"CUTANEOUS MANIFESTATIONS IN NEONATES- A ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL"

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LIST OF ABBREVIATIONS USED

VC	Vernix caseosa
SGH	Sebaceous gland hyperplasia
ETN	Erythema toxicum neonatorum
VEN	Verrucous epidermal nevi
CMN	Congenital melanocytic nevi
MS	Mongolian spot
AD	Atopic dermatitis
EB	Epidermolysis bullosa
EBS	Epidermolysis bullosa simplex
DEB	Dystrophic epidermolysis bullosa
JEB	Junctional epidermolysis bullosa
SSSS	Staphylococcal scalded skin syndrome
HSV	Herpes simplex virus
CMV	cytomegalovirus
PF	purpura fulminans
NLE	neonatal lupus erythematosus
PRP	pityriasis rubra pilaris
SCID	severe combined immunodeficiency

ABSTRACT

Background

Skin lesions in neonatal period range from transient self limiting conditions to serious dermatoses requiring specific therapies. They can cause significant psychological distress to parents. The awareness of the fact that most of these conditions are benign and transient is important so that parents can be reassured. Since studies on neonatal dermatoses are limited, this study has been planned to know the spectrum of cutaneous lesions in neonates, both physiological and pathological.

Methods

All neonates less than 28 days old attending KLEs Dr. Prabhakar Kore Hospital and MRC, Belgaum were recruited into the study. Newborns admitted in the NICU were excluded from the study. A written informed consent was obtained from the mother. Study design was non randomized cross sectional study. Sample size calculation was done using the Chi square test. Analysis of data was performed by STATA 11.2 and SPSS software. An ethical committee clearance was obtained prior to the start of the study.

Results

104 neonates were enrolled in the study, out of which 49 (47%) neonates were male and 55 (53%) were female. 51 (49%) neonates were born through normal vaginal delivery and 53 (51%) by caesarian section. 3 (2.88%) neonates were born preterm, 5 (4.81%) post term and 96 (92.31%) neonates were born at term. 99 (95%) had physiological changes and 5 (5%) had pathological changes. The most common physiological change observed was mongolian spot in 34 (33%) of neonates followed

by erythema toxicum neonatorum (ETN) in 27 (26%) neonates and physiological desquamation in 21 (20%) . Other less common physiological skin changes observed were milia, miliaria, hypertrichosis lanuginosa, vernix caseosa and sebaceous gland hyperplasia. Pathological skin changes were observed in only five neonates, out of which one had bullous impetigo, one had birth trauma, 1 had furunculosis, 1 had intertrigo and 1 was a collodion baby. Mongolian spot was found to be more common among low birth weight (<2.5kgs) whereas ETN was more common among neonates with normal birth weight and among males. There was no statistically significant association found with other parameters like birth order and maturity of the neonate.

Conclusion

In this study, physiological changes were found in 99 (95.19%) and pathological changes in 5 (4.81%). Although skin changes in neonates are common, majority are benign and transient and require no treatment. The parents need to be reassured about the self limiting nature of such lesions. Less often, more serious conditions may be found where early diagnosis and prompt institution of treatment may even be life saving.

Key words: neonate, dermatoses, physiological, pathological, mongolian spot.

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INTRODUCTION

Neonatal period encompasses the first four weeks of extrauterine life.[1]

During this time, the skin undergoes rapid adaptation to assume an important role as a defense barrier and of thermoregulation.[2]

Skin lesions have been found in around 94-96% of neonates in various studies published across the world. [2,3]

They may range from benign and transient self limiting conditions to severe, life threatening disorders. Various factors like race, nutrition, hygiene, socioeconomic status, maternal factors, heredity, climate etc influence the pattern of skin changes in the newborn.[2]

A broad classification of skin lesions in newborn include physiological conditions, transient eruptions, birth marks, cutaneous infections and inherited disorders.[2]

Majority of the skin changes in neonates are physiological and transient requiring no treatment. However these cause undue concern to the parents as well as to the pediatricians who may be unfamiliar with these skin changes.[3]

Few lesions can be cutaneous manifestations of potentially life threatening systemic disorders, so early diagnosis is crucial to initiate specific therapy at the earliest.[4]

OBJECTIVES

1. To study the spectrum of cutaneous manifestations in neonates; both physiological and pathological.

2. To find an association between the cutaneous changes and various parameters like birth weight, maturity, birth order and gender of the neonate.

REVIEW OF LITERATURE

Skin is a vital structure of the body. It has various functions; providing a barrier against invading microbes, maintaining fluid balance and core body temperature are amongst the most important.

Neonatal period encompasses the first four weeks of extrauterine life. Infants born between 37-40 weeks are considered term babies. Infants born before 37th week and after 40th week are preterm and post term respectively. This term is used irrespective of birth weight. Infants with a birth weight less than 2.5 kgs are considered to be low birth weight.

Skin of the neonate has some differences from that of an adult, which are enumerated in the following table:[5]

Skin structure	Preterm Skin	Term Skin	Adult Skin
Epidermis	Thin, compressed stratum corneum with low melanin content	Adherent stratum corneum with more layers than preterm skin	Normal structure and thickness of epidermis, normal melanin content
Dermo-Epidermal Junction	Less cohesion between epidermis and dermis	Less cohesion between epidermis and dermis	Good epidermo-dermal cohesion
Dermis	Thinner, less developed elastic fibres	Thinner, less developed elastic fibres	Fully developed elastic fibres
Permeability	Highly permeable to fat soluble substances and increased absorption due to increased surface area: body weight ratio.	Highly permeable to fat soluble substances and increased absorption due to increased surface area: body weight ratio.	Good barrier protection and resistance to penetration.

Hair	Lanugo hair	Vellus hair	Terminal and vellus hair
Sweat glands	Secretory cells are undifferentiated with decreased sweating capacity	Dense distribution of sweat glands, decreased sweating capacity	Less dense distribution of sweat glands with normal sweating capacity.
Sebaceous glands	Large and active	Large and active	Large and active
CNS,CVS	Disorganized, unmyelinated nerves	Unmyelinated nerves,vascular system gets organized at 3 months	Normal.

The skin serves several important functions in a newborn:

Barrier function

The skin of a full term neonate has a fully functional stratum corneum with adequate barrier protection. But in case of preterms, especially those born before 34 weeks of gestation, the barrier function is impaired due to the anatomical immaturity of the epidermis. However, the barrier function appears to improve rapidly after birth to normal level by 2^{nd} to 3^{rd} week after birth.

There is a high risk of percutaneous absorption of topically applied substances especially in preterm. The other factors which contribute are- high surface area to volume ratio, occlusive environment, eg. in the diaper area, underlying skin disease, and high ambient temperature and humidity.[6]

All newborns experience transepidermal water loss (TEWL) in the 1st month of gestation. In a full term infant it is around 6-8g/m^2/hr whereas in a preterm infant born at 25 weeks it is 60g/m^2/hr. By 32^{nd} week it normalizes to that of a mature full term

infant. TEWL depends on: ambient temperature, humidity, phototherapy, activity of the baby, skin trauma and gestational age of the baby.[6]

Due to defective barrier function, preterm infants have the risk of systemic dissemination of cutaneous infections like candidiasis.[6]

Sebaceous gland secretion

Sebaceous gland activity is high in the first month and decreases steadily thereafter; it is due to the stimulation by maternally transferred androgens especially dehydroepiandrosterone. The sebaceous secretions form an important component of the vernix caseosa which also contains various anti-microbial peptides and functions as a natural barrier cream.[7]

Eccrine Sweating

Sweating in neonates is inefficient as a thermoregulatory mechanism. Therefore care must be taken to make sure that a baby, especially preterm kept in incubators, is not overheated. The site of thermally induced sweating is the forehead and that of emotional sweating, eg in response to arousal, is the palms and soles. This is fully developed at birth in term neonates but not in preterm.[7]

Cutaneous disorders in the human neonate were first described by Ballantyne, an obstetrician from Edinburg in 1895.

Neonatal skin disorders may vary from transient response to physiological stimuli to severe life threatening conditions.

Neonatal skin lesions may be divided into [8]

A. PHYSIOLOGICAL

1. Vernix caseosa
2. Cyanosis
3. Desquamation of the skin
4. Erythema neonatorum
5. Sebaceous gland hyperplasia
6. Physiological Jaundice
7. Milia
8. Epstein pearls
9. Harlequin colour changes
10. Cutis marmorata
11. Neonatal occipital alopecia

B. TRANSIENT NON INFECTIVE DISEASE

1. Erythema toxicum neonatorum
2. Miliaria
3. Transient neonatal pustular melanosis
4. Neonatal acne
5. Infantile acropustulosis
6. Sucking blisters
7. Eosinophilic pustulosis
8. Subcutaneous fat necrosis

C. NAEVI AND OTHER DEVELOPMENTAL DEFECTS

1. Epidermal naevi

- Verrucous naevi
- Sebaceous naevi

2. Dermal and subcutaneous naevi

- Congenital melanocytic naevi
- Dermal melanosis
- Nevus simplex
- Nevus flammeus

3. Hemangiomas of infancy

4. Supernumerary nipples

5. Accessory tragus

6. Neural tube dysraphism

- Spinal dysraphism
- Cranial dysraphism

7. Aplasia cutis congenita

D. GENODERMATOSIS AND OTHER HERITABLE DISEASES

1. Ichthyosis

- Ichthyosis vulgaris
- Lamellar Ichthyosis

- X linked Ichthyosis
- Epidermolytic hyperkeratosis
- Collodion baby
- Rare syndromes

2. Epidermolysis bullosa

- Epidermolysis bullosa simplex
- Junctional Epidermolysis bullosa
- Dystrophic Epidermolysis bullosa

3. Ehler Danlos Syndrome

4. Cutis laxa

5. Incontinentia pigmenti

6. Piebaldism

7. Cafe-au-lait macules

8. Congenital Erythropoietic porphyria

9. Oculocutaneous Albinism

10. Ectodermal dysplasia

- Hidrotic Ectodermal dysplasia
- Hypohidrotic Ectodermal Dysplasia
- Ectodermal dysplasia-ectrodactyly-cleft lip/palate syndrome

E. INFECTIONS

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- Mastitis
- Omphalitis
- Congenital Syphilis
- Necrotizing fasciitis
- Listeriosis
- Noma neonatorum

2. Fungal

- Candida
- Dermatophytosis

3. Viral

- Herpes simplex
- Varicella zoster
- Human parvovirus B19
- Rubella
- Enterovirus
- Cytomegalovirus

4. Parasitic

- Scabies
- Toxoplasmosis
- Leishmaniasis

5. Arthropods

- bites and stings.

F. INFANTILE ECZEMA

1. Infantile seborrheic dermatitis
2. Atopic Dermatitis
3. Diaper Dermatitis

G. NUTRITIONAL DERMATOSES

1. Acrodermatitis Enteropathica
2. Neonatal Biotin Deficiency

H. IMMUNODEFICIENCY SYNDROMES

1. Severe combined immunodeficiency
2. Omenn's syndrome
3. Di George anomaly
4. Wiskott Aldrich Syndrome
5. Chronic granulomatous disease
6. Hyperimmunoglobulin E Syndrome
7. Acquired Immunodeficiency Syndrome

I. MISCELLANEOUS

1. Neonatal Lupus Erythematosus
2. Bronze baby syndrome
3. Purpura fulminans
4. Neonatal Pemphigus
5. Purpuric phototherapy induced eruption
6. Langerhans cell histiocytosis
7. Pityriasis rubra pilaris
8. Erythroderma

PHYSIOLOGICAL SKIN CHANGES

1. VERNIX CASEOSA

Vernix caseosa is a protective layer which forms a biofilm over the skin surface of the fetus in the last trimester It is a creamy white deposit derived from the stratum corneum, sebaceous glands and remnants of the epitrichium. The vernix is composed of water (80.5%), lipids (10.3%) and proteins (9.1%). [9] It also contains antimicrobial peptides like cathelicidins and defensins [8,9] along with other proteins like lysozyme, lactoferrin, secretory leukocyte protease inhibitor and psoriasin. [10,11] Vernix is also rich in lipids, the composition of which varies - in males there is a higher proportion of squalene and wax esters and in females it contains more cholesterol and cholesterol esters. [14] The vital proteins involved in host defence are cystatin A, UGRP-1, and calgranulin A,B & C. They have antifungal activity, opsonizing capacity, inactivate protease and inactivate parasite. [15,16] Hence vernix is an important host defense barrier, protecting the neonate against infection. [17]

2. CYANOSIS

Cyanosis is derived from the greek word “kuaneos” which means “dark blue”. It may be peripheral; referred to as acrocyanosis or central. Peripheral cyanosis is a relatively common findings in young infants and it is frequently physiological, due to the large arteriovenous oxygen difference that results during slow flow through the peripheral capillary beds.[16] Acrocyanosis is more common among term infants; other risk factors for developing cyanosis are hypothermia, polycythemia and other hyperviscosity symptoms. This bluish discoloration disappears on warming. It should be differentiated from cyanosis due to cardiac/ respiratory cause. It usually resolves in the first week and requires no active treatment. [17]

3. SUPERFICIAL CUTANEOUS DESQUAMATION

It is seen in upto 75% of normal newborns. It is also known as physiological scaling of the newborn. It is usually localised initially, starting to develop around the ankles and gradually becomes widespread by the end of the first week. It is more common in post term babies and is unusual in neonates born before 39 weeks of gestation.[7]

4. ERYTHEMA NEONATORUM

It refers to generalized hyperemia that develops within a few hours after birth and resolves spontaneously within 24 to 48 hours. It occurs due to reflex vasodilatation of cutaneous capillaries due to reduced sympathetic tone. [18,19]

5. SEBACEOUS GLAND HYPERPLASIA

It is seen clinically as multiple, uniform, pin point yellowish papules that are most common over the nose, cheeks, upper lip and forehead. In 40% of patients it may be

associated with milia. It usually disappears by the end of the first week although rarely it may persist longer.[7]

6. PHYSIOLOGICAL JAUNDICE

Jaundice refers to yellowish discoloration of the skin and other tissues of neonate having bilirubin level more than 5mg/dL. It consists of two phases-

Phase 1: It lasts for upto 10 days in term infants with serum bilirubin rising upto 12 mg/dL and for around two weeks in preterm infants with serum bilirubin rising till 15 mg/dL.

Phase 2: Bilirubin levels reduce to 2mg/dL for two weeks, but in preterm and exclusively breast fed infants it can last for more than a month.

Mechanism of Physiological Jaundice :

A. Low activity of glucuronosyltransferase which converts unconjugated bilirubin to conjugated bilirubin.

B. Short lifespan of fetal RBCs i.e 80-90 days , compared to 120 days in adults.

C. Low conversion of bilirubin into urobilinogen leading to reabsorption into the circulation.

Neonates with total serum bilirubin greater than 21mg/dL should receive phototherapy. [20,21]

7. MILIA

Milia are tiny yellowish white keratin filled cysts seen at the opening of each pilosebaceous follicle. The most common sites involved are the nose and cheeks . The increase in sebaceous gland volume , size and total number of sebaceous cells is due to maternal androgen stimulation in utero. Extensive, persistent and atypical milia are associated with orofacial digital syndrome Type-1, Marie Unna type of congenital hypotrichosis or the X linked Bazex-Dupre-Christol syndrome .[22]

8.EPSTEIN PEARLS

They are also known as gingival cysts of the newborn. They are yellowish white cystic lesions that can be seen over the gums and roof of the mouth in a newborn . They spontaneously resolve within 1 to 2 weeks of birth. [23]

9.HARLEQUIN COLOR CHANGE

This bizarre benign episodic color change was first reported in 1952 by Neligan and Strang. It is a migratory pink color involving one lateral half of the infants body with simultaneous pallor of the other half separated by a strikingly sharp line of demarcation along the midline. It spontaneously resolves within minutes.

It is seen most commonly between the second and fifth day of life. The color change appears suddenly and lasts for 30 seconds to 20 minutes. The pathogenesis is thought to be due to an imbalance in the autonomic regulatory mechanism of cutaneous vessels. Hypoxia, transient tachypnea and heart anomalies have been implicated as possible trigger factors. It does not require any specific treatment. [23]

10. CUTIS MARMORATA

It refers to mottled/ marbled appearance of the skin of neonates in the first few months of life. It occurs due to immaturity of nerve supply to the superficial cutaneous vasculature. It is a common finding in babies with Downs syndrome. It becomes more prominent on cooling the skin which causes some cutaneous vessels to contract and the others to dilate, giving a mottled or marbled appearance. It disappears on rewarming, in contrast to cutis marmorata telangiectatica congenita[7]

11. NEONATAL OCCIPITAL ALOPECIA

Some neonates develop a clearly delimited, elongated oval alopecic patch over the occipital region around the second week of life. This occurs due to the normal shedding of occipital telogen hair [22]

TRANSIENT NON INFECTIVE DISEASES IN THE NEWBORN

1. ERYTHEMA TOXICUM NEONATORUM (ETN)

Syn: toxic erythema, erythema neonatorum allergicum, urticaria neonatorum, erythema papulatum of the newborn

It is a common benign and transient rash located over the trunk of healthy neonates. In 70% of the cases the rash consists of irregular yellowish white papules on an erythematous base and in the remaining 30%, the lesions are predominantly pustular. It may involve any part of the trunk and limbs but usually the palms and soles are spared. It usually begins between the first and fourth day of life and lasts for 2 to 3 days. On histopathology, the main inflammatory infiltrate within the lesions was found to be eosinophils. [24]

The cause is still unclear. It has been proposed that ETN might correspond to a minor graft versus host reaction caused due to materno-fetal transfer of lymphocytes that can occur either prior to or during delivery. In a recent study on the expression of human anti-bacterial peptide in skin biopsies of infants with ETN, it was found that the innate antimicrobial system is strengthened during the first few days of life. A positive direct correlation was found between the length of labour and the severity of ETN. This could be explained by- stimulation of human antibacterial peptide expression or just due to eosinophil attraction to the dermis due to prolonged contact of the neonatal skin with vaginal secretions.[25]

2. MILIARIA

It is a vesicular eruption that occurs due to accumulation of sweat into obstructed eccrine ducts. Neonates have a greater density of eccrine glands which predisposes them to develop miliaria. Other contributory factors are excessive and tightly wrapping the baby and phototherapy given to infants nursed in NICU.

It can be classified into 4 types; miliaria crystallina, miliaria rubra, miliaria pustulosa and miliaria profunda. Miliaria crystallina consists of very superficial and thin walled non- inflammatory vesicles which can rupture easily. In miliaria rubra small erythematous grouped papules may be found commonly over the skin folds. Miliaria pustulosa consists of pustular lesions and miliaria profunda consists of mildly inflammatory papules arising within the dermal portion of the eccrine duct. When the fluid from these lesions are stained with Wright stain, predominent lymphocytes are found in case of miliaria rubra. Skin biopsy is confirmatory. The lesions may aggravate with the use of occlusive emollients . They generally resolve with reduction in the environmental temperature. [26]

3. TRANSIENT NEONATAL PUSTULAR MELANOSIS

It was first described in 1976 by Ramamurthy *et* al. [28] The etiology is unknown. Males and females are affected equally.

Clinically the neonates present with vesiculopustular lesions without surrounding erythema at birth or on the first day of life. These lesions rupture easily to leave behind pigmented macules with a characteristic collarette of scale which usually take 3 to 4 weeks to fade. The most common sites involved are the chin, neck, upper part of the chest, the lower back , buttocks, abdomen , thighs, even the palms and soles. The lesions are not associated with any systemic symptoms. [29] A Grams stain or Tzanck smear taken from the lesion shows neutrophils and a few eosinophils. Histopathology shows hyperkeratosis with intra corneal and sub corneal pustules containing neutrophils and occasional eosinophils .

These lesions resolve spontaneously and require no specific treatment. Parents should be counseled about the benign nature and course of the lesions.[27]

4. NEONATAL ACNE

It is also referred to as neonatal cephalic pustulosis. Prevalence has been found to be around 20% .[28,29] Most common presenting feature is erythematous papules and pustules with a typical absence of comedones except in cases of androgen derived neonatal acne. Lesions are found most commonly over the face and less often over other sites such as the scalp, neck and upper chest. Most cases are mild and heal spontaneously within three months. Exact etiology is not known. It is hypothesized to occur as a consequence of overgrowth of lipophilic yeasts like Malassezia furfur at

birth leading to an inflammatory eruption of papules and pustules due to follicular occlusion.[30,31]

5. INFANTILE ACROPUSTULOSIS

This condition is characterized by intensely pruritic recurrent vesiculopustular eruption mainly involving the palms and soles of infants. They may also occur over ankles, wrist, scalp and less frequently over buttocks. The lesions begin as papules and quickly progress to form pustules within 24 hours. These lesions last for around 7 to 14 days and tend to recur after 2 to 4 weeks. Spontaneous resolution is seen by 2 to 3 years of age.[32,33]

Etiology is unknown. An association has been found between infantile acropustulosis and atopic dermatitis and peripheral blood eosinophilia.[34]

On histopathology, the intra-epidermal pustules were found to be sterile, containing only polymorphonuclear neutrophils.

Dapsone has been found to be effective in a dose of 2mg/kg/day in two divided doses.[35]

6. SUCKING BLISTERS

Vigorous sucking by the infant during fetal life may result in the formation of calluses or erosions over the lips. It is believed to occur due to the effect of epithelial hyperplasia as well intracellular edema that occurs as an adaptive response to the mechanical effects of sucking. It is a benign and self limiting condition.[36,37]

7. EOSINOPHILIC PUSTULOSIS

This entity was first described by Ofuji in 1970. Characteristically, papules and pustules are seen which show centrifugal extension and central clearing with new papules arising at the border of older ones. The lesions may or may not be pruritic and they heal with residual pigmentation. It is not associated with systemic manifestations however peripheral blood eosinophilia is found in most cases. Histopathological examination shows eosinophilic infiltrate surrounding the hair follicle which may aggregate to form subepidermal pustules. The lesions are distributed on the head, neck and trunk as discrete, follicularly oriented erythematous papules. Other atypical forms that exist are non follicular erythematous papules , urticarial plaques and large erythematous patches. [38]

The etiology is unknown. Various theories have been proposed, one of which is infectious etiology. [39] Other theories are the immune theory and the hypersensitivity theory . One study found IgE antibodies to the dust mite Dermatophagoides pteronyssium in 3 patients with infantile eosinophilic pustulosis.[40]

Infants show an increase in sebum secretion which may contain chemokines for eosinophils involved in the pathogenesis of eosinophilic pustulosis.[41]

8. SUBCUTANEOUS FAT NECROSIS

It is a rare form of panniculitis in which erythematous indurated plaques and nodules may be found over the extremities, gluteal region, back and thighs and rarely over the face. They are self limiting and occur mostly in term and post term neonates. Other associated conditions are hypoglycemia, anemia, thrombocytopenia,

hypertryglyceridemia and most importantly hypercalcemia which may be life threatening.[42]

The etiopathogenesis of this condition is unclear. It has been found to be associated with various conditions like perinatal hypoxia, hypothermia, meconium aspiration, sepsis, obstetric trauma, gestational DM, pre-eclampsia and Rh incompatibility.

On histopathological examination , necrosis of adipocytes with chronic inflammatory infiltrate including foreign body giant cells may be seen. Alternatively fine needle aspiration cytology may be used to supplement the diagnosis.

Sclerema neonatorum is the main differential diagnosis of this condition. [42]

NAEVI AND OTHER DEVELOPMENTAL DEFECTS

Naevi are circumscribed, permanent non neoplastic lesions of the skin and/or mucosa; whereas developmental defects are tumor like non neoplastic proliferation of abnormal mixture of normal components of a tissue which arise due to errors in morphogenesis.

Naevi can be divided into epidermal naevi and dermal or subcutaneous naevi; the former includes verrucous and sebaceous nevi and the latter includes congenital melanocytic naevi, mongolian spot, nevus simplex and nevus flammeus. Other naevi- connective tissue naevi, smooth muscle naevi, fat naevi and vascular naevi. [7]

Epidermal nevi show a prevalence of about one in 1000 live births with equal sex incidence. One third of individuals also have involvement of other organ systems, hence it is considered to be an epidermal nevus syndrome, reported in upto 10% of individuals with epidermal nevi.[43]

Six syndromes have been described in association with epidermal nevi. These are: Proteus syndrome, congenital hemidysplasia with ichthyosiform nevus and limb defect (CHILD syndrome), phakomatosis pigmentokeratotica, sebaceous nevus, Becker's nevus and nevus comedonicus syndromes.[44]

1. VERRUCOUS EPIDERMAL NAEVI (VEN)

VEN are congenital, noninflammatory cutaneous hamartomas composed of keratinocytes. Each lesion occurs as a result of a single mutant keratinocyte.VEN can present as well defined patches, linear streaks or whorls that typically follow Blaschko's lines. This suggests that they occur as a result of post zygotic mutations. Most common site of involvement is the trunk and limbs followed by involvement of the head and neck rarely. Linear verrucous epidermal nevi (LVEN) appear usually at birth or during infancy as skin colored to brown, flat, velvety soft papillomatous lesions, which may become more keratotic/ hard during adolescence especially over the joints and flexural areas. Inflammatory linear verrucous epidermal nevi (ILVEN) consist of erythematous and itchy lesions in a linear pattern. ILVEN may be diffuse i.e systematized epidermal nevus or it may be unilateral i.e nevus unius lateralis. Histopathological examination shows orthohyperkeratosis, acanthosis, papillomatosis and a well demarcated expanding papillary dermis. Immunohistochemical studies help to differentiate ILVEN from other non inflammatory types of VEN. Treatment is often difficult and unsatisfactory. Topical therapies include corticosteroids, tars, retinoids, 5-fluorouracil and podophyllin. Surgical modalities of treatment like excision with full thickness skin graft, cryotherapy, carbon dioxide laser ablation and erbium-YAG laser therapies have been tried.[45]

2. SEBACEOUS NAEVI

Syn. Nevus sebaceous of Jadassohn, organoid nevus syndrome.

It was first described by Jadassohn in 1895. They are elevated, waxy orange-brown plaques with a granular surface present over the face and scalp most commonly. They grow slowly and develop a warty surface during infancy.[46] Risk of developing basal cell carcinoma is higher (6.5%) in nevus sebaceous as compared to the more common VEN.[45] Schimmelpenning-Feuerstein-Mims syndrome consists of sebaceous nevus associated with cerebral anomalies, coloboma, and lipodermoid of the conjunctiva.[47] Treatment of choice is surgical excision.[46]

3. CONGENITAL MELANOCYTIC NAEVI (CMN)

CMN is believed to occur due to disrupted migration of melanocyte precursors from the neural crest. Incidence is 0.2 to 2% . They appear as brown/black flat lesions, but in rare cases may also be raised. They have a potential risk for malignancy.[48,49] CMN greater than 20cm in size have been described as giant CMN/ garment nevi/bathing trunk nevi. They are rare and occur in less than one in 20,000 births. They have a 4-6% risk of becoming malignant. Hence, any nevus showing a change in shape, colour, surface or thickness should be immediately evaluated to rule out melanoma, which may even occur at a site away from the original site of the nevus in one third of cases .[48] Thus regular follow up is essential even after removal of the nevus.[50]

4. MONGOLIAN SPOT

Syn. Congenital dermal melanocytosis [51]

Mongolian spot is a congenital, developmental anomaly of the skin which occurs due to entrapment of melanocytes in the dermis during their migration from the neural crest into the epidermis.Prevalence of mongolian spot has a racial predilection-it is found in 90% of Native Americans, 80-90% of Asians, 80% of East Africans, 40-70% of Hispanics and less than 10% of Whites.[51,52]

It is usually present at birth but it may also appear within the first few weeks of the neonatal period.[51]

Clinically they appear as blue-grey macules/patches, few centimeters in diameter, solitary/ multiple. Most common location is the lumbosacral area, but the buttocks, shoulders, limbs, chest, back and flanks may also be affected. The typical bluish discoloration is due to the location of the pigment deep in the dermis which imparts a Tyndall effect of scattering of light.

Several variants exist- persistent mongolian spot, aberrant mongolian spot (unusual location) persistent aberrant mongolian spot (macular type blue nevi) and superimposed mongolian spot (dark mongolian spot overlying a lighter one).[51]

Mongolian spots usually fade in the first year of life. Risk factors for persistence of lesions are- large size i.e >10cm, extrasacral location, dark colored patches and multiple patches. At times they may persist indefinitely. These aberrant mongolian spots may be at an increased risk for inborn errors of metabolism like Hurlers syndrome, GM1 gangliosidosis type 1, Niemann-Pick disease and Hunters syndrome.[51]

These lesions are benign and require no treatment. Opaque cosmetics may be used to camouflage persistent and large lesions over exposed areas of the body. Q switched alexandrite laser, Q switched ruby laser and the Intense pulsed light laser have been tried for the treatment of aberrant mongolian spots.[51]

5. PORT-WINE STAIN

Also known as nevus flammeus, it is a vascular birthmark occuring in an estimated 0.3% of neonates.[53] They appear as flat, reddish purple patches visible at birth. They do not fade over time as seen in hemangiomas; in contrast, they may even deepen in color, develop varicosities , nodules or granulomas. Microstructure shows an increase in the diameter of the vessels without an increase in the number as is seen in hemangiomas. Port wine stain which occurs in the ophthalmic division of the trigeminal nerve may be associated with ipsilateral glaucoma either in isolation or in association with Sturge Weber Syndrome (triad of glaucoma, seizures and port wine stain with angiomas of the brain and meninges). Other associated syndromes are Parkes Weber syndrome, Klippel-Trenaunay Syndrome, Proteus syndrome, hyperkeratotic cutaneous capillary-venous malformation and PHACES syndrome (posterior fossa anomalies, hemangiomas, arterial anomalies, cardiac defects, eye anomalies, sternal anomalies). Patients seek treatment mainly for cosmetic purpose. Pulsed dye laser may be used to lighten the lesion, ideally before one year of age.[54] Beta blockers like oral propranolol 2mg/kg/day in two to three divided doses; and topical 0.5% timolol (beta blocker) can also be used for the management of port wine stain.[55]

6. HEMANGIOMA OF INFANCY

They are commonly referred to as birthmarks and are the most common benign tumors of childhood. They consist of rapidly proliferating endothelial tumor cells . They occur due to abnormal angiogenesis leading to excessive proliferation of vascular structures. Most of them occur sporadically as a result of developmental errors between the 4th to 10th week of gestation. It is found in around 3% of neonates, more frequently among preterm neonates.[56] Most common location is the head and neck (59%) followed by the trunk (24%) lower limbs (10%) and upper limbs (7%). They slowly begin to resolve , with 50% showing resolution by age 5, 70% by age 7 and 90% by age 9.[57]

7. NEVUS SIMPLEX

Synonyms : stork bite, angel kiss, salmon patch.

They are flat, blanchable often bilaterally symmetrical salmon colored lesions which may be located over the eyes, scalp and neck. They are benign lesions found in an estimated 33% of newborns with majority resolving within 18 months of age. [53]

8. ACCESSORY TRAGUS

The tragus of the external ear is a derivative of the first branchial arch, dorsal portion. Accessory tragi (pre-auricular tags: misnomer) are pedunculated flesh colored soft papules found anywhere along the line of fusion of the maxillary and mandibular branches of the first branchial arch. Goldenhar syndrome manifests as a triad of epibulbar dermoids, vertebral anomalies and accessory tragi. Treatment is by careful surgical excision. [58]

9. SUPERNUMERARY NIPPLES

It may consist of true glandular tissue, areola, nipples or their combination along the course of the embryonic breast line which runs from the axilla to the inner thigh. Diagnosis is usually clinical, which may be confirmed by histopathological demonstration of mammary tissue. Complete surgical excision is recommended especially if glandular tissue is present as it may enlarge at puberty and cause pain and embarassment to the patient.[59]

10. NEURAL TUBE DYSRAPHISM

The skin and the nervous system are both derived from the ectoderm. Separation of the neural and cutaneous ectoderm corresponds to the time of fusion of the neural tube. This explains the association of neural with cutaneous malformations.[59]

A. CRANIAL DYSRAPHISM

Syn : cephalocele/ cutaneous neural heterotopia

Leptomeningeal or glial tissue found in the dermis or subcutis due to faulty closure of the neural tube.

Cephalocele appear as soft, compressible, round or pedunculated nodules that increase in size when the baby cries or with Valsalva maneuver. They may be covered by normal skin or by a blue glistening translucent surface. They are midline defects and may be seen in the frontal, parietal and occipital regions. [59]

Cephalocele may be associated with hypertrichosis (hair collar sign), hemangiomas, capillary malformations, hemangiomas, cutaneous dimples, sinuses or overlying red blanchable patches. Treatment is by surgical correction. [59]

B. SPINAL DYSRAPHISM

It results due to an incomplete closure of the spinal axis. Large defects such as the meningomyelocele are apparent at birth whereas smaller occult lesions may be asymptomatic showing very subtle signs. It is highly essential to make an early diagnosis as it can prevent irreversible neurological damage. The cutaneous clues to the diagnosis are; hypertrichosis, dermoid cyst, lipoma hemangioma aplasia cutis, telangiectasias, capillary malformations and melanocytic nevus. The most common site is the lumbosacral region; however it may also occur over the cervical and thoracic region. Definitive diagnosis may be made only at the time of surgery. [59]

11. APLASIA CUTIS CONGENITA

Congenital absence of the skin most commonly aftects the scalp but may also affect other areas like the trunk and the extremities. Mode of inheritance is sporadic in most cases but may also show an autosomal dominant or recessive type of inheritance. It may be associated with other anomalies like cleft lip and palate, cutaneous organoid nevi, limb anomalies and epidermolysis bullosa. Other associations are teratogenic factors like methimazole, intrauterine varicella, herpes infection, elevated alfa feto protein in the maternal serum and amniotic fluid. It may also be associated with various syndromes like trisomy 13, Johanson-Blizzard syndrome, amniotic band disruption complex and ectodermal dysplasias.

The lesions are sharply marginated midline defects usually in the parietal or occipital areas of the scalp. They may clinically manifest with ulcers, bullae or scars which may be strikingly symmetrical. Large scalp defects may penetrate deep into the dura

or the meninges leading to complications like meningitis. intracranial bleed and venous thrombosis.

Cutaneous and bony lesions need only observation as these defects heal spontaneously over a few weeks to months. Larger , deeper lesions need to be monitored carefully for development of severe complications and may need prophylactic excision. [59]

GENODERMATOSES

Genodermatoses has been defined as genetically determined skin disorders , the course of which is determined by chromosomal aberrations or a single gene factor. They may be classified into three main groups: due to chromosomal aberrations, single gene defects and multifactorial disorders. Genetic disorders due to chromosomal aberrations may be further divided into numerical (eg. trisomy, monosomy etc) and structural (eg. translocations, deletions and duplications) Single gene defects may be inherited as autosomal dominant, recessive or X linked[59]

1. ICHTHYOSIS

It consists of a heterogenous group of inherited disorders of cornification clinically manifesting with large scaly lesions over the entire body. It is named after the Greek word ‘icthys’ which means fish due to the appearance of fish-like scales over the body. There are 4 major types of ichthyosis- Ichthyosis vulgaris, lamellar ichthyosis, X-linked ichthyosis and congenital ichthyosiform erythroderma. Frost and Van Scott initially grouped these disorders into those due to retention hyperkeratosis and those due to epidermal hyperproliferation.[59]

A. ICHTHYOSIS VULGARIS

This is the most common type showing clinical manifestations of the disease in 95% of the cases before the age of five years. It is inherited as an autosomal dominant trait.

Clinically the baby presents with fine whitish scales involving predominantly the extensors of the limbs and less commonly the trunk. The flexures and the face are usually spared. Palms and soles show features of hyperlinearity and mild hyperkeratosis.

On histopathological examination, the epidermis is found to be of normal thickness. The stratum granulosum is either reduced or absent. The keratohyaline granules are small and crumbly in appearance. The clinical severity of the disease is directly proportional to the filaggrin content of the stratum corneum. [60]

B. X-LINKED ICHTHYOSIS

It is an X-linked recessive disorder , usually manifesting within one to three weeks of age with predominant involvement of extensors more than flexures and a characteristic involvement of the preauricular areas. The skin lesion show a marked improvement in the summer season. It may be associated with features of asymptomatic corneal opacities, cryptorchidism, increased risk of testicular cancer, delayed developmental milestones, anosmia and hypogonadotropic hypogonadism.

Microscopic examination reveals hyperkeratosis with or without hypergranulosis. Keratohyaline granules appear small and numerous. Melanosomes are more in quantity in the horny cells due to the deficiency of the steroid sulfatase enzyme leading to an elevated level of cholesterol sulfate in the serum and the stratum

corneum. This causes abnormal hydrogen bonding leading to increased intracorneal cohesion. manifesting clinically as corneal opacity. [60]

C. LAMELLAR ICHTHYOSIS

Mode of inheritance is autosomal recessive. The baby presents with large plate like scales all over the body including the flexures associated with ectropion and palmoplantar keratoderma. Sometimes features of collodion baby may be seen. On light microscopy, hyperkeratosis, moderate degree of acanthosis , papillomatosis and prominent capillaries in the dermal papillae may be seen. On performing biochemical tests, free sterols and ceremide are found to be increased. [60]

D. NONBULLOUS ICHTHYOSIFORM ERYTHRODERMA

Mode of inheritance is autosomal recessive . It is the most common cause of collodion baby. The baby is born with extensive scaling all over the body which is large and plate like over the extensors and fine over the face, scalp and trunk. An increased incidence of premature birth was noted in this condition. It may be associated with cicatricial alopecia and secondary nail dystrophies. The infants are at increased risk of fluid and electrolyte imbalance, heat intolerance, growth retardation. Histopathology reveals hyperkeratosis, focal or diffuse parakeratosis and acanthosis with a marked increase in the turnover rate of epidermal cells.[60,61]

E. COLLODION BABY

In majority of the cases it is inherited as an autosomal recessive trait. In this condition, newborns are found encased within a tight, translucent parchment like sheet of skin called collodion membrane. [61] This term was coined by Hallopeau in 1884. Since then only 270 cases have been reported approximately.[62]

Congenital ichthyosiform erythroderma is the most common cause followed by lamellar ichthyosis and harlequin ichthyosis. Rarely it may be associated with bullous congenital ichthyosiform erythroderma, Sjogren-Larsson syndrome and Gaucher's disease.

Pathogenesis : It occurs due an epidermal cornification disorder due to keratinocyte protein and lipid metabolism defects.

The collodion membrane which covers the newborn like an armor, restricts both the respiration as well as the suckling of the baby.It may constrict the limbs and lead to loss of their function. This membrane peels off in a week or two leaving behind dry and rough skin with fissures. As a result the baby is more prone to serious complications like life threatening infections, fluid and electrolyte imbalance, temperature instability and hyponatremic dehydration. In babies with ectropion, keratitis and eventually blindness may occur if not managed carefully.

Microscopic examination reveals hyperkeratosis with hypogranulosis . Dense intracytoplasmic granules with convoluted corneocytes may be found in the upper layers of the stratum corneum. Numerous lamellar bodies are found in the intercellular spaces.

Treatment includes careful monitoring and supportive care, correction of any fluid and electrolyte balance if present and providing analgesics and eye care. Ideally these babies should be nursed in humidified incubators with water dressings and application of regular emollients. [61] Since these babies are at a high risk of infection, a careful selection of antibiotics should be considered. Ventilatory support should be provided to those with respiratory failure. Keratolytics like lactic acid, salicylic acid and

propylene glycol can be used. Patients with localised lesions may obtain some relief with topical retinoids whereas those with extensive lesions require treatment with systemic retinoids. [61]

F. EPIDERMOLYTIC HYPERKERATOSIS

This is a rare disorder inherited as autosomal dominant or it may be sporadic. The affected baby manifests soon after birth with erythema, scaling and blister formation. Scaling is thick and grey-brown showing a verrucous appearance over the flexures. Blistering reduces with age. The skin over the flexures may develop a rigid, rippled appearance.

On microscopy, hyperkeratosis with accentuated rete ridges is seen, with variable sized keratohyaline granules in the stratum granulosa and vacuolar degeneration of the basal layer. Dermis shows edema and lymphohistiocytic infiltrate.The disorder is thought to result due to an abnormality in keratin synthesis.[60]

G. OTHER RARE ICHTHYOSIFORM SYNDROMES

It includes- Harlequin type, Lamellar dominant type, Ichthyosis hysterix, Netherton type, Sjogren-Larsson type, Refsum's disease, Chananrin-Dorfam syndrome, multiple sulfatase deficiency, trichothiodystrophy, KID syndrome (keratitis, ichthyosis and deafness), CHILD syndrome (congenital hemidysplasia, ichthyosiform hemidysplasia, defect of extremities), Erythrokeratolysis heimalis, erythrokeratoderma variabilis, peeling skin type and Darier type.[60]

2. EPIDERMOLYSIS BULLOSA (EB)

It refers to a group of inherited mechanobullous disorders which have the common features of development of vesicles and bullae at the sites of trivial trauma. It may be divided into three types depending on the level of the blister- EB simplex, Junctional EB (JEB) and dystrophic EB (DEB). Severe forms of EBS Dowling Meara type, Herlitz type JEB and recessive DEB can be lethal in the neonatal period. [60]

A. EPIDERMOLYSIS BULLOSA SIMPLEX (EBS)

It has an autosomal dominant mode of inheritance with an estimated incidence around 1 in 5,00,000 births. The child may be affected at birth or shortly thereafter with improvement in 2 to 3 months and tendency to recur later in childhood. Clinically the baby has blisters all over the body with a tendency for acral distribution. The lesions heal without scarring unless secondarily infected. The condition shows seasonal variation- worsens in the summer and improves during winter season. [60]

Variants of EBS include-

- EBS localisata (Weber-Cockayne)
- EBS herpetiformis : lesions follow a herpetiform pattern and heal without scarring. It may be associated with hyperkeratosis of the palms and soles.[60]
- EBS with mottled pigmentation: hypo/hyperpigmented macules present at birth which gradually fade with age.
- EBS Ogna variant: manifests at birth with small acral traumatic sanguineous blebs.[60]

B. JUNCTIONAL EPIDERMOLYSIS BULLOSA

Autosomal recessive inheritance. Clinical presentation ranges from very mild signs to severe life threatening forms. Histopathology reveals a split at the level of the lamina lucida with decreased number of hemidesmosomes and absent sublamina densa. [60]

EB lethalis : Characterised clinically by generalised blistering with relative sparing of the hands. There is characteristic perioral and perinasal hypertrophic granulation tissue. Systemic involvement of the GIT, gall bladder and vagina has been reported. Mortality can be high due to predisposition to infection. [60]

- EB atrophicus generalisata mitis : Blisters first appear at birth and then improve with age. Mucosal involvement along with cutaneous atrophy and enamel hypoplasia have been reported. [60]

- EB atrophicus inversa : Pyoderma like blistering can be seen in the neonatal period which improves in a few months. A characteristic finding in this condition is an elevated white streak called ‘albostriata lesion’. It may also be associated with nail dystrophy and corneal opacity. [60]

C. DERMOLYTIC EPIDERMOLYSIS BULLOSA

- Epidermolysis bullosa dystrophica of the hyperplastic type : autosomal dominant mode of inheritance with serosanguineous blisters limited to the extremities, predominantly over the extensors. The lesions heal with milia formation and scarring. Dystrophy of the nails is a common finding. [60]

- Epidermolysis bullosa dystrophica of the albopapuloid type : autosomal dominant inheritance with blistering at birth or early infancy. Lesions are termed

'albopapuloid' due to the characteristic appearance of white papules over the trunk and extremities during adolescence. In both these forementioned conditions, the primary defect lies in the anchoring fibrils.

- Recessive dystrophic EB : neonate with this condition presents with multiple subepidermal blisters over the body with characteristic scarring milia and nail dystrophy. It has been further subclassified into- recessive EB gravis, recessive EB mitis, recessive EB inversa, recessive EB of the centripetal variety and transient EB of newborns. [60]

Gold standard for diagnosis of epidermolysis bullosa in transmission electron microscopy. However, immunofluorescence is preferred as it is easy to perform and less time consuming. There is no specific treatment for epidermolysis bullosa. The newborn should be nursed in a neonatal intensive care unit specialized for EB patients where use of incubators or radiant warmers is avoided. Mainly supportive management is given with due importance to prevention of mechanical trauma, minimal handling and counseling with respect to wound management and its prevention. [62]

3. EHLER-DANLOS SYNDROME

It is a heterogenous group of connective tissue disorders characterised by hyperextensible, fragile skin and hypermobile joints. Fragility of the skin leads to easy bruising, bleeding, gaping wounds and healing with cigar paper scars. Primary pathogenesis is due to defect in the biosynthesis of collagen. It is divided into six major types : classic type, hypermobile type, kyphoscoliosis type, vascular type , arthrochalasia type and the dermatosparaxis type. This classification is of prognostic

significance as certain types are associated with fatal complications- eg. the vascular type is associated with arterial, bowel and uterine rupture. [60]

4.CUTIS LAXA

It is a condition characterized by generalized elastolysis which can be inherited as autosomal recessive trait more commonly, or sometimes as autosomal dominant trait. It occurs due to reduced elastin which may be a consequence of decreased synthesis by fibroblasts, synthesis of structurally abnormal elastin or due to an overactivity of the elastase enzyme. Histological examination shows sparse and inelastic fibres which may be short, fragmented and clumped or may show granular degeneration. In the autosomal recessive form, babies are born with a characteristic facies- slanting palpebral fissues, broad flat nose, large ears and sagging cheeks. Prominent skin folds may be seen over the abdomen, thighs and knees. Other less commonly associated features include hernias, gastrointestinal and urinary tract diverticulas, pulmonary emphysema, hip dislocation, osteoporosis and genital anomalies. In the autosomal dominant form, the skin changes may develop at any age. The child may show intrauterine growth retardation, lax ligaments and delayed closure of the cranial fontanelles. [60]

5. INCONTINENTIA PIGMENTII

It is also known as Bloch-Sulzberger Syndrome. It is a rare, multisystem neuroectodermal disorder with X-linked dominant mode of inheritance. It predominantly affects the skin, eyes, teeth, hair, nails and the central nervous system. It is caused due to a mutation in the nuclear factor-kappa B essential modulator gene (NEMO) which is located on chromosome Xq28. This gene encodes a protein which

regulates the expression of various cytokines, chemokines and adhesion molecules which is essential for protection against tumour necrosis factor induced apoptosis. There are four stages of the disease, first stage : erythematous vesicular lesions following the lines of Blaschko which may be present at birth or appear shortly after, usually lasting for two weeks to four months. In the second stage: verrucous lesions appear over the distal limbs. Third stage : whorled slate grey to brown hyperpigmented lesions may be seen following the lines of Blaschko and in the fourth stage, permanent pale anhidrotic hairless lines free of sweat glands most frequently over the posterior aspect of the legs and arms may be seen.[63] Criteria for diagnosis of incontinentia pigmentii have been suggested by Landy and Donnai. [64]

Major neurological findings include seizures, delayed psychomotor development, hemiplegia, hemiparesis, mental retardation, spasticity and microcephaly. Eye abnormalities are vascular retinopathy leading to retinal detachment.

Characteristic histopathological findings include eosinophilic spongiosis and apoptosis which will be present at birth.

Management is aimed at early diagnosis of these patients and prompt instigation of neurological and ophthalmic care. [65]

6. PIEBALDISM

It is an autosomal dominant disorder of congenital leukoderma characterized by a white forelock, which is seen in 90% of the cases. Other areas that may be devoid of pigment include central forehead, chin and ventral aspect of the trunk. Other areas that may be depigmented include eyebrows, mid arm and mid leg.

The basic molecular level defect in this disorder is of the c-kit proto-oncogene . The affected areas show an absence of melanocytes.

The leukoderma and white forelock remain constant throughout life. Cosmetic camouflage is the treatment option provided to infants and children. [26]

7. ECTODERMAL DYSPLASIA

They are a heterogenous group of inherited disorders with defective development of atleast two structures of ectodermal origin. The structures most commonly involved include the hair follicles, nail, teeth, conjunctiva, sweat glands, sebaceous glands and lens. A total of 154 different types of ectodermal dysplasia have been included in the classification based on the specific structures affected. Out of these, there are three types which show characteristic features:

A. Hypohidrotic Ectodermal Dysplasia (Christ-Siemens-Touraine syndrome)

X-linked recessive disorder with congenital absence of sweat glands leading to heat intolerance and episodes of hyperpyrexia. In the neonatal period they may present with features of severe peeling of skin all over the body. Thereafter they may develop pale, dry skin with prominent venous pattern over the body and hyperpigmented, wrinkled skin over the periorbital area.

The characteristic craniofacial features include frontal bossing, depression of the midface, flattened nasal bridge, thick protruding lips, prominent chin and hypodontia. Other features include atopic dermatitis, atrophic rhinitis, diminished lacrimation and hoarseness of voice.

Management includes counselling of parents so as to prevent the neonates from overheating. Other supportive measures include instillation of lubricant tear drops, saline drops or irrigation of the nasal cavity and regular dental evaluation. Reconstructive surgery is required for the correction of facial deformities.[26]

B. Hidrotic Ectodermal Dysplasia (Clouston Syndrome)

It is an autosomal dominant disorder characterized by abnormalities of the skin, hair and nails with normal functioning of the eccrine and sebaceous glands. Clinical features include thick, conical nails with widening of the distal periungal area ; hyperpigmented skin overlying the joints and a variable degree of alopecia.[26]

C. Ectodermal dysplasia-Ectrodactyly-Cleft lip/palate syndrome:

It is caused by a mutation of the p63 gene that leads to cutaneous and appendageal anomalies diffuse hypopigmentation of the skin and hair, scanty scalp and eyebrow hair, dystrophic nails and small teeth. Other less common features include conjunctivitis, blepharitis, xerostomia, conductive hearing loss and mental retardation.[26]

8. OCULOCUTANEOUS ALBINISM

Group of congenital disorders that occur due to an absence of pigment of the skin, hair and eyes due any defect that interfers with either the synthesis or transport of melanin. It is inherited as autosomal recessive, except for type 1.

Affected infants have decreased pigmentation of the skin with varying pigmentation of the hair and iris. Eye changes include nystagmus, photophobia and impaired visual

acuity. Other less common features include small stature, defective mentation and hemorrhagic diathesis.

Treatment is supportive with counseling regarding sun protection and even complete avoidance if possible.[26]

9. CAFE-AU-LAIT MACULES

Syn. Circumscribed cafe au lait hypermelanosis, Recklinghausen spot.

They are well circumscribed, pigmented macules to patches varying in size from 1mm to more than 20 cm in diameter. Overall prevalence of café-au-lait macules varies with race: it has been reported to be present in 0.3% of Caucasian, 0.4% of Chinese, 3% of Hispanic and 18% of African Americanneonates. Finding one or two cafe au lait macules in a neonate is normal. However, the presence of multiple cafe au lait, large segmental lesions, associated facial dysmorphism, other unusual findings on clinical examination should alert the physician to the possibility of an associated syndrome, most common amongst which is neurofibromatosis type 1. Other less common syndromes include Legius syndrome, Noonan syndrome and other neuro-cardi-facial cutaneous syndromes.[66]

10. CONGENITAL ERYTHROPOIETIC PORPHYRIA

Syn. Gunther disease

It is a rare autosomal recessive disorder that occurs due to deficient activity of uroporphyrinogen III synthase . This leads to non enzymatic conversion of hydroxymethylbilane to uroporphyrinogen I which is further converted to coproporphyrinogen I . These substrates are further oxidized to uroporphyrin I (URO-

I) and coproporphyrin-I (COPRO-I) which are phototoxic. They accumulate in the erythrocytes resulting in massive hemolysis and release into the blood, skin, bones and teeth and are excreted in large amounts in the urine and feces.[67]

The clinical severity of the disease is highly variable. It can range from hydrops fetalis to mild cutaneous symptoms with onset in adulthood. Usual presentation is with complains of photosensitivity and increased skin fragility since birth or early infancy with development of vesicles and bullae over the areas exposed to either sunlight or artificial sources of light. Other common features include scarring alopecia of the scalp, hypertrichosis of the face and extremities and pigmentary skin changes. Over a period of time they may develop severe mutilation of facial structures especially the nasal and auricular cartilage, ectropion, eclabium as well as shortening of the digits.

Histological examination reveals subepidermal blister with accumulation of PAS positive, diastase resistant, homogenous hyaline material (porphyrins) perivascularly, with minimal inflammatory infiltrate. [68]

Prenatal diagnosis can be made by amniotic fluid analysis for uroporphyrin I or direct gene mutation analysis. [69]

Management includes strict sun protection, splenectomy if required in cases of severe hemolytic anemia, bone marrow transplantation and replacement of the defective enzyme through cord blood infusion. Replacement gene therapy has been tried successfully in vitro. [70,71]

INFECTIONS

A. BACTERIAL INFECTIONS

1. BULLOUS IMPETIGO

Bullous impetigo is caused only by infection with exfoliative or epidermolytic toxin producing, coagulase positive staphylococcus aureus. In some neonates and immunocompromised adults, it may become generalised and this was erroneously termed as pemphigus neonatorum in the past. It is characterized by large, fragile, flaccid bullae that can rupture and ooze yellow fluid. The pathognomonic collarette of scales on its periphery develops after the bullae rupture, leaving a thin, brown crust with no scarring. Histopathological examination reveals intraepidermal blister at the level of the granular layer.

Diagnosis can be established by the typical clinical presentation, Gram staining, bacterial culture and sensitivity of the blister fluid and skin biopsy.

These patients require prompt treatment with appropriate antibiotics in order to prevent systemic complications such as pneumonia, osteomyelitis, septic arthritis and septicemia. [67]

2. STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Syn: Ritter's disease

Staphylococcal scalded skin syndrome (SSSS) occurs due to infection or colonization of *S. aureus* group 2 phage type 71 that produce circulating exfoliative exotoxinsA (89%), B (4%) and both A and B (7%). Immunohistochemical studies show that exfoliative toxin-A binds and lyses fillagrin in the keratohyaline granules leading to epidermal splitting possibly due to proteolytic rupture of the desmosomes.

It was first described by Ritter et al in 1878 who termed the disease 'dermatitis exfoliativa neonatorum'.

It is usually seen in infants and children less than five years and rarely seen in older age group. Within 48 hours of a prodrome of sore throat or severe conjunctivitis, follow fever, malaise and extremely tender erythematous patches on the face , neck and other flexural areas. Within these lesions flaccid bullae develop eventually that rupture easily revealing a moist erythematous "scalded" base. The lesions heal without scarring. Nikolsky's sign is positive. Patients with extensive blistering may develop complications like hypothermia, hypotension, electrolyte imbalance, respiratory distress and secondary infection.

Histopathology reveals a subcorneal split at the level of the granular layer. Immunohistochemical studies show exfoliative toxin A binding to fillagrin in the keratohyalin granules of the granular cell layer.

Treatment is by early institution of appropriate antibiotics along with supportive care. [68]

3. MASTITIS

The most common causative organism is Staphylococcus aureus. It is seen in neonates with a peak at three weeks of age. It is usually unilateral and not associated with fever or any systemic symptoms. Early cases may be treated with antibiotics but surgical drainage is required if an abscess is formed. Other complications include cellulitis, fasciitis, osteomyelitis, brain abscess and sepsis. [69]

4. OMPHALITIS

The estimated incidence of omphalitis in developed countries is around 0.7% in developed countries and 6.18% in developing countries. [70,71] Risk factors include protracted labour, prolonged rupture of membranes, unsterile delivery, prematurity and low birth weight; [71,72] and use of umbilical catheters or topical application of ash/ other material as a cultural practice. [73]

The umbilicus becomes colonized by bacteria from the maternal genital tract or from the external environment soon after birth. The most common organisms in developing world include Staph aureus, Staph epidermidis, Streptococcus Group A & B , E.coli, Pseudomonas and Clostridium difficile.

It is characterized by peri-umbilical edema, erythema and tenderness, with or without discharge. [74] In some cases it may progress to cause cellulitis, fasciitis and lymphangitis of the abdominal wall; and rarely serious complications like sepsis, necrotizing fasciitis, abcesses, peritonitis and hepatic vein thrombosis.[75]

Management includes supportive measures along with institution of antibiotics according to the pus culture and sensitivity report.[76]

5. CONGENITAL SYPHILIS

Syphilis is a sexually transmitted disease caused by the bacteria Treponema pallidum. If mother is infected with the bacteria, the organism can cross the placenta and infect the fetus from 14 weeks of gestation; with the risk on infection increasing with gestational age.[77] The manifestation of the infection in neonates depends on various factors- gestational age of fetus at the time of acquiring the infection, stage of

maternal syphilis, treatment received by the mother and immunological status of the fetus.

Congenital syphilis can lead to abortion, still birth or preterm delivery; 60% of neonates are asymptomatic at birth . It may be divided into early (symptoms appear within first two years of life) and late congenital syphilis (symptoms appear over the first two decades).

In early congenital syphilis, infants may present with small coppery red maculopapular lesions with peeling of skin and crusting developing over the span of 1 to 3 weeks. Rhinitis (syphilitic snuffles) nasal discharge, nasal septal perforation are other early features. After 2 to 3 weeks, the perioral and perineal area may develop wart like lesions known as condyloma lata which can lead to fissures and scarring (rhagades). The neonate may or may not show any signs of neurosyphilis. Diagnosis can then be made by a reactive CSF-VDRL. Ocular manifestations include chorioretinitis, uveitis, glaucoma, cataract, salt and pepper fundus, and eyelid chancre. Other features include hepato-spleenomegaly, hemolytic anemia and jaundice.

Late congenital syphilis occurs in approximately 40% of untreated children. The affected neonates have peg shaped notched central incisors (Hutchison's teeth) and multi cuspid first molars (mulberry molars) with increased chance of dental caries. Saddle nose deformity is another late stigmata of the disease which occurs as a result of nasal septal perforation.

Prenatal diagnostic methods include amniocentesis, percutaneous umbilical cord sampling and USG abdomen. The amniotic fluid sample is subjected to dark field microscopic examination.

Adequate treatment of mother is effective for preventing transmission to the fetus as well as for treating fetal infection. Drug of choice is Penicillin G .[78]

6. NOMA NEONATORUM

It is a gangrenous disease with Pseudomonas aeruginosa implicated as the most common causative agent in developing countries. Predisposing factors include prematurity, low birth weight, malnutrition, previous illness and underlying immunodeficiency state like severe combined immunodeficiency. The infants present with ulcers over the nose, lips, mouth, anus, scrotum and eyelids which is of abrupt onset and in severe cases may lead to severe mutilating deformities.

Treatment is with appropriate antibiotics and surgical debridement.[79]

B. VIRAL INFECTIONS

1. HERPES SIMPLEX (HSV)

Neonatal HSV infections are acquired through vertical transmission from the mother either in utero, during delivery or after delivery through contact with infected material. Maternal genital HSV infections are associated with preterm labour and low birth weight infants. Neonatal HSV infection is caused by HSV-2 virus in the majority of cases but in 15-30% of cases it is caused by HSV-1.

Congenitally acquired infection can lead to microcephaly, hydrocephalus, chorioretinitis and vesicular skin lesions. Infection acquired during or after birth may show one of the following three patterns- localized to the skin and mucosa, encephalitis with or without skin and mucosal involvement and widespread disseminated infection.

Diagnosis can be difficult in the early stages as the neonate presents with non specific findings. Delay in diagnosis increases the morbidity as well as mortality rate despite anti viral therapy.

Intrauterine infection can be prevented by treating the maternal genital herpes with antivirals . Infants who are suspected of or are diagnosed with HSV infection should receive antivirals- acyclovir 60mg/kg/day in three divided doses IV for 14 days in case of limited disease and for 21 days in case of disseminated disease. [80]

2. VARICELLA ZOSTER

If mother gets infected in her first trimester of pregnancy, the offspring develops congenital varicella associated with multiple anomalies. 'Fetal varicella syndrome' develops in <2% of newborns born to mothers who acquire the infection between 7-28 weeks of pregnancy. It is associated with a mortality rate of 30%. Clinically the newborn presents with low birth weight, cutaneous scars in a dermatomal distribution, ocular anomalies like cataract, microphthalmia and chorioretinitis, bone and muscle hypoplasia, neurological anomalies like mental retardation, seizures, microcephaly, hydrocephaly and cortical atrophy ; apart from other features like Horner's syndrome and bowel and bladder dysfunction.

If the mother develops varicella 5 days before to 2 days after delivery the neonate develops 'neonatal varicella'. 30% of such cases may develop fulminant varicella with systemic dissemination. In such cases VZIG i.e varicella zoster immunoglobulin 625 units I.M should be given prophylactically immediately after birth. Acyclovir 10-15mg/kg/day should be infused iv every 8th hourly for 5-7 days. If possible, the

delivery date should be postponed so as to allow adequate time for passage of protective maternal antibody. [81]

3. RUBELLA

Maternal infection can be transmitted to the fetus in utero during any period of gestation but is most common in the first trimester and is rare afterwards. The virus affects rapidly dividing cells and hence leads to a defect in organogenesis. The neonate presents with a rash consisting of 2 to 20 mm raised, erythematous, soft spongy lesions with a characteristic 'blueberry muffin' appearance. Petechiae and purpura may also be found.

Confirmative diagnostic method is take a pharyngeal swab for viral culture. Maternal serological tests can be done to detect the virus. Treatment consists of only supportive management as no specific antivirals exist for rubella. Immunization of children against rubella is specifically given to prevent development of this congenital rubella syndrome in their offspring. [82]

4. HUMAN PARVOVIRUS B19

Syn: Erythema infectiosum, Fifth disease

This virus causes a benign self limiting viral exanthem during childhood consisting of a typical rash with slapped cheek appearance followed by a reticulate, lacy, truncal eruption. Infection during pregnancy has been associated with an increased risk of abortion, fetal hydrops, Intrauterine growth retardation and pleural or pericardial effusion.[82] Symptomatic neonatal infection is rare. Most common finding includes pallor, maceration and subcutaneous edema.

Prenatal diagnostic methods include Radioimmunoassay and ELISA to detect IgG/IgM antibodies. Affected symptomatic neonates need supportive care and blood transfusion if required .[83]

5. CYTOMEGALOVIRUS (CMV)

CMV infection can be acquired congenitally, perinatally or postnatally. Severity of the infection in the neonate depends on the time of acquiring the infection , with more severe manifestations occurring if the infection is acquired in the first trimester. It is a double stranded DNA virus belonging to the herpes virus group. The skin lesions resemble that of congenital rubella syndrome with 'blueberry muffin spots' rash and petechiae.

Viral culture of urine/saliva is the easiest method of confirmation of the diagnosis. Serological tests include detection of IgM and persistently increasing levels of IgG in the serum. Other tests include PCR for detection of CMV-DNA. Currently the best antiviral available for treatment of this infection is ganciclovir. [84]

C. FUNGAL INFECTIONS

1. CANDIDIASIS

Depending on the time of acquiring the infection it may be divided into two main types- congenital cutaneous candidiasis and neonatal candidiasis.

Congenital cutaneous candidiasis results due to spread of the infection in utero . The classical presentation is a generalized erythematous naculopapular rash with or without pustules over an erythematous base, distributed predominantly over the back, flexures, palms and soles, and extensor surface of the limbs. The oral mucosa is rarely

involved. The diaper area is spared. These lesions gradually evolve into pustules, vesicles and bullae over a span of few days and later resolve with desquammation over 1-2 weeks.

Neonatal candidiasis results due to spread of the infection during passage through the infected birth canal. It manifests after one week of life with lesions localized to the oral cavity and diaper area. However in low birth weight and immunocompromised infants it may progress to severe systemic candidiasis.

Treatment with antifungals has been recommended for systemic disease. First line drug is IV amphotericin B; Other drugs include fluconazole IV 6mg/kg/day, voriconazole, caspofungin and micafungin. [85]

2. DERMATOPHYTOSIS

Tinea in neonates is rare, possibly due to the high rate of sebum secretion which has anti bacterial and anti fungal properties. The causative agents reported in various case studies are Trichophyton rubrum, Microsporum canis, Microsporum gypseum, Trichophyton violaceum and Trichophyton mentagrophytes. Most common site affected in neonates is the scalp and face. Tinea capitis presents with erythematous scaly lesions with patchy alopecia. [86,87] Lesions may vary from the non-inflammatory black dot alopecia to the inflammatory ‘kerion’. It is usually associated with a posterior cervical lymphadenopathy. Tinea diaper dermatitis presents as annular plaques with scaling over the borders. Diagnosis can be confirmed by a simple KOH examination of the skin scrapings. Controversy remains on whether to treat neonatal tinea with either systemic or topical anti-fungals. Topical therapy is considered safer. It has also been proposed that topical therapy is more effective in neonatal period due

to increased permeability of the skin. Topical 1% clotrimazole for 2 to 3 weeks is effective.[86]

MITES AND PARASITES

1. SCABIES

It is caused by the mite Sarcoptes scabiei var hominis. Neonatal scabies usually manifests with atypical presentation. As the neonate is unable to scratch, presenting features may be irritability, poor feeding and insomnia. On examination, lesions are pleomorphic and consist of papules, pustules and vesicles, classically seen over the palms and soles. However any part of the body may be involved. Nodules and crusted lesions may also be seen. Diagnosis can be confirmed by obtaining a skin scraping and observing under a drop of mineral oil.[88] Treatment for infants less than two months of age is with topical application of 10% crotamiton in petrolatum for 3 to 5 days, to be washed after 24 hours and 5% sulphur cream daily for 2 to 3 days. [87] Topical 5% permethrin is FDA approved only after two months of age. Treatment of the mother and other child handlers should also be undertaken[88]

2. TOXOPLASMOSIS

This infection is caused by a protozoa Toxoplasma gondii. The risk of transmission from the mother to the fetus is 15% in first trimester, 30% in second and 60% in third trimester. Acquiring the disease in early neonatal period results in fetal death or severe neurological or ophthalmological disease; whereas those infected in the second and third trimester have mild or subclinical manifestations.

Cutaneous manifestations are variable- they include macules, papules, pustules and vesiculobullous eruptions. Diagnosis can be supported with serological investigations. Prenatal transmission from the mother can be prevented by maternal treatment with the drug spiramycin. Symptomatic congenital toxoplasmosis may be treated with pyrimethamine, sulfadiazine and folinic acid, which should be given for atleast a year.[89]

ARTHROPODS

A variety of skin lesions may result due to bite or sting by an arthropod. Clinically, erythematous urticarial papules and pustules with a central punctum may be seen predominantly over the exposed areas of the body. These lesions are typically arranged in groups. Persistence of these lesions for weeks to months is a rare occurence. Presence of fever and pus discharge from the lesion would suggest the presence of a secondary infection and it warrants institution of antibiotic therapy.[90]

ECZEMA

1. INFANTILE SEBORRHEIC DERMATITIS

Syn. Cradle cap

It is a type of endogenous eczema affecting the seborrheic areas of the body. P.ovale, a lipophilic yeast found as a commensal on normal skin has found found to play a role in its pathogenesis. The most common regions involved in a neonate is the face, scalp and the diaper area. Flexural areas may or may not be affected. It may be localized or disseminated. Dark skinned neonates most commonly present with hypopigmentation and in some cases fissures may develop which can get secondarily infected. It spontaneously resolves in most cases by the end of the first year of life. Topical

agents like ketoconazole cream/shampoo with zinc pyrithione or selenium sulphide can be used in symptomatic cases.[26]

2. ATOPIC DERMATITIS (AD)

In 60% of the cases of atopic dermatitis, signs and symptoms of the disease begin at infancy. However the affected infant may not show all the signs required for making a definite diagnosis. It starts as erythema and scaling of the cheeks, sometimes extending to involve the forehead, chin and scalp but sparing the perioral and periorbital area. It may mimic seborrheic dermatitis, but it classically spares the diaper region .These infants are at increased risk of developing cutaneous bacterial, viral, fungal and viral infections. Staphylococcus aureus plays an important role in the exacerbation of AD. Treatment is mainly symptomatic, and is aimed at maintaining hydration of the skin, preventing any triggers and controlling the pruritis. Prolonged breast feeding has been found to have a protective role especially in those with a strong family history of atopy.[26]

3. DIAPER DERMATITIS

Syn. Napkin dermatitis, Irritant diaper dermatitis, nappy or diaper rash

It is a type of irritant contact dermatitis that occurs due to irritation caused by exposure of the skin to prolonged wetness, usually urine or feces. This causes an alteration in the skin pH and resultant breakdown of the epidermis. It is characterized by patches of erythema and scaling over the convex surface of the gluteal region with characteristic sparing of the skin folds. In case of secondary bacterial/fungal infection even the skin folds may get involved showing large areas of beefy red erythema with satellite pustules. Candida is the most common opportunistic organism that has been

implicated; others are Proteus mirabilis, Pseudomonas aeruginosa and enterococci. The most effective treatment option is to discontinue the use of diapers. Barrier creams or ointments especially zinc oxide based are effective in prevention due to their drying effect, antiseptic and astringent role. In severe cases, a mild steroid with antifungal combination cream can be given. [91]

NUTRITIONAL DERMATOSIS

1. ACRODERMATITIS ENTEROPATHICA

It is a rare autosomal recessive disorder that causes a defective zinc uptake by the deodenum and jejunum. The defective gene is SLC39A4. The characteristic triad, seen in only 20% of the cases, consists of symmetrical acral and periorificial dermatitis, diarrhea and alopecia. The onset of symptoms usually coincides with the time of weaning. The skin lesions which are initially erythematous patches later becoming vesiculobullous, pustular or erythemato-squamous. Mucosal lesions include gingivitis, glossitis and stomatitis. There may be complete absence of scalp hair eyelashes and eyebrows. The gastrointestinal symptoms may be associated with anorexia and growth failure. Neuropsychiatric symptoms like irritability, lethargy and depression are the last to appear. Treatment is with oral zinc supplementation at the dose of 5-10mg/kg/day followed by a maintenance dose of 1-2mg/kg/day.[92]

2. NEONATAL BIOTIN DEFICIENCY

This occurs due to holocarboxylase synthetase (HCS) deficiency which manifests within the first six weeks of life. Biotin is required for functioning of the carboxylase enzymes involved in fatty acid synthesis, gluconeogenesis and amino acid catabolism. HCS deficiency causes severe, bilaterally symmetrical well demarcated periorificial and intertriginous dermatitis, universal alopecia, severe metabolic acidosis and

aciduria. Other manifestations include seizures, lethargy, projectile vomiting, athetosis, abnormal muscle tone, progressive loss of consciousness and coma. It can be fatal if not diagnosed and treated early.[93,94]

Diagnosis can be established by evaluating the urinary organic acid excretion, chiefly 3-hydroxyisovaleric acid. Enzyme assays can be used to detect carboylase deficiency. Fibroblast analysis can be used to detect poor incorporation of biotin into carboxylase and low transfer of biotin by holocarboxylase synthetase. Treatment is with lifelong biotin suplementation at the dose of 20-40mg/day.[94,95]

IMMUNODEFICIENCY SYNDROMES [96]

SYNDROME	DEFECTIVE GENE	CUTANEOUS FEATURES	ASSOCIATED FINDINGS
Severe combined immunodeficiency	T-cell deficiency	Erythroderma, seborrheic dermatitis, morbilliform rash	Failure to thrive diarrhea pneumonia
Omenn's syndrome	RAG gene mutation	Erythroderma, alopecia	Failure to thrive, hepatospleenomegaly, lymphadenopathy, elevated IgE, eosinophilia
Wiskott-Aldrich Syndrome	WAS gene	Eczema, petechiae, purpura, erythroderma	thrombocytopenia, auto immune disease, lymphoreticular malignancy
Chronic granulomatous disease	NADPH oxidase defect	Granulomas	respiratory tract infections, hepatospleenomegaly, hepatic abcess
DiGeorge Anomaly	Monosomy 22q11 deletion 10p14	Eczematous dermatitis, erythroderma, GVHD	Thymic aplasia/ hypoplasia, hypoparathyroidism, cardiac anomalies, cleft palate
Hyperimmunoglobuli n syndrome	STAT3 mutations	severe dermatitis, abcesses	sinopulmonary infections, fractures, elevated IgE

MISCELLANEOUS

1. BRONZE BABY SYNDROME

It is a rare complication of phototherapy causing grey-brown discoloration of the skin, serum and urine. This is due to photo-oxidation of bilirubin or copper-bound porphyrins or due to accumulation of biliverdin. This discoloration fades spontaneously over time and requires no treatment. Laboratory evidence of conjugated hyperbilirubinemia can be found. [97]

2. PURPURIC PHOTOTHERAPY INDUCED ERUPTION

It is a benign, transient purpura that occurs in infants who receive both blood transfusion and phototherapy. Pathogenesis of this condition is unknown. They present with non blanchable raspberry colored lesions over the photoexposed areas of the body one to four days after receiving phototherapy. Histologically there is RBC extravasation into the dermis without any epidermal damage. It usually undergoes spontaneous resolution. [98]

3. PURPURA FULMINANS (PF)

Hereditary PF is an autosomal recessive disorder which occurs due to a mutation of PROC gene which results in complete deficiency of protein C. It can also be caused by septicemia caused by Group B streptococci, staphylococci, E coli and Enterobacter. It is an acute syndrome characterized by widespread, rapidly progressing necrosis of the skin with disseminated intravascular coagulation (DIC).

The affected neonates manifest clinically with sudden onset of widespread purpura, most commonly affecting the extremities. These lesions enlarge and form

hemorrhagic bullae. the lesions are well defined, erythematous and indurated. Consequently the skin undergoes necrosis resulting in the formation of a black eschar. These skin lesions culminate in DIC and internal bleeding. The thrombosis can affect the skin, CNS, kidney and the eyes.

Diagnosis of hereditary PF can be established by estimation of protein C activity. Indicators of DIC are- increased levels of D-dimer, low fibrinogen, raised fibrinogen degradation products, low platelet count and prothrombin fragment. In acute infectious PF, blood culture has to be undertaken.

PF should be diagnosed and treated at the earliest. It consists of monitoring of vitals, supportive care along with immediate heparinization and infusion of fresh frozen plasma at 15ml/kg every 12 hours. Prostacycline at an initial dose of 5ng/kg/min which is later increased to 15ng/kg/min for 48 hours has been used for the treatment of acute infectious PF. Mortality is 100% for untreated cases of hereditary PF and 50% cases of acute infectious PF. [99]

4. NEONATAL LUPUS ERYTHEMATOSUS (NLE)

The incidence of NLE is approximately 1 in 20,000 live births. Male: female ratio of cutaneous LE is 1:2. Anti-Ro IgG antibodies are found in 98% of affected neonates and around 30% also have anti-La IgG antibodies.

Around half the neonates develop cutaneous lesion, half have congenital heart block and around 10% have both. The skin lesions usually appear during the first few weeks of life but in some they may be seen at birth. Erythematous scaly raised or flat lesions, are most commonly located over the face, scalp and extremities and less often over the trunk. A classic distribution of lesions peri orbitally with ‘owl eye distribution’ is

characteristic of NLE. These lesions are associated with photosensitivity in greater than half of the patients. Crusted lesions have a direct correlation with extra cutaneous features such as cholestasis and hematological abnormalities.

Biopsy from the lesion shows vacuolar degeneration of the basal layer with a sparse superficial dermal mononuclear cell infiltrate. Direct immunofluorescence shows granular deposit of IgG.

Parents should be counseled about the transient nature of these lesions. Strict sun protection must be advised along with mid to low potency topical steroids. [100]

5. NEONATAL PEMPHIGUS

Infants of mothers with pemphigus may also develop the disease due to the transplacental spread of auto antibodies.The skin lesions mimic those of the mother i.e vesicles or erosions over the whole body. Skin biopsy features include intraepithelial blisters with acantholytic cells. Direct and indirect immunofluorescence was positive in majority of the cases. Treatment is mainly symptomatic and conservative; there were no cases which required systematic treatment and in all cases the skin lesions resolved by three weeks of age except one, which resolved by six weeks.[101]

6. PITYRIASIS RUBRA PILARIS (PRP)

Type V PRP- Atypical juvenile variant shows an early age of onset (i.e from birth upto 5 years of age) and a chronic course. The characteristic features include erythema, follicular plugging and keratoderma. Erythroderma is a rare complication. It shows no tendency for spontaneous resolution. It is often resistant to treatment.[102]

7. NEONATAL ERYTHRODERMA

It is a rare and potentially fatal condition characterized by erythema and scaling affecting greater than 90% of the body surface area. Causes include :

- Icthyoses - bullous and non bullous congenital icthyosiform erythroderma, lamellar icthyosis, Netherton syndrome
- Immunodeficiency disorders- Omenn syndrome, sever combined immunodeficiency, graft versus host disease, Wiskott-Aldrich syndrome
- Papulosquamous disorders- pityriasis rubra pilaris, psoriasis
- Others- drug induced erythroderma, diffuse cutaneous mastocytosis

These neonates are at increased risk of hypernatremic dehydration, severe systemic infections, hypoalbuminemia and temperature dysregulation. Laboratory evaluation showing elevated IgE levels gives a diagnostic clue for Netherton and Omenn Syndrome. Histopathology acts as a supplement to aid in the diagnosis . Serum electrolyte and albumin levels should be monitored. Pus and blood culture and sensitivity should be undertaken and appropriate antibiotics instituted. Bland emollients help in maintaining the barrier function of the epidermis. Specific therapy can be started once the underlying cause has been identified. Systemic retinoids have been used in case of icthyosis. SCID and Omenn syndrome may require bone marrow transplantation. [99]

MATERIALS AND METHODS

This non randomized cross-sectional study was conducted between Jan 2016-Dec 2016 by the department of skin & VD, Dr. Prabhakar Kore Charitable Hospital, Belagavi.

Institute ethical committee clearance was obtained prior to the start of the study.

A total of 104 neonates were enrolled for the study on a random basis from the post natal ward and from the skin OPD.

Newborns admitted in the NICU were excluded from the study.

A detailed history was obtained from the mother with regard to the age and mode of onset of the skin lesions, any significant antenatal or postnatal history, birth order of the baby and mode of delivery.

The neonates were examined thoroughly from top to bottom to look for any signs of skin lesions, including a detailed examination of the hair, nails, mucosal sites and general physical examination of the vital signs.

OBSERVATION AND RESULTS

The statistical analysis was performed by STATA 11.2 (College station TX USA) and SPSS software. Chi-square test was used to measure the association between the cutaneous findings (physiological and pathological) with maturity, birth order, gender and birth weight. P value <0.05 was considered as statistically significant.

Total 104 neonates were enrolled in this study.

A) GENDER WISE DISTRIBUTION OF NEONATES

49 (47%) neonates were male and 55 (53%) were female. (Table.1, Figure.1)

Gender	Number of Cases	Percentage
Male	49	47%
Female	55	53%
Total	104	

TABLE.1

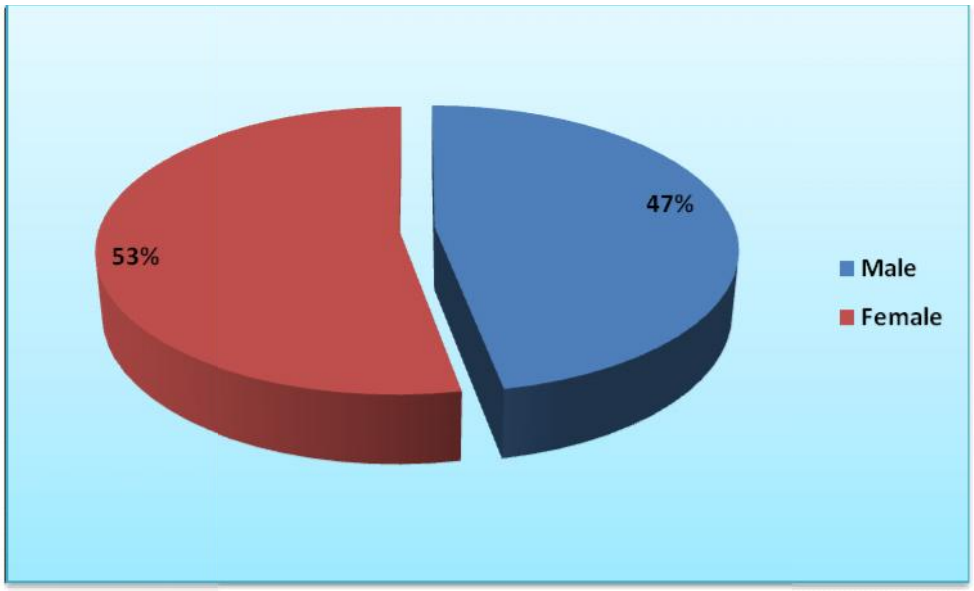

FIGURE.1

B) DISTRIBUTION OF NEONATES ACCORDING TO THE MODE OF DELIVERY

51 (49%) neonates were born through normal vaginal delivery and 53 (51%) through caesarian section. (Table.2, Figure.2)

Mode of delivery	Number of Cases	Percentage
Caesarean	53	51%
Normal	51	49%
Total	104	

TABLE.2

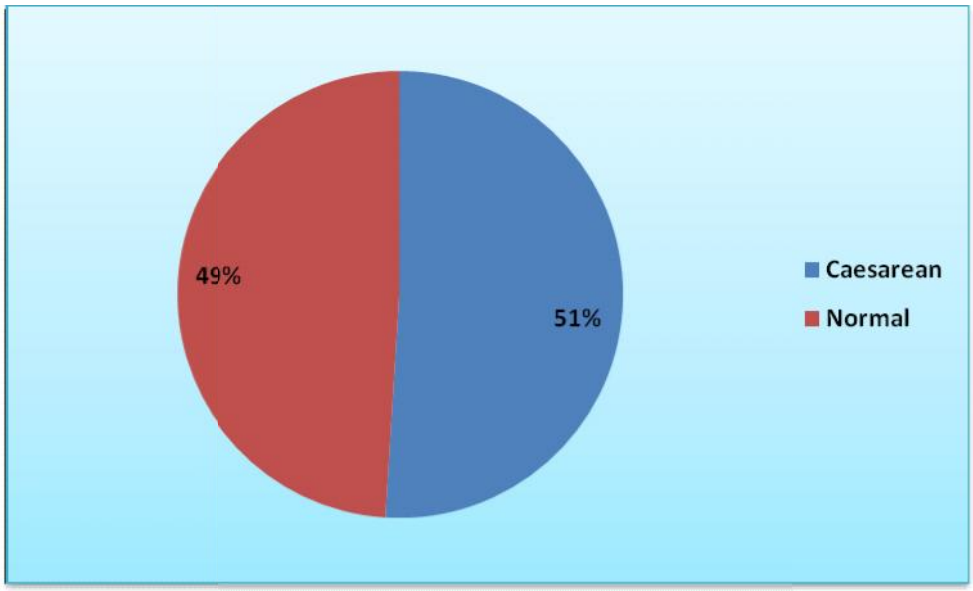

FIGURE.2

C) DISTRIBUTION OF NEONATES ACCORDING TO MATURITY

3 (2.88%) neonates were born preterm, 5 (4.81%) post term and 96 (92.31%) neonates were born at term. (Table.3, Figure.3)

Maturity of baby	Number of Cases	Percentage
Pre term	3	2.88%
Post Term	5	4.81%
Term	96	92.31%
Total	104	

TABLE.3

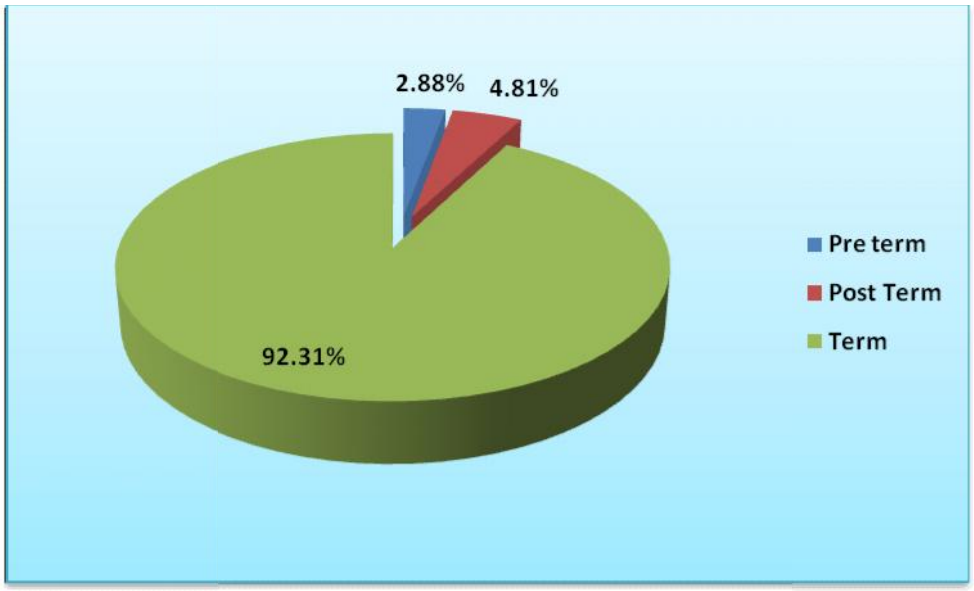

FIGURE.3

D) DISTRIBUTION OF NEONATES ACCORDING TO BIRTH WEIGHT

18 (15%) neonates were underweight i.e <2.5kgs. (Table.4, Figure.4)

Birth weight (kgs)	Number of Cases	Percentage
<2.5	16	15%
>2.5	88	85%
Total	104	

TABLE.4

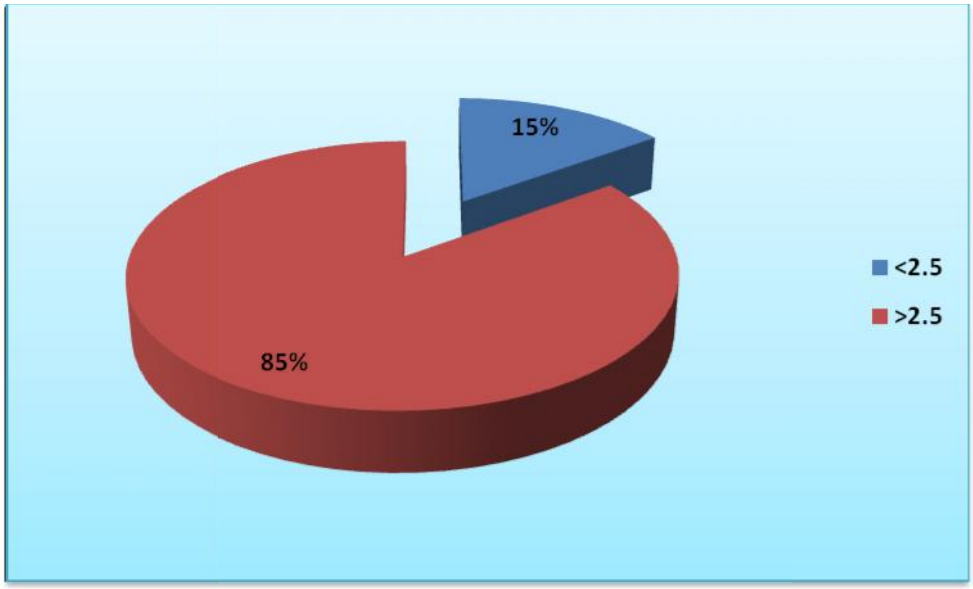

FIGURE.4

E) DISTRIBUTION OF NEONATES ACCORDING TO BIRTH ORDER

46 (44.23%) neonates were first born and out of the remaining 58 (55.77%) : 42 (40.38%) were second born, 13 (12.5%) were third born and 3 (2.88%) were fourth born. (Table.5, Figure.5).

Birth Order	Number of Cases	Percentage
1	46	44.23%
2	42	40.38%
3	13	12.50%
4	3	2.88%
Total	104	

TABLE.5

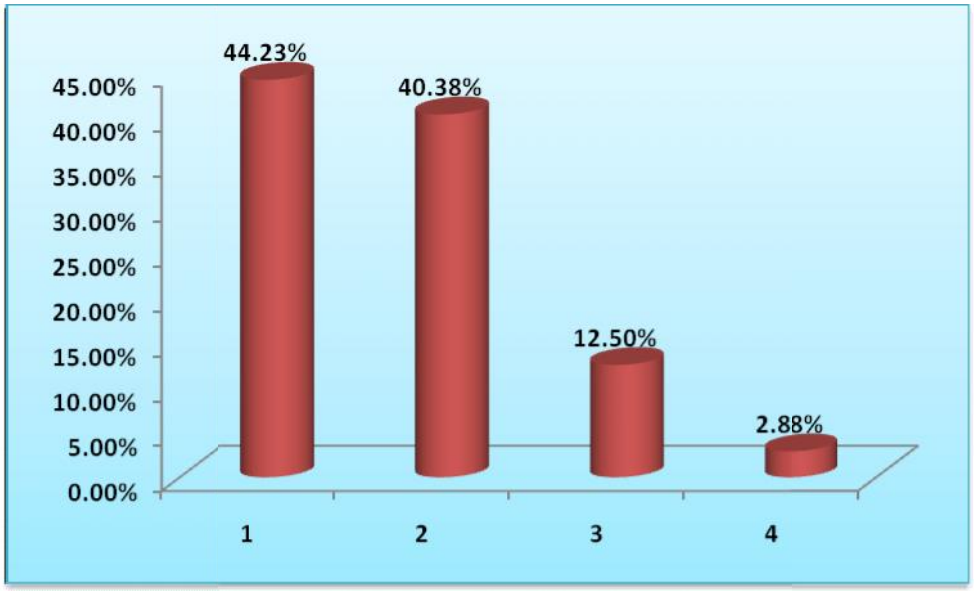

FIGURE.5

F) DISTRIBUTION OF MOTHERS ACCORDING TO AGE

102 (98%) mothers were ≤ 30 years of age and 2 (2%) were >30 years of age. (Table.6, Figure.6)

46 (44.23%) mothers were primigravida and 58 (55.77%) were multigravida.

Age of mother (yrs)	Number of Cases	Percentage
≤30	102	98%
>30	2	2%
Total	104	

TABLE.6

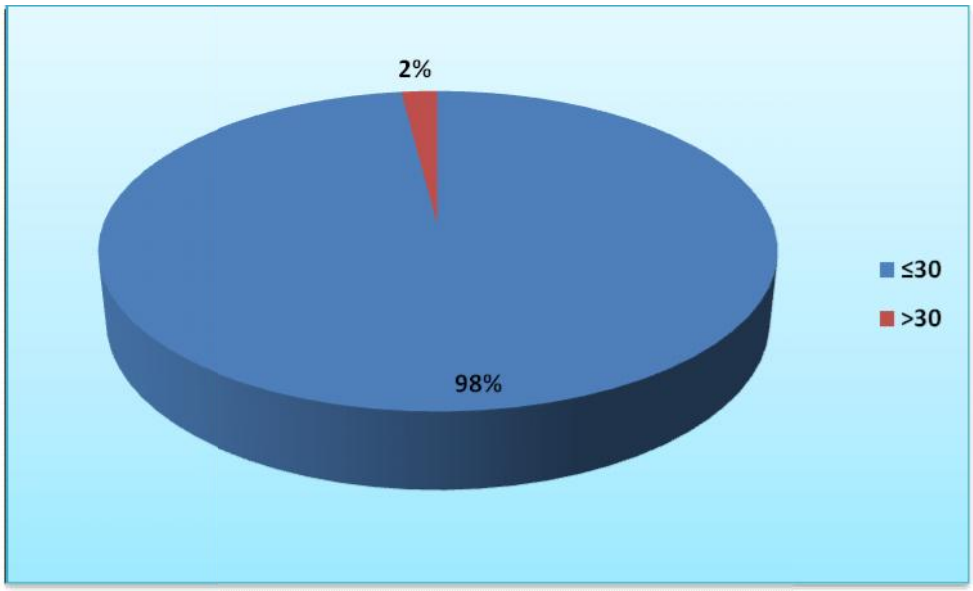

FIGURE.6

G) DISTRIBUTION OF NEONATES ACCORDING TO THE SKIN CHANGES OBSERVED

Out of 104 neonates, 99 (95%) had physiological changes and 5 (5%) had pathological changes. (Table.7, Figure.7)

Skin changes	Number of cases	Percentage
Physiological	99	95%
Pathological	5	5%

TABLE.7

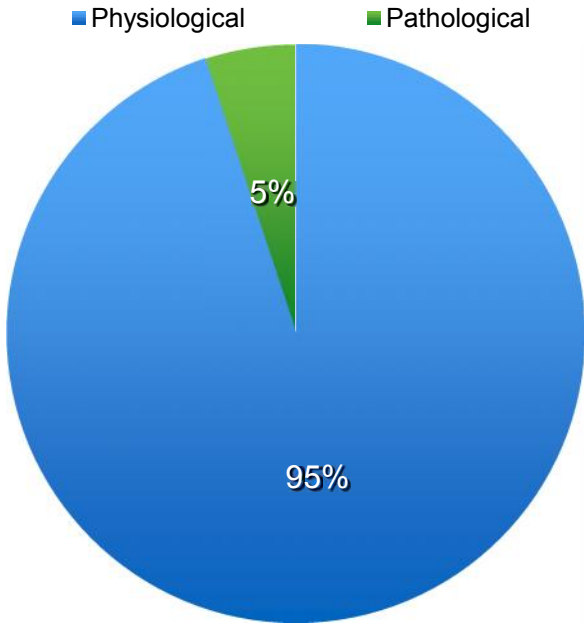

FIGURE.7

H) PHYSIOLOGICAL CHANGES

The most common physiological change observed was mongolian spot which was found in 34 (33%) neonates.

Out of these 34 neonates, 24 (23.08%) neonates had only mongolian spot, 4 (3.85%) also had erythema toxicum neonatorum, 3 (2.88%) also had desquamation of skin, 1 (0.96%) had desquamation of skin along with vernix caseosa, 1 (0.96%) had erythema toxicum neonatorum and milia, and 1 (0.96%) had hypertrichosis lanuginosa .

The second common physiological change observed was erythema toxicum neonatorum, which was seen in 27 (26%) neonates. Out of these 27, 22 (21.15%) neonates had only ETN, 1 also had desquamation of skin, 4 also had mongolian spot .

The third common physiological change observed was physiological desquamation of skin which was seen in 21(20%) neonates. Out of these 21 neonates, 14 had only desquamation, 2 neonates also had milia, 2 also had ETN 3 also had mongolian spot, and 1 also had mongolian spot with vernix caseosa. (Table.8, Figure.8A and Figure.8B)

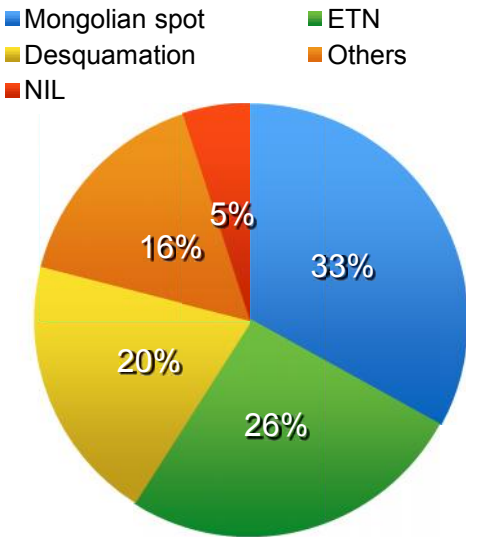

Figure.8A

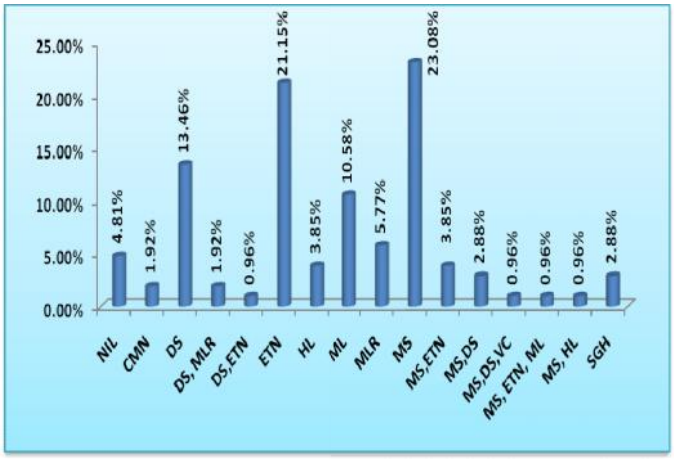

Figure.8B

Physiological changes	Number of Cases	Percentage
NIL	5	4.81%
Congenital melanocytiv nevi	2	1.92%
Desquammation of skin only	14	13.46%
Desquammation of skin,miliaria	2	1.92%
Desquammation of skin,erythema toxicum neonatorum	1	0.96%
Erythema toxicum neonatorum only	22	21.15%
Hypertrichosis lanuginosa	4	3.85%
Milia	10	9.62%
Miliaria only	6	5.77%
Mongolian spot only	24	23.08%
Mongolian spot, erythema toxicum neonatorum	4	3.85%
Mongolian spot, desquammation of skin	3	2.88%
Mongolian spot, desquammation of skin, vernix caseosa	1	0.96%
Mongolian spot, Erythema toxicum neonatorum, Miliaria	1	0.96%
Mongolian spot, hypertrichosis lanuginosa	1	0.96%
Sebaceous gland hyperplasia	3	2.88%
Total	104	

TABLE.8

I) PATHOLOGICAL CHANGES

99 (95.19%) neonates had no pathological changes. One (0.96%) neonate had bullous impetigo, One had birth trauma, one collodion baby, one had furunculosis and one neonate had intertrigo. (Table.9A, Figure.9A, Figure.9B)

	Number of Cases	Percentage
Nil	99	95.19%
Bullous impetigo	1	0.96%
Birth trauma	1	0.96%
Collodion baby	1	0.96%
Furunculosis	1	0.96%
Intertrigo	1	0.96%
Total	104	

TABLE.9A

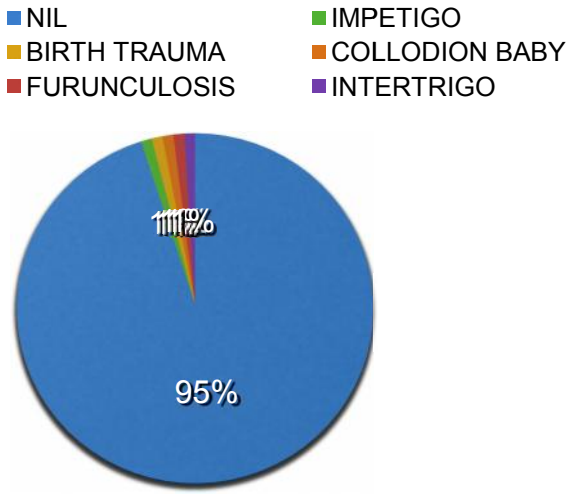

FIGURE.9B

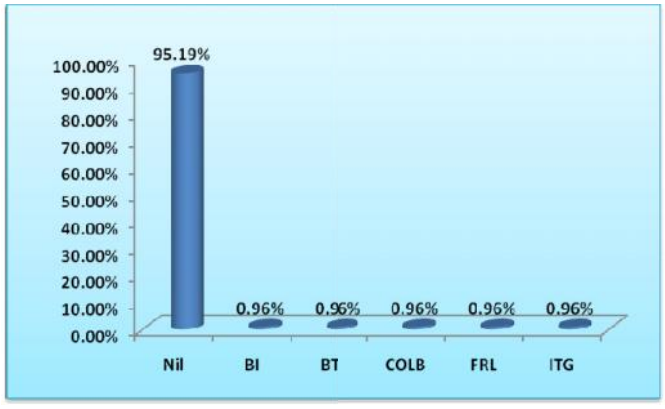

FIGURE.9A

J) SUMMARY OF SKIN CHANGES OBSERVED- BOTH PHYSIOLOGICAL AND PATHOLOGICAL

SKIN CHANGES	NUMBER OF CASES	PERCENTAGE
MONGOLIAN SPOT	34	32.69%
ERYTHEMA TOXICUM NEONATORUM	27	25.96%
PHYSIOLOGICAL DESQUAMATION	21	20.19%
MILIA	10	9.62%
MILIARIA	9	8.65%
HYPERTRICHOSIS LANUGINOSA	5	4.81%
SEBACEOUS GLAND HYPERPLASIA	3	2.89%
CONGENITAL MELANOCYTIC NEVI	2	1.92%
VERNIX CASEOSA	1	0.96%
BULLOUS IMPETIGO	1	0.96%
BIRTH TRAUMA	1	0.96%
COLLODION BABY	1	0.96%
FURUNCULOSIS	1	0.96%
INTERTRIGO	1	0.96%
MORE THAN ONE DERMATOSIS (OVERLAP)	13	12.5%

TABLE.9B

K) DISTRIBUTION OF SKIN CHANGES ACCORDING TO THE AGE OF THE NEONATE

Neonatal period can be divided into early and late neonatal period. Early period consists of the first seven days of extrauterine life, whereas late neonatal period consists of 8-28 days of extrauterine life. The skin changes observed in neonates can vary considerably in these two age groups.

EARLY NEONATE (<7 DAYS)	LATE NEONATE (8-28 DAYS)	TOTAL
88 (85%)	16 (15%)	104

TABLE.10

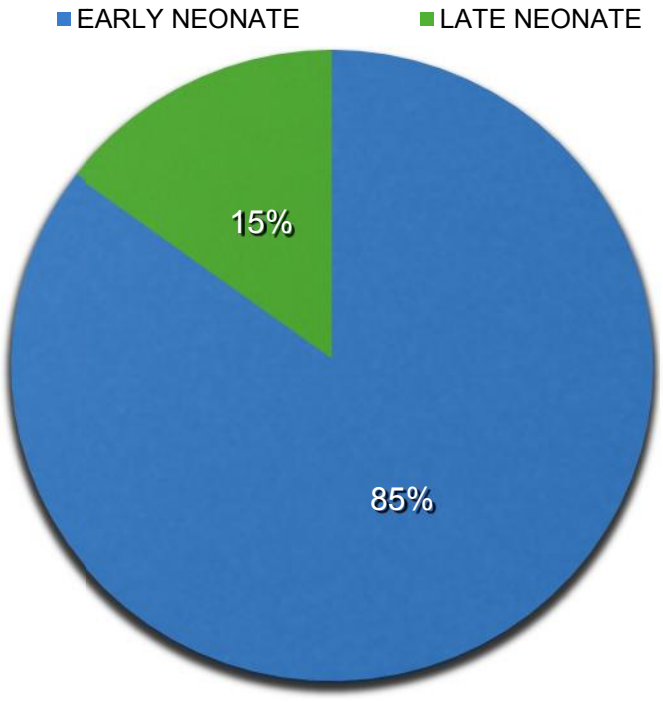

FIGURE.10

1. CO-RELATION OF MONGOLIAN SPOT WITH VARIOUS PARAMETERS

Figure.11A and Table.11A shows the co-relation of mongolian spot with birth weight of the neonate. 9 (56%) low birth weight neonates and 25 (28%) normal birth weight neonates had mongolian spot. **The P-value was found to be 0.029 which is statistically significant.**

	MONGOLIAN SPOT			
Birth weight (kgs)	Present	Absent	Total	P -Value
<2.50	9 (56%)	7 (44%)	16	0.029
>2.50	25 (28%)	63 (72%)	88	
Total	34	70	104	

TABLE.11A

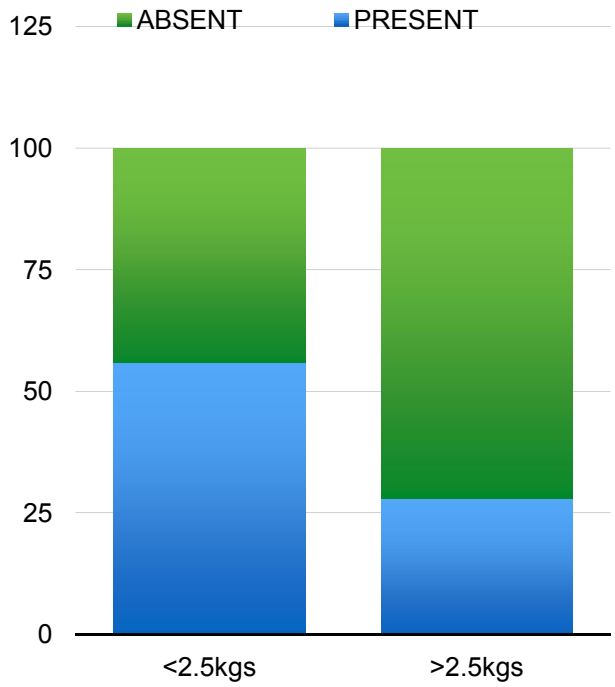

FIGURE.11A

Figure.11B and Table.11B shows the co-relation of Mongolian spot with maturity of the neonate. Mongolian spot was found in 1 (33%) preterm neonate, 1 (20%) post term neonate and 32 (33%) term neonates. The P-value was found to be 0.825 which is not statistically significant.

	MONGOLIAN SPOT			
Maturity	Present	Absent	Total	P -Value
Pre term	1 (33%)	2 (67%)	3	0.825
Post term	1 (20%)	4 (80%)	5	
Term	32 (33%)	64 (67%)	96	
Total	34	70	104	

TABLE.11B

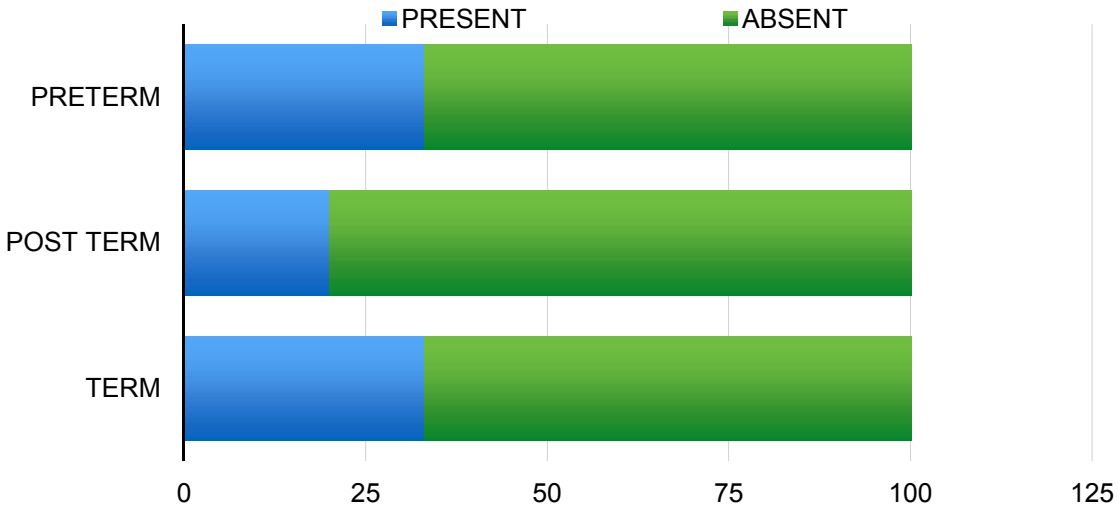

FIGURE.11B

Table.11C and Figure.11C shows the co-relation of Mongolian spot with gender. 18 (37%) male neonates and 16 (29%) female neonates had mongolian spot. The P-value was found to be 0.407 which is not statistically significant.

	MONGOLIAN SPOT			
Gender	Present	Absent	Total	P -Value
Male	18 (37%)	31 (63%)	49	0.407
Female	16 (29%)	39 (71%)	55	
Total	34	70	104	

TABLE.11C

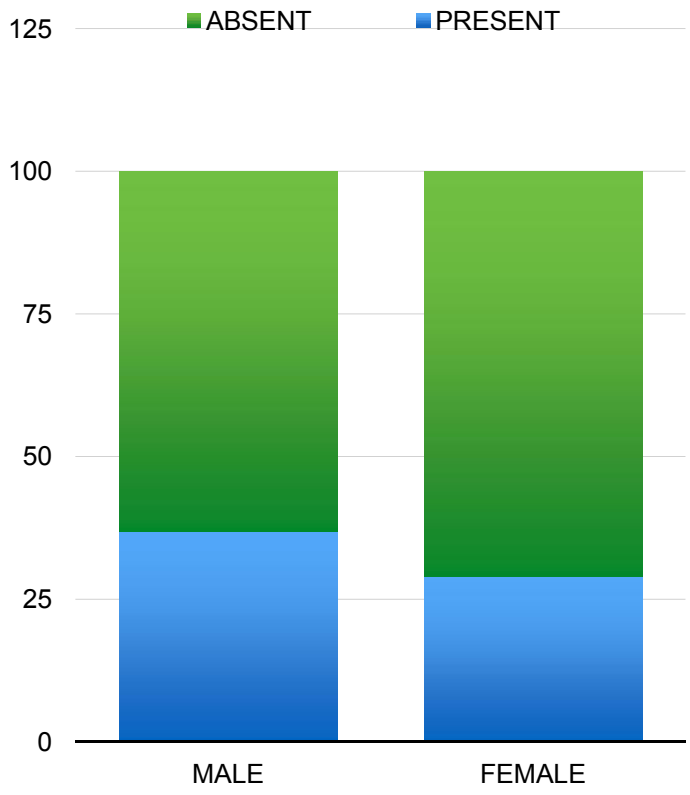

FIGURE.11C

Table.11D and Figure.11D shows the co-relation of Mongolian spot with birth order. Out of 34 neonates with Mongolian spot, 10 were first born, 16 were second born, 7 were third born and 1 was fourth born. The P-value was 0.127 which is statistically insignificant.

	MONGOLIAN SPOT			
Birth order	Present	Absent	Total	P -Value
1	10 (22%)	36 (78%)	46	0.127
2	16 (38%)	26 (62%)	42	
3	7 (54%)	6 (46%)	13	
4	1 (33%)	2 (67%)	3	
Total	34	70	104	

TABLE.11D

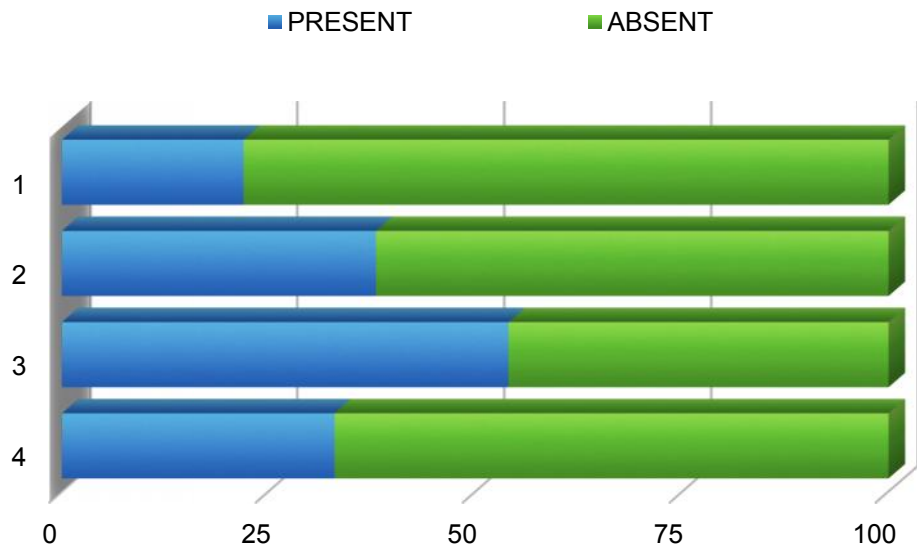

FIGURE.11D

2. CO-RELATION OF ERYTHEMA TOXICUM NEONATORUM WITH VARIOUS PARAMETERS

Table.12A and Figure.12A shows the co-relation of erythema toxicum neonatorum with birth weight. All 27 neonates with Erythema toxicum neonatorum had birth weight >2.5kgs and **this association was found to be statistically significant**.

	ERYTHEMA TOXICUM NEONATORUM			
Birth weight (kgs)	Present	Absent	Total	P -Value
<2.50	0	16 (100%)	16	0.010
>2.50	27 (31%)	61 (69%)	88	
Total	27	77	104	

TABLE.12A

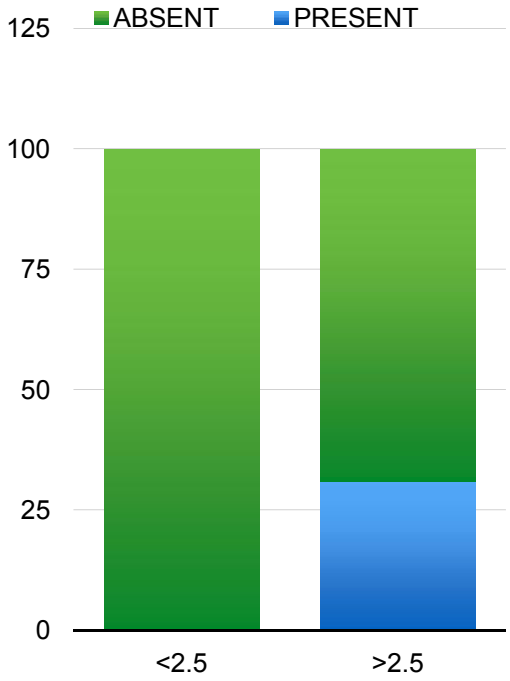

FIGURE.12A

Table.12B and Figure.12B shows the association between erythema toxicum neonatorum and maturity of the baby. Erythema toxicum neonatorum was found only in term neonates i.e in 27 (28%) out of 96 term neonates. P-value was not found to be statistically significant.

	ERYTHEMA TOXICUM NEONATORUM			
Maturity	Present	Absent	Total	P -Value
Pre term	0	3 (100%)	3	0.219
Post term	0	5 (100%)	5	
Term	27 (28%)	69 (72%)	96	
Total	27	77	104	

TABLE.12B

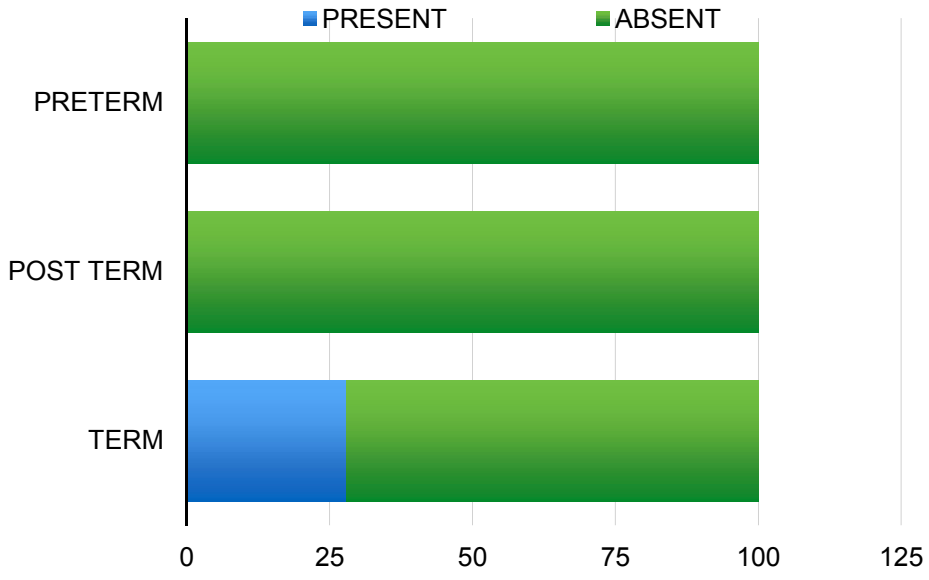

FIGURE.12B

Table.12C and Figure.12C shows the association between erythema toxicum neonatorum and gender. It was found in 27 neonates out of which 18 were males and 9 were females. The P-value was calculated and was found to be **statistically significant**.

	ERYTHEMA TOXICUM NEONATORUM			
Gender	Present	Absent	Total	P -Value
Male	18 (37%)	31 (63%)	49	0.018
Female	9 (16%)	46 (84%)	55	
Total	27	77	104	

TABLE. 12C

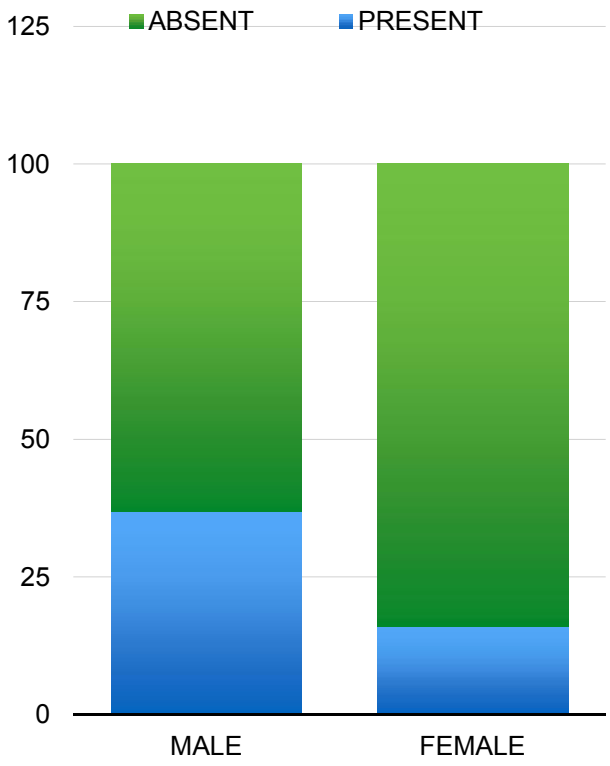

FIGURE.12C

Table.12D and Figure.12D shows the co-relation of erythema toxicum neonatorum with birth order of the neonate. Out of 27 neonates with ETN, 13 were first born and 14 were second born. This association was found to be statistically insignificant.

	ERYTHEMA TOXICUM NEONATORUM			
Birth order	Present	Absent	Total	P -Value
1	13 (28%)	33 (72%)	46	0.074
2	14 (33%)	28 (67%)	42	
3	0	13 (100%)	13	
4	0	3 (100%)	3	
Total	27	77	104	

TABLE.12D

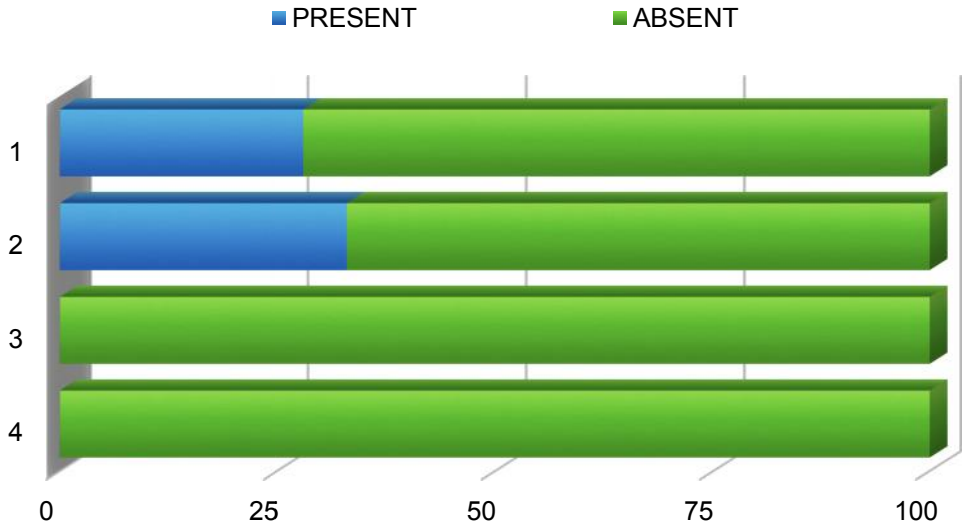

FIGURE.12D

3. CO-RELATION OF PHYSIOLOGICAL DESQUAMATION OF SKIN WITH VARIOUS PARAMETERS

Table.13A and Figure.13A shows the co-relation between desquamation of skin and birth weight of the neonate. It was present in 19% of neonates with birth weight <2.5kgs and in 20% of neonates with birth weight >2.5kgs. The P-value was found to be 0.876 which is not statistically significant.

	DESQUAMATION			
Birth weight (kgs)	Present	Absent	Total	P -Value
<2.50	3 (19%)	13 (81%)	16	0.876
>2.50	18 (20%)	7 0 (80%)	88	
Total	21	83	104	

TABLE.13A

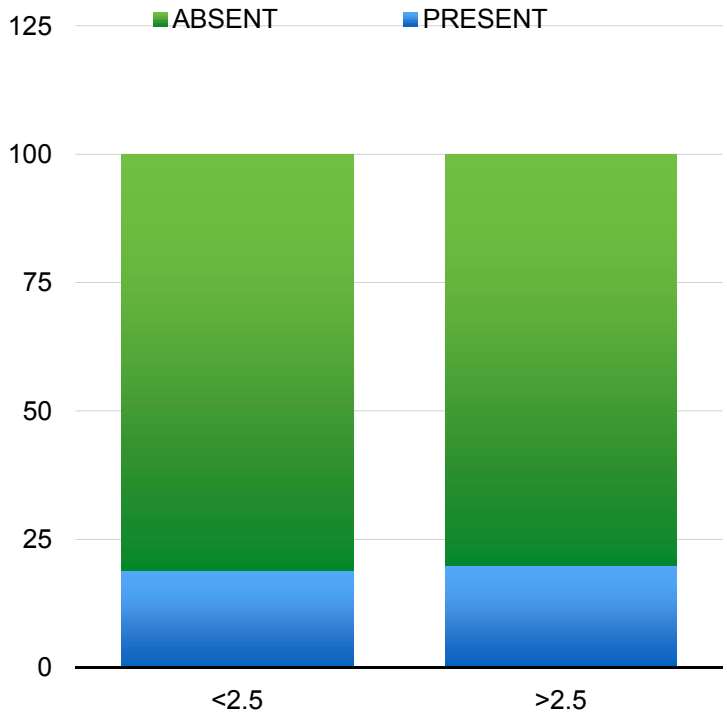

FIGURE.13A

Table.13B and Figure.13B shows the co-relation between Desquamation and maturity of the neonate. Out of 21 neonates with desquamation, 19 were term neonates and 2 were post term neonates. The co-relation was not statistically significant.

	DESQUAMATION			
Maturity	Present	Absent	Total	P -Value
Pre term	0	3 (100%)	3	0.370
Post term	2 (40%)	3 (60%)	5	
Term	19 (20%)	77 (80%)	96	
Total	21	83	104	

TABLE.13B

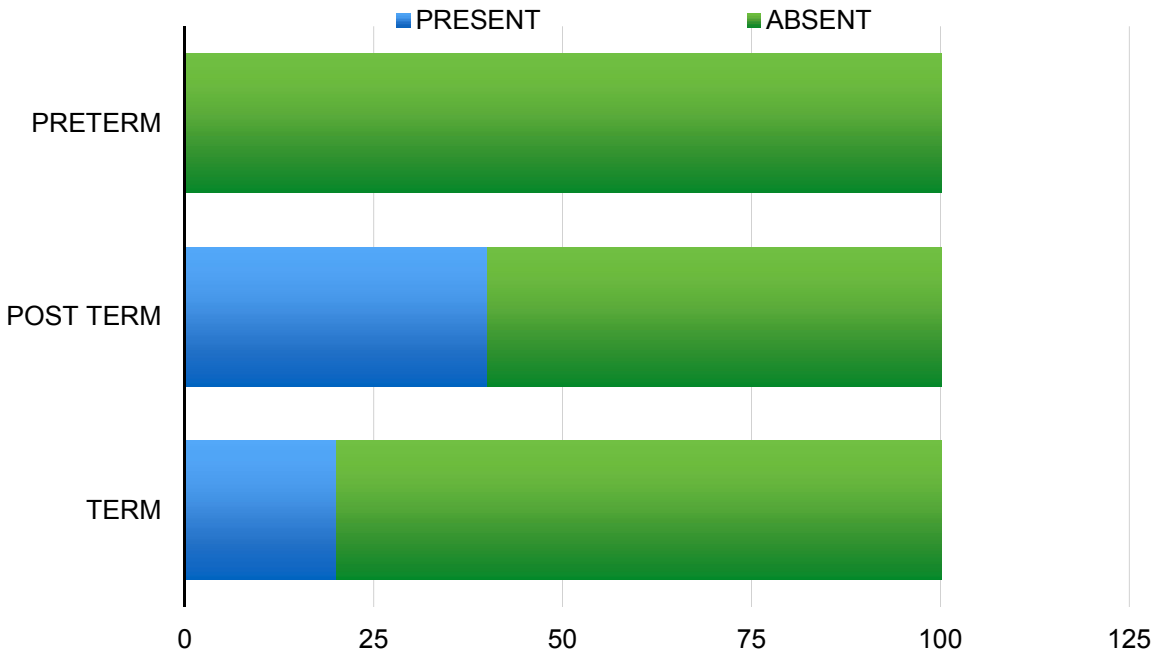

FIGURE.13B

Table.13C and Figure.13C shows the co-relation between desquamation of skin and gender of the neonate. Out of 21 neonates who had desquamation of skin, 5 were males and 16 were females. The co-relation was not found to be statistically significant.

	DESQUAMATION			
Gender	Present	Absent	Total	P -Value
Male	6 (12%)	43 (88%)	49	0.057
Female	15 (27%)	40 (73%)	55	
Total	21	83	104	

TABLE.13C

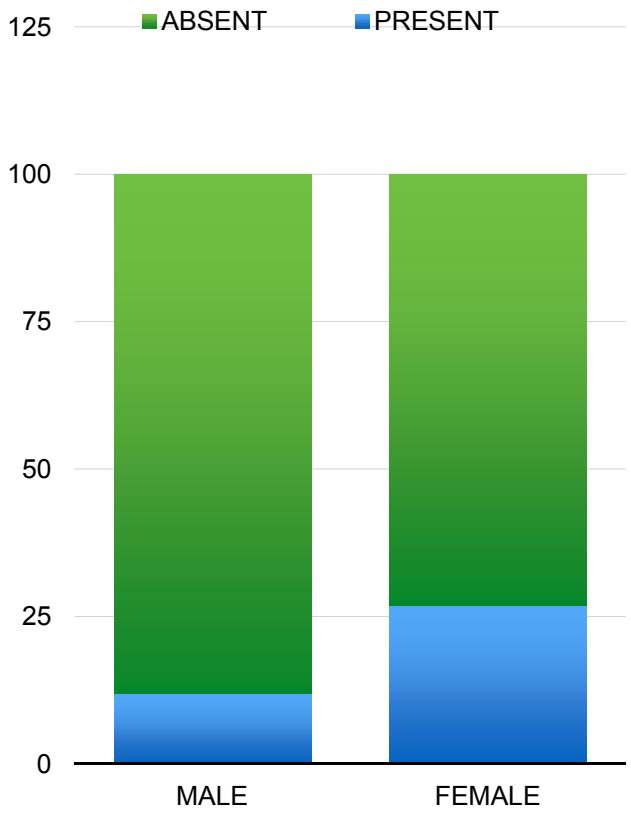

FIGURE.13C

Table.13D and Figure.13D shows the co-relation between desquamation of skin and birth order of the neonate. Out of 21 neonates with desquamation, 10 were first born, 5 were second born, 5 were third born and one was fourth born. The P-value was found to be 0.181 i.e this co-relation was not statistically significant.

	DESQUAMATION			
Birth order	Present	Absent	Total	P -Value
1	10 (22%)	36 (78%)	46	0.181
2	5 (12%)	37 (88%)	42	
3	5 (38%)	8 (62%)	13	
4	1 (33%)	2 (67%)	3	
Total	21	83	104	

TABLE.13D

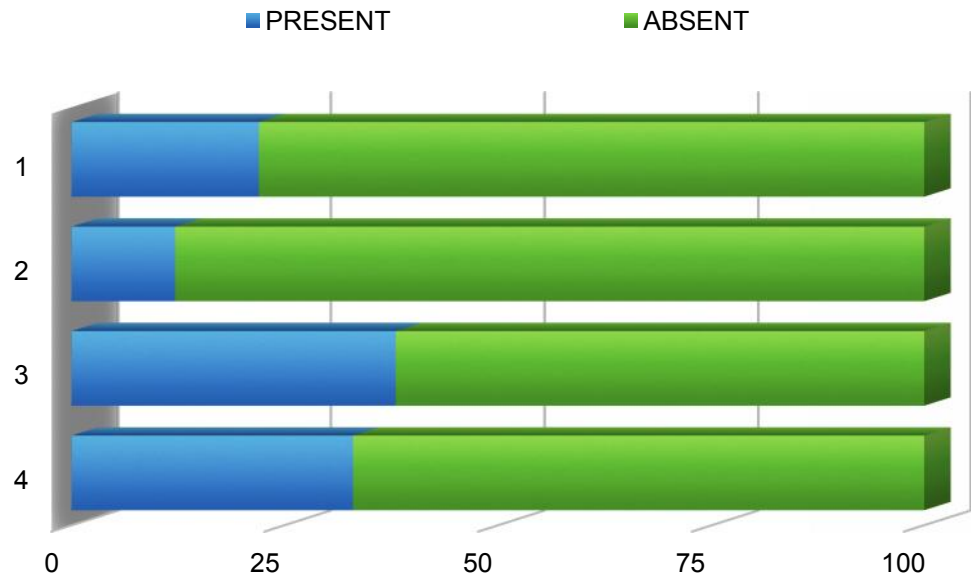

FIGURE.13D

4. CO-RELATION OF MILIA WITH VARIOUS PARAMETERS

Table.14A and Figure.14A shows the co-relation between milia and birth weight. Out of 10 neonates with milia, 2 weighed <2.5kgs and 8 weighed >2.5kgs. The P value was 0.597 which is not statistically significant.

	MILIA			
Birth weight (kgs)	Present	Absent	Total	P -Value
<2.5	2 (13%)	13 (87%)	15	0.597
>2.5kgs	8 (9%)	81 (91%)	89	
Total	10	94	104	

TABLE.14A

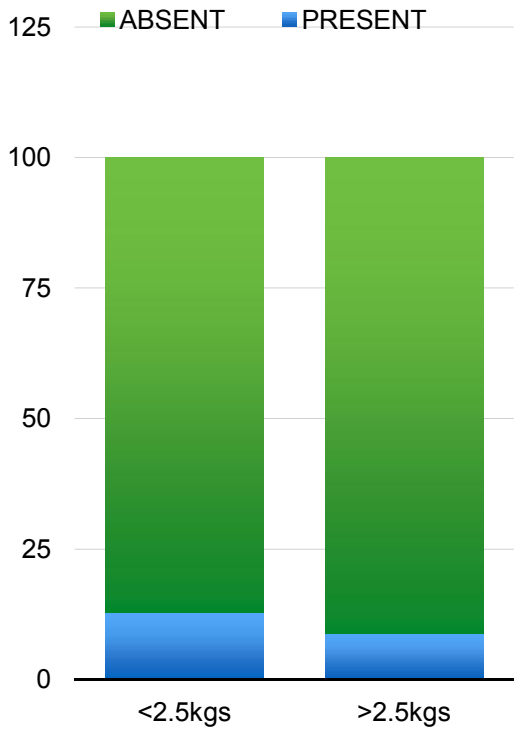

FIGURE.14A

Figure.14B and Table.14B shows the co-relation between milia and maturity of the neonate. Out of ten neonates with milia, two were preterm, two were post term and six were term neonates. The P value was 0.744 which was not statistically significant.

	MILIA			
Maturity	Present	Absent	Total	P -Value
Preterm	2 (67%)	1(33%)	3	0.744
Term	6(6%)	89(94%)	95	
Post Term	2(33%)	4(67%)	6	
Total	10	94	104	

TABLE.14B

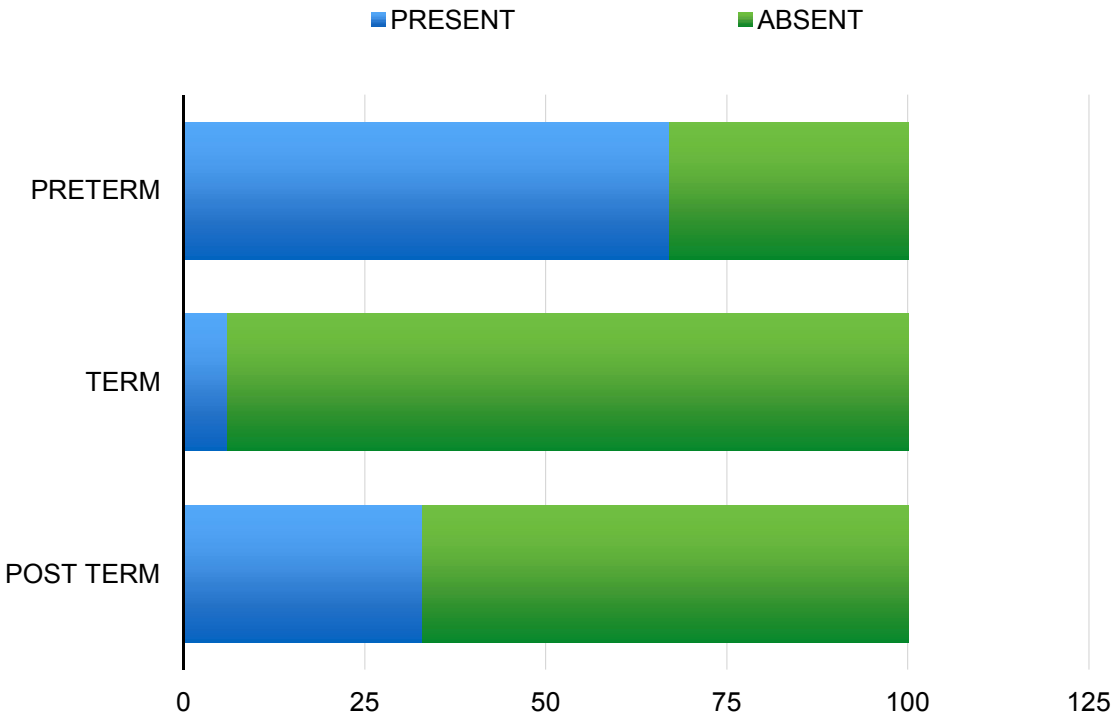

FIGURE.14B

Table.14C and Figure.14C shows the co-relation between milia and gender. Out of ten neonates with milia, six were males and three were females. The P value was found to be 0.391 which is not statistically significant.

	MILIA			
Gender	Present	Absent	Total	P -Value
Male	6 (12%)	43 (88%)	49	0.391
Female	4 (7%)	51 (93%)	55	
Total	10	94	104	

TABLE.14C

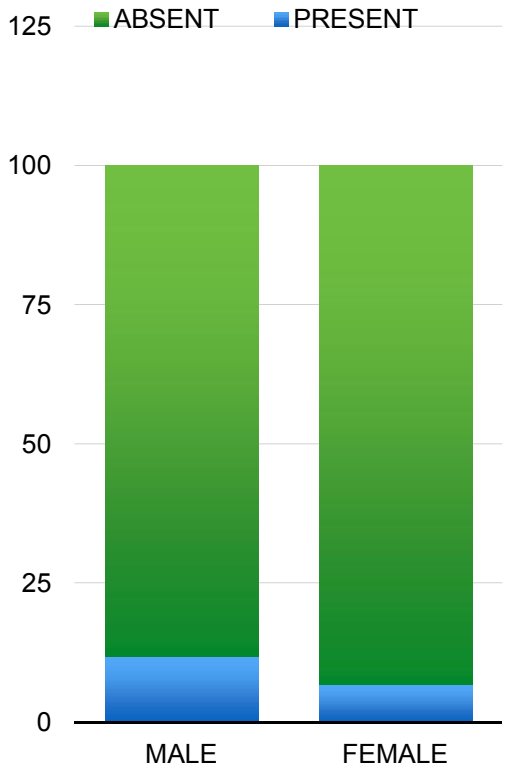

FIGURE.14C

Table.14D and Figure.14D shows the co-relation between milia and birth order. P value was found to be 0.066 which is not statistically significant.

	MILIA			
Birth Order	Present	Absent	Total	P -Value
1	2 (4%)	44 (96%)	46	0.066
2	7 (17%)	35 (83%)	42	
3	0 (0%)	13 (100%)	13	
4	1 (33%)	2 (67%)	3	
Total	10	94	104	

TABLE.14D

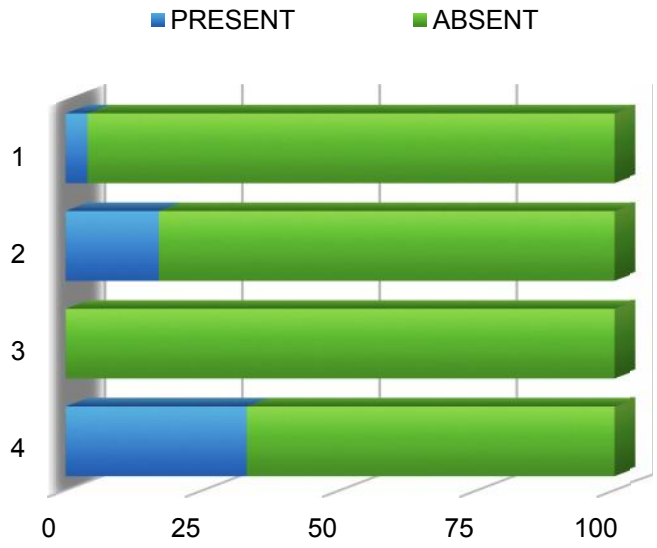

FIGURE.14D

5. CO-RELATION OF MILIARIA WITH VARIOUS PARAMETERS

Table.15A and Figure.15A shows the co-relation between miliaria and birth weight of the neonate. All nine neonates had a birth weight of more than 2.5kgs. The P-value was found to be 0.198 which is not statistically significant.

	MILIARIA			
Birth weight (kgs)	Present	Absent	Total	P -Value
< 2.5	0 (0%)	15 (100%)	15	0.198
> 2.5	9 (10%)	80 (90%)	89	
Total	9	95	104	

TABLE.15A

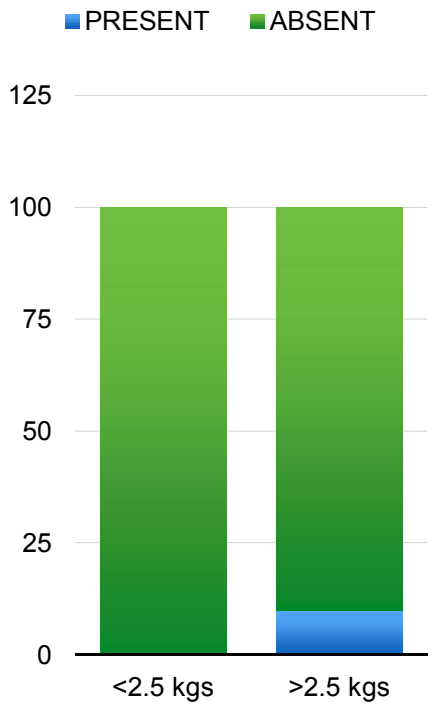

FIGURE.15A

Table.15B and Figure.15B shows the co-relation between miliaria and maturity of the neonate. All nine neonates with miliaria were born at term. The P-value was found to be 0.627 which is not statistically significant.

	MILIARIA			
Maturity	Present	Absent	Total	P -Value
Preterm	0 (0%)	3 (100%)	3	0.627
Term	9 (9.5%)	86 (91.5%)	95	
Post Term	0 (0%)	6 (100%)	6	
Total	9	95	104	

TABLE.15B

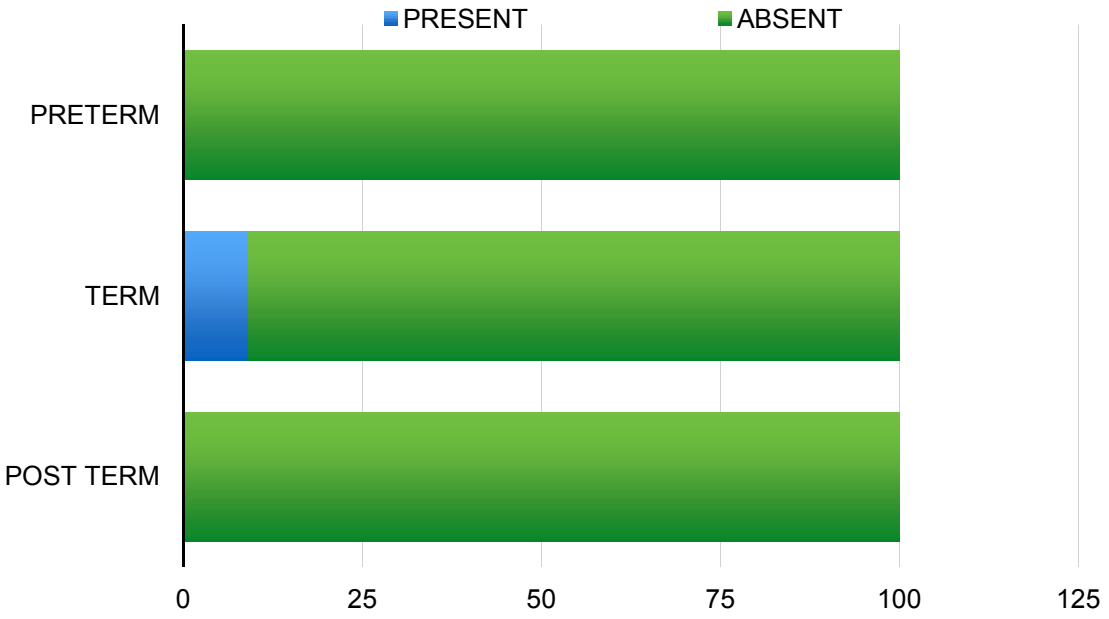

FIGURE.15B

Table.15C and Figure.15C shows the co-relation between miliaria and gender of the neonate. Out of nine neonates with miliaria, four were males and five were females. The P value was found to be 0.867 which is not statistically significant.

	MILIARIA			
Gender	Present	Absent	Total	P -Value
Male	4 (8%)	45 (92%)	49	0.867
Female	5 (9%)	50 (91%)	55	
Total	9	95	104	

TABLE.15C

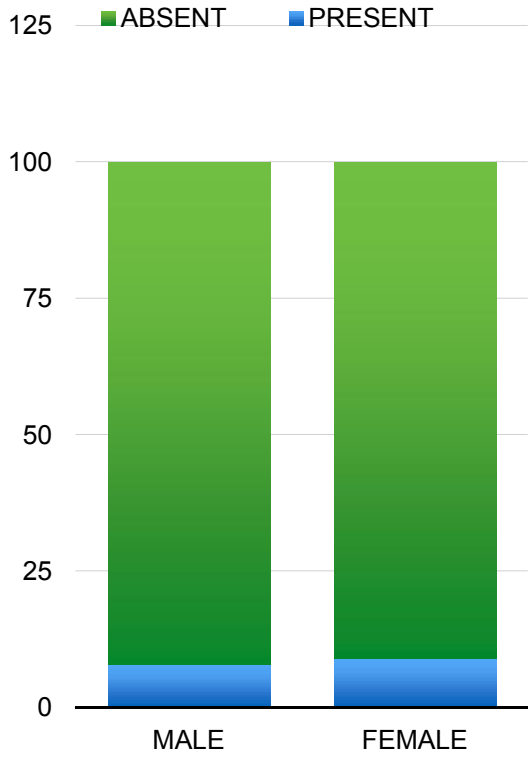

FIGURE.15C

Table.15D and Figure.15D shows the co-relation between miliaria and birth order of the neonate. No statistically significant association was found.

	MILIARIA			
Birth Order	Present	Absent	Total	P -Value
1	4 (9%)	42 (91%)	46	0.76
2	3 (7%)	39 (93%)	42	
3	2 (15%)	11 (85%)	13	
4	0 (0%)	3 (100%)	3	
Total	9	95	104	

TABLE.15D

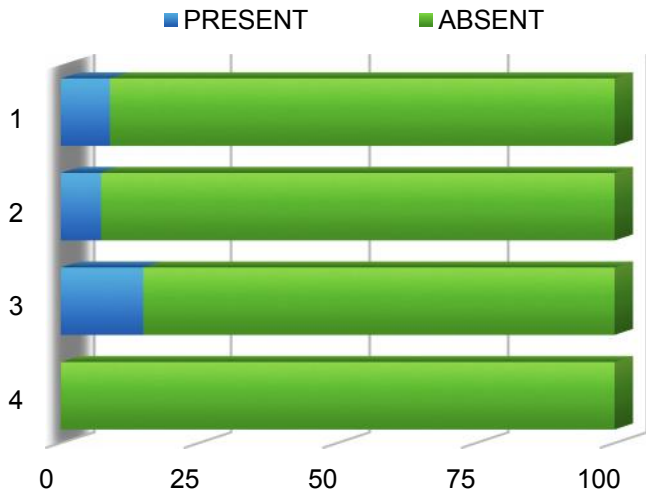

FIGURE.15D

6. CO-RELATION OF SEBACEOUS GLAND HYPERPLASIA WITH VARIOUS PARAMETERS

Table.16A and Figure.16A shows the co-relation between sebaceous gland hyperplasia and birth weight of the neonate. All three neonates with sebaceous gland hyperplasia weighed more than 2.5kgs at birth. P value was found to be 0.471 which is not statistically significant.

	SEBACEOUS GLAND HYPERPLASIA			
Birth weight (kgs)	Present	Absent	Total	P -Value
<2.5	0 (0%)	15 (100%)	15	0.471
>2.5	3 (3.3%)	86 (96.7%)	89	
Total	3	101	104	

TABLE.16A

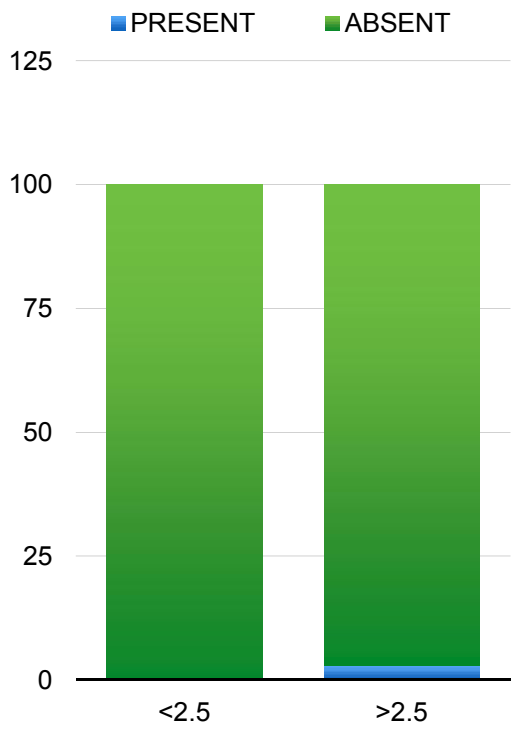

FIGURE.16A

Table.16B and Figure.16B shows the co-relation between sebaceous gland hyperplasia and maturity of the neonate. All the three neonates with sebaceous gland hyperplasia were found to be term neonates. However the P value was not found to be statistically significant.

	SEBACEOUS GLAND HYPERPLASIA			
Maturity	Present	Absent	Total	P -Value
Preterm	0 (0%)	3 (100%)	3	0.864
Term	3 (3%)	92 (97%)	95	
Post Term	0 (0%)	6 (100%)	6	
Total	3	101	104	

TABLE.16B

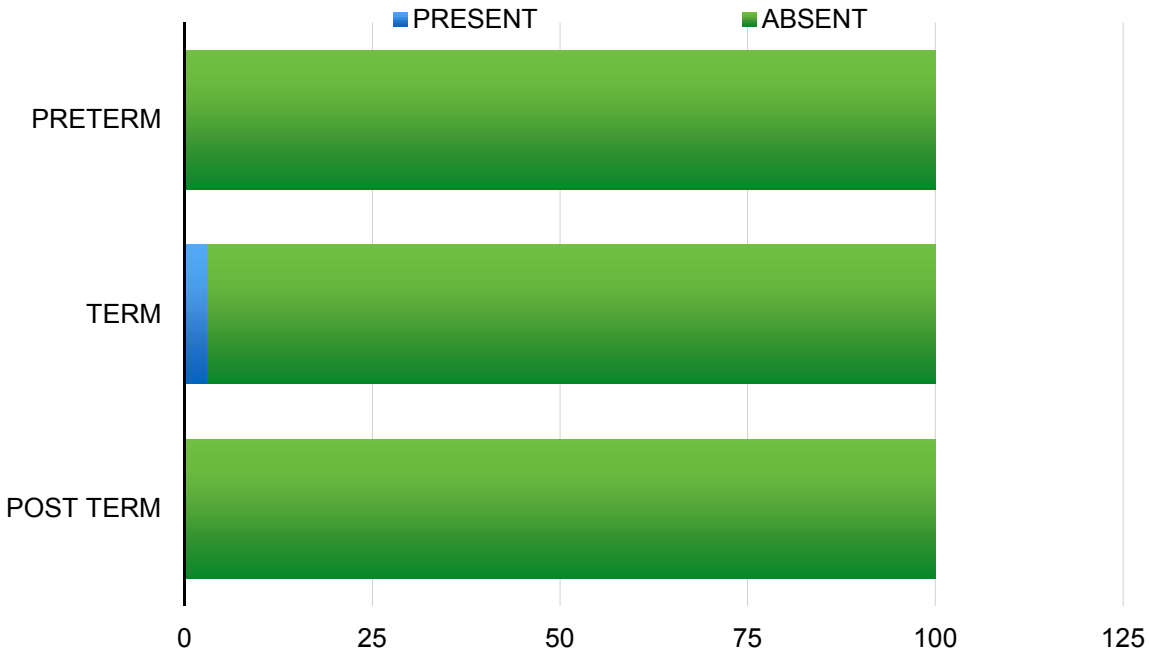

FIGURE.16B

Table.16C and Figure.16C shows the co-relation between sebaceous gland hyperplasia and gender of the neonate. Out of the three neonates with sebaceous gland hyperplasia, one was male and two were female. The P value was found to be 0.627 which is not statistically significant.

	SEBACEOUS GLAND HYPERPLASIA			
Gender	Present	Absent	Total	P -Value
Male	1 (2%)	48 (98%)	49	0.627
Female	2 (4%)	53 (96%)	55	
Total	3	101	104	

TABLE.16C

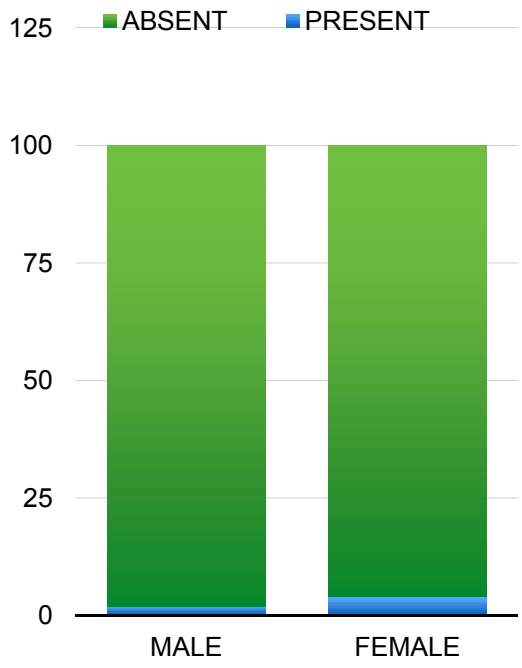

FIGURE.16C

Table.16D and Figure.16D shows the co-relation between sebaceous gland hyperplasia and birth order. P value was found to be 0.733 which is not statistically significant.

	SEBACEOUS GLAND HYPERPLASIA			
Birth Order	Present	Absent	Total	P -Value
1	1 (2%)	45 (98%)	46	0.733
2	1 (2%)	41 (98%)	42	
3	1 (7.7%)	12 (92.3%)	13	
4	0 (0%)	3 (100%)	3	
Total	3	101	104	

TABLE.16D

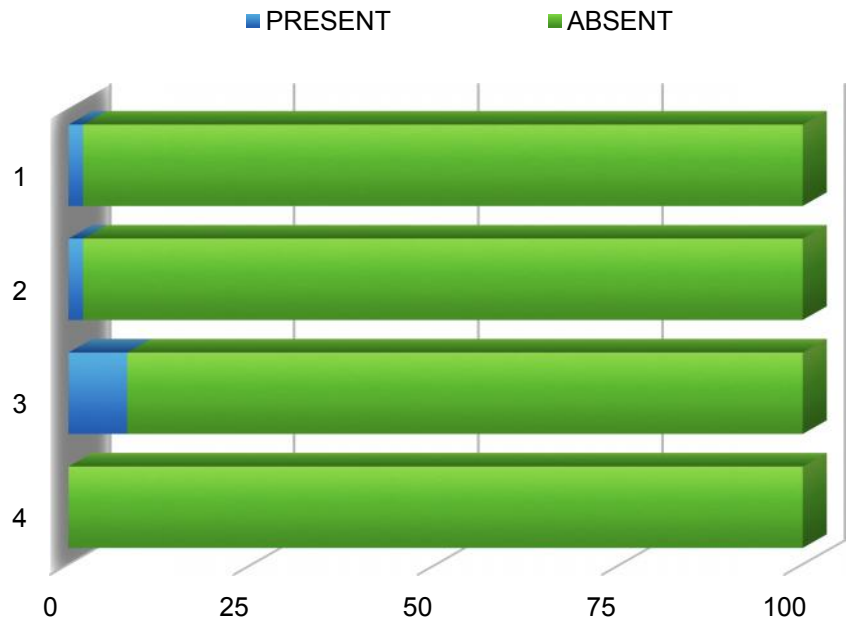

FIGURE.16D

7. CO-RELATION OF HYPERTRICHOSIS LANUGINOSA WITH VARIOUS PARAMETERS

Table.17A and Figure.17A shows the co-relation between hypertrichosis lanuginosa and birth weight. Out of five neonates, two had low birth weight (<2.5 kgs) and three had normal birth weight (>2.5 kgs). The P value was found to be 0.095 which is not statistically significant.

	HYPERTRICHOSIS LANUGINOSA			
Birth weight (kgs)	Present	Absent	Total	P -Value
<2.5	2 (13%)	13 (87%)	15	0.095
>2.5	3 (4%)	86 (96%)	89	
Total	5	99	104	

TABLE.17A

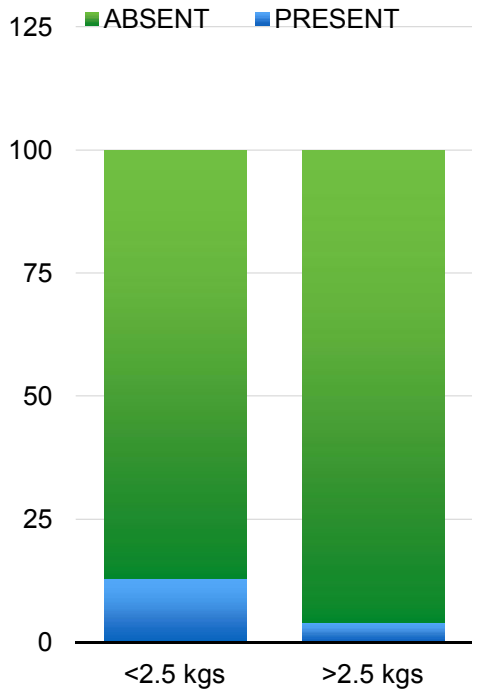

FIGURE.17A

Table.17B and Figure.17B shows the co-relation between hypertrichosis lanuginosa and maturity of the neonate. Out of five neonates, four were term and one was post term neonate. The P value was found to be 0.355 which is not statistically significant.

	HYPERTRICHOSIS LANUGINOSA			
Maturity	Present	Absent	Total	P -Value
Preterm	0 (0%)	3 (100%)	3	0.355
Term	4 (4%)	91 (96%)	95	
Post Term	1 (17%)	5 (83%)	6	
Total	5	99	104	

TABLE.17B

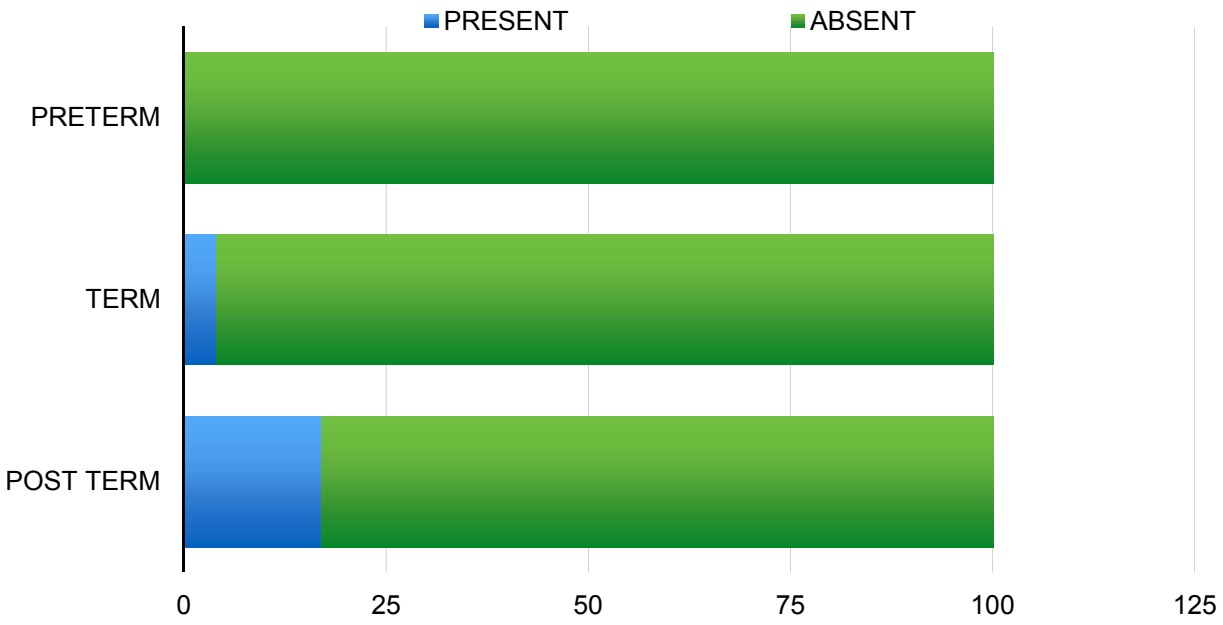

FIGURE.17B

Table.17C and Figure.17C shows the co-relation between hypertrichosis lanuginosa and gender of the neonate. The P value was found to be 0.744 which is not statistically significant.

	HYPERTRICHOSIS LANUGINOSA			
Gender	Present	Absent	Total	P -Value
Male	2 (4%)	47 (96%)	49	0.744
Female	3 (5.5%)	52 (94.5%)	55	
Total	5	99	104	

TABLE.17C

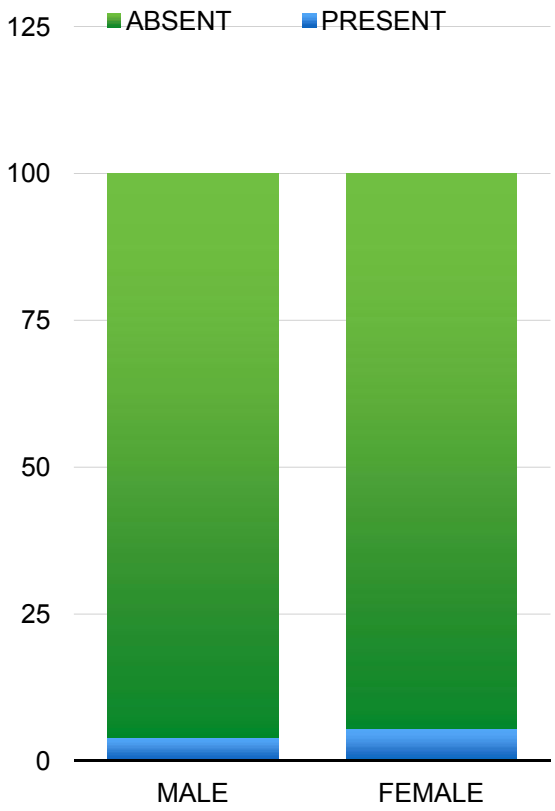

FIGURE.17C

Table.17D and Figure.17D shows the co-relation between hypertrichosis lanuginosa and birth order of the neonate. The P value was found to be 0.747 which is not statistically significant.

	HYPERTRICHOSIS LANUGINOSA			
Birth Order	Present	Absent	Total	P -Value
1	3 (6.5%)	43 (93.5%)	46	0.747
2	1 (2%)	41 (98%)	42	
3	1 (8%)	12 (92%)	13	
4	0 (0%)	3 (100%)	3	
Total	5	99	104	

TABLE.17D

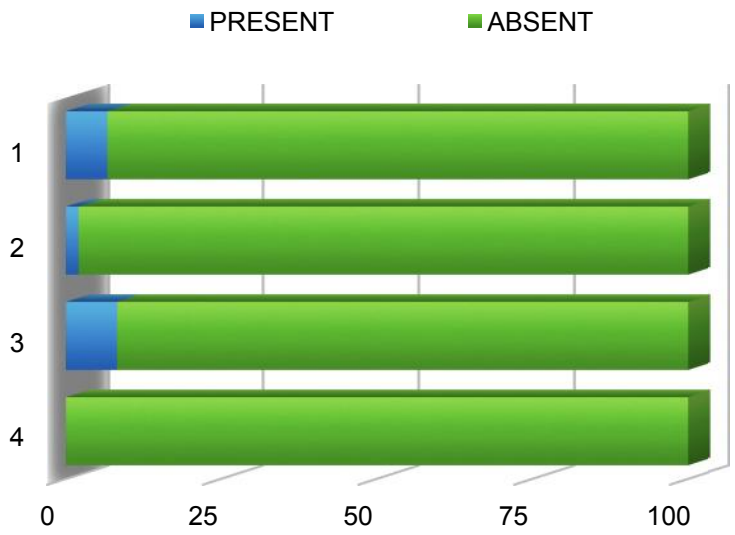

FIGURE.17D

DISCUSSION

Mongolian Spot

In the present study, mongolian spot was found to be the most common dermatosis, seen in 34 (33%) out of 104 neonates.

Mongolian spot was also found to be the most common dermatosis in a study done by Jain N et. al (incidence- 45%) [103] and Zagne V et. al (incidence-50.74%).[104]

The incidence of mongolian spot, ranges from 20.1 to 84.7% according to various studies[103,104,105,106,107,108]

This marked variation may be due to the racial difference in the incidence of mongolian spot. [52]

In our study, mongolian spot was observed more commonly in neonates with low birth weight (i.e <2.5kgs) and this was found to be statistically significant (P-value 0.029). However, in a study done by Basnet S et. al[105] and Kurrey VK et. al[106], mongolian spot was found to be more common in neonates with a birth weight >2.5kgs.

Our study shows no significant co-relation between mongolian spot and maturity of the neonate. In a study done by Basnet S et. al[105], mongolian spot was found to be more common amongst term neonates.

No significant co-relation between mongolian spot and gender of the neonates was found by us. This was similar to the findings by Khoshnevisasl P et. al[107] and Gokdemir et. al[108]

The co-relation between mongolian spot and birth order of the neonate was not found to be statistically significant. However, there are no earlier studies which have been done to compare these findings.

Erythema toxicum neonatorum

Erythema toxicum neonatorum was found in 27 (26%) neonates out of 104. The incidence is similar to the findings in studies done by Haveri FTS et. al[3] and Jain N et. al[103] where the incidence of ETN was found to be 23.2% and 23.33% respectively.

The frequency of erythema toxicum neonatorum varies from 1.3 to 46.8% in various studies.[,3,103,110,111]

A significant co-relation between erythema toxicum neonatorum and normal birth weight (>2.5kgs) was noted by us. This is similar to a study done by Kurrey VK et. al.[106]

There was no significant co-relation seen between erythema toxicum neonatorum and maturity of the neonate. This was against the findings observed by Kurrey VK et. al[106] who noticed it more commonly amongst term neonates.

On the co-relation between erythema toxicum neonatorum and gender, erythema toxicum neonatorum was found to be more common among male neonates which was similar to the findings of previous studies done by Basnet S et. al[105] and Ahsan U et. al.[2]

The reason for male predominance is not clearly understood, but the increased level of adrenal and gonadal androgens in male newborns, may have a direct effect on hair

follicle and on sebaceous glands, which are involved in pathogenesis of erythema toxicum neonatorum.[24]

The co-relation between erythema toxicum neonatorum and birth order of the neonate was not found to be statistically significant. There are no studies in literature so far which have noted and compared these findings.

Desquamation of skin

Desquamation of skin was noted in 21 (20%) neonates out of 104 which was more or less in accordance with the finding of 22.73% observed by Gokdemir G et. al.[108]

The incidence of physiological desquamation as observed in other studies varied from 7.2 to 83%. [113,114,115,116,117]

The incidence of physiological desquamation varies depending on the day of examination, being more in studies where babies were followed up for more than five days. The day of examination (5th –7th day) and onset of physiological desquamation showed statistical significance.[118]

The co-relation between desquamation of skin and birth weight showed no significant association in this study. However in a study done by Kosaraju et. al[119] and Jain N et. al[103] it was found to be more commonly associated with neonates >2.5kgs birth weight.

On the co-relation between desquamation of skin and maturity of the neonate, it was not seen in preterm neonates, and it was seen in 40% of post term neonates and 20% of term neonates in our study. However, this association was not statistically significant. In a study done by Jain N et. al[103] and Sadana H et. al[111], desquamation

was noted more commonly amongst term neonates whereas in a study done by Gokdemir et. al[108], it was found to be more common among post term neonates.

On the co-relation between desquamation of skin and gender, it was found in 27% of females and only 12% of males. However the association was not statistically significant. No sexual predominance was noted by Kurrey VK et. al.[106]

The co-relation between desquamation of skin and birth order was not statistically significant. There are no studies which have been done previously to compare these findings.

Milia

Milia was noted in 10 (9.6%) neonates out of 104. The prevalence of milia as reported by previous studies varied from 7.5 to 36%.[117,120,121]

When co-relating milia with birth weight and maturity of the neonate, no statistically significant co-relation was found in our study. However, in a study done by Gorur DK et. al,[118] milia was seen more in term babies and in babies weighing more than 2.5 kg.

The co-relation between milia and gender of the neonate was not found to be statistically significant. Similar finding was also mentioned by Nobby B et al[113] and Kulkarni et al.[116] But female predominance was observed by Gokdemir G et al.[108]

Miliaria

In this study, miliaria was present in 9 (8.65%) out of 104 neonates.

In other studies incidence of miliaria ranged from 1.7 – 28.3%.[2,103,114,122]

The frequency of miliaria was observed to be 1.7% in american neonates[123] and 4.5% in japanese newborns.[121]

This difference in frequency might be due to the different climatic conditions. Secondly, Indian cultural and social practices of overwrapping the babies, use of massage oils and heat therapy might also be responsible for this difference. Racial differences in the distribution and number of eccrine sweat glands might be an another factor for this difference.[124]

In this study all the neonates with miliaria were term neonates, however this association was not statistically significant. A similar finding was also reported by Sachdeva M et. al,[122] Haveri FTS et. al[3] and Jain N et. al.[103]

No significant association was found between miliaria and birth weight or gender in this study. Miliaria was found to be higher in males in studies done by Haveri FTS et. al[3] and Jain N et. al[103]Miliaria was more in newborns with birth weight ≥ 2.5kg, in a study done by Ahsan U et al[2]

No statistically significant association was found between miliaria and birth order of the neonate. There have been no studies done previously to compare these findings.

Sebaceous gland hyperplasia

Sebaceous gland hyperplasia was observed in three neonates (2.88%) out of 104. This was similar to a study done by Shehab M et. al[123] where the incidence was 3%.

In this study, all the neonates with sebaceous gland hyperplasia weighed >2.5kgs, however, the P-value was insignificant. In studies done by Gorur DK et. al[118] and Kosaraju SC et. al[119] sebaceous gland hyperplasia was found to be more common among neonates with birth weight >2.5kgs.

All the 3 neonates were born at term in this study however the P value is insignificant. This is similar to a study done by Haveri FTS et. al[3] in which majority of the neonates were born at term.

No association was found between sebaceous gland hyperplasia and gender of the neonate. However, it was found to be significantly more in male newborn as observed by Kosaraju SC et. al[119] and Ahsan U et. al.[2]

No statistically significant association was found between sebaceous gland hyperplasia and birth order. There are no studies done previously to compare these findings.

Hypertrichosis lanuginosa

In the present study, hypertrichosis lanuginosa was observed in 5 (4.8%) out of 104 neonates. The incidence of lanugo hair observed in other studies varied from 7-14.6%.[108,113,114]

There was no statistically significant association found between hypertrichosis lanuginosa and various parameters like birth weight, birth order, maturity and gender of the neonate in this study. In a study done by Kurrey VK et.al[106] Jain et.al[103] Sachadeva et.al[123] and Dash K et.al[114] hypertrichosis lanuginosa was found more commonly among preterm neonates. In a study done by Basnet S et.al[105] and El-Moneim et.al[112] hypertrichosis lanuginosa was found to be more common among neonates with normal birth weight. There have been no studies done previously to co-relate hypertrichosis lanuginosa with birth order.

Others

Other less common conditions in this study were: physiological changes like congenital melanocytic nevi in 2 (1.92%) neonates and vernix caseosa in 1 (0.96%) neonate; and pathological changes like bullous impetigo in 1 (0.96%), birth trauma in 1 (0.96%), collodian baby in 1 (0.96%), furunculosis in 1 (0.96%) and intertrigo in 1 (0.96%) neonate each.

In other studies congenital melanocytic naevi in newborns showed a prevalence of 0.4–15.6%.[85]and vernix caseosawas observed in 2.9% to 9.8%.[104,109,107,118]

Vernix caseosa is more prominently seen in term babies with normal birth weight, as low birth weight and preterm neonates possess very immature and incompetent stratum corneum and sebaceous glands.[109]

Although vernix caseosa is a common finding among newborns, with a reported incidence of 2.9% to 9.8%[3,104,107,118] it was observed in only 1 (0.96%) neonate in this study. This could be attributable to differences in cultural practices as majority of the mothers gave a history of wiping off the vernix after birth.

Impetigo was reported in 3.4% and 11.4% of neonates in a study done by Basnet S et.al[105] and Ahsan U et.al[2] respectively. But in studies done on american newborns[123] it was reported to be 0.02% and was found to be 1% in Iranian neonates.[121]

Jain et.al[103] found intertrigo in 3.33% neonates.

In a study done by Javed M[110] collodion baby was found in 0.34% of the neonates.

As the number of cases were less, they could not be co-related with other parameters like birth weight, maturity, gender and birth order of the neonate.

CONCLUSION

Based on the findings in this study, the following conclusions were made:

- Physiological changes were found in 99 (95.19%) and pathological changes in 5 (4.81%)

- The most commonly observed physiological change was mongolian spot in 34 (33%) neonates followed by erythema toxicum neonatorum in 27 (26%) and physiological desquamation in 21 (20%) neonates.

- There was a statistically significant association between mongolian spot and low birth weight. (<2.5 kgs)

- There was a statistically significant association between erythema toxicum neonatorum and normal birth weight (>2.5 kgs), as well as gender i.e it was more commonly observed among males in this study.

- Although skin changes in neonates are common, majority are benign and transient and require no treatment. The parents and care takers need to be reassured about the self limiting nature of these lesions.

- Less often, more serious cutaneous lesions may be seen such as erythroderma, infections like staphylococcal scalded skin syndrome, genodermatosis like epidermolysis bullosa and collodion baby, etc. In such cases early and correct diagnosis with prompt institution of appropriate treatment may even be life saving.

SUMMARY

This study “Cutaneous manifestations in neonates: A one year cross-sectional study in a tertiary care hospital” was conducted between Jan 2016-Dec 2016 by the department of Skin & VD, Dr. Prabhakar Kore Charitable Hospital, Belagavi.

A total of 104 neonates were enrolled for the study from the post natal ward and from the skin OPD. Newborns admitted in the NICU were excluded from the study.

In the present study, out of 104 neonates

- 49 (47%) neonates were male and 55 (53%) were female.
- 53 (51%) neonates were delivered by caesarian section and 51 (49%) were delivered by normal vaginal delivery.
- 3 (2.88%) neonates were preterm, 5 (4.81%) were post term and 96 (92.31%) were full term neonates.
- 16 (15%) neonates had a birth weight less than 2.5kgs and 88 (85%) had a birth weight more than 2.5kgs.
- 46 (44.23%) were first born, 42 (40.38%) were second born, 13 (12.5%) were third born and 3 (2.88%) neonates were fourth born.
- Majority of the neonates i.e 88 (85%) were early neonates (less than 7 days old) and 14 (15%) were late neonates.
- 99 (95.19%) neonates had physiological changes and 5 (4.81%) had pathological changes.

- Mongolian spot was found to be the most common cutaneous change, which was seen in 34 (33%) neonates. It was found to be more common among neonates with a low birth weight (<2.5kgs). This association was found to be statistically significant.

- Erythema toxicum neonatorum was the second most common change observed, present in 27 (26%) neonates. All the neonates had a birth weight >2.5kgs and it was found to be more common among male neonates, with a statistically significant association.

- Physiological desquamation of skin was noted in 21 (20%) neonates . No statistically significant co-relation was found between physiological desquamation and birth weight, maturity, gender and birth order of the neonate.

- Other less commonly observed physiological changes were milia in 10 (9.62%), miliaria in 9 (8.65%), hypertrichosis lanuginosa in 5 (4.8%), sebaceous gland hyperplasia in 3 (2.88%), congenital melanocytic nevi in 2 (1.92%) neonates and vernix caseosa in 1 (0.96%) neonate; and pathological changes like bullous impetigo in 1 (0.96%), birth trauma in 1 (0.96%), collodian baby in 1 (0.96%), furunculosis in 1 (0.96%) and intertrigo in 1 (0.96%) neonate.

LIMITATIONS OF THIS STUDY

Neonates were randomly enrolled into the study without uniformity in the age.

Among 104 neonates, 88 (85%) were early neonates (less than 7 days old) and 16 (15%) were late neonates (7 to 28 days old) which could have lead to variability in results as some physiological changes like physiological desquamation and erythema toxicum neonatorum are more common among early neonates, as observed in this study.

As this was a cross sectional study, the neonates were not followed up to assess if they developed any new lesions in the subsequent late neonatal period.

As per the exclusion criteria, neonates admitted in NICU were excluded from the study. Hence there were less pre-terms enrolled in this study and no cases of hyperbilirubinemia were observed.

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ANNEXURE- I - INFORMED CONSENT FORM

Statement of Consent:

I.D.NO:_____

I Mr/Mrs mother/father of the baby volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's parents name:

Signature or left thumb print of participants parent:

Witness name:

Signature of witness & Date :

Signature of the investigator & Date :

ANNEXURE- II - PROFORMA

Date:

OPD/IPD No.

Name: Age/Sex:

Mothers name: Age:

Fathers name: Age:

Address:

Date of delivery:

Chief Complaints:

Obstetric History:

GRAVIDA	PARA	ABORTION/STILL BIRTH

Antenatal History:

	YES	NO
DIABETES		
HYPERTENSION		
APH		
DRUG INTAKE		
IRRADIATION		
VAGINAL DISCHARGE		
BLOOD TRANSFUSION		
IMMUNIZATION		

Natal History:

Premature rupture of membranes: YES/NO

Type of labour: Spontaneous/induced/normal/prolonged

Type of delivery: Normal/instrumental/C section

Maturity: preterm/term/post term

Postnatal History :

	PRESENT	ABSENT
Cry Breathing		
Activity		
Pallor Icterus		
Cyanosis		
Convulsions Passage of urine		
Passage of meconium		

General Physical examination:

Weight: General appearance:

Pulse: Temperature:

Respiration: Cyanosis : central/peripheral

Head: Skeletal System:

Eyes: Ears:

Systemic Examination:

Cardiovascular system:

Respiratory System:

Abdomen:

Central Nervous System:

Mucocutaneous Examination:

O/E :

Clinical features	YES	NO
Sebaceous gland hyperplasia Epstein pearls		
Mongolian spot		
Desquammation of skin Erythema toxic neonatorum		
Miliaria Milia		
Salmon patch		

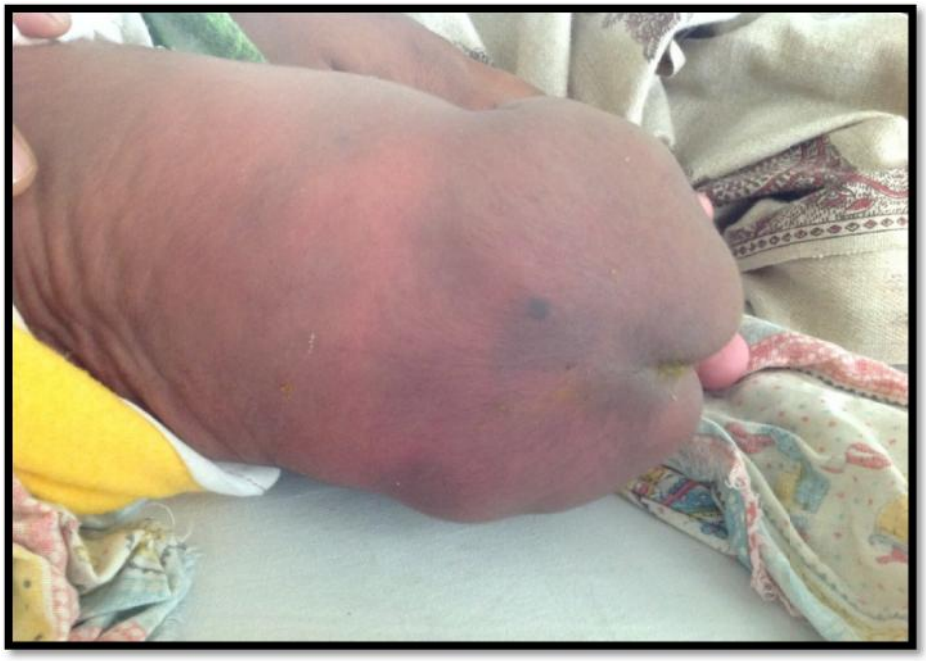
Birth trauma Napkin Dermatitis		
Vernix Caseosa		
Hypertrichosis Birth marks		
Pigmentary changes		
Jaundice Eczema		
Developmental defects		

OTHERS:

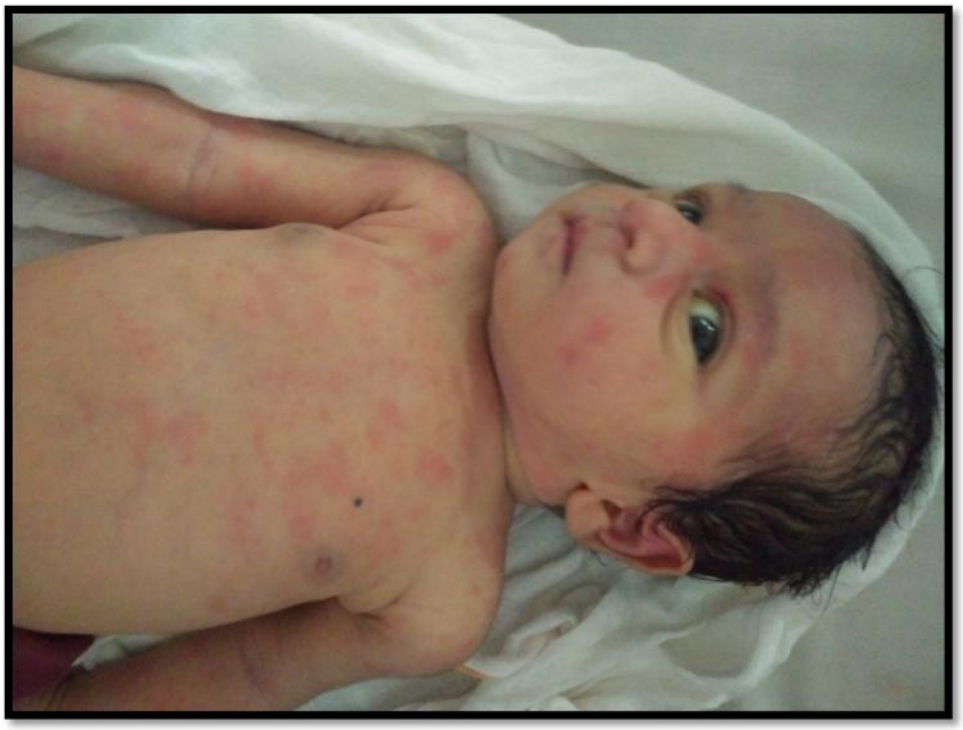
Hair:

Nails:

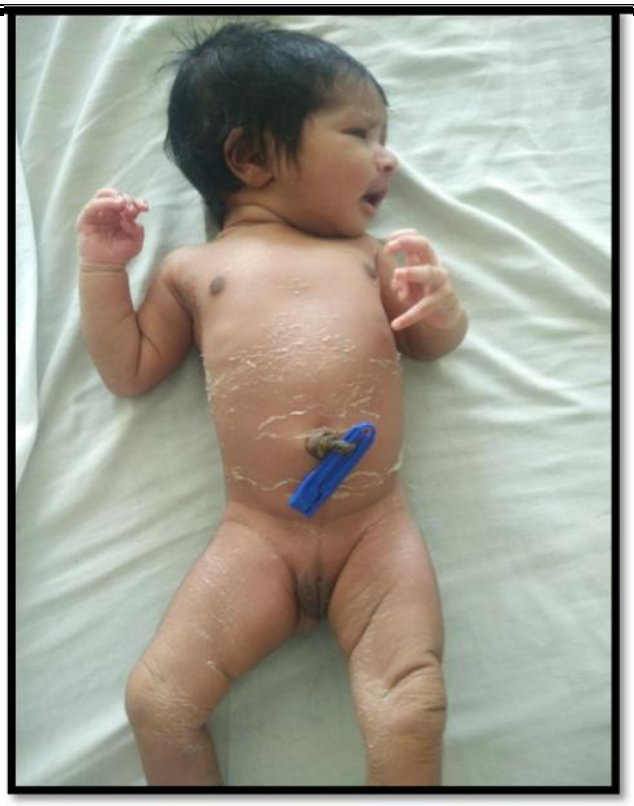
ANNEXURE- III – PHOTOGRAPHS



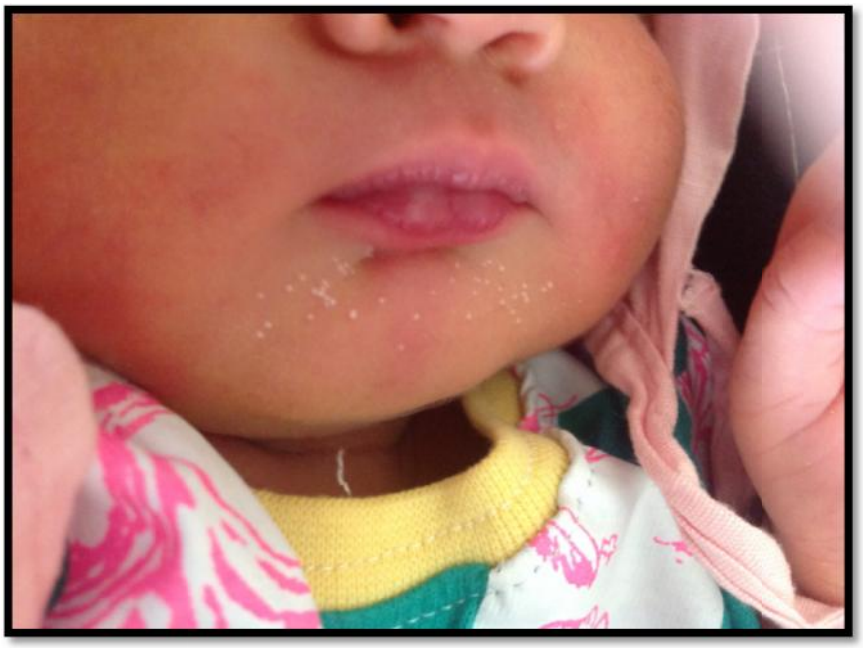
Mongolian spot



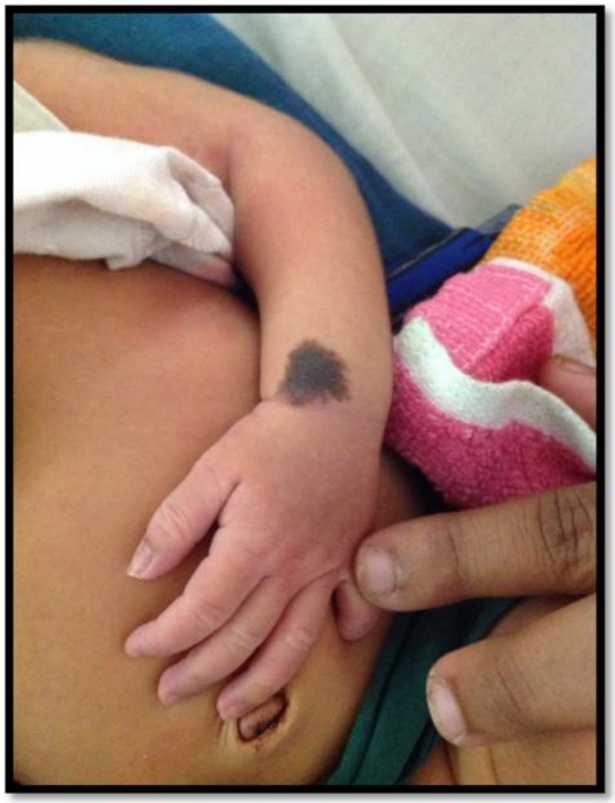
Erythema toxicum neonatorum



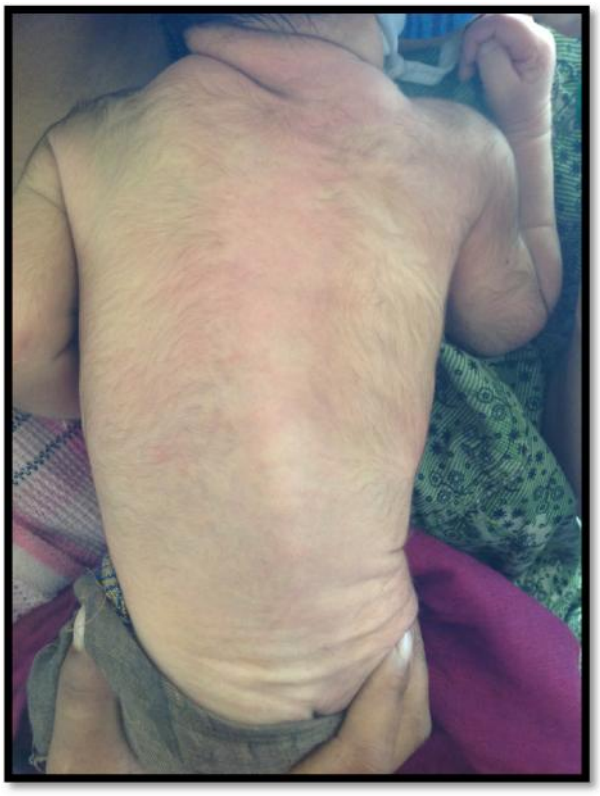
Physiological desquamation



Sebaceous gland hyperplasia



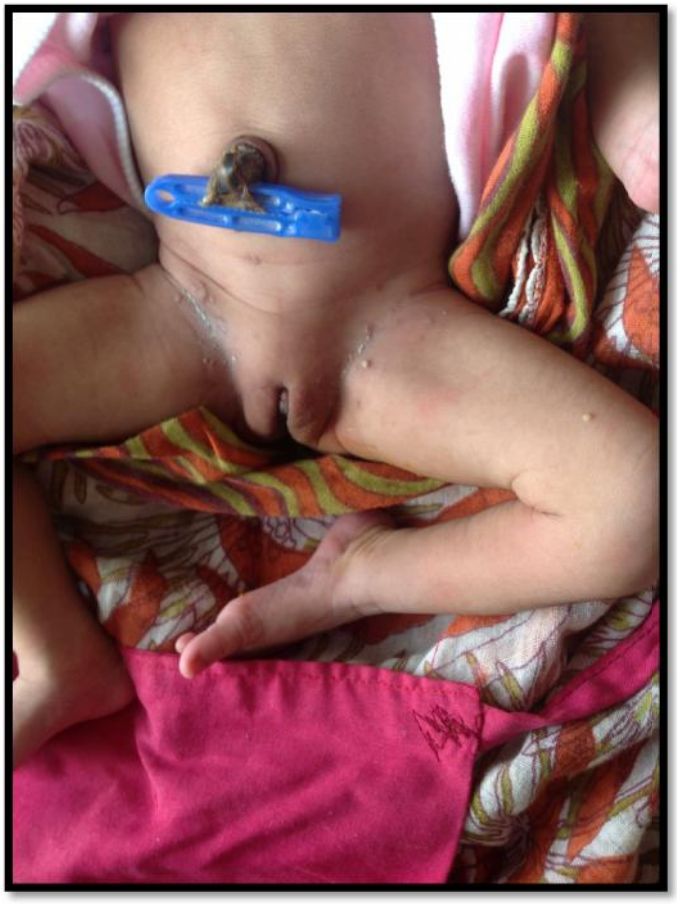
Congenital melanocytic nevi



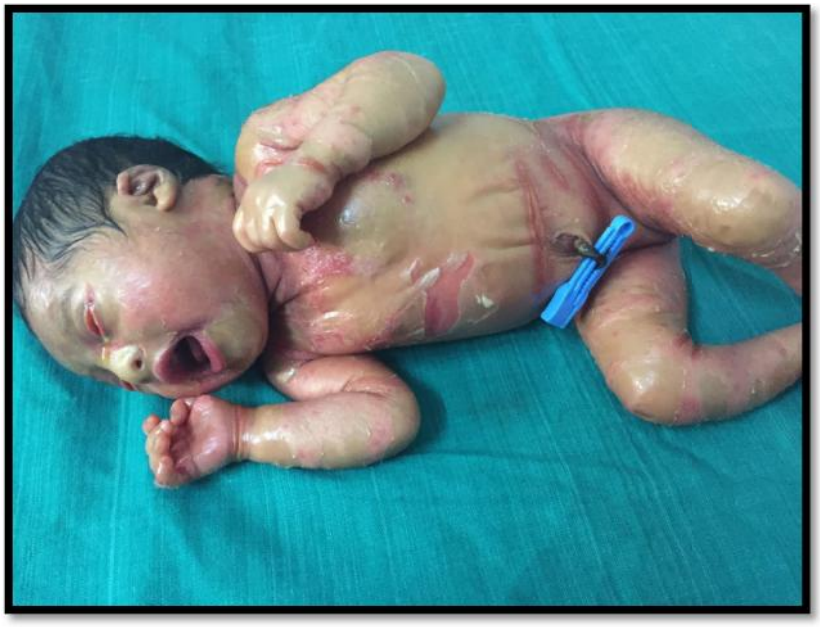
Hypertrichosis lanuginosa

Milia

Miliaria



Intertrigo



Collodian baby

ANNEXURE - IV - MASTER CHART

SI NO.	MATERNAL AGE (yrs)	BIRTH ORDER	MATERNAL DISEASE	MODE OF DELIVERY	MATURITY OF BABY	BIRTH WEIGHT (kgs)	AGE (days)	GENDER	PHYSIOLOGICAL CHANGES	PATHOLOGICAL CHANGES
1	<30	1	-	Normal	Term	>2.5	20	Female	Desquamation of skin,miliaria	-
2	<30	1	-	Caesarian	Term	>2.5	5	Female	Desquamation of skin	-
3	>30	1	-	Caesarian	Term	>2.5	6	Male	Erythema toxicum neonatorum	-
4	<30	2	-	Caesarian	Term	>2.5	4	Male	Erythema toxicum neonatorum	-
5	<30	3	-	Normal	Term	>2.5	9	Female	Miliaria	-
6	<30	4	-	Caesarian	Term	>2.5	5	Female	Desquamation of skin	-
7	<30	1	-	Normal	Term	>2.5	3	Male	Erythema toxicum neonatorum	-
8	<30	1	-	Normal	Term	>2.5	8	Female	Desquamation of skin	-
9	<30	1	-	Normal	Post Term	>2.5	4	Male	Desquamation of skin	-
10	<30	1	-	Caesarian	Term	>2.5	3	Male	Erythema toxicum neonatorum	-
11	<30	1	-	Caesarian	Term	>2.5	3	Male	Erythema toxicum neonatorum	-
12	<30	1	-	Normal	Term	>2.5	1	Female	-	Birth trauma
13	<30	2	-	Caesarian	Term	>2.5	2	Female	Erythema toxicum neonatorum	-
14	<30	2	-	Normal	Term	>2.5	3	Male	Miliaria	-
15	<30	1	-	Caesarian	Term	>2.5	3	Female	Erythema toxicum neonatorum	-
16	<30	3	-	Normal	Term	>2.5	4	Female	Desquamation of skin	-
17	<30	1	-	Normal	Term	>2.5	3	Female	Desquamation of skin	-
18	<30	1	-	Caesarian	Term	>2.5	6	Male	Erythema toxicum neonatorum	-
19	<30	2	-	Normal	Term	>2.5	2	Female	Desquamation of skin	-
20	<30	1	-	Normal	Term	>2.5	1	Male	Erythema toxicum neonatorum	-
21	<30	4	-	Caesarian	Term	<2.5	6	Male	Milia	-
22	<30	1	-	Normal	Term	>2.5	3	Male	Erythema toxicum neonatorum	-
23	<30	1	-	Caesarian	Term	<2.5	11	Male	Desquamation of skin	-
24	<30	1	-	Caesarian	Term	>2.5	5	Female	Miliaria	-
25	<30	1	-	Normal	Term	>2.5	9	Female	-	Furunculosis
26	<30	2	-	Normal	Term	>2.5	3	Female	-	Collodion baby
27	<30	2	-	Normal	Term	>2.5	4	Male	Miliaria	-
28	<30	1	-	Normal	Term	>2.5	3	Male	Erythema toxicum neonatorum	-
29	<30	2	-	Caesarian	Term	>2.5	3	Female	Erythema toxicum neonatorum	-
30	<30	1	-	Normal	Term	>2.5	3	Female	Miliaria	-
31	<30	2	-	Caesarian	Term	>2.5	2	Female	Erythema toxicum neonatorum	-
32	<30	2	-	Normal	Preterm	>2.5	1	Female	Milia	-
33	<30	2	-	Caesarian	Term	>2.5	15	Female	Desquamation of skin	-
34	<30	1	-	Normal	Term	>2.5	5	Female	Desquamation of skin	-
35	<30	2	-	Caesarian	Term	>2.5	5	Male	Mongolion spot, ETN	-
36	<30	3	-	Normal	Term	>2.5	10	Female	Desquamation of skin	-
37	<30	2	-	Caesarian	Term	>2.5	6	Female	Congenital melanocytic nevus	-
38	<30	2	-	Caesarian	Term	>2.5	5	Female	Mongolion spot, desquamation	-
39	<30	1	-	Caesarian	Post Term	<2.5	5	Female	Hypertrichosifs lanuginosa	-

SI NO.	MATERNAL AGE (yrs)	BIRTH ORDER	MATERNAL DISEASE	MODE OF DELIVERY	MATURITY OF BABY	BIRTH WEIGHT (kgs)	AGE (days)	GENDER	PHYSIOLOGICAL CHANGES	PATHOLOGICAL CHANGES
40	<30	2	-	Normal	Term	<2.5	4	Male	Mongolion spot	-
41	<30	3	-	Normal	Term	>2.5	4	Female	Mongolion spot	-
42	<30	1	-	Normal	Term	>2.5	4	Male	Mongolion spot	-
43	<30	1	-	Caesarian	Term	>2.5	9	Female	Desquamation of skin	-
44	<30	2	-	Normal	Term	>2.5	9	Male	Mongolion spot,ETN,milia	-
45	<30	2	-	Caesarian	Term	>2.5	5	Female	Mongolion spot	-
46	<30	1	-	Normal	Term	>2.5	2	Male	Miliaria	-
47	<30	2	-	Caesarian	Term	>2.5	21	Male	Mongolion spot	-
48	<30	3	-	Caesarian	Term	>2.5	6	Male	Desquamation,miliaria	-
49	<30	1	-	Normal	Term	>2.5	4	Male	Mongolion spot, ETN	-
50	<30	1	-	Normal	Term	>2.5	3	Male	Mongolion spot	-
51	<30	2	-	Normal	Term	>2.5	2	Female	Miliaria	-
52	<30	4	-	Caesarian	Term	>2.5	11	Male	Mongolion spot	-
53	<30	2	-	Caesarian	Post Term	>2.5	7	Female	Milia	-
54	<30	2	-	Caesarian	Term	>2.5	6	Female	Erythema toxicum neonatorum	-
55	<30	2	-	Normal	Term	<2.5	3	Female	Milia	-
56	<30	2	-	Normal	Term	>2.5	3	Male	Mongolion spot, ETN	-
57	<30	2	-	Normal	Term	>2.5	2	Male	Milia	-
58	<30	3	-	Normal	Term	<2.5	3	Male	Mongolion spot	-
59	<30	2	-	Normal	Term	>2.5	6	Female	Mongolion spot	-
60	<30	3	-	Caesarian	Term	>2.5	4	Female	Mongolion spot	-
61	<30	1	-	Caesarian	Term	>2.5	12	Male	-	Bullous Impetigo
62	<30	3	-	Caesarian	Term	>2.5	5	Female	Sebaceous gland hyperplasia	-
63	<30	2	-	Caesarian	Term	>2.5	6	Female	Mongolion spot, desquammation, ETN	-
64	<30	1	-	Normal	Term	<2.5	6	Female	Desquamation of skin	-
65	<30	2	-	Caesarian	Term	>2.5	5	Male	Milia	-
66	<30	2	-	Caesarian	Term	<2.5	6	Female	Hypertrichosis lanuginosa	-
67	<30	3	-	Normal	Term	<2.5	4	Female	Mongolion spot	-
68	<30	2	-	Normal	Term	>2.5	3	Female	Erythema toxicum neonatorum	-
69	<30	1	-	Normal	Term	>2.5	3	Female	Mongolion spot	-
70	<30	1	-	Caesarian	Post Term	>2.5	26	Female	Milia	-
71	<30	2	-	Normal	Term	>2.5	2	Male	Erythema toxicum neonatorum	-
72	<30	3	-	Normal	Term	>2.5	10	Male	Mongolion spot, desquamation	-
73	>30	2	HepB Antigen+	Caesarian	Term	>2.5	3	Female	Mongolion spot	-
74	<30	2	-	Caesarian	Term	>2.5	4	Female	Mongolion spot	-
75	<30	1	-	Caesarian	Term	>2.5	4	Male	Hypertrichosis lanuginosa	-
76	<30	2	-	Caesarian	Term	>2.5	2	Female	Mongolion spot, ETN	-
77	<30	1	-	Normal	Term	>2.5	3	Female	Erythema toxicum neonatorum	-
78	<30	3	-	Caesarian	Term	>2.5	4	Female	Mongolion spot,hypertrichosis	-
79	<30	1	-	Caesarian	Term	>2.5	3	Male	Hypertrichosis lanuginosa	-
80	<30	2	-	Caesarian	Term	>2.5	3	Male	Erythema toxicum neonatorum	-

SI NO.	MATERNAL AGE (yrs)	BIRTH ORDER	MATERNAL DISEASE	MODE OF DELIVERY	MATURITY OF BABY	BIRTH WEIGHT (kgs)	AGE (days)	GENDER	PHYSIOLOGICAL CHANGES	PATHOLOGICAL CHANGES
81	<30	2	-	Caesarian	Term	>2.5	4	Female	Mongolion spot	-
82	<30	2	-	Caesarian	Term	>2.5	5	Male	Erythema toxicum neonatorum	-
83	<30	1	-	Normal	Term	>2.5	5	Female	Mongolion spot	-
84	<30	3	-	Caesarian	Term	>2.5	16	Female	Desquamation, Erythema toxicum neon	-
85	<30	1	-	Caesarian	Term	>2.5	5	Male	Mongolion spot	-
86	<30	1	-	Caesarian	Post Term	>2.5	8	Male	Desquamation of skin	-
87	<30	1	-	Normal	Term	>2.5	3	Male	Erythema toxicum neonatorum	-
88	<30	1	Diabetes	Caesarian	Post Term	>2.5	5	Female	Mongolion spot	-
89	<30	1	-	Caesarian	Term	>2.5	3	Male	Milia	-
90	<30	2	-	Caesarian	Preterm	<2.5	4	Male	Mongolion spot	-
91	<30	1	-	Caesarian	Term	<2.5	4	Male	Mongolion spot	-
92	<30	1	-	Normal	Term	>2.5	1	Female	Mongolion spot	-
93	<30	1	Hypertension	Caesarian	Term	>2.5	5	Male	Congenital melanocytic nevi	-
94	<30	2	-	Caesarian	Term	>2.5	3	Male	Sebaceous gland hyperplasia	-
95	<30	2	-	Caesarian	Term	>2.5	3	Female	Erythema toxicum neonatorum	-
96	<30	3	-	Normal	Term	<2.5	1	Male	Mongolion spot	-
97	<30	1	-	Normal	Term	>2.5	3	Female	Sebaceous gland hyperplasia	-
98	<30	2	-	Normal	Term	<2.5	3	Male	Mongolion spot, desquamation	-
99	<30	2	-	Normal	Term	<2.5	1	Female	Mongolion spot	-
100	<30	1	-	Normal	Term	<2.5	3	Male	Mongolion spot	-
101	<30	1	-	Caesarian	Term	>2.5	6	Female	-	Intertrigo
102	<30	2	-	Normal	Term	<2.5	4	Male	Milia	-
103	<30	2	-	Normal	Term	>2.5	4	Male	Mongolion spot	-
104	<30	1	-	Normal	Term	>2.5	4	Male	Erythema toxicum neonatorum	-