11	11	12	3
F	16	14	14	3
Total	27	25	26	6
Percentage(%)	69.23	64.10	66.66	15.38

Out of 39 children, 31 had positive Auspitz sign. A positive family history was present in 5 patients (12.8%) and all of them were second degree relatives. Thirty children (76.92%) had classical silvery white scale. None of the children showed positive Koebner's response. The body surface area (BSA) involvement was calculated by rule of nine. Twenty four (61.54%) children had upto 25% of BSA involvement, 7 children had 50% of BSA involvement, 4 children had 70% of BSA involvement and, 6 children had > 90% of BSA involvement. Twenty two children had nail changes like pitting, Beau's lines and oil drop sign which has been shown in table 8.

Table 8: Nail changes in children with psoriasis

Types	M	F	Total	Percentage(%)
Pitting	12	7	19	48.71
Beau's Lines	2	0	2	5.1
Onychodystrophy	1	0	1	2.6

Investigations:

Hemogram and routine urine analysis were normal in all the cases. Antistreptolysin-O titer was positive in 2 children. Throat swab for culture sensitivity showed *Streptococcus species* in one child.

Histopathology:

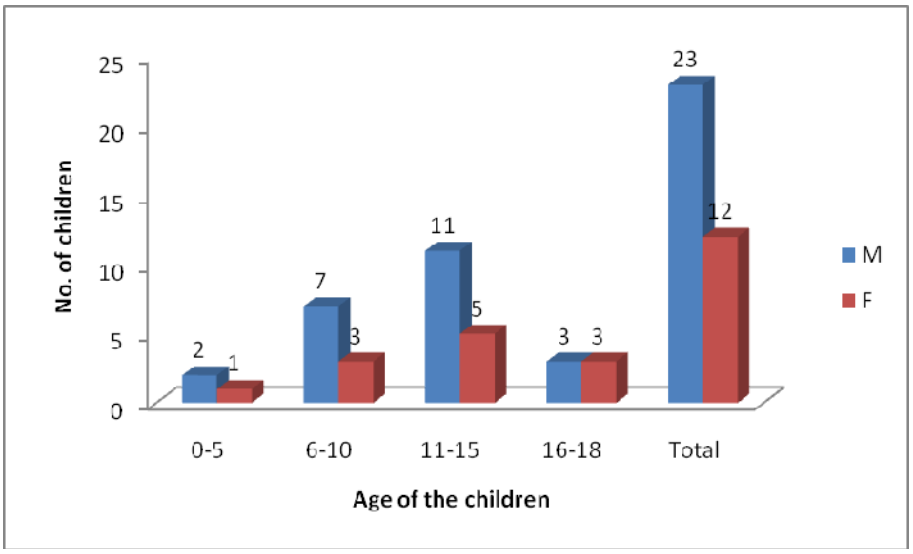
Histopathological analysis was done in 35 cases (89.74%). Skin biopsy was taken from the scaly lesions. The common epidermal features were micro Munro abscess which were present in 13 specimens. Elongation of rete ridges and epidermal hyperplasia was seen in 36 specimens. Confluent parakeratosis was seen in 27 specimens. Pustules of Kogoj was seen in 24 specimens. Suprapapillary thinning was seen in 27 specimens.

Lichen planus:

Age incidence and distribution:

Among 125 children, thirty five (28%) were affected with lichen planus, the age of the patients ranging from 2 to 18 years (mean 10yrs). Incidence of lichen planus was highest among 11-15years of age group. There was male preponderance in the occurrence of LP (M-23, F-12), ratio being 1.9:1. Gender wise age and distribution of the children with LP has been presented in figure 24 .

Figure 24: Gender wise age distribution of children with lichen planus



Clinical types:

Classical pattern of lichen planus was the most frequent type encountered at the time of presentation. Nineteen children (54.28%) presented with this type of lesions, followed by 4 cases, who had hypertrophic, actinic and follicular LP and 2 children had linear lichen planus and eruptive LP. Clinical variants of LP among the patients have been presented in table 9.

Table 9: Clinical variants of lichen planus

S.No	Types	Male	Female	Total
1	Classical LP	13	6	19
2	Actinic LP	3	1	4
3	Follicular LP	3	1	4
4	Hypertropic LP	2	2	4
5	Linear LP	1	1	2
6	Eruptive LP	1	1	2
	Total	**23**	**12**	**35**

Extremities were the commonest site of involvement (n=30) followed by trunk (n=14) face (n=9), genitalia (n= 8) and scalp (n=1), as shown in table 10 and figure 25.

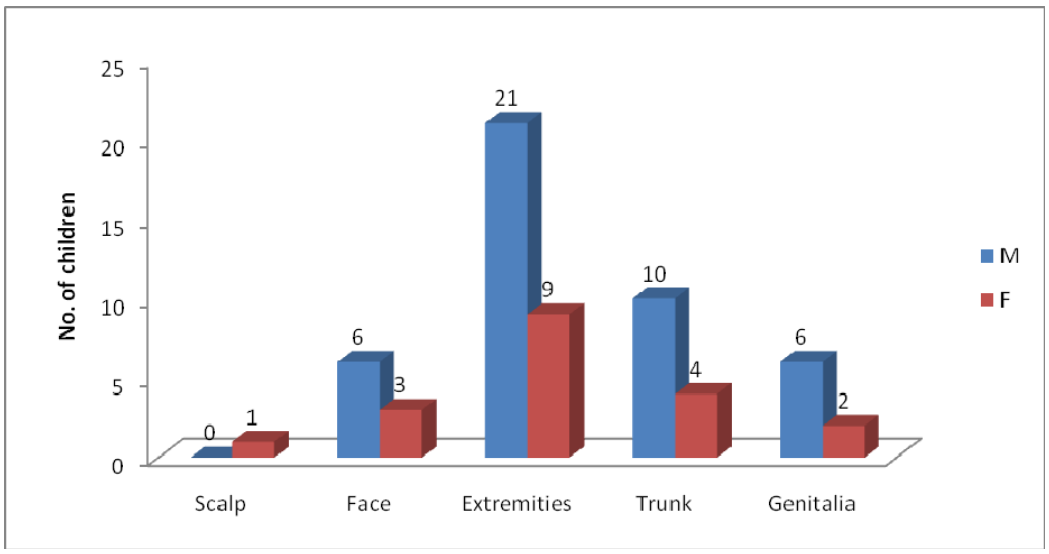
Mucous membrane lesions were seen in 3 patients. Nail changes were seen; pitting was the commonest finding seen in 4 children, Beau's line and onychodystrophy were seen in one child each. Wickham's striae was seen in 25 children (71.42%). Koebner's phenomenon was seen in 11(31.42%) children.

Twenty three (65.71%) children had < 25% of BSA involvement,6 (17.14%) had < 50% of BSA involvement and 10 (28.57%) children had > 50% but <100% of BSA involvement.

Table 10: Distribution of LP lesions over various body sites

Sex	Scalp	Face	Extremities	Trunk	Genitalia
M	0	6	21	10	6
F	1	3	9	4	2
Total	1	9	30	14	8

Figure 25:Gender wise distribution of lesions of LP over various body sites



Investigations:

Hemogram and routine urine analysis were normal in all the cases. Hepatitis B surface antigen and hepatitis C antibodies were done wherever necessary but these were negative in all the cases.

Histopatological study was done in 34 children, of which 15 specimens showed saw toothed appearance of the rete ridges. Wedge shaped hypergranulosis was seen in 19 specimens. Vacuolar degeneration of basal layer was present in 26 specimens. Pigment incontinence was seen in 22 specimens and band like lymphohistiocytic infiltration was seen in 25 specimens.

Lichen nitidus:

Total 11(8.8%) children were affected with lichen nitidus. The age of onset of lichen nitidus in these children ranged from 3 to18 years. There was male preponderance (M= 7,F=5), the ratio being 1.4:1. Extremities were the commonest sites affected in all the 11 children, followed by trunk (n=9) and genitalia (n=6). Koebner's phenomenon was seen in 3 children. Distribution of skin lesions of LN has been presented in table 11.

Table 11: Distribution of lesions of LN over various body sites

Sex	Extremities	Trunk	Genitalia
M	7	5	4
F	4	4	2
Total	11	9	6

Investigations:

Skin biopsy was done in 9 children and histopathology showed focal parakeratosis in 4 specimens. Diminished granular layer was seen in 4 specimens. Vacuolar degeneration of basal layer was seen in 9 specimens. "Claw clutching the ball" appearance was seen in 5 specimens and mixed lymphohistiocytic infiltration was seen in 8 specimens.

Lichen striatus:

Total 15(12%) children were affected with lichen striatus. The age of onset of the disease ranged from 2 to 17 years (mean 9.5 years). Females were commonly affected (F:M = 1.14:1) Lesions were distributed over extremities in 13 children, over trunk in 2 children and on face in one child. Distribution of skin lesions over various body sites has been presented in table12.

Table 12: Distribution of LS lesions over various body sites

Sex	Face	Rt hand	Lt hand	Rt leg	Lt. leg	Trunk
M	1	1	1	2	3	1
F	0	1	2	1	2	1
Total	1	2	3	3	5	2

Investigations:

Histopatological study was done in 9 children, among which spongiosis was seen in 8 specimens. Acanthosis was present in 6 specimens. Focal parakeratosis was present in 4 specimens. Vacuolar degeneration of basal layer was present in 7 specimens and mixed lymphohistiocytic infiltration was present in 9 specimens.

Pityriasis rosea:

Total 20 (16%) children were affected with PR. Age of onset of PR ranged from 5 years to 18 years. There was male preponderance (M=14, F=6), the ratio being 2.3:1. Lesions were most commonly distributed over the trunk (n=16), followed by extremities (n=6) and face (n=3). "Christmas tree pattern" was seen 6 children and herald patch was seen in 3 children. Collarette of scale was present in all except in 2

children. Nail changes like pitting was seen in one child. Thirteen children (65%) had 0-25% of BSA involvement, 5 (25%) children had 26-50% of BSA involvement, 2 (10%) children had more than 50% of body surface area involvement.

Investigation:

Histopathological analysis was done in 17(85%) children. Focal parakeratosis was seen in 11 specimens. Acanthosis was seen in 8 specimens. Extravasation of RBC was seen in 6 specimens.

Pityriasis rubra pilaris:

Four children (3.2%) had PRP (M-2, F-2). Family history of PRP was not present in any of these children. Typical morphology and distribution of lesions like follicular hyperkeratotic papule and plaque were seen on extremities in all the children followed by on trunk, face and scalp. Three children had palmoplantar keratoderma (PPK). Nail changes like subungual hyperkeratosis and onychodystrophy were seen in 2 children.

Investigations:

Histopathological study was done in all 4 children. Alternating orthokeratosis / parakeratosis was seen in 3 specimens. Acanthosis with broad and short rete ridges was seen in 3 specimens. Spongiosis was seen in 3 specimens. Focal or confluent hypergranulosis was seen in 2 specimens and perivascular lymphocytic infiltration was seen in 2 specimens.

DISCUSSION

Papulosquamous disorders are not commonly encountered in children. However, recently there are reports of more frequent occurrence of these disorders in children, which may be attributed to genetic factors, viral or bacterial infections or it may be of autoimmune etiology.

Various papulosquamous disorders that may occur in children are psoriasis, lichen planus, lichen nitidus, lichen sriatus, pityriasis rosea, PRP and PLC. These disorders are of universal occurrence, without any geographical predilection. Age and sex variation may be observed in various ethnic groups. Since these disorders, especially psoriasis and lichen planus run a chronic, relapsing course, occurrence of these among pediatric patients may impair their quality of life adversely.

In the present study papulosquamous disorders were more prevalent among school going children. Eighty five children (68%) were in this age group. Incidence was less among infants and adolescents.

In an epidemiological study on incidence of childhood dermatoses, conducted in JIPMER, Pondicherry, India, it was observed that 34 (1.6%) children were affected with papulosquamous disorders.[14] In another hospital - based study on childhood dermatoses from Ahmedabad, out of 700 children aged < 14 years who were examined, 22 (3.15%) were affected with papulosquamous disorders.[13] Psoriasis, lichen planus and p.rosea were the commonly encountered papulosquamous disorders in these two studies.

In the present hospital-based study, children suffering from only papulosquamous disorders were included. Hence, as compared to the above studies, the incidence of

psoriasis, lichen planus and p.rosea appears to be higher. A comparison of these data has been presented in table 13. Moreover, in the above quoted two studies, the disorders included were only psoriasis, lichen planus and p.rosea, whereas in the present study other papulosquamous disorders like lichen nitidus, lichen striatus, pityriasis rubra pilaris and PLC have also been included.

Table 13: Incidence of papulosquamous disorders in different epidemiological studies

Studies	Incidence
Patel et al [13]	LP -2.16% Psoriasis - 0.99%
Karthikeyan et al[14]	Psoriasis -1.4% PR - 0.2% LP-0.2% PRP -0.14%
Present Study	Psoriasis - 31.2% LP - 28% PR -16% PRP-3.2%

Psoriasis

Psoriasis is a chronic inflammatory skin disease with a strong genetic basis, characterized by erythematous scaly papules and plaques due to alteration in epidermal proliferation and differentiation.

Psoriasis constituted the commonest papulosquamous disorder in the present study (31.2%). Gender distribution of the patients was comparable to other studies (Table 14). The peak age of onset was 6-10 years in the present study as well as in others. Family history was present in 12.8%, which is higher as compared to another Indian study, whereas, much lower as compared to two western studies (Table 15).

Though it is conventionally thought that guttate type of psoriasis is the common clinical pattern among children, chronic plaque psoriasis was the commonest variant observed in this study (66.6%). Even, this is the commonest finding encountered by other authors (Kumar et al, 60.6%,[15] Morris et al, 34%[30]).

Rare clinical variant, like acute generalized pustular psoriasis of von Zumbusch was seen in a girl. Scalp psoriasis without involvement of other body parts was seen in 5 children. Nail changes were observed in 22 children (56.41%), pitting being the commonest finding. Clinical types like psoriatic erythroderma or psoriatic arthritis were not seen in this study.

Table 14: Gender ratio of children affected with psoriasis in different studies

Studies	M:F ratio
Morris et al [30]	1:1.4
Stefanaki et al[17]	1.4:1
Kumar et al [15]	1.09:1
Present study	1:1.16

Table 15: Occurrence of positive family history of psoriasis in various studies.

Studies	Positive family history
Morris et al [30]	43%
Stefanaki et al[17]	16%
Kumar et al [15]	4.5%
Present study	12.8%

Histopahological examination was done in 35 patients and following features were commonly seen ; micro Munro abscess, elongation of rete ridges and epidermal hyperplasia, confluent parakeratosis, Pustules of Kogoj and suprapapillary thinning .

Lichen planus

Lichen planus is a papulosquamous disorder of unknown etiology, that affects skin, hair, nail and mucous membrane, characterized by violaceous, flat topped, polygonal, pruritic, papule.

Lichen planus constituted the second commonest papulosquamous disorder encountered in this study (28.2%). The results of the present study showed boys were more commonly affected than girls, which is similar to the findings of the study conducted by Sharma et al.[62] Some other authors like Handa et al[35] and Luis-Montoya et al [34] have shown nearly equal gender ratio in the occurrence of childhood lichen planus in their studies. Table 16 presents gender ratio of children suffering from LP in various studies. Classical LP was the commonest clinical variant observed in all the above reported studies.[34,35,60] In the present study also classical LP was the commonest variant encountered (54.28%). The second in order was lichen planus hypertrophicus, followed by actinic LP, follicular LP, linear LP and eruptive LP.

Actinic LP is considered rare in children; however in the present study it accounted for 11.42%, which is comparable to another Indian study (Handa et al,11.5%).[35] Sharma et al [60] have reported occurrence of actinic LP in only 2% of the studied children and Luis-Montoya et al[34] have encountered this clinical type in only one child. The higher occurrence of actinic LP among the children in this study may be because of geographical location of this area and relative lack of awareness among parents regarding photo-protection in children.

Mucous membrane lesions were seen in only 3 children (8.5%). This is far lower as compared to most of the studies except the one by Kanwar et al (6%). [61] Incidence of mucous membrane lesions in various studies has been presented in table 17 .

Wickham's striae was present in 25(71.42%) children. Koebner's phenomenon was present in 11(31.42%) children, which is higher as compared to the findings by Handa et al (26.5%).[35]

Genital lesions were seen in 8 children (22.85%) in this study. None of reported studies except one have quoted occurrence of genital lesions in children with LP. Nnoruka et al [62] have reported genital involvement in 7.7% of the studied children.

Non-specific nail changes like Beau's lines and onychodystrophy were found in 5 (14.28%) children with LP. There was no child with only nail LP. Nail involvement in children with LP in various Indian studies ranged from 0-19%.

Table 16: Gender ratio in children with LP observed in various clinico-epidemiological studies

Studies	M:F ratio
Handa et al[35]	1.1:1
Luis Montoya et al[34]	1:1.2
Sharma et al [60]	2:1
Present Study	1.9:1

Table 17: Incidence of mucous membrane lesion in various studies

Studies	M:F ratio
Sharma et al [60]	30%
Handa et al[35]	13%
Kanwar et al [61]	6%
Luis Montoya et al[34]	4.3%
Present Study	8.5%

Histopathological examination was done in 33 patients and common findings were; saw toothed appearance of the rete ridges, wedge shaped hypergranulosis , vacuolar degeneration of basal layer, pigment incontinence and band like lymphohistiocytic infiltration.

Lichen nitidus:

Lichen nitidus is a papulosquamous disorder, characterized by multiple, discrete, 1 to 2 mm, flesh-colored, shiny papules. It commonly involves the penis, arms,

forearms, chest, and abdomen. The lesions are usually asymptomatic, or sometimes mildly pruritic.

None of the studies on papulosquamous disorders in children has included lichen nitidus as an entity included. In this study, lichen nitidus was seen in 11 children with male preponderance (M=7, F=4). Extremities were the most commonly affected sites, followed by trunk and genitalia. Koebnerization was present in 3(27.27%) children. Mucous membrane involvement is rare in LN but in this study one child had shown mucosal pigmentation. However, whether this finding was related to the disease itself or of some other etiology could not be assessed because of lack of mucosal biopsy. Mild pruritus was seen in all children. LN occurs in association with LP in 25% to 30% children.[36] In the present study no such association was recorded.

Histopathological examination was done in 9 patients and common findings were; epidermal changes like, focal parakeratosis, diminished granular layer and vacuolar degeneration of basal layer. Dermal changes like "claw clutching the ball" appearance and mixed lymphohistiocytic infiltration were present.

Lichen striatus:

Lichen striatus is a papulosquamous disorder which manifests as unilateral linear eruptions along the Blaschko's lines on the extremities, trunk and neck. Individual lesions are characterized by minute, slightly raised, erythematous papules, which may have a scaly surface and are closely aggregated. The lesions heal with or without treatment leaving behind linear hypopigmentation

Lichen striatus was not included in any of the studies on childhood papulosquamous disorders. Lichen striatus was seen in 15 children in this study. Though a male preponderance in the occurrence of childhood LS has been reported by Patrize et al [46] and Abagge et al,[63] almost equal sex distribution (M=7, F=8) was recorded in this

study. Commonest site of involvement was extremities followed by trunk and face. Bilateral occurrence of LS is rare but one child presented with lesions over both the thighs. There was no family history of atopy in any of the children and the lesions were symptomatic in all of them.

Histopathological examination was done in 9 patients and common findings were; spongiosis, acanthosis , focal parakeratosis ,vacuolar degeneration of basal layer and mixed lymphohistiocytic infiltration.

Pityriasis rosea

Pityriasis rosea is a self-limiting papulosquamous disorder of unknown etiology, manifested by appearance of herald patch followed by generalized crops of lesions with collarette of scales.

PR was the third in the order of occurrence of childhood papulosquamous disorders included in this study (16%).

Twenty children were affected with PR showing male preponderance (M=14, F=6). In an yet unpublished study by Ganguly et al,[53] male preponderance was recorded among children with PR. Herald patch was seen in 15% of the cases in this study. It is lower as compared to the usual occurrence of herald patch. Trunk was the most common site of involvement (80%) in the present study. “Christmas tree pattern” was seen in 6 (30%) children. No precipitating factor could be detected in any of the children included in this study (Table 18).

Table 18: Comparison of various parameters of PR with other study

Studies	No. and % of patients	M:F	Herald Patch	Area of involvement
Ganguly et al [53]	41(56.16%) children	1.7:1	27(65.85%)	Extremities (40%) Trunk (38.2%) Face (8.82%)
Present study	20(16%) children	2.3:1	3 (15%)	Trunk (80%) Extremities (30%) Face (15%)

Histopathological examination was done in 17 patients and common findings were; focal parakeratosis, acanthosis and extravasation of RBCs

Pityriasis rubra pilaris

Pityriasis rubra pilaris is a heterogeneous group of disorder characterized by circumscribed follicular papules with branny scale, orange red erythema and palmoplantar keratoderma. It is a chronic and relapsing disorder with relative resistance to treatment.

Four children included in this study were affected with PRP. As reported by Gelmetti et al[64] and Allison et al,[65] in children with PRP, most frequent sites of involvement were the palms and soles. Gelmetti et al[64] have proposed that palmoplantar involvement may be considered as the second most important diagnostic feature for the classification of juvenile PRP. In this study 3 children among the 4 had PPK. Alisson et al[65] have reported that 17% of the children with PRP in their study had

erythroderma, and 13% had ectropion. In the present study none of the affected children had erythroderma and one child had only mild ectropion.

Histopathological examination was done in 3 patients and common finding were; alternating orthokeratosis / parakeratosis, acanthosis with broad and short rete ridges, spongiosis, focal or confluent hypergranulosis and perivascular lymphocytic infiltration.

Pityriasis lichenoides chronica

Pityriasis lichenoides chronica (PLC) is a papulosquamous disorder occurring largely in children and young adults. The characteristic lesions are small, firm lichenoid papules with adherent 'mica-like' scale. Post-infl ammatory hypopigmentation may occur and persist for long time.

One female child included in this study had PLC. She presented with bilateral symmetrical hypopigmented macules and patches with mild scaling over the lesions.

Histopathological examination showed hyperkeratosis, parakeratosis, acanthosis and hydropic degeneration of the basal cell. Exocytosis of lymphocytes and extravasation of RBCs were seen.

CONCLUSION

There is considerable difference in epidemiology of papulosquamous disorders among adults and children. This is regarding etiological factors, clinical patterns and treatment options and impact of disease on patient's life. Epidemiological data on adult papulosquamous disorders are abundantly available but it is not so in children. Hence, it was prudent to conduct a study on the clinico epidemiological aspects of these chronic disorders in children from this region of India.

As the present study shows, papulosquamous disorders are not uncommon in the pediatric age group. Most of these disorders are disfiguring when occurs over exposed body parts. The course of these disorders, especially psoriasis and lichen planus are chronic and relapsing. Because of the tender age, all the therapeutic modalities available for these disorders cannot be used in children. Hence occurrence of any of these disorders in a pediatric member of a family may impose both psychological and economic burden upon the family members. Moreover, for the affected children the disease may be stigmatizing causing hindrance to social interaction with peers resulting in introvert personality, impairing their quality of life.

Hence it is important for the treating clinicians to be well conversant with the epidemiology and clinical features of papulosquamous disorders in children, so that effective management strategies can be undertaken.

SUMMARY

A hospital based, cross-sectional, analytical study on clinic-epidemiological pattern of papulosquamous disorders in children was conducted during the period of October 2010 to September 2012. Children up to 18 years of age were included and detailed history of illness, regarding age, duration, onset, symptoms, recurrence, family history of the diseases, preexisting medical conditions and any history of drug intake were recorded.

Each patient was subjected to a complete systemic and cutaneous examination. Relevant information about preceding history of fever, cough, sore throat was noted.

Following are the salient findings of this study:

- School going children were the commonest age group suffering from papulosquamous disorders.
- Overall male: female ratio was 1.3:1.
- Psoriasis was the commonest papulosquamous disorder, followed by lichen planus and PR.
- Incidence of psoriasis was highest among children of 6-10 years of age.
- Family history was positive in 12.8% of children suffering from psoriasis.
- Antistreptolysin-O titer was positive in 2 children with chronic plaque psoriasis.
- Incidence of lichen planus was highest among 11-15years of age group.
- Wickham's stria was seen in 25 children and Koebner's phenomenon was seen in 11 children with LP.

- Lichen nitidus was seen in 11 children of whom Koebnerization was present in 3.
- Lichen striatus was seen in 15 children, of whom one child had bilateral lesions.
- PR was seen in 20 children, of whom only 6 presented with classical 'christmas tree pattern.'
- Four children were affected with PRP of whom 3 had PPK.

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ANNEXURES

PROFORMA:

SCHEME OF CASE TAKING

B.L.D.E.U'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR.

Department of Dermatology, Venereology and Leprosy.

SL.NO: Date: Sex: Male/Female

Name: Age:

Father's name: Age of onset:

Address :

1. Complaints with duration :
2. Presenting features :
   a) Symptoms with duration : Fever / sore throat / joint pain / jaundice
   b) Itching : Mild / moderate / severe
   c) Number of episodes :
   d) Any history of : Blood transfusion / drug intake

e) Family history : First degree family members /

Second degree family members.

f) History of trauma : Yes / No

g) Immunization status :

3. General Physical Examination : Weight- Height-

Pallor- Cyanosis- Clubbing-

Icterus - Edema - Lymphadenopathy-

4. Cutaneous examination :

a) Type of lesions : Macule / papule / plaque / pustule

b) Distribution of lesion : Generalized / localized

Symmetrical / asymmetrical

Photo exposed area / flexural

Palmoplantar / nail / scalp / trunk

Christamas tree pattern

Innverse PR pattern

c) Herald patch : Site / size / shape

d) Type of scale : Silvery white / adherent / collarette

e) Auspitz sign : Present / absent

f) Koebner's phenomenon : Present / absent

g) Wickham's striae : Present / absent

h) Body surface area involved:

i) Mucous membrane :

j) Nail changes : Pitting / SUHK / Oil drop sign / Pterygium/

Splinter hemorrhage

k) Hair changes :

5. Systemic Examination :

Respiratory system :

Cardiovascular system :

Central nervous system :

GI system :

6. Diagnosis :

7. Investigations :

Hb% :

Total leucocyte count :

Differential count :

ESR :

Peripheral blood smear :

Urine analysis :

Albumin- :

Sugar- :

Microscopy- :

Skin biopsy for histopathological examination

In psoriasis

- C-reactive Protein
- ASO titer
- Swab for culture from pharynx and perianal area.

In lichen planus

- Hepatitis B surface antigen
- Hepatitis C antibodies

SAMPLE INFORMED CONSENT FORM BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR-586 103

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT:- A CROSS SECTIONAL CLINICAL STUDY OF PAPULOSQUAMOUS DISORDERS IN CHILDREN

PG GUIDE :- DR. ARUN C. INAMADAR.

PG STUDENT :- DR. SWAROOPA S. LAGALI.

PURPOSE OF RESEARCH :-

I have been informed that this project will study the epidemiologic characters and clinical pattern of papulosquamous disorders in children.

BENEFITS:-

I understand that my child's participation in this study will help the investigator to understand the disease better and will help in the management of the disease.

PROCEDURE :-

I understand that relevant history will be taken and my child will undergo detailed clinical examination after which necessary investigation will be done whenever required.

RISK AND DISCOMFORTS:-

I understand there is no risk involved and my child will experience minimal pain during the procedures performed.

CONFIDENTIALITY:-

I understand that medical information produced by this study will become a part of my child's hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs, videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:-

I understand that I may ask more questions about the study at any time concerned. The researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:-

I understand that my child's participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that the researcher may terminate my child's participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my child's continued care by my own physician if this is appropriate.

INJURY STATEMENT:-

I understand that in the unlikely event of injury to my child resulting directly from my child's participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I understand that by my agreement for my child's participation in this study, I am not waiving any of my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that my child will undergo, and the possible risks and discomforts as well as benefits that I may experience. I have read

and I understand this consent form. Therefore, I agree to give my consent for my child's participation as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

KEY TO MASTER CHART

Sl. No –	Serial number
M –	Male
F –	Female
Ps –	Psoriasis
LP –	Lichen planus
LPH -	Lichen planus hypertrophicus
LPP –	Lichen planopilaris
LN –	Lichen nitidus
LS –	Lichen striatus
PR -	Pityriasis rosea
PRP –	Pityriasis rubra pilaris
PLC –	Pityriasis lichenoides chronic
BSA –	Body surface area
C/ S –	Culture and sensitivity
CRP –	C-reactive protein
ASO –	Antistreptolysin –O
SUHK –	Subungual hyperkeratosis

MASTER CHARTS (CLINICAL PARAMETERS)

Psoriasis

Sl. No.	Diagnosis	M/F	Age in yrs	Age of onset (yr)	Family history	Distribution Of lesions				Type of scale	Auspitz sign	Koebner's Phen.	BSA	Nail changes					Investigations		
						Scalp	Extremities	Trunk	Genitalia					Pitting	Beau's Lines	SUHK	Oil drop Sign	Splinter Haemorrhage	CRP	ASO titer	Swab for C/S from pharynx & Perianal Area
1	Chr. Plaq Ps	F	12	11	+	+	+	+	--	+	+	_	80%	+	+	--	--	--	--	--	--
2	Chr. Plaq Ps	F	18	16	--	+	+	+	--	+	+	--	90%	+	--	--	--	--	--	+	--
3	Chr. Plaq Ps	F	15	14	--	+	+	+	--	--	+	--	30%	--	--	--	--	--	--	--	--
4	Chr. Plaq Ps	M	18	12	--	+	--	+	--	+	+	--	50%	+	--	--	+	--	--	--	--
5	Chr. Plaq Ps	M	16	16	+	+	+	+	--	+	+	--	12%	+	--	--	--	--	--	--	--
6	Chr. Plaq Ps	M	5	5	--	+	--	+	--	+	+	--	50%	+	--	--	--	--	--	--	--
7	Chr. Plaq Ps	F	18	16	--	+	+	--	--	--	--	--	10%	+	--	--	--	--	--	--	--
8	Chr. Plaq Ps	F	7	7	--	+	+	+	--	+	+	--	25%	--	--	--	--	--			+
9	Chr. Plaq Ps	M	12	11	--	+	+	--	--	+	--	--	15%	+	--	--	--	--	--	--	--
10	Chr. Plaq Ps	M	7	6	--	+	+	+	+	+	+	--	7%	+	--	--	--	--	--	--	--
11	Chr. Plaq Ps	F	4	4	--	+	+	--	+	+	+	--	14%	--	--	--	--	--	--	--	--
12	Chr. Plaq Ps	F	16	16	--	--	+	+	--	--	+	--	10%	--	--	--	--	--	--	--	--
13	Chr. Plaq Ps	M	10	10	--	--	+	--	--	--	+	--	8%	--	--	--	--	--	--	--	--
14	Chr. Plaq Ps	F	8	6	--	+	--	+	--	--	+	--	60%	+	--	--	--	--	--	--	--
15	Chr. Plaq Ps	F	10	10	--	+	+	--	+	+	+	--	70%	+	--	--	--	--	--	--	--
16	Chr. Plaq Ps	F	4	4	--	+	+	+	+	+	+	--	80%	+	--	--	--	--	--	--	--
17	Chr. Plaq Ps	M	14	12	--	--	+	--	--	+	+	-	18%	+	--	--	--	--	--	--	--
18	Chr. Plaq Ps	M	8	4	--	--	+	--	--	+	+	-	10%	--	--	--	--	--	--	--	--
19	Chr. Plaq Ps	M	13	13	--	--	+	+	--	+	+	-	8%	--	--	--	--	--	--	--	--
20	Chr. Plaq Ps	M	5	5	--	--	--	+	--	+	+	-	80%	--	--	--	--	--	--	--	--
21	Chr. Plaq Ps	F	10	10	--	+	--	+	--	+	+	-	30%	--	--	--	--	--	--	--	--
22	Chr. Plaq Ps	F	15	15	--	+	+	+	--	+	+	-	50%	--	--	--	--	--	--	--	--
23	Chr. Plaq Ps	F	15	12	--	--	+	+	--	+	+	-	30%	--	--	--	--	--	--	+	--
24	Chr. Plaq Ps	M	8	8	+	+	+	+	+	+	+	-	90%	+	--	--	--	--	--	--	--
25	Chr. Plaq Ps	M	9	9	--	--	+	--	--	+	-	-	16%	--	--	--	--	--	--	--	--
26	Chr. Plaq Ps	M	8	6	+	+	+	+	+	+	-	-	80%	+	--	--	--	--	--	--	--
27	Guttate ps	M	12	12	+	+	+	+	--	+	+	--	80%	+	--	--	--	--	--	--	--

28	Guttate Ps	M	10	7	--	+	--	+	--	--	+	--	20%	--	--	--	--	--	--	--	--
29	Guttate Ps	M	6	6	--	+	--	+	--	+	+	--	12%	+	--	--	--	--	--	--	--
30	Guttate Ps	F	18	17	--	+	+	+	--	+	+	--	12%	--	--	--	--	--	--	--	--
31	Guttate Ps	F	10	9	--	--	+	+	--	+	+	-	70%	--	+	--	--	--	--	--	--
32	Scalp Ps	M	17	17	--	+	--	--	--	+	+	--	2%	+	--	--	--	--	--	--	--
33	Scalp Ps	F	4	4	--	+	--	--	--	+	+	--	2%	--	--	--	--	--	--	--	--
34	Scalp Ps	F	6	2	--	+	--	--	--	+	+	--	5%	--	--	--	--	--	--	--	--
35	Scalp Ps	F	15	15	--	+	--	--	--	--	-	--	3%	--	--	--	--	--	--	--	--
36	Scalp Ps	F	9	9	--	+	--	--	--	+	-	--	4%	+	--	--	--	--	--	--	--
37	Pustular ps	F	12	12	--	--	+	+	--	--	-	--	60%	--	--	--	--	--	--	--	--
38	Follicular Ps	M	6	5	--	--	--	+	--	+	+	--	10%	+	--	--	--	--	--	--	--
39	Inverse Ps	F	10	6 mon.	--	--	--	+	--	--	-	--	5%	--	--	--	--	--	--	--	--

Lichen planus:(LP)

Sl. No.	Diagnosis	M/F	Age in years	Age of on set (Yr.)	No.of episodes	Distribution of Lesion					Wickham's Striae	Koebner's Phenomenon	Mucous membrane	Nail change				BSA
						Scalp	Face	Extremities	Trunk	Genitalia				Pitting	Beau's Line	Onychodystrophy	Pterygium	
1	LP	M	12	7	2nd	--	--	+	+	+	+	--	--	+	--	--	--	30%
2	LP	M	7	7	1st	--	--	+	+	--	--	--	--	--	+	--	--	40%
3	LP	M	12	12	--	--	--	+	+	--	--	--	--	--	--	--	--	20%
4	LP	M	11	11	1st	--	--	+	--	+	+	--	--	--	+	+	--	70%
5	LP	M	12	12	1st	--	--	+	+	--	--	+	+	--	--	--	--	5%
6	LP	F	10	9	2nd	--	--	+	--	--	+	--	+	--	--	--	--	10%
7	LP	M	5	5	1st	--	--	+	+	--	+	--	--	--	--	--	--	60%
8	LP	M	10	10	1st	--	--	+	--	--	+	+	--	--	--	--	--	15%
9	LP	M	9	9	1st	--	+	--	+	+	--	+	--	+	--	--	--	30%
10	LP	F	15	15	1st	--	--	+	--	--	+	--	--	--	--	--	--	10%
11	LP	M	17	17	1st	--	--	+	+	--	+	+	--	--	--	--	--	30%
12	LP	M	9	9	2nd	--	--	+	--	+	+	--	--	--	--	--	--	10%
13	LP	F	15	14	3rd	--	+	+	+	+	+	+	--	--	--	--	--	90%
14	LP	M	15	15	1st	--	--	+	+	+	+	+	--	--	--	--	--	50%
15	LP	M	15	15	1st	--	--	+	+	+	+	+	--	--	--	--	--	40%
16	LP	F	11	11	1st	--	--	+	--	--	+	--	--	--	--	--	--	10%
17	LP	F	9	9	1st	--	+	+	+	+	+	+	--	--	--	--	--	25%
18	LP	F	18	18	--	--	--	+	--	--	+	--	--	--	--	--	--	10%
19	LP	M	12	12	1st	--	--	+	--	--	+	--	--	--	--	--	--	4%
20	Eruptive LP	F	18	14	4th	--	--	+	+	--	+	+	--	--	--	--	--	90%
21	Eruptive LP	M	8	8	1st	--	+	+	--	--	+	--	+	--	--	--	--	20%
22	L P H	M	13	10	3rd	--	--	+	--	--	+	--	--	--	--	--	--	5%
23	L P H	F	10	10	1st	--	--		+	--	+	--	--	--	--	--	--	10%
24	L P H	M	18	17	2nd	--	--	+	--	--	+	--	--	--	--	--	--	20%
25	L P H	F	12	10	3rd	--	--	+	--	--	+	--	--	--	--	--	--	2%
26	Linear LP	M	2	2	1st	--	--	+	--	--	--	--	--	--	--	--	--	2%

27	Linear LP	F	17	16	2nd	--	--	post. Aspect of right leg	--	--	+	+	--	--	--	--	--	20%
28	Actinic LP	F	5	5	1st	--	+	+	--	--	+	--	--	+	--	--	--	50%
29	Actinic LP	M	8	8	1st	--	+	+	--	--	+	+	--	--	--	--	--	20%
30	Actinic LP	M	13	13	1st	--	+	+	--	--	+	--	--	--	--	--	--	15%
31	Actinic LP	M	14	14	1st	--	+	+	--	--	--	--	--	--	--	--	--	16%
32	Follicular LP	M	11	11	1st	--	--	+	+		--	--	--	--	--	--	--	10%
33	Follicular LP	M	10	10	1st	--	+	--	--	--	--	--	--	--	--	--	--	2%
34	Follicular LP	F	14	12	4th	+	--	--	--	--	--	--	--	+	--	--	--	6%
35	Follicular LP	M	18	18	1st	--	--	+	--	--	--	--	--	--	--	--	--	8%

LN

1	L N	M	11	11	1st	--	--	+	+	--	--	--	+	--	--	--	--	5%
2	L N	M	12	12	1st	--	--	+	+	--	--	--	--	--	--	--	--	20%
3	L N	F	11	11	1st	--	--	+	+	+	--	--	--	--	--	--	--	18%
4	L N	F	4	4	1st	--	--	+	+	+	--	--	--	--	--	--	--	20%
5	L N	M	13	13	1st	--	--	+	+	+	--	+	--	--	--	--	--	10%
6	L N	F	5	5	1st	--	--	+	+	--	--	+	--	--	--	--	--	8%
7	L N	M	8	8	1st	--	--	+	--	+	--	+	--	--	--	+	--	4%
8	L N	M	6	3	4th	--	--	+	--	--	--	--	--	--	--	--	--	30%
9	L N	M	18	18	1st	--	--	+	+	+	--	--	--	--	--	--	--	30%
10	L N	F	11	11	1st	--	--	+	+	--	--	--	--	--	--	--	--	10%
11	L N	M	6	6	1st	--	--	+	+	+	--	--	--	--	--	--	--	18%

PLC

1	PLC	F	18	18	1st	--	--	+	+	--	--	--	--	--	--	--	--	20%

Lichen Striatus(LS)												
Sl. No.	Diagnosis	Gender	Age in year	Age of onset (Yr.)	Family histor	Distribution Of lesion						BSA
						Face	Hand		Leg		Trunk	
							Right	Left	Right	Left		
1	LS	F	7	7	--	--	--	--	+	--	--	2%
2	LS	F	5	5	--	--	--	+	--	--	--	2%
3	LS	F	8	8	--	--	--	--	--	+	--	2%
4	LS	M	13	13	--	--	--	--	--	+	--	3%
5	LS	M	6	6	--	+	--	--	--	--	--	2%
6	LS	F	6	6	--	--	--	--	+	--	--	2%
7	LS	F	14	14	--	--	--	+	--	--	--	2%
8	LS	F	14	14	--	--	--	--	--	--	+	3%
9	LS	M	12	12	--	--	--	--	--	--	+	4%
10	LS	M	13	13	--	--	--	+	--	--	--	3%
11	LS	M	2	2	--	--	--	--	+	+	--	8%
12	LS	M	3.5	3.5	--	--	+	--	--	--	--	5%
13	LS	M	4	4	--	--	--	--	--	+	--	3%
14	LS	F	5	5	--	--	--	--	--	+	--	3%
15	LS	F	17	17	--	--	+	--	--	--	--	9%

Pityriasis rosea (PR)

Sl. No.	Diagnosis	Gender	Age	Age of onset in (yr)	Distribution of lesions				Christmas tree	Type of scale	Herald patch	Nail change Pitting	BSA
					Face	Trunk	Extremities	Flexural					
1	PR	M	10	10	--	+	--	--	+	+	--	--	80%
2	PR	F	6	6	--	+	--	--	+	+	--	--	90%
3	PR	M	13	13	--	+	--	--	--	--	--	--	36%
4	PR	M	16	16	--	+	--	--	--	+	+	--	30%
5	PR	F	9	9	--	+	--	--	--	+	--	--	10%
6	PR	M	13	13	--	+	+		+	+	--	--	30%
7	PR	F	14	14	+	--	--	--	--	+	--	--	5%
8	PR	M	12	12	--	+	--	--	+	+	+	--	24%
9	PR	F	10	10	--	+	+	--	--	+	--	--	10%
10	PR	M	11	11	--	+	+	--	--	+	-	--	12%
11	PR	F	11	11	--	+	--	--	--	+	--	--	20%
12	PR	M	10	10	--	+	--	--	--	--	--	+	6%
13	PR	M	16	16	--	+	+	--	--	+	--	--	25%
14	PR	M	8	8	+	+	--	--	--	+	--	--	30%
15	PR	F	5	5	--	+	--	--	--	+	--	--	50%
16	PR	M	12	12	--	--	--	--	+	+	--	--	20%
17	PR	M	18	18	--	+	--	--	--	+	--	--	20%
18	PR	M	5	5	+	--	+	--	--	+	--	--	20%
19	PR	M	10	10	--	+	+	--	--	+	+	--	20%
20	PR	M	10	10	--	--	--	--	+	+	--	--	24%

Pityriasis rubra pilaris (PRP)

Sl. No	Diagnosis	Gender	Age	Age of onset	Family history	Distribution Of lesion				PPK	Nail change		BSA
						Scalp	Face	Extremities	Trunk		SUHK	Onychodystrophy	
1	PRP	F	17	17	--	+	+	+	+	+	+	--	80%
2	PRP	M	13	13	--	--	--	+	--	+	--	--	10%
3	PRP	M	4	4	--	+	+	+	+	+	+	+	80%
4	PRP	F	3	3	--	--	--	+	+	--	--	--	30%

MASTER CHARTS (HISTOPATHOLOGICAL PARAMETERS)

PS

S.No.	O.P. No.	Confluent Parakeratosis	Micro Munroabscess	elongation of rete ridges	Pustules of Kogoj	Suprapapillary thinning
1	80/12	+	--	+	+	+
2	117/11	+	--	+	+	--
3	640/11	+	+	+	+	+
4	764/11	+	+	+	+	+
5	948/11	+	--	+	+	+
6	1507/11	+	+	+	--	--
7	1506/11	+	--	--	+	+
8	1539/11	+	--	+	--	+
9	1708/11	+	--	+	+	+
10	2157/11	+	--	+	--	+
11	1847/12	+	+	+	+	--
12	2742/12	+	--	+	+	--
13	2256/12	--	--	+	+	+
14	285/12	--	--	+	+	+
15	1800/12	+	--	+	+	+
16	242/11	+	--	+	--	--
17	270/12	+	--	+	+	+
18	1800/11	+	+	+	--	+
19	2344/11	+	+	+	+	+
20	540/12	+	+	+	--	+
21	2554/12	+	+	+	+	+
22	807/12	+	+	+	+	+
23	2993/11	+	+	+	+	+
24	308/11	--	--	+	+	+
25	3079/11	--	+	+	--	+
26	3697/11	+	--	+	--	--
27	3776/11	+	--	+	+	+
28	2804/11	--	--	+	+	+
29	3665/11	+	+	+	+	+
30	3580/11	+	--	+	+	+
31	3697/12	+	+	+	+	+
32	1535/11	+	--	+	--	--
33	3776/12	--	--	+	--	+
34	9776/11	--	--	+	+	+
35	4548/11	--	--	+	--	--

LP

S.No.	O.P. No.	Saw toothed apperance	Wedge shaped hypergranulosis	vacuolar degeneration of basal layer	Pigment incontinence	Band like lymphohistiocytic infiltration
1	4578/11	--	--	+	+	--
2	1844/12	--	--	+	+	+
3	2817/12	+	+	+	+	+
4	2562/12	+	+	+	+	+
5	568/12	+	--	+	--	+
6	2416/11	+	+	+	+	+
7	2738/12	+	--	+	--	--
8	2738/11	+	+	+	+	+
9	1311/12	--	--	+	+	+
10	2981/12	--	+	+	+	+
11	1251/12	+	+	+	+	+
12	3130/11	--	+	+	+	+
13	3325/11	--	+	+	--	+
14	1209/11	--	+	+	+	+
15	2657/12	+	+	+	+	+
16	2915/12	--	+	+	+	+
17	131/12	--	+	+	+	+
18	3360/11	+	+	+	+	+
19	1085/11	+	+	+	+	+
20	3775/11	+	+	+	+	+
21	4578/11	--	--	+	+	+
22	3519/11	+	--	--	+	+
23	4680/11	--	+	+	+	+
24	4666/11	+	--	+	--	+
25	2327/12	+	+	+	+	+
26	2420/11	+	+	+	+	+
27	3759/11	--	+	+	--	+
28	3180/11	+	+	+	+	--
29	4505/11	+	+	+	+	+

Lichen Striatus(LS)						
S.No.	**O.P. No.**	**Spongiosis**	**Acanthosis**	**Focal parakeratosis**	**vacuolar degeneration of basal layer**	**Mixed lymphohistiocytic infiltration**
1	3054/11	+	+	+	+	+
2	3663/11	+	--	--	--	+
3	1537/12	--	--	--	+	+
4	2909/12	+	+	--	+	+
5	973/12	+	+	+	+	+
6	299/12	+	--	--	+	+
7	5013/11	+	+	+	+	+
8	3699/11	+	+	+	+	+
9	1837/11	+	+	--	--	+

Pityriasis rosea (PR)

S.No.	O.P. No.	Focal parakeratosis	Acanthosis	Extravasation of RBC's
1	3202/12	+	+	+
2	3094/11	+	+	+
3	3723/11	+	+	+
4	4711/11	+	+	--
5	1617/12	+	--	--
6	496/11	--	--	+
7	186/12	+	+	--
8	4261/11	+	--	-
9	2369/12	+	+	--
10	4347/11	+	+	--
11	1115/12	+	+	+
12	2153/12	--	--	--
13	2916/11	--	--	--
14	2322/12	--	--	--
15	2003/12	--	--	+
16	307/11	+	--	--
17	946/11	--	--	--

Pityriasis rubra pilaris (PRP)

S.No.	O.P. No.	Alternating ortho keratosis/parakeratosis	Acanthosis with broad short ridges	spongiosis	Focal or confluent hypergranulosis	Perivascular lymphocytic infiltration
1	337/11	+	+	+	+	+
2	4395/11	+	+	+	+	+
3	2617/11	+	+	+	--	--
4	1449/12	+	+	--	--	+