A CLINICAL STUDY ON PAPULOSQUAMOUS DISORDERS IN CHILDREN LESS THAN 12 YEARS

Dissertation submitted to

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 032

APRIL 2015

in partial fulfillment of the regulations required for the award of

M.D. DEGREE

IN

DERMATOLOGY, VENEREOLOGYAND LEPROLOGY

(BRANCH XII)

DEPARTMENT OF DERMATOLOGY, VENEREOLOGY

AND LEPROLOGY

COIMBATORE MEDICAL COLLEGE HOSPITAL

DECLARATION

I **Dr. PREETHA PRASAD** solemnly declare that the dissertation entitled **"A CLINICAL STUDY ON PAPULOSQUAMOUS DISORDERS IN CHILDREN LESS THAN 12 YEARS"** was done by me in the Department of Dermatology and Venereology at Coimbatore Medical College Hospital during the period from August 2013 to July 2014 under the guidance & supervision of **Dr. P. P. RAMASAMY M.D., D.D.,** Professor & Head of Department, Department of Dermatology and Venereology, Coimbatore Medical College Hospital, Coimbatore. The dissertation is submitted to Tamil Nadu Dr. MGR Medical University, Chennai towards the partial fulfillment of the requirement for the award of M.D., degree in Dermatology, Venereology and Leprology.I have not submitted this dissertion on any previous occasion to any university for the award of any degree.

PLACE: **Dr. PREETHA PRASAD**

DATE:

CERTIFICATE

This is to certify that the dissertation entitled **"A CLINICAL STUDY ON PAPULOSQUAMOUSDISORDERS IN CHILDREN LESS THAN 12 YEARS"** is a record of bonafide work done by **Dr. PREETHA PRASAD**, Post graduate student in the Department of Dermatology, Venereology and Leprology,Coimbatore Medical College Hospital, Coimbatore under the guidance of **Dr.P.P.Ramasamy M.D.,D.D.,** Professor & Head of Department, Department of Dermatology, Coimbatore Medical College Hospital, Coimbatore in partial fulfillment of the regulations of the Tamilnadu Dr.M.G.R Medical University, Chennai towards the award of M.D., degree(Branch XII) in Dermatology, Venereology and Leprology.

Date :

GUIDE,
Dr.P.P.Ramasamy M.D., D.D.,
Professor, Department of Dermatology,
Coimbatore Medical College & Hospital.

Date :

Dr. P. P. Ramasamy M.D., D.D.,
Professor & Head of the Department ,
Department of Dermatology,
Coimbatore Medical College & Hospital

Date :

Dr.S.Revwathy MD.DGO., DNB
Dean,
Coimbatore Medical College & Hospital
Coimbatore.

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : PREETHA PRASAD.

Course : M.D - DERMATOLOGY & VENEROLOGY.

Period of Study : 2012 - 2015

College : COIMBATORE MEDICAL COLLEGE

Dissertation Topic : A CLINICAL STUDY ON PAPULO-SQUAMOUS DISORDERS IN CHILDREN LESS THAN 12 YEARS

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not accepted~~ and you are permitted / ~~Not permitted~~ to proceed with the above Study.

DEAN
Coimbatore Medical College & Hospital,
Coimbatore

turnitin

Digital Receipt

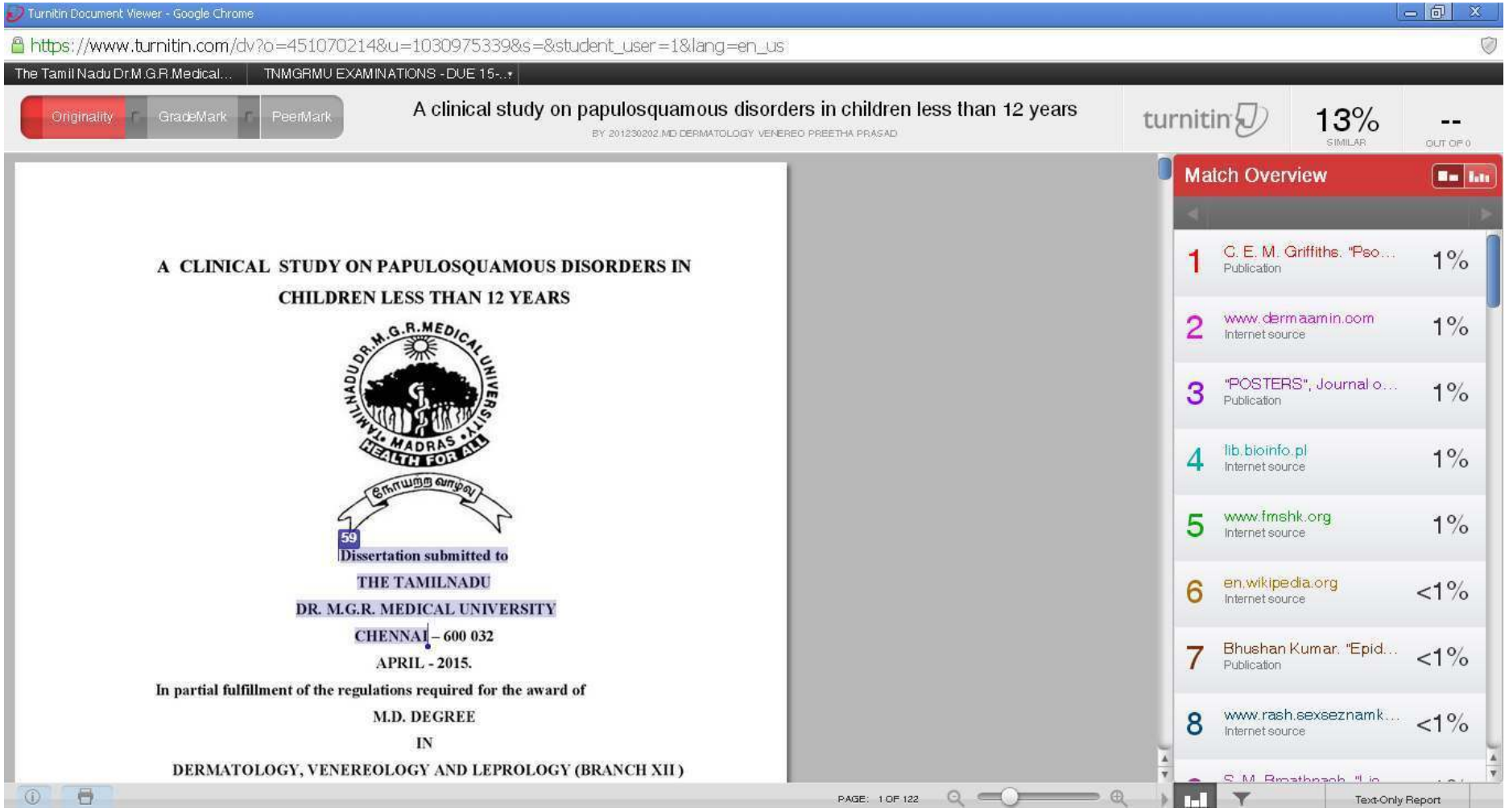
This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201230202.md Dermatology Venereo..
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	A clinical study on papulosquamous ..
File name:	ON_PAPULOSQUAMOUS_DISORD.
File size:	294.53K
Page count:	122
Word count:	17,692
Charader count:	101,973
Submission date:	19-Sep-2014 05:24AM
Submission ID:	451070214

A CLINICAL STUDY ON PAPULOSQUAMOUS DISORDERS IN CHILDREN LESS THAN 12 YEARS

Coimbatore Medical College Hospital,


Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?o=451070214&u=1030975339&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical...
TNMGRMU EXAMINATIONS - DUE 15-...
Originality
GradeMark
PeerMark
A clinical study on papulosquamous disorders in children less than 12 years
BY 201230202.MD DERMATOLOGY VENEREO PREETHA PRASAD
turnitin
13%
SIMILAR
--
OUT OF 0
A CLINICAL STUDY ON PAPULOSQUAMOUS DISORDERS IN
CHILDREN LESS THAN 12 YEARS
Dissertation submitted to
THE TAMILNADU
DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600 032
APRIL - 2015.
In partial fulfillment of the regulations required for the award of
M.D. DEGREE
IN
DERMATOLOGY, VENEREOLOGY AND LEPROLOGY (BRANCH XII)
PAGE: 1 OF 122
Match Overview
1 C. E. M. Griffiths. "Pso... Publication 1%
2 www.dermaamin.com Internet source 1%
3 "POSTERS", Journal o... Publication 1%
4 lib.bioinfo.pl Internet source 1%
5 www.fmshk.org Internet source 1%
6 en.wikipedia.org Internet source <1%
7 Bhushan Kumar. "Epid... Publication <1%
8 www.rash.sexseznamk... Internet source <1%
Text-Only Report

ACKNOWLEDGEMENT

First of all, I Thank the Almighty for what I am today. I wish to thank the Dean of this institution, **Dr. S. Revwathy MD., D.G.O., DNB,** for permitting me to undertake this study. I would like to thank my guide, **Dr.P.P.Ramasamy M.D., D.D., Professor** and Head of Department of Dermatology, Venereology and Leprology from the bottom of my heart for his scholary advice and timely assistance during the preparation of the thesis.

My special thanks to **Dr. M. Revathy M.D.,** Assistant Professor of Department of Dermatology for her constant encouragement and valuable suggestions during the period of this study.

I would like to express my gratitude to our **Prof. Dr. K. Mahadevan, M.D, D.V.,** Department of STD for his support andguidance .

I owe great debt of gratitude to **Dr. R. Madhavan M.D., and Dr.S.Bharathi M.D.,** Assistant Professors in Department of Dermatology for their kind support and encouragement. I sincerely thank **Dr.B.Eswaramoorthy M.D.,** and **Dr.A.P.Balaji, Assistant Professors** in Department of Dermatology and Venereology for their valuable guidance and help.

I extend my sincere thanks to all my postgraduate colleagues for their help and co-operation for collecting datas in the study. I am very grateful to all patients for their co-operation and participation in the study.

On a personal note I thank my parents, sister, husband and his parents for their constant moral support, which helped me to make this study a reality.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF CHARTS

LIST OF ABBREVIATIONS

IL-1	–	Interleukin-1
TNF alpha	–	Tumour Necrosis Factor alpha
MHC	–	Major Histocompatibility Complex
IFN	–	Interferon
GPP	–	Generalised Pustular Psoriasis
TNF	–	Tumour Necrosis Factor
FDA	–	Food and Drug Administration
HHV	–	Human Herpes Virus
DNA	–	Deoxy ribonucleic Acid
HHV 7	–	Human Herpes Virus 7
EBV	–	Epstein Barr Virus
PR	–	Pityriasis Rosea
LP	–	Lichen Planus
HLA	–	Human Leukocyte Antigen
HSV 2	–	Herpes Simplex Virus 2
HIV	–	Human Immunodeficiency Virus
HCV	–	Hepatitis C Virus
HPV	–	Human Papilloma Virus
CD8	–	Cluster of Differentiation 8
TGF	–	Transforming Growth Factor
CD95L	–	Cluster of Differentiation 95
CD95	–	Cluster of Differentiation 95

NBUVB	–	Narrow Band Ultraviolet B
PUVA	–	Psoralene Ultraviolet A
PRP	–	Pityriasis Rubra Pilaris
HAART	–	Highly Active Antiretroviral Therapy
IL–1B	–	Interleukin – 1B
ILVEN	–	Inflammatory linear verrucous epidermal naevi
LN	–	Lichen Nitidus
MEN 2C	–	Multiple Endocrine Neoplasia 2C
CD4	–	Cluster of Differentiation 4
CD8	–	Cluster of Differentiation 8
LN	–	Lichen Nitidus
UVB	–	Ultraviolet B
LPP	–	Lichen Plano Pilaris
MF	–	Mycosis fungoides
PLC	–	Pityriasis lichenoides chronica
PLEVA	–	Pityriasis Lichenoideset Varioliformis Acuta
IgM, C3	–	Immunoglobulin M, Complement 3
MMR	–	Mumps Measles Rubella
DPT	–	Diphtheria Pertussis Typhoid
PAC	–	Papular acrodermatitis of chilhood
GCS	–	Gianotti Crosti Syndrome
KOH	–	Potassium Hydroxide
VDRL	–	Venereal Disease Research Laboratory
OPD	–	Out Patient Department

COLOUR PLATES

ABSTRACT

Title:

A Clinical study on papulosquamous disorders in children less than 12 years,

Background and objectives

Among the wide spectrum of skin diseases in children, papulosquamous disorders form quite a common group. Papulosquamous disorders during childhood can present a vast array of clinical findings. This study was done to know the hospital based prevalence and the clinical patterns of papulosquamous disorders among children less than 12 years of age presenting to skin OPD, Coimbatore Medical College Hospital.

Methodology

This is a descriptive study conducted from August 2013 to July 2014 at the outpatient Department of Skin and STD, Coimbatore Medical College Hospital.105 consecutive children with papulosquamous disorder were included in the study. Routine investigations were done for all cases and special investigations like potassium hydroxide mount, culture and skin biopsy were done for relevant cases.

Results

Papulosquamous disorders constituted 0.81%% of the pediatric dermatoses during the study. M: F ratio was 1.1:1.0. Majority of the patients belonged to 10-12years age group. Among papulosquamous disorders, psoriasis was the most common disease (25.7%), followed by pityriasisrosea (22.95%) and lichen planus(18.09%).

CONCLUSION

Papulosquamous disorders are common in children and have varied presentations. Genetic inheritance of papulosquamous disorder is less significant. Papulosquamous disorders in children require a separate view from adult dermatoses as there are important differences in clinical presentation, treatment and prognosis. By understanding the morphological characterestics of papulosquamous diseases and differentiating the diseases clinically , we can explain the prognosis of the disease to the parents, which will alleviate their worry.

KEY WORDS:

Papulosquamous, childhood, psoriasis, pityriasis rosea

INTRODUCTION

Papulosquamous diseases are typically characterized by well-demarcated areas of papules and scales typically on an erythematous background. Papulosquamous disorders form a common group among the wide spectrum of skin diseases in children. The disease which comes under this group varies from the inflammatory skin disease like psoriasis to infections like syphilis and from self-resolving pityriasis rosea to the treatment resistant parapsoriasis.

The diseases which come under papulosquamous group mimic each other. Atypical presentations can be there in individual diseases. Diagnosing these atypical variants clinically will save the time,resources and avoid procedures like biopsy in children.

Children are not little adults. A child's skin is different from that of adults. The skin of children is thinner. They are more prone for damage from external insults. Surface to volume ratio of child's skin is greater than that of adults, so more amount of chemicals can be absorbed by them.

The presentation of the same disease may differ in children and adults. Prevalence of subtypes in the same disease will vary between adults and children.

Though there are various studies on paediatric dermatosis and individual papulosquamous disorders, there is a paucity of studies regarding papulosquamous disorders in children.

Thus studying the prevalence and clinical features of papulosquamous disorders in children helps dermatologists to understand better the papulosquamous disorders in children and to manage them appropriately.

AIM AND OBJECTIVES

1. To study the prevalance of papulosquamous disorders in children less than 12 years of age

2. To study the prevalence, age sex distribution, morphology of individual papulosquamous disorders in children less than 12 years of age

REVIEW OF LITERATURE

The papulosquamous diseases,characterized by scaly papules or plaques, constitute the largest conglomerate group of disease seen by dermatologist. The word “Papule” is derived from the latin word papula which means pimple and “scale”is derived from latin words squames.The nosology of these disorders is based on a descriptive morphology of clinical lesions characterized by scaly papules and plaques.[1]These diseases assume considerable importance because of their frequency of occurrence .Because all are characterized by papules,patches,plaques and scaling,clinical confusion may result in their differentiation. Separation of each of these disease becomes important because the treatment and prognosis for each tends to be disease specific.

In a study of pattern of pediatric dermatoses in Rajasthan,papulosquamous disorders constituted 1.66% of all the dermatoses.[2]In a similar study in Saudi Arabia, papulosquamous disorders constituted 8.1% of all dermatoses.[3]

Papulosquamous disorders of children can present with various clinical manifestations. The frequency of distribution of papulosquamous disordes in children varies in different age groups also. For example

seborrheic dermatitis is relatively more common during early ages of life. Certain morphological types of these disorders will be more common in children than that of adults. Guttate psoriasis is more common in children than adults.

The papulosquamous disorders have classic and distinct clinical features. But sometimes the morphology of the lesion will appear atypical.The diseases which are classified under papulosquamous disorder can present with non papular non scaly form also. For example, bullous lichen planus, purpuric pityriasis rosea and pustular psoriasis.

Some of the diseases in this group are chronic and relapsing. This necessitates periodical examination of the children and long term follow up. So parent education is an important for the better management of these group of disorders. They should be advised regarding adherence to the treatment and avoidance of the trigerring factors such as minor trauma.

Various authors have included different set of disorders in their study of papulosquamous disorders.

Gibson et al , perry H.O considered Psoriasis, Pityriasis Rubra Pilaris, Pityriasis Rosea, Lichen Planus, Lichen Nitidus , lichen striatus and parpsoriasis in their study.[4] Toussaint S. and Kamino H considered

the same set of diseases in their study.[5] But Hall,[6]includedpsoriasis, Pityriasis rosea,Lichen Planus, tinea versicolor, seborrheic dermatitis, secondary syphilis and drug eruptions in his study.

ICD classification of papulosquamous disorders[7]

Psoriasis

- Psoriasis vulgaris
- Generalized pustular psoriasis
- Impetigo herpetiformis
- Von zumbusch's disease
- Acrodermatitis continua
- Pustulosis Palmaris et plantaris
- Guttate psoriasis
- Arthropathic psoriasis
- Other psoriasis
- Psoriasis.unspecified

Parapsoriasis

- Pityriasis lichenoides et varioliformis acuta
- Mucha habermann disease

- Pityriasis lichenoides chronica
- Lymphomatoid papulosis
- Small plaque parapsoriasis
- Large plaque parapsoriasis
- Retiform parapsoriasis
- Other parapsoriasis
- Parapsoriasis unspecified

Pityriasis rosea

Lichen planus

Other papulosquamous disorders

- Pityriasis rubra pilaris
- Lichen nitidus
- Lichen striatus
- Lichen ruber moniliformis
- Infantile papular acrodermatitis(Gianotti-Crosti syndrome)
- Other specified papulosquamous disorders
- Papulosquamous disorder ,unspecified

Papulosquamous disorders in disease classified elsewhere

Psoriasis

Psoriasis is a common, chronic, disfiguring, inflammatory and proliferative condition of the skin, in which both genetic and environmental influences have a critical role.[8] It is characteristized by red, scaly, sharply demarcated, indurated plaques, present particularly over extensor surfaces and scalp. The disease is enormously variable in morphology, duration, periodicity of flares, severity and extent. Plaque psoriasis is the most frequent type in children, as in adults. However initial lesions are often smaller, thinner and less scaly.[9]When compared to adults certain clinical variants like erythroderma, arthropathy, and localized and generalized pustular psoriasis are rare in children.[10]

Epidemiology

A definitive paucity of studies existson the epidemiological background of childhood psoriasis. Psoriasis accounts for about 4% of all dermatoses in children less than 16yrs of age and occurs at all ages in 2-3% of the population.[11,12]

Psoriasis constituted 3.4% of all skin disorders in a study conducted in Kashmir[13]. A study from south India in children less than

14 years showed a prevalence of 1.4%.[14] A prospective study in Kuwait showed a prevalence of 4% among paediatric dermatoses.[15]In a study conducted at Saudi Arabia over a period of 24 months psoriasis constituted about 22.6% among the papulosquamous disorders in children less than 13 yrs.[3]

About 31-45% of adults with psoriasis have noted the onset during first two decades of life. As much as 40% of adult patients with psoriasis have reported manifestations of psoriasis in childhood, with at least one-third of the patients demonstrating features of psoriasis before the age of 16.1 years.[17]. A study conducted in north India showed, the average age of onset of psoriasis in boys is 6-10years and that of girls is 10-14 years.[18]

Psoriasis has been reported to be more common in girls.[19] However Nanada et al observed an equal gender distribution in childhood[20] which is similar to results of Morris et al[21] and Kumar et al[18] studies.

Seventy percentage of affected paediatric population have a family history of psoriasis and affected twins have been described.[22] But history of occurrence in the family varies in different races. A study conducted in north India showed a positive family history of 4.5% among children.[18]A study conducted in China also showed a family history of only 8%.[23] But a study conducted in United States showed a family

history of 51.4%.[24]The lifetime risk of developing psoriasis is thought to be 4% if no parent is affected,28% if one parent is affected and 65% if both parents are affected. [25]

Chronic plaque type psoriasis is the most common presentation in children which is similar to adults. Guttate psoriasis is more common in children. The severity of the condition may vary from mild localized disease to life threatening neonatal pustular dermatoses or exfoliative dermatoses.

Etiopathogenesis[8]

Henseler and Christophers demonstrated that the bimodal peak in disease onset could be taken as evidence for the existence of two pathogenetically distinct forms of the disease, similar to the model for diabetes mellitus. Thus, type 1 is hereditary, strongly HLA associated (particularly HLA-Cw6), early onset and more likely to be severe. Type II is sporadic, HLA unrelated, of late onset and usually mild.Childhood psoriasis have more familial preponderance than adult onset psoriasis.

Physical, chemical, electrical, surgical, infective and inflammatory insults have been recognized to trigger psoriatic lesions.Drugs like lithium, antimalarials, beta blockers, ACE Inhibitors, NSAIDs are reported to be responsible for onset or exacerbation of psoriatic

lesions.Acute guttate psoriasis may be associated with past history of streptococcal throat infection.

In general sunlight is beneficial to psoriatic lesions but in a minority of patients, psoriasis can be exacerbated by sunlight.

In HIV infection, severity of psoriasis and incidence of psoriatic arthropathy will be more.

Pathogenetic mechanism

Pathogenesis of psoriasis can be explained by the following four factors;

- **Epidermal proliferation**

There is an increase in the proliferation of cells in the basal and suprabasal layers of epidermis. There is a sevenfold increase in the number of cycling cells. Transforming growth factor is an important mediator for keratinocyte proliferation.

- **Vascular changes**

Vascular growth or angiogenesis is an important factor in the pathogenesis of psoriasis. Immunohistochemical studies have shown that there is a fourfold increase in the endothelium of superficial dermis with a proliferating index of 3%.

In vivo models of angiogenesis have demonstrated that epidermal keratinocytes are the primary source for angiogenesis. These cells will produce various angiogenic factors including vascular endothelial growth factor. VEGF is overexpressed in psoriatic epidermis and receptors are increased in psoriatic microvasculature.

Inflammatory mediators like E cadherin and intercellular adheshion molecule (ICAM 1) is overexpressed in dermal capillaries of psoriatic skin, which cause accumulation of lymphocytes in lesional epidermis and dermis. Other inflammatory mediators which induce leukocyte homing in skin are histamine, neuropeptides, IL-1 and TNFα.

- **Molecular genetics**

Psors1, which is located within MHC on chromosome 6p is the major psoriasis genetic determinant .It accounts for about 35-50% of heritability of the disease.

- **Immunology and inflammation**

There is a dysregulation in the innate immune mechanism in psoriatic patients. Innate immunomediators like antimicrobial peptides are overexpressed in psoriatic lesions. This will lead to antigen driven T cell expansion and activation. The most important subsets that are

activated in psoriasis are Th1 and Th17. Th17 clone of Tcells will overexpress IL23, which will induce psoriatic phenotype. Avtivated T cells will also secrete IFN gamma, which causes keratinocyte proliferation.

Histopathology[5,26]

A fully developed plaque of psoriasis will show hyperkeratosis with confluent parakeratosis. Stratum corneum will show micromunro abscess.spongiform pustules of Kogoj will be there in the stratum malphigi.. There will be hypogranulosis with regular acanthosis and

Suprapapillary thinning of epidermis Dermis will show elongation and edema of dermal papillae with dilated tortuous dermal capillaries and

lymphocytic infiltration.

CLINICAL FEATURES

Chronic plaque psoriasis

This is the most common morphological variant of psoriasis seen in children. The lesions will be well defined erythematous plaques with easily removable silvery white scales distributed over extensor aspect of knee, elbow and trunk.As compared to adults, in children the plaques are

often smaller and scales are finer and softer[27]. Children will have severe itching than adults.

Removal of scales over the plaque with a glass slide will reveal a red glistening membrane called Buckley's membrane. Successive scraping of the plaque will produce fine punctate bleeding points. This phenomenon is called Auspitz sign. Koebnerisation is a process in which isomorphic cutaneous lesions develops at the line of trauma. Koebner phenomenon can develop at the sites of trauma in psoriasis.

Facial involvement is more common in children than adults. It occurs in about 4-5% of patients. Periorbital area is the most typical site affected.[27]

Guttate psoriasis

The term guttate psoriasis is derived from the Latin word *gutta* ,which means "a drop". The lesions of guttate psoriasis will be erythematous papules and plaques which vary from 2-3mm to 1cm with overlying silvery white scales, which are distributed almost symmetrically over trunk and proximal part of extremities. Guttate psoriasis mostly occurs in children ,often a week after streptococcal pharyngitis.

Various studies have shown that about 40% of children with guttate psoriasis may progress to chronic plaque psoriasis.[28]

Scalp psoriasis[29]

In children scalp is frequently the first affected site (20-40%). The lesions will be similar to that occurring in other parts of the body. The psoriatic lesion over the scalp and eyebrows can be greasy and more salmon coloured, often termed sebopsoriasis. Lesions of psoriasis frequently extend beyond the hairline, to forehead, preauricular, postauricular and nuchal region. This feature of psoriasis is in contrast to seborrheic dermatitis which confines to the hairline.

Tinea amiantacea , also called as pityriasis amiantacea is a variant of scalp psoriasis occurring in children, which is characterized by large plates of firmly adherent scales(asbestos –like) on scalp and hair. It usually begins in childhood and about 2-15% may progress to more typical psoriasis.

Flexural psoriasis

Flexural psoriasis involves groins, vulva , axillae, submammary folds, gluteal cleft and other body folds. It is more common in adults than in children. In children the most frequently affected area is napkin area

and axillae. It presents as sharply demarcated bright red plaques. Scales may not be evident clinically but may be revealed on gentle scrapping. Many infants with diaper area psoriasis may show psoriatic plaques in other regions.

Nail psoriasis[29]

Nail involvement is seen in 25-50% of pediatric patients with psoriasis. Pitting is the most common manifestation and other features like discolouration onycholysis, subungual hyperkeratosis, splinter haemorrhages can also be seen . Nail bed and hyponychium may show circular areas of discoloration, which resembles an 'oil drop'. Secondary infection with bacteria, candida and dermatophytes can be seen with increased frequency. Nail disease will be more severe if onset of psoriasis is early and familial.

Erythrodermic psoriasis: [27]

It is a severe variant of psoriasis characterised by generalised erythema and scaling. It accounts for about 16-24% of all cases of erythroderma cases. It can develop in a patient with pre existing chronic plaque psoriasis or as a manifestation of unstable psoriasis precipitataed by drugs, infection, steroid withdrawal or overuse of tar or dithranol. Erythrodermic psoriasis is relatively rare in chilren.

Pustular psoriasis

Classification of pustular psoriasis is[8]

1 **Localized pustular psoriasis**:

(a) palmoplantar pustulosis

(b) acrodermatitis continua

2 **Generalized pustular psoriasis:**

(a) acute

(b) of pregnancy

(c) infantile and juvenile

(d) circinate

(e) localized (not hands and feet).

Infantile and juvenile pustular psoriasis[8]

Pustular psoriasis is a variant of psoriasis described by Von zumbusch in 1910. In children, pustular psoriasis is very rare and accounts for about 1.1% of childhood psoriasis. Average age of onset of pustular psoriasis in children is 2-10 years,but cases with onset in the first year of life have also been described. Two case of congenital

generalised pustular psoriasis have been reported. Male preponderance is seen in childhood GPP with a ratio of 3:2.

Infantile pustular psoriasis is usually benign. Generally systemic symptoms are absent. Fever and symptoms of acute toxicity can develop in some children. Annular and circinate forms are the most common presentations. Lesions begin as discrete areas of erythema, which become raised and oedematous. Pustules appear at the periphery which desiccate, leaving a trailing fringe of scale as the lesion slowly advances. Von zumbusch pattern also be seen in children, which have an abrupt onset with features of toxicity. In older children the pattern will resemble that of adults.

The lesions will subside within few weeks but recurrent episodes can be there. Von zumbusch type can progress to erythroderma. The prognosis of pustular psoriasis in children is variable.

Extracutaneous involvement [29]

Arthritis : Psoriatic arthritis is biphasic in pediatric population. Younger children will present with dactylitis and small joint involvement. Most of them will be female children. The affected fingers and toes will have a "sausage shaped" appearance due to the juxtaarticular swelling. Radiological examination of metatarsophalangeal and

metacarpophalangeal joint will show "pencil-in-cup" or "pencil-and-goblet" deformity.

Older children with psoriatic arthritis will have axial joint involvement and enthesitis.

A study of 211 children with moderate and severe psoriasis showed that 9% of affected children had joint involvement.

Uveitis:14-17% of children with psoriatic arthritis will have asymmetric anterior uveitis .

Others : Recent studies have shown that there is a clear link between psoriasis and metabolic syndrome and cardiovascular risk. Studies in pediatric patients also showed that risk of obesity, cardiovascular complications begins in childhood and adolescence.

Differential diagnosis:

Table 1:Differential diagnosis of psoriasis in childhood[30]

TYPE OF PSORIASIS	DIFFERENTIAL DIAGNOSIS
Psoriasis vulgaris	Discoid eczema Tinea corporis SCLE Lichen simplex chronicus SPEK Cutaneous Tcell lymphoma
Guttate psoriasis	Pityriasis rosea Pityriasis lichenoides chronicus Lichen planus Secondary syphilis Drug eruption
Flexural psoriasis	Intertrigo Candidiasis Hailey-Hailey disease
Erythrodermic psoriasis	Drug induced erythroderma PRP Eczema CTCL

Pustular psoriasis	Impetigo Superficial candidiasis Subcorneaal pustular dermatosis Transient neonatal pustular melanosis Acropustulosis of infancy AGEP
Scalp psoriasis	Tinea capitis Atopic dermatitis Seborrheic dermatitis
Nail psoriasis	Tinea unguium Nail dystrophy Lichen planus

Treatment [31]

Treatment of psoriasis in children is a therapeutic challenge. There is no definite guidelines for the management of psoriasis in children and many therapeutic agents are not approved for use in children. The aim of treatment of psoriasis is to control the disease and improve the quality of life of the children. Success of treatment depends on compliance to the treatment which can be enhanced by parent education about nature of disease and treatment options.

Topical vitamin D analogue(calcipotriol and calcitriol) is an effective well tolerated treatment option for mild to moderate childhood psoriasis. Studies have shown that calcipotriol is safe and effective in treating childhood psoriasis ,involving<30% of body surface area. The maximum recommended dose is 50g/week/m^2 .Vitamin D analogue induces differentiation of keratinocytes , inhibits their proliferation and inhibits immunological mediators in the pathogenesis of psoriasis. It should not be applied on the face,scalp,genital area and areas under occlusion. Hypercalcemia can result from application of large quantities.

Tacrolimus is an imunomodulator which inhibits calcineurin and prevents the production of inflammatory cytokines from T-cells. Topical 0.1% tacrolimus is an effective treatment option for psoriasis over the face and intertriginous areas.

Guttate psoriasis and pustular psoriasis can be precipitated by streptococcal infections. So appropriate antibiotics can be given in these forms of psoriasis.Even tonsillectomy have been advised in recurrent pustular psoriasis.

Methotrexate can be used in children with severe and extensive psoriasis. It will be more beneficial when psoriasis is associated with arthritis. Retinoids and ciclosporin are effective in pustular psoriasis.

Children on long term retinoids are at a risk of premature epiphyseal closure. So they require radiological monitoring at an interval of 1 year. Hypertension, nephrotoxicity,hypertrichosis and gingival hyperplasia are the adverse effects of ciclosporin.Phototherapy can be used in extensive disease as an alternative to systemic treatment, but it has the potential side effect of carcinogenicity and premature ageing of the skin.

Biologicals play an important role in the treatment of childhood psoriasis which are resistant to topical and systemic therapy. TNF alpha blocking biologicals are etanercept, infliximab and adalimumab . T cell targeting biologicals are efalizumab and abatacept. Efalizumab ,amonoclonal antibody directed against CD11a was FDA approved for treatment of moderate to severe plaque psoriasis. Abatacept, a Tcell co stimulation modulator is used to treat psoriatic arthritis.

Prognosis [30]

Guttate psoriasis in children is usually a self limiting disease, which resolves within 12-16 weeks. In contrast to this, chronic plaque psoriasis has a prolonged course with remission and exacerbation. The other variants like erythrodermic and psoriatic arthropathy have poor prognosis. The disease will be severe and persistent.

PITYRIASIS ROSEA

Pityriasis rosea is a common,self limiting skin eruption that typically begins as a single thin oval scaly plaque on the trunk, known as herald patch. The word pityriasis means scales and rosea means pink. Camille melchoir gibert coined the name and gave the accurate description of pityriasis rosea in 1860.[32]Alfred Blashko described peripheral scaling in 1899. Emile vidal described pityriasis circinata et marginata in 1882.[33]

Epidemiology:

The incidence of pytiriasis rosea ranges from 0.39[34] to 4.8[35] per 100 dermatology cases. The incidence of pityriasis rosea in paediatric patients is 1.02 per 100 patients.[36] The prevalence of pityriasis rosea reported in adolescents is 0.6%. [37]

Most of the cases are seen between the ages of 10 and 35.[38]The youngest reported age of pityriasis rosea was 3 months.[39]It is very rare in those who are less than 2 years and more than 65 years. Male and female are affected equally. But various studies have shown a slight female preponderance of approximately 1.5:1.[38,40]

Aetiology

Pityriasis rosea is a disease of unknown origin. The natural course of the disease, prodromal symptoms, primary herald patch , secondary eruptions followed by complete remission suggests an infectious etiology. This is also strengthened by the observation of clustering of the disease in some communities.[41]

Studies have shown that there is a relationship between pityriasis rosea and HHV 6 and 7. Electron microscopic examination of lesional biopsies have shown evidence of human herpes virus DNA.[42] HHV7 PP85 antigen can be detected in lesional biopsies of pityriasis rosea.[43]

Other viruses like cytomegalovirus, EBV, adenovirus, influenza virus, parainfluenza virus, parvovirus B19, picornavirus have also been suspected to be associated with pityriasis rosea.[41] Bacteria which are suspected to be associated with PR are legionella, mycoplasma and chlamydia.

T lymphocytotoxic antibodies[44] and antinuclear antibodies[45] are also seen in patients with pityriasis rosea, which suggests an autoimmune etiology.

Clinical features:

The initial lesion of PR is known as primary plaque of PR or herald patch. Herald patch is seen in 50-90% of the inividuals with PR.[45] The characteristic lesion of herald patch is a 2-4 cm well demarcated oval or round salmon coloured, erythematous or hyperpigmented plaque with fine collarete of scale, just inside the periphery of the plaque. It is usually situated on the thigh or upper arm, the trunk or the neck; rarely it may be seen on the face, scalp or the penis. A gap of 2 days to 2 months may be there between herald patch and secondary eruption. But usually secondary eruption occurs within 2 weeks of appearance of mother patch.

The secondary eruption can be of two types. i) small plaques resembling miniature form of primary plaque distributed along the line of clevage in a Christmas tree pattern. ii) small red non scaly papules that spreads peripherally. Secondary eruptions occurs in crops at intervals of a few days to reach a maximum in about 10 days. The lesions are mainly distributed over anterior aspect of chest, abdomen, back. Sometimes the lesions may be seen over the neck and proximal extremities.

In children, there will be predominantly papular or urticarial lesions in the early stages, which are later surmounted by an inconspicuous ring of fine scales.[46] The centre of the lesion may be covered with scales in children which gives a lichenoid appearance .

An atypical presentation may be seen in about 20 percentage of the patients.[45] The herald patch may be absent or present as double or multiple lesions. The primary plaque may be the only manifestation of the disease. Papular PR is the most common atypical presentation in children. Vesicular, pustular,purpuric, haemorrhagic, erythema multiforme like and urticarial like atypical morphological types may be seen. In children distribution of the secondary eruptions mayoccur exclusively over the extremities,and face. Localized forms of Pityriasis rosea may be seen over scalp , axillae, vulva and groin.

Severe pruritis is seen in about 25 percentage of the patients. Usually children do not complain of intense pruritis. Flu like symptoms like generalised malaise, headache, nausea , loss of appetite , fever and arthralgia are reported in a minority of patients.

DIAGNOSTIC CRITERIA OF PITYRIASIS ROSEA[47]

Essential clinical features:

1. Discrete circular or oval lesions
2. Scaling on most lesions
3. Peripheral collarette scaling with central clearance on at least two lesions

Optional clinical features (at least one has to be present):

1. Truncal and proximal limb distribution, with less than 10% of lesions distal to midupper-arm and mid-thigh and secondary eruptions.
2. Orientation of most lesions along direction of the ribs
3. A herald patch (not necessarily the largest) appearing at least two days before the generalized eruption

Exclusional clinical features:

1. Multiple small vesicles at the center of two or more lesions
2. Most lesions on palmar or plantar skin surfaces
3. Clinical or serological evidence of secondary syphilis

Histopathology:[5]

Epidermis will show mounds of parakeratosis with plasma, decreased granular layer, spongiosis, moderate acanthosis. Dermis will show perivascular infiltrate predominantly of lymphocytes, eosinophils and histiocytes and extravastion of RBCs. Exocytosis is also seen. Herald patch will show pronounced acanthosis, deeper and denser perivascular inflammatory infiltrate and papillary dermal edema.

Differential diagnosis:[45]

Guttate psoriasis, tinea corporis , pityriasis versicolor , nummular dermatitis, parapsoriasis ,pityriasis lichenoides chronica, drug eruptions, viral rashes and secondary syphilis.

Treatment :

No active treatment is necessary as pityriasis rosea have a self limiting course.[45]We should educate the patient about the disease process and reassure them. If the child is symptomatic , topical antipruritics, topical steroid and oral anti histamines can be given. If the patient presents with flu like symptoms in the earlier stage or with extensive disease , a high dose of acyclovir will hasten the resolution.[42] Phototherapy is also useful in some patients.[48]Earlier oral antibiotics like

erythromycin and azithromycin were thought to have beneficial role in PR.The use of oral erythromycin antibiotic (1 g four times a day for 2 weeks for adults) was reported to clear the disease[49] within 2weeks of treatment but later studies have proven that there is no significant efficacy for these antibiotics.[50,51]

Prognosis:[45]

All patients with PR will have a spontaneous resolution of the disease , normally between 4-10 weeks after the onset of the disease. Post inflammatory hypopigmentation and hyperpigmentation can be there. Patients who are treated with phototherarpy have more chance of developing post inflammatory hyperpigmentation.

LICHEN PLANUS

Lichen planus is a pruritic papulosquamous disease of unknown etiology which is characterised by violaceous flat topped polygonal scaly papules involving the flexor aspect of wrists, legs, oral and genital mucosa.[52]Lichen planus is derived from the greek word *leichen* meaning tree moss and latin word *planus* meaning flat.[53] The word lichen planus is first coined by Erasmus Wilson in 1869.[54]

Epidemiology

Lichen planus is distributed worldwide. Overall prevalence of LP in general population is less than 1 percent .[55] Lichen planus is more common in adults above the age of 30.About 60-65% of cases occur in adults above 30 years of age. Both oral LP and cutaneous LP are reported rarely in children. Various studies have shown a prevalence of 2-11% in children and adolescents.[55] In a study conducted in children in Birmingham ,UK there was an over representation of south Asians in the series.[56] The youngest age of LP was reported in a 3 weeks infant.[57]

Although LP is sporadic in origin, cases with familial predisposition are recorded. In the literature , less than 100 cases of familial lichen planus have been reported.[58]

About 10-15% of adult patients with cutaneous LP will have nail involvement.[59]In children nail involvement is rare. Nail involvement in children ranges from 0-16.6%.[55] In a study conducted in nail LP in children, out of 15 cases, 10 children had typical nail matrix lesions, 2 children had 20-nail dystrophy (trachyonychia), and 3 children had idiopathic atrophy of the nails.[60]

Mucosal involvement is seen in upto 40% of the paediatric patients as compared to 50-70% of adult patients.[55,61.] Oral lichen planus without cutaneous lesions will occurin 20-30% 0f patients.[58]In paediatric population,mucosal involvement is more common in children in India and Kuwait.[55]Follicular involvement of LP is rare in childhood.

Aetiopathogenesis

Various theories have been put forwarded to explain the etiology of LP but exact mechanism has yet to be elucidated.

In 1977,Black proposed an autoimmune mechanism in the etiopathogenesis of LP. This is based on clinical and histological similarity of LP with LE, association of other autoimmune disease with LP and immunoglobulin staining of basement membranes and colloid bodies which is similar to autoimmune disease.[62]

Specific HLA types have been found associated with LP. HLA A3, HLA A5, HLA Bw35and HLA DR1have been found in close association with LP. HLA haplotypes reported in familial cases are HLA B7, -Aw19 ,-B18 and –Cw8. HLA B8 is more strongly associated with oral lichen planus and HLA Bw5 with cutaneous LP.[63]

The pathogenesis of LP can be explained in the following steps;

Antigen recognition:[55]

The nature of the antigen which stimulates the pathogenetic process is unknown. The antigens may be a contact allergen, drug , viral or infectious agents or an unidentified immunogenic target.

The most import contact allergen proposed as an antigen is mercury in dental amalgam. Other metals which act as contact sensitiser are gold, palladium and beryllium. Viral etiologies in lichen planus are HSV2 , HIV, HCV and HPV. Bacterial infections like syphilis, helicobacter pylori are also suggested in the etiology of LP.

Cytotoxic lymphocyte activation:[55]

Antigen recognition will lead to activation,clonal expansion and proliferation of $CD8^+$ T cells. As a result , cytokines and chemokines like interleukins , interferon gamma, TNF α and TGF β1 are secreted. Both pro and anti inflamatory cytokines are secreted simultaneously. Clinical behaviour is determined by the balance between lymphocytic activation and downregulation. The lymphocytes interacts with basement membrane resulting in apoptosis, basement membrane disruption , reduplication and subepidermal clefting.

Keratinocyte apoptosis [55]

The activated cytotoxic T cells will trigger apoptosis of the keratinocytes. TNFα and granzyme B which are secreted by T cell will lead to keratinocyte apoptosis. Binding of CD95L to CD95 on keratinocyte will also trigger the process.Matrix metalloproteinase secreted by T cells will block the cell survival signals to keratinocyte and induces apoptosis, thus disrupting the epithelial basement membrane.

CLINICAL FEATURES

Classical LP lesions appear as purple or violaceous flat topped polygonal papules which variesin size from pin point to 1.5cm. The typical site of involvement in both adult and children are volar aspect of wrists, around ankles and lumbar region,often bilateral and symmetrical.Koebnerisation may be positive in lichen planus.In a report of fifty cases of lichen planus 28% of the children showed koebner phenomenon.[64]Wickhams striae may be evident over the papules or plaques which appear as white reticular network.LP lesions are generally mildly to intensly pruritic,but in children the lesions are often non pruritic. Hypertrophic lesions will have severe itching.

Childhood onset LP will be similar in presentation to classical LP, with lesions typically affecting lowerlimbs. The course of LP in children

is similar to the adult course with majority of disease resolving within 1 year. [65]

Variants of LP are hypertrophic,follicular, actinic, linear, LP pigmentosus, annular,atrophic,LP of palms and soles. Mucosal,nail and scalp involvement can be seen in LP.

Linear LP is more common in childhood.[65]It may present as a few papules arranged linearly in a few centimetres or can extend to involve a whole limb. Multiple linear LP lesions following Blaschko lines has been reported in some individuals. Multiple linear LP was documented in a patient with human immunodeficiency virus (HIV) infection.Hypertrophic LP is a chronic variant of LP which presents as intensely pruritic thick verrucous hyperkeratotic plaques which are distributed bilaterally symmetrical over the shins.This variant is frequently reported in children.[65]

Annular LP is an uncommon clinical variant of LP which is characterized by a ring like cluster of purple and polygonal flat topped papules with a central normal or atrophic skin. Actinic LP presents as annular or discoid patches with deeply pigmented centre and peripheral halo of hypopigmentation. Actinic LP generally occurs in children or young adults in tropical countries.

Follicular LP is rare in childhood.[65]Clinically it presents as perifollicular scaling,erythema,hyperkeratotic papules and loss of follicular orifices. Most common site of involvement on the scalp is parietal and vertex areas.

Lichen planus pemphigoides is a rare autoimmune blistering disease in which typical lichen planus lesions evolve into bullous lesions with a mean lag time of 8 weeks. The most common site involved areextremities and about half of the affected children will have palmoplantar lesions.

Nail involvement in children is relatively rare as compared to adults. Finger nails are involved more than that of toe nails.The nail changes include exaggeration of the longitudinal lines ,linear depressions, loss of lustre,thinning of nail plate,splitting or nicking of nail margin, atrophy,pterygium,onycholysis , subungual hyperkeratosis. Violaceous lines or papules in the nail bed may occasionally seen through the nail plate.LP has been shown to cause idiopathic atrophy of nails in children.Rarely Lp can cause severe ulceration of soles which can lead to complete loss of toe nails.[66]

Mucosal involvement in LP in children is less common than adults. Most common sites involvedare the buccal mucosa and tongue. Mucous

membrane of genitalia,anus and larynx can also be involved. Rare cases of tympanic membrane and oesophagus involvement have also been reported[67]. The morphological type of mucosal LP are reticular, erosive, atrophic,bullous and plaque type. Reticular patches and white plaques on the buccal and gingival mucosa and violaceous papules over the lips are the most common types described. Ulcerated variant is very rare in children.

Histopathology: [5]

The papules or plaques of lichen planus will show compact orthokeratosis,wedge shaped hypergranulosis,irregular acanthosis giving a saw toothed appearance of rete ridges,vacoular alteration of basal layer and band like dermal lymphocytic infiltrate close to the epidermis. Lower epidermis will show necrotic keratinocytes, which are also called as colloid orhyaline or cytoid or civatte bodies. They will have a homogenous eosinophilic appearance measuring an average of 20um in diameter.The dermal infiltrate is composed almost entirely of lymphocytes intermingled with macrophages. A few eosinophils or plasma cells may be there. As a result of damage to basal cells, the dermis will show pigmentary incontinence and melanophages. An artifactual separation of epidermis and dermis termed Max joseph space is seen occasionally.

Differential diagnosis[58,68]

Table 2: Differential diagnosis of lichenplanus in children

Type of lichen planus	**Differential diagnosis**
Classical lichen planus	Psoriasis Lichen simplex chronicus Drug eruption
HypertrophicLichen Planus	Lichen simplex chronicus Prurigo nodularis Lichen amyloidosis Lichenoid psoriasis
Follicular	Darier's Disease Keratosis pilaris Lichen scrofulosorum Lichen nitidus
Linear	Lichen Striatus Linear psoriasis Inflammatory linear verrucous epidermal nevus
Actinic	Annular psoriasis Granuloma annulare Tinea corporis
Atrophic	Lichen sclerosus et atrophicus
Guttate	Guttate psoriasis
Lichen planus of oral mucosa	Candidiasis Leukoplakia Contact dermatitis to dental amalgam Healing oral erosions of Pemphigus vulgaris
Lichen planus of palms and soles	Psoriasis Focal palmoplantar keratoderma

Treatment

Idiopathic LP oftenhas a self resolving natural course. Some patients will have remission and exacerbation of the disease.

In paediatric population ,first line of therapy in cutaneous or oral LP is potent and super potent topical steroids with or without antihistamines. Topical steroids will help in reducing pruritis and flatten skin lesions. Topical steroids have to be continued for several weeks to achieve these effects.But there is risk of atrophy in long term use.

Topical calcineurin inhibitors such as tacrolimus and pimecrolimus can be tried in those who require prolonged therapy.[68]

In oral LP, avoidance of triggers and maintenance of good oral hygiene should be observed. Topical steroids in an adhesive base is needed for oral LP for prolonged period. Other treatment options for oral LP in children are topical tretinoin , isotretinoin, tacrolimus and pimecrolimus.[68]

Oral corticosteroids should be considered in severe generalised LP of childrenor if topical treatment fails to relieve the symptoms. Oral corticosteroids should be given in a dose of 0.5-1mg/kg for 2-6 weeks with gradual tapering thereafter. Other treatment options in children are

dapsone ,griseofulvin, metronidazole,oral retinoids,cyclosporine and thalidomide. NBUVB and PUVA therapy is also used in generalise LP.

PROGNOSIS[58,67]

Skin lesions in LP will most often subside within 9-18 months. Occasionally lichen planus may persist for years. About 10-20% of patients sufferfrom one or more recurrences of their skin lesions. Hypertrophic LP and oral LP will take long time to resolve. The lesion may tend to flatten or resolvewith hyperpigmentation, which may persist for months or years.

LP lesions may recur in about 15-20% of the patients. Recurrences are more common in generalised LP. Malignant transformation is seen in less than 1 percent of persistent oral mucous lesions on long term follow up.

PITYRIASIS RUBRA PILARIS

Pityriasis rubra pilaris (PRP) was first described in 1856 by Devergie and was named by Besnier in 1889.[69] Pityriasis rubra pilaris (PRP) is a group of keratinisation disorders that have in common, circumscribed follicular keratoses, branny scale and an orange-red erythema surrounded by normal skin, and palmoplantar keratoderma .[70]

Epidemiology

In India , incidence of PRP is 1 in 50,000 as compared to 1 in 5000 in Great Britain ,which makes clear that there is a racial variation.[71,72] There is a bimodal age of onset without sex predilection. The most common type of PRP is adult onset classical type.

Aetiopathogenesis:

The aetiology and pathogenesis of PRP are still poorly understood. It represents a disorder of keratinisation. Most childhood cases of PRP are acquired without any family history,but an autosomaldominant inheritance is also reported.[73] Clinically and histologically, acquired and hereditary forms are indistinguishable, but autosomal dominant form will have less severe onset from birth to early childhood.

Vitamin A deficiency, infection and trauma are the various postulated causes of PRP. [74,75,76]Studies have shown that there is an association between PRP and decreased retinol binding protein. A study conducted on PRP in children by Allison D S et al showed that 27% of patients had preceeding upper respiratory tract infection and 10% had preceeding trauma.[76] Epidermal thymidine labelling is increased from an average normal 3% to 27%.

PRP may be a result of dysregulation of immune system and abnormal response to various antigenic triggers .[77] Bacterial or viral infection beforethe eruption are commonly noticed in juvenile PRP. PRP is also associated with rheumatism and malignancy.[78] Photoaggravation is also a suggested triggering factor.[79]

Down's syndrome, osteoporosis,arthropathy , coeliac sprue, protein losing enteropathy,hypothyroidism,hyperparathyroidism and myasthenia gravis are rare associations of PRP.[80]

Griffiths classification:[71]

Based on clinical and morphological appearance,Griffiths proposed a classification of five types

Type 1: Adult onset, classical

Type II: Adult onset, atypical

Type III: Juvenile onset, classical

Type IV: juvenile onset,circumscribed

Type V: Juvenile onset, atypical

Juvenile onset PRP is discussed in detail

Type III (Juvenile onset, classical)[70]

The age of presentation of type III PRP is 5-18 years. Type III PRP accounts about 10% of the total PRP cases. Type III resembles type 1 PRP clinically, but type III have a better prognosis than type 1.It starts as orange red macule on head, neck or upper trunk with fine scales which later spread cephalocaudally to involve whole body. Later, perifollicular erythematous papules with central acuminate keratinous plug develops. A characteristic feature of widespread PRP is "islands of sparing". Palms and soles will show thick yellow keratoderma which is referred to as "PRP sandal". Scalp may be covered with pityriasiform scales. Ectropion may be seen. Nails may be normal or yellow,thickened and curved.

Pruritis may be seen in the erupting stage. Sometimes burning sensation will be there. The symptoms will improve as the disease stabilises.Rarely type III can get transformed to typeIV.

Type IV: Juvenile onset, circumscribed[70]

The age of onset of type IV PRP is usually less than 12years.It presents as asymptomatic,erythematous grouped follicular papules over extensor aspect of knees and elbows. A few erythematous scaly macules or follicular keratotic plugs can be seen in the trunk. Marked Palmoplantar keratoderma can be seen in some cases.the lesions are

nonpruritic. Scalp is spared in this type. These lesions does not progress to type I and type III.

Type V : Juvenile onset ,atypical[70]

This type usually present since birth or in the first few years of life. It is characterised by follicular hyperkeratosis with only minimal erythema. Scleroderma like changes of the digits have also been reported in many patients. This type usually persists throughout life.Majority of familial PRP cases are of type V . It presents as follicular ichthyoses and erythrokeratoderma.

HIV associated

PRP like eruptions have been documented in all age groups with HIV infection. it is usually diagnosed in known HIV patients. But sometimes the eruption can be a sign of HIV infection.

This type is associated with acne conglobata , cystic acne or hidradenitis suppurativa. It is usually refractory to therapy but early intiation of HAART may be effective.[81]

Histopathology[5]

A fully developed lesion shows alternating orthokeratosis and parakeratosis oriented in both vertical and horizontal directions,slight spongiosis,focal or confluent hypergranulosis,acanthosis with broad and short rete ridges,thick suprapapillary plates in the epidermis. Dermis will how mildly dilated vessels and superficial perivascular lymphocytic infiltrate.

Differential diagnosis[81]

Psoriasis, lamellarichthyosis , keratosis pilaris, erythrokeratoderma variabilis

Treatment

Mainstay of treatment for PRP is topical treatment.General measures like use of gentle bathing soap,liberal use of emollients should be advised. Topical steroids, keratolytics, calcipotriene and retinoids are commonly used.

Oral retinoids like acitretin(0.5-1mg/kg) and isotretinoin (0.75-1.5mg/kg) can be used in severe disabling disease.Methotrexate and cyclosporine alsohave been reported to be effective.[80]

Prognosis

Type III PRP often resolves with in an average period of 1-2 years. Couse of type IV PRP is uncertain, but some cases clear in the late teens. Type V PRP have a chronic course. It usually resolves with retinoids but relapses on stopping the drug.[81]

LICHEN STRIATUS

Lichen striatus is a self limiting disease of unknown origin characterized by inflammatory papular eruptions in a linear distribution or Blaschkoid distribution. [82]

Epidemiology

It is more common in children between the age of 5-15years,with a female preponderance. [83]About two third of the cases will have lesions over the limbs. Pruritis is moderate and occurs in about 10% of the cases.

Aetiology

Definitive aetiology of lichen striatus is unknown. Lichen striatus is an acquired disease. Trauma, ultraviolet light and contact dermatitis have been implicated. Seasonal variation was reported in an Australian study, and was found to be more frequent in the spring season.[84]

Studies on lichen striatus of children have shown a high association with atopic dermatitis.Various studies have shown that 60-80%of children with lichen striatus have a history of atopy.[85]The abnormal immune status associated with atopy may contribute to the development of lichen striatus.

Viral infection has also been suggested as a trigerring factor. Lichen striatus have shown an increased amount of IL-1β,whichsupports an in situ inflammation driven process,trigerred by infectious pathogens.[86]

An epigenetic mechanism has been suggested in the occurrence of familial cases of lichen striatus. Eventhough familial cases are rare, cases of simultaneous occurrence in siblings was reported . This also suggests a common environmental trigger.[87]

Congenital presence of an abnormal skin clone due to a postzygotic mutation has also been proposed as a possible explanation for LS .[88] The cells bearing the mutation will be quiescent. A trigger, most commonly a viral infection will cause this aberrant clone to express a novel antigen, resulting in the disease manifestations..

Clinical features

Lichen striatus initially presents as asymptomatic, discrete tiny erythematous and lichenoid papules which later coalesce to form a irregular linear band. The surface of the papules may have an associated scaling. Common sites involved are extremities and trunk. It usually presents as a unilateral single lesion,although rare cases of multiple and bilateral involvement are reported.[89.] Facial involvement is also seen.

The lesion progress for 2to 3weeks and resolves within a period of 3-12 monthsleaving a postinflammatory hypopigmentation or hyperpigmentation.

Nail involvement can be seen, frequently on the fingers, especially the thumb. Longitudinal ridging, splitting and thinning with onychodystrophy are common nail manifestations . Nail lichen striatus may preceed the onset of cutaneous lesions.[90]

Histopathology[5]

Epidermal changes include focal parakeratosis, spongiosis, intracellular edema often associated with exocytosis. Dermis will show superficial perivascular inflammatory infiltrate of lymphocytes with histiocytes. Focally in the papillary dermis the infiltrate may have a band like distribution with extension into the the lower portion of epidermis.

The corresponding regions of epidermis will show vacuolar alteration of the basal layer and necrotic keratinocytes with melanophages in the papillary dermis.

Differential diagnosis[91]

Linear psoriasis, linear Darier's disease, linear lichen planus,linear porokeratosis and other linear nevi, are to be considered in the differential diagnosis. ILVEN is the most important differential diagnosis for lichen striatus,but it will have an earlier onset and will persist indefinitely.

Hypomelanosis of Ito and vitiligo are the differential diagnosis in the hypopigmented stage.

Treatment

Benign and self limiting nature of the disease should be explained to the parents. Intralesional steroid can be used in persistent case. If nails are affected, potent topical steroids can be used. Topical tacrolimus ointment will hasten the resolution of lesion.

Prognosis

Lichen striatus is a benign condition. The mean duration of the lesion is 6-9 months. Relapses can occur within 4 years. The lesions may heal with post inflammatory hypopigmentation.[91]. Nail matrix lichen

striatus will not cause permanent damage and will resolve spontaneously.[92]

SEBORRHEIC DERMATITIS

Seborrhoeic dermatitis is a common chronic papulosquamous dermatosis,which is characterised by self limiting erythematous scaly eruptions that occurs primarly over the face, postauricular, presternal and intertrigenous areas. The term seborrheic dermatitis was first coined by Unna in 1887.[93]

Epidemiology

The exact incidence of seborrheic dermatitis is unknown, but the disorder is quite common. Seborrheic dermatitis showed an incidence of 1.6% among children , in a study conducted in India[94]. In United States it affects about 3-5% of general population.[95] Seborrheic dermatitis have bimodal peak, one in infancy and second around fourth to seventh decade. Males are affected more frequently than females. Seborrheic dermatitis is found in upto 85% of patients with HIV infection.

Aetiopathogenesis

The aetiology of seborrheic dermatitis is not well understood. However many factors are implicated in the pathogenesis of seborrheic dermatitis.

A positive correlation with sebum and sebaceous have been postulated. It is evidenced by predilection for areas of high sebaceous density and correlation of activity with increased hormonal levels during first year of life and adolescents.

Theories have suggested that Malassezia furfur plays an important role in the etiology of seborrheic dermatitis. Ruiz maldonao et al detected Malassezia furfur in 73% of patients with seborrheic dermatitis.[96]

Studies done in infantile seborrheic dermatitis have demonstrated abnormality with regard to essential fatty acids. A transient impairment in delta-6-desaturase was detected in these children.[96]

Although a family history of seborrhea is quite often reported , there is no conclusive evidence that heredity plays a role.

Seborrheic dermatitis is associated with parkinsonism and neurological disorders where sebum production is increased. It is also associated with some medical conditions like malabsorbtion, epilepsy, obesity , alcoholic pancreatitis.[97,98] Seborrheic dermatitis is an established marker of HIV infection and also seen in local cutaneous immunosuppression.[99]

Clinical features

The various clinical patterns of seborrheic dermatitis are shown in Table 3.

Table 3. CLINICAL PATTERNS OF SEBORRHEIC DERMATITIS[100]

INFANTILE	**SCALP** (CRADLE CAP) **TRUNK** **LEINER'S DISEASE** • .Non-familial • Familial C5 dysfunction
ADULT	**SCALP** • Dandruff • Inflammatory **FACE** **TRUNK** • Petaloid • Pityriasiform • Flexural • Eczematous plaques • Follicular **GENERALISED**

Infantile seborrheic dermatitis appears between second and third week of life and peak incidence at the age of 3 months. Infantile seborrheic dermatitis in scalp manifests as cradle cap, i.e. yellowish or

whitish greasy thick adherent , frequently confluent scales occurring mostly over vertex or frontal regions. Pruritis is usually absent or may be mild.

On the face ,it appear as small round scaly erythematous areas mainly over forehead,eyebrows, retroauricular region and nasolabial folds. The lesions may also develop over neck, axillae,umbilicus,inguinal and intergluteal folds. In the intertriginous areas the lesions will become macerated and eroded. So scaling will be prominent only at the peripheries. These areas may have secondary candidal or bacterial infection evidenced by erythematous papules or pustule.Blepharitis is also a common manifestation of seborrheic dermatitis in infants.[100]

Occasionally, seborrhoeic dermatitis may become generalized, resulting in erythroderma in infants.Leiners disease is a severe type of seborrheic dermatitis which is associated with diarrhoea, failure to thrive and erythroderma.A possibility of immunodeficiency should be suspected when it is severe, generalised and exfoliative,in adults.

Histopathology: [101]

The histopathological features of seborrheic dermatitis vary according to different clinical stages.

In acute and chronic stage, there will be orthokeratosis, parakeratosis follicular plugging, spongiosis, psoriasiform hyperplasia. Dermis will show superficial perivascular infiltrate of histiocytes and lymphocytes.In chronic stage superficial plexus of dermis will show markedly dilated capillaries and venules,

Differential diagnosis

Atopic dermatitis, psoriasis, intertrigo, Langerhans'cell histiocytosis, multiple carboxylase deficiency,zinc deficiency and tinea capitis.

Treatment

Infantile seborrheic dermatitis can be best managed by frequent shampooing , with a gentle 'no tears' shampoo.Thick adherent scale can be removed by application of baby oil or mineral oil followed by gentle scraping with a toothbrush and shampooing.If there is severe inflammation, a low potent topical steroid .For non scalp areas a low potent topical steroid or antifungal will be usually effective.Topical anti bacterial and candicidal will be useful if superadded infection is there

Blepharitis can be managed by warm compressess, non irritating shampoo or mechanical removal of scales when necessary.

Prognosis

Infantile seborrheic dermatitis has an excellent prognosis. The lesions will clear within 3-4 weeks in some patients. Most of the patients will have spontaneous clearance of the lesion by the age of 8-12 months

LICHEN NITIDUS

Lichen nitidus was first described by Pinkus in 1907.[103]In Latin, the word nitidus means "shiny" or "glistening". Lichen nitidus is a self limiting chronic inflammatory papulosquamous disorder defined more often in children and young adults.

Epidemiology

Epidemiological data of lichen nitidus is not well documented. Lichen nitidus occurs more common in blacks than Caucasians. Children andyoung adults are affected more than that of elderly. Lichen nitidus is more common in males than females. A 25 year survey of skin disease in African-Americans reported an incidence of 3.4 cases/10,000 population for lichen nitidus..[104]

Aetiopathogenesis

The exact aetiology of lichen nitidus is unknown. LN is sporadic in origin but familial cases have been reported. In the earlier days it was thought to be a tuberculoid reaction. Lichen nitidus was once thought to

be a variant of lichen planus , as the two diseases coexisted in the same individual and also due to the development of lichen planus following generalised lichen nitidus.

Recently postulated mechanism for the pathogenesis of lichen nitidus is that an allergen may intiate the pathological process. Allergen will cause epidermal and dermal antigen presenting cells to activate cell mediated response , initiates accumulation of lymphocytes and form discrete inflammatory papules.[103]

Clinical features

The lesions of lichen nitidus are characterised by multiple grouped and discrete pinpoint to pinhead sized ,fleshcoloured to slight pink flat,shiny papules,commonly distributed over forearm, penis, chest and abdomen. The papules will be hypopigmented in blacks. Scaling may be minimal and can be elicited by rubbing the surface of the papules. Koebnerisation and linear arrangement can be seen.

Rare forms of lichen nitidus are generalised,linear,actinic, vesicular, hemorrhagic, perforating, purpuric and spinous follicular. Lichen nitidus may rarely affect mucous membrane,palms,soles and nails. In children rare variants like generalised lichen nitidus, perforating type and actinic lichen nitidus are reported. [103]

Atopic dermatitis , Down's syndrome and juvenile chronic arthritis have been reported to be associated with lichen nitidus in children.[105,106,107] In adults, lichen nitidus are reported to be associated with Crohn's disease, erythema nodosum and MEN2e.

Histopathology[5]

Epidermis will be flattened wth focal parakeratosis, diminished granular layer, vacoular alteration of basal layer and focal subepidermal clefting. . Colloid bodies are seen rarely. Dermis will show a well circumscribed mixed cell granulomatous infiltrate . At each lateral margin of the infiltrate rete ridges tend to bend inward and seem to clutch the infiltrate in a manner of a " claw clutching a ball". The infiltrate is composed of lymphocytes,monocytes and a few multinucleated epitheloid histiocytes. Transepidermal elimination of the infiltrate through the thinned epidermis can occur. Capillary wall degeneration and extravasation of RBCs are seen in purpuric and hemorrhagic lesions.

Differential diagnosis[65]

Lichen planus, plane wart, keratosis pilaris ,Darier's disease, lichen spinulosus, lichen scrofulosorum, id reaction are considered as differential diagnosis.

Prognosis

LNis a chronic self limiting disease with an average duration of 1 year. New lesions may continue to develop as older lesions resolve. Lesions heal without scar formation or pigmentary abnormalities. The eruption may persist rarely.

Treatment

Medical treatment is considered when the patient is symptomatic or when the lesions are generalised and persistent. Topical treatment includes mid to high potent steroids and tacrolimus. Immunotherapy with dinitrochlorobenzene has been used. Studies have shown that oral isotretinoin is effective in generalised LN and acitretin in palmoplantar involvement.

In refractory cases NBUVB, PUVA, astemizole, enoxaparin and etretinate have been used. Systemic retinoids can cause premature epiphyseal plate closure and stop normal growth in children.

PARAPSORIASIS

The term parapsoriasis was coined by Brocq in 1902 to encompass a group of conditions that clinically resembled psoriasis and seborrhoeic dermatitis.[108] Parapsoriasis is classified into large plaque parapsoriasis and small plaque psoriasis.

Epidemiology

Parapsoriasis is a disease predominantly seen in adults. Males are affected more commonly than females. Small plaque psoriasis shows a definite male predominance of approximately 3-4: 1.[109] Parapsoriasis is rarely seen in children. In a study, about 5% of small plaque psoriasis and 20% of large plaque psoriasis were reported in children.

CLINICAL FEATURES[65,108]

Small plaque parapsoriasis

Chronic superficial scaly dermatitis, persistent superficial dermatitis digitate dermatosis and xanthoerythroderma perstans are the synonyms for small plaque parapsoriasis.

The lesions are usually insidious in onset and asymptomatic. Most common sites involved are trunk and limbs. The typical lesions are hyperpigmented or yellowish red,round or oval, patches with well defined sharp borders of size 1-5cm. The patches will be covered with fine moderately adherent scales. In the digitate variant of small plaque parapsoriasis elongated fingerprint like lesions oriented along the cleavage lines are seen commonly over the flanks. In this type the long axis of the lesions will be more than 5 cm. A variant of parapsoriasis, known as xanthodermia perstants will have a yellowish hue .

Large plaque parapsoriasis

Parakeratosis variegate,retiform parapsoriasis,atrophic parapsoriasis and poikilodermatous parapsoriasis are the synonyms for large plaque parapsoriasis.

Large plaque psoriasis commonly occurs in middle aged males with a peak incidence in fifth decade. Unexposed areas, especially trunk and flexures are the most commonly affected areas. Extremities and breasts in females are other common sites. The lesions will present as persistent ,reddish brown or salmon pink patches or plaques with small and scanty scaling which are more than 5cm, often measuring more than 10cm. The lesion are stable in size but will increase in number. Later atrophic changes along with mottled pigmentation and telangiectasia can occur over patches and plaques. The surface will have wrinkling – "cigarette paper wrinkling."

A rare variant , known as retiform parapsoriasis which refers to a net-like or zebra-like pattern of scaly macules and papules that eventually becomes poikilodermatous in appearance. An icthyosiform variant of large plaque parapsoriasis is also reported in the literature.

Histopathology[5]

Elongated mounds of parakeratosis with collection of plasma above a basket weave cornified layer is a characteristic finding. Epidermis will also show acanthosis,spongiosis and exocytosis of lymphocytes. Papillary dermis will show mild superficial perivascular lymphocytic infiltrate.

Early stages of large plaque parapsoriasis will show similar features as that of small plaque parapsoriasis. Advanced lesions will have epidermal atrophy , marked exocytosis of lymphocytes, and a band-like infiltrate of lymphocytes in the papillary dermis. Dilated vessels and melanophages can be seen in dermis.

Differentialdiagnosis[10,110]

Small plaque parapsoriasis should be differentiated from nummular eczema, psoriasis vulgaris and guttate psoriasis, pityriasis rosea, pityriasis lichenoides chronica and secondary syphilis

Large plaque psoriasis should be differentiated from plaque type psoriasis, tinea corporis, contact dermatitis, poikilodermatous skin conditions such as: Rothmund-Thomson syndrome, Bloom's syndrome, dyskeratosis congenita, systemic lupus erythematosus, dermatomyositis, radiodermatitis ,patch stage of mycosis fungoides.

Treatment

Patients with small plaque parapsoriasis should be reassured about the benign nature of the disease. Small plaque parapsoriasis can be treated with emollients, topical corticosteroids , topical tar products, and narrow band UVB therapy. Treatment response is variable and temporary.

LPP requires aggressive treatment. The goal of treatment is to suppress the disorder to prevent possible progression to overt MF. In large plaque parapsoriasis topical corticosteroids should be used with caution because of atrophic nature of plaques.Emollients ,NBUVB and PUVA therapy can be used.

Repeated examinationwith sequential biopsies of suspicious lesion is needed every 3 months initially and then every 6 months to 1 year for evidence of progression.[110] If the histopathological section shows early stages of early MF , it can be treated with topical nitrogen mustard, bexarotene gel, imiquimod or carmustine. Narrow band UVB and broad band UVB therapy can also be used in such situations.

Prognosis

Small plaque parapsoriasis may persist for years to decades, without treatment. Malignant transformation is rarely reported. Various studies have shown that about 10-35% of large plaque parapsoriasis can

turn to mycosis fungoides. Retiform variant may progress to overt MF in virtually all cases.[110] So children with large plaque parapsoriasis should undergo serial biopsies to rule out malignant transformation.

PITYRIASIS LICHENOIDES

Pityriasis lichenoides is a spectrum of cutaneous eruption of unknown etiology that mainly affects children and young adult. Jadassohn and Neisser first described pityriasis lichenoides in 1894 in two separate reports.[111,112] Juliusberg in 1899 described the chronic form in 1899 and gave the name PLC. Mucha in 1916 and Haberman in 1925 described the acute form of pityriasis lichenoides and Degos in 1966 reported a severe febrile ulceronecrotic subtype now usually called febrile ulceronecrotic Mucha Habermann disease (*FUMHD*)

Gelmetti *et al*.[113] suggested an alternative classification. Based on anatomic distribution, they described three patterns: a diffuse form involving glabrous skin over the whole body surface, a central variety involving the neck, trunk, and proximal extremities, and a peripheral form with an acral distribution. Acral involvement including the face is more common in children.

Epidemiology

The prevalence and incidence of pityriasis lichenoies is not well documented.There is a male predominace of 1.5:1to 3:1.[110]A case of pityriasis lichenoides has been described at birth. PLC is seen in about 37.5% of paediatric patients with pityriasis lichenoides. PLC is three times more common than PLEVA. About 50% of reported cases of febrile ulceronecrotic Mucha Habermann disease have been seen in children.

Aetiopathogenesis

Pityriasis lichenoides is a disorder of unknown etiology. Infectious agents such as Toxoplasma gondii, Epstein-Barr virus, cytomegalovirus,parvovirus B19 and HIV have been associated with some cases.[114] Certain studies have demonstrated T cell clonality in pityriasis lichenoides ,which suggests that it is a benign lympho proliferative disease in which a vigorous host immune response prevents the condition from evolving into lymphoma.

Pityriasis lichenoidess is removed from parapsoriasis group of disorders and now considered as a lymphocytic vasculitis.[115,116]

Epidermis of piryriasis lichenoides lesions have shown a reduction in CDla+ antigen-presenting dendritic (Langerhans) cells in immunohistologic studies. CD8+ cells predominate PLEVA lesions and PLC lesions will have both CD8+ and CD4+ cells. Majority of these Tcells will express cytolytic enzymes(granzyme band TIA-1)and memory proteins(CD45RO) . Presence of IgM, C3 deposition in the dermoepidermal junction and in the dermal vessel walls in the early acute lesions suggest a humoral response. These data suggest that PL may be due to persistent antigen stimulation triggered by an infectious agent resulting in immune complex formation.[113]

Clinical features

PLEVA

PLEVA is characterised by acute onset of firm reddish papules of 2-10mm size. Later the papules will become hemorrhagic, vesicular, necrotic and crusted,leaving scars with hyperpigmentation or hypopigmentation. The eruption may sometimes be associated with fever, headache, malaise and lymphadenopathy. Most common sites affected are anterior aspect of trunk, flexors, and proximal part of extremities. Lesions are usually non pruritic. The disease usually lasts for several weeks to months. After an acute course ,PLEVA can progress to PLC.

PLC

PLC may begin de novo or may evolve from PLEVA. It is characterised by recurrent crops of lichenoid, reddish brown papules which are covered by mica like scales. The papules will involute within 3-6 weeks leaving hyperpigmented macules which gradually fades. The lesions may last from several months to several years with frequent remissions and exacerbations.

In febrile ulceronecrotic Mucha Haberman disease, there will be acute onset of fever with purpuric papules and nodules. Later multiple lesions will coalesce to form diffuse large necrotic ulcers.

Histopathology[5]

In PL, epidermis will show confluent parakeratosis, mild spongiosis, necrotic keratinocytes and basal cell vacoulation.PLEVA will have more pronounced vacoular degeneration with marked exocytosis.Dermis will have perivascular lymphocytic infiltrate which is more deep in PLEVA. Papillary dermis will show melanophages and extravasated RBCs.

Differential diagnosis.[110]

PLC: guttate psoriasis,pityriasis rosea,lichen planus and secondary syphilis.

PLEVA:Arthropod bites, Lymphomatooid papulosis, vasculitis, varicella

FUMHD:Varicella,Lymphomatoid papulosis,Lymphoma

Treatment

Ultraviolet therapy is the most effective therapy for pityriasis lichenoides. Satisfactory outcome is seen with oral antibiotics, particularly azithromycin and erythromycin. Topical corticosteroids and oral antihistamine have a role in reducing the associated pruritis. In persistent cases and ulceronecrotic variants,methotrexate and cyclosporine can be used.

Prognosis and complications

Pityriasis lichenoides have variable outcome. It may recover spontaneously within weeks or may persist and recur for years. The duration of the disease in children correlate better with the clinical distribution of lesions. The longest duration will be seen in peripheral distribution and shortest with diffuse pattern.[117] PLC will persist longer than PLEVA. Malignant transformation is very rare although case of PLC transforming to Tcell lymphoma has been reported.

Most common complication of pityriasis lichenoides is secondary infection. In febrile ulceronecrotic variant the patient may develop arthralgia , gastrointestinal and central nervous system manifestations.

LYMPHOMATOID PAPULOSIS

Lymphomatoid papulosis was first described by Macauly in 1968[118]. It is a benign recurrent self healing dermatosis with histologic features suggestive of lymphoma.

Epidemiology

It is a rare disease with an incidence of about 1.2-1.9 per 1,000,000 in adults.[119] Boys affected with lymphomatoid papulosis have a younger age of onset than girls. The course of lymphomatoid papulosis issimilar in children and adults, including the risk of lymphoid malignancies. But some studies have shown that the disease in children will resolve more spontaneously than in adults.

Clinical features

The lesions typically involve trunk and extremities. There will be recurrent crops of numerous reddish brown papules or vesiculopustules over a period of months to several years. The lesions will characteristically develop central hemorrhagic necrosis and crusting which gradually resolves with hyperpigmentation or hypopigmentation. Occasionally varioliform scars may develop. Rarely large ulcerating nodules, plaques or nonulcerating papules occur. Regional distribution

has been described in children although more generalised spread develops later.

Histopathology[120,121]

There are three histological types described in lymphomatoid papulosis.

Type A (histiocytic type): This consist of scattered or grouped pleomorphic or anaplastic lymphoid cells. Inflammatory cells like eosinophils, neutrophils, histiocytes may be seen in the dermis.**Type B**(Lymphocytic): There will be epidermotrophic infiltrate of small to medium sized lymphoid cells.**Type C**(ALCL- type): There will be nodular infiltrate of large atypical lymphoid cells arranged in sheets. Inflammatory cells may be present.

Differential diagnosis[122]

The differential diagnosis includespityriasis lichenoides,arthropod reaction, pseuolymphoma and anaplastic large cell lymphoma.

Treatment and prognosis[122]

Topical ultrapotent corticosteroids for 2-3 weeks followed by weekly pulse is effective in clearing the lesions. Low dose oral methotrexate(5-10mg/week) will suppress the development of new

lesions. PUVA therapy has also been reported to have beneficial effect on the disease. There may be relapse of the lesions after discontinuation of the therapy. So, in patients with relatively few and non scarring lesions,long term follow up without active treatment should be considered.

Prognosis

About 10-20% of adult patients will develop malignant lymphoma and there is increased risk of developing non lymphoid malignancies. A study conducted in 35 paediatric patients with lymphomatoid papulosis showed that 9% of them developed non Hodgkins lymphoma,[122] which emphasize the need for long term surveillance.

PAPULAR ACRODERMATITIS OF CHILDHOOD

Papular acrodermatitis of childhood was initially described by Gianotti in 1955.[123]Subsequently in 1956 Crosti and Gianotti described the entity and since then the disorder has been commonly as Gianotti Crosti syndrome. They described the disorder as symmetrical distribution of erythematous papular eruption over face, buttocks and extremities of children.

Epidemiology

Gianotti crosti syndrome is distributed world wide. The age group affected are 1-6 years with 90% being affected before the age of 4 years.[123]There is no specific gender or race predisposition. Adult cases are rarely reported. (Pac)

Aetiology [124]

The etiology of PAC has long been suspected to be viral in origin. The common viruses implicated are Ebstein Barr virus, cytomegalovirus, coxsackie virus, parvovirus B19,rotavirus, and HHV 6. A possible association withvaccines has also been suggested. MMR vaccine, DPT,oral polio, hepatitis B vaccines are reported to be associated with PAC.Bacterial pathogens include Mycoplasma pneumoniae, Boreliaburgdorfei, Bartonnella henselae, groupA B-hemolytic streptococcus.

The exact mechanism of pathogenesis of PAC is not well understood. It may be due to a viral antigenemia and circulating immune complexes.

Clinical features[124]

Papular acrodermatitis is seen predominantly in children between the age of 1and 6 years. There will be a history of upper respiratory tract infection in majority of the children. The eruption is characterised by monomorphous,erythematous, edematous papules and papulovesicles distributed symmetrically over face ,buttocks and extensor surface of upper and lower extremities. The papules may coalesce into large erythematous plaques. Lesions are usually non pruritic or mildly pruritic. It may take 8-12 weeks to resolve completely. Post inflammatory hypopigmentation may occasionally persist for several months.

Diagnostic Criteria for Gianotti-Crosti Syndrome[123]

Cuhh ATT proposed a diagnostic criteria for PAC.

A Patient is diagnosed to have Gianotti Crosti syndrome if he/she exhibits all positive clinical features on at least one occasion or clinical encounter, and does not exhibit any negative clinical feature on any occasion or clinical encounter related to rash, and no differential diagnosis is considered more likely than diagnosis of GCS based on clinical judgment, and if lesional biopsy is performed, findings are consistent with GCS

Positive Clinical Features

1. 1.Monomorphous, flat-topped, pink-brown papules or papulovesicles 1–10 mm in diameter.Any 3 or all 4 sites involved: cheeks, buttocks, extensor surfaces of forearms, extensor surfaces of legs.
2. Symmetry
3. Duration of 10 days or more

Negative Clinical Features

1. Extensive truncal lesions
2. Scaly lesions

Differential diagnosis[125]

Acrodermatitis enteropathica, erythema infectiosum, erythema multiforme, hand-foot-and-mouth disease,Henoch-Schönlein purpura, Kawasaki disease, lichen planus, papular purpuric gloves-and-socks syndrome, papular urticarial and scabies.

Histopathology

Epidermis will show parakeratosis, focal spongiosis, and lymphocytes. There is a superficial and mid dermal perivascular infiltrate of lymphocytes and histiocytes. Sometimes the inflammatory infiltrate

may also present as lichenoid infiltrate. Papillary edema and extravasation of RBCs may also be seen.

Treatment and prognosis

Gianotti crosti syndrome is a self limiting disease. Treatment is mainly supportive. In hepatitis B associated PAC, a self resolving anicteric viral hepatitis may evolve after 2 weeks of onset of eruptions

METHODOLOGY

The study was conducted in the outpatient department of dermatology, Coimbatore medical college hospital between the time period of August 2013 and July 2014. This is a clinical study of papaulosquamous disorders in children less than 12years who were clinically diagnosed to have papulosquamous disorder as per ICD 10 classification .Ethical committee clearance was obtained. A written informed consent was obtained from the parents.

Inclusion criteria :

All children less than 12 years who presented to the skin OPD with papulosquamous morphology of skin lesions.

Exclusion criteria:

Children whose parents did not give consent for the study.

One hundred and five children were clinically diagnosed to have papulosquamous disorder during the study period. A detailed history including age of onset, duration of the disease, progression and associated symptoms were obtained from the patients and their parents. Relevant family history and history of drug intake prior to the onset of disease were recorded.General examination and systemic examination were done.

Dermatological examination was done. Morphology, distribution, characters of scales and any special features were recorded. Hair, nails, oral and genital mucosa were examined in detail.

All routine investigations including haemoglobin, total blood count, differential count, erythrocyte sedimentation rate were done for each patient.

Special investigations like KOH mount,VDRL and biopsy were done for relevant patients

Children from 0-12 years were divided into four groups. The findings were recorded in the profoma and tabulated in the masterchart. The results were analysed and discussed in detail.

RESULTS

One hundred and five patients under the age of 12 years with papulosquamous disorders attending the outpatient department of Dermatology,venereology and leperology , Coimbatore medical college hospital formed the study material.The duration of study was from August 2013 to July 2014.

During the study period of 1 year, 69445 patients attended the outpatient department, of which 12835 were children

Table 4 No of total patients and pediatric patients attended the OPD

Total number of patients attendedthe skin opd	Total number of paediatric patients attended skin opd	Percentage of paediatric patients attended skin opd
69445	12835	18.48%

Out of the paediatric patients, 105 patients had papulosquamous disorders.

Table 5 Percentage of pediatric patients with papulosquamous disorder

Total number of paediatric patients attening skin opd	Total number of patients with papulosquamous disorders	Percentage of papulosquamous disorder in children
12835	105	0.81%

SEX RATIO

Total number of patients included in the study were 105, out of which 55(52%) were male and 50(48%) were female.

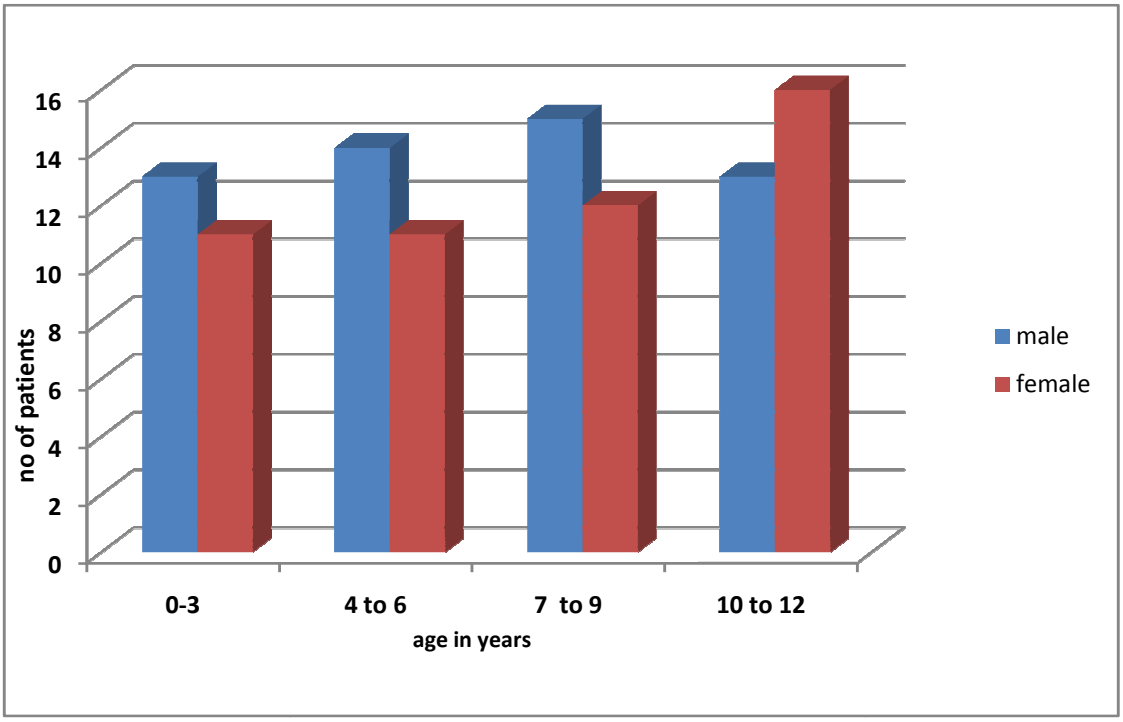
The male to female ratio in the study was 1.1:1

AGE AND SEX DISTRIBUTION

Table 6Age sex distribution of children with papulosquamous disorders

Age(years)	Male	Female	Total
0-3	13 (23.64%)	11 (22%)	24 (22.86%)
4-6	14 (25.45%)	11 (22%)	25 (23.81%)
7-9	15 (27.27%)	12 (24%)	27 (25.71%)
10-12	13 (23.64%)	16 (32%)	29 (27.62%)
Total	**55 (100%)**	**50 (100%)**	**105 (100.00%)**

CHART: 1. Age sex distribution of children with papulosquamous disorders



The maximum number of patients were in the age group 7 to 9 years and least number of patients were seen in 0-3 years age group.

The youngest patient was of age 1 month and oldest was 11 years.

The highest percentage of male patients were in the age group 7 to 9(27.27%) years and least were in the age group 0-3 years and 10-12 years(23.64%)

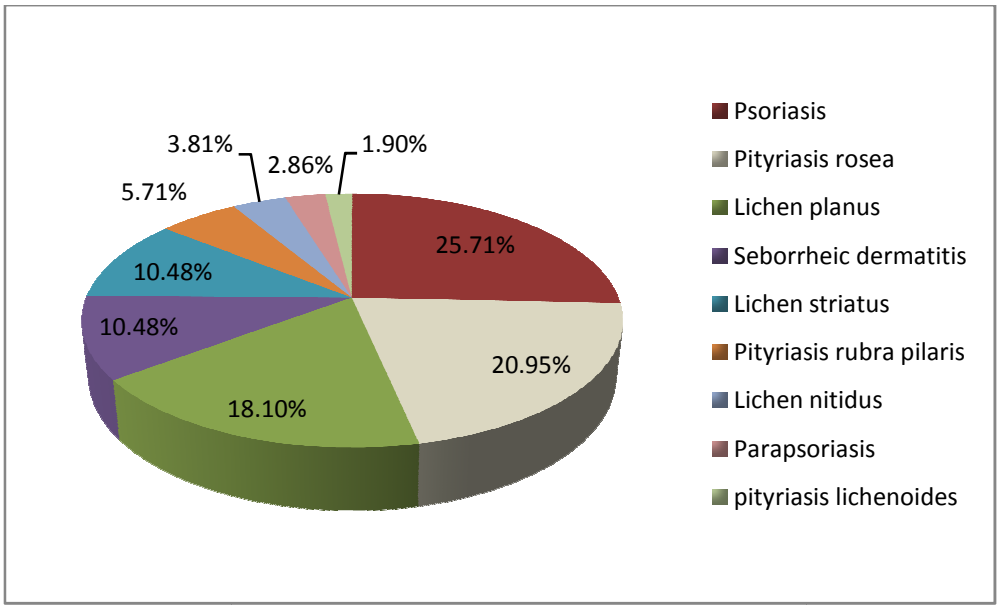
The highest percentage of female patients were in the age group 10-12(27.62%) years and least were in the age group 0-3years and 4-6 years(22.86%).

The various papulosquamous disorders recorded in the decreasing order of frequency were as follows

Table 7Frequency of various papulosquamous disorders

Sl no	Papulosquamous disorder	Number of cases	Percentage
1	Psoriasis	27	25.71
2	Pityriasis rosea	22	20.95
3	Lichen planus	19	18.09
4	Seborrheic dermatitis	11	10.47
5	Lichen striatus	11	10.47
6	Pityriasis rubra pilaris	6	5.71
7	Lichen nitidus	4	3.8
8	Parapsoriasis	3	2.85
9	Pityriasis lichenoides	2	1.9
	TOTAL	**105**	**100**

CHART 2: Frequency of papulosquamous disorders



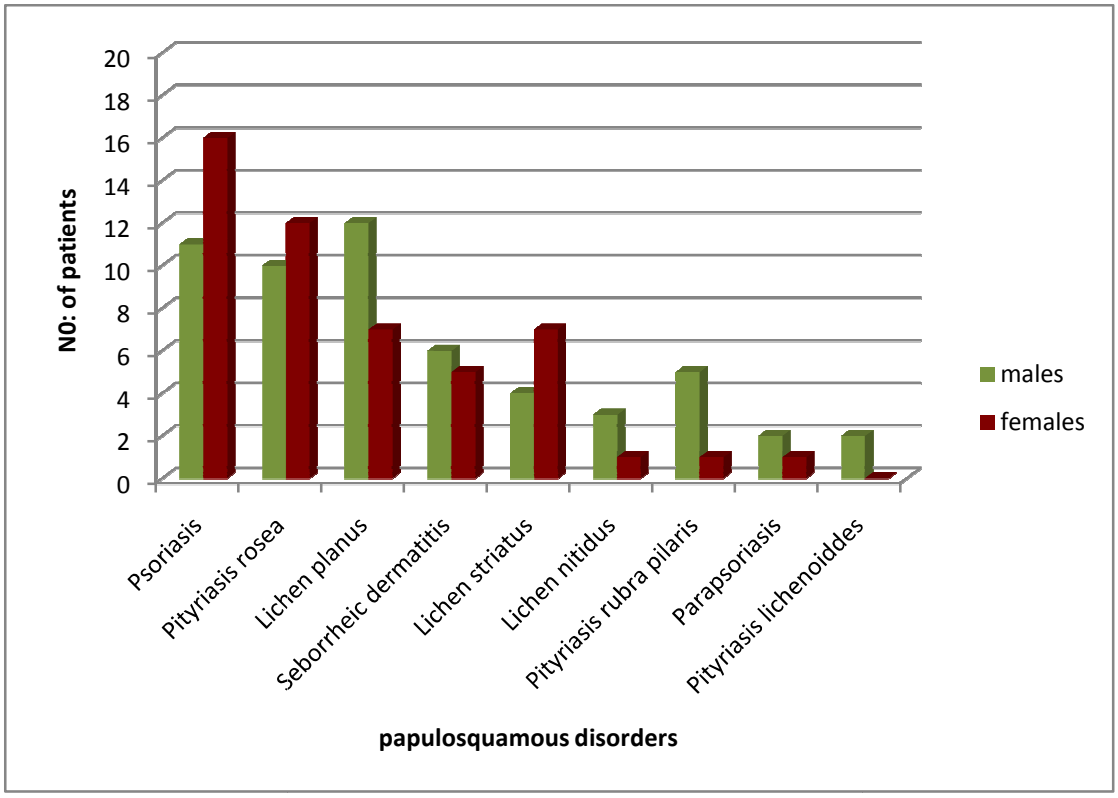
Psoriasis was the most common papulosquamous disorder with 27 cases (25.71%) ,followed by pityriasis rosea with 22 cases(20.95%) and pityriasis lichenoides was the least common with 2 cases(1.9%).

The distribution of papulosquamous disorders in males and females in the study is as follows.

Table 8Frequency of papulosquamous disorders in males and females

Sl no	Papulosquamous disorder	Males	Females
1	Psoriasis	11(20%)	16(36%)
2	Pityriasis rosea	10(18.18)%	12(24%)
3	Lichen planus	12(21.82)%	7(14%)
4	Seborrheic dermatitis	6(10.91)%	5(10%)
5	Lichen striatus	6(10.91)%	5(10%)
6	Lichen nitidus	3(5.45)%	1(2%)
7	Pityriasis rubra pilaris	5(9.09)%	1(2%)
8	Parapsoriasis	2(3.64)%	1(2%)
9	Pityriasis lichenoiddes	2(3.64)%	0
	TOTAL	**55(100%)**	**50(100%)**

Chart 3 Distribution of papulosquamous disorders in males and females



In males the most common papulosquamous disorder was lichen planus (21.82%), followed by psoriasis (20%). Parapsoriasis and pityriasis lichenoides (3.64%) were the least common diseases recorded.

Psoriasis(36%) was the most common papulosquamous disorder seen in females followed by pityriasis rosea(24%). Pityriasis rubra pilaris , parapsoriasis and lichen nitidus were seen in 2% of female patients.

Seasonal variation of papulosquamous disorders is shown in the below table:

Table 9Seasonal variation of papulosquamous disorders

Disease	Winter (Dec-Feb)	Summer (Mar-May)	Monsoon (June-Nov)
Psoriasis	15	5	7
Pityriasis rosea	11	5	6
Lichen planus	5	9	5
Seborrheic dermatitis	3	6	2
Lichen striatus	4	5	2
Pityriasis rubra pilaris	1	3	2
Lichen nitidus	1	1	2
Parapsoriasis	1	0	2
Pityriasis lichenoides	1	1	0
Total	**42**	**35**	**28**
Percentage	**40%**	**33.33%**	**26.67%**

Incidence of papulosquamous disorder was high during the winter season(40%). 33.33% of patients visited during the summer season and 26,67% of the patients visited during monsoon season.

The incidence of psoriasis(55.56%) and pityriasis rosea (50%) were more during the winter season.

PSORIASIS

In the study, out of 12835 patients ,psoriasis was reprted in 27 patients. The prevalence of childhood psoriasis in the study is 0.21%. The prevalence of childhood psoriasis among papulosquamous disorder is 25.71%.

There were 11 boys and 16 girls with a male to female ratio of 1:1.45

AGE SEX DISTRIBUTION OF PATIENTS WITH PSORIASIS

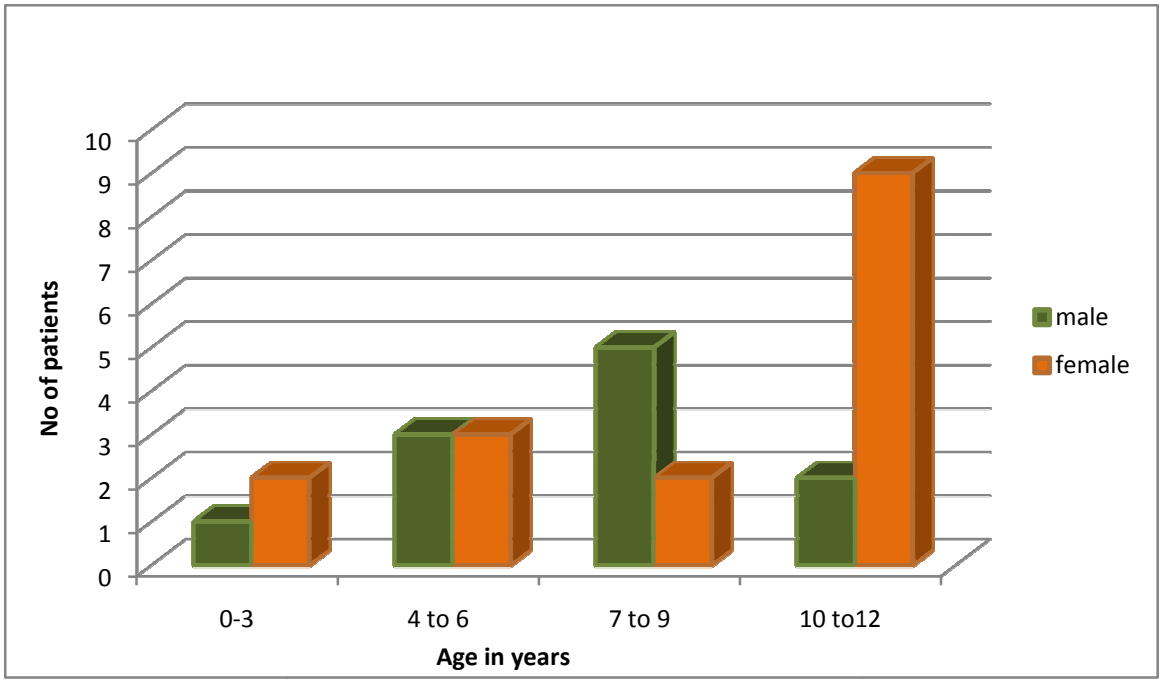
The table shows the age sex distribution of psoriasis

Table 10: Age sex distribution of patients with psoriasis

Age(in years)	Male	Female	Total
0-3	1(9.09%)	2(12.50%)	3(11.11%)
4-6	3(27.27%)	3(18.75%)	6(22.22%)
7-9	5(45.45%)	2(12.50%)	7(25.93%)
10-12	2(18.18%)	9(56.25%)	11(40.74%)
Total	11(100%)	16(100%)	27(100%)

CHART 5:

Age Sex Distribution of Patients with Psoriasis



The youngest age of psoriasis in the study was 6 months. Majority of the children belonged to the age group 10-12 year(40.74%). Mean age of onset of psoriasis in our study was 6.83 years.

Majority of the male patients with psoriasis belonged to the age group 7 to 9 years and female patients belonged to the age group 10-12 years. Mean age of onset of psoriasis in boys were 6 years and 7.4 years in girls.

A patient with guttate psoriasis and 2 patients with chronic plaque psoriasis gave history of fever and sore throat prior to the onset of lesions.

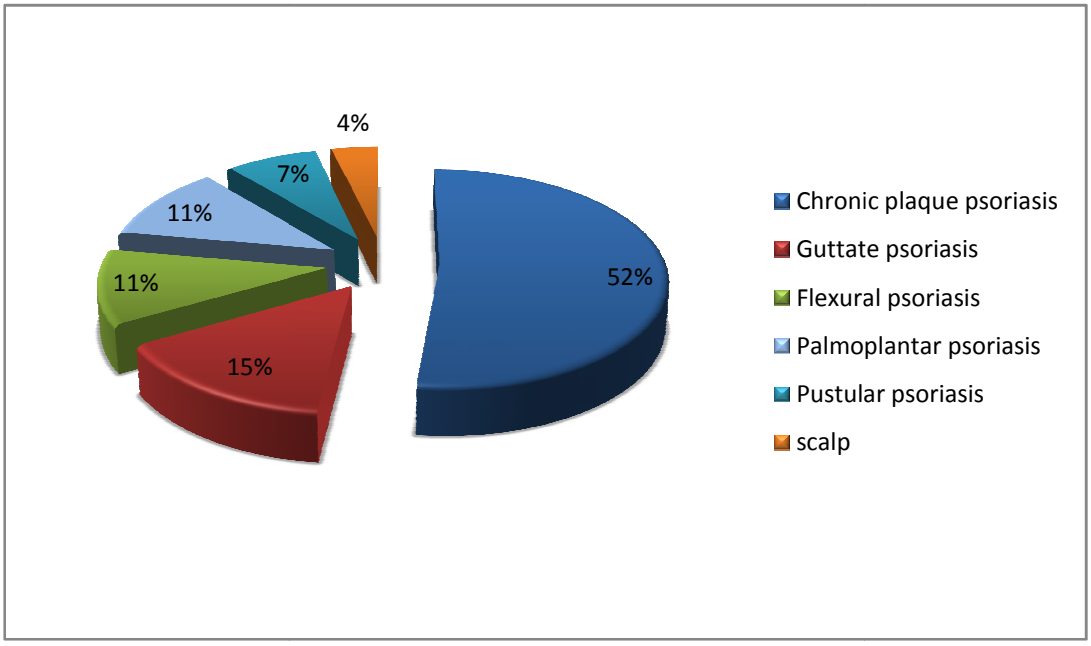
A positive family history was present in 2(7.41%) patients, one patient gave history of psoriasis in the sibling and the other patients father had history of psoriasis.

Pruritis was present in 16(59.25%) patients.

Table No 11: Percentage of various morphological type of psoriasis

Type of psoriasis	Number of cases	Percentage
Chronic plaque psoriasis	14	51.85%
Guttate psoriasis	4	14.81%
Flexural psoriasis	3	11.11%
Palmoplantar psoriasis	3	11.11%
Pustular psoriasis	2	7.41%
Scalp psoriasis	1	3.70%
Total	**27**	**100.00%**

CHART 6:Percentage of morphological types of psoriasis



In this study chronic plaque psoriasis,14(51.85%) was the most common type of psoriasis seen in children .

Exclusive guttate psoriasis was seen in 4(14.81%) children. But guttate lesion co existed with chronic plaque psoriasis in 7(25.92%) chidren. Pustular psoriasis was seen in 7.41% of children.

The nails were involved in 5(18.51%) cases. Pitting was the most common nail change.Other nail changes observed in the study are subungual hyperkeratosis and thickening of nail plate

Exclusive scalp psoriasis was seen only in 1(3.7%) patient but 4 (14.81%) patients had scalp lesions along with other site involvement.

Infantile psoriasis was present in 2(7.4%) children.

Koebnerisation was present in 12(44.44%) patients.Auspitz sign was positive in all patients except the patients with pustular psoriasis, flexural and palmoplantar psoriasis .

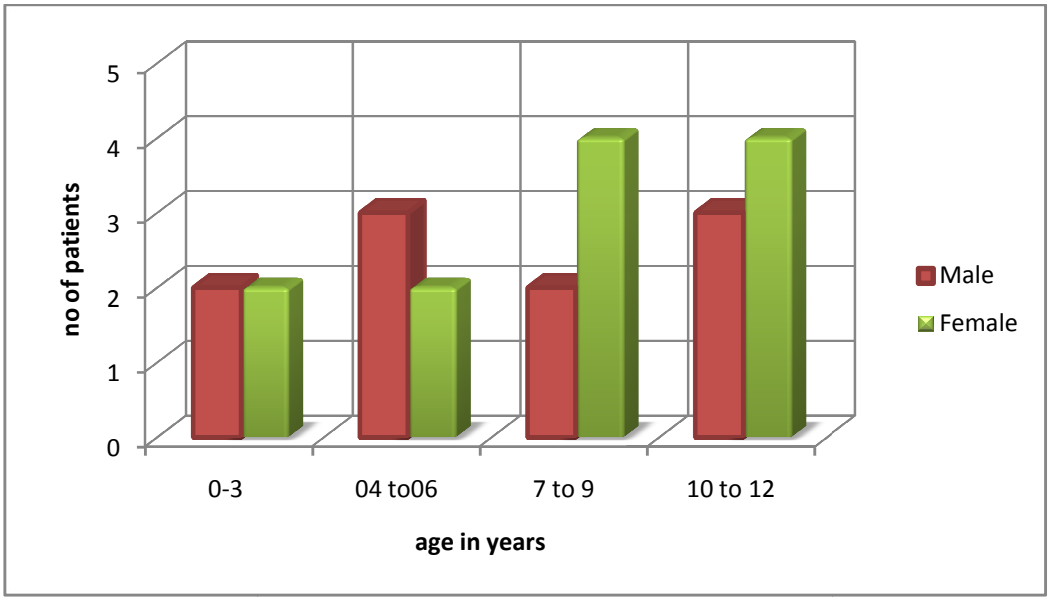
PITYRIASIS ROSEA

In the study , pityriasis rosea was seen in 22 patients (20.95%). There were 10 male patients and 12 females patients with male to female ratio 0.83: 1.

Table 12 Age sex distribution of children with pityriasis rosea

Age in years	Male	Female
0-3	2(20%)	2(16.67%)
4-6	3(30%)	2(16.67%)
7-9	2(20%)	4(33.33%)
10-12	3(30%)	4(33.33%)
TOTAL	**10(100%)**	**12(100%)**

CHART 7:Age-sex distribution of pityriasis rosea



One year was the youngest age affected and eldest was of 11 years. Maximum number of patients belonged to the age group 10-12 years.

In the study,6 (27.27%) patients had history of prodromal symptoms of upper respiratory tract infection

Herald patch was seen in 12(54.54%) patients and absent in10 patients(45.46%). Most common site of herald patch in the study was trunk. The median time between the appearance of herald patch and wide spread eruption was 1 week(range4 days-10days)

Table 13: frequency of various types of PR

Sl no	Type of pityriasis rosea	Number of patients	Percentage
1	Classical	18	81.81%
2	Inverse	3	13.6%
3	lichenoid	1	4.54%

Most common type of PR in the study was classical PR, seen in18(81.81%) patients. Inverse type of PR was seen in 3(13.6%) patients in the study . Lichenoid PR was seen in 1 (4.54%) patient.

One(4.54%) patient had eczematous changes associated with PR lesions

.

Table14:Site of involvement of pityriasis rosea

Sites	No of patients	Percentage
Face	6	27.27%
Neck	7	31.81%
Chest	10	45.46%
Back	18	81.81%
Upperlimb	13	59.09%
Lower limb	7	31.81%

In the study ,most common site affected was back(81.81%). Upperlimb lesions were seen in 12(59.09%) patients. Lowerlimb and neck lesions lesions were seen in 7 (31.81%)of patients. Least common affected site was face,6(27.27%) children.

Lichen planus

During the study period of 1 year,out of 12835 attended the OPD, 19 children had lichen planus. Thus the prevalence of lichen planus in children in our hospital was 0.14%. Out of 105 patients with papulosquamous disorders 19(18.09%) patients had lichen planus.In the study,12(63.16%)were male and 7(36.84%) were female with male female ratio 1.7:1

Table15:Age sex distribution of patients with lichen planus

Age(years)	Male	Female	Total
0-3	1(8.33%)	0(0%)	1(5.26%)
4-6	4(33.33%)	4(57.1%)	8(42.11%)
7-9	5(41.67%)	1(14.28%)	6(31.58%)
10-12	2(16.67%)	2(28.57%)	4(21.05%)
Total	**12(100%)**	**7(100%)**	**19(100%)**

In the study youngest age of onset was 3 years and eldest was 11 years. Maximum number of patients belonged to age group 7 to 9 years.

Table16 : Percentage of morphological types of lichen planus

Type	No. of cases	Percentage
Classical	11	57.89%
Generalised	5	26.32%
Linear	3	15.79%

The most common morphologic variant of LP was classical LP (57.89%). Five patients (26.32%) had generalised LP and 3(15.79%) had linear LP. Koebnerisation was seen in 68.42% 0f patients.

Nail involvement was seen in 3 patients(15.79%). Two patients ha thinning of nail plate and one patient had naildystrophy of finger nails.Oral mucosal involvement were seen in 4 patients.(21.05%).

Reticular pattern was seen in 3 patients and 1 patient had violaceous plaque

SEBORRHOEIC DERMATITIS

Seborrheic dermatitis was seen in 11(10.48%) patients in the study. There were 6 boys(54.54%) and 5 girls(45.54%) with a male to female ratio of 1.2:1

Table17:Age sex distribution of patients with seborrhoeic dermatitis

Age in months	Male	Female	Total
0-2	4(66.66%)	1(20%)	5(45.46%)
3-5	1(16.67%)	3(60%)	4(36.36%)
6-8	1(16.67%)	1(20%)	2(18.18%)
total	**6(100%)**	**5(100%)**	**11(100%)**

Among the children with seborrheic dermatitis, highest number of cases belonged to age group of 0-2 months. Youngest child was of age 1 month and eldest was of age 7 months.

Five (45.45%) children had onset of their disease before the age of 2 months.

Males had earlier age of onset as compared to females. Mean age of onset of disease in males is 2.6 months and in females is4.4 months.

Table18:Site of involvement in seborrheic dermatitis

Site	No: of patients	Percentage
scalp	10	90.91%
Face	5	45.46%
Neck	3	27.27%
Trunk	3	27.27%
Intertrigenous areas	2	18.18%

In the study,10(90.91%) children with seborrheic dermatitis had scalp involvement. Face was involved in 5(45.46%) children. Neck and trunk were in involved in 3(27.27%) children and intertriginous area in 2(18.18%.)Erythroderma was seen in one patient with seborrheic dermatitis

LICHEN STRIATUS

Lichen striatus constituted 10.91% of all papulosquamous disorders in the study. There were 4males and 7 females. The male female ratio was 0.57:1

Table19:Age-sex distribution of patients with lichen striatus

Age in years	Male	Female	Total
0-3	2(60%)	2(28.57%)	4(36.36%)
4-6	0	2(28.57%)	2(18.18%)
7-9	1(40%)	3(42.86%)	4(36.36%)
10-12	1(40%)	0	1(9.10%)
Total	4(100%)	7(100%)	11(100%)

In this study minimum age of onset was at 2 years and maximum at 11years. Eight (72.72)% 0f the patients had associated pruritis.

All cases had unilateral distribution.

Table20 :Site of distribution of lichen striatus

Site	No. of cases	Percentage
Face	2	18.18
Trunk	2	18.18
Upperlimb	3	27.27
Lowerlimb	4	36.36

In the study,most common site of involvement of lichen striatus was lowerlimb,4(36.36%) patients followed by upperlimb, 3(27.27%) patients. Face and trunk were involved in 2(18.18%) patients.

One patient had eczematous change over the lichen striatus.

PITYRIASIS RUBRA PILARIS

In this study 6 children presented with pityriasis rubra pilaris, which accounts for 5.71% of papulosquamous disorder. There were 5 males and 1female with male female ratio 5:1

Table 21:Site of involvement and type of PRP

Sl no	Age/Sex	Site of involvement	Type
1	3Yrs/Mch	Chest, back, abdomen	Type IV
2	6yrs/Fch	Chest,Abdomen, back	Type IV
3	8yrs/Mch	Elbow, knees	TYPE IV
4	11yrs/Mch	Chest, abdomen, back	TYPEIII
5	11yrs/Mch	Chest, abdomen, back	TYPE IV
6	11yrs/Mch	Scalp. back, arms	TYPE 1V

The youngest age of onset was 3 years and eldest age of onset was 11 years.

In our study TypeIV PRP , was the most common type noticed. One patient had scalp involvement and 2(33.33%) patient had palms and soles thickening.

One child had lichen nitidus and one had associated lichen striatus along with PRP lesions.

LICHEN NITIDUS

Lichen nitidus constituted 3.80% of all patients with papulosquamous disorders in the study. There were 3(75%) males and 1(25%)female with a male female to ratio of 3:1.

Table 22 Site of involvement in lichen nitidus

Sl.no	Age in years	Gender	Site
1	4	Mch	Elbow
2	6	Mch	Forearm,genitals
3	7	Fch	Trunk, extremities
4	9	Mch	Elbow,forearm

Upperlimbs were the most common site of involvement in the study. Out of the 3 male patients 1 patient had classical LN lesions over the genitals.

Koebner phenomenon was seen in 3(75%) patients.

One patient, 7 yrs old female child had generalised lichen nitidus

PARAPSORIASIS

Parapsoriasis was seen in 3 patients(2.85%) in the study. There were 2 male and 1 female patient with male to female ratio of 2:1.

Table 23Site of involvement in parapsoriasis

Sl no	Age/sex	Site of distribution
1	11yrs/Fch	Face, trunk, upper limbs and lower limbs
2	11yrs/Mch	face, trunk, upper limbs and lower limbs
3	4yrs/Mch	Tunk,upperlimb

All the three patients had small plaque parapsoriasis. The age of onset of the patients were 11 years,7 years and 3 years with mean age of onset of 7 years. Out of the 3 patients 2 patients had generalised involvement,including the face and 1 patient had lesions limited to trunk and upperlimbs.

PITYRIASIS LICHENOIDES

In the study, pityriasis lichenoides constituted 1.9%(2 patients) of papulosquamous disorders. Both of them were male patients.

One patient had pityriasis lichenoides chronica and one patient had the acute form,PLEVA.

8 year old male patient presented with hypopigmented and brown coloured scaly patches over chest, back and arms. Histopathological examination revealed features suggestive of pityriasis lichenoides chronica.

11 year old male patient presented with erythematous papules over chest and back, with central necrosis and mild scaling. There was history of fever prior to the onset of disease. Histopathological examination revealed features suggestive of PLEVA.

COLOUR PLATES

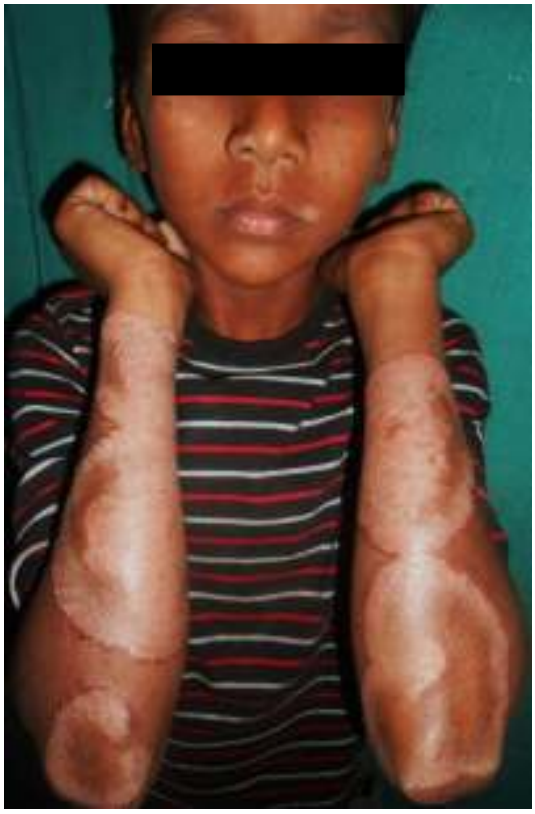


Fig. 1 Chronic Plaque Psoriasis

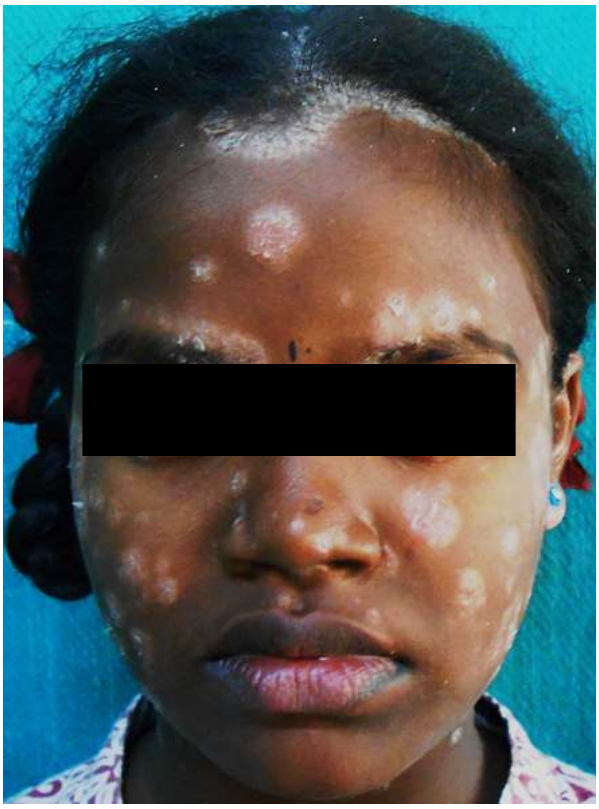


Fig. 2 Psoriasis – Scalp & Face

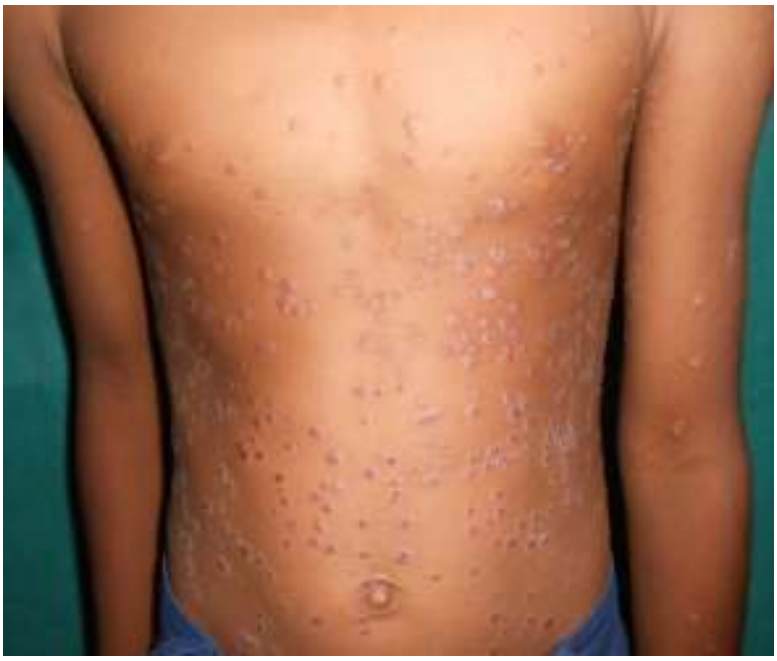


Fig. 3 Guttate Psoriasis

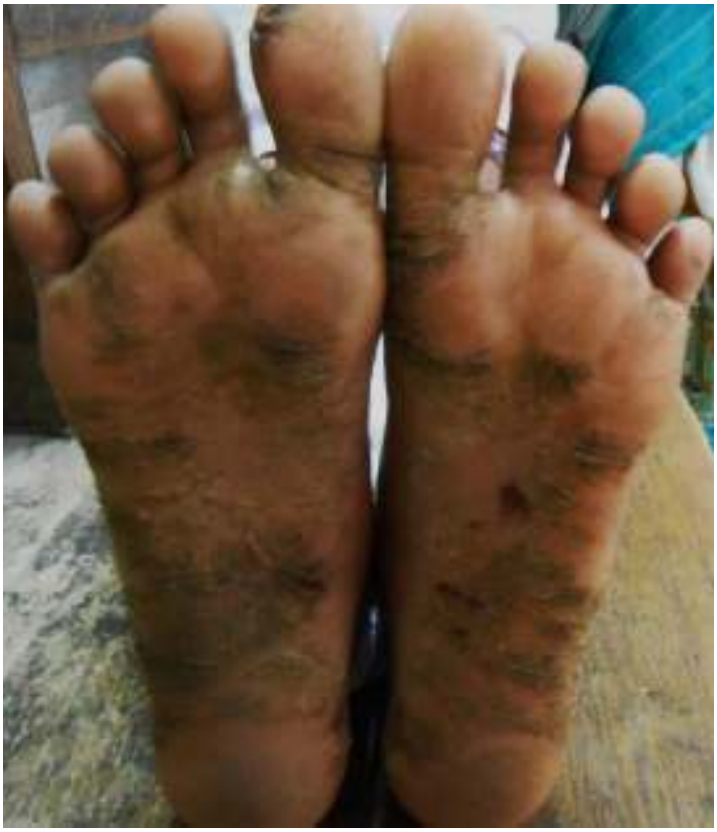


Fig. 4 Plantar Psoriasis

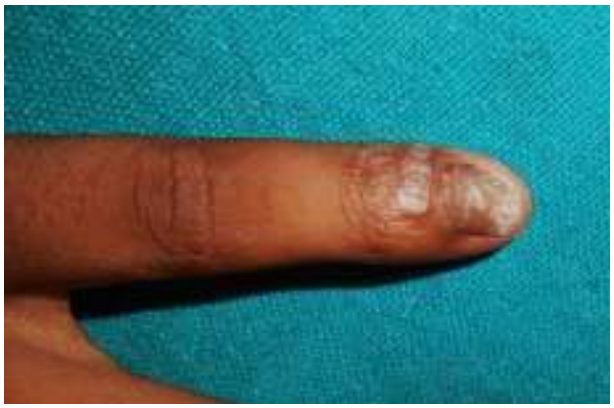


Fig. 5 Psoriasis – Nail

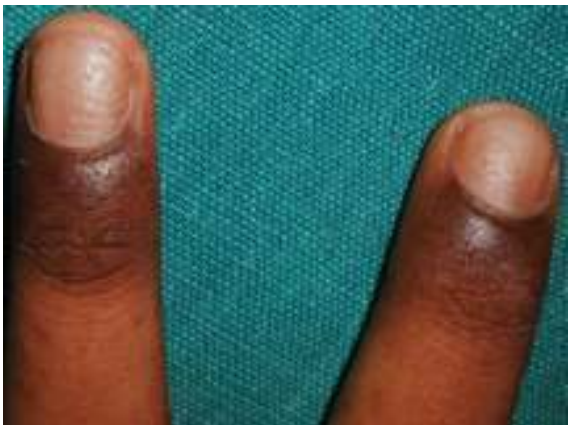


Fig. 6 Nail Pitting in Psoriasis

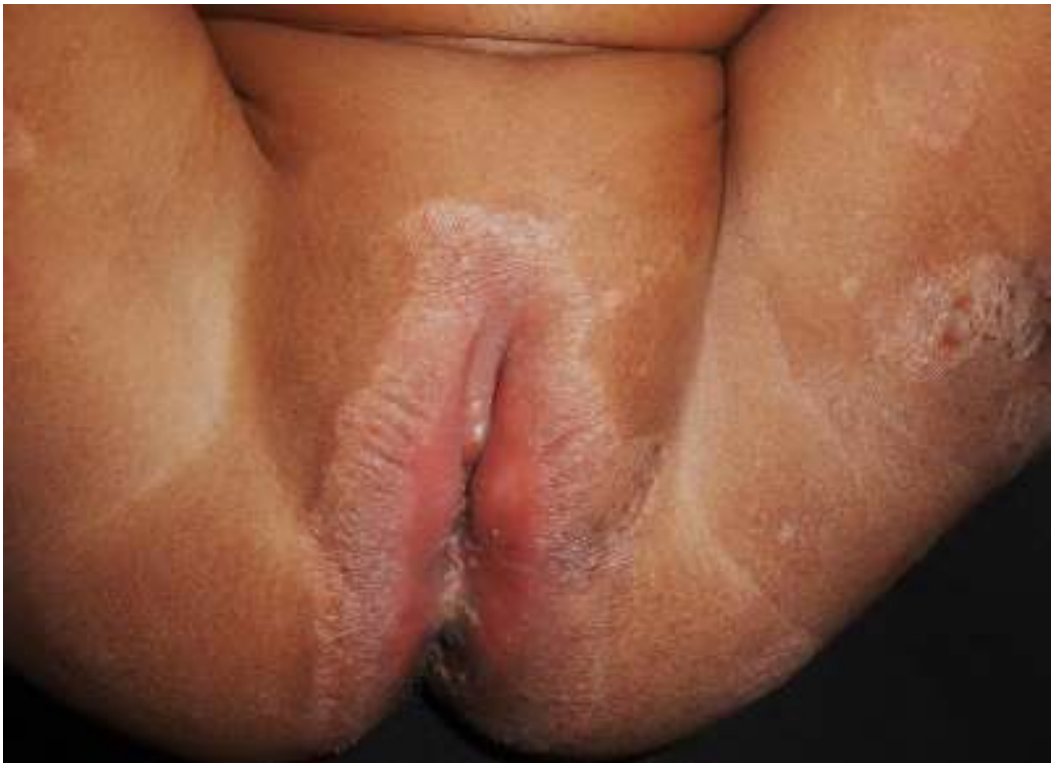


Fig. 7 Flexural Psoriasis

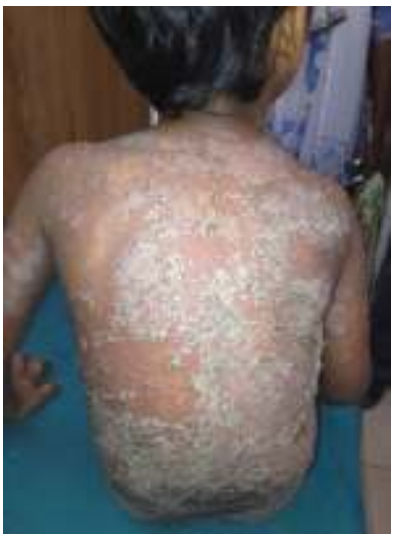


Fig. 8. Pustular Psoriasis

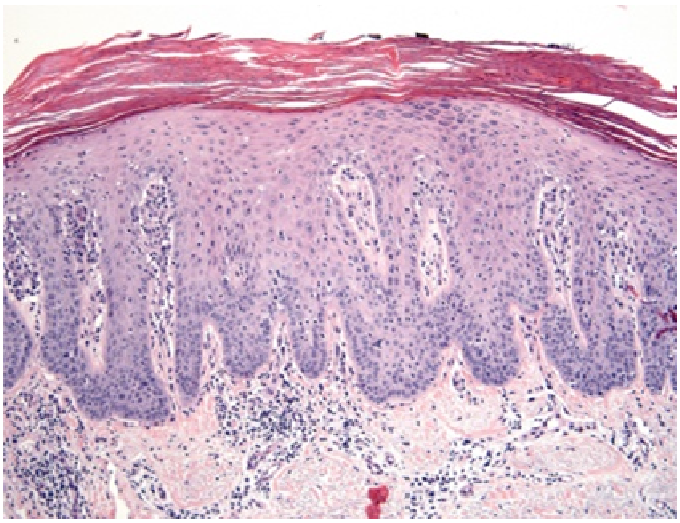


Fig. 9 Histopathology of Psoriasis

Showing Hyperkeratosis ,Parakeratosis,micro munro abscess, regular Elongation of Rete ridges and Lymphocytes in the dermis

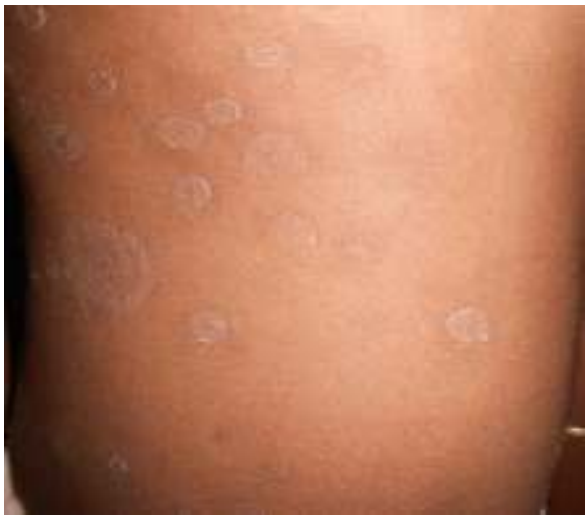


Fig. 10 Pityriasis Rosea

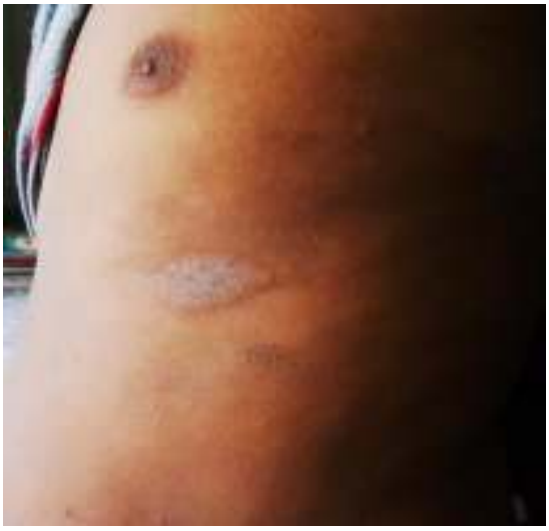


Fig.11 Herald Patch

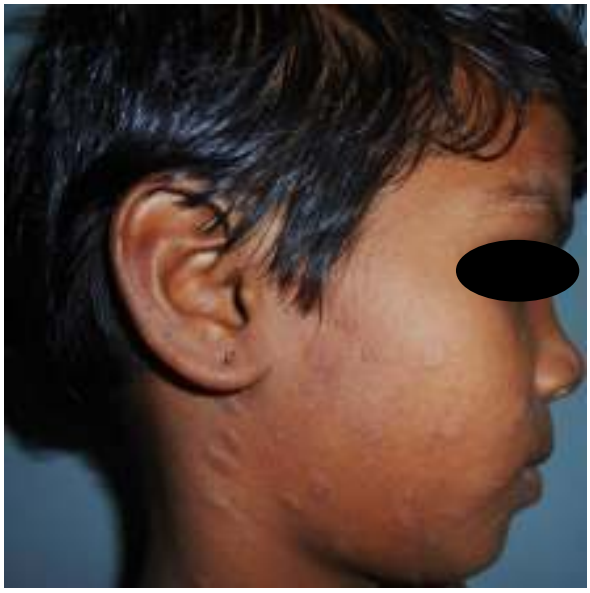


Fig. 12 Pityriasis Rosea-face

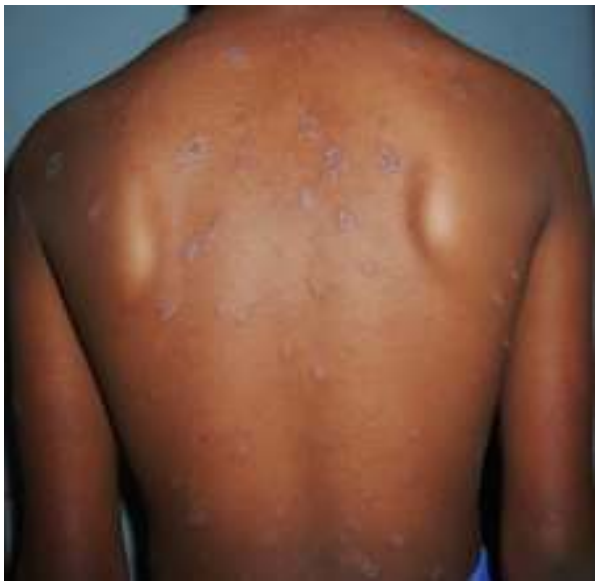


Fig. 13 Lichenoid Pityriasis Rosea

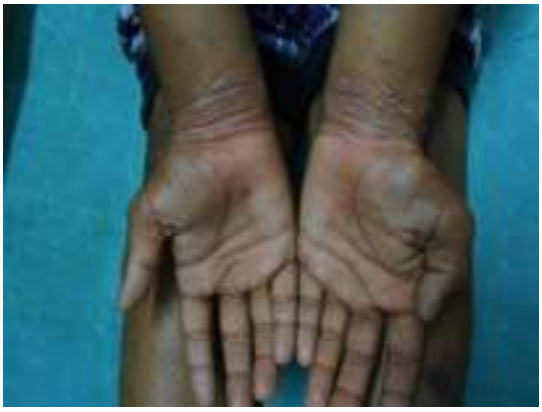


Fig. 14 Classical LP

Fig. 15 Generalised LP

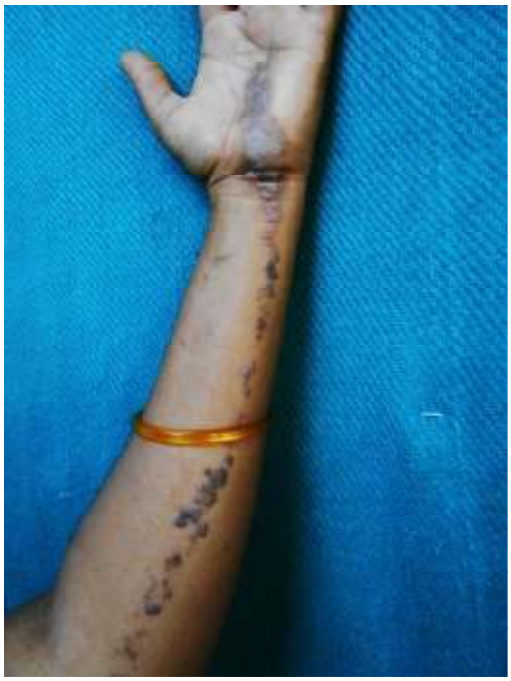


Fig. 16 Linear LP extending to Palms

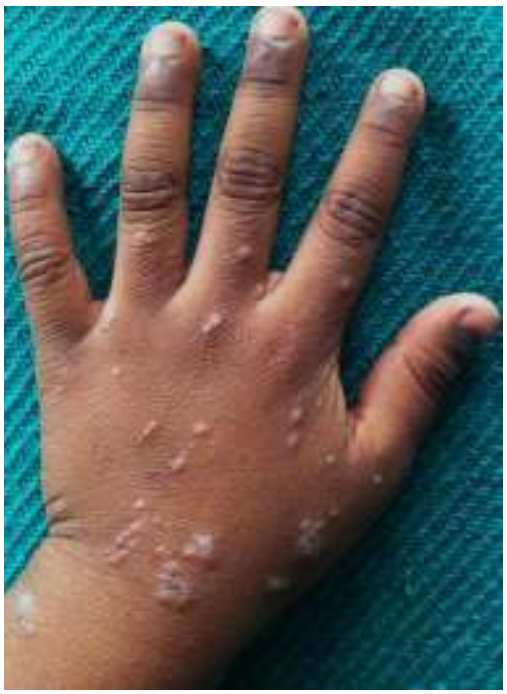


Fig. 17 LP – Koebnerisation

Fig. 18 LP-Violaceous Plaque over Lip involvement

Fig. 19 LP – Genital

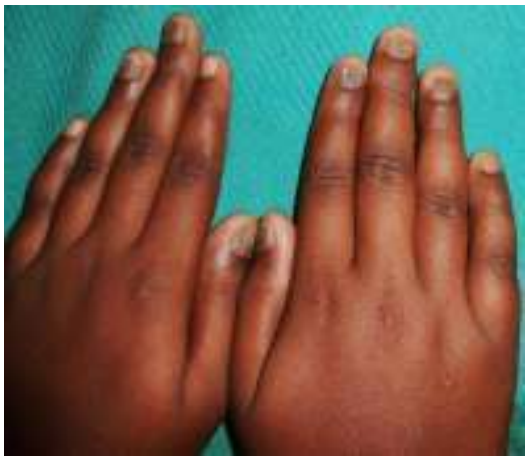


Fig. 20 LP - Nail Dystrophy

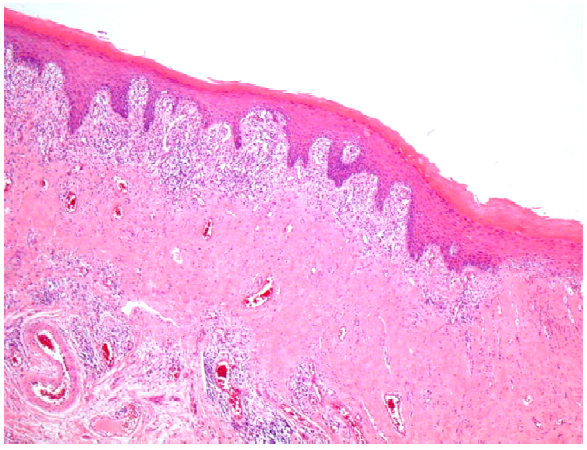


Fig. 21 Histopathology of LP
shows saw toothening of rete ridges with with Bandlike infiltrate in the dermis

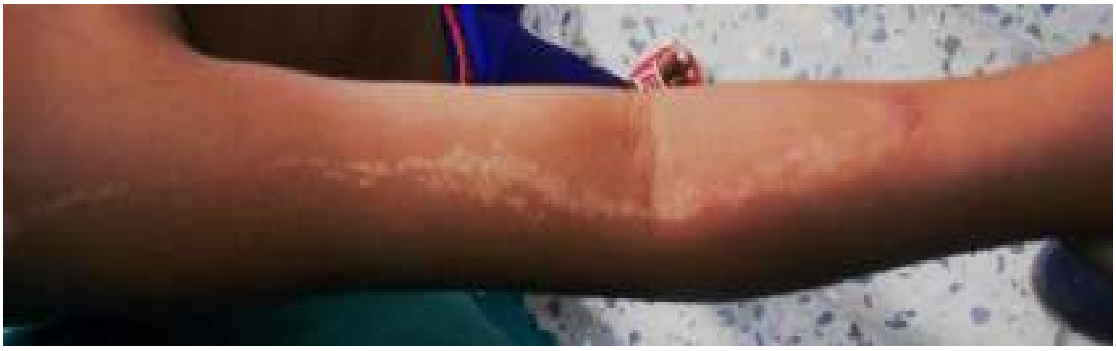


Fig. 22 Lichen Stratitus - Upperlimb

Fig. 23 Lichen Striatus – Thigh

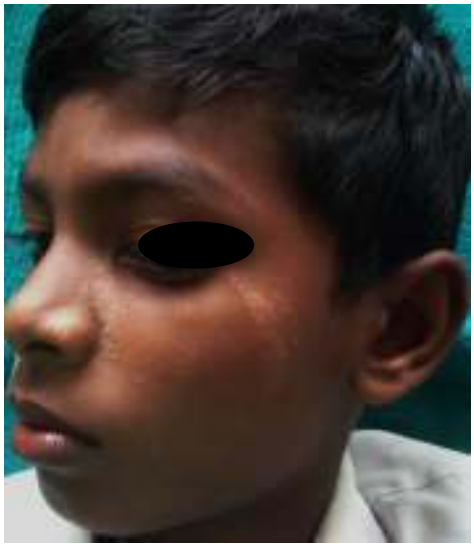


Fig. 24 Lichen Striatus – Face

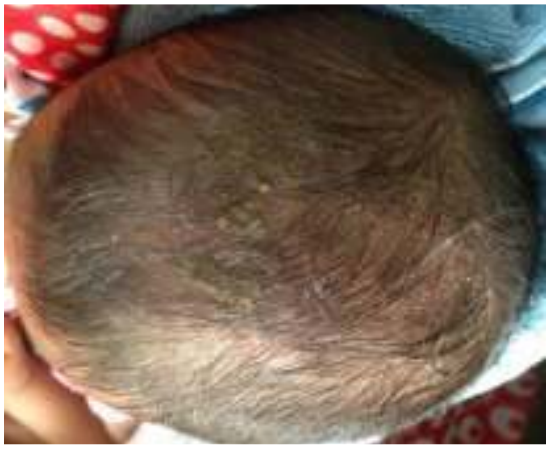


Fig. 25 Seborrhoeic Dermatitis-Scalp Yellow greasy scales

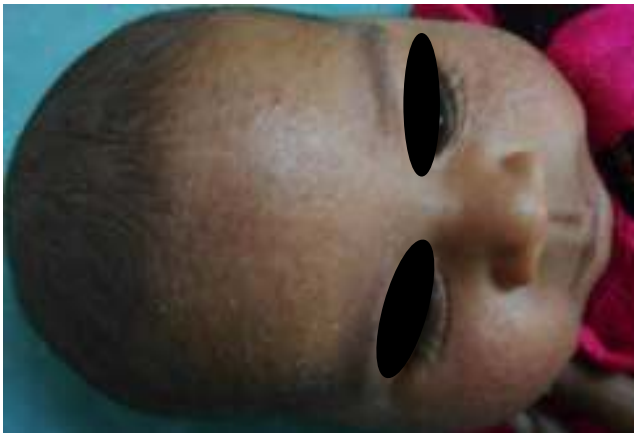


Fig. 26 Seborrhoeic Dermatitis – Face

Fig. 27 Seborrhoeic Dermatitis – Erythroderma

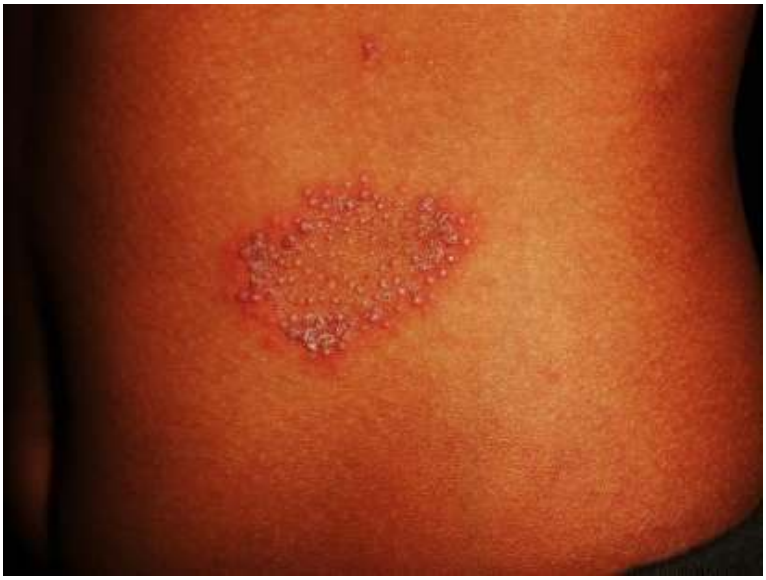


Fig 28 Pityriasis Rubra Pilaris – Type IV

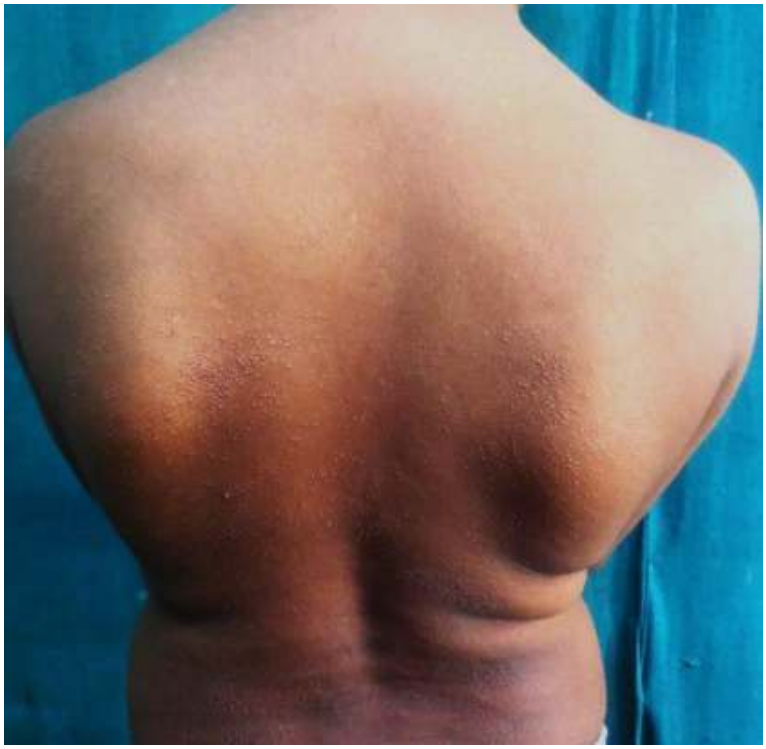


Fig 29 Pityriasis Rubra Pilaris – Type III

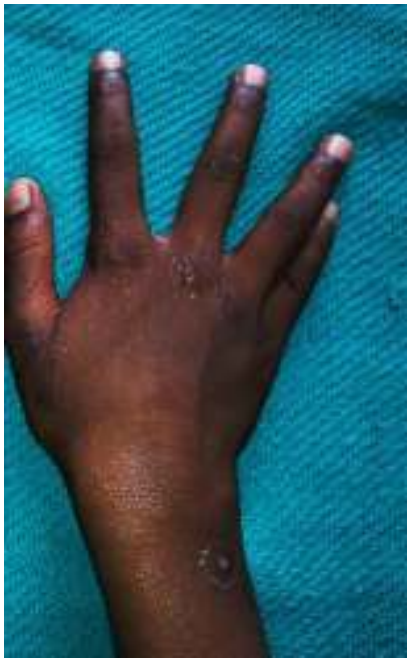


Fig. 30 Lichen Nitidus

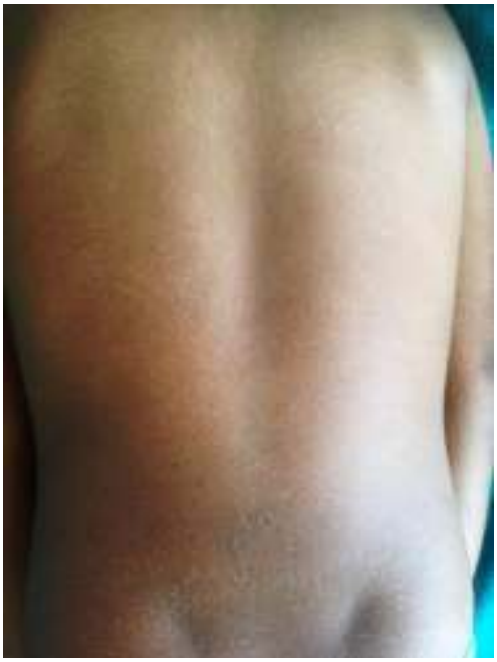


Fig. 31 Generalised LN

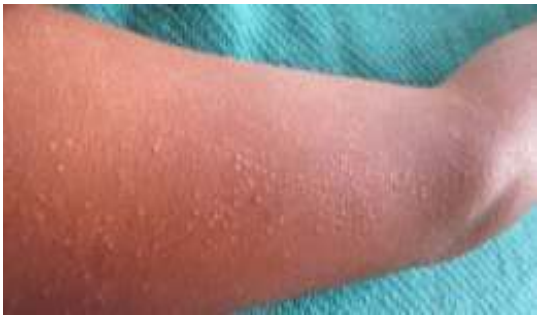


Fig. 32 LN with Koebnerisation

Fig. 33 LN – Genitals

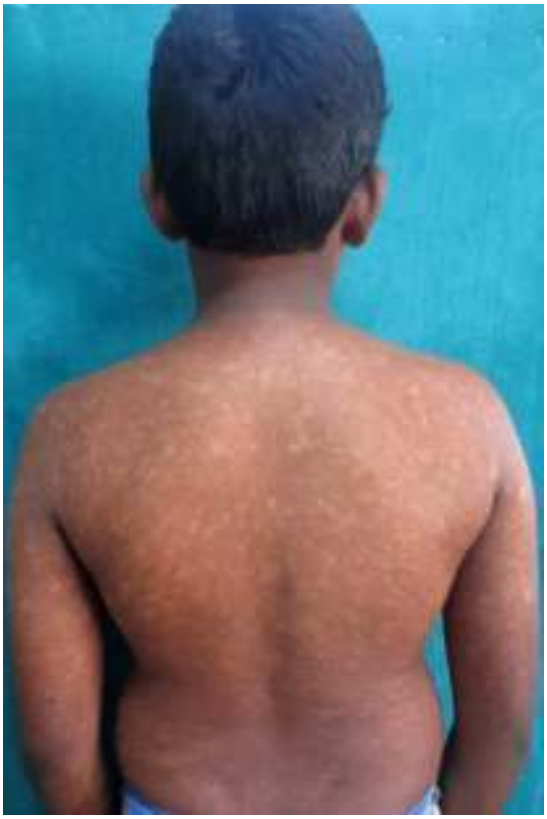


Fig. 34 Small Plaque Parapsoriasis

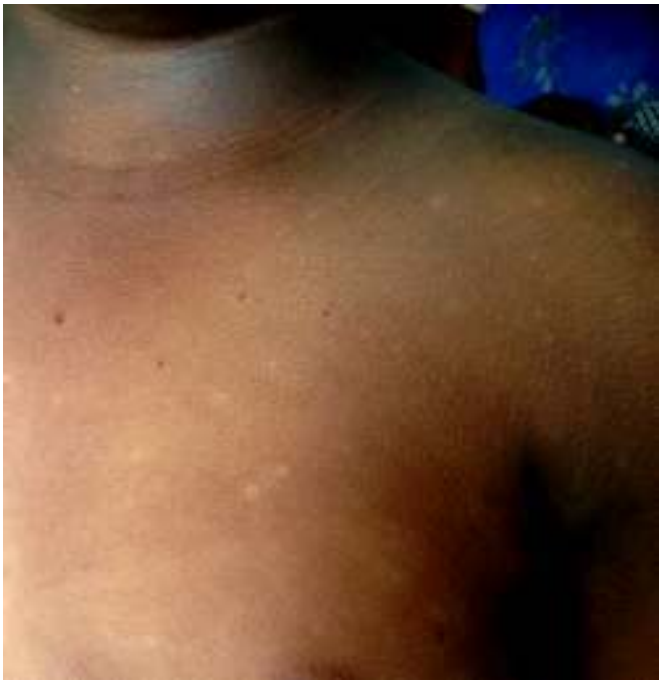


Fig. 35 PLEVA
Erythematous Papules with Central Necrosis

DISCUSSION

Dermatological disease forms an important part in the out patient attendance of paediatric population. Skin diseases in children are associated with significant morbidity and they form a major health problem in the paediatric age group. Skin diseases contribute to 30% of outpatient visits to paediatrician and 30% of patients consulting a dermatologist will be from paediatric age group.[126,127]

The prevalence of paediatric dermatoses in various parts of India ranges from 8.7 to 35% in school based surveys.[14]In the present study, hospital based prevalence of skin disease among children was 18.48%.

Papulosquamous disorders constituted 0.81% of paediatric dermatosis in the present study. In an Indian study conducted in Rajasthan, papulosquamous disorders constituted 1.66% of paediatric dermatosis.[2]In a recent study from Turkey[128] papulosquamous disorders constituted 6.9% of all paediatric dermatosis and a similar study[129] showed a prevalence of 14.2% among the paediatric dermatosis.

The difference in the prevalence rate may be due to the short duration of the study and geographical variations.

AGE -SEX DISTRIBUTION

In the present study male patients(52%) outnumbered the female patients(48%) with a male female ratio of 1.1:1which is similar to the study by Vetrichevvel etal,[130] where boys (57.5%) outnumbered girls (42.5%). But in another study on papulosquamous disorders in children, females(54.9%) outnumbered males(45.1%)[131]

In our study highest percentage of patients belonged to the age group. 10-12 years(27.62%). In Kalyani vasram vadite study, highest percentage of patients belonged to the age group 5-9 years.[131]

FREQUENCY OF VARIOUS PAPULOSQUAMOUS DISORDERS

In the present study, the various papulosquamous disorders seen in decreasing order of frequency were, psoriasis(25.71%), pityriasis rosea(20.95%), lichen planus(18.09%), seborrheic dermatitis(10.47%). Lichen striatus(10.47%), pityriasis rubra pilaris(5.71%),lichen nitidus(3.8%) , parapsoriasis (2.85%) and pityriasis lichenoides(1.9%).

In a similar study conducted by Kalyani vasram vadite,[131] the frequency of papulosquamous disorders in decreasing order were lichen planus(28.43%), pityriasis rosea(25.49%), seborrheic dermatitis(19.61%), psoriasis vulgaris(16.67%), lichen striatus(3.92%), pityriasis rubra pilaris(3.92%) and lichen nitidus(1.96%).

Vetrichevvel et al[130] reported pityriasis rosea as the commonest disease in their study followed by lichen planus, seborrheic dermatitis(11.11%), psoriasis (11.1%), pityriasis rubra pilaris (10.2%), lichen nitidus(9.2%), lichen striatus (6.4%), pityriasis lichenoides chronica (2.7%), acrodermatitis enteropathica and drug induced erythroderma (one case each, 0.9%) and a total of 5 cases of erythroderma due to papulosquamous causes were noted.

An Egyptian study[132] reported pityriasis rosea as the commonest disease in papulosquamous disorder followed by psoriasis and and PRP .

Psoriasis

During the study period of 1year, out of 12385 patients attended dermatology OPD 27 patients had psoriasis. Thus the hospital based prevalence of childhood psoriasis was 0.21%. This is comparable to the study conducted in Karnataka which showed a prevalence of 0.38%[131]and low as compared to a South Indian study in paediatric population which showed a prevalence of 1.4%.[14]

A study conducted in Saudi arabia by Ghada A. Ben Saif and Salha A. Al Shehab[3] showed a prevalence of 1.8% among paediatric dermatosis. In a Turkish study[133] and study by Gul et al[128] showed higher prevalence of 3.8% and 5.4% respectively.

In this study, among 105 patients with papulosquamous disorders, 27(25.71%) were psoriasis. This is comparable to a study conducted in Saudi Arabia[3] which showed a prevalence of 22.6% childhood psoriasis among the papulosquamous disorders. But Vetrichevvel et al[130]noted a lower prevalence of 11.11% of childhood psoriasis among papulosquamous disorders.

In the present study females out numbered males with a male to female ratio of1: 1.45.Mercy et al[134] reported a similar male to female ratio of 1:1.48 among childhood psoriasis where as kumar et al[18] showed a male to female ratio of 1.09:1.A survey of 1262 childhood psoriasis in Australia[21] reported a male to female ratio of 1:1.

The age of onset in our study ranged from 6 months to 12 years. Kumar et al[18] reported age of onset from 4days to 14 years.

The mean age of onset in our study was 6.83years.In a study from Denmark[135] mean age of onset of childhood psoriasis was 8.1 years which is slightly higher than the present study. Kumar et al[18] reported a mean age of onset of 8.1±2.1 years in boys and 9.3±2.3 in girls. In the present study mean age of onset of psoriasis in boys was 6 years and 7.4 years in girls. So boys had an earlier age of onset than girls.

In our study,the peak age of onset in males was 7 to 9 years where as in females, peak age of onset was 10-12 years. Kumar et al[18] reported a peak age of onset of 6-10 years in males and 10-14 years in females.

Chronic plaque psoriasis was the most common presentation in our study(51.85%) as observed by various previous studies.[18,20,22,23]

Table 24Comparison of frequency of chronic plaque psoriasis in various studies

Sl.no	Author	Place of study	Percentage
1	Nanda A et al[20]	Chandigarh ,India	69.6%
2	Fan X et al[22]	China	68.6%
3	Kumar et al[18]	India	60.6%
4	Yan wu et al[23]	China	52.6%
5	Present study	coimbatore	51.85%

In contrast to the above studies, guttate psoriasis was the most common type observed in Denmark.[21]In the present study guttate psoriasis was seen in 14.81%. Nanda et al[20] observed guttate psoriasis in 25.9% of childhood psoriasis and Yan wu et al[23] reported a frequency of 25.5% of guttate psoriasis in china.

The palms and soles have been reported to be uncommon sites of psoriasisinvolvement in children. However, in our study, palmoplantar psoriasis was observed in 11.11% of patients. Kumar B et al[18] reported

palmoplantar psoriasis in 12. 8% of childhood psoriasis. Morris et al[21] reported plantar psoriasis in 4% ,however Fan X et al[22] did not observe plantar psoriasis in their study.

In our study,pustular psoriasis was observed in 7.41% of the psoriasis patients . Yan Wu et al[23] observed in 10.90% of children with psoriasis.Seyhan M et al[136] reported pustular psoriasis in 13.1% of childhood psoriasis.

In our study infantile psoriasis was present in 7.4% of the patients. In Kalyani vasram study[131] infantile psoriasis was seen in 5.9% of patients with psoriasis

Nail involvement was seen in 5(18.52%) patients which is low as compared to Yan wu et al[23],who reported 25.5%. Pitting was the most common finding in the study.

Variable familial incidence has been reported in various studies of childhood psoriasis which ranges from 9.8 to 89%.In our study family history was seen in 2(7.4%) patients. Kumar et al[18] and Yan wu et al[23] reported a familial incidence of 4.5% an 8% in childhood psoriasis respectively. But a study conducted in United States by Mercy et al [134] showed a familial incidence of 51.4% in childhood psoriasis.

Yan wu et al[23] and Seyhan et al[136] reported that upper respiratory tract infection was the most common triggering factor in childhood psoriasis. In our study precipitating factor was seen in 11.11% patients, which was upper respiratory tract infection in all the cases.

Pruritis was the most common symptom in our study(59.25%) kumar et al[81] studies reported pruritis in.87.1% of the patients.

Koebnerisation was seen in 12 patients (44.44%) which was higher than the observation in kumar et a1[8l] study,which was 27.9%.

Psoriatic arthropathy and erythrodermic psoriasis were not observed in our present study.In a study from Karnataka[131]also there were no cases of psoriatic arthropathy ans erythroderma.A north Indian study reported psoriatic arthropathy in 1.1% of their subjects. Mercy et al[134]study reported psoriatic arthritis in 10.5% of all the subjects.Nanda et al[20] study reported psoriatic erytroderma in 1.8% of their patients. A study from china[23]showed , psoriatic erythroderma in 5.1% of their patients. These study shows that prevalence of psoriatic erythroderma have regional and racial variation

Pityriasis rosea

Pityriasis rosea is an acute self limiting inflammatory papulosquamous disorder of unknown etiology.

In the present study .pityriasis rosea contributed 0.17% of all the paediatric dermatosis attending our skin OPD. A study from South India reported ,[14] pityriasis rosea in 0.2% of all the paediatric dermatosis. Another study conducted in north eastern region of India[94] showed a frequency of 1.2%. but a study from Iraq[129] showed a higher frequency of 2.9% among children.

In a clinical study of papulosquamous disorders in children by Vetrichevvel et al[130], pityriasis rosea was the most common disorder(32.4%) where as in our study PR was the second common condition seen in 20(22.95%) patients. This is comparable to a clinical study of papulosquamous disorders in children in Karntaka(25.49%)[130].

In our stuy there were 10 males and 12 females with male to female ratio of 0.83: 1 which is similar to study conducted by Kalyani vasaram valdite[131] with male to female ratio 0.7:1.In a clinical study on pityriasis rosea conducted by Egwin AS et al[137] male to female ratio was 1.5:1 and by Gunduz et al[138] was 1.1:1

The youngest age of pityriasis rosea in Andrew etal[139] study was 3 months old and 1 year in our study.

The maximum number of children with pityriasis rosea belonged to the age group 10-12 years(31.8%). Gunduz et al[138] and kalyani vasram vadite[131] observed peak incidence in 6-11 years age group with 49% and 53.8% patients respectively

Seasonal variation has been found in pityriasis rosea. Gunduz et al[138] and Kalyani Vasram Vadite[131] observed a peak incidence during winter and autumn months. In our study peak incidence was during winter season(54.55%) similar to the observation of Vetrichevvel et al.[130]

A history of fever and sorethroat suggestive of upper respiratory tract infection was seen in 27.27% of patients.Gunduz et al[138] observed prodromal symptoms in 33% of patients .

Pruritis was present in 16(72.72%) of the patients.Gunduz et al[138] observed pruritis in 69% of their patients.

Herald patch was seen in 54.46% of patients in our study, most common over the trunk with a median period between appearance of herald patch and widespread eruptions of 7days(4-12days). Gunduz et al[138] study observed herald patch in 45% of patients, which was located

most often over the trunk. The median period between appearance of herald patch and widespread eruptions was 4 days(range 0-30 days).

Most common site of involvement in our study was back 81.81%. Least affected site in our study was face(27.27%) .In a clinical study by Kalyan vasaram valdite[131], back was the most common site(88.5%) and neck was the least affected site(23.1%)

In our study,most common type of pityriasis rosea was classical type(81.81%). Inverse PR was seen in 3 patients and lichenoid PR in 4.54% of patients.

We have observed eczematous changes in 1(4.54%) patient with pityriasis rosea.

Lichen planus

Lichen planus is a papulosquamous disorder of unknown etiology.Eventhough lichen planus is very rare in children,most reports of LP in children have come from the Indian subcontinent, suggesting that children of South Asian origin are more susceptible to developing LP.(lp7) The frequeny of lichen planus varies from 2.1% to 11.2% in the paediatric population. The prevalence of childhood lichen planus in children in our study was 0.15%. This is low when compared to study conducted by Handa et al[52] in North India ,who reported 2.5% among the

total paediatric patients. A hospital based study conducted at Iraq[129]showed , lichen planus formed 3.9% of the total number of paediatric dermatosis in a 6 month period.

In the study of Vetrichevvel et al[130] lichen planus constituted 14.8% of various papulosquamous disorders where as in our study lichenplanus constituted 18.09%of the total papulosquamous disorders.Kalyani vasaram valdite[131] reported a higher frequency of 28.43% of lichen planus among the papulosquamous disorers.

Most of the studies show an equal sex prediliction in lichen planus or a slight male preponderance. Handa et al[52] and Kalyani vasaram valdite[131] reported a male female sex ratio of 1.1:1 . In our study the male female ratio was higher than these studies,ie; 1.7:1. Sharma and Maheswari[64] have also reported a higher male female ratio of 2:1.

In our study, age group ranged from 3-12 years with mean age of onset of 6.6 years. In Handa et al[52] study of lichen planus in 87 children, the age group ranged from 8 months to 12 years with mean age of onset of 7.1 years.

The most common clinical type in our study was classical lp(57.89%) which is similar to Handa et [a52] and Sharma et al[64] who observed classical LP in 60.9% and 60%of their LP patients respectively. Linear

LP was observed in 15.79 patients in our study. Linear LP was observed from 8-30% in various studies[52,55,64.]One case of linear LP ha involvement of palms also.In our study we observed acute generalised LP in 26.32 % of all LP patients which is higher than Handa et al[52] , who observed 6.9% and Sharma and Maheswari[64] who observed 4%.

Various studies on childhood lichen planus had reported a frequency of 41-70% of involvement of extremities.[52,64,68]In our study, the most common site to be involved by lichen planus was extremities(78.95%), more commonly lower limbs.

Oral mucosal involvement in Lp ranges from 4.3 to 39% in various studies[52,64,140.]Oral mucosal involvement was seen in 4 patients(21.05%) in our study which is comparable to Nnoruka et al[141] who observed oral involvement in 23.1% of their patients.

In our study nail involvement was seen in 3 patients(15.79%). Kanwar and de[68] observed nail involvement in 19.5% of their patients. But in other studies it ranges from 0-7.7%.[52,55,,64]

Koebnerisation is seen commonly in childhood LP, which ranges from 24 to 28%.In our study we observed a higher percentage of koebnerisation of 68.42%. But Kanwar and [68] observed koebneriastion in only 6% of their patients.

Pruritis was present in 84% of the patients in our study. This is comparable to findings of other authors who found pruritis in 87%, 89.6% and 100% of their patients respectively.[64,68,142]

Lichen striatus

Lichen striatus is a self limiting papulosquamous disorder of unknown etiology in which ,the lesions follow the Blaschko lines.

Lichen stratus constituted 10.91% of all the papulosquamous isorders in our study, which is high as compared to the study of Vetrichhevel et al[130] who observed lichen striatus in 6.4% children.

Lichen striatus is more common in females than in males. In our study male: female ratio was 0.57: 1 which is similar to Patrizzi et al[143] who noted male to female ratio of 0.5:1.

Lichen striatus is more common in the age group 5-15 years.In our study maximum number of patients were seen in the age group0-3 years and 7-10 years with mean age of 5.5 years. Patrizzi et al[143] reported a mean age of 4.5 years

In our study the most common site of presentation was extremities(63.63%); lower limb (36.36%) more than upperlimb(27.27%). This is similar to Taniguchi et al[144] study in which

lesions were predominant in the lowerlimbs. In Patrizzi et al[143] study, extremities were the most frequent site with no substantial difference between upperlimbs and lower limbs were seen. Taieb et al[88] study reported upperlimbs to be the most common site.

One patient with lichen striatus had eczematous changes.

SEBORRHEIC DERMATITIS

In our study seborrheic dermatitis was seen in 11(10.48%) patients. This is comparable to Vetrichevvel et al[131] who noted seborrheic dermatitis in 11.11% of their patients with papulosquamous disorders.

Seborrheic dermatitis affects males more often than female children. In our study 54.54% of the cases were male and 45.45% cases were female with a male to female ratio of 1.2: 1.Wananukul et al[145] study also showed that 51.66% of the cases were male and 48.33% were male to female ratio was 1.06:1.But in Kalyani vasram vadite[130] study females(55%) outnumbered males(45%) with male to female ratio of 0.82:1

The youngest child was of age 1 month and eldest of age 7 months. Majority of the children belonged to 0-2 months age group. Wananukul et al[145] also noticed 60% of the children were less than 2 months.

In our study , the scalp (90.91%) was the most common site to be involved which is comparable to Foley et al[146] and Kalyani vasram vadite[131] study, where scalp involvement was seen in 86% an 95% of the children respectively.

Generalised seborrheic dermatitis was noted in 1 patient in our study who was a 2 year old male child. In a study, infantile seborrheic dermatitis constituted 10% of the causes of erythroderma[10].

PITYRIASIS RUBRA PILARIS

Pityriasis rubra pilaris is an idiopathic inflammatory papulosquamous disorder characterised by follicular hyperkeratosis, palmoplantar karatodema and erythroderma. In our study 6(5.71%) children presented with pityriasis rubra pilaris. Vetrichevvel et al[130] noted pityriasis rubra pilaris in 10.92 % of the children in their study and kalyani vasram vadite noted 3.92% of the children in their study.

In our study mean age was 8 years, slightly higher than yang et al who reported mean age of 6.5 years in their study of 28 cases of juvenile pityriasis rubra pilaris.

The male to female ratio in our study was 5:1.

In our study Type IV ,juvenile onset circumscribed pityriasis rubra pilaris was the most common type (83.33%) followed by type III. Yang

et al studies also reported a similar observation. There were no cases of type V PRP in our study. There were no case of erythroderma due to PRP in our study.

Table 25Comparison of patients with juvenile pityriasis rubra pilaris in different studies

Sl no:	**Present study**	**Yang et al[147]**	**Gelmetti et al[148]**
Number of cases	6	28	31
Age of onset(in years)	3-11	0.67-18	2-13
Type III	16.67%	14.33%	34%
Type IV	83.33%	85.7%	66%
Type V	0	0	0
Site of involvement			
Scalp	16.67%	25%	32%
Palms	33.33%	89%	84%
Soles	33.33%	86%	84%
Trunk	50%	39%	25%
Elbows	66.67%	64%	77%
Knees	50%	89%	77%

Lichen nitidus

Lichen nitidus is a rare chronic papulosquamous disorder of uncertain etiology seen primarily in the paediatric population. In our study , 4(3.8%) patients presented with lichen nitidus which is low when

compared to vetrichevvel et al[131] who reported lichen nitidus in 9.2% of children.

In lichen nitidus, there is an equal male to female ratio or slight male predominance. In our study male to female ratio was 3:1 which is similar to Zapata et al[149] study in which male to female ratio was 3:1. Lapins et al also reported a higher male to female ratio of 4:1.

In our study mean age of onset was 6.5 years . Mean age of onset of males was 6.1 years which is comparable to Lapins[150] study where mean age of onset was 7 years for males. Mean age of onset in female in lapins et al study was 13 years[150]. In our study only one female was noticed who was 7 years age old.

Extremities were the most common site of involvement in our study which is similar to kalyana vasram vadite study.. Zapata et al[149] noted upperlimbs and trunk to be the most common site of involvement in their study.

Generalised lichen nitidus is a rare variant of lichen nitidus.In our study one patient, 7 year old female had generalised lichen nitidus.

Parapsoriasis

Parapsoriasis is a group of uncommon inflammatory disorders characterised by persistent scaly inflammatory eruptions. In our study

there were 3 (3.80%)cases of parapsoriasis.Vetrichevvel et al reported parapsoriasis in There were 2 males and 1 female.All the three cases were of small plaque parapsoriasis. Out of the 3 patients ,2 patients had involvement all over the body including face and 1 patient had trunk and extremities involvement.

Pityriasis lichenoids

Pityriasis lichenoies is a papulosquamous disorder of unknown etiology with acute and chronic presentations.

In our study, we noticed pityriasis lichenoides in 2(1.9%) patients Vetrichevvel et al[131] also reported pityriasis lichenoides in 2.7% of the children with papulosquamous disease in their study.

Pityriasis lichenoides is more common in males. In various studies male to female ratio varies from 1.4:1 to 2:1.[151,152] In our study both of them were male patients.

One patient had pityriasis lichenoides chronica and the other had the acute form,PLEVA.

SUMMARY

A clinical study on papulosquamous disorders of children less than 12 years was done from August 2013 to July 2014. The age-sex prevalence, age of onset, symptoms,morphology and site of involvement of lesions of each diseases were recorded, analysed and compared with other studies and discussed.

- Papulosquamous disorders constituted 0.81% of the paediatric dermatoses during the study.Majority of the patients belonged to the age group10-12 years. The youngest patient was 1 year old at the time of presentation. Boys outnumbered girls with male to female ratio of 1.1:1

- Psoriasis was the most common papulosquamous disorder (25.71%) followed by pityriasis rosea(20.95%). Pityriasis lichenoides was seen least in number(1.90%).Psoriasis patients in our study showed a female predominance with a male to female ratio of 1:1.45. Chronic plaque psoriasis was the most common type(51.85%).Guttate psoriasis was seen in (14.81%%) of patients. Palmoplantar psoriasis was seen in 11.11%. Nail involvement was seen in 18.52% and scalp psoriasis in 3.7% .Pruritis was seen in 55.55% and Koebneriastion in 40.74% patients.

- Pityriasis rosea was seen in 22.95% patients, with male to female ratio of 1.5:1. It was common in 10-12 years age group(31.8%) .Youngest patient was of age 1 year .Pruritis was seen in 72.72% patients.Herald patch was seen in 54.46% patient with most common site of involvement over the chest.Avaerage time period between appearance of herald patch and widespread eruptions was 7 days.Most common type was classical PR seen in 81.81% patients. Inverse PR was seen in-13.6% and lichenoid PR was seen in4.54% patients.

- Lichen planus was seen in 18.09% of the total 105 patients with male to female ratio of 1.7:1. Mean age of onset was 6.6years. Most common clinical type was classical LP(57.89%). Generalised LP was seen in 26.32% patients and linear LP was seen in 15.79% patients. Most common site involved was extremities(78.95%), more commonly lowerlimbs. Oral mucosal involvement was seen in 21.05% patients. Nail involvement was seen in 15.79%. pruritis was seen 84% of the patients and koebnerisation in 68.42% patients.

- Seborrheic dermatitis was seen in 10.48% of patients with male to female ratio of 1.2: 1. Children with seborrheic dermatitis belonged to 1 months and 7 months. Majority of children belonged to 0-2 months age group. Most common site to be involved was scalp(90.91%). Erythroderma was present in 1 patient.

- Lichen striatus constituted 10.48% of patients with papulosquamous disorders with male to female ratio of 0.5:1. Mean age of onset was 5.5 years. Most common sites involved were extremities.Most common pattern observed was linear pattern.

- Pityriasis rubra pilaris was seen in5.71% of the patients with male to female ratio of 5:1.Most common type of PRP noticed was Type IV(83.33%). Most common sites observe was elbows(66.67%). Palms and soles were involved in 33.33% of patients an scalp was involved in16.67%

- Lichen nitidus was seen in 3.8% of patients with male female ratio of 3:1. Most common site involved was extremities.One of the three male patients had typical lesions over genitalia. Generalised lichen nitidus was seen in one patient.

- Parapsoriasis was seen in 2.85% of patients in the study. All the patients had small plaque parapsoriasis. The mean age of onset of parapsoriasis in our study was 7 years. Two patients had generalised involvement including the face. One patient had distribution of the lesions over the trunk and upperlimbs.

- Pityriasis lichenoides was seen in 21.9% of the patients. One patient had pityriasis lichenoides chronica and one patient had PLEVA. In our study,all the patients were male.

CONCLUSION

Papulosquamous disorder is a common dermatological group of disorders which is more predominant in male children . Various factors like age, gender and seasonal variation play a role in the occurrence and exacerebration of these group of disorders. Genetic inheritance plays negligible role in the occurrence of these disorders. The prevalence rate of papulosquamous disease varies from region to region. This regional variation can be due to the climatic and racial variation.

The prevalence of papulosquamous disorders in our study was 0.81%. The most common papulosquamous disorder in the study was psoriasis

Understanding the morphology , distribution of the lesions, characteristics of the scales and special features like koebnerisation, Auspitz sign, herald patch etc will help to differentiate the individual diseases clinically. This will avoid invasive procedures like biopsy particularly in paediatric age group, as the patients and the parents will be anxious . When it is difficult to differentiate the diseases clinically, biopsy and histopathological examination will come in hand. Individual diseases in this group have variable prognosis..

By understanding the morphological characterestics of papulosquamous diseases and differentiating the diseases clinically ,we can explain the prognosis of the disease to the parents, which will alleviate their worry.

LIMITATION OF THE STUDY:

- As the study was of short duration, follow up and response to treatment could not be evaluated.
- Natural course of the disorders could not be studied as the study was of short duration.

BIBLIOGRAPHY

1. Fox BJ, Odom RB.BalaiPapulosquamous diseases: a review.J Am AcadDermatol. 1985 Apr;12(4):597-624.

2. M,KhareAK,Gupta LK, Mittal A, KuldeepCM.Pattern of paediatric dermatoses in a teritiary care centre of South West Rajasthan.Indian J dermatol 2012;57:275-8.

3. Ghada A. Ben Saif and Salha A. Al Shehab Pattern of Childhood Dermatoses at a Teaching Hospital of Saudi ArabiaInt J Health Sci(Qassim).Jul 2008:63-74.

4. Gibson LE,PerryHO.Papulosquamous and exfoliative dermatitis .In Moschella SL, Hurley HJ , editors. Dermatology.3rd ed. Philadelphia: WB Saunder's company ; 1992.p607-51

5. MobiniN,ToussaintS,Kamino H. Non infectious erythematous popular and squamous disorders. In Elder DE,ElensistsasR,JohnsonJr.Bl, Murphy GF, editors. Lever's histopathology of skin.9thed Philadelphia: Lippincott Williams and Wilkns ;2006.p179-214.

6. Hall JC. Saurer's manual of skin disease. 8thed, Philadelphia:Lippincot Williams and Wilkins; 2000.

7. Papulosquamousdisorders(L40-45).Chapter XII Diseases of skin and subcutaneous tissue(L00-L99).International Statistical

Classification of Diseases and Related Health Problems 10th Revision (ICD-10) Version for 2010.cited on September 2014. Available from http://apps.who.int/classifications/icd10/browse/2010/em#/L40-45

8. C.E.M.Griffiths and J.N.W.N.Baker. In Tony Burns,Stephenbreathnach, Neil cox, Christopher Griffiths editors..Rooks text book of dermatology. 8th edition.Chapter 20,Psoriasis: 20.1-20.60

9. Benoit S,HammH.Psoriasis in childhood and adolescence. Clinical features and therapy.Hautarzt 2009;60:100-8

10. Dogra S, De D, Ragunatha S. Disorders of keratinisation. In: InamadarAC,Sacchidanand S, eds. Textbook of Pediatric Dermatology, 1st ed. New Delhi:Jaypee publications;2009:pp334-59

11. Patel S, Paller AS: Pediatric psoriasis. In: Yoo JY, Lee CS, Lebwohl MG, et al ed. Psoriasis, New York: Informa Healthcare; 2009:219-238.

12. Rogers M: Childhood psoriasis. CurrOpinPediatr 2002; 14(4):404-409.

13. QaziMasood,Iffat Hassan. Pattern of skin disorders in Kashmir valley. Indian J DermatolVenerolLeprol 2002; 47(3):147-8

14. Karthikeyan K, Thappa DM, Jeevankumar B. Pattern of pediatricdermatoses in a referral centre in South India. In Pediatrics 2004;41:373-7.

15. Nanda A, Hasawi FA, Alsaleh QA. A prospective survey of pediatric dermatology clinic patients in Kuwait: An analysis of 10,000 cases. PediatrDermatol 1999;16:6-11.

16. Jain N, Khandpur S. Paediatric dermatoses in India.Indian J Dermatol,Venerol,Leprol 2010; 76: 451-4.

17. SandipanDhar, Raghubir Banerjee, Nilesh Agrawal,Sharmila Chatterjee, and RajibMalakar. Psoriasis in children: An insight. Indian J Dermatol. 2011 May-Jun; 56(3): 262–265.

18. Kumar B, Jain R, Sandhu K, Kaur I, Handa S. Epidemiology of childhood psoriasis: A study of 419 patients from northern India. 2004;43:654-8. Int J Dermatol. 2004;43:654–8.

19. Verbow J.Psoriasis in childhood. Arch Dis Child 1992;67:75-6.

20. Nanda A,KaurS,Kaur I et al . Childhood psoriasis: an epidemiological survey of 112 patients. Paediatr Dermatology 1990;7:19-21.

21. Morris A, Kaur S, Kaur I et al. Chilhood psoriasis: a clinical review of 1262 cases. Paediatrdermatol 2001;18: 188-98.

22. Fan X, Xiao FL, Yang S, et al: Childhood psoriasis: a study of 277 patients from China. J EurAcadDermatolVenereol 2007; 21(6):762-765.

23. Wu Y, Lin Y, Liu HJ, Huang CZ, Feng AP, Li JW. Childhood psoriasis: A study of 137 cases from central China. World J Pediatr. 2010;6:260–4.

24. Mercy et al.Clinical manifestations of pediatric psoriasis: Results of a multicentre study in the United States.PediatrDermatol. 2013 July ; 30(4): 424–428.

25. SwanbeckG,InterotA,Martinssont,Enerbackk C, EnlundF,Samuelsson et al.Genetic counselling in psoriasis: Empirical data on psoriasis among first degree relatives of 3095 psoriatic probans.Br J Dermatol 1997;137: 9339-42.

26. David weedon.Thepsoriasiformreation pattern. Weedon's Skin pathology. 3rd edition, pg.78-79

27. Flora B.de Waard-van der Spek and Arnold P Oranje. Psoriasis. Harper's Textbook of Paediatric Dermatology, 3rd edition.80.1-80.6.

28. Naldi L, Peli L, Parazzini F, et al: Family history of psoriasis, stressful life events, and recent infectious disease are risk factors for a first episode of acute guttate psoriasis: results of a case-control study. J Am AcadDermatol 2001; 44(3):433-438.

29. Amy S. Paller and Anthony J. Mancini. Psoriasis and related papulosquamous disorders. Hurwitz clinical paediatric dermatology. 4th edition.pg.71-91.

30. Johann E,Gujonsson and james T elder.Psoriasis. Fitzpatrik's textbook of ermatology in general medicine. 8th ed. Vol1: pg197-231

31. Flora B.de Waard-van der Spek and Arnold P Oranje. Psoriasis:Treatments. Harper's Textbook ofPaediatric Dermatology. 3rd edition.82.1-80.7.

32. Gibert CM: TraitePratique de Maladies de la Peauet de la Syphilis, 3rd edition. Paris, Plon, 1860, p. 402.

33. KlauderJV.PityriasisRoseawith particular reference to its unusual manifestations .JAMA 1924;82:178-83.

34. De Souza Sittart JA, Tayah M, Soares Z. Incidence pityriasisrosea of Gibert in the Dermatology Service of the Hospital do ServidorPúblico in the state of São Paulo .Med CutanIberoLat Am. 1984;12(4):336-8.

35. Olumide Y. Pityriasisrosea in Lagos. Int J Dermatol. 1987 May;26(4):234-6.

36. Giam YC. Skin diseases in children in Singapore. Ann Acad Med Singapore. 1988 Oct;17(4):569-72.

37. Traore A1, Korsaga-Some N, Niamba P, Barro F, Sanou I, Drabo YJ. Pityriasisroseain secondary schools in Ouagadougou, Burkina Faso. Ann DermatolVenereol. 2001 May;128(5):605-9.

38. Chuh AA et al: Temporal case clustering in pityriasis rosea: A regression analysis on 1379 patients in Minnesota, Kuwait, and Diyarbakir, Turkey. Arch Dermatol 141:767, 2005.

39. HYATT HW Sr. Pityriasisrosea in a three month old infant. Arch Pediatr. 1960 Sep;77:364-8.

40. Drago F, Vecchio F, Rebora A: Use of high-dose acyclovir in pityriasisrosea. J Am AcadDermatol2006;54:82,

41. Antonio A.T.Cuhh and Vijay Zawar. PityriasisRosea, Harper's Textbook of Paediatric Dermatology. 3rd edition;84.1-84.5

42. Drago F et al: Human herpes virus-like particles in pityriasisrosea lesions: An electron microscopy study. J CutanPathol 2002;29:359.

43. Broccolo F, Careddu AM et al.Additional evidence that Pityriasis is associated with reactivation of human harpes virus-6 and -7.J Invest Dermatol 2005;124:1234-40.

44. Hosokawa H,HorioS,TakiuchiY,MaruyamaN,AsadaY.Naturally occurring T lymphocytotoxic antibody in viral and related skin diseases.ActaDermVenerol 1984;64:275-80.

45. Andrew Blauvelt. PityriasisRosea. Fitzpatrik's Dermatology in General Medicine.8th ed: 362-6

46. J.C. Sterling. Virus Infections.In: Burns T, BreathnachS,Cox N, Griffiths C, eds. Rook textbook of Dermatology.8th ed. Oxford:Blackwell science;2010:33.78-33.81

47. Chuh A, Lee A, Zawar V, Sciallis G, Kempf W. Pityriasisrosea - An update.Indian J DermatolVenereolLeprol 2005;71(5):311-5

48. Neoh CY et al: Characterization of the inflammatorycell infiltrate in herald patches and fullydeveloped eruptions of pityriasisrosea. ClinExpDermatol 35:300, 2010

49. Sharma PK, Yadav TP, Gautam RK et al. Erythromycin in pityriasisrosea: Adouble-blind, placebo-controlled clinical trial. J Am AcadDermatol 2000; 42:241–4.

50. Rasi A1, Tajziehchi L, Savabi-Nasab.Oral erythromycin is ineffective in the treatment of pityriasisrosea. J Drugs Dermatol. 2008 Jan;7(1):35-8.

51. Amer A1, Fischer H. Azithromycin does not cure pityriasisrosea. Pediatrics. 2006 May;117(5):1702-5.

52. Handa S, Sahoo B. Childhood lichen planus: A study of 87 cases. Int JDermatol 2002;41(7):423-7.

53. VenketramanMysore. Lichen Planus and lichenoid dermatoses. Dermatological diseases: A practical approach.1st ed. 59-63

54. Wilson E.On lichen planus. J cutan Med Dis Skin1869;3:117-32

55. Luis-Montoya P, Domínguez-Soto L, Vega-Memije E: Lichen planus in 24 children with review of the literature. PediatrDermatol 2005; 22(4):295-298.

56. Balasubramaniam P, Ogboli M, Moss C. Lichen planus in children: review of 26cases. ClinExpDermatol 2008; 33: 457–9.

57. Pusey WA.Lichenplanus in an infant less than 6 month old.ArchDermatolSyph 1929;19: 671-2

58. MarkR .Pittelkow, MazenSDaoud.Lichen Planus. Fitzpatrik's textbook of dermatology in general medicine 8thedition.Vol 1: 244-255

59. Vega ME et al: Ashy dermatosis versus lichen planuspigmentosus: A controversial matter. Int J Dermatol 31:87, 1992

60. Nail lichen planus in children: clinical features, response to treatment, and long-term follow-up. Arch Dermatol. 2001 Aug;137(8):1027-32. Tosti A, Piraccini BM, Cambiaghi S, Jorizzo M)

61. Usman A, Kimyai-Asadi A, Stiller MJ, et al: Lichenoid eruption following hepatitis B vaccination: first North American case report. PediatrDermatol 2001; 18(2):123-126.

62. Black MM. What is going on in lichen planus? ClinExpDermatol. 1977 Dec;2(4):303-10.

63. Mahood JM: Familial lichen planus. Arch Dermatol.119:292, 1983

64. Sharma R, Maheshwari V. Childhood lichen planus: a report of fifty cases. PediatrDermatol. 1999 Sep-Oct;16(5):345-8.

65. Harper N Price and Andrea L Zaenglein. Lichen Planus and Lichen Nitidus. Harper's Textbook of Paediatric Dermatology 3rdedition.Vol 1: 85.1-85.17.

66. Vasquez Garcia J, Pérez Oliva N, PeireioFerreirós MAM et al. Lichen planusfollicularistumidus with cysts and comedones. ClinExpDermatol 1992; 17:346–8.

67. S.M. Breathnach. Lichen Planus and Lichenoid Disorders .chapter 41.Rook's Textbook of Dermatology.8th ed.Vol3: 41.1-41.28

68. Kanwar AJ, De D. Lichen planus in children. Indian J DermatolVenereolLeprol 2010;76(4):366-72

69. Hogan PA. Papulosquamous disease. In: Schachner LA, Hansen RC, eds. Pediatric Dermatology. 3rd ed. Philadelphia: Elsevier publishers;2003:pp643-81.

70. M.R. Judge1, W.H.I. McLean2 & C.S. Munro.In Tony Burns,Stephenbreathnach, Neil cox, Christopher Griffiths editors..Rooks text book of dermatology. 8th edition. Chapter 19.Disorders of keratinisation.19.76-19.80.

71. Griffiths WA et al. Pityriasisrubrapilaris. ClinExpDermatol. 1980;105-12.

72. Sehgal VN, Jain MK, Mathur RP. Pityriasisrubrapilaris in Indians.Br J Dermatol. 1989 Dec;121(6):821-2.

73. Vanderhooft SL, Francis JS, Holbrook KA, et al: Familial pityriasisrubrapilaris. Arch Dermatol 1995; 131(4):448-453.

74. Finzi AF, Altomare G, Bergamaschini L, Tucci A Pityriasisrubrapilaris and and retinol-binding protein. Br J Dermatol. 1981 Mar;104(3):253-6.

75. vanVoorst Vader PC, van Oostveen F, Houthoff HJ, Marrink J Pityriasisrubrapilaris, vitamin A and retinol-binding protein: a case study.ActaDermVenereol. 1984;64(5):430-2.

76. Allison DS, el-Azhary RA, Calobrisi SD, Dicken CH. Pityriasirubrapilaris in children. J Am AcadDermatol 2002;47:386-9.DECEMBER 2008.

77. Betlloch I et al: Acute juvenile piryriasisrubrapilaris:Asuperantigen mediateddisease PediatrDermatol18:41.12.,0 01.

78. KurzydloAM, Gillespie R. Paraneoplasticpityriasisrubrapilaris in association with bronchogenic carcinoma. Australas J Dermatol. May 2004;45(2):130-2.

79. Evangelou G, Murdoch SR, Palamaras I, Rhodes LE. Photoaggravatedpityriasisrubrapilaris. PhotodermatolPhotoimmunolPhotomed 2005;21:272-4.

80. Melinda Jen and Mary Wu Chang.PityriasisRubraPiaris. Harper's Textbook of Dermatology,3rd edition.Vol.1:83.1-83.8

81. DanielaBruch-GerharzThomas Ruzick .PityriasisRubraPilaris, Fitzpatrik's textbook of Dermatology in general medicine.7th ed. Vol 1: 232-6.

82. Kanegaye JT, Frieden IJ. Lichen striatus: simultaneous occurrence in siblings. Pediatrics 1992; 90: 104-6.

83. Patrone P, Patrizi A, Bonci A, Passarini B. Lichen striatus: studio clinicoedistologico di 8 casi. G Ital DermatolVenereol 1990; 125: 267-70.)

84. Kennedy ,RogersM,Lichenstriatus.Paeiatr Dermatology1996;13:95-9

85. Toda K,Okamoto H, HorioT. Lichen Striatus. Int J Dermatol 1986;70: 200-6.

86. Zhang Y, McNuttNS. Lichenstriatus. Histological, immune histochemical and ultrastructural study of 37 cases. JCutan Pathol2001;28: 65-71.

87. HappleR.Transposable elements and the lines of blaschko: A new perspective,Dermatology 2002;204;4-7.

88. Taïeb A, Youbi E, Maleville J. Lichen striatus: a Blaschko linear acquired inflammatory skin eruption. J Am AcadDermatol 1991; 25: 637-42.

89. Gelmetti C, Cerri D,Gianotti R et al.Bilateral multiple lichen stiatus.PaeiatrDermatolNews(Bari)1998;7:97.

90. GoskowickMo, EichenfieldLF. Onychodystrophy with lichen striatus. PaediatrDermatol 1994;11: 282-3

91. Alain Taeib and EouardGrosshans. Lichen striatus. Harper's Textbook of Dermatology. 3rd edition. Vol 1: 86.5

92. Tosti A, Peluso AM, Misciali C, Cameli N. Nail lichen striatus: clinical features and long-term follow-up of five patients. J Am AcadDermatol 1997; 36: 908-13.

93. Bernard LA, Eichenfield LF. Eczematous and papulosquamous disorders. In:Eichenfield LF, Frieden IJ, Esterly NB, eds. Neonatal Dermatology, 2nd ed.Philadelphia: Elsevier; 2008: pp236-44.

94. M M Huda, Pranabkumarsaha. Pattern of dermatosis among paediatric patients attending a medical college hospital in the North eastern of India. Indian J of Dermatol 2004;49(4) 189-191.

95. Johnson M-LT, Roberts J. Prevalence of Dermatological Diseases Among Persons 1–74Years of Age. Washington DC: US Department of Health Education, NationalCenter for Health Statistics, 1978: PHS 79–1660.

96. Ruiz Maldonado R ,Lopez Martinez R,Chavarria P et al.Pityriosporumovale in infantile seborrheic dermatitis.PaediatrDermatol 1989;6:16-20.

97. Tager A. Seborrhoeic dermatitis in acute cardiac disease. Br J Dermatol 1964; 76:367–9.

98. Barba A, Piubello W, Vantini I et al. Skin lesions in chronic alcoholic pancreatitis.Dermatologica 1982; 164: 322–6.

99. Cohen BA. Papulosquamous eruptions. In: Cohen BA, eds. PediatricDermatology. 3rd ed. Philadelphia: Elsevier; 2005.pp67-99

100. Holden CA, Berth-Jones J. Eczema, lichenification, prurigo and erythroderma.In: Burns T, Breathnach S, Cox N, Griffiths C, eds. Rook textbook ofDermatology.7th ed. Oxford: Blackwell science; 2004: pp17.10-17.15.

101. Oranje AP, van Joost T, van Reede EC, Vuzevski VD, Dzoljic-Danilovic G, ten Kate FJ, Stolz E. Infantile seborrheic dermatitis. Morphological and immunopathological study. Dermatologica. 1986;172(4):191-5.

102. Carlo M Gelmetti and Ramon Grimalt. Infantile seborrheic dermatitis. Harper's textbook of dermatology.3rd d. vol 1.chapter 35; 35.5-35.6

103. Pittelkow MR, Daoud MS. Lichen nitidus. In: Wolff K , Goldsmith LA, KatzSI, Gilchrest BA, Paller AS, Leffell DJ, eds. Fitzpatrick's Dermatology inGeneral Medicine 7th ed. NewYork: McGraw Hill; 2007: pp255-8

104. Hazen HH: Syphilis and skin diseases in the American Negro: Personal observations. Arch DermatolSyphilol 31:316, 1935

105. LakshmishaC,ThappaDM.Generalised lichen nitidus with downs syndrome.JEurAcaddermatol Venerol2006;20(9):1156-7

106. Lestringant GG et al: Coexistence of atopic dermatitis and lichen nitidus in three patients. Dermatology 192:171, 1996Pediatr Dermatol. 1999 Sep-Oct;16(5):406-7.

107. Bercedo A, Cabero MJ, García-Consuegra J, Hernado M, Yaez S, Fernández-LlacaH.Generalized lichen nitidus and juvenile chronic arthritis: an undescribed association.PediatrDermatol. 1999 Sep-Oct;16(5):406-7.

108. Sarveswari KN, Yesudian P. The conundrum of parapsoriasis versus patchstage of mycosis fungoides. Indian J DermatolVenereolLeprol 2009;75(3):229-35.

109. Tasić S1, Stojanović S, Poljacki M, Vucković N. . Small-plaque parapsoriasis: case report.Med Pregl. 2001 May-Jun;54(5-6):278-81.

110. Gary S. Wood, Chung-Hong Hu, & Rosemarie Liu .Parapsoriasis and PityriasisLichenoides.Fitzpatrik's textbook of dermatology in general medicine.8th ed. 285-295.

111. Neisser A. ZurFrage der lichenoidenEruptionen. VerhDtschDermatolGes 1894;4:495-9.

112. Jadassohn J. Ube reineingenartigespsoriasiformes undlichenoidesExanthem. VerhDtschDermatolGes 1894;4:524-9.

113. Gelmetti C, Rigioni C, Alessi E, et al.: Pityriasislichenoidesin children: a long term follow-up of eighty-nine cases. J AmAcadDermatol 23: 473-478, 1990.

114. ZlatkovNB,AndrewVC.Pityriasislichenoides and toxoplasmosis.Br J Dermatol 1977;87:1114-6

115. Hood AF, Mark EJ. Histopathological diagnosis of pityriasislichenoidesetvarioliformisacuta and its co-relation. Arch Dermatol 1982;118:478-82.

116. Longly J, Demar L, Feinstein RP, Miller RL, Silvers DN. Clinical and histopathological features of pityriasislichenoides et varioliformisacuta in children. Arch Dermatol 1987;123:1335-9.

117. Devang G. Patel, MDGeorge Kihiczak, MD,Robert A. Schwartz, MD, MPH,Camila K. Janniger, MD,W. Clark Lambert, MD, PhD PityriasisLichenoides.Paediatr Dermatol.2000(66):17-23.

118. Whittaker SJ, MacKie RM. Cutaneous lymphomas and lymphocyticinfiltrates. In: Burns T, Breathnach S, Cox N, Griffiths C, eds. Rook textbookof Dermatology.7th ed. Oxford: Blackwell science; 2004:pp54.25-54.27.

119. ChaliAdssaf,WolframS terry .CutaneousLymphoma.Fitzpatrik's textbook of Dermatology 7th ed.vol 1.145:1392.

120. El Shabrawi-Caelen L, Kerl H, Cerroni L. Lymphomatoid papulosis: reappraisal of clinicopathologic presentation and classification into subtypes A, B, and C. *Arch Dermatol*. Apr 2004;140(4):441-7.

121. Willemze R, Beljaards RC. Spectrum of primary cutaneous CD30 (Ki-1)-positive lymphoproliferative disorders. A proposal for classification and guidelines for management and treatment. *J Am Acad Dermatol*. Jun 1993;28(6):973-80.

122. Nijsten T1, Curiel-Lewandrowski C, Kadin ME. Lymphomatoidpapulosis in children: a retrospective cohort study of 35 cases. Arch Dermatol. 2004 Mar;140(3):306-12.

123. . Seetharam KA, Zawar V. Viral infections and exanthems. In: Inamadar AC,Sacchidanand S, eds. Textbook of Pediatric Dermatology, 1st ed. New Delhi:Jaypee publications;2009:pp156-8

124. Amy S.Paller, Anthony J Mancini. Exanthematous disease of childhood.Hurwitz clinical paediatric dermatology 4th ed.16:380-382.

125. Lima DA, Rocha DM, Miranda MFR. Gianotti-Crosti syndrome: clinical,laboratorial features, and serologic profiles of 10 cases from Belém, State ofPara, Brazil. An. Bras. Dermatol 2004;79(6):699-707.

126. Thappa DM. Common skin problems in children. In J Pediatr 2002;69:701-06.

127. Federman DG, Reid MC, Feldman SR, Greenhoe J, Kirsner RS. The primary care provider and the care of skin disease. Arch Dermatol 2001;137:25-9.

128. Gül U, Cakmak SK, Gönül M, Kiliç A, Bilgili S. Pediatric skin disordersencountered in a dermatology outpatient clinic in Turkey. Pediatr Dermatol.2008;25(2):277-8

129. M. D. Al-Mendalawi and J.G. Ibrahim .Pattern of dermatoses in Iraqi children.EMHJ.2012 ;18(4):365-371.

130. Vetrichevvel TP, Thappa DM. A clinical study of papulosquamous diseases inchildren. In 34th national conference of Indian association of dermatologists,venereologists and leprologists; 2006 Feb 1-5, Hyderabad, Andhra Pradesh,India; 2006. p.333 (Abstract no. P241)

131. KalyaniVasramvadite.Clinical and epidemiological stuy of papulosquamous disorders in children attendin dermatology op. rajiv Gandhi university of health, sciences.2011

132. FawziaFaragMostafa , Aida Abdel Hamid Hassan,Mohamed Ibrahim Soliman, Amani Nassar, Randa Hassan Deabes Prevalence of skin diseases among infants and children in Al Sharqia Governorate, Egypt.EDOJ June 2012. Vol 8 No 1: 4,

133. Tollefson MM, Crowson CS, McEvoy MT, MaraditKremers H. Incidence ofpsoriasis in children: A population-based study. J Am AcadDermatol .2010;62(6):979-87.

134. Mercy et al. Clinical manifestations of paediatric psoriasis: Results of a multi center study in the united states.PediatrDermatol. 2013 July ; 30(4): 424–428.

135. Nyfors A, Lemholt K. Psoriasis in children. A short review and a survey of245 cases. Br J Dermatol 1975;92(4):437-42.

136. Seyhan M1, Coşkun BK, Sağlam H, Ozcan H, Karincaoğlu Y. Psoriasis in childhood and adolescence: evaluation of demographic and clinical features. Pediatr Int. 2006 Dec;48(6):525-30.

137. Egwin AS, Martis J, Bhat RM, Kamath GH, Nanda KB. A clinical study on pityriasisrosea. Indian J Dermatol 2005;50:136-8

138. Gündüz O, Ersoy-Evans S, Karaduman A. Childhood pityriasisrosea. PediatrDermatol 2009;26(6):750-1

139. Hendricks AA, Lohr JA. PityriasisRosea in Infancy. Arch Dermatol1979;115(7):896-7

140. . Nanda A, Al-Ajmi HS, Al-Sabah H, Al-Hasawi F, Alsaleh QA. Childhood lichen planus: a report of 23 cases. PediatrDermatol 2001;18(1):1–4.117

141. NonurkaEN.Lichenplanus in African Children. a study of 13 cases.Paediatrdermatol 2007;24:495-8.

142. . Kumar V, Garg BR, Baruah MC, Vasireddi SS. Childhood lichen planus. JDermatol 1993;20(3):175-7.

143. Patrizi A, Neri I, Fiorentini C, Bonci A, Ricci G. Lichen striatus: clinical andlaboratory features of 115 children. PediatrDermatol 2004;21(3):197-204

144. Taniguchi Abagge K, ParolinMarinoni L, Giraldi S, Carvalho VO, de OliveiraSantini C, Favre H. Lichen striatus: description of 89 cases in children. PediatrDermatol 2004;21(4):440-3

145. Wananukal s et al.Malassezia furfur in infantile seborrheic dermatitis.Asian Pac J Allergy immunol.2005;23:101-5.

146. Foley P, Zuo Y, Plunkett A, Merlin K, Marks R. The frequency of commonskin conditions in preschool-aged children in Australia: seborrheic dermatitis and pityriasiscapitis (cradle cap). Arch Dermatol 2003;139(3):318-22

147. Yang CC, Shih IH, Lin WL, Yu YS, Chiu HC, Huang PH, et al. Juvenilepityriasisrubrapilaris: Report of 28 cases in Taiwan. J Am AcadDermatol 2008;59(6):943-8

148. Gelmetti C, Schiuma AA, Cerri D, Gianotti F. Pityriasisrubra pilaris in childhood: a long-term study of 29 cases. Pediatr Dermatol 1986;3:446-51.

149. Zapata Román G, Ruiz-Maldonado R, Tamayo L. Lichen nitidus in children.Study of 17 cases. Med CutanIberoLat Am 1981;9(5):323-8

150. N. A. Lapins, C. Willoughby, and E. B. Helwig, “Lichen nitidus. A study of forty-three cases,” Cutis, vol. 21, no. 5, pp. 634–637, 1978.

151. Nair PS. A clinical and histolopathological study of pityriasis lichenoides. Indian J DermatolVenereolLeprol 2007;73:100-2

152. Longly J, Demar L, Feinstein RP, Miller RL, Silvers DN. Clinical and histopathological features of pityriasislichenoides et varioliformisacuta in children. Arch Dermatol 1987;123:1335-9.

APPENDIX 1

PROFORMA

Name:…….....Sl. No:……

Age:…. OP No……....

Sex -M/F Date:……..

Informant:……..:

Address:

PRESENTING COMPLAINT :-

H/o skin lesions

H/o itching

H/o other symptoms

HISTORY OF PRESENTING ILLNESS :

SKIN LESIONS

Mode of onset :Sudden/Rapid/Insidious

Distribution :

Itching : Mild/Moderate/Severe

Seasonal Variation

Aggravating / Relieving factors

Remissions / Exacerbations

OTHER SYMPTOMS:Fever/joint pain/others

OTHER SITES:

Treatment History: Topical/ Systemic/ Others

Past History : Similar illness / Any other contributing illness

Family History : Similar illness / Any other contributing illness**.**

Personal History : Diet:Veg / Mixed

Appetite – Good / reduced

Sleep – Sound /disturbed

Bowel – Regular / irregular

Bladder – Regular /variability.

EXAMINATION :

General Physical Examination:

Height: Weight:

Temp: Febrile / Afebrile. Resp Rate- / min

Pulse: / min. BP- mmHg

Built: Poor / Moderate / Well.

Nourishment: Poor / Moderate / Well

Cyanosis: Present / Absent Icterus: Present / Absent Pallor: Present /Absent

Pedal oedema: Generalised Lymphadenopathy:

Cutaneous Examination ;

Types of Lesions:

Pattern:

Distribution: Symmetrical / Asymmetrical

Scales : Fine/ Coarse

Colour

Size

Adherence: Loosely/ Firmly

Special characters:

OTHER SITES

Palms

Soles

Hairs

Nails

Mucosa membrane: Conjunctival /Genital/Oral/ Anal/Nasal

Systemic Examination :-

P/A

RS

CVS

CNS

Provisional Diagnosis:

Associated diseases if any: (A) Cutaneous (B) Systemic

Investigations

Blood: Hb% - TC-DC-ESR-

Urine routine KOH

Skin biopsy

FINAL DIAGNOSIS:

CONSENT FORM

Dr. Preetha Prasad, post graduate student in the Department of Dermatology, Venereology and Leprosy, Coimbatore medical college hospital is conducting a study on **"A CLINICAL STUDY ON PAPULOSQUAMOUS DISORDERS IN CHILDREN LESS THAN 12 YEARS"**. The study and test procedures were explained to me clearly. I hereby give my consent to participate my child in this study and to give blood sample and to take biopsy if needed. The data obtained herein may be used for research and publication.

Place:

Date: Signature of the Parent

(Name:)

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் தோல் மருத்துவ துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவி அவர்கள் மேற்கொள்ளும் "**குழந்தைகளுக்கு செதில் உறிதல் நோய்**" குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் என் குழந்தையை பரிசோதனை செய்ய முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

என் குழந்தைக்காக செய்யும் இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்தில் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :

KEY TO MASTER CHART

Sl No	-	Serial number
yrs	-	Years
m	-	months
F	-	Female
M	-	Male
Fe	-	Fever
ST	-	Sore throat
Oz	-	Oozing
Pu	-	Papule
Pl	-	Plaque
Pa	-	Patch
Ma	-	Macule
Fs	-	Fissure
Ps	-	Pustules
A	-	Annular
L	-	Linear
Pf	-	Perifollicular
G	-	Grouped
W	-	White
Fi	-	Fine
SW	-	Silvery white
PC	-	Peripheral collarete

Y	-	yellow
Gr	-	Greasy
Th	-	Thickened
As	-	Auspitz sign
Kb	-	Koebnerisation
Hp	-	Herald patch
PV	-	Psoriasis vulgaris
GP	-	guttate psoriasis
FP	-	Flexural psoriasis
SP	-	Scalp psoriasis
PPS	-	palmoplantar psoriasis
FP	-	flexural psoriasis
PUPS	-	Pustular Psoriasis
PR	-	Pityriasisrosea
LP	-	Lichen planus
GLP	-	generalised LP
LLP	-	Linear lichen planus
SD	-	seborrhoeic dermatitis
LS	-	Lichen striatuus
PRP	-	Pityriasis rubrapilaris
LN	-	Lichen nitidus
PLEVA	-	pityriasis lichenoides et varioliformis acuta
PLC	-	pityriasis lichenoides chronic
PaPS	-	Parapsoriasis

SL No.	NAME	Age/Sex	CLINICAL FEATURES																								DIAGNOSIS
			HISTORY					MORPHOLOGY																			
											SITES															
			AGE OF ONSET	Month of onset	PRURITIS	OTHER SYMPTOMS	FAMILY HISTORY	LESION	PATERN	NECK	FACE	CHEST	ABDOMEN	BACK	UPPERLIMB	LOWERLIMB	FLEXURES	PALMS	SOLES	NAILS	SCALP	ORAL MUCOSA	GENITALS	SCALING	TYPE OF SCALES	SPECIAL FEATURES	
1	Rani	5yrs/F	4yrs	february	+	-	-	Pu,Pl	A	-	-	+	+	-	+	+	-	-	-	-	+	-	-	+	SW	AS.Kb	PV
2	Eeswari	6yrs/f	6yrs	December	-	Fe	-	pa,pu	-	-	+	-	-	-	+	+	-	-	-	-	-	-	-	+	PC	HP	PR
3	Amritha	3m/F	3m	March	-	-	-	pa	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	+	Y,Gr	Kb	SD
4	Parvathi	4yrs/F	4yrs	April	+	-	-	pu,pl	L	-	-	-	-	-	-	+	-	-	-	+	-	-	-	+	Fi,W	Kb	LLP
5	Maari	11yrs/F	11yrs	March	+	-	-	pa	A	+	-	-	-	+	+	+	-	-	-	-	-	-	-	+	PC	-	PR
6	Goutham	5yrs/M	4yrs	December	+	-	-	pu	-	-	-	+	+	+	-	-	-	-	-	-	-	+	-	+	Fi,W	Kb	LP
7	Raman	11yrs/M	12yrs	January	+	-	-	pu	L	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	Fi	-	LS
8	Pavithra	11yrs/F	12yrs	January	+		+	Pl	A	-	-	-	-	-	+	+	-	-	-	+	+	-	-	+	SW	AS,Kb	PV
9	Sankar	7yrs/M	7yrs	february	+	-	-	pu	L	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	Fi	-	LS
10	Roja	8yrs/F	7yrs	March	+	-	-	pu	L	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	Fi	Kb	LLP
11	Bharath	8yrs/M	7yrs	October	+	-	-	pu	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+ +	Fi Fi	-	PRP LN
12	Aaditya	5yrs/M	5yrs	April	-	-	-	pa	A	+	+	+	-	+	-	-	-	-	-	-	-	-	-	+	PC	HP	PR
13	Jaiharini	8yrs/F	8yrs	February	+	-	-	pu,pl	A	-	-	+	+	+	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
14	Sabira	11yrs/F	11yrs	December	+	Fe	-	Pu,Pl	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	+	SW	AS,Kb	GP
15	Jennifer	7yrs/F	5yrs	December	+	-	-	PU	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	SW	AS,Kb	GP
16	Arun	6yr/M	6yrs	December	-	-	-	pu	G		-	-	-	-	+	-	-	-	-	-	-	-	-	+	Fi	-	LN
17	Chithra	7yrs/F	7yrs	September	+	oz	-	pu	L,G	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	Fi	-	LS with Eczema
18	Thilakaraj	5yrs/M	4yrs	May	+	-	-	pu,pl	-	-	-	-	-	-	+	+	+	-	-	-	-	+	-	+	Fi	Kb	GLP
19	Kaasi	4yrs/M	4yrs	May	-	-	-	pa	-	+	+	+	-	-	-	+	+	-	-	-	-	-	-	+	PC	-	IPR
20	Vaani	11yrs/F	11yrs	January	+	Fe	-	Pl	A	-	-	-	-	-	-	+	-	-	+	+	-	-	-	+	SW	AS	PV
21	Paramjothi	3yrs/M	3yrs	December	-	-	-	pu	A	-	-	+	-	+	+	-	-	-	-	-	-	-	-	+	Fi	-	PRP with LS
22	Kabil	10yrs/M	8yrs	January	+	-	-	pu,pl	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	Fi	Kb	LP
23	Thanushri	6yrs/F	5yrs	December	+	-	-	Pu,Pl	A	-	-	-	-	-	+	-	-	-	+	-	+	-	-	+	SW	AS, Kb	PV
24	Nageswari	6m/f	6m	March	-	-	-	Pl	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	FP
25	Manimekala	3yrs/F	3yrs	December	+	-	-	pu	L	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	Fi	-	LS
26	Harish	9yrs/M	9yrs	january	-	Fe, ST	-	pu,pa	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	PC	Ez	PR
27	Ilakiya	11yrs/F	10yrs	September	+	-	-	Pu,Pl	A	-	-	+	-	+	+	+	-	-	-	+	-	-	-	+	SW	AS,Kb	PS
28	Ganesh	11yrs/m	11yrs	March	-	Fe	-	pa	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	PLEVA

29	Kavitha	5yrs/F	5yrs	April	+	-	-	Pl	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	SW	AS	SP
30	Malathi	5yrs/F	5yrs	february	-	-	-	pu	L	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	Fi	-	LS
31	Padma	7yrs/F	7yrs	february	+	-	-	pa	A	-	-	+	+	+	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
32	kalayarasi	6yrs/F	6yrs	March	-	-	-	pu	G,Pf	-	-	-	-	-	+	+	-	-	-	-	+	-	-	+	Fi	-	PRP
33	Ramkumar	4m/M	4m	April	-	-	-	pa	-	-	+	+	-	-	-	-	+	-	-	-	+	-	-	+	Y,Gr	-	SD
34	Gunasekar	8yrs/M	7yrs	february	+	-	-	pa	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	+	Fi,Br	-	PLC
35	Suresh	11yrs/M	10yrs	April	+	-	-	pu	G,Pf	-	-	+	+	+	+	+	-	+	+	-	-	-	-	+	Fi	-	PRP
36	Gaurav	4yrs/M	4yrs	June	-	-	-	PL,Fs	L	-	-	-	+	-	-	-	-	-	+	-	-	-	-	+	Th Fi	-	PPS
37	Mathiyan	4yrs/M	4yrs	october	-	-	-	Pl,Fs	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	Th	-	PPS
38	Paimala	8yrs/F	6yrs	May	+	-	-	Pu,Pl	A	-	-	-	-	+	+	-	-	-	-	+	+	-	-	+	SW	AS.Kb	PV
39	Anandi	11yrs/F	10yrs	February	+	-	-	Pu,pl	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	Fi	Kb	LP
40	karthik	11yrs/M	11yrs	August	-	-	-	Pl,Fs	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	+	Th	-	PPS
41	Poorni	4yrs/F	4yrs	March	-	-	-	Pu	L	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	Fi	-	LS
42	Loganathan	9yrs/M	9yrs	December	-	-	-	pu,pl	-	-	-	+		+	+	+	-	-	-	-	-	-	-	+	Fi	-	LP
43	Pandi	2yr/M	-	April	+	-	-	pu	G,L	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	Fi	-	LS
44	Maalavika	6yrs/F	6yrs	June	+	-	-	pu,pl	-	-	-	+	+	+	+	+	-	-	-	+	-	-	-	+	Fi	Kb	GLP
45	Poomani	2yrs/F	2yrs	November	-	Fe	-	pu,pa	A	-	-	-	-	+	+	+	-	-	-	-	-	-	-	+	PC	-	PR
46	Madhan	8yrs/M	7yrs	February	+	Fe	+	Pl,F	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	+	Th SW	AS	PV
47	Rajeswari	10yrs/F	10yrs	January	+	-	-	Pa	A	-	-	+	+	+	-	-	-	-	-	-	-	-	-	+	PC	HP	PR
48	Ramalingam	8yrs/M	8yrs	April	+	-	-	Pu	-	-	-	+	+	-	+	-	-	-	-	-	-	-	-	+	SW	AS,Kb	GP
49	Priya	11yrs/F	11yrrs	March	-	Fe,ST	-	pu,pa	-	+	+	+	+	+	-	+	-	-	-	-	-	-	-	+	PC	-	PR
50	Vasudev	8yrs/M	7yrs	December	+	-	-	Pu	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	+	SW	AS	GP
51	Thangavel	4yrs/M	4yrs	April	-	-	-	pu	-	-	-	-	-	-	+	+	-	-	-	-	-	-	+	+	Fi	-	LN
52	Vaishnav	8yrs/M	7yrs	February	-	-	-	Pu,Pl	A	-	-	-		+	+	-	-	-	-	-	-	-	-	+	SW	AS, Kb	PV
53	Saavanth	9yrs/M	4yrs	September	-	-	-	pu	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	FI	Kb	LN
54	Vasanthi	9yrs/F	9yrs	february	+	-	-	pu,pl	-	-	+	-	-	+	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
55	Aadiraj	3yrs/M	3yrs	May	+	-	-	pu	-	-	-	+	-	-	+	+	-	-	-	-	-	+	-	+	Fi	Kb	LP
56	Selvi	8yrs/F	8yrs	May	+	-	-	pu	L	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	Fi	-	LS
57	Arjun	11yrs/M	11yrs	July	-	-	-	Pl	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	Fi	-	FP
58	Madhusri	11yrs/F	11yrs	May	-	Fe, ST	-	pu,pa	-	+	+	-	-	-	-	-	+	-	-	-	-	-	-	+	PC	-	IPR
59	Lekshmi	10yrs/f	9yrs	January	-	-	-	Pu,Pl	-	-	-	+	+	+	+	+	-	-	-	-	-	-	-	+	SW	-	PUPS
60	Thayyal	7yrs/F	7yrs	December	+	-	-	pa	A	-	-	-	+	+	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
61	Ajith	4yrs/M	3yrs	January	+	-	-	pu,pl	-	-	-	-	-		-	+	-	-	-		-	-	-	+	Fi	-	LP
62	Muthu	2yrs/M	2yrs	March	+	-	-	Pu	L	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	Fi	-	LS
63	Anitha	11yrs/F	11yrs	December	+	-	-	Ma,Pa	-	-	+	+	+	+	+	+	-	-	-	-	-	-	-	+	Fi	-	PaPS
64	Narmada	7yrs/F	6yrs	October	+	-	-	Pa	-	-	-	+	+	+	+	+	-	-	-	-	-	-	-	+	Fi	kb	LN
65	Lakshman	11yrs/M	11yrs	May	-	-	-	Pl,pu	G,Pf	-	-	-	-	+	+	-	-	+	+	-	-	-	-	+	Fi		PRP
66	Janani	10yrs/F	9yrs	December	-	-	-	Pl	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	Th	-	PPS
67	Rahul	12yrs/M	12yrs	February	-	-	-	Pu,Pa	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	PC	-	PR

68	Pradeep	11yrs/M	7yrs	February	-	-	-	Ma,Pa	-	+	+	+	+	+	+	+	-	-	-	-	-	-	-	+	Fi	-	PaPS
69	Satheesh	8yrs/M	7yrs	March	+	-	-	pu,Pl	-	-	-	-	+	+	+	+	-	-	-	-	-	-	+	+	Fi	Kb	GLP
70	Maalini	10yrs/F	9yrs	May	+	-	-	Pl,Fs	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	Th	-	PPS
71	Balamurugan	6m/M	6m	May	-	-	-	Pl	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	Y,Gr	-	SD
72	Charles	8yrs/M	7yrs	February	+	-	-	Pu,Pl	A	-	-	+	-	+	+	-	-	-	-	-	-	-	-	+	SW	AS,Kb	PV
73	Dhanesh	9yrs/M	9yrs	July	+	-	-	Pu,Pl	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	Fi	Kb	LP
74	Ramya	8yrs/F	8yrs	April	+	-	-	Pu	L	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	Fi	-	LS
75	Veni	8m/F	8m	January	-	-	-	Pl	-	-	-	-	-	-	-	+	+	-	-	-	-	-	+	+	-	-	FP
76	Jagadeep	11yrs/M	11yrs	September	-	-	-	Pu	G,Pf	-	-	+	+	+	-	-	-	-	-	-	-	-	-	+	Fi	-	PRP
77	Dhanesh	10yrs/M	8yrs	September	+	-	-	Pu	L	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	Fi	KB	LLP
78	Gauri	4yrs/F	4yrs	October	-	-	-	Pa	A	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	PC	HP	PR
79	Krithika	5yrs/M	4yrs	October	+	-	-	Pu,Pl	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	Fi	kb	LP
80	Sahaana	4yrs/M	3yrs	October	+	-	-	Ma,Pa	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	+	Fi,W	-	PaPS
81	Vishnu	3m/M	3m	october	-	-	-	Pl,Pa	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	Y,Gr	-	SD
82	Junaidh	2yrs/M	2yrs	january	+	-	-	Pa	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	+	PC	-	PR
83	Revathi	6yrs/F	6yrs	November	-	-	-	Pa,Pl	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	Fi	-	LP
84	Bharath	4yrs/M	4yrs	September	-	-	-	Pa	A	-	-	-	-	+	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
85	Lakshan	3m/M	3m	September	-	-	-	Pa	-	+	+	+	-	+	+	+	-	-	-	-	+	-	-	+	Y,Gr	-	SD
86	Kapilan	11yrs/M	12yrs	December	+	-	-	Pa	A	+	-	+	-	+	-	-	-	-	-	-	-	-	-	+	PC	-	PR
87	Dharani	6m/F	6m	March	-	-	-	Pl	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	Y,Gr	-	SD
88	Madhav	6yrs/M	5yrs	July	-	-	-	Ps,Pl	A	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	PUPS
89	Balaji	11yrs/M	11yrs	February	-	-	-	Pa	A	-	-	+	-	+	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
90	Mithila	5m/F	5m	April	-	-	-	Pa	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+		Y,Gr		SD
91	Anusree	11yrs/F	11yrs	April	+	-	-	Pu,Pl	-	-	-	+	+	+	-	+	-	-	-	+	-	-	-	+	Fi	Kb	GLP
92	Iqbal	2m/M	2m	May	-	-	-	Pa	-	-	+	-	-	+	-	-	-	-	-	-	+	-	-	+	Y,Gr	-	SD
93	Stalin	5yrs/M	5yrs	May	+	-	-	Pl	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	+	Fi	kb	LP
94	Katherine	6m/F	6m	January	-	-	-	Pa	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	Y,Gr	-	SD
95	Saranya	10yrs/F	10yrs	January	+	-	-	Pu,Pl	A	-	-	-	-	-	+	+	-	-	-	-	-		-	+	SW	AS,Kb	PV
96	Saravanan	9yrs/M	8yrs	April	-		-	Pl	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	+	Fi	-	LP
97	Babu	7m/M	7m	December	-	-	-	Pa	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	Y,Gr	-	SD
98	Ali	1yr/M	1yr	August	+	-	-	Pa	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	+	PC	-	LPR
99	Alice	11yrs/F	10yrs	December	+	-	-	Pu,Pl	A	-	-	-	-	+	+	+	-	-	-	-	-	-	-	+	SW	AS,Kb	PV
100	Parveen	9yrs/F	9yrs	March	+	-	-	Pu,Pl	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	Fi	-	LP
101	Meena	3yrs/F	3yrs	July	+	Fe	-	Pa	-	+	-	+	-	+	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
102	Sankar	10m/M	10m	November	-	-	-	Pl		-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	W	-	FP
103	Gayathri	2yrs/F	2yrs	November	-	-	-	Pu	L	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	Fi,W	-	LS
104	venkat	7yrs/M	7yrs	June	-	-	-	Pa	-	-	-	+	+	-	+	-	-	-	-	-	-	-	-	+	PC	HP	PR
105	Kala	4m/F	4m	January	-	-	-	Pa	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	+	Y,Gr	-	SD