

**“A CLINICO-AETIOLOGICAL STUDY OF DIAPER AREA
DERMATOSES IN CHILDREN”**

By

Dr. BINDUSHREE R. M.B.B.S.



DISSERTATION SUBMITTED TO THE SRI DEVARAJ URS ACADEMY OF
HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE
DEGREE OF

**DOCTOR OF MEDICINE
IN
DERMATOLOGY, VENEREOLOGY AND LEPROSY**

Under the Guidance of
Dr. RAGHAVENDRA B.N. M.D
Associate Professor



**DEPARTMENT OF DERMATOLOGY, VENEREOLOGY
AND LEPROSY,**

**SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101**

APRIL 2016

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled “**A CLINICO-AETIOLOGICAL STUDY OF DIAPER AREA DERMATOSES IN CHILDREN**” is a bonafide and genuine research work carried out by me under the guidance of **Dr. RAGHAVENDRA B N**, M.D. Associate Professor, **Dept of DERMATOLOGY, VENEREOLOGY, LEPROSY**, Sri Devaraj Urs Medical College, Kolar.

Date:

Dr. BINDUSHREE R.

Place : Kolar

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND
RESEARCH, TAMAKA, KOLAR, KARNATAKA**

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation “**A CLINICO- AETIOLOGICAL
STUDY OF DIAPER AREA DERMATOSES IN CHILDREN**” is a bonafide
and genuine research work carried out by **Dr. BINDUSHREE R** in partial
fulfillment of the requirement for the degree of **M.D** in **DERMATOLOGY,
VENERELOGY AND LEPROSY**.

SIGNATURE OF THE GUIDE

Dr.RAGHAVENDRA B.N. M.D,

Associate Professor

Department of Dermatology, Venereology
and Leprosy,

Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date :

Place : Kolar

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND
RESEARCH, TAMAKA, KOLAR, KARNATAKA**

CERTIFICATE BY THE CO-GUIDE

This is to certify that the dissertation “**A CLINICO- AETIOLOGICAL
STUDY OF DIAPER AREA DERMATOSES IN CHILDREN**” is a bonafide
and genuine research work carried out by **Dr. BINDUSHREE R** in partial
fulfillment of the requirement for the degree of **M.D** in **DERMATOLOGY,
VENERELOGY AND LEPROSY**.

SIGNATURE OF THE CO-GUIDE

Dr.K.N.V.PRASAD M.D,

Professor & HOD,
Department of Paediatrics,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date :

Place : Kolar

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND
RESEARCH, TAMAKA, KOLAR, KARNATAKA**

**ENDORSEMENT BY THE HOD,
PRINCIPAL / HEAD OF THE INSTITUTION**

This is to certify that the dissertation “**A CLINICO- AETIOLOGICAL
STUDY OF DIAPER AREA DERMATOSES IN CHILDREN**” is a bonafide
research work done by **Dr. BINDUSHREE R.** under the guidance of
Dr.RAGHAVENDRA B.N. M.D Associate Professor, Department Of
Dermatology, Venereology & and Leprosy Sri Devaraj Urs Medical College,
Kolar, in partial fulfillment of University regulation for the award “**M.D. IN
DERMATOLOGY, VENEREOLOGY AND LEPROSY.**

”.

Dr.RAJASHEKAR T.S. MD

Professor & HOD

Department Of Dermatology, Venereology
& Leprosy

Sri Devaraj Urs Medical College,
Tamaka, Kolar

Date:

Place:Kolar

Dr. RANGANATH.B.G MD

Principal,

Sri Devaraj Urs Medical College
Tamaka, Kolar

Date:

Place: Kolar

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND
RESEARCH, TAMAKA, KOLAR, KARNATAKA**

ETHICS COMMITTEE CERTIFICATE

This is to certify that the ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved, **Dr. BINDUSHREE R** student in the Department of Dermatology, Venereology and Leprosy at Sri Devaraj Urs Medical College, Tamaka, Kolar to take up the dissertation work titled “**A CLINICO-AETIOLOGICAL STUDY OF DIAPER AREA DERMATOSES IN CHILDREN**” to be submitted to Sri Devaraj Urs Academy of Higher Education and Research, Kolar.

Date:

Signature of Member Secretary

Place: Kolar

Ethical Committee

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND
RESEARCH, TAMAKA, KOLAR, KARNATAKA**

COPY RIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic /research purpose.

Date :

Place : Kolar

Dr. BINDUSHREE R.

ACKNOWLEDGEMENT

I thank the almighty for showering his blessings on me.

*My deep felt gratitude to my **Dear Parents** and my **Sister** for their constant support and encouragement without whom I would not be the person I am today.*

*I am deeply indebted and grateful to my guide **Dr. Raghavendra B.N**, MD, Associate professor, Department of Dermatology, Venereology and Leprosy, Sri Devaraj Urs Medical College, Tamaka, Kolar, for his able guidance, timely advice and constant encouragement throughout the period of the study.*

*I sincerely thank **Dr. Rajashekar T.S**, M.D, Professor & Head, Department of Dermatology, Venereology and Leprosy, Sri Devaraj Urs Medical College, for his frank, timely advice and relentless support during the preparation of this study and my course.*

*I would like to express my sincere gratitude to my co- guide **Dr. K N V Prasad**, MD, Professor and Head, Department of pediatrics, Sri Devaraj Urs Medical College, for his constant help and expert advice during the course of this study.*

*I thank **Dr. Rajesh G**, **Dr. Satish S**, **Dr Suresh**, Department of Dermatology, Venereology and Leprosy, for their constant encouragement and support.*

I am also thankful to my postgraduate colleagues, seniors , juniors and friends for their constant motivation and co-operation.

Last but not the least, I thank all my patients involved in this study, without whose co-operation, this study would not have been possible.

Dr. BINDUSHREE R.

ABSTRACT

BACKGROUND: Diaper area dermatoses is a broad term used to describe various skin conditions that can occur in diaper area. The anatomical area for diaper dermatoses encompasses the lower abdomen, lower lumbar region, gluteal area, genitalia and inner aspects of thigh. Diaper area eruptions can be subdivided into primary diaper area dermatitis, an acute inflammation of skin in diaper area with an ill-defined and multifactorial etiology, and secondary diaper area dermatitis, a term which encompasses eruption in diaper area with defined etiologies. These eruptions are directly related to the wearing of diapers, those aggravated by wearing diapers, and those that occur in the diaper region irrespective of whether diapers are worn or not.

OBJECTIVES: To document the various clinical patterns of diaper area dermatoses in children below 5 years.

To determine the etiological factors in conditions where it is defined.

MATERIAL AND METHODS: The study was undertaken from December 2013 to July 2015. All children below 5 years of age with eruptions involving the diaper area reporting to the Department Of Dermatology, Sri R.L.Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar were enrolled in the study. A written consent was taken from the informant. A detailed history including chief complaints, recent infections, history of allergy, family history of rash like conditions, general physical examination and dermatological examination was done and recorded in a proforma. Tests such as Tzanck smear, skin biopsy, KOH preparation, culture sensitivity, Gram staining and

appropriate hematological investigations were done whenever necessary depending on the presenting condition.

RESULTS: In the present study on children below 5 years, majority of Cases were infants (45%), followed by toddlers (33%), pre school children (19%) and least were neonates (3%). Males (52.3%) were affected marginally more than females (47.7%). The most common diaper area involved in our study population is gluteal region (65.4%). A total of 23 various dermatoses were encountered. ICD (22.3%) was the most common dermatoses, followed by scabies (14.6%), impetigo (13.8%), papular urticaria (11.5%). Infectious etiology (28.46%) was the commonest, followed by inflammation (26.15%) and arthropod bite (26.15%). Amongst the infective dermatoses, bacterial infection (14.61%) was most common entity followed by viral (10.76%) and fungal infection (3.1%). Amongst inflammatory dermatoses, irritant contact dermatitis (22.3%) was most common followed by seborrhoeic dermatitis (3.8%). Among arthropod bites, scabies was seen in 14.6% patients, and papular urticaria was seen in 11.5% patients.

CONCLUSION: Our study brings to light various dermatoses in diaper area in pediatric population. Three disorders which were very common were irritant contact dermatitis, scabies and impetigo. This study proves that aetiology is multifactorial and is not because of diaper alone.

KEY WORDS: diaper area dermatoses, irritant contact dermatitis, impetigo, scabies

LIST OF ABBREVIATIONS

ACD	Allergic contact dermatitis
GABHS	Group A beta haemolytic streptococcus
GCS	Gianotti-crosti syndrome
GGI	Granuloma gluteale infantum
GPC	Gram positive cocci
HFMD	Hand foot and mouth disease
HLA	Human leucocyte antigen
HPV	Human papilloma virus
HSV	Herpes simplex virus
ICD	Irritant contact dermatitis
ISD	Infantile seborrhoeic dermatitis
KOH	Potassium hydroxide mount
LCH	Langerhans cell histiocytosis
MGC	Multinucleated giant cell
PPPN	Perianal pseudo verrucous papules and nodules
SCFN	Subcutaneous fat necrosis
SD	Seborrhoeic dermatitis
SJS	Stevens Johnson syndrome
SSSS	Staphylococcal scalded skin syndrome
VZV	Varicella zoster virus

TABLE OF CONTENTS

Sl. No.		Page No.
1.	INTRODUCTION	01
2.	AIM OF THE STUDY	02
3.	REVIEW OF LITERATURE	03
4.	METHODOLOGY	39
5.	RESULTS	40
6.	DISCUSSION	71
7.	CONCLUSION	75
8.	SUMMARY	76
9.	BIBLIOGRAPHY	78
10.	ANNEXURES	86
	• PROFORMA	86
	CONSENT FORM	88
	KEY TO MASTER CHART	90

LIST OF TABLES

TABLE NO	CONTENTS	PAGE NO
1	Clinical types of diaper Dermatoses	40
2	Age group distribution.	42
3	Gender distribution of subjects	43
4	4:Distribution of various Dermatoses according to age 4A: Distribution of various Dermatoses according to age for Four Major Diagnosis in the study.	45
5	5:Distribution of Dermatoses according to Gender 5A: Distribution of Dermatoses according to Gender in Four common conditions	46
6	Distribution of Subjects according to presenting complaints	47
7	Distribution of Subjects according to Duration	48
8	Distribution according to Initial site involved	49
9	Examination of Diaper area site	50
10	Morphology of Primary lesion in diaper area	52
11	Morphology of Secondary lesion in diaper area	53
12	Demographic and disease characteristics of Irritant Contact Dermatitis (ICD)	54
13	Demographic and disease characteristics of Scabies	57
14	Demographic and disease characteristics of Impetigo	60
15	Demographic and disease characteristics of Papular Urticaria	66

16	Aetiology of Diaper area dermatoses	66
17	Aetiology-Infections	67
18	Aetiology-Inflammatory,congenital and other aetiological factors.	68

LIST OF GRAPHS

GRAPH NO	TITLE	PAGE NO
1	Aetiology of Diaper area dermatoses	41
2	Aetiology-Infections	42
3	Aetiology-Inflammatory,congenital and other aetiological factors.	43
4	Aetiology of Diaper area dermatoses	47
5	Aetiology-Infections	48
6	Aetiology-Inflammatory,congenital and other aetiological factors.	49
7	Aetiology of Diaper area dermatoses	50
8	Aetiology-Infections	51
9	Aetiology-Inflammatory,congenital and other aetiological factors.	52
10	Aetiology of Diaper area dermatoses	53
11	Bar diagram showing morphology of secondary lesions	56
12	Gender Distribution of ICD cases	56
13	Age Distribution in ICD cases	57
14	Bar diagram showing Site of Lesion in ICD cases	58
15	Gender Distribution of Scabies patients	58
16	Age distribution of Scabies Cases	59
17	Bar Diagram Showing Site of Lesion in Scabies	
18	Gender distribution of Impetigo cases	61
19	Bar diagram showing Age distribution of Impetigo cases	61

20	Bar diagram showing Site of Lesion in Impetigo cases	62
21	Pie diagram showing Gender distribution in Papular Urticaria cases	64
22	Bar diagram showing Age distribution of Papular Urticaria	64
23	Bar diagram showing Site of Lesion in papular urticaria cases	65
24	Pie chart showing etiology of diaper area dermatoses.	66
25	Pie chart showing infectious aetiology.	67
26	Impetigo involving gluteal region	92
27	Impetigo involving inner aspects of thigh and lower abdomen	92
28	GPC in clusters on grams stain in a case of impetigo	93
29	Staphylococcus aureus growth on blood agar	93
30	Candidiasis involving skin folds in diaper area	94
31	Candidiasis with satellite pustules	94
32	Gram positive budding yeast cells on gram stain	95
33	KOH mount showing hyphae	95
34	Irritant contact dermatitis due to diaper	96
35	Case of irritant contact dermatitis	96
36	Case of irritant contact dermatitis	96
37	Stevens johnson syndrome with genital area involvement	97
38	Papular urticaria involving diaper area	97
39	Scabies involving genital area	98
40	Case of infantile hemangioma	98

41,42	Seborrhoeic dermatitis involving scalp and diaper area	99
43,44 45,46	Case of tuberous sclerosis	100
47,48	Case of lamellar ichthyosis	101

INTRODUCTION

Diaper area dermatoses is a broad term used to describe various skin conditions that can occur in diaper area.¹ Eruptions in the diaper region have diverse origins. Many lesions like vesicles, pustules, bullae, erosions and ulcerations may arise in the diaper area.²

Eruptions in the diaper area are the most common dermatological problem in infancy.³

There are many diseases that may also involve other areas of the body and coincidentally affect the diaper area.

Diaper area eruptions can be subdivided into primary diaper area dermatitis, an acute inflammation of skin in diaper area with an ill-defined and multifactorial etiology, and secondary diaper area dermatitis, a term which encompasses eruption in diaper area with defined etiologies.³

These eruptions are directly related to the wearing of diapers, those aggravated by wearing diapers, and those that occur in the diaper region irrespective of whether diapers are worn or not.⁴

The occurrence of diaper area dermatoses in an infant or child causes panic in parents. An early and accurate diagnosis of these disorders will help the dermatologist to avoid unwanted investigations, unnecessary hospitalization and treatment.

As there is a paucity of studies on diaper area dermatoses in the current literature, a clinical study of these diaper area dermatoses can unravel the common clinical manifestations in our population.

AIMS AND OBJECTIVES

1. To document the various clinical patterns of diaper area dermatoses in children below 5 years.
2. To determine the aetiological factors in conditions where it is defined.

REVIEW OF LITERATURE

The anatomical area for diaper dermatoses encompasses the lower abdomen, lower lumbar region, gluteal area, genitalia and inner aspects of thigh. The most important factors in development of primary diaper area dermatitis are water, moisturizer, friction, urine, feces, microorganism.³ Secondary diaper area dermatitis includes a variety of other inflammatory and infectious processes that can occur in the diaper area.³

AETIOLOGY OF DIAPER AREA DERMATOSES

Inflammatory conditions

- Irritant diaper dermatitis
- Seborrhoeic dermatitis
- Atopic dermatitis
- Psoriasis
- Psoriasiform diaper dermatitis with Id reaction
- Erosive perianal eruption
- Pseudoverrucous papules
- Granuloma gluteale infantum
- Allergic contact dermatitis due to diaper components
- Diaper dye dermatitis
- ‘Lucky Luke’ dermatitis

Infections

- Candidiasis
- Dermatophyte infections
- Bullous impetigo

- Staphylococcal scalded skin syndrome
- Perianal streptococcal dermatitis/Streptococcal intertrigo
- Ecthyma gangrenosum
- Herpes virus infection
- HPV infections
- Molluscum contagiosum
- Hand foot and butt exanthem caused by Coxsackie virus infection

Metabolic causes

- Nutritional abnormalities
- Zinc deficiency
- Acrodermatitis enteropathica
- Acrodermatitis enteropathica like eruptions
- Methylmalonic acidemia
- Propionic acidemia
- Glutaric aciduria (type I)
- Maple syrup urine disease
- Ornithine transcarbamylase deficiency
- Citrullinemia
- Biotin deficiency
- Holocarboxylase deficiency

Miscellaneous

- Langerhans cell histiocytosis
- Kawasaki disease
- Granular parakeratosis
- Pyramidal perianal protrusion

- Nascent hemangioma
- Lichen sclerosus
- Pyoderma gangrenosum
- Chronic bullous disease of childhood
- Bullous pemphigoid
- Ichthyosis
- Papular urticaria
- Scabies
- Miliaria

INFLAMMATORY CONDITIONS

IRRITANT CONTACT DERMATITIS

Jacquet gave the first description of diaper dermatitis in 1905. Onset of irritant contact dermatitis is generally between 3 weeks and 2 years of age, with highest prevalence between 9 and 12 months.

The condition was previously common, affecting 25% of children, but the incidence has decreased remarkably in western cultures owing to the advent of disposable diapers.²

Aetiology/pathogenesis

In most cases, it is thought to be a reaction to irritants in the diaper environment, such as friction, occlusion, dampness, maceration, urine, feces, or chemicals. The main irritants in this situation are fecal proteases and lipases, whose activity is increased greatly by elevated pH. An acidic skin (neutral or low pH) surface is essential for the maintenance of the normal microflora, which provides innate antimicrobial protection against invasion by pathogenic bacteria and yeasts. Fecal lipase and protease activity is also greatly increased by acceleration of gastrointestinal transit; this is the reason for the high incidence of irritant diaper dermatitis observed in babies who have had diarrhoea in the previous 48 hours. The wearing of diapers causes a significant increase in skin wetness and increase in the pH level. Prolonged wetness leads to maceration (softening) of the stratum corneum, which is associated with extensive disruption of intercellular lipid lamellae. Weakening of physical integrity makes the stratum corneum more susceptible to damage by friction from the surface of the diaper, and local irritants. The normal pH of the skin is between 4.5 and 5.5. Elevated pH levels increase the hydration of the

skin and make the skin more permeable. At full term, the skin of infants is an effective barrier to disease and is equal to adult skin with regard to permeability. However, dampness, lack of air exposure, acidic or irritant exposures, and increase in skin friction begin to break down the skin barrier.^{3,4}

Clinical features

Irritant diaper dermatitis presents as erythema on the convex surfaces of the inner upper thigh, the lower abdomen, and buttock areas, the areas most in contact with the diaper. The intertiginous creases are usually spared. It varies in severity from mild, with erythema with or without scale, to moderate, with more papules or macerated lesions. An ulcerative form, known as ammoniacal ulcers or jacquet erosive dermatitis, is characterized by well-demarcated, punched out ulcers or erosions that have elevated borders. In more severe cases the erythema may be accompanied by a glistening or glazed appearance and a wrinkled surface.²⁻⁴

Differential diagnosis⁵

Seborrhoeic dermatitis

Psoriasis

Candidiasis

Atopic dermatitis

Acrodermatitis enteropathica

Treatment

The key to prevent irritant diaper dermatitis is to keep the skin in the diaper area protected from urine and feces, which act synergistically to cause most cases of

diaper dermatitis. This can be managed by increasing the frequency of diaper changes and gently cleansing the skin. It makes sense, however, that the skin be cleaned gently and rinsed with warm tap water after urination and a mild, nonperfumed soap used to help remove residual fecal material.

Barrier creams such as zinc oxide paste may help minimize urine and fecal contact with the skin. More frequent diaper change, and ultraabsorbent disposable diapers should be recommended if the dermatitis is recurrent or particularly severe.

A non- fluorinated, low-potency corticosteroid ointment or cream, such as hydrocortisone 1%, should be applied four times daily with diaper changes.

If the rash is severe or present for more than 72 hours, anticandidal agents such as nystatin, clotrimazole, or ketoconazole should also be used.

Thickly applied barrier creams may be helpful as adjunctive therapy and afterwards to prevent recurrences.^{4,6}

SEBORRHOEIC DERMATITIS

Seborrheic dermatitis was first described by Unna in 1887.⁷ SD is a common chronic inflammatory disease characterized by erythema accompanied by greasy scales in the seborrheic region, which includes the scalp, forehead/forehead, eyebrows, malar eminences, nasolabial folds, retroauricular area, chest, **diaper area** and intertriginous area.

SD occurs most frequently in infants and adults aged 30 to 60 years⁸.

Aetio-pathogenesis:

Although the aetiology of seborrhoeic dermatitis is not definitely known, the most accepted hypothesis is the alteration in colonization and metabolism of cutaneous microflora.

A more causal link seems to exist between seborrhoeic dermatitis and the proliferation of *Malassezia* species (e.g., *Malassezia furfur*, *Malassezia ovalis*) found in normal dimorphic human flora.⁹ The causal relationship is implied because of the ability to isolate *Malassezia* in patients with seborrhoeic dermatitis and by its therapeutic response to antifungal agents.¹⁰ Yeasts of this genus predominate and are found in seborrhoeic regions of the body that are rich in sebaceous lipids (e.g., head, trunk, upper back). Yeast of the genus *Malassezia* is also increased in the scaly epidermis of dandruff and seborrhoeic dermatitis. Although it has been suggested that this is secondary to the increase in size of the habitat provided by the scaling, it is generally accepted that the presence of yeast of the genus *Malassezia* causes the condition.¹¹

The *Malassezia* species that have been most commonly associated with seborrhoeic dermatitis are *M. globosa* and *M. restricta*, both of which are commensal yeasts that require an exogenous source of lipids.¹² A cell mediated immune response to *M. furfur* has been found in normal individuals also.

Seborrhoeic dermatitis is also associated with nutritional deficiencies, but there is no firm linkage. Overgrowth of *M. furfur* may lead to inflammation, either through introduction of yeast-derived metabolic products into the epidermis or as a result of the presence of yeast cells on the skin surface.^{11,12} The mechanism of production of inflammation is through activation of Langerhans cells and T lymphocytes by *Malassezia* or its by-products. When *M. furfur* comes into contact

with serum, it can activate complement via the direct and alternative pathways and this may play some part in the role of inflammation.¹²

Clinical features:

SD usually develops in neonates within the first 3 to 4 weeks of life. Spontaneous recovery generally occurs at about 6 to 7 months of age, though persistence until 2 years of age can be seen. SD in adults affects men more often than women; ISD shows no gender predilection. The occurrence of SD in prepubertal children (aged 2–5 years) is uncommon.⁸

The most commonly involved sites are the scalp and diaper area.⁸

Morphological variants:

Clinical patterns of seborrhoeic dermatitis are-

- Scalp (cradle cap)
- Trunk (including flexures and napkin area)
- Leiner's disease
- Non-familial
- Familial C5 dysfunction

In infants, seborrhoeic dermatitis may present as thick, greasy scales on the vertex of the scalp (cradle cap). The condition is not pruritic in infants.

The scales may vary in color, appearing white, off-white, or yellow. Infants with large, dry scales often have psoriasiform seborrhoeic dermatitis. This presentation often is the only sign of seborrhoeic dermatitis in infants and usually occurs in the third or fourth week after birth. However, the scalp, central face, forehead, and ears may have fine, widespread scaling. **In diaper area lesions consist**

of erythematous, well defined patches involving the creases, but sometimes affecting the whole region. Generalized seborrhoeic dermatitis is uncommon in otherwise healthy children and usually is associated with immune deficiencies. Immunocompromised children with generalized seborrhoeic dermatitis often have concomitant diarrhoea and failure to thrive (Leiner's disease); therefore, infants with these symptoms should be evaluated for immunodeficiencies.¹³

Differential diagnosis^{13,14}

Infantile seborrhoeic dermatitis must be differentiated from:

- Atopic dermatitis
- Irritant diaper dermatitis
- Langerhans cell histiocytosis.

Treatment

The usual therapeutic approach for ISD of the scalp is conservative. In mild cases, an emollient such as white petrolatum or mineral oil may be used to soften the cradle cap so that it can be gently removed by brushing off the scales.¹⁵ Crusts are soaked overnight with slightly warmed oil and washed off in the morning. A mild non medicated shampoo should be used at the start of therapy in conjunction with brushing off scales with a baby's toothbrush. If a mild shampoo is not helpful, a shampoo containing ketoconazole 2% can be used. Coal tar-based shampoos must be avoided because of the carcinogenicity of coal tar. Mild topical corticosteroid lotions can be used adjunctively to reduce erythema of the scalp. ISD involving intertriginous areas is treated with gentle skin care and topical medicaments. Topical ketoconazole or nystatin are safe and effective therapies, particularly when combined with a mild topical corticosteroid.^{15,16}

PSORIASIS

Psoriasis is being increasingly recognized in early infancy and often affects the diaper area, particularly in those under 1 year.¹⁷

Aetiology/pathogenesis

Pathogenesis is thought to involve an imbalance of T-helper cells resulting in a Th 1-type cytokine reaction pattern.

There is an increased cell turn over period of 4 days that is characteristic of psoriasis.¹⁸

Clinical features

Skin lesions in the diaper area has 2 distinctive patterns: Localized psoriatic diaper rash with well demarcated bright red rashes and psoriatic diaper rash with dissemination, where the initial presentation is well defined erythematous plaques localized to the diaper area slowly progressing to involve other areas. More sharply demarcated lesions, positive family history, presence of lesions in other areas and nail involvement favors the diagnosis of psoriasis.¹⁷

Differential diagnosis¹⁷⁻¹⁹

- Seborrhoeic dermatitis
- Irritant contact dermatitis
- Candida albicans infection

Treatment

Mild to moderate topical corticosteroid three times a day usually produces rapid resolution of the lesions. Salicylic acid is not recommended in neonates and infants, as systemic absorption can lead to salicylism.¹⁸

GRANULOMA GLUTEALE INFANTUM

Granuloma gluteale infantum, originally described by Tappeiner and Pfleger is a rare dermatosis of infancy of unknown etiology.

Clinical features

Infants present with oval red-brown to purple dermal nodules on the gluteal surface and diaper area. The diagnosis of GGI should be considered in longstanding and unresponsive cases of diaper dermatitis in infants as the inflammation can easily mask the underlying characteristic nodules suggestive of the diagnosis.²⁰

Irritant contact dermatitis, candidiasis, occlusion from diapers and the prolonged use of benzocaine and fluorinated steroids have been considered as contributing factors for the development of GGI.

Histopathology

The histological picture is that of a non-specific perivascular dermal infiltrate composed of neutrophils, lymphocytes, histiocytes, plasma cells and eosinophils; with an overlying hyperplastic epidermis and parakeratosis.²¹

Differential diagnosis²¹

- Allergic contact dermatitis
- Jacquet's erosive dermatitis
- Candidiasis
- Langerhans cell histiocytosis
- Seborrhoeic dermatitis

Treatment

Underlying cause should be corrected

Frequent use of a barrier product

Lesions generally resolve completely and spontaneously after a period of several months if the source of chronic irritation can be removed.²¹

PSEUDOVERRUCOUS PAPULES

Perianal pseudo verrucous papules and nodules (PPPN) is a rare entity, first described by Goldberg et al in the setting of chronic diaper dermatitis, encopresis or peristomal skin irritation.²²

Clinical features

PPPN usually occurs in infants rather than newborns. It presents clinically as multiple well-demarcated, dome-shaped papules, 2-10 mm in size, with a shiny smooth surface, over the perianal region, buttocks, vulvar, and scrotal area or around entero-stomal region. The lesions may become ulcerated or friable and there is a risk of secondary infection.²²

Differential diagnosis²³

- Candidiasis
- Granuloma gluteale infantum
- Condylomata acuminata
- Histiocytosis x.

Histopathology

Histopathological examination shows epidermal hyperplasia with marked acanthosis and altered cornification with parakeratosis, hypogranulosis, and pale keratinocytes in the epidermis.²²

Treatment

Treatment should be aimed at reducing the irritation and prevention of secondary infection by removal of precipitating factor and recovery of skin barrier function.

The peristomal skin should be swabbed in all cases for microbiological examination because treatable primary and secondary infections are relatively common.^{22,24}

ALLERGIC CONTACT DERMATITIS

Allergic contact dermatitis may occur in the diaper region after exposure to fragrances, dyes, and to components of the diaper itself.

Aetiology

Allergens that infants and children have become sensitized to include urushiol(poison ivy), nickel, thimersol, neomycin, chromates, balsam of peru, formaldehyde and related preservatives.^{25,26}

Clinical features

The lesions begin with erythema and small vesicles leading to an eczematous eruption with red papules and vesicles overlying areas of edema.²

Differential diagnosis

- Irritant contact dermatitis
- Seborrhoeic dermatitis
- Candidiasis involving diaper area.

Treatment

Treatment with a medium-strength topical steroid provides rapid relief of symptoms, but removing the offending allergen is key to prevent recurrences.²

ATOPIC DERMATITIS

Lesions of atopic dermatitis spare the diaper area during infancy, which aids in the diagnosis of atopic dermatitis. The sparing of diaper area in atopic dermatitis probably reflects a combination of increased hydration, protection from triggers, and inaccessibility to scratching and rubbing.^{27,28}

INFECTIONS

IMPETIGO

Impetigo is one of the most common skin infections in children accounting for approximately one tenth of all cutaneous problems presenting to pediatric clinics.

Impetigo is superficial skin infection mainly caused by staphylococcus aureus or group A beta haemolytic streptococcus (GAβHS)

Impetigo is divided into 2 clinical types: bullous and non bullous or crusted impetigo.^{31,32}

Clinical features

Bullous impetigo presents, often on previously normal skin, as small vesicles that may develop into bullae of more than 1 cm in diameter, with little or no surrounding erythema. The thin roof of the bulla soon breaks, revealing an erythematous moist base that quickly dries, leaving a shiny lacquered appearance.^{31,33}

Non bullous or crusted impetigo starts as small vesicle or pustule that is very transient, soon rupturing to be replaced by an expanding golden crust .Removal of the crust reveals a moist pink surface. The lesion may be surrounded by a small halo of erythema.³³

Pathogenesis

Bullous impetigo is caused by *Staphylococcus aureus*. The usual strain involved is group 2, phage type 71, although other strains that have the ability to produce exfoliative toxin can be involved.

The primary etiological agent in most cases of non bullous impetigo is *GAβHS*. However recent studies indicate that, in many parts of the United states, and in several other countries, *S.aureus* must be now regarded as the primary pathogen of nonbullous and bullous impetigo.³³⁻³⁶

Differential diagnosis^{34,35}

- Herpes simplex infection
- Epidermolysis bullosa
- Congenital candidiasis

Management

Ideally bacterial culture and sensitivity testing should be obtained in all cases and a decision on which antibiotic to be used should be delayed until the results are available. Oral antibiotics such as cloxacillin, cephalixin, and erythromycin are all effective as 10-day course and lead to rapid cure in 5-7 days. Mupirocin in a 2 percent concentration has antibacterial activity against *S.aureus* and *GAβHS*.^{33,36}

CANDIDIASIS

Diaper area candidiasis usually affects infants between 6 and 24 months. Both sexes are equally affected.

Aetiology

The most common organisms implicated are *candida albicans* followed by *C.parapsilosis*, *C .pulcherrima*, *C. guilliermondii* and *C. zeylanoides*.³⁷

Clinical features

Candidiasis initially manifests as maceration of anal mucosa and perianal skin. Later, it develops into sharply marginated confluent zones of erythema with papules, pustules, and satellite pustules. The presence of satellite pustules allows diaper dermatitis caused by candida to be distinguished from other types of dermatoses in diaper area. The condition can present on the thighs, genital creases, abdomen and genitalia. Distant and distinct satellite lesions are most likely to provide material that will yield positive cultures.³

Differential diagnosis^{37,38}

- Irritant contact dermatitis
- Infantile seborrhoeic dermatitis
- Granuloma gluteale infantum
- Psoriasis

Laboratory diagnosis³⁸

1. Direct examination: The scrapings are kept in 15% KOH aqueous solution till adequately dissolved. The smears are examined under a microscope as such or after staining with Gram's stain. Smears from the exudate are more suitable for staining with PAS, Gram's stain or Gridley method. Microscopic examination reveals yeast cells of 4–8 mm with budding and pseudohyphae.
2. Culture: Culture can be made on Sabouraud's dextrose agar incubated at 37°C temperature. The typical creamy white, smooth and pasty colonies appear in 3–4 days.
3. Electron microscopy: Ultrathin sections display *C. albicans* on the surface of keratinized cells as well as within them. Scanning electron microscopy allows more rapid identification of *C. albicans* than with cultures.

4. Germ tube test: Germ tubes of *Candida* species can be demonstrated by “Reynold’s Braude phenomenon”. Incubation of the culture of *Candida* species with human or sheep serum at 37°C produces germ tubes within 2–4 hours. Tube-like projections without a constriction at the point of attachment to yeast cell (pseudohyphae) are seen extending from the cell wall.
5. Biochemical tests: Biochemical tests like sugar fermentation and assimilation are of immense importance in the identification of the yeast.
6. Serology and immunology: Antigen tests based on ELISA, RIA, PHA (phytohemagglutination) have been developed to detect either cell wall mannan or the cytoplasmic component.

Treatment³⁸

Topical imidazoles like 1% clotrimazole, 2% ketoconazole are usually effective but proper hygiene is most important.

HAND, FOOT AND MOUTH DISEASE

Hand, foot and mouth disease (HFMD) is caused by human enteroviruses. It occurs in outbreaks in nursery schools or kindergartens, usually during summer and autumn months.

Aetiology

Coxsackie virus A16 (CA16) and enterovirus 71 (EV71) are two major causative agents and coxsackie viruses A4, A5, A8, A10, B3, and B7 are usually associated or are minor etiologies. This fact was reported for the first time in Toronto, in 1957.³⁹

Clinical features

The incubation period is 3-5 days. The clinical feature is characteristic and classically consists of a combination of exanthem and enanthem. Papulovesicular lesions are present over the hand, feet, buttocks, knee and oral mucosa. There may be a mild fever.³⁹

Differential diagnosis^{39,40}

- Papular urticarial
- Varicella
- Measles

Treatment⁴⁰

It is a self-limiting disease. Specific treatment is not necessary.

GIANOTTI-CROSTI SYNDROME

Gianotti-Crosti syndrome (GCS) is a relatively common dermatoses, seen worldwide, primarily affecting children between 2 and 6 years of age. The classic physical finding is an asymptomatic self-limited papulovesicular exanthem; on rare occasions there are associated systemic signs and symptoms. GCS is usually associated with viral infections or immunization, but the pathogenesis remains a mystery.

Since the initial description by Gianotti in 1955, the classification of GCS has varied over the decades, primarily reflecting increased sophistication in virology.⁴¹

Aetiology

Epstein-Barr virus, Hepatitis A, B, or C viruses, Cytomegalovirus, Human herpesvirus 6, Coxsackie virus A16, B4, B5, Rotavirus, Parvovirus B19, Molluscum

contagiosum , Respiratory syncytial virus, Echovirus , Mumps virus, Parainfluenza virus, HIV.

Bacteria also appear capable of triggering GCS, although it is a rare event.⁴²

Clinical features

The disease usually starts abruptly with cutaneous lesions. Sometimes there may be a prodrome with pharyngitis, upper-airway infection, or diarrhea. The cutaneous signs and symptoms seem to depend more on the individual characteristics of the patient than on the causative virus. The classic finding is multiple, monomorphous pink to red-brown papules or papulovesicles, which may be slightly pruritic and can become confluent . The individual lesions are 1 to 5 mm in diameter and rarely exceed 10 mm, often flat-topped , and occasionally with hemorrhage or, less often, scale. The papules are symmetrically distributed on the cheeks, extensor aspects of the extremities, and buttocks . They last for many weeks. The trunk, knees and elbows, along with palms and soles are usually spared, but can occasionally show a few lesions, so that their involvement does not exclude the diagnosis of GCS.^{41,43}

Diagnostic criteria of GCS⁴⁴

A patient is diagnosed as having GCS or papular acrodermatitis if:

On at least one occasion or clinical encounter, he/she exhibits all the positive clinical features and on all occasions or clinical encounters related to the rash, he/she does not exhibit any of the negative clinical features and none of the differential diagnoses is considered to be more likely than GCS on clinical judgment and if lesional biopsy is performed, the findings are consistent with GCS

The positive clinical features are:

Monomorphic, flat-topped, pink-brown papules or papulovesicles 1-10 mm in diameter on at least three of the following four sites: Cheeks, buttocks, extensor surfaces of the forearms, extensor surfaces of the legs.

Being symmetrical

Lasting for at least 10 days

The negative clinical features are:

- Extensive truncal lesions
- Scaly lesions

Differential diagnoses are:

- Acrodermatitis enteropathica
- Erythema infectiosum
- Erythema multiforme
- Hand-foot-and-mouth disease
- Henoch-Schönlein purpura
- Papular urticarial
- Scabies

Treatment

The lesions blanch and resolve more rapidly with mid potent topical corticosteroids applied once daily for 7 to 14 days but this effect has never been established in a controlled setting. Although worsening of findings with topical corticosteroids has been described, it must be very rare and does not pose a contraindication. Systemic corticosteroid pulse therapy has been recommended for severe cases. If the lesions are pruritic, oral antihistamines or topical antipruritics may be useful.^{41,44}

HERPES SIMPLEX INFECTION

Neonatal herpes may present in diaper region following breech delivery of an infant whose mother has genital HSV. The diagnosis of genital HSV in an older infant or child may raise the suspicion of sexual abuse, although innocent transmission from an infected care giver or parent may occur.

Primary herpes infection presents with painful vesicles, clustered on an erythematous base, 2-8 days after contact with an infected individual.⁴⁵⁻⁴⁷

VARICELLA

Varicella is a febrile illness caused by varicella zoster virus, characterized by a generalized, pruritic vesicular rash and is most prevalent in childhood.⁴⁸

Clinical features

Chickenpox lesions evolve from erythematous macules to papules and then to small vesicles on an irregular erythematous base, giving the “dew drop on a rose petal” appearance. The vesicular fluid soon becomes cloudy with the influx of inflammatory cells to form a pustule. It then dries, beginning in the center, first producing an umbilicated pustule and then a crust. Crusts fall off spontaneously in 1 to 3 weeks, leaving a shallow depression that gradually disappears. Scarring is rare, unless it is picked or secondarily infected with bacteria. The lesion, as it heals, is hyperpigmented and then hypopigmented and disappears. Mucous membrane involvement is not rare, with involvement of the mouth, nose, pharynx, larynx, trachea, gastrointestinal tract, urinary tract and vagina.⁴⁸⁻⁵⁰

Diagnosis

A Tzanck smear showing multinucleated giant cells, epithelial cells with acidophilic intranuclear inclusion bodies, suggests a diagnosis of VZV infection.

Punch biopsy of the lesion may show ballooning degeneration of the granular cells as well as cells of the deeper layers of the stratum spinosum.

Treatment

Oral acyclovir -20 mg/kg body weight q.i.d. for 5 days.

ANOGENITAL WARTS

Verrucae are caused by cutaneous infection with the human papilloma virus(HPV).

Infants can acquire warts from maternal genital condyloma, at the time of delivery with resulting genital disease.⁵¹

Lesions appear as asymptomatic, flesh coloured papules that may coalesce to form plaques. They have a characteristic verrucous , velvety surface. There have been reports of condyloma acuminata in young children, which may be the result of sexual abuse.⁵¹

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Staphylococcal scalded skin syndrome (SSSS) is a rare toxin-mediated manifestation of localized infection with certain strains of *Staphylococcus aureus*, which cause blistering and desquamation of the skin. SSSS occurs mostly in children younger than 5 years possibly due to immunological immaturity and less renal clearance of toxin.⁵²

Aetiopathogenesis

SSSS starts from a localised staphylococcal infection that is a producer of the two causative exotoxins, either or both exfoliative toxins (A and B) - carried by around 3% of all *S.aureus*. In children the infectious focus is usually in the nasopharynx or conjunctiva. Exfoliative toxins are glutamate specific serine proteases

that specifically bind and cleave desmoglein 1 causing blistering and denudation by disruption of the epidermal granular cell layer in skin.⁵³

Clinical features

SSSS usually starts with fever, irritability, and widespread redness of the skin. Other symptoms may include tender and painful areas around the infection site, weakness, and dehydration. Areas of involvement can be limited to neck and diaper area. Within 24-48 hours fluid-filled blisters form with demonstrable Nikolsky's sign. These rupture easily, leaving an area that looks like a burn. SSSS usually follows a benign course when diagnosed and treated appropriately. Otherwise, severe infections such as sepsis, cellulitis, and pneumonia may develop. Death can follow severe infection.^{52,53}

Differential diagnosis

- Sunburn
- Epidermolysis bullosa
- Toxic epidermal necrolysis
- Kawasaki disease
- Extensive bullous impetigo
- Viral exanthem

Treatment

Oral and IV antibiotics for 5-10 days.

In methicillin-resistant cases it may be feasible to use fucidic acid or mupirocin if the lesions are localized.

SCABIES

Scabies is a highly contagious skin disease which affects both males and females of all socioeconomic status and race. It is related primarily to poverty and overcrowding. Scabies is caused by an arthropod, *Sarcoptes scabiei hominis*, an ectoparasitic mite.⁵⁴

Aetiology

The scabies mite, *Sarcoptes scabiei hominis*, was identified by Bonomo using light microscopy in 1687.⁵⁵ The mite has a lifespan of 30 days and lays 2 to 3 eggs per day. The average mite population per patient with classic scabies is 20.⁵⁶

Clinical features

The sources of infection in scabies can be an infected family member, sexual partner or friends (in school or otherwise) and is spread commonly by direct contact, fomites and sexual route.⁵⁷

After an incubation period ranging from 3 to 4 weeks, the patient presents with intense itching which is worse at night. Sites of predilection of the lesions are the Interdigital clefts of hands, wrists, elbows, anterior axillary folds, the periumbilical area, the areolae of the breast, the buttocks, genitalia. In infants, scabies usually affects the axillae, head, diaper region, and, occasionally, the palms and soles.^{58,59}

Pathognomonic sign of this disease is the presence of a burrow several millimeters in length. These serpiginous, gray-white lines may, upon close inspection, reveal a black speck at its leading end, indicating the presence of the adult mite. Burrows are typically located on the interdigital spaces of the hand, the flexor surfaces of the wrists and elbows, and genitalia.⁵⁹

Various types of lesion have been seen and reported in literature e.g., papular, papulovesicular, eczematous and nodular. Though rare, erythrodermic and crusted

scabies (Norwegian variety) have also been seen. Scabies occurs in all ages and all groups of person of both sexes are susceptible.⁵⁷

Diagnosis

Intense pruritus should raise the suspicion that an individual may have scabies. The index of suspicion should be further heightened if several members of a family report similar symptoms. Scabies may be diagnosed from physical examination revealing burrows in typical locations, including the hands, wrists, elbows, and genitalia.

Burrows may be barely visible to the naked eye. The burrow ink test may aid in identification. In this procedure, a washable felt-tip marker is rubbed across the typical locations, and the ink is subsequently removed with alcohol or water. If burrows are present, they will absorb ink and be readily apparent.

Diagnosis of scabies should be confirmed by the presence of the mite, its eggs, or its scybala. The mite may be visible at the leading end of a burrow. Direct microscopic examination of skin scrapings mounted on a slide is ideal for identifying the mite and its products. Potassium hydroxide, saline, and mineral oil are possible solutions for the preparation. Potassium hydroxide provides the clearest visualization of the mites and eggs, but this is at the expense of dissolving the scybala. Because the fecal material is the most sensitive product to detect, saline or mineral oil may be better solutions to use in the procedure.⁶⁰

Differential diagnosis⁵⁸⁻⁶⁰

- Atopic dermatitis
- Contact dermatitis
- Folliculitis

- Infantile acropustulosis
- Papular urticaria
- Seborrhoeic dermatitis

Treatment

All family members must be treated at the same time to avoid the ‘ping pong’ effect, i.e. transmission of scabies by the asymptomatic member.

Avoid hot scrub bath before application of the scabicide agent (this can increase the systemic toxicity). Ideally, the scabicide agent should be applied on cool, dry skin.

The scabicide cream or lotion should be applied at bedtime, all over the body from head to toes in infants and neck downwards in older children.⁵⁸⁻⁶⁰

Permethrin 5% cream is commonly recommended as the drug of choice.

Other treatment options are crotamiton 10% cream, lindane 1% lotion, benzyl benzoate (BB) 25% lotion.

Mass treatment of scabies either by oral ivermectin (200 Microgram/kg) or topical permethrin is suggested. Contact tracing is an important approach for scabies control and prevention.^{61,62}

PAPULAR URTICARIA

Papular urticaria was originally described by Bateman in 1813. Papular urticaria is a common and often distressing childhood disorder manifested by chronic or recurrent papules caused by a hypersensitivity reaction to the bites of mosquitoes, fleas, bedbugs, and other insects.⁶³

Pathogenesis

First exposure to the bites does not produce reaction. On subsequent exposures, host response to salivary or contactant proteins causes eruption. Exact immune mechanism is unknown; evidence supports involvement of types I, III, and IV hypersensitivity.⁶⁴

Evidence supporting involvement of type I hypersensitivity includes time frame of immediate reaction, formation of wheals, elevated mosquito saliva-specific IgE in papular urticaria caused by mosquito bites, correlation of immediate wheals and flares with mosquito salivary gland-specific IgE levels, and high levels of saliva-specific IgE levels in systemic reactions.

Type I hypersensitivity reaction may cause the wheal, while delayed papule may result from type IV hypersensitivity.

Appearance of new lesions following new bites may be accompanied by lesions appearing on previously sensitized sites, indicating role of circulating antigen(s) which stimulate cutaneous T cells in previously sensitized sites.⁶⁴

Clinical features

Papular urticaria is seen primarily in children between 2 and 7 years of age, particularly in those with a history of atopic dermatitis. Although cases have been described in infants as young as 2 weeks. The disorder usually appears in late spring and summer. Papules may occur on any part of the body but tend to be grouped in clusters on exposed areas, particularly the extensor surfaces of the extremities. These papules may also be seen on the face and neck, trunk, thighs, and buttocks and generally spare the genital, perianal, and axillary regions. However, location depends on the arthropod involved.^{65,66}

Individual lesions are seen as 3 to 10-mm, firm urticated papules, often with a central punctum. They may be excoriated, lichenified, or secondarily infected with crust formation. The lesions recur in crops and all stages of development may be noted. Most lesions persist for 2 to 10 days and, after resolution, may result in temporary post inflammatory erythema or pigmentation.⁶⁶

Pathology

The histopathology of papular urticaria consists of mild subepidermal edema, extravasation of erythrocytes, interstitial eosinophils, and exocytosis of lymphocytes. Depending on the predominant cellular infiltrate 4 subtypes (lymphocytic, eosinophilic, neutrophilic, and mixed) may be recognized.⁶⁷

Differential diagnosis.^{63,68}

- Papular forms of atopic dermatitis
- Allergic contact dermatitis
- Drug-induced reaction
- Id reaction
- Miliaria rubra
- Papulovesicular polymorphous light eruption
- Papular acrodermatitis of childhood (Gianotti- crosti syndrome)
- Pityriasis lichenoides et varioliformis acuta.

Treatment

Three “Ps” of management include protective clothing, pruritus control, and patience. Treatment of papular urticaria is symptomatic, with topical corticosteroids and antihistaminics for mild reactions, short course of systemic corticosteroid for severe reactions, and management of anaphylaxis if it occurs.⁶⁹

MILIARIA

Miliaria is one of the common noninfectious eruption of diaper area.

Aetiology/pathogenesis

Miliaria occurs when the sweat gland ducts get plugged due to dead skin cells or bacteria such as staphylococcus epidermidis, a common bacterium that occurs on the skin.

The condition can occur in infants wearing disposable diapers and tape line has an occlusive effect underneath it, obstructing the discharge of eccrine secretions, which results in miliaria.²⁹

Clinical features

Miliaria appears as tiny red papules, papulo-vesicles and/or fragile vesicles. In neonates it most commonly appears in diaper area, neck and intertriginous areas.

The condition usually occurs at the elasticized openings of the diaper and at the superolateral abdomen, where the diaper tapes attach to the front panel of the diaper.³⁰

Differential diagnosis³

- Irritant contact dermatitis
- Impetigo
- Scabies
- Papular urticaria

Treatment

The condition is self-limited; precipitating factors should be avoided.

INFANTILE HEMANGIOMA

Hemangiomas of infancy are the most common vascular nevi of childhood.

Aetio- pathogenesis

The aetio-pathogenesis of infantile hemangiomas has not yet been completely defined. Angiogenetic factors, mast cells, endogenous steroid hormones, placental injury during prenatal chorionic villous sampling have all been suggested as factors.^{70,71}

Clinical features

Hemangiomas of infancy are usually solitary, soft, well-defined, round, dome-shaped, scarlet-red swellings with a smooth or lobulated surface. Occasionally they may be multiple. These lesions have a predilection for the head and neck (60%), although they can occur anywhere on the body. Infantile hemangioma located in the perineum frequently become ulcerated, and this may actually be the presenting finding in a minority of patients.⁷¹

Treatment

Prednisolone is used in a dose of 2-4 mg/kgbw over 4-8 weeks followed by gradual tapering.

Surgical excision may be required for lesions that fail to involute or respond to other therapy, for larger angiomas and for those causing tissue destruction.

Recent literatures support the use of propranolol in these cases, but the long-term results are awaited.⁷²

ACRODERMATITIS ENTEROPATHICA

Acrodermatitis enteropathica is a rare, autosomal recessive disorder classically characterized by zinc deficiency due to defective absorption. It is associated with ocular, gastrointestinal, and mucocutaneous symptoms with variable frequency, and can be fatal without zinc therapy.

Clinical features

Infants with acrodermatitis enteropathica present with the classic triad of diarrhoea, skin rash with alopecia and extreme irritability with depression. It is common in premature babies due to insufficient body stores of zinc as well as high zinc requirements at the time of weaning. It is rare in breastfeeding infants because of zinc binding ligand in breast milk which generally protects breastfed infants against this condition by increasing zinc bioavailability. Onset is approximately 1-2 weeks after weaning or 4-10 weeks of age in bottle-fed infants. Infants with acrodermatitis enteropathica have a classical erythematous, scaly, psoriasiform and sometimes vesiculo-erosive eruption located periorificially (i.e. around the mouth, eyes and genital area). Refractory diaper dermatitis may be the presenting complaint. Nail changes consisting of paronychia as well as dystrophy of the nail plate may occur. Superadded staphylococcal and candidal infections are very common, and candida may not respond to topical therapy until zinc deficiency is corrected.⁷³

Diagnosis

Samples for determining zinc levels must be carefully collected in special acid washed or plastic tubes so that exogenous zinc present on ordinary glassware will not contaminate the specimen. Serum zinc levels are usually less than 50 µg/dl.

Low alkaline phosphatase levels can be a valuable marker of zinc deficiency even when the plasma zinc levels are normal as it is a zinc dependent enzyme.^{73,74}

Treatment

Treatment is with oral zinc, 3-5 mg/kg/day. Life-long zinc supplementation is required with a diet rich in zinc in the form of unmilled cereals, beans, cheese, whole wheat bread and animal proteins like lean red meat is necessary to prevent recurrences.⁷³

LANGERHANS CELL HISTIOCYTOSIS

Langerhans cell histiocytosis (LCH) is a disorder of antigen-presenting cells and is the commonest disorder of the mononuclear phagocytic system. LCH is characterized by clonal proliferation and excess accumulation of pathologic Langerhans cells. The disease varies widely in clinical presentation from localized involvement of a single bone to a widely disseminated life-threatening disease.⁷⁵

Clinical presentation

Skin involvement occurs in 50% of patients with isolated ‘skin-only’ disease in about 10%. LCH should be considered whenever seborrheic dermatitis or diaper dermatitis fails to respond to therapy, or keeps recurring. The commonest presentation is with a ‘seborrhoea-like’ eruption, which may or may not be purpuric, often initially misdiagnosed as ‘cradle-cap’. Other skin manifestations include papules, vesicles, crusted plaques, nodules and purpuric nodules. Hashimoto–Pritzker disease (congenital self-healing reticulohistiocytosis) is a skin-only LCH associated with spontaneous involution. There are no reliable pathologic criteria that distinguish congenital self-healing reticulohistiocytosis from skin LCH, and a recent study failed to show a significant difference in histology or expression of markers such as E-cadherin, Ki-67 and phosphorylated histone 3.^{75,76}

Diagnosis

The diagnosis is clinicopathologic, based on classical clinical findings and histologic/immunohistochemical criteria, to avoid misdiagnosis of reactive normal Langerhans cells found within nodes in response to a variety of diseases including neoplasms.

Previously absolute criteria for diagnosis depended on finding CD1a by immunohistochemistry or Birbeck granules by electron microscopy. Currently, the presence of Birbeck granules is assumed by immunohistochemical demonstration of langerin (CD207), a mannose-specific lectin whose intracellular component is found in association with Birbeck granules with 100% concordance .

Positivity of one or both of these markers now defines the Langerhans cell phenotype.^{75,76}

Differential diagnosis

- Erythema toxicum neonatorum
- Neonatal pustular melanosis
- Acropustulosis of infancy
- Seborrhoeic dermatitis.

Treatment⁷⁷

The diagnosis should be established based on biopsy and skin markers CD1a and S100 staining.

Limited involvement of the skin is left untreated. Extensive and symptomatic cutaneous changes demand prednisolone therapy. PUVA therapy and topical nitrogen beam therapy is useful for recalcitrant ulcerated plaques which are usually unresponsive to systemic treatment. Radiation therapy is also recommended in the

presence of diabetes insipidus and severe pulmonary lesions. Chemotherapy is indicated in case of generalized involvement.

LICHEN SCLEROSUS

Lichen sclerosus, also known as white spot disease or Csillag's disease, is an idiopathic inflammatory dermatosis of unknown etiology. Cases during infancy are uncommon.⁷⁸

Aetiology

Etiology of LS is unknown but several theories are postulated. Occasional finding of autoimmune antibodies and association with autoimmune disease suggests an autoimmune phenomenon. A genetic basis for the disease is also suggested and is supported by increased incidence of HLA DQ 7, HLA BO8-B18 and familial occurrence. Role of *Borrelia burgdorferi* is controversial. Anatomical abnormality (hypospadias) and trauma seem to be contributory factors.⁷⁹

Clinical features

The eruption usually presents with pruritus in the genital area or with constipation in girls and phimosis in boys. The classical clinical picture is characterised by small, porcelain white, sclerotic lesions with accentuation of the veins occurring at any site on genital areas.⁸⁰

Treatment

Symptomatic treatment with emollients and antipruritic medications (sedative antihistaminics) help to control pruritus.

Ultrapotent corticosteroid ointment in the form of clobetasol propionate applied initially once every night for 4 weeks followed by applying every alternate night for 4 weeks and then twice per week for 4 weeks controls pruritus.

Secondary bacterial and candidial infection should be treated. Other modalities include testosterone ointment oral stanazolol, liquid nitrogen cryotherapy, CO₂ laser,

ACTH, acitretin and isotretinoin. Surgery is indicated whenever medical treatment fails and includes circumcision, frenuloplasty, meatotomy and sophisticated plastic repair.^{2,3}

BULLOUS PEMPHIGOID

A rare disease in childhood, bullous pemphigoid is an acquired blistering disorder.

Tense bullae on a normal or erythematous base, with urticarial lesions, are typical.

Common areas of involvement include the flexures, particularly the inner thighs, the forearms the axillae and the diaper area.²

The diagnostic criteria used to diagnose childhood BP so far in literature are:⁸¹

- o Patients 18 years of age or younger with the clinical appearance of tense bullae on erythematous or non-erythematous skin with or without mucous membrane involvement
- o Routine histopathologic study showing subepidermal bulla formation with a variable amount of eosinophils.
- o DIF showing linear deposition of IgG and/or C₃ as the major immunoreactant(s) at the BMZ and/or a positive IIF showing IgG antibodies reactive with antigen(s) at the BMZ.

Treatment

Systemic or topical steroids have been used with good effect.

Tetracycline and nicotinamide are used in combination with steroids.⁸¹

LICHEN STRIATUS

Lichen striatus (LS) is linear papular dermatoses that primarily occurs in children between four months and 15 years of age.⁸²

Lichen striatus presents as unilateral, asymptomatic, flesh-colored to red-brown flat topped papules.

Although various hypotheses such as the course of blood vessels, lymphatics, peripheral nerves, blaschko lines, and direction of body hairs were given for the linear distribution in lichen striatus, none could explain the various clinical morphology of the lesions.

Lesions are more commonly seen over the arms, legs, trunk, and less commonly over the face in decreasing order of frequency. Pruritus is rare and hypopigmentation is a frequent sequelae.

Lichen striatus is a T-cell-mediated inflammatory disease and tacrolimus ointment may be an effective alternative treatment for this disease.⁸²⁻⁸⁴

MATERIALS AND METHODS

The study was undertaken from December 2013 to July 2015. All children below 5 years of age with eruptions involving the diaper area reporting to the Department Of Dermatology, Sri R.L.Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar were enrolled in the study. A written consent was taken from the informant. A detailed history including chief complaints, recent infections, history of allergy, family history of rash like conditions, general physical examination and dermatological examination was done and recorded in a proforma.

Tests such as Tzanck smear, skin biopsy, KOH preparation, culture sensitivity, Gram staining and appropriate hematological investigations were done whenever necessary depending on the presenting condition.

INCLUSION CRITERIA:

-Children below 5 years with eruptions involving diaper area.

EXCLUSION CRITERIA:

-Patients who have taken treatment earlier.

STATISTICAL ANALYSIS:

Data was compiled in Microsoft excel after coding and was analyzed using SPSS 20 version software. Qualitative data was represented by frequencies and proportions and analyzed

OBSERVATIONS AND RESULTS

A total of 130 cases having diaper area lesions fulfilling inclusion criteria attending to dermatology OPD at R. L. Jalappa Hospital and Research centre, Tamaka, Kolar district, Karnataka during the period of December 2013– July 2015 were enrolled in this clinical study.

Table 1: Clinical types of diaper dermatoses in the study

Sl.no	Clinical Dermatoses	Frequency	Percent
1	Irritant contact dermatitis	29	22.3
2	Scabies	19	14.6
3	Impetigo	18	13.8
4	Papular urticaria	15	11.5
5	Varicella	9	6.9
6	Miliaria	8	6.2
7	Seborrhoeic dermatitis	5	3.8
8	Candidiasis	4	3.1
9	Hand foot and mouth disease	3	2.3
10	Acute urticaria	2	1.5
11	Erythema multiforme	2	1.5
12	Gianotti crosti syndrome	2	1.5
13	Hemangioma	2	1.5
14	Lamellar ichthyosis	2	1.5
15	Lichen striatus	2	1.5
16	Vitiligo	1	0.8
17	Tuberous sclerosis(Shagreen patch, Ash leaf macule)	1	0.8
18	Staphylococcal scalded skin syndrome	1	0.8
19	Stevens-johnson syndrome	1	0.8
20	Subcutaneous fat necrosis	1	0.8
21	Nevus depigmentosus	1	0.8
22	Lipoatrophy	1	0.8
23	Acrodermatitis enteropathica	1	0.8
	Total	130	100.0

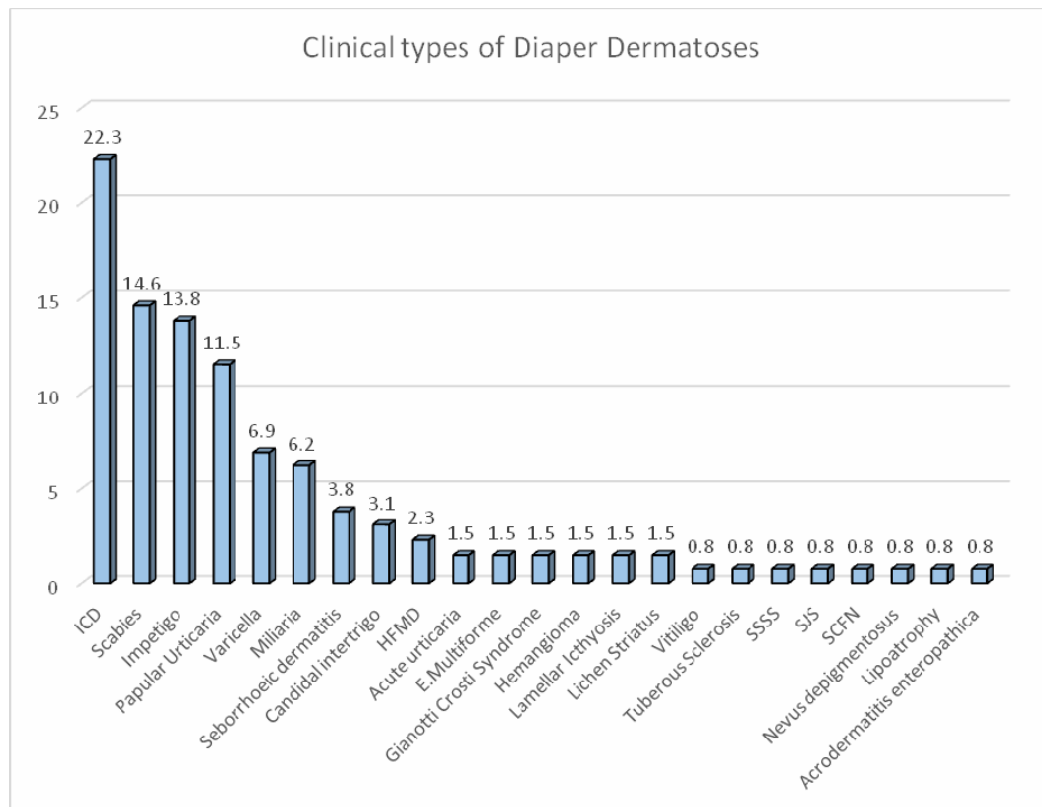


Figure 1: Bar diagram showing clinical types of diaper dermatoses

In the study a total of 23 dermatoses were encountered. ICD (22.3%) was the most common dermatoses, followed by Scabies (14.6%), Impetigo (13.8%), Papular urticaria (11.5%) and other causes as mentioned in table 1.

Table 2: Age group distribution.

		Frequency	Percent
Age Groups	Neonates	4	3.1
	Infants	59	45.4
	Toddlers	43	33.1
	Preschool	24	18.5
	Total	130	100.0

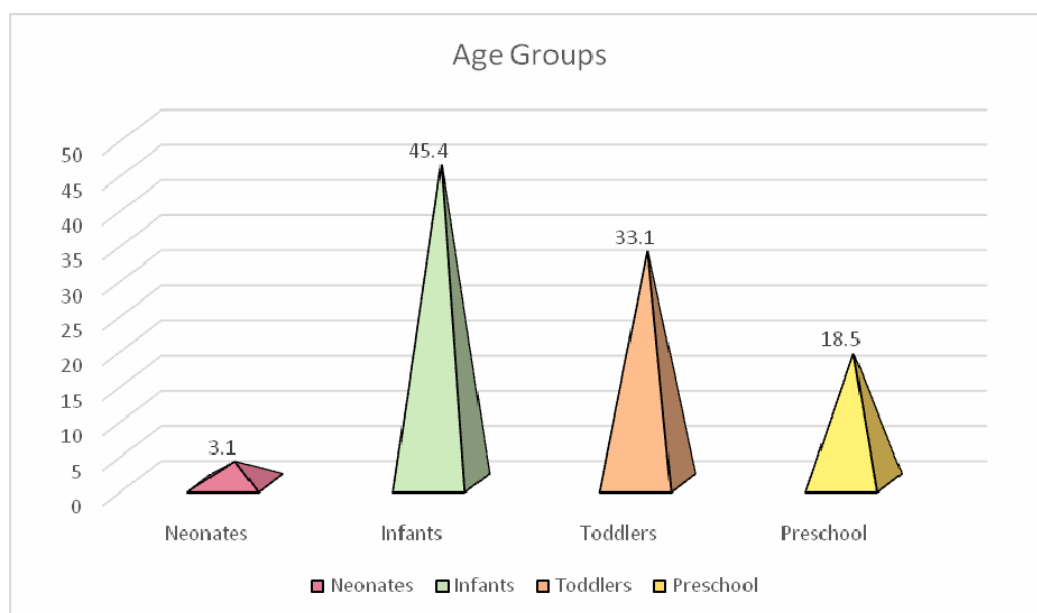


Figure 2: Diagram showing age group distribution

In the study majority of cases were infants (45%), followed by toddlers (33%), pre school children (19%) and least were neonates (3%).

Table 3: Gender distribution of subjects

		Frequency	Percent
Gender	Female	62	47.7
	Male	68	52.3
	Total	130	100.0

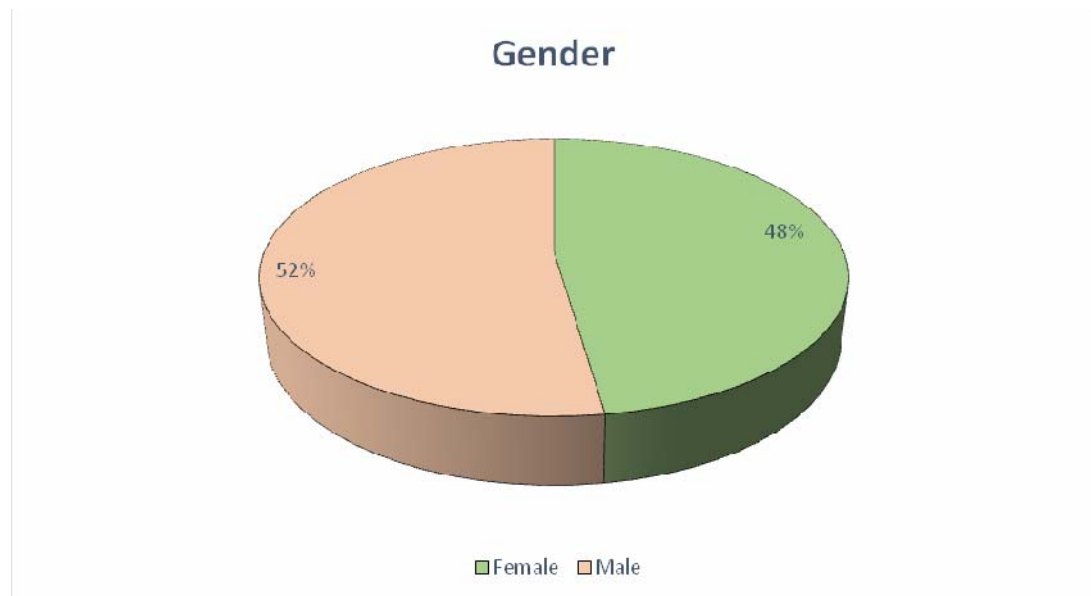


Figure 3: Pie diagram showing gender distribution of subjects

A total of 62 females (47.7%) and 68males (52.3%) were affected, indicating slight male preponderance

Table 4: Distribution of various dermatoses according to age

		Age Group				Total
		Neonates	Infants	Toddlers	Preschool	
Diagnosis	ICD	0	18	11	0	29
	Scabies	0	4	12	3	19
	Impetigo	2	8	3	5	18
	Papular urticaria	0	3	5	7	15
	Varicella	0	4	3	2	9
	Miliaria	1	4	2	1	8
	Seborrhoeic dermatitis	0	4	1	0	5
	HMFD	0	1	1	1	3
	Acute urticaria	0	0	1	1	2
	Candidal intertrigo	0	4	0	0	4
	E.multiforme	0	1	1	0	2
	Gianotti crosti syndrome	0	1	1	0	2
	Hemangioma	0	2	0	0	2
	Lamellar ichthyosis	0	1	0	1	2
	Lichen striatus	0	0	2	0	2
	Vitiligo	0	0	0	1	1
	Tuberous sclerosis	0	0	0	1	1
	SSSS	0	1	0	0	1
	SJS	0	0	0	1	1
	SCFN	1	0	0	0	1
	Nevus depigmentosus	0	1	0	0	1
	Lipoatrophy	0	1	0	0	1
	Acrodermatitis Enteropathica	0	1	0	0	1
Total		4	59	43	24	130

$\chi^2 = 104.00$, $df = 66$, $p = 0.002^{***}$ [84 cells (91.3%) have expected count less than 5]

Table 4A: Distribution of various dermatoses according to age for four major diagnosis in the study.

		Age groups				Total
		Neonates	Infants	Toddlers	Preschool	
Diagnosis	ICD	0	18	11	0	29
	Scabies	0	4	12	3	19
	Impetigo	2	8	3	5	18
	Papular Urticaria	0	3	5	7	15
Total		2	33	31	15	81

$\chi^2 = 31.755$, $df = 9$, $p < 0.0001^{***}$ [7 cells (43.8%) have expected count less than 5]

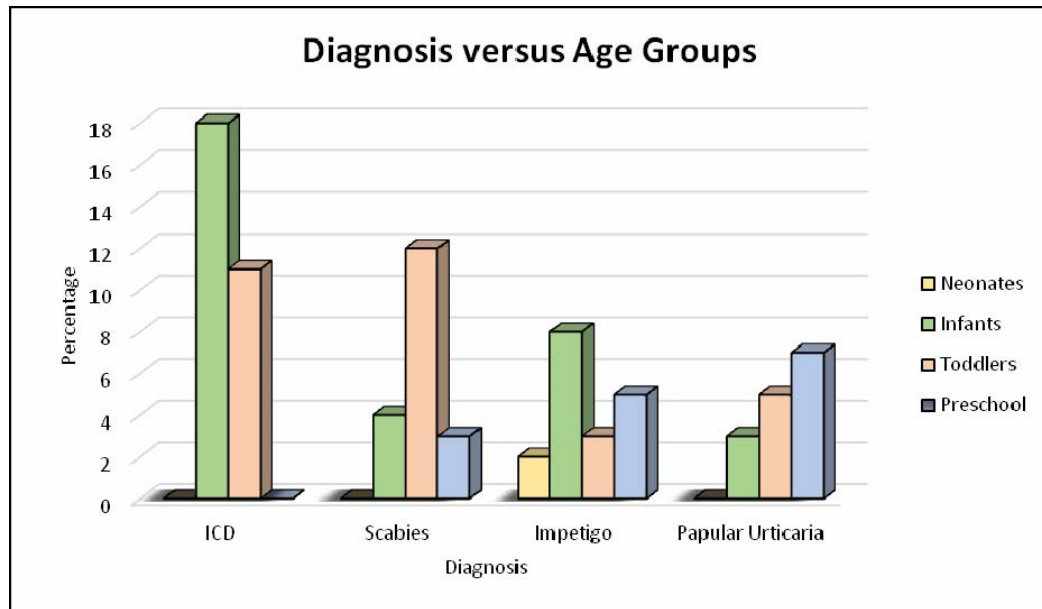


Figure 4: Bar diagram showing association between clinical dermatoses and age group

In the study it was observed that majority of cases were infants and ICD was the most common dermatoses. Scabies was common in toddlers and Papular urticaria was common in preschool children. This observation was highly statistically significant.

Table 5: Distribution of dermatoses according to gender

		Gender		Total
		Female	Male	
Diagnosis	ICD	14	15	29
	Scabies	4	15	19
	Impetigo	11	7	18
	Papular urticaria	9	6	15
	Varicella	6	3	9
	Miliaria	1	7	8
	Seborrhoeic dermatitis	2	3	5
	HMFD	1	2	3
	Acute urticaria	0	2	2
	Candidal intertrigo	2	2	4
	E.multiforme	2	0	2
	Gianotti crosti syndrome	1	1	2
	Hemangioma	0	2	2
	Lamellar ichthyosis	1	1	2
	Lichen striatus	2	0	2
	Vitiligo	1	0	1
	Tuberous sclerosis	1	0	1
	SSSS	0	1	1
	SJS	1	0	1
	SCFN	1	0	1
	Nevus depigmentosus	1	0	1
	Lipoatrophy	0	1	1
	Acrodermatitis enteropathica	1	0	1
Total		62	68	130

$\chi^2 = 29.711$, $df = 22$, $p = 0.126$

Table 5A: Distribution of dermatoses according to gender in four common conditions

		Gender		Total
		Female	Male	
Diagnosis	ICD	14	15	29
	Scabies	4	15	19
	Impetigo	11	7	18
	Papular urticaria	9	6	15
Total		38	43	81

$\chi^2 = 7.61$, $df = 3$, $p = 0.055$

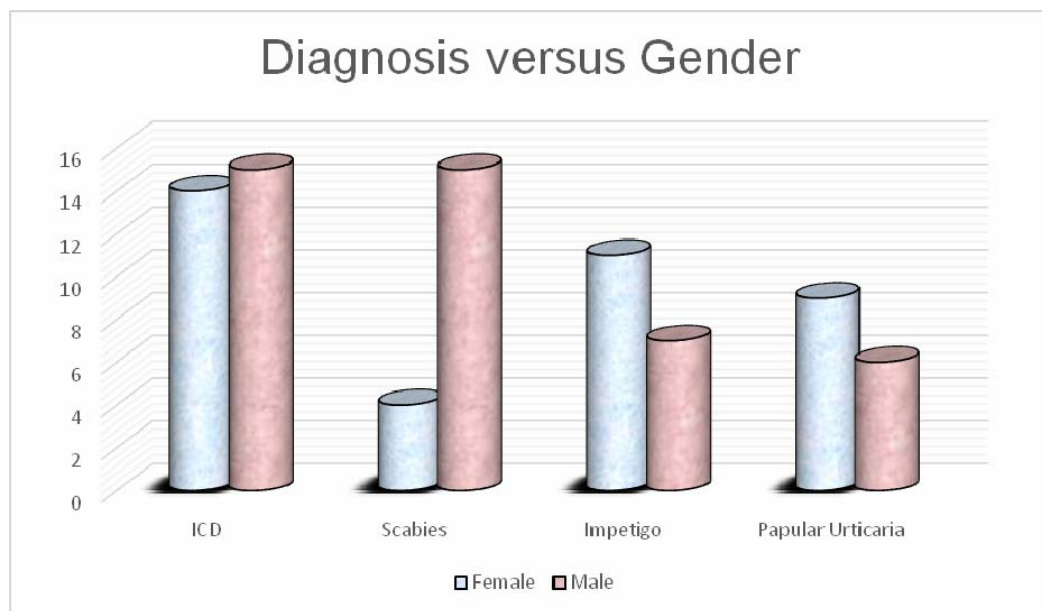


Figure 5: Diagram showing association between gender and diagnosis

In the study majority of ICD and scabies cases were seen in males and impetigo and papular urticaria were common in females. This observation was not statistically significant.

Table 6: Distribution of Subjects according to presenting complaints

Presenting complaint	Patients (n =130)	Percentage (%)
Reddish lesions	70	53.8
Itching	45	34.6
Scaling	28	21.5
Fluid filled lesions	23	17.7
Red raw area	13	10
Burning sensation	4	3.1
Pigmentation	2	1.5
Pain	0	0
Asymptomatic	6	4.6

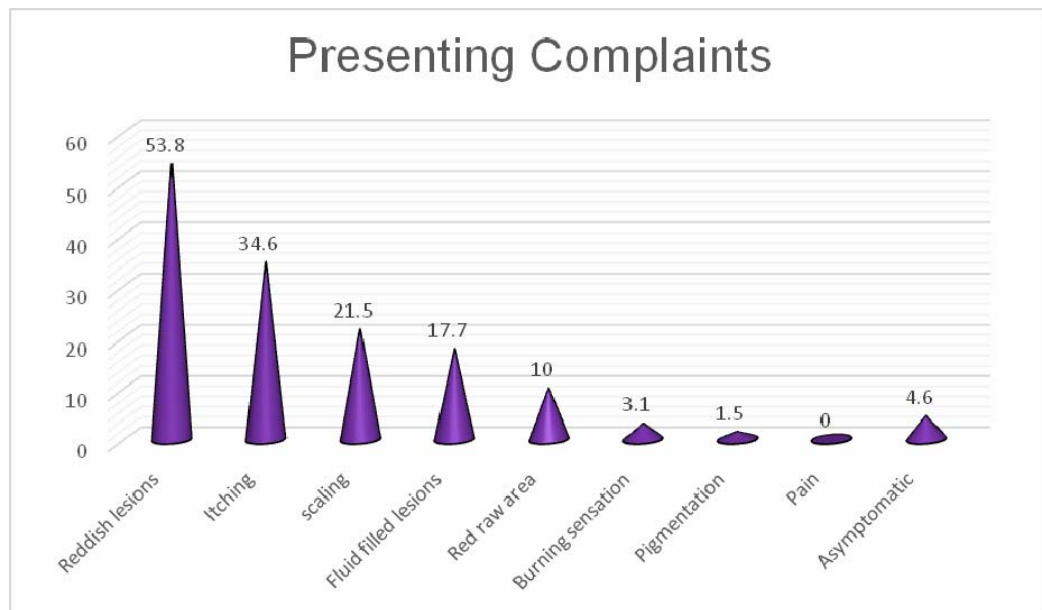


Figure 6: Diagram showing presenting symptoms in the study subjects

Majority of subjects presented with reddish lesions (53.8%), followed by itching (34.6%), scaling (21.5%), 17.7% with fluid filled lesions and other symptoms such as red raw area, burning sensation, pigmentation.

Table 7: Distribution of subjects according to duration

		Frequency	Percent
Duration	Since Birth	6	4.6
	< 6 weeks	121	93.1
	> 6 weeks	3	2.3
	Total	130	100.0

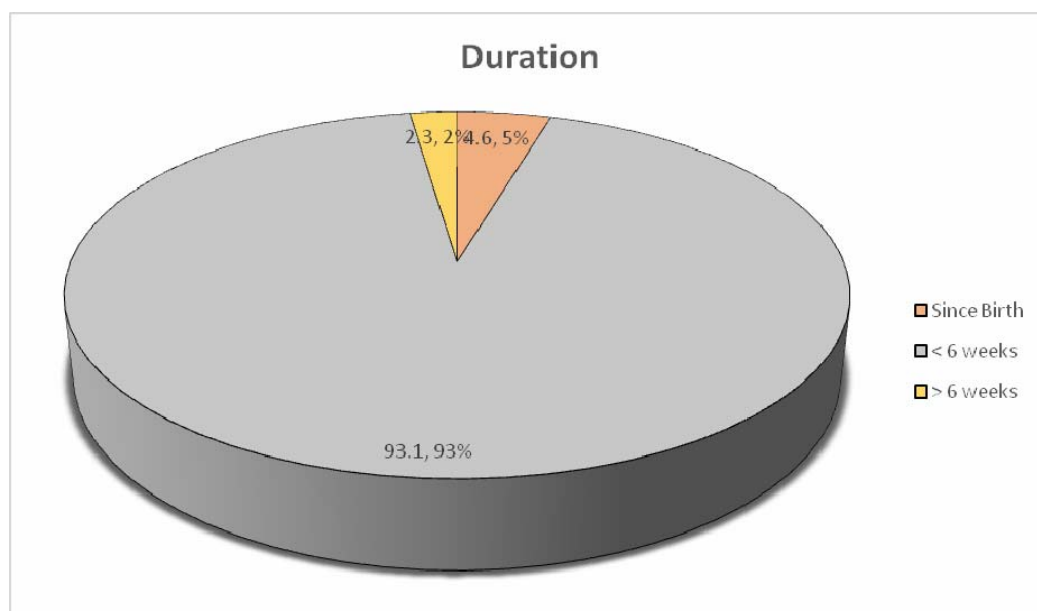


Figure 7: Pie diagram showing distribution of subjects according to duration

Majority of lesions were <6weeks in duration (93.1%), 4.6% had lesions since birth and only 2.3% of them had duration >6 weeks.

Table 8: Distribution according to initial site involved

		Frequency	Percent
Initial site	Diaper area	49	37.7
	Upper limbs	26	20.0
	Trunk	21	16.2
	Lower limbs	12	9.2
	Face	7	5.4
	Scalp	7	5.4
	Neck	4	3.1
	All Sites	2	1.5
	Oral cavity	2	1.5
	Total	130	100.0

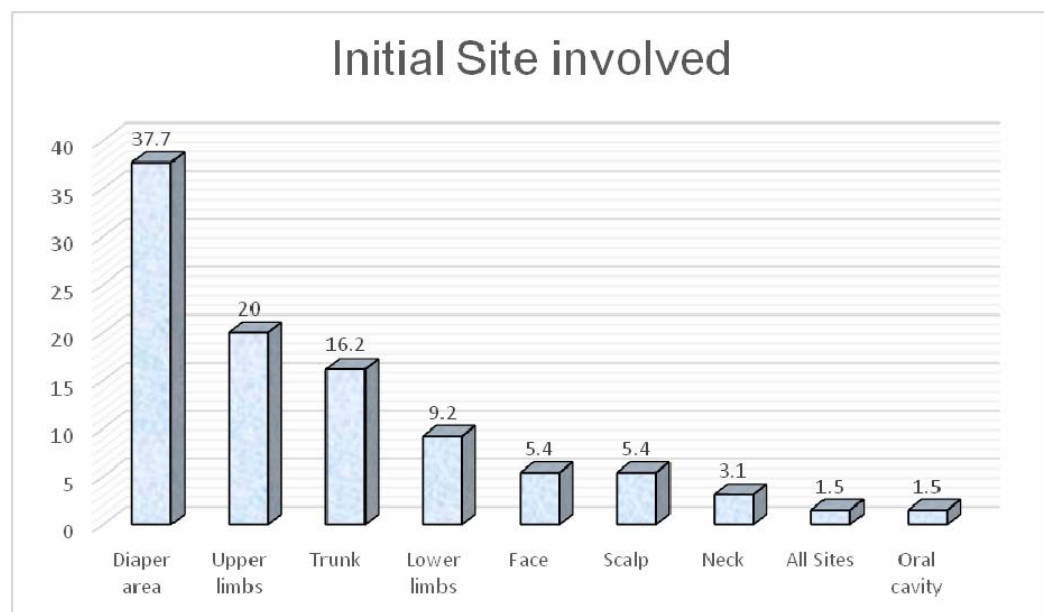


Figure 8: Diagram showing initial site involvement

In majority of subjects initial site of involvement was diaper area in 37.7% patients, followed by upper limbs in 20%, trunk in 16.2%, lower limbs in 9.2%. Exclusive diaper area involvement was seen in 41(31.53%) cases.

Table 9: Examination of diaper area site

Area	Frequency	Percentage
Gluteal area	85	65.4
Inner aspect of thigh	60	46.2
Lower abdomen	56	43.1
Genitalia	28	21.5
Lower lumbar region	23	17.7

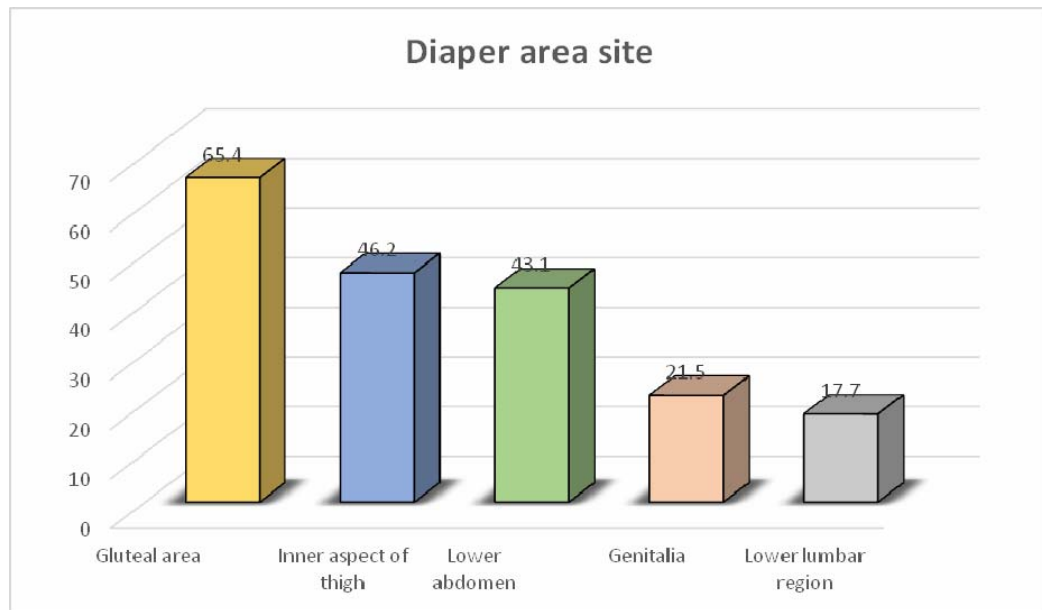


Figure 9: Bar diagram showing diaper area site

In Diaper area gluteal region(65.4%) was the commonest site, followed by inner aspect of thigh (46.2%), lower abdomen (43.1%), genitalia (21.5%) and lower lumbar region (17.7%).

Table 10: Morphology of primary lesion in diaper area

	Primary lesion	Patients (n=130)	Percentage (%)
A	Macule	42	32.3
B	Papule	49	37.7
C	Plaque	12	9.2
D	Pustule	11	8.5
E	Nodule	3	2.3
F	Cyst	0	0
G	Vesicle	25	19.2
H	Bullae	9	6.9
I	Target lesion	2	1.5
J	Burrow	1	0.8
K	Wheals	2	1.5

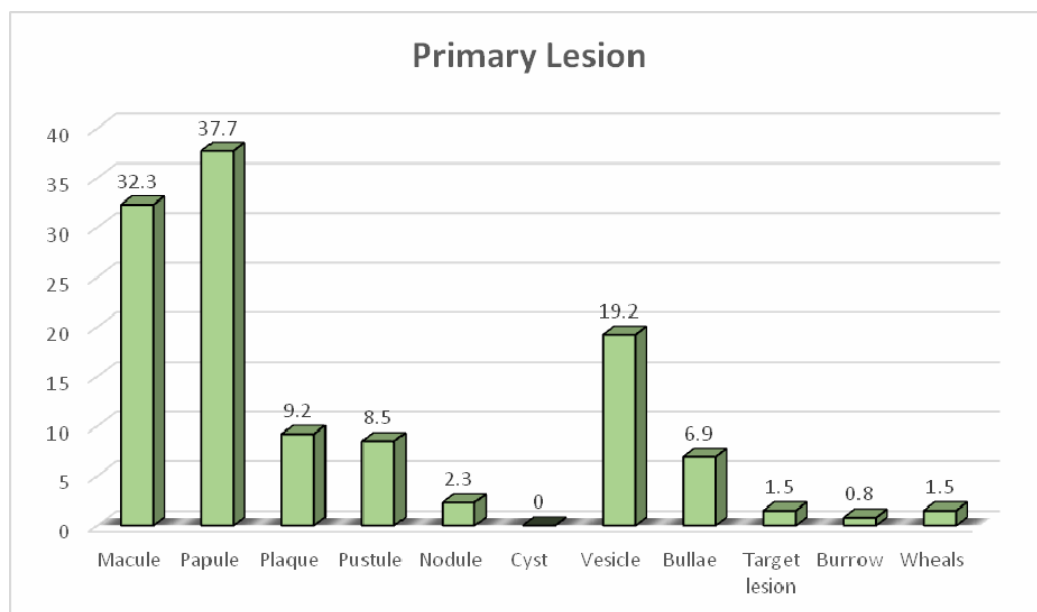


Figure 10: Bar diagram showing morphology of primary lesion

Most common primary lesion was papule (37.7%), followed by macules (32.3%), vesicle (19.2%) and other lesions were less common.

Table 11: Morphology of Secondary lesion in diaper area

	Secondary lesion	Frequency	Percentage
A	Scaling	36	27.7
B	Crusting	18	13.8
C	Ulcer	0	0
D	Erosion	30	23.1
E	Excoriation	42	32.3
F	Hyperpigmentation	3	2.3
G/H	Hypo/Depigmentation	4	3.1

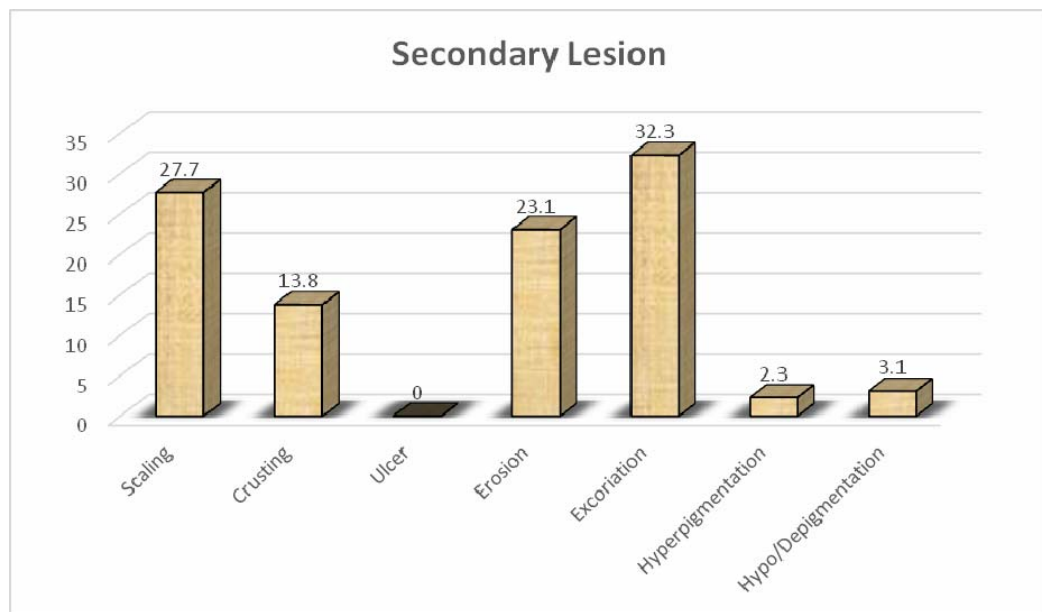


Figure 11: Bar diagram showing morphology of secondary lesions

In the secondary lesion majority of them had excoriation (32.3%), scaling (27.7%), erosion (23.1%) and 13.8% had crusting.

Table 12: Demographic and disease characteristics of Irritant contact dermatitis

(ICD) ICD (n=29)

AGE GROUP

	Male	Female	Total
Gender	15 (51.7%)	14 (48.3%)	29

Age group

	Male	Female	Total
<1 Yr	11	7	18
1 to <3 Yrs	4	7	11
Total	15	14	29

$\chi^2 = 1.675$, df = 1, p = 0.196

SITE

	Male	Female	Total	p value
Gluteal	14	8	22	0.023
Inner Aspect of Thigh	8	9	17	0.550
Lower Abdomen	6	7	13	0.588
Genitalia	4	4	8	0.909
Lower Lumbar	1	2	3	0.501

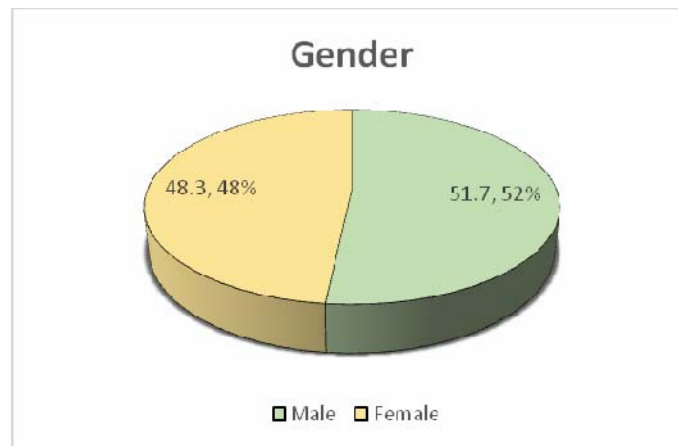


Figure 12: Gender distribution of ICD cases

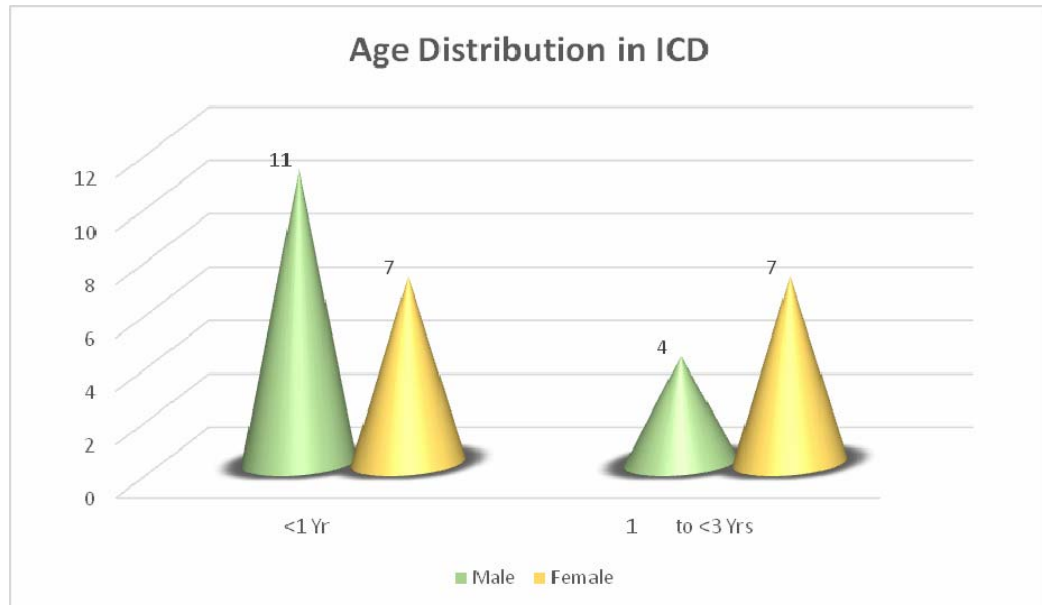


Figure 13: Age distribution in ICD cases

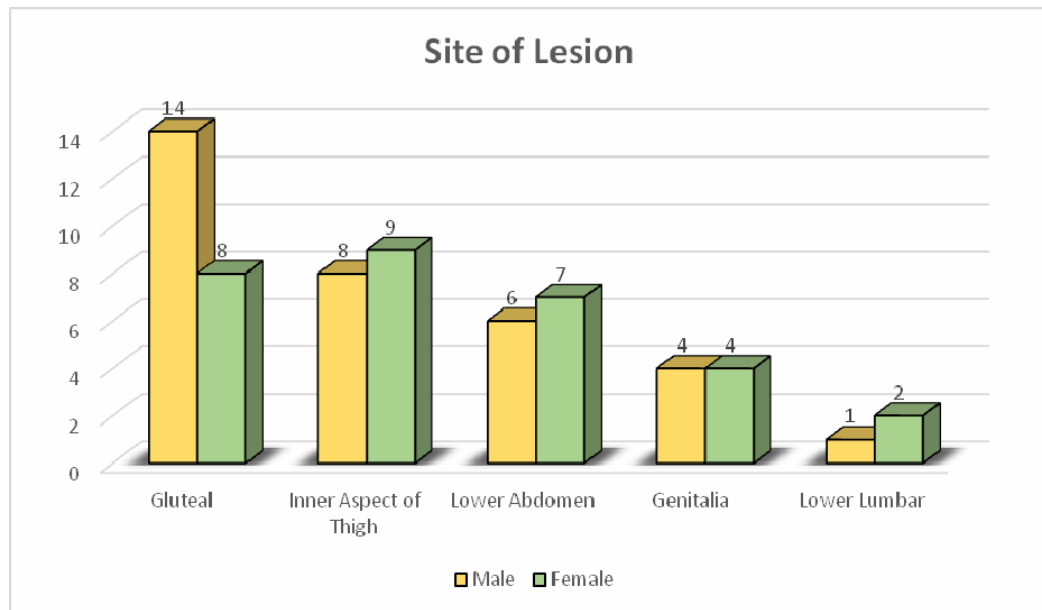


Figure14: Bar diagram showing site of lesion in ICD cases

- ❖ The incidence of ICD in our study is 22.3%; seen in 15 males (51.7%) and 14 females (48.3%).
- ❖ Commonest region of diaper area involved was gluteal area followed by inner aspect of thigh.
- ❖ ICD was more prevalent among infants than among toddlers

Table 13: Demographic and disease characteristics of Scabies

Scabies (n=19)

	Male	Female	Total
Gender	15 (78.9%)	4 (21.1%)	19

Age group

	Male	Female	Total
<1 Yr	3	1	4
1 to <3 Yrs	9	3	12
3 to 5 Yrs	3	0	3

$\chi^2 = 0.950$, $df = 2$, $p = 0.622$

Site of Lesion

	Male	Female	Total	p value
Lower abdomen	6	2	8	0.719
Gluteal	6	2	8	0.719
Genitalia	10	2	12	0.539
Inner aspect of thigh	7	0	7	0.086

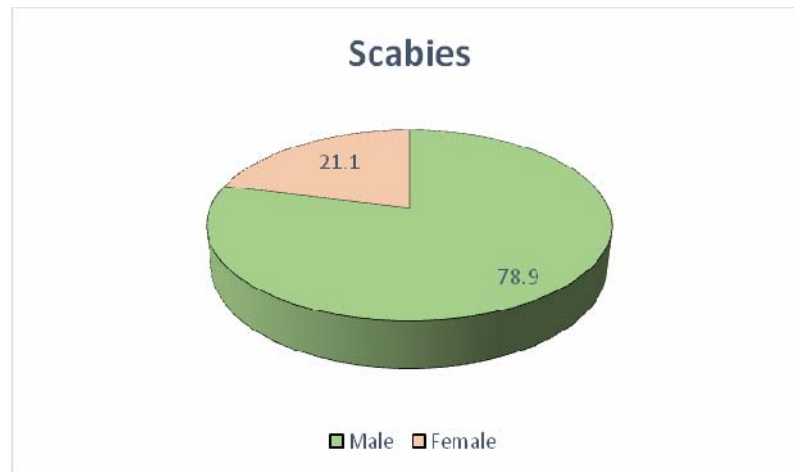


Figure 15: Gender distribution of scabies patients

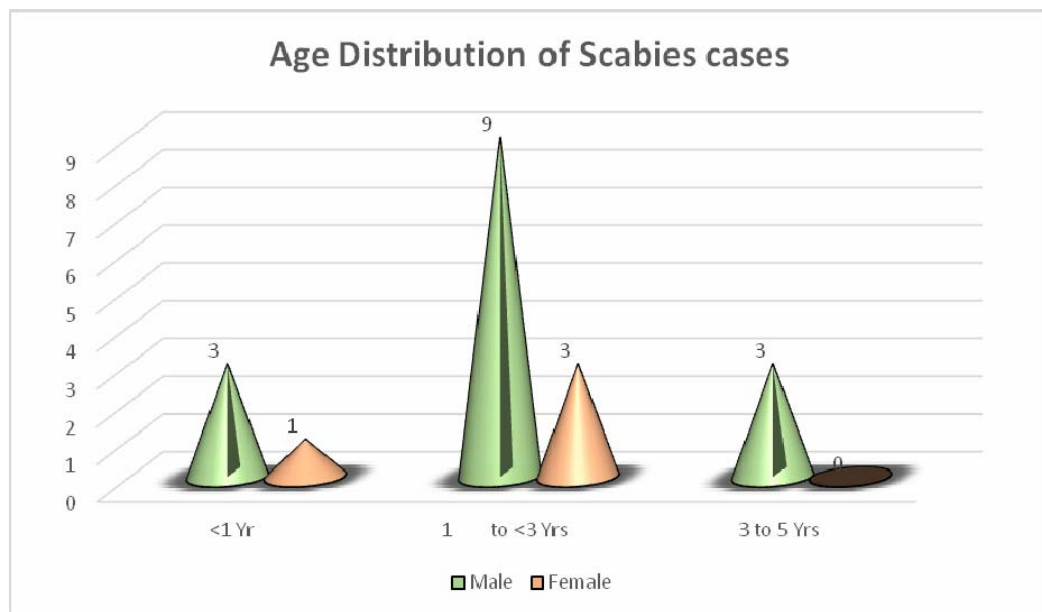


Figure 16: Age distribution of scabies cases

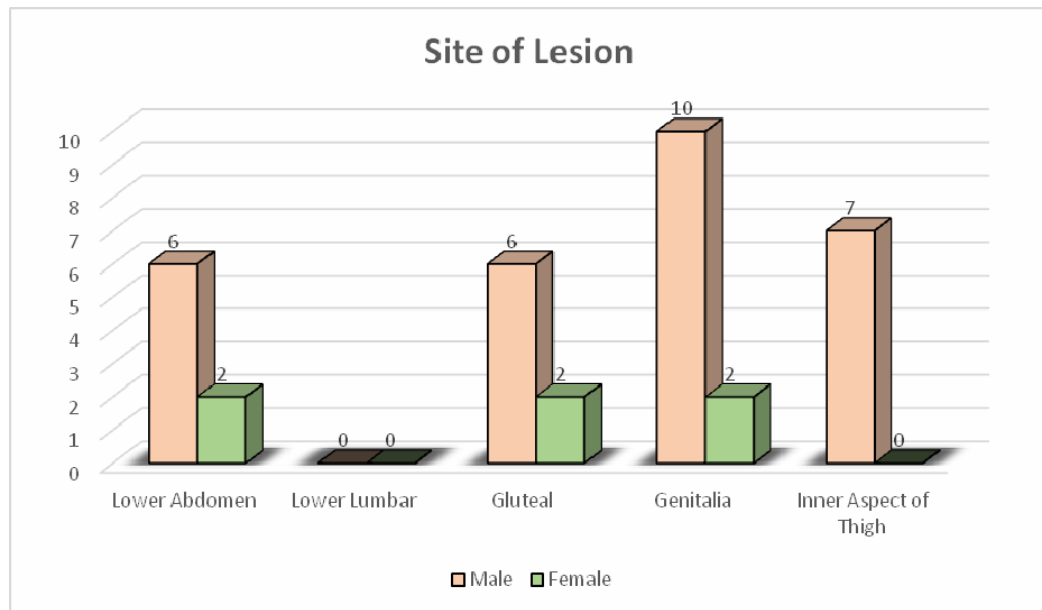


Figure 17: Bar diagram showing site of lesion in scabies

- ❖ The incidence of scabies in our study is 14.6%; seen in 15 males (78.9%) and 4 females (21.1%).
- ❖ Commonest region of diaper area involved was genitalia followed by lower abdomen and gluteal area.
- ❖ Scabies was more prevalent among toddlers, followed by infants and preschool children.

Table 14: Demographic and disease characteristics of Impetigo

	Male	Female	Total
Gender	7 (38.8%)	11 (61.2%)	18

Age group

	Male	Female	Total
<28 days	2	0	2
<1 yr	2	6	8
1 to <3 yrs	0	3	3
3 to 5 yrs	3	2	5

$\chi^2 = 6.639$, $df = 3$, $p = 0.084$

Site

	Male	Female	Total	p value
Lower Abdomen	5	5	10	0.280
Lower Lumbar	1	0	1	0.197
Gluteal	6	9	15	0.829
Inner Aspect of Thigh	2	5	7	0.474

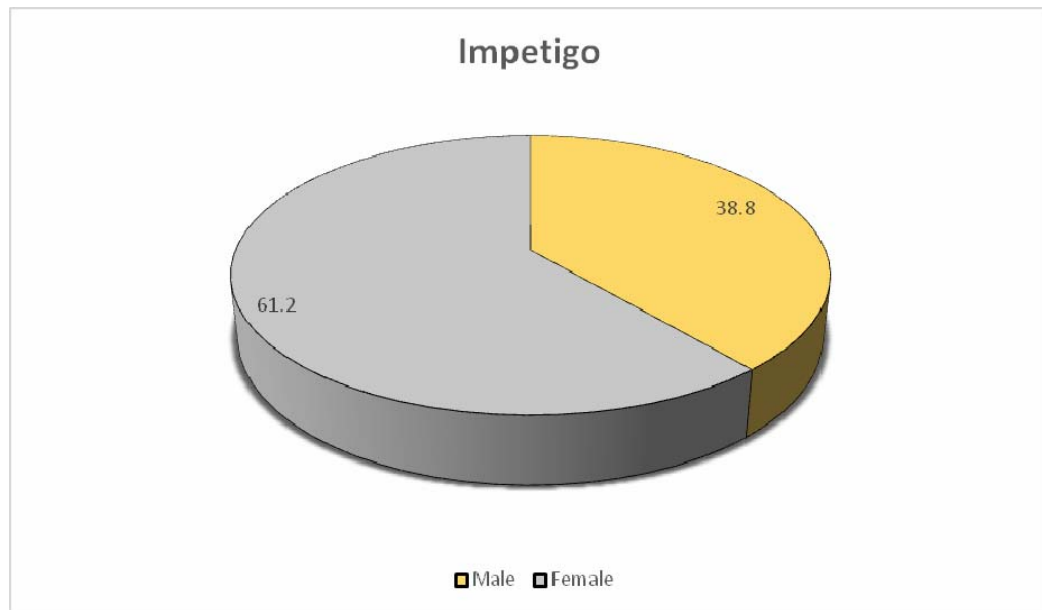


Figure 18: Gender distribution of impetigo cases

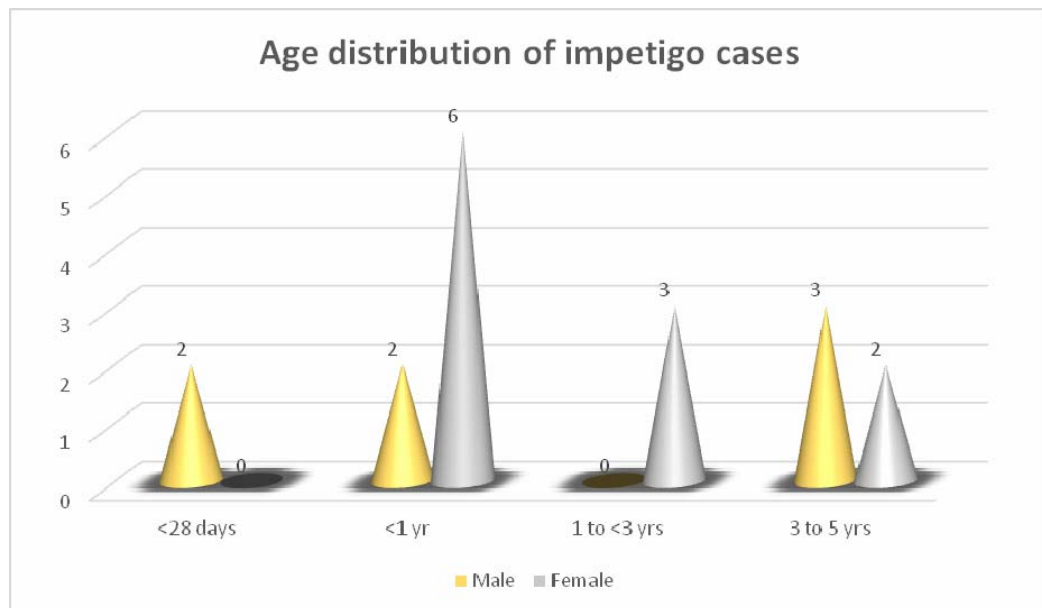


Figure 19: Age distribution of impetigo cases

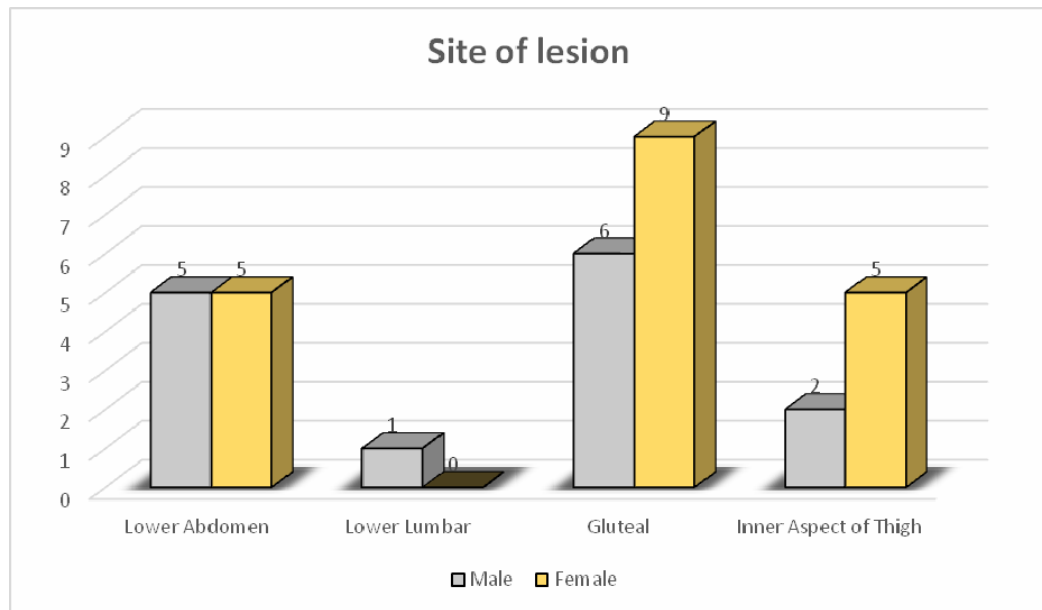


Figure 20: Bar diagram showing site of lesion in impetigo cases

- ❖ The incidence of impetigo in our study is 13.8%; seen in 7 males (38.8%) and 11 females (61.2%).
- ❖ Commonest region of diaper area involved was gluteal area followed by lower abdomen.
- ❖ Impetigo was more prevalent among infants, followed by preschool children and toddlers.

Table 15: Demographic and disease characteristics of papular urticarial

	Male	Female	Total
Gender	6 (40%)	9 (60%)	15

Age group

	Male	Female	Total
<1 Yr	3	0	3
1 to <3 Yrs	1	4	5
3 to 5 Yrs	2	5	7

$\chi^2 = 5.714$, $df = 2$, $p = 0.057$

Site

	Male	Female	Total	p value
Lower Abdomen	1	5	6	0.132
Lower Lumbar	1	5	6	0.132
Gluteal	5	5	10	0.264
Inner Aspect of Thigh	1	3	4	0.475

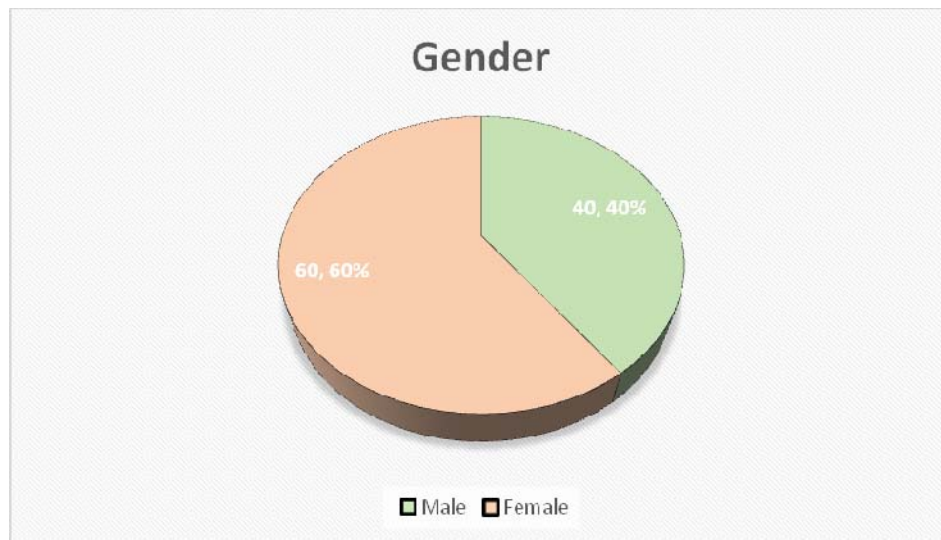


Figure 21: Gender distribution in papular urticaria cases

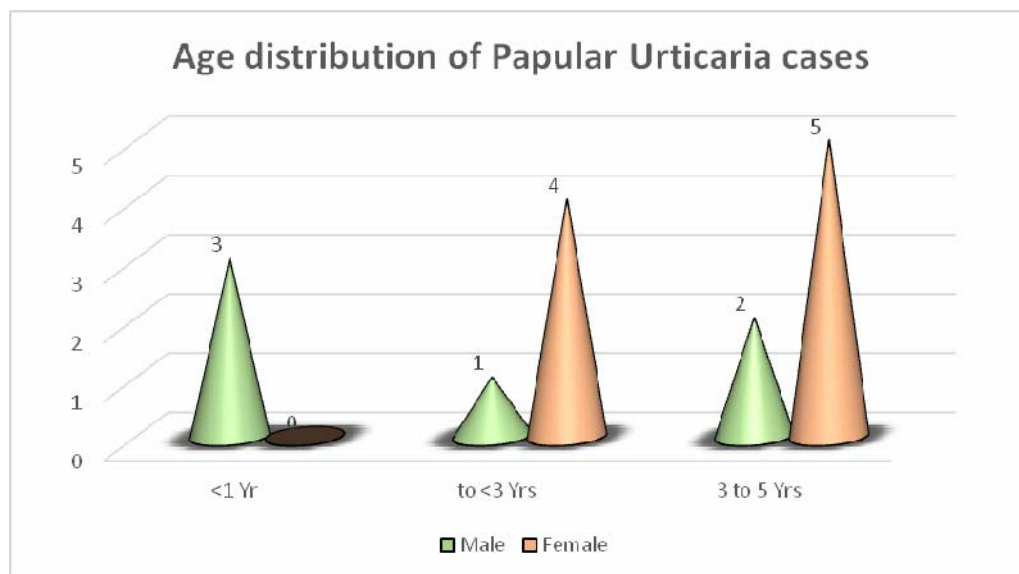


Figure 22: Age distribution of papular urticaria

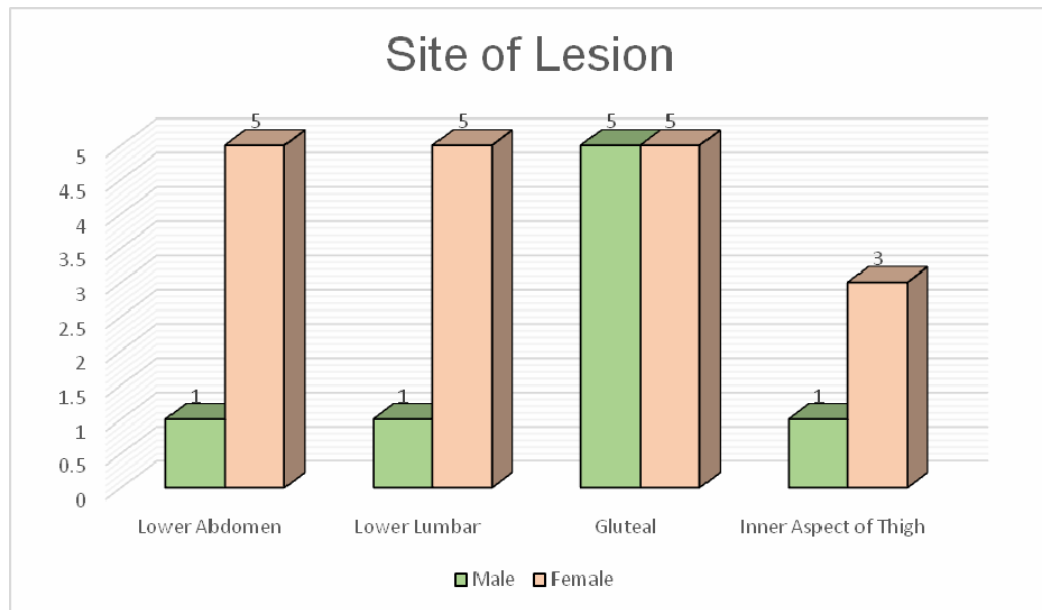


Figure 23: Bar diagram showing site of lesion in papular urticaria cases

- ❖ The incidence of papular urticaria in our study is 11.5%; seen in 6 males (40%) and 9 females (60%).
- ❖ Commonest region of diaper area involved was gluteal area followed by lower abdomen and lower lumbar region.
- ❖ Papular urticaria was more prevalent among preschool children, followed by toddlers and infants.

TABLE 16- Aetiology of diaper area dermatoses

Aetiology	Total patients	Percentage
Inflammation	34	26.15%
Infection	37	28.46%
Arthropod bite	34	26.15%
Congenital/Genetic	7	5.38%
Cutaneous vascular response	4	3.07%
Miscellaneous	14	10.76%
Total	130	100%

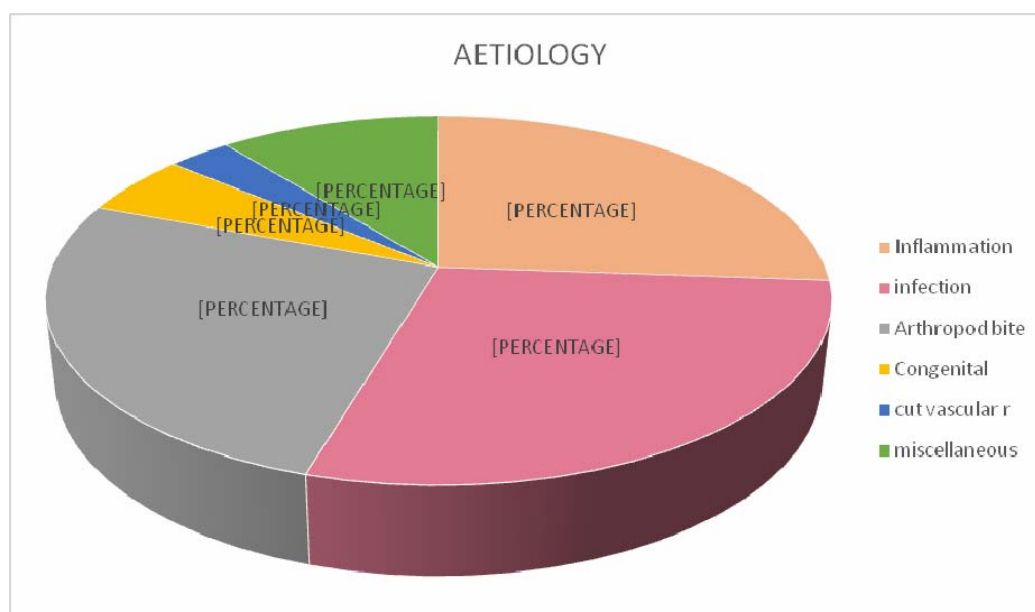


Figure 24: Pie diagram showing aetiology of diaper area dermatoses.

The most common aetiological factor was infection(28.46%) followed by inflammation and arthropod bite.

TABLE 17-Aetiology-Infections

CONDITION INFECTION	AETIOLOGY	PATIENTS	PERCENTAGE
			28.46%
BACTERIA		19	14.61%
Impetigo	Grams stain-S.Aureus	15	13.8%
	-B H Streptococcus	03	
SSSS	Grams stain-S.Aureus	01	0.8%
VIRAL		14	10.76%
Varicella	Tzanck smear-MGC	09	6.9%
HFMD		03	2.3%
GCS		02	1.5%
FUNGAL		04	3.1%
Candidiasis	KOH-C.albicans	04	3.1%

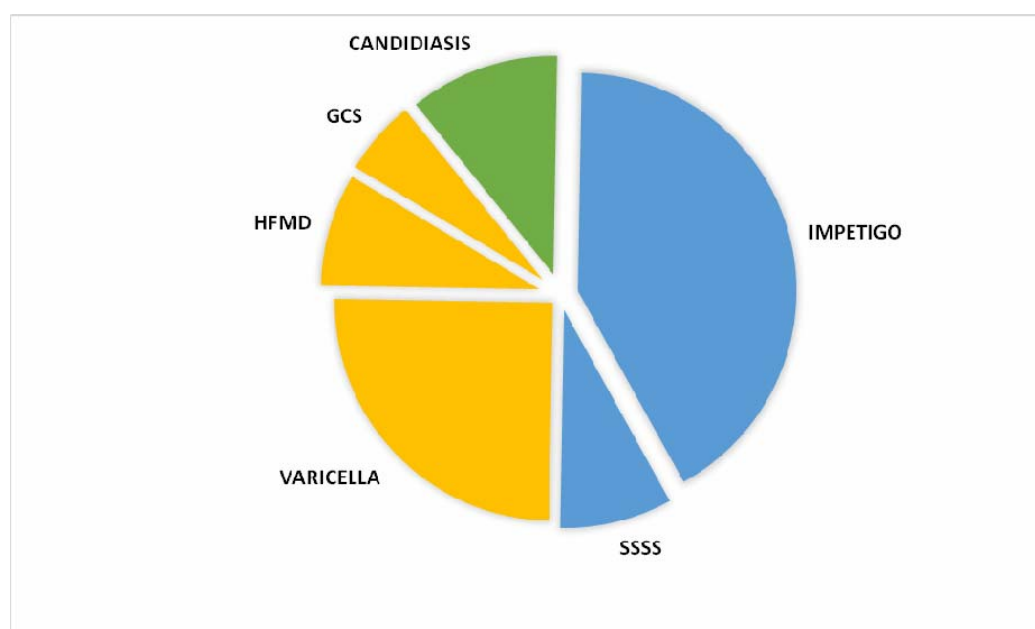


Fig 25-Pie diagram showing infectious aetiology.

TABLE 18-Aetiology-Inflammatory,congenital and other aetiological factors.

CONDITION	AETIOLOGY	PATIENTS	PERCENTAGE
INFLAMMATORY		34	26.15%
ICD	Diaper Diarrohea	24 5	22.3%
SD		5	3.8%
CONGENITAL/GENETIC		07	5.38%
Lamellar ichthyosis	-	2	1.5%
Hemangioma	-	2	1.5%
SCFN	Fetal hypoxia	1	0.8%
Nevus.depigmentosus	-	1	0.8%
Tuberous sclerosis	-	1	0.8%
ARTHROPOD BITE		34	26.15%
Scabies	S.scabei(KOH-mite)	19	14.6%
Papular Urticaria	-	15	11.5%
CUTANEOUS VASCULAR RESPONSE		04	3.07%
Acute urticaria	Post varicella NSAID	1 1	1.5%
Erythema multiforme	HSV	2	1.5%
MISCELLANEOUS		14	10.76%
Miliaria		8	6.2%
SJS	phenobarbitone	1	0.8%
Vitiligo	-	1	0.8%
Lipoatrophy	trauma	1	0.8%
Lichen striatus	-	2	1.5%
Acrodermatitis enteropathica	zinc deficiency(low zinc levels)	1	0.8%

AETIOLOGY

1. Infectious aetiology was the commonest, seen in 37(28.46%) children.

- ❖ Bacterial infection(14.61%) was most common followed by viral(10.76%) and fungal infection(3.1%).
- ❖ Out of bacterial infection, impetigo was the commonest entity seen in 19(14.61%) children followed by SSSS seen in 1 (0.8%) patient and in impetigo, staphylococcus aureus was the causative organism in 15 cases and Group A beta hemolytic streptococcus, in 3 cases.
- ❖ Out of various viral infections , varicella was seen in majority 9(6.9%) of the cases and multinucleate giant cells were seen on Tzanck smear. Hand foot mouth disease and gianotti crosti syndrome were seen in 3(2.3%) and 2(1.5%) patients respectively.
- ❖ Among Fungal infections candidiasis was seen in 4(3.1%) patients and KOH and Fungal culture was positive for candida albicans.

2. Inflammatory aetiology was seen in 34(26.15%) patients.

- ❖ ICD(29, 22.3%) secondary to diapers was seen in 24 patients and 3 cases was secondary to diarrhoea.
- ❖ Seborrhoeic dermatitis was seen in 5 (3.8%) patients.

3. Among arthropod bites scabies was seen in 19 (14.6%), and papular urticaria was seen in 15(11.5%) patients.

4. Congenital/genetic condition was seen in 7 (5.38%) patients.

- ❖ Autosomal recessive lamellar ichthyosis was seen in 2(1.5%) patients.
- ❖ Vascular malformation, hemangioma was seen in 2(1.5%) patients.
- ❖ Subcutaneous fat necrosis secondary to fetal hypoxia was seen in 1 (0.8%) patient.

- ❖ Nevus depigmentosus and Tuberous sclerosis (Ash leaf macule and shagreen patch) in the diaper area was seen in 1 (0.8%) patient each.

5. Lesions secondary to cutaneous vascular response was seen in 4(3.07%) patients.

- ❖ Erythema multiforme secondary to HSV infection was seen in 2(1.5%) patients
- ❖ Acute urticaria involving diaper area was seen in 2(1.5%) patients one was secondary to varicella infection and the other was due to ibuprofen.

6. Among the miscellaneous conditions

- ❖ Miliaria was seen in 8 (6.2%) patients.
- ❖ SJS due to phenobarbitone was seen in 1(0.8%) patient.
- ❖ Lichen striatus was seen in 2(1.5%) patients.
- ❖ Lipoatrophy secondary to trauma was seen in 1 (0.8%) patient.
- ❖ Acrodermatitis enteropathica due to zinc deficiency was seen in 1 (0.8%) patient.

DISCUSSION

Dermatoses in diaper area are very common in general practice. They occur as primary diseases of the diaper area, as part of a generalised inflammatory skin disease, or as part of a systemic disease.

Many common conditions of diaper area have similar symptoms, clinical features and in some cases it may be a sign of a more substantial medical problem, thereby complicating diagnosis; so accurate diagnosis is critical to determine proper treatment.

There are no comprehensive studies on the various clinical patterns of diaper area dermatoses in the Indian and Western literature. Hence, clinical and etiological studies are required to assist in the selection of appropriate treatment and the prevention of important complications of diaper area dermatoses.

In our study, a total of 130 children below 5 years with diaper area lesions were enrolled. Detailed history was taken; clinical examination was done along with appropriate investigations. Predisposing factors, preexisting co-morbidities and other associated conditions were enquired about and noted. The following observations are made and are compared with other similar studies.

A total of 23 various dermatoses were recorded in 130 children below 5 years of age.

In our study, a total of 62 females (47.7%) and 68 males (52.3%) were affected, indicating slight male preponderance which is in concordance with the study on the pattern of paediatric dermatoses by Balai and his team.⁸⁵

In the present study, majority of cases were infants (45%), followed by toddlers (33%), pre school children (19%) and least were neonates (3%). Whereas toddlers (42%) outnumbered infants and pre school children in another study.⁸⁶

Majority of our patients (93.1%) had symptoms of less than 6 weeks duration which is consistent with other studies.^{85,87}

In majority of subjects initial site of involvement was diaper region in 37.7% patients. Exclusive diaper area involvement was seen in 41(31.53%) cases.

The most common diaper area involved in our study population is gluteal region(65.4%) , followed by inner aspect of thigh (46.2%), lower abdomen (43.1%), genitalia (21.5%) and lower lumbar region (17.7%).

Various morphological types were observed in our study, with papule (37.7%) and excoriation (32.3%) being the predominant primary and secondary types respectively.

These findings are consistent with the morphological types seen in previous studies of scabies and papular urticaria.^{61,62,67}

Among the various etiologies encountered, infections (28.46%) were most common followed by inflammation (26.15%) and arthropod bite (26.15%), which were similar to various other studies on dermatoses in paediatric population.^{85,86,88}

Of the infective dermatoses, bacterial infection (14.61%) was most common followed by viral (10.76%) and fungal infection (3.1%) which was similar to a study done in south west Rajasthan.⁸⁵

While viral infections out numbered bacterial and fungal infections in another study.⁸⁹

In the present study, impetigo (13.8%) was the commonest bacterial infection like many other studies.^{45,46,85}

Impetigo was seen in 7 males (38.8%) and 11 females (61.2%). Commonest region of diaper area involved was gluteal area followed by lower abdomen. Impetigo was more prevalent among infants, followed by preschool children and toddlers.

Whereas impetigo occurred much less commonly in infants compared to other age groups in another study.⁸⁶

Candidiasis (3.1%) was the most common fungal infection, which is consistent with other study.⁸⁷

Out of various viral infections, varicella was seen in majority (6.9%) of the cases in our study whereas, herpes simplex was the commonest viral infection in another study.³

Pattern of inflammatory disorders revealed ICD (22.3%) to be the commonest, followed by seborrhoeic dermatitis (3.8%). ICD was seen in 15 males (51.7%) and 14 females (48.3%). Commonest region of diaper area involved was gluteal area followed by inner aspect of thigh and was more prevalent among infants. Seborrhoeic dermatitis was seen in 3 males(60%) and 2 females(40%). Similar findings was documented by Hayden and his team.⁹⁰ whereas infantile seborrhoeic dermatitis (10.49%) was most common in another study.⁸⁹

Among arthropod bites scabies was seen in 19 (14.6%), and papular urticaria was seen in 15 (11.5%) children.

The incidence of scabies in our study is 14.6%; seen in 15 males (78.9%) and 4 females (21.1%). Commonest region of diaper area involved was genitalia followed by lower abdomen and gluteal area. Scabies was more prevalent among toddlers, followed by infants and preschool children. Almost a similar occurrence (10.61%) has been reported in other studies.^{85,90} Whereas no significant age difference was found in the study by Banerjee and his team.⁸⁶

The incidence of papular urticaria in our study is 11.5%; seen in 6 males (40%) and 9 females (60%). Commonest region of diaper area involved was gluteal area followed by lower abdomen and lower lumbar region. Papular urticaria was more

prevalent among preschool children, followed by toddlers and infants. Papular urticaria was more frequent in rainy season.

In a study on papular urticaria by Naeem and his team, 13.8% paediatric patients presented with papular urticaria and there was no significant difference between gender of the children, and lesions of papular urticaria were commonly present over exposed parts of the body. However, majority of patients had lesions of papular urticaria over covered parts of the body, either involving only covered parts or in addition to involvement of exposed parts of the body and lower trunk was the most commonly involved covered site of the body.⁶⁷

In our study, miliaria was seen in 8 (6.2%) patients ,out of which 7 were males (87.5%) and 1 female(12.5%).It was more prevalent among infants(50%) . However in another study by Banerjee , miliaria was found to be more prevalent among toddlers than infants..

Of the nutritional disorders, acrodermatitis enteropathica was the only entity recorded in 1 (0.8%)patient. A higher incidence (3.6%) of acrodermatitis enteropathica has been reported in a study from Karachi.⁹¹

Other conditions recorded in our study with diaper area dermatoses in children were Hand foot and mouth disease (3 cases , 2.3%), Acute urticaria (2 cases, 1.5%) , Erythema multiforme (2 cases ,1.5%), Gianotti crosti syndrome(2 cases ,1.5%) ,Hemangioma(2 cases ,1.5%) , Lamellar ichthyosis (2 cases ,1.5%), Lichen striatus(2 cases ,1.5%), Vitiligo(1case, 0.8%), Tuberous sclerosis(Shagreen patch, Ash leaf macule) (1case, 0.8%), Staphylococcal scalded skin syndrome(1case, 0.8%), Stevens-johnson syndrome(1case, 0.8%),Subcutaneous fat necrosis (1case, 0.8%), Nevus depigmentosus (1case, 0.8%) Lipoatrophy (1case, 0.8%) which are not reported in any other study.

CONCLUSION

Our study brings to light various dermatoses in diaper area in paediatric population. Three disorders which were very common were irritant contact dermatitis, scabies and impetigo.

This study emphasizes the fact that many of the common dermatoses can significantly involve the diaper area and can have overlapping symptoms and presentations making the diagnosis difficult.

This study proves that aetiology is multifactorial and is not because of diaper alone.

Awareness of these conditions over diaper area and attention to a comprehensive physical and cutaneous examination will help the clinician to arrive at an accurate diagnosis.

SUMMARY

- Diaper area dermatoses is a broad term used to describe various skin conditions that can occur in diaper area. Eruptions in the diaper region have diverse origins.
- The anatomical area for diaper dermatoses encompasses the lower abdomen, lower lumbar region, gluteal area, genitalia and inner aspects of thigh.
- A total of 130 children below 5 years with diaper area lesions who presented to the department of Dermatology at R.L. Jalappa hospital and research centre, attached to Sri Devaraj Urs Medical College, Tamaka, Kolar between December 2013 to July 2015 were studied.
- In the present study on children below 5 years, majority of cases were infants (45%), followed by toddlers (33%), pre school children (19%) and least were neonates (3%).
- Males (52.3%) were affected marginally more than females (47.7%).
- Majority of the patients (93.1%) presented with symptoms of less than 6 weeks duration.
- Diaper area was the initial site of involvement in 37.7% of the cases studied. Lesions exclusively over the diaper area were seen in 31.5% of children.
- The most common diaper area involved in our study population is gluteal region (65.4%).
- Papule (37.7%) and excoriation(32.3%) were the commonest morphological presentation in our study.
- A total of 23 various dermatoses were encountered. ICD (22.3%) was the most common dermatoses, followed by scabies (14.6%), impetigo (13.8%), papular urticaria (11.5%).

- Irritant contact dermatitis and impetigo were common in infants. Scabies was common in toddlers and papular urticaria was common in preschool children.
- Infectious etiology was the commonest, seen in 37(28.46%) children, followed by inflammation(26.15%) and arthropod bite(26.15%).
- Amongst the infective dermatoses , bacterial infection (14.61%) was most common entity followed by viral (10.76%) and fungal infection (3.1%).
- Amongst inflammatory dermatoses, irritant contact dermatitis (22.3%) was most common followed by seborrhoeic dermatitis (3.8%).
- Among arthropod bites, scabies was seen in 14.6% patients , and papular urticaria was seen in 11.5% patients.
- This study gives a precise clinical insight into diaper area dermatoses and thereby helps us manage the patient better.

BIBLIOGRAPHY

1. Daniel BW, Alan BF, Steven RF, Daniel PK. Characterization Of Diaper Dermatitis in the United states. Arch Pediatr Adolesc Med 2000; 154: 943-946.
2. Alfrons K, Bernice K. Diaper area eruptions. In: Lawrence FE, Ilona JF, Nancy BE(eds). Neonatal dermatology. 2nd edition. Philadelphia: Saunders; 2008: p 245-263.
3. Scheinfeld N. A review and brief survey of eruptions of diaper area. Am J Clin Dermatol 2005; 6: 273-281.
4. Srisupalak S, Ilona JF. Diaper dermatitis. Pediatr Rev 1995; 16:142-147.
5. Rasmussen JE. Classification of diaper dermatitis: an overview. Pediatrician 1987; 14:6-10.
6. Bhat RM, Chavda R. To evaluate the efficacy and safety of RV2427B cream in Irritant dermatitis care. Indian Dermatol online J 2013; 4: 180-4.
7. Unna PG. Seborrhoeic eczema. J Cutan Dis 1887; 5: 499.
8. Diana elish, Nanette B. Infantile seborrheic dermatitis. Cutis 2006; 77: 297-300.
9. Schwartz RA. Superficial fungal infections. Lancet 2004; 364: 1173-82.
10. Heng MC, Henderson CL, Barker DC, Haberfelde G. Correlation of Pityosporum ovale density with clinical severity of seborrheic dermatitis as assessed by a simplified technique. J Am Acad Dermatol 1990; 23: 82-6.
11. McGinley KJ, Leyden JJ, Marples RR, Kligman AM. Quantitative microbiology of the scalp in non-dandruff, dandruff and seborrhoeic dermatitis. J Invest Dermatol 1975; 64: 401.
12. Erchiga VC, Martos OJ, Cassano AV. Malassezia globosa as the causative organism of pityriasis versicolor. Br J Dermatol 2000; 143:799–803.

13. Schwartz RA, Janusz CA, Janniger CA. Seborrheic Dermatitis: An Overview. *Am Fam Physician* 2006; 74: 125-30.
14. Plewig G, Jansen T. Seborrheic Dermatitis. In: Wolff K, Goldsmith LA, Katz S, Gilchrest B, Paller A, Leffell D (eds). *Fitzpatrick's Dermatology in General Medicine*. 7th edition. New York : Mc Graw Hill; 2008: p 219.
15. Janniger CK. Infantile seborrheic dermatitis: an approach to cradle cap. *Cutis* 1993;51:233-235.
16. Cohen S. Should we treat infantile seborrheic dermatitis with topical antifungals or topical steroids? *Arch Dis Child* 2004;89: 288-289.
17. Lokre S, Hiremagalore R. Childhood psoriasis - Clinical manifestations. *Indian J Paediatr Dermatol* 2012; 13:3-8.
18. Henseler T. The genetics of psoriasis. *J Am Acad Dermatol* 1997; 37:S1-11.
19. Morris A, Roger M, Fisher G, Williams K. Childhood psoriasis: A clinical review of 1262 cases. *Pediatr Dermatol* 2001; 18:188-98.
20. De Zeeuw R, Van Praag MC, Oranje AP. Granuloma gluteale infantum: a case report. *Pediatr Dermatol* 2000; 17:141-3.
21. Al-Faraidy NA, Al-Natour SH. A forgotten complication of diaper dermatitis: Granuloma gluteale infantum. *J Fam Community Med* 2015 ; 17:107-9.
22. Dandale A, Dhurat R, Ghatge S. Perianal pseudoverrucous papules and nodules. *Indian J Sex Transm Dis* 2013; 34:44-6.
23. Amiry SA, Pride HB, Tyler WB. Perianal pseudoverrucous papules and nodules mimicking condylomata acuminata and child sexual abuse. *Cutis* 2001; 67:335-8.
24. Goldberg NS, Esterly NB, Rothman KF, Fallon JD, Cropley TG, Szaniawski W, et al. Perianal pseudoverrucous papules and nodules in children. *Arch Dermatol* 1992; 128:240-2.

25. Friedlander SF. Contact dermatitis. *pediatric Rev* 1998;19:166-171.
26. Fisher AA. Allergic contact dermatitis in early infancy. *Cutis* 1994;54:300-2.
27. Williams HC, Pembroke AC, Foresdyke H, et al. London-born black Caribbean children are at increased risk of atopic dermatitis. *J Am Acad Dermatol* 1995; 32:212-17.
28. Sandipan dhar. Atopic dermatitis In: Valia RG, Valia AR eds. *IADVL Textbook of Dermatology*. 3rd edition. Mumbai: Bhalani Publishing House; 2010 p.223-51.
29. Mowad CM, McGinley KJ. The role of extracellular polysaccharide substance produced by *staphylococcus epidermidis* in miliaria. *J Am Acad Dermatol* 1995;33:729-33.
30. Boiko S. Treatment of diaper dermatitis. *Dermatol clin* 1999;17: 235-40.
31. Darmstadt G, Lane AT. Impetigo. An overview. *Pediatr Dermatol* 1994;11:293-303.
32. Hellgren L, Hersle K. Impetigo contagiosa: statistical evaluation of clinical and laboratory data in 3167 patients and matched healthy controls. *Acta Derm Venereol* 1964; 44:356-61.
33. Wesley KG. Bacterial infections. In: Schachner LA, Hansen RC. *Textbook of Paediatric Dermatology*. 3rd ed., Edinburgh Mosby Publication: Edinburgh; 2003 p. 683.
34. Khalid M. A retrospective study of some clinical and epidemiological features of impetigo patients. *Journal of family and community medicine* 2006; 13:32-36.
35. Malhotra SK, Malhotra S, Dhaliwal GS, Thakur A. Bacteriological study of pyoderms in tertiary care dermatological center. *Indian J Dermatol* 2012;57:358-61.
36. Thappa DM. Common skin problems. *Indian J Pediatr* 2002; 69:701-6.

37. Chirac A, Brzezinski P, Chiriac AE, Foia L. Autosensitisation (Autoeczematisation) reactions in a case of diaper dermatitis candidiasis. *Niger Med J* 2014; 55: 274-5.
38. Amrinder J. Kanwar ,Dipankar De .Superficial fungal Infections In: Valia RG, Valia AR eds. *IADVL Textbook of Dermatology*. 3rd Ed. Mumbai: Bhalani Publishing House; 2010 p.223-51
39. Sasidhran CK, Sugathan P, Agarwal R, Khare S, Lal S, Jayaram Paniker CK. Hand-foot and mouth disease in Calicut. *Indian J Pediatr* 2005; 72:17-21.
40. Nilendu Sarma, Abhijit Sarkar.Epidemic of hand, foot and mouth disease in west bengal India august, 2007:A multicentric study. *Indian J Dermatol*2009;54:26-30.
41. Brandt O, Abeck D, Gianotti R, Burgdorf W. Gianotti-Crosti syndrome. *J Am Acad Dermatol* 2006;54:136-45.
42. Patrizi A, Di Lernia V, Neri I, Ricci G. An unusual case of recurrent Gianotti-Crosti syndrome. *Pediatr Dermatol* 1994;11:283-4.
43. Chuh AA. Diagnostic criteria for Gianotti-Crosti syndrome: A prospective case-control study for validity assessment. *Cutis* 2001;68:207-13.
44. Shylaja S. A case of Gianotti-Crosti syndrome .*Muller Journal of Medical Science and Research* 2014; 5:88-89.
45. Karthikeyan K, Thappa DM, Jeevankumar B. Pattern of pediatric dermatoses in a referral center in South India. *Indian Pediatr* 2004; 41:373-7.
46. Sayal SK, Bal AS, Gupta CM. Pattern of skin diseases in paediatric age group and adolescents. *Indian J Dermatol Venereol Leprol* 1998; 64:117-9.
47. Giam YC. Skin diseases in children in Singapore. *Ann Acad Med Singapore* 1988; 17:569-72.

48. Arvin AM. Cell mediated immunity to varicella zoster virus. *J Infect Dis* 1992; 166:S35–S46.
49. Boyd AS, Neldner KH, Zemnstov A, et al. photolocalized varicella. *J Am Acad Dermatol* 1992; 26: 772–4.
50. Shelly WB, Shelly ED. Immunization, abrasion and sunburn as localizing factors in chickenpox. *Pediatr Dermatol* 1987; 4:102–4.
51. Usman N. Anal condyloma acuminata in eleven months old infant. *Indian J Sex Transm Dis* 1985;6:61-62.
52. Gemmell CG. Staphylococcal scalded skin syndrome. *J Med Microbiol* 1995;43: 318-27.
53. Sethuraman KM, Avabratha KS, Varghese AD, Rai BS. Staphylococcal scalded skin syndrome: A dermatological emergency in pediatrician's hand. *Med J DY Patil Univ* 2014;7: 189-91.
54. Mohammed NS, Suleman H. Prevalence of scabies among school-aged children in Katanga rural community in Kaduna state, Northwestern Nigeria. *Annals of Nigerian Medicine* 2012 ; 6 : 26-29.
55. Sterling GB, Janniger CK, Kihiczak G, et al. Scabies. *Am Fam Physician* 1992; 46: 1237- 1241.
56. Chosidow O. Scabies and pediculosis. *Lancet* 2000; 355:819-826.
57. Sudip Das, Tapas Chatterjee, G Banerji. Evaluation of the commonest site, demographic profile and most effective therapy in scabies. *Indian J Dermatol* 2006; 51(3); 186-188.
58. Rashmi Sarkar, Amrinder JK. Three common dermatological disorders in children. *Indian paediatrics* 2001; 38:995-1008.
59. Vinay A, Michael JM. Pediatric scabies. *Cutis* 2003; 71:193-196.

60. Rasmussen JE. Scabies. *Pediatr Rev* 1994; 15:110-114.
61. Abdulghani Mohamad Alsamarai. Frequency of Scabies in Iraq: Survey in a Dermatology Clinic. *J Infect Dev Ctries* 2009; 3(10):789-783.
62. Narendra P. Bachewar, Vijay R. Thawani, Smita N. Comparison of safety, efficacy, and cost effectiveness of benzyl benzoate, permethrin, and ivermectin in patients of scabies. *Indian J Pharmacol* 2009 ;41: 9-14.
63. Stibich AS, Schwartz RA. Papular urticaria. *Cutis* 2001; 68: 89-91.
64. Heng MC, Kloss SG, Habberfelde GC. Pathogenesis of papular urticaria. *J Am Acad Dermatol* 1984;10: 1030-4.
65. Howard R, Frieden IJ. Papular urticaria in children. *Pediatr Dermatol* 1996; 13: 246-9.
66. Steen CJ, Carbonaro PA, Schwartz RA. Arthropods in dermatology. *J Am Acad Dermatol* 2004; 50: 819-62.
67. Naeem R, Muhammad Saqib L. A Clinical Study of Papular Urticaria. *Journal of the College of Physicians and Surgeons Pakistan* 2008; 1: 147-150.
68. Hood AF, Mark EF. Histopathologic diagnosis of pityriasis lichenoides et varioliformis acuta and its clinical correlation. *Arch Dermatol* 1982;118 :478-482.
69. Singh S, Mann BK. Insect bite reactions. *Indian J Dermatol Venereol Leprol* 2013;79:151-64.
70. Metry DW, Hebert AA. Benign cutaneous vascular tumors of infancy: When to worry, what to do. *Arch Dermatol* 2000;136:905-14.
71. Garzon MC, Enjolras O, Frieden I. Vascular tumors and vascular malformations: Evidence for an association. *J Am Acad Dermatol*. 2000; 42:275–9

72. Nitin Sharma, Shasanka Shekhar Panda. Use of oral prednisolone with or without propranolol in the management of infantile hemangioma: A critical appraisal. Indian Journal of Paediatric Dermatology 2013; 14 : 19-22.
73. Michaelsson G. Zinc therapy in acrodermatitis enteropathica. Acta Derm Venereol 1974; 54:377-381.
74. Sharma NL, Sharma RL, Gupta KR, et al. Self limiting acrodermatitis enteropathica. A follow-up study of three inter-related families. Int J Dermatol 1988; 27:485-486.
75. McLelland J, Broadbent V, Yeomans E, Malone M, Pritchard J. Langerhans cell histiocytosis: The case for conservative treatment. Arch Dis Child 1990;65: 301-3.
76. Caldemeyer KS, Parks ET, Mirowski GW. Langerhans cell histiocytosis. J Am Acad Dermatol. 2001; 44:509–11.
77. Minkov M, Grois N, Heitger A, Pötschger U, Westermeier T, Gadner H; DAL-HX Study Group. Response to initial treatment of multisystem Langerhans cell histiocytosis; An important prognostic indicator. Med Pediatr Oncol. 2002; 39: 581–5.
78. Powell JJ, Wojnarowska F. Lichen sclerosus . Lancet 1999; 353:1777-83.
79. Shah R, Ghiya R, Iyer A, Marfatia Y S. Lichen sclerosus: A case report with review of literature. Indian J Sex Transm Dis 2007;28:40-2.
80. Gupta S, Malhotra AK, Ajith C. Lichen sclerosus: Role of occlusion of the genital skin in the pathogenesis. Indian J Dermatol Venereol Leprol 2010;76:56-8.
81. Sharma R, Nadeem M, Chandra M. Bullous pemphigoid in childhood. Indian J Dermatol Venereol Leprol 1996; 62:100-2.
82. Nayak S, Acharjya B, Devi B, Sethi G. Lichen striatus in a rare pattern. Indian J Dermatol 2007;52:67-8.

83. Krishnegowda SY, Reddy SK, Vasudevan P. Lichen striatus with onychodystrophy in an infant. *Indian Dermatol Online J* 2015;6:333-5.
84. Kennedy D, Rogers M. Lichen striatus. *Pediatr Dermatol* 1996; 13:95-9.
85. Balai M, Khare AK, Gupta LK, Mittal A, Kuldeep CM. Pattern of pediatric dermatoses in a tertiary care centre of South West Rajasthan. *Indian J Dermatol* 2012;57: 275-8
86. Banerjee S, Gangopadhyay DN, Jana S, Mitra C. Seasonal variations in pediatric dermatoses. *Indian J Dermatol* 2010; 55: 44-6
87. Sharma RC, Mendiratta V. Clinical profile of cutaneous infections and infestations in the paediatric age group. *Indian J Dermatol* 1999;44:174-8.
88. Gul U, Cakmak SK, Gonul M, Kilic A, Bilgili S. Pediatric skin disorders encountered in a dermatology outpatient clinic in Turkey. *Pediatr Dermatol* 2008;25:277-8.
89. Sardana K, Mahajan S, Sarkar R, Mendiratta V, Bhushan P, Koranne RV . The spectrum of skin disease among Indian children. *Pediatr Dermatol* 2009; 26:6-13.
90. Hayden GF. Skin diseases encountered in a pediatric clinic. *Am J Dis Child* 1985;139: 36-8.
91. Javed M, Jairamani C. Pediatric dermatology: An audit at Hamdard University Hospital, Karachi. *J Pak Assoc Dermatol* 2006; 16:93-6.

ANNEXURES

PROFORMA

SL NO-

Name : Hosp No:

Age : Date:

Sex : Religion:

Address:

PRESENTING COMPLAINTS:

DURATION:

HISTORY OF PRESENTING COMPLAINT:

1. Onset : Sudden/insidious
2. Progression : Stationary/infection/drugs
3. Initial site :
4. Provoking factors : drugs/infection
5. Seasonal variation : increased/decreased/no change
6. Any other associated disease

PAST HISTORY : history of similar illness: present/absent

FAMILY HISTORY:

PERSONAL HISTORY:

Diet: Appetite: Sleep:

Bowel: Bladder:

General physical Examination:

Weight-

Pallor, icterus , lymphnodes , cyanosis, clubbing

Vital signs:

Pulse: Respiratory rate:

Blood pressure: Temperature:

DIAPER AREA EXAMINATION:

a. Morphology of lesion-

b. Size-

c. Shape-

d. colour-

e. scaling

f. surrounding skin-

Examination of other sites.

SYSTEMIC EXAMINATION

CVS : GIT :

CNS : RS :

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS:

1. Blood:

2. Urine:

3. Stool:

4. KOH Preparation:

5. Skin Biopsy:

6. Gram staining:

7. Bacterial culture and sensitivity:

8. Fungal culture:

FINAL DIAGNOSIS:

**PARENTAL/GUARDIAN CONSENT FORM FOR CHILD
PARTICIPATION IN PROJECT ON DIAPER AREA DERMATOSES.
CONSENT FORM**

I
being over the age of 18 years hereby consent to my child
participating, as requested, for the dissertation project on diaper area dermatoses.

1. I have been explained about the nature and purpose of the study.
2. Details of procedures and any risks have been explained to my satisfaction.
3. I agree to video recording of my child's information and participation.

I understand that:

- My child may not directly benefit from taking part in this research.
- My child is free to withdraw from the project at any time and is free to decline to answer particular questions.
- While the information gained in this study will be published as explained, my child will not be identified, and individual information will remain confidential.
- Whether my child participates or not, or withdraws after participating, will have no effect on any treatment or service that is being provided to him/her.
- Whether my child participates or not, or withdraws after participating, will have no effect on his/her progress in his/her course of study, or results gained.

Parent/guardian signature/Thumb impression

Date.....

I certify that I have explained the study to the volunteer and consider that she/he understands what is involved and freely consents to participation.

Researcher's name.....

Researcher's signature.....Date.....

KEY TO MASTER CHART

1. SERIAL NUMBER:
2. HOSPITAL NO:
3. AGE :
4. SEX : M- Male F- Female
5. RELIGION: H-Hindu M-Muslim C-Christian
6. CHIEF COMPLAINTS : A- Itching B- Pain
 C- Burning sensation D- Reddish lesions
 E-Fluid filled lesions F-Scaling
 G-Pigmentation H-Red raw areas
 I-Asymptomatic lesions
7. DURATION : A- since birth
 B- <6 weeks
 C->6 weeks
8. INITIAL SITE : S- Scalp F- Face N-Neck U- Upper limbs
 T- Trunk L- Lower limbs O-Oral cavity
 D-Diaper area
9. OTHER SITES INVOLVED : S- Scalp F- Face N-Neck
 U- Upper limbs T- Trunk L- Lower limbs
 O-Oral cavity A-All D-Diaper area
10. PROVOKING FACTORS: A- Trauma B-Drugs C-Infection
 D- Diaper E-Heat F-Insect bite
11. FAMILY HISTORY: +/-
12. DIAPER AREA SITE: A-Lower abdomen B-Lower lumbar region

C-Gluteal area

D-Genitalia

E-Inner aspect of thigh

13. PRIMARY LESION: A- Macule B- Papule C- Plaque

D- Pustule

E- Nodule

F- Cyst

G-Vesicle

H-Bullae

I-Target lesion

J-Burrow

K-Wheals

14. SECONDARY CHANGES : A- Scaling B- Crusting C- Ulcer

D- Erosion

E-Excoriation

F-Hyperpigmentation

G-Hypopigmentation

H-Depigmentation

15. OTHER SITES : F- Face U- Upper limbs O- Oral cavity

T- Trunk L- Lower limbs

16. INVESTIGATION: A-Clinical WL – Woods lamp FC-Fungal culture

GS-Gram stain BC-Bacterial culture

17. DIAGNOSIS:



Figure 26: Impetigo involving gluteal region



**Figure 27: Impetigo Involving Inner Aspects Of Thigh And Lower
Abdomen**

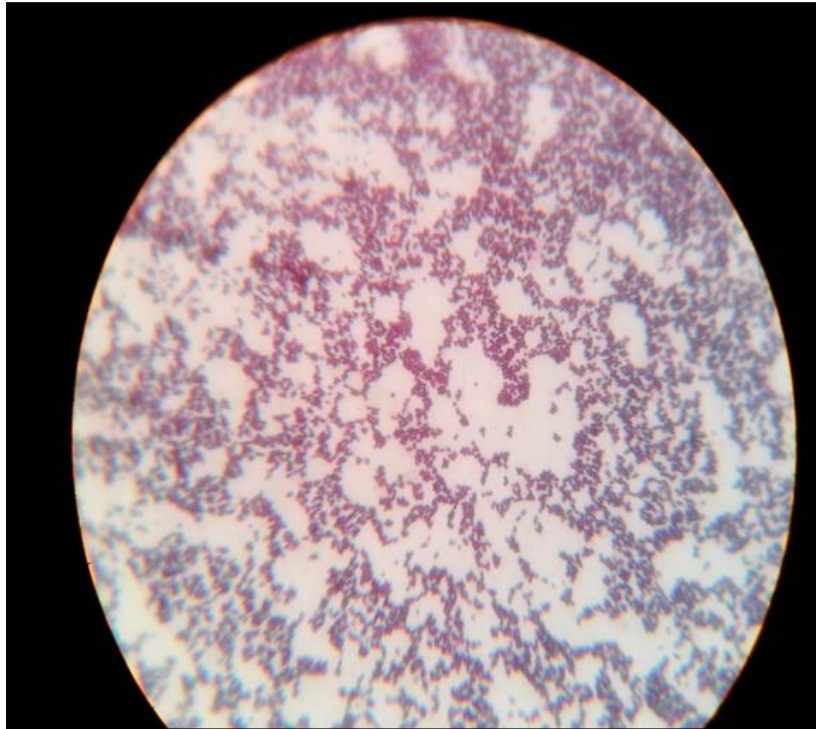


Figure 28: GPC in clusters on grams stain in a case of impetigo



Figure 29: Staphylococcus Aureus Growth On Blood Agar



Figure 30: Candidiasis Involving Skin Folds In Diaper Area



31: Candidiasis With Satellite Pustules

Figure



Figure 32: Gram Positive Budding Yeast Cells On Gram Stain In A Case Of Candidiasis

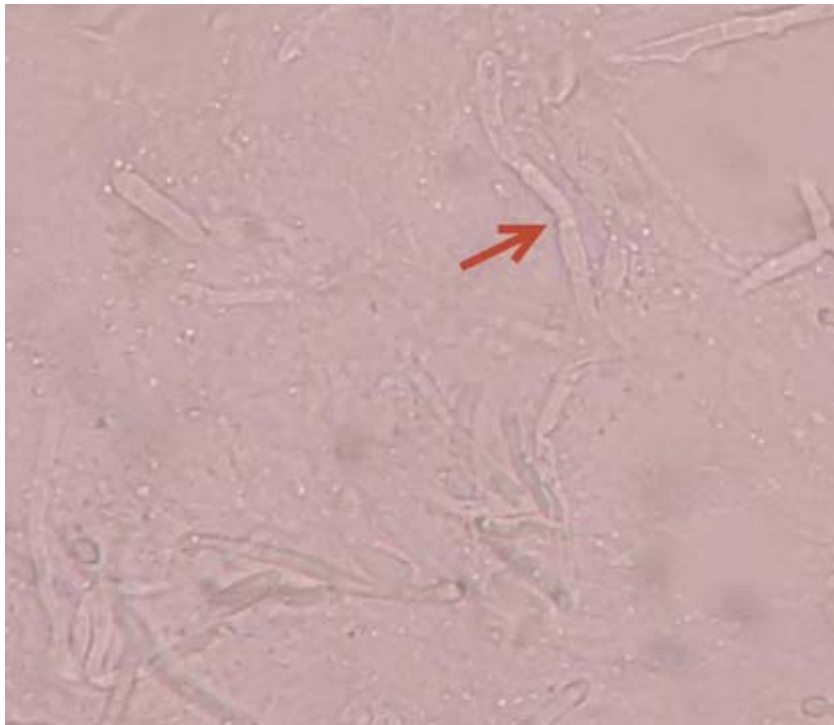


Figure 33: KOH Mount Showing Hyphae



**Figure 34: Irritant Contact
Dermatitis Due To Diaper**



Figure 35: ICD



Figure 36: Irritant Contact Dermatitis



Figure 37: Stevens Johnson Syndrome With Genital Area Involvement



Figure 38: Papular Urticaria Involving Diaper Area



Figure 39: Scabies Involving Genital Area



Figure 40: Ulcerated Infantile Hemangioma



Figure 41



Figure 42

**Fig 41 and 42: Seborrhoeic Dermatitis Involving Scalp And Diaper
Area**



Figure 43 –Adenoma sebaceum



Figure 44-Ash Leaf Macules



Figure 45-Shagreen patch



**Figure 46-Positive Family
History**

Tuberous Sclerosis



Figure 47 and 48: Lamellar Ichthyosis

