Chapter 1

Transient Physiological Skin Disorders in Neonates and Infants

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INTRODUCTION

Cutaneous lesions are of common occurrence in the newborn period. The expression newborn baby defines babies who have just been born up to their 28th day of extra uterine life.[1]

Skin is the largest organ system in the body. It comprises approximately 13% of body weight in the neonate as compared to 3% of the body weight of an adult.[2] Just as the other organs are immature with decreasing gestational age, the skin also is immature; however, with exposure to the postnatal environment, skin undergoes changes rapidly to enhance maturation. Neonatal skin plays a pivotal role in transition from an aqueous to an air-dominant environment by providing mechanical protection and assisting thermoregulation, immune-surveillance, and fluid balance.[3] Mature skin acts as a barrier to invading microbes, while immature skin may systemically absorb substances that result in adverse effects.

Skin, oral mucosa, genitals, hair and nails have been found to be affected nearly universally, in up to 99.3% neonates[4] in dermatoses ranging from transient self-limiting conditions rarely examined by a dermatologist to serious disorders requiring specific therapies, genetic counseling, and family planning. Hence, their correct identification is important for proper management.

Evolution of neonatal skin diseases is much more rapid compared to adults. Some apparently serious skin lesions turn out to be trivial and vice versa. Many transient phenomenons have been clinically recorded on neonatal skin but remain poorly understood. A thorough knowledge of the neonatal skin biology and the cutaneous lesions is expected from those providing neonatal care.

The neonatal skin changes show a wide geographic and ethnic variation. Some skin lesions are common in darker skin race. It is important to know the pattern of dermatosis prevalent among Indian children in the neonatal period.

PHYSIOLOGY OF NEWBORN SKIN

The ability of the infant to adjust to the external environment after delivery mainly depends on the maturity. Full term infants have a well developed epidermis and dermis similar to the skin of adults which is essential for prevention of water loss and defense against micro organisms that begin to colonize the skin. During the late gestation, the number of epidermal layers and the thickness of the stratum corneum increases with the fetal age.[5] The extent of transepidermal water loss and risk of infections with skin colonising organisms in preterm infants are thus directly proportional to the degree of prematurity.

NEONATAL SKIN DISORDERS

Skin disorders of the newborn can be classified as follows:

1. Transient skin disorders that require no treatment
2. Congenital disorders (e.g. birth mark) and geno-dermatoses
3. Acquired skin disorders specific to neonatal period
4. Iatrogenic dermatologic complications seen in the neonatal intensive care unit

TRANSIENT PHYSIOLOGICAL CHANGES AND CUTANEOUS LESIONS IN THE NEWBORN

Skin lesions are common in neonates, and most are transient, benign or physiological conditions. Careful physical examination and complete history helps to differentiate the transient physiological conditions from

potentially life threatening diseases to avoid expensive and unnecessary evaluations and treatment. Transient neonatal dermatoses display many unique characteristic skin lesions that reflect immaturity and transition from intrauterine life. Most of these are normal and self-resolution is common (Table 1.1). Some transient dermatoses are present at birth such as vernix caseosa, physiologic cutis marmorata, "Harlequin" phenomenon, milia, sebaceous gland hyperplasia, Epstein pearl, transient pustular melanosis, "sucking blister", mongolian spots, and "salmon patches". Conditions which manifest later include erythema toxicum of the newborn, vaginal discharge, and breast enlargement.

VERNIX CASEOSA

It is a protective white greasy biofilm covering the fetal skin during the last trimester which acts as a mechanical shield against maceration by amniotic fluid and bacterial infection. It may cover the entire skin or be found concentrated only in the skin folds and back. After a few hours or days of life, it disappears. Vernix is mainly composed of water (80.5%), proteins and lipids (8–10%). These lipids are derived from two sources: wax esters formed in the sebaceous glands and epidermal barrier lipids derived from keratinocytes.[6] Neonatal skin hydration decreases rapidly postnatally and then increases, indicating adaptive changes in stratum corneum water handling properties. Neonatal skin with retained vernix caseosa (Fig. 1.1) is more hydrated than skin with vernix removed. The strategic location of the vernix on the fetal skin surface suggests participation in multiple overlapping functions required at birth, such as barrier to water loss, skin hydration, as a lubricant facilitating passage through birth canal, wound healing, temperature regulation and innate immunity. It contains antimicrobial peptides which protects the fetus prenatally and helps skin colonization by non-pathogenic bacteria in the postnatal period. Vernix seems to perform various integral roles during transition of the fetus from intra-uterine to extra-uterine life. Vernix caseosa which acts as a natural barrier also has antioxidant, disinfectant, and skin cleansing functions.[7] The color of vernix caseosa reflects intrauterine problems. Brownish yellow discoloration is frequently seen in post term infants, contact with meconium and in hemolytic disease of the newborn. Foul smelling infected vernix caseosa can be a sign of neonatal septicaemia. It is normally shed within few hours or within a weeks time after birth. It sheds on its own and should not be removed by wiping or bathing.

TABLE 1.1: Transient skin disorders in neonates and infants

Physiological skin conditions	Vernix caseosa Physiological desquamation Lanugo Sebaceous gland hyperplasia Milia Miniature puberty Physiological jaundice
Transient vascular physiological changes	Cutis marmorata Salmon patch Harlequin colour change Erythema neonatorum and acrocyanosis
Transient vesicopustular eruptions	Erythema toxicum neonatorum Miliaria Infantile acropustulosis Transient neonatal pustular melanosis Neonatal acne Benign neonatal cephalic pustulosis Eosinophilic pustulosis
Oral lesions	Epstein pearls Bohn nodules Sucking pads of lip
Pigmentary skin lesion	Mongolian spots Pigmentary lines of the newborn
Miscellaneous	Sucking blisters Pedal papules of infancy Neonatal occipital alopecia Subcutaneous fat necrosis of newborn Sclerema neonatorum

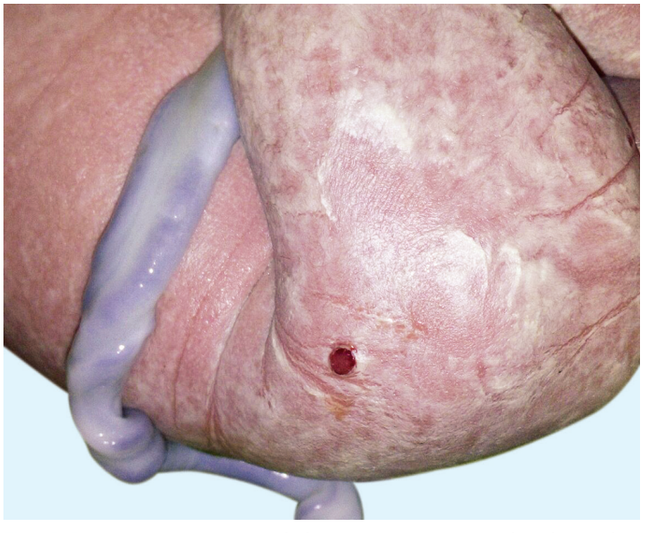

Fig. 1.1: Vernix caseosa with hemangiomatosis: Whitish, sticky, waxy film coating the neonatal skin

PHYSIOLOGICAL DESQUAMATION

Superficial cutaneous desquamation, often termed physiological scaling (Fig. 1.2) of the newborn, occurs in up to 75% of normal neonates. This fine desquamation usually first appears around the ankles on the first day of life, and is most commonly confined to the hands, feet and ankles. It may remain localized or may gradually become more widespread, usually reaching its maximum extent and intensity by the sixth to tenth day. It may persist upto first 3 months of life. In healthy term neonates it begins on the first or second day of life while in preterm babies it starts after 2–3 weeks of birth. It tends to be more thick and generalized in post term babies. Physiological scaling may occasionally need to be distinguished from congenital ichthyosis, X-linked hypohidrotic ectodermal dysplasia and congenital syphilis which may present with severe or persistent skin desquamation and needs further evaluation.[8]

SEBACEOUS GLAND HYPERPLASIA

Sebaceous gland hyperplasia (Fig. 1.3) is a physiological event in the newborn, reflecting the influence of maternal androgens in causing increased activity of sebaceous glands. It is present at birth in 50–89% of newborns, and is more common in full term neonates. It presents as multiple, uniform, pinpoint, yellowish papules with no surrounding erythema that is most prominent around nose, cheek, forehead and upper lip. The papules differ from milia, which are usually discrete, solitary, and whiter in color. Other differentials include neonatal acne and miliaria crystallina. No treatment is needed as spontaneous resolution is seen in few weeks after birth.

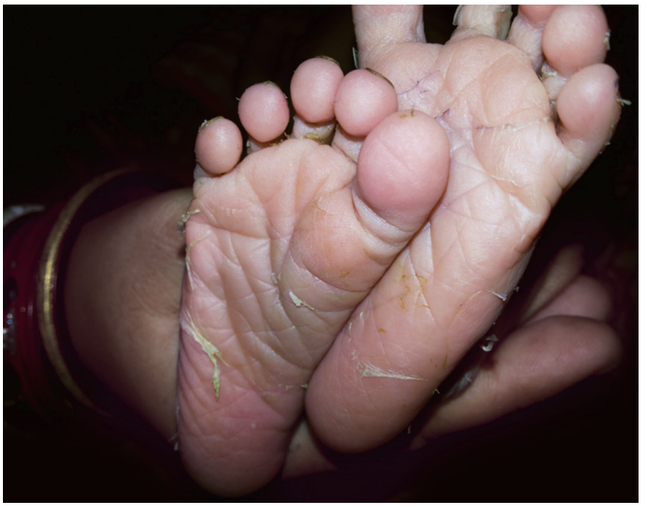

Fig. 1.2: Physiological desquamation predominant on extremities

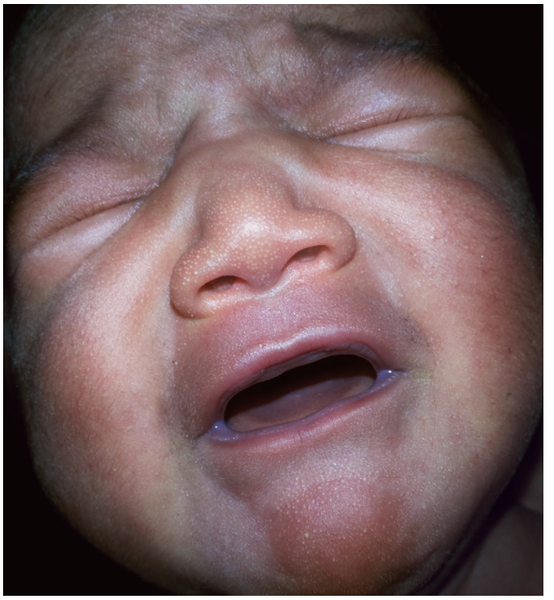

Fig. 1.3: Sebaceous gland hyperplasia (*Courtesy:* Dr Jayesh Kavadya, Raipur)

LANUGO

Often at birth, newborn is covered with fine, soft, non-pigmented and unmedullated immature hair termed as lanugo. It is replaced by vellus hairs during the first month of life. Usually these hairs are shed in utero during last trimester of gestation and so it is seen mostly in preterm neonates. In congenital hypertrichosis lanuginose, lanugo hair are present all over the body except palms and soles and is not replaced by terminal hairs. Lanugo is differentiated from congenital hypertrichosis universalis by presence of terminal hairs over the body.

MILIA

Milia are tiny white smooth surfaced papules, which are usually discrete, but their numbers vary from a few to many (Figs 1.4 and 1.5). Milia are epidermal cysts derived from the pilosebaceous follicle.

Neonatal milia is usually primary milia that are associated with pilosebaceous units arising from the infundibula of vellus hair. Secondary milia usually appear after trauma and originate from a variety of epithelial structures, such as hair follicles, sweat duct, sebaceous ducts, or epidermis.[9] They exfoliate in 3 to 4 weeks without any scarring. Their persistence in unusual sites or widespread distribution needs evaluation for underlying genodermatosis as in facial-digital syndrome, dystrophic form of epidermolysis bullosa, hereditary trichodysplasia and pachyonychia congenital.[10]

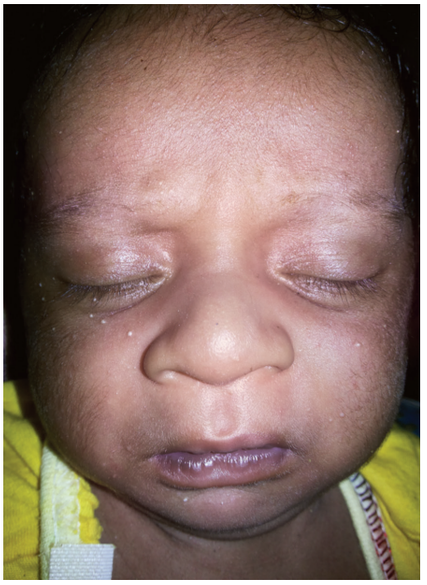

Fig. 1.4 : Milia present over cheeks

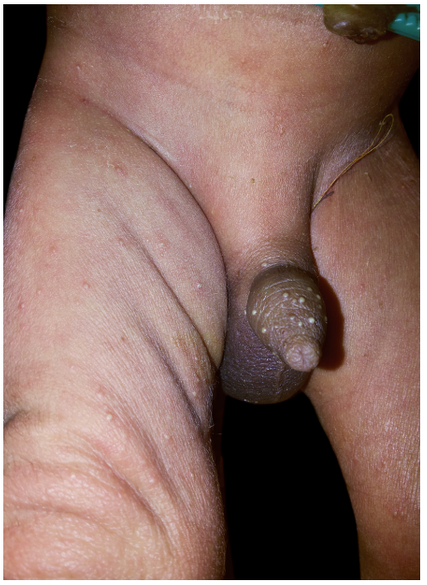

Fig. 1.5 : Multiple milia present over genitals (*Courtesy:* Dr Manas Ranjan Puhan, Cuttack)

MINIATURE PUBERTY

The influence of maternal and placental hormones on the fetus gives rise to a group of phenomena, varying greatly in degree, described as 'miniature puberty'. In the newborn female, the genitalia appears succulent and mucoid vaginal discharge is common (Fig. 1.6). A few days after birth, the hyperplastic vaginal epithelium desquamates to leave a more normal infantile mucosa. This desquamation may manifest as a creamy white

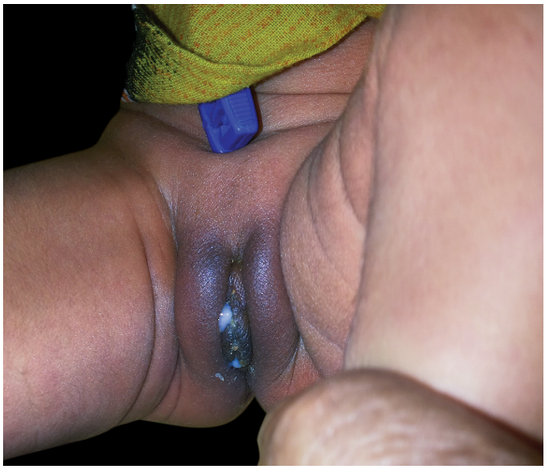

Fig. 1.6 : Miniature puberty (*Courtesy:* Dr Smita Rani Samal, Cuttack)

discharge. Frank withdrawal bleeding may occur from the uterus on the third or fourth day, usually lasting for 2 or 3 days. The male genitalia appears similarly large and well developed at birth. Sometimes pigmentation is prominent of areolas and external genitalia due to influence of hormones. Both sexes show hypertrophy of mammary glands at birth. After 2 or 3 days the breasts may become engorged and lactation of so-called 'witch's milk' may occur. The swelling subsides during the second week and usually become undetectable by the end of the fourth week.

PHYSIOLOGICAL JAUNDICE

Physiological jaundice results from transient elevation of serum bilirubin resulting in a generalized yellow discoloration of the skin, in the few days of life. It fades away after bilirubin returns to normal. Meconium staining will often darken the vernix; it can also leave patchy yellow brown pigmentation especially on desquamating epidermis.[11] Hyperbilirubinemia (>5 mg/dL) occurs in 50% of the neonates during the 1st week. It is biphasic, rising during the first phase of about 10 days to 12 mg/dL and about 2 weeks to 15 mg/dL in term and preterm infants respectively and declining to 2 mg/dL during second phase of about 2 weeks in term and lasting over a month in preterm and exclusively breastfed neonates.[12]

BOHN'S NODULE AND EPSTEIN'S PEARLS

The Bohn's nodule and the Epstein's pearls are micro-keratocysts which can be single or multiple whitish lesions present in oral mucosa considered as analogous to milia. Epstein pearls are located at the junction of soft and hard palates or at palatal midline. Bohn's nodule is located on

the buccal or lingual aspects of alveolar ridges. They disappear within few weeks to months without any treatment.

They need to be differentiated from other oral lesions like dental lamina cysts which are greater in size, fluctuant, solitary and more transparent. Natal and neonatal teeth are rare premature eruption of tooth and are usually located in the lower incisor region.

SUCKING PADS OF LIPS

Sucking pads are considered as physiological adaptive change of lips in response to sucking. They are painless, whitish and hyperkeratotic swelling of lips predominantly in middle of upper lip. They disappear in 3 to 6 months after breastfeeding is stopped.

RUBOR AND ACROCYANOSIS

These represent manifestations of vasomotor instability, as newborns adjust from intra amniotic to extra uterine life. Generalized rubor is often seen in first several hours of life and reflects cutaneous vasodilatation and hyperemia. Rubor or erythema neonatorum is thought to be reflex vasodilation of cutaneous capillaries due to decreased sympathetic tone present at birth. It is prominent when newborn cries, is handled and during bath.

Acrocyanosis in contrast is characterised by bilateral and symmetric bluish discoloration of hands and feet that is intermittent and variable in intensity. The coloration is blanched with pressure and decreases by warming of the extremities. Acrocyanosis is more pronounced in hypothermia, polycythemia and other hyperviscosity syndromes. Usually no treatment required.[11] It should be distinguished from other conditions causing central cyanosis like congenital cardiac disease or respiratory disease.

CUTIS MARMORATA

A transient, benign, reticulate, mottled bluish discoloration of the skin that may last minutes to hours is termed cutis marmorata. It is exaggerated response to hypothermia. Profound or persistent cutis marmorata has been reported with Down syndrome, trisomy 18, hypothyroidism and Cornelia de Lange syndrome.[13] Physiologic cutis marmorata is distinguishable from cutis marmorata telengectatica congenita (CMTC), a persistent vascular anomaly that presents at birth. However, this is fixed and does not disappear after warming (unlike physiological cutis marmorata). In cutis marmorata no specific therapy is required, but rewarming may be required if environmental temperature and core body temperature are not appropriate.

HARLEQUIN COLOR CHANGE

Harlequin color change is defined as transient erythema involving one half of the infant's body with simultaneous blanching of the other side and a sharp demarcation in the midline. It occurs in 10–15% of neonates. The harlequin color change is most frequently seen 3 to 4 days of birth. Seen mostly in healthy child, the episode may last for 30 seconds to 20 minutes.

SALMON PATCH (NEVUS SIMPLEX, CAPILLARY ECTASIA)

Erythematous macules and patches occurring over the occiput, eyelids (Fig. 1.7) glabella, and to lesser extent the nose and on the upper lip. They are minor vascular malformations consisting of ectatic capillaries in the upper dermis in the normal overlying skin. Most resolve over several months to years, but 25–50% of nuchal lesions and a much percent of glabellar lesions may persist in the adult life. The primary differential of this benign lesion is port wine stain which is usually more lateral in location, does not resolve and often continue to darken and thicken with age.

ERYTHEMA TOXICUM NEONATORUM (ETN)

Erythema toxicum is also called toxic erythema or urticaria neonatorum, erythema neonatorum is the most common transient rash in healthy neonates (Fig. 1.8).

Etiology

The exact cause is not known. Initially it was thought to be an allergic response but failed to establish allergic agents. Some thought it may correspond to minor graft versus

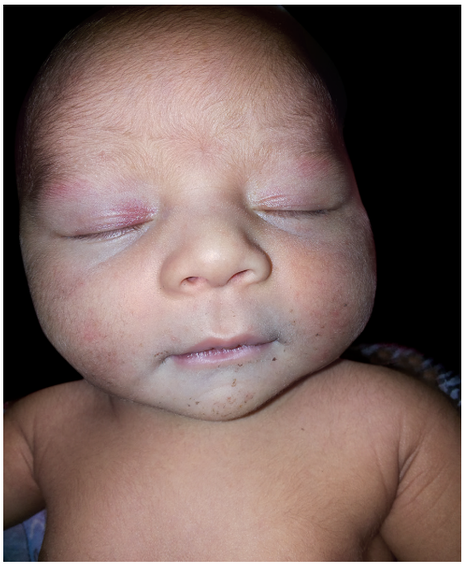

Fig. 1.7: Salmon patch on right upper eyelid

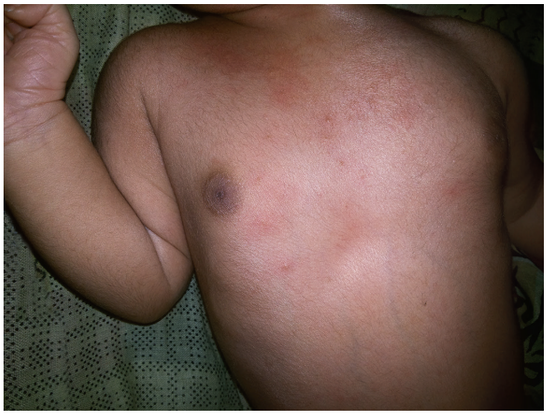

Fig. 1.8 : Erythema toxicum neonatorum

host reaction against maternal lymphocytes but failed to demonstrate any maternal cell in lesions. A variety of inflammatory mediators such as IL-1 alpha, IL-1 beta, IL-8, exotoxin, aquaporins 1 and 3, nitric oxide syntheses 1, 2 and 3 and HMGB-1 have been associated immunohistochemically with erythema toxic neonatorum.[14]

The human skin is equipped with an antimicrobial skin defense system active prior to birth, and that this antimicrobial system is strengthened during the first days after birth by an acute cellular inflammatory response, leading to ETN rash.[15]

Clinical Features

The incidence is directly correlated with several parameters of maturity, including birth weight and also the number of pregnancies. Time of onset is between first and fourth day of life, delayed onset as late as 10 days has also been reported. Two types of eruptions are encountered: erythematous papular lesions and pustular variant. The most frequent type consists of erythematous papular lesions with characteristic flea bitten appearance that is white-yellow papules on an erythematous base. In 30% of cases the eruptions are predominantly pustular. The sterile pustules are white, 1–2 mm in diameter and occasionally surrounded by an erythematous rim. Lesions usually comes in crop and are evanacent. Lesions are commonly located on trunk, with predilection for back, upper arm and face. Palms and soles are spared. Atypical ETN with pustules localized to genital areas is reported.[16]

Histology reveals follicular centered eosinophils. ETN has to be differentiated from other infectious conditions like impetigo, listeriosis, herpes, candidiasis and non infectious conditions like incontinentia pigmenti, eosinophilic pustulosis and histiocytosis. No treatment is required as lesions are generally self limiting.

TRANSIENT NEONATAL PUSTULAR MELANOSIS (TNPM)

TNPM is characterized by pustules that are present at birth and evolve into areas of macular pigmentation. TNPM is more common in dark neonates. The higher incidence in black neonates may be related to an accelerated stimulation of Negroid melanocytes due to cytokines and release of growth factor by cells in the epidermal infiltrate.

Clinical Features

It is characterized by the presence of multiple pustules or vesicles measuring 1–5 mm over a normal-appearing skin (Fig. 1.9). Lesions are found singly or in clusters. New lesions cease to appear after birth and vesicopustules break down, leaving a fine collarette of scale. Days later the lesions resolve leaving behind hyperpigmented macules.[17] No treatment is required.

MILIARIA

Miliaria occurs when the flow of eccrine sweat gland is impeded by obstruction of the sweat duct. Relative immaturity of the sweat duct may be an important predisposing factor in early infancy, as may a tendency for infants to be nursed in excessively warm and humid conditions. The clinical manifestations of miliaria vary, depending on the level of obstruction.

Miliaria crystallina (Fig. 1.10) appears to reflect obstruction of the sweat duct within the stratum corneum itself. Miliaria rubra appears to be caused by sweat duct obstruction deeper within the epidermis, induced

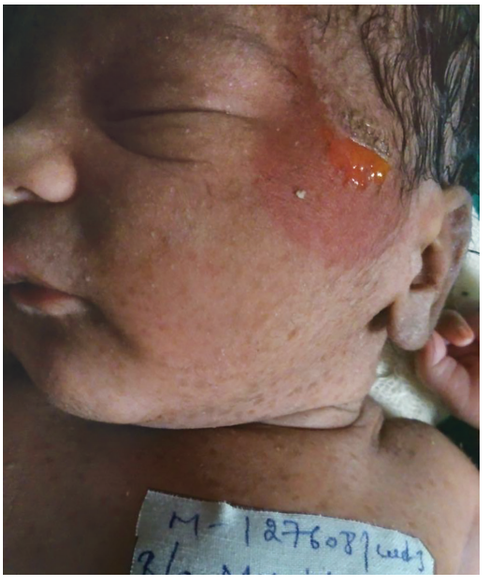

Fig. 1.9 : Transient neonatal pustular melanosis (*Courtesy:* Dr Varsha Mishra, Delhi)

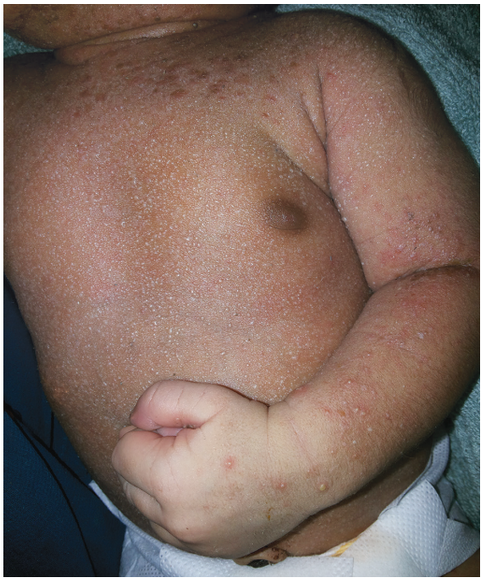

Fig. 1.10: Miliaria crystallina

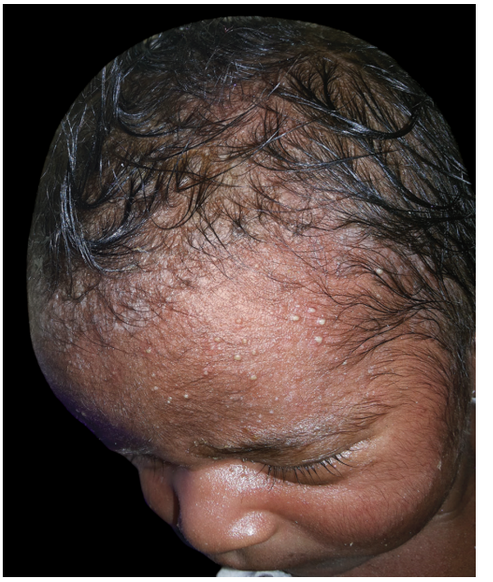

Fig. 1.11: Miliaria pustulosa (*Courtesy:* Dr Binodini Behera, Cuttack)

perhaps by increased activity of the intraductal micro flora.

Clinical Features

Miliaria crystallina presents, within the first few weeks of life as asymptomatic, non-inflammatory, fluid-filled vesicles 1 to 2 mm in diameter, clustered on the face, neck, and trunk. These vesicles often occur during a febrile illness or hot humid enviornment.[18]

Miliaria rubra ('prickly heat') comprises erythematous papules and papulovesicles about 1–4 mm in diameter, on a background of macular erythema. Crops of lesions arise symmetrically, most often in flexural areas, around the neck and in the groins and axillae. The face, scalp and upper trunk are also affected along with areas of occlusion. Each crop of lesions will subside within 2–3 days, but recurrences are common. Pustular variant of miliaria rubra is termed as miliaria pustulosa (Fig. 1.11).

When it occurs during the first few days of life, miliaria rubra is often confused with toxic erythema. Miliaria rubra can be distinguished by its flexural predominance, by the frequent presence of vesicular lesions and by its tendency to recur. When pustular lesions are prominent in miliaria, there may be confusion with infantile acne or with folliculitis, but pustules in miliaria are not follicular oriented. Miliaria resolves following elimination of environmental and physical factors that cause sweat gland occlusion or increase in local temperatur. No specific therapy is indicated. Cotton clothing, cool baths and soothing agents like calamine lotion may be helpful.

Miliaria profunda, the third and deepest level of sweat gland obstruction, has occlusion at or below the dermoepidermal junction and is rare in newborn period.

NEONATAL ACNE

Neonatal acne (Fig. 1.12) may occur at birth and usually disappears within the first 2–3 weeks of life. Clinically presents as inflammatory and non-inflammatory acne lesions. Lesions can be closed comedones and sometimes open comedones and inflammatory lesions like

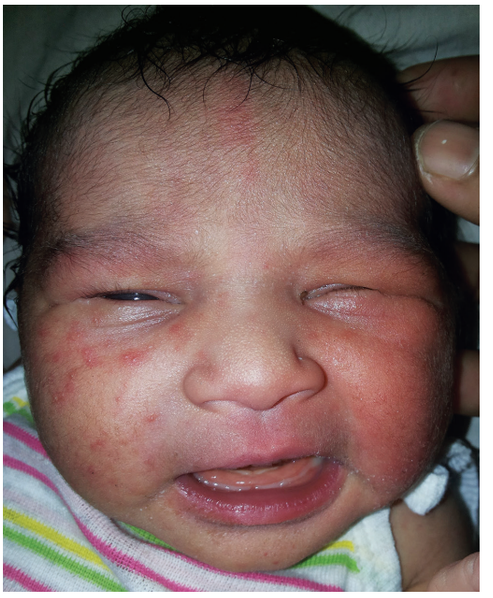

Fig. 1.12: Neonatal acne

erythematous papules and pustules can be seen. Rarely cyst and nodules can be seen leading to scarring. Face is usually affected specifically cheeks and forehead. Etiology includes genetic factor, hormonal factor and also role of *Malassezia furfur*. Clinical differentiation from miliaria rubra and benign cephalic pustulosis is difficult. Neonatal acne resolves spontaneously without treatment.

BENIGN CEPHALIC PUSTULOSIS

Neonatal cephalic pustulosis is characterized by a papulopustular facial eruption usually concentrated on the cheeks, but forehead, chin, eyelids, neck, upper chest and scalp may also be affected. The mean age of onset is 2–3 weeks with some cases beginning as early as first week of life. *Malassezia furfur* and *M. sympodialis* has been suspected as one of the causes of this condition.[19] Treatment is with topical ketoconozole and low potency steroids.

INFANTILE ACROPUSTULOSIS

Infantile acropustulosis (Fig. 1.13) is an uncommon disorder which may begin during the neonatal period and continue throughout infancy and early childhood.[20]

Extremely pruritic vesicles and pustules concentrated on hands and feet makes the neonate restless and irritable. Clinical manifestations are limited to the skin and lesions begin as small red papules, evolving within 24 hours into vesicles and pustules which are several millimeters in diameter. They last for 7 to 10 days, and appear in crops every 2 to 3 weeks. A Tzanck smear or Gram stain of pustular content reveals numerous neutrophils, occasional eosinophils and no bacteria.[21] Topical corticosteroids may be valuable. In severe cases dapsone at a dose of 1 to 2 mg/kg/day may be effective.

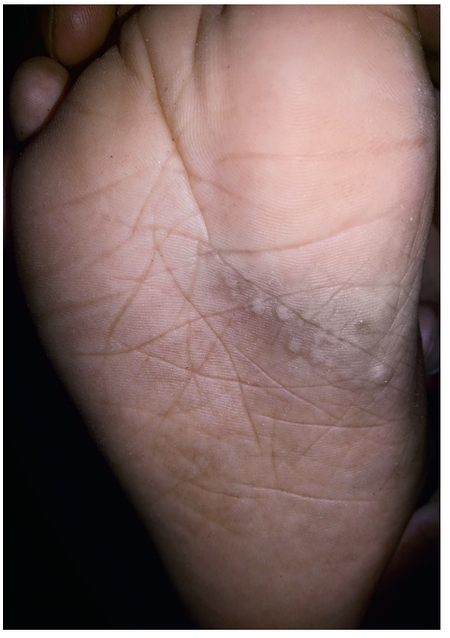

Fig. 1.13: Infantile acropustulosis (*Courtesy:* Dr Prasenjeet Mohanty, MD Skin and Vd, SCB Medical College, Cuttack)

EOSINOPHILIC PUSTULAR FOLLICULITIS

Eosinophilic pustular folliculitis is a rare pustular disorder. Several cases have been reported in young infants, including a few cases with onset at birth or in the first few days of life.[22] In the neonatal cases, pustules develop primarily on the scalp and face but also intermittently on the trunk or extremities. The lesions are usually very pruritic. After successive crops, the disease resolves spontaneously within 1 month to 3 years. Blood eosinophilia is present at the beginning of flares in 70% patients.[23]

MONGOLIAN SPOT

Mongolian spots (Figs 1.14 and 1.15) are due to collections of melanocytes located in the dermis. The pigmentation is macular, diffuse and more or less uniform, slate blue to grey and usually relatively faint. This is the most common variety, being almost a normal finding in Asian babies. The patches are usually rounded or oval in shape, up to 10 cm or so in diameter, and usually single but occasionally multiple. The lumbosacral region is the common site, and the buttocks, flanks or even shoulders and lower legs may be affected in extensive lesions. The skin over the sacrum and lower back is blue in color but is otherwise normal. Occasionally, the blue pigmentation may be more extensive, involving the limbs. With age, the melanocytes disappear and the pigment usually resolves, often by the age of about 4 years. Occasionally the pigment persists into adult life. Rarely, the Mongolian spots may occur as part of phakomatosis

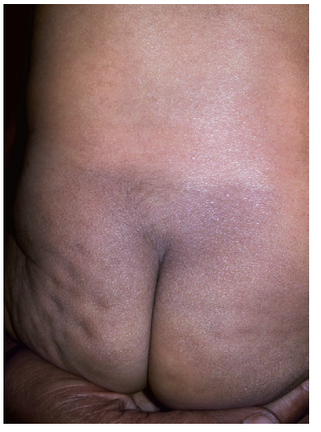

Fig. 1.14: Mongolian spot at lumbosacral region

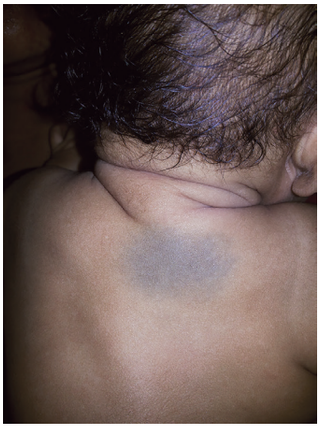

Fig. 1.15: Mongolian spot at nape of neck

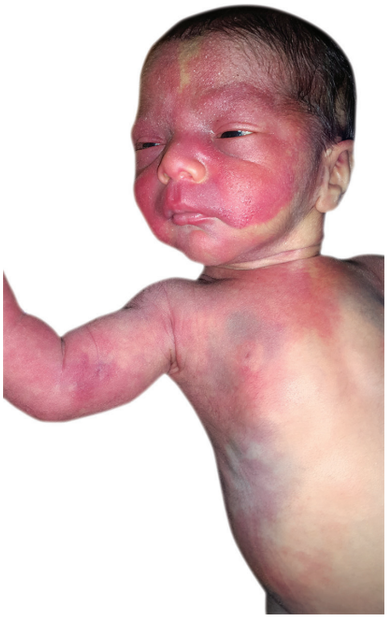

Fig. 1.16 : Phakomatosis pigmentovascularis

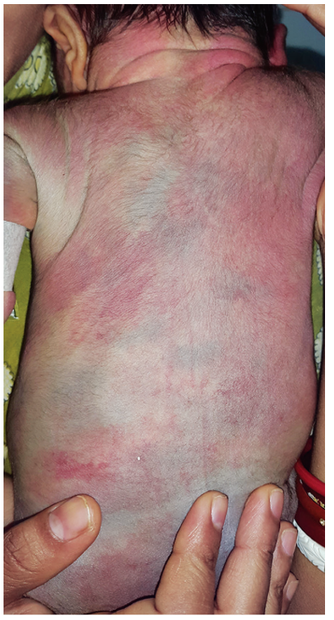

Fig. 1.17: Mongolian spot and salmon patch covering the trunk in 8th day baby of phakomatosis pigmentovascularis

pigmentovascularis[24,25] (Figs 1.16 and 1.17) in which there are vascular lesions as well as Mongolian spot or naevus spilus. The vascular lesions include naevus flammeus, naevus anemicus and cutis marmorata.

These lesions that appear blue in color is because of the optical effects of light reflecting off the melanin deep in the dermis. The skin absorbs blue light less readily than the rest of the visible spectrum and it is therefore more likely to be reflected by deeper melanin (the Tyndall effect).[26] There have been studies suggesting that Mongolian spots manifest more often in children with certain inborn errors of metabolism, including mucopolysaccharidosis, GM1 gangliosidosis (GM1 gangliosidase also called beta galactosidase 1 deficiency) and in Hurler syndrome.[27] No treatment is required.

EPIDERMAL HYPERPIGMENTATION

In more darkly pigmented neonates, transient nearly black hyper-pigmentation can be observed in genital regions on the labia and scrotum, in a linear fashion on the lower abdomen (linea nigra), around the areola, in the axillae, on the pinna and at the base of finger nails. This is due to stimulation by melanocyte stimulating hormone *in utero*.

SCLEREMA NEONATORUM

It is a rare disorder of panniculus that is seen usually in premature, low birth weight or sick babies in the first few weeks of life. Clinically it is characterized by woody induration of skin which usually starts from thigh or cheeks and can spread to rest of the body. The skin becomes yellowish, mottled, stony hard. Limbs become immobile and face is mask like. Usually palms, soles and genitalia are spared. Histopathology shows increase thickness of adipocytes and needle shaped clefts in adipocytes is characteristic.

The infants are sluggish, feed poorly, presents with clinical signs of shock. Treatment is supportive like thermal regulation, fluid and electrolyte balance and treatment of underlying cause.

SUBCUTANEOUS FAT NECROSIS

It is a benign, self limiting disease affecting healthy, full term infants. Clinically lesions start as single or multiple, localized, sharply demarcated, painless areas of induration with no deeper attachment. Sometimes they may be tender to touch. It is seen usually on the cheeks, back, buttocks, thigh and arms.

Etiology is unknown and can be related to maternal pre-eclampsia, perinatal trauma, asphyxia, meconium aspiration, hypothermia and hypercalcemia. Histopathology shows large fat globules and inflammatory infiltrate, needle shaped cleft in adipocytes, necrosis, and calcification. All infants should undergo serum calcium level to exclude hyperparathyroidism.

Generally no treatment is required if hypercalcemia is ruled out. Restricted calcium intake, vitamin D,

systemic steroids and furosemide can be used in case of hypercalcemia.

PEDAL PAPULES OF INFANCY

They present as symmetric, painless, flesh coloured nodules on medial aspects of an infant's heels. Very few cases are known so pathogenesis is poorly understood. The lesions should be differentiated from lipoma, fibrous hamatoma etc.

SUCKING BLISTERS

It results from vigorous sucking by the infant during the fetal life. The lesions are usually present at birth and are not associated with other abnormalities. The bullae are usually flaccid, varying in size from 5–15 mm and may evolve rapidly to become superficial linear or round erosions. Characteristic locations include the forearm, wrist and hand including the dorsal thumb and index finger. The diagnosis can be suspected by typical skin lesions on characteristic sites and absence of vesicles on other body parts. Sucking pads are often confused with sucking blisters but can easily be differentiated as they are clearly blisters not hyperkeratotic lesions and generally present on accessible areas like thumb or arm.[28] Other differentials include bullous impetigo, epidermolysis bullosa etc. Lesions resolve spontaneously in one or two weeks and do not recur aiding in diagnosis.

NEONATAL OCCIPITAL ALOPECIA

It is physiological shedding of occipital hairs usually seen at 2 to 3 months of age. It was thought to be due to friction but now it is considered as part of physiological shedding. No treatment is required.

TAKE HOME MESSAGE

1. Cutaneous manifestations are frequent in newborns.Usually no treatment is required for transient conditions
2. Early recognition is important to distinguish these lesions from more serious skin disorders
3. Parents are more apprehensive for birthmarks as compared to transient conditions of neonates
4. Neonatal dermatoses require proper training and the close liaison between the pediatrician and the dermatologist to save neonate from unnecessary intervention
5. Counseling of parents is of utmost importance while dealing with neonatal dermatoses

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