Prevalence of skin disorders among neonates

Thesis
Submitted for partial fulfillment of master degree in
dermatology and veneorology
By

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Introduction:

The skin is the most visible and easily accessible organ of the body. It serves many purposes, acting as a barrier against infection, protecting internal organs, contributing to thermoregulation, storing insulating fats, excreting electrolytes and providing tactile sensory input **(Shwayder and Akland, ٢٠٠٥).** Neonatal skin plays an important role with vernix caseosa through their antimicrobial properties to protect the neonate in utero and after birth **(Marchini et al., ٢٠٠٢).**

Neonatal period is the first ٢٨ days of life after birth. Skin disorders are commonly seen during the neonatal period and they may be benign transient lesions, napkin dermatitis and related disorders, lesions of infections, blistering dermatoses, genodermatoses, or birth marks **(Mallory, ٢٠٠١).**

Benign transient cutaneous lesions include; transient vascular phenomena and transient pustular or papulopastular dermatosis which are common skin findings in neonates. Napkin dermatitis is commonly seen in neonates and it should be differentiated from other disorders that may involve the napkin area like psoriasis or seborreic dermatitis **(Treadwell, ١٩٩٧).**

Skin infections are extremely common during the neonatal period. Neonates are more exposed to bacterial infections especially the contagious ones such as impetigo. Viral infections can affect the neonates, while most of them are fatal in this age group. Fungal and parasitic infections are less commonly to occur in neonates and they need strong family history especially in the mothers (**Stevens et al., ٢٠٠٥).**

Blistering dermatoses are classified into mechanobullous diseases (Epidermolysis bullosa) and autoimmune blistering diseases. Some of these disorders denote life threatening condition such as neonatal lupus erythematosus, so it is important to recognize it early. Genodermatoses are inherited genetic skin conditions that include wide variety of diseases; Icthyosis is one of the most common genodermatoses **(Treadwell, ١٩٩٧).**

Birthmarks include two main categories; pigmented and vascular birthmarks. Pigmentation birthmarks include; hyperpigmentation disorders include; freckles, ephelids, café au lait spots or congenital nevi, while hypopigmentation disorders include; albinism, piebaldism, hypomelanosis of Ito or nevus depigmentosus. Vascular birthmarks include; vascular malformations, vascular tumours eg. hemangiomas and hamartomas **(Osburn et al., ٢٠٠٠).**

Aim of the study

The aim of this work was to study the prevalence of different skin disorders among neonates over one year and to detect their risk factors.

Skin structure

In the earliest days of fetal life, the epidermis is composed of two layers; the outer layer is the periderm and the inner layer is the stratum germanitivum. Stratum germanitivum is responsible for the development of the basal cell layer and eccrine sweat glands. The primary epithelial germ cells give rise to the sebaceous glands, apocrine glands and hair follicles **(Chang and Orlow, ٢٠٠٨).**

The skin of neonate has a smooth appearance and the histologic features are mainly like adult skin with some variations. The main differences include immaturity of collagen, hair follicles and sebaceous glands which undergo more modification with increase of age**.** The dermoepidermal adhesions in babies are less than in adults. This explains the stronger skin reactions of neonates in response to certain stimuli such as in bullous papular urticaria due to insect bites or other stimuli **(Shwayder and Akland, ٢٠٠٥).**

Skin functions

The most important functions of the skin include; barrier against infections and loss of fluids, thermoregulation, photo protection and excretion of toxic substances with sweat. It has immunological function mediated by Langerhans cells, acts as sensory organ and helps vitamin D synthesis from its precursors under the effect of sunlight (**Mazurek et al., ١٩٩٩).**

The anatomical barrier is presented by the integrity of stratum corneum. Birth triggers lipid and DNA synthesis that is followed by cornification of keratinocytes and formation of stratum corneum. It takes one month for the skin of a full-term infant to develop its full barrier function and even longer in preterm babies. Metabolic processes of the stratum corneum, forms acid mantle that inhibits the growth of pathogenic bacteria and maintains epidermal barrier integrity **(Chang and Orlow, ٢٠٠٨).**

Vernix contains several antibacterial polypeptides that are active against common bacterial and fungal pathogens (**Akinbi et al., ٢٠٠٤).** World Health Organization guidelines for newborn care recommend that vernix should not be removed from the skin of newborn for at least ٦ hours after birth because it has been approved that leaving vernix on the skin of the newborn produces earlier skin acidification (**Visscher et al., ٢٠٠٥).**

The anatomical barrier of the skin is presented by the stratum corneum, the acid environment, commensal microflora and antimicrobial peptides **(Shwayder and Akland, ٢٠٠٥).**

The thermoregulation mechanism in neonate differs from that in adult skin. The differences are mainly due to high area-to-body volume, minimal subcutaneous fat and an immature nervous system of the newborn, so cold stress is considered a major risk especially to naked preterm baby. Sweating is not an effective mechanism for thermoregulation in full-term or preterm infants for at least several postnatal weeks; this is due to immature autonomic nervous system and functional immaturity of sweat glands **(Fiala and Stohrer, ٢٠٠١).**

The skin surface of neonates tends to be less pigmented for first few postnatal months although melanocytes are actively synthesizing and transferring pigment to epidermal keratinocytes. As a consequence, newborns have less natural protection from sunlight and are more likely to develop sunburn **(Plensdorf and Martinez, ٢٠٠٩).**

Cutaneous lesions of newborn may be benign transient lesions, napkin dermatitis and its related disorders, lesions of infections, blistering dermatoses, genodermatoses or birth marks **(Mallory, ٢٠٠١).**

I) Benign transient lesions of newborn

There are number of benign rashes that occur in infants in transient manner however; they are a major source of parental anxiety **(Chang and Orlow, ٢٠٠٨).**

According to **Paller et al., ٢٠٠٦** transient lesions of newborn is classified into:

١. Transient vascular phenomena:

a) Acrocyanosis
b) Cutis marmorata
c) The harlequin color change

٢. Benign pustular dermatoses:

a) Erythema toxicum neonatorum
b) Transient neonatal pustular melanosis
c) Acropustulosis of infancy
d) Eosinophilic pustular folliculitis

٣. Papulopustular dermatoses:

a) Sebaceous gland hyperplasia
b) Miliaria
c) Milia
d) Neonatal acne

٤. Miscellaneous:

a) Sucking Blisters
b) Subcutaneous fat necrosis of the newborn

١-Transient vascular phenomena

During the first ٢–٤ weeks of life, cold stress may be associated with acrocyanosis and cutis marmorata. Both patterns usually resolve with warming of the skin and recurrence is unusual after ١ month of age (**Paller et al., ٢٠٠٦).**

a) Acrocyanosis is common initially after delivery in the preterm and full term newborn while intervention normally is not required. In acrocyanosis, the hands and feet become variably and symmetrically blue in color without edema or other cutaneous changes (**Chang and Orlow, ٢٠٠٨).**

b) Cutis marmorata is identified by the characteristic reticulated cyanosis or marbling of the skin. This phenomenon is caused by instability or immaturity of the nerve supply to the superficial capillary blood vessels in the skin. This causes the blood vessels in some regions of the skin to dilate, producing a red color of the skin, while other regions are contracting, producing pale skin; hence the marbling appearance of the skin. Cutis marmorata symmetrically involves the trunk and extremities **(O'Connor et al, ٢٠٠٨).** Cutis marmorata telangiectatica congenita (or congenital phlebectasia) may mimic cutis marmorata. However, the lesions are persistent in a localized patch on the trunk or extremities. The eruption may extend in a dermatomal pattern and occasionally widespread lesions and reticulated cutaneous atrophy may be present (**Kienast and Hoeger, ٢٠٠٩)**

c) The harlequin color change is noted when the infant lies horizontally and the dependent half of the body turns bright red in contrast to the pale upper half. The color shifts when the infant is rolled from side to side. This phenomenon lasts from seconds to ٢٠ minutes while recurrences are common until ٣–٤ weeks of life. The cause is unknown but it is not associated with serious underlying disease **(O'Connor et al, ٢٠٠٨).**

٢-Benign pustular dermatoses

Several pustular eruptions must be differentiated from potentially serious infectious dermatoses.

a) Erythema toxicum neonatorum

Erythema toxicum neonatorum is a benign self-limited pustular eruption occurring primarily in healthy newborns in the early neonatal period with high prevalence in males than in females. It occurs in up to ٧٠٪ of full-term infants and in ٥٪ of preterm infants. Although lesions usually appear on the second or third day of life, onset has been reported up to ٢–٣ weeks of age (**Morgan et al., ٢٠٠٩**).

Typically, it begins with ٢–٣ mm erythematous, blotchy macules and papules, which may evolve over several hours into pustules on a broad erythematous base to give a flea-bitten appearance. Lesions may be isolated or clustered on the face, trunk, and proximal extremities and it usually fades over ٥-٧ days without permanent sequelae while recurrences may occur for several weeks **(Treadwell, ١٩٩٧).**

The cause of erythema toxicum neonatorum is unknown. It may be due to inflammatory cells tending to concentrate around hair follicles with cocci-like microbes **(Marchini et al., ٢٠٠٥**). The high frequency (١٥–٢٠٪ of patients) of eosinophilia suggests an allergic basis occurring due to an immediate hypersensitivity reaction to a substance passed transplacentally **(Schwartz and Janniger, ١٩٩٦).**

A Tzanck smear or Gram stain from intralesional contents is essential for diagnosis. Inflammatory cells are present; with greater than ٩٠٪ eosinophils and occasional neutrophils. It is a benign self-limited disorder requiring no treatment, just reassurance of the parents **(Van Praag et al., ١٩٩٧).**

b) Transient neonatal pustular melanosis

Transient neonatal pustular melanosis is a benign, self-limited condition of unknown etiology. Historically, the disorder was called pemphigus neonatorum **(O'Connor et al., ٢٠٠٨).**

Transient neonatal pustular melanosis is characterized by vesicles and superficial pustules ٢–٥ mm diameter which rupture easily leaving a spot of hyperpigmentation with a collarette of fine scale. The lesions are commonly present at birth and are most likely to appear on the chin, neck, forehead, chest, and back. The vesicles and pustules usually resolve within ٤٨ hours, while the brown macules may persist for several months and often it is the only manifestation of the eruption **(Van Praag et al., ١٩٩٧).** Smears from the pustules reveal neutrophils with absence of organisms and no specific therapy is indicated (**Tunzi and Gray, ٢٠٠٧).**

c) Acropustulosis of infancy

Acropustulosis of infancy is a chronic pustular eruption that appears on the palms and soles, but may also involve the scalp, trunk, buttocks, and extremities. Onset may occur during the newborn period or in early infancy. Disease-free periods tend to lengthen until the rash resolves by age of ٢–٣years. During flares, infants are usually fussy and pruritus is severe **(Dorton and Kaufmann, ١٩٩٦).**

Although the cause is unknown in some infants, scabies may precede the eruption. Histopathology of the lesions reveals sterile, intraepidermal pustules. Wright stain shows numerous neutrophils and occasional eosinophils **(Mazereeuw-Hautier, ٢٠٠٤).**

Oral dapsone (١–٣ mg/ kg/day) suppresses lesions and symptoms in ٢٤–٤٨ hours. Brief courses of moderate- to high potency topical corticosteroids to the palms and soles may provide safe temporary relief (**Braun-Falco et al., ٢٠٠١).**

d) Eosinophilic pustular folliculitis (EPF)

Eosinophilic pustular folliculitis or Ofuji disease is a rare, self-limiting condition of unknown etiology. It is characterized by recurrent episodes of ٢–٣ mm follicular white vesicles and pustules on a red base, which are usually found on the scalp and forehead and occasionally, the lesions spread to the trunk. It occurs almost exclusively in boys of age ٥–١٠ months while several neonatal cases have also been described. Although affected infants experience intense pruritus and irritability, EPF is not associated with systemic disease **(Buckley et al., ٢٠٠١).**

Smears from the pustules reveal large numbers of eosinophils with absence of organisms. The strong association of EPF with human immunodeficiency virus infection seen in adults has not been noted in infants. Treatment of Eosinophilic pustular folliculitis is only symptomatic treatment **(Garcia-Patos et al., ١٩٩٦).**

٣) Papulopustular dermatoses

The benign pustular dermatoses may also be confused with several other papulopustular rashes, including sebaceous gland hyperplasia, miliaria, milia and acne **(Wenzel and Horn, ١٩٩٨).**

a) Sebaceous gland hyperplasia

Sebaceous gland hyperplasia is a common finding over the nose and cheeks of full-term infants. Lesions consist of multiple ١–٢ mm yellow papules that result from maternal or endogenous androgenic stimulation of sebaceous gland growth. The eruption resolves spontaneously within ٤–٦ months **(Deplewski and Rosenfield, ٢٠٠٠).**

b) Miliaria

Miliaria is a common disorder of the eccrine sweat glands that often occurs in conditions of increased heat and humidity **(Ale et al., ٢٠٠٩).**

Neonates are thought to have immature eccrine ducts that easily rupture when sweating is induced. Miliaria is classified according to the level at which obstruction of the sweat duct occurs into miliaria crystalline where the duct ruptures superficially, miliaria rubra (prickly heat), where the obstruction occurs in the mid epidermis and miliaria profunda, when the duct ruptures at the dermal–epidermal junction. Miliaria crystalline occurs by age of ١ week and miliaria rubra occurs with a mean age of ١١-١٤ days **(Moosavi and Hosseini, ٢٠٠٦).**

Miliaria crystalline is presented with superficial, ١–٢ mm vesicles on non-inflamed skin. Small papules and pustules are typical presentations of miliaria rubra (prickly heat. Deep-seated papulopustular lesions of miliaria profunda occur rarely in neonates with high risk of heat exhausion **(Wenzel and Horn, ١٩٩٨).**

Treatment of these conditions is warranted. The prevention and treatment of miliaria primarily consists of controlling heat and humidity so that sweating is not stimulated. Measures may involve treating a febrile illness, removing occlusive clothing and providing air conditioning **(Haas et al., ٢٠٠٢).** Topical treatments involve lotions containing calamine, boric acid, or menthol. Topical corticosteroids and topical antibiotics may be needed. Topical application of anhydrous lanolin has resulted in dramatic improvement in patients with miliaria profunda **(Huda and Saha, ٢٠٠٩).**

c) Milia

Milia are very common, benign tiny white papules seen on the face and scalp of the neonate that are usually discrete. They areusually presented at birth or appear subsequently in ٥٠٪ of newborns. They usually resolve within the first month without treatment but they may persist for several months **(Berk and Bayliss, ٢٠٠٨).**

Histology demonstrates epidermal inclusion cysts that arise from the pilosebaceous apparatus of vellus hairs. No topical or systemic medications are effective, so milia can be safely left alone without complication **(Clemons, ٢٠٠٠).**

d) Neonatal acne (Neonatal cephalic pustulosis)

Mild acne develops in up to ٢٠٪ of newborns. Lesions may be present at birth or develop over the first ٢-٤ weeks of life. There is controversy over whether it is truly acne or represents a form of pustular disorder in the newborn period. As a result, the term neonatal cephalic pustulosis has been used instead **(Bergman and Eichenfield, ٢٠٠٢).**

The condition consists of pustules over the cheeks primarily, but may involve other areas of the face and the scalp. Papules and pustules are the main types of acne in newborns. In some cases, the newborn baby is affected by whiteheads (closed comedones) **(Bernier et al., ٢٠٠٢).**

Maternal and endogenous androgens probably play a role in neonatal acne. Some studies implicate infection with Pityrosporum species in some infants. Lesions usually involute spontaneously within ١–٣ months; however, topical application of ٢٪ ketoconazole cream for ١٥ days can be helpful **(Hernane and Ando, ٢٠٠٣).**

٤) Miscellaneous

a) Sucking Blisters

These lesions are present at birth, most often over the dorsal and lateral aspect of the wrist. They may appear like bruises or may be vesicular. The infant is noted to exhibit excessive sucking activity. The absence of lesions in other parts of the body and the well appearance of the baby would rule out pathological disorders presenting with similar lesions **(Wallach, ٢٠٠٣).**

b) Subcutaneous fat necrosis of the newborn

Fat necrosis is a rare, self-limited process that usually occurs in otherwise healthy newborn during the first few weeks of life. Discrete red or hemorrhagic nodules and plaques up to ٣ cm in diameter appear most commonly over areas exposed to trauma, such as the cheeks, back, buttocks, arms and thighs. Lesions are usually painless and resolve without scarring in ١–٢ months. However, lesions occasionally become fluctuant, drain and heal with atrophy **(Mahe et al., ٢٠٠٧).**

Although the cause is unknown there may be a role of mechanical, cold and hypoxic injury to fat**.** Histopathology demonstrates necrosis of fat with a foreign body giant cell reaction. Remaining fat cells contain needleshaped clefts and calcium deposits that are scattered throughout the subcutis **(Fenniche et al., ٢٠٠٤).**

II) Napkin dermatitis (Contact dermatitis) and related disorders

The napkin dermatitis is one of the most common skin disorders in neonates, either due to irritant or allergic causes. Persistent napkin dermatitis may result from other disorders such as seborrheic dermatitis, infantile psoriasis and candidiasis. In particular, these should be considered when the intertriginous areas are involved. Some systemic diseases are manifested by napkin dermatitis e.g. Letterer-Siwe disease and acrodermatitis enteropathica **(Kazaks and Lane, ٢٠٠٠)).**

١) Napkin dermatitis (Contact dermatitis)

a) Irritant contact dermatitis

The napkin area is prone to napkin dermatitis because it is usually bathed in urine and feces and is occluded by plastic diaper covers. Although ammonia was first thought to play a leading role in the pathogenesis of napkin dermatitis, there is an evidence points to feces as the principal cause. Red, scaly, and occasionally erosive irritant reactions are usually confined to convex surfaces of the perineum, lower abdomen, buttocks and proximal thighs while the intertriginous areas are usually spared. Gentle cleansing of the area and the application of lubricants and barrier pastes (e.g. zinc oxide) usually result in clearing of dermatitis. A tapering course of low-potency topical corticosteroid (e.g. hydrocortisone) may speed resolution of in severe dermatitis **(Darmstadt and Dinulos, ٢٠٠٠).**

b) Allergic contact dermatitis

Preservatives and emulsifiers in topical baby products may be associated with allergic reactions in the napkin area. It is less common than irritant dermatitis. Avoidance of the offending agent, aggressive use of emollients and use of low-potency topical corticosteroids may clear these reactions within several days **(Adam, ٢٠٠٨).**

Contact napkin dermatitis is occasionally complicated by secondary infection. The presence of thin-walled pustules on an erythematous base should alert to staphylococcal pustulosis. The lesions rupture rapidly and dry to produce a collarette of scale around a denuded red base **(Prasad et al., ٢٠٠٣).**

٢) Persistent napkin dermatitis

a) Seborrheic dermatitis

Seborrheic dermatitis is characterized by salmon-colored patches with greasy, yellow scales that begin in the intertriginous areas, especially the napkin area, axillae and scalp, thick, adherent scales on the occiput are referred to as cradle cap. Even in severe cases affected infants remain healthy and asymptomatic **(Schwartz et al., ٢٠٠٦).** In the napkin area, red, greasy, scaly patches extend from the skin creases to involve the genitals, perineum, suprapubic area and thighs. Secondary candidiasis or impetigo may mask the underlying process **(Elish and Silverberg, ٢٠٠٦).**

The cause of seborrheic dermatitis is unknown. However, the yeast Pityrosporum has been implicated in seborrhea and identified in the scalp of infants with cradle cap. In addition, this dermatitis is linked to Malassezia due to abnormal immune response of the body. The contribution of Malassezia species to seborrheic dermatitis may come from its lipase activity releasing free fatty acids **(Zisova, ٢٠٠٩).**

Seborrheic dermatitis may clear without treatment by ٢–٣ months of age, but often persists until ٨–١٢ months. Mild keratolytics found in antiseborrheic shampoos (zinc pyrithione, sulfur and salicylic acid) are helpful in the management of cradle cap. Emollients and low-potency topical corticosteroids fasten the resolution of cutaneous lesions. Topical antifungal may be useful particularly in infants with associated candidiasis **(Cohen, ٢٠٠٤).**