

Review

Physiologic Skin Findings of Newborn

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Abstract

Background: Neonatal skin presents many unique features that reflect immaturity and the transition from intrauterine life. Identification of these features is crucial so that we can distinguish them from the pathological ones. In this article we want to discuss common physiologic skin findings of newborn.

The first 30 days of life is named as neonatal period. Skin conditions of newborns that tend to resolve in 30 days considered to be transient. These conditions are very common and almost expected in newborns [1]. The practical issue posed by skin eruptions in newborns relates to the process of ruling out infection. Most transient vesicular or pustular eruptions of newborns are non-infective but the rare newborn with systemic bacterial disease may die if such a disease is not suspected. For this reason newborns should be examined very carefully [2].

Miscellaneous skin lesions that tend to be common in newborns are adopted physiologic. We want to summarize the most common transient skin lesions of newborns.

Vernix Caseosa

During the last trimester of gestation, the fetus is covered by a protective membrane called vernix caseosa. It may cover whole of the body, sometimes only limited to the in-

guinal folds [3]. It functions as a mechanical barrier against bacteria and maceration in amniotic fluid. Vernix caseosa composed of dead epithelial cells, lanugo hairs and sebaceous secretions, lubricates the skin and facilitates passage through the birth canal [4]. Vernix becomes thicker with gestational age approaching term. Interestingly, post-mature infants are usually devoid of vernix [2, 4].

The lipid composition of vernix is measured and highly variable. These lipids are derived from two sources: wax esters formed in sebaceous glands and epidermal barrier lipids derived from keratinocytes. Vernix from term infants contains more squalene and a higher wax ester to sterol ester ratio than vernix from preterm infants. This change reflects increased sebaceous function in fetal skin as term approaches [4, 5].

Vernix is suggested to have an antibacterial and antifungal function since it contains sebum which is known to have these properties. It is demonstrated that vernix provides a mechanical barrier for bacterial passage

and according to this vernix be left on the newborn until it is spontaneously shed [4]. Furthermore, the other physiologic functions of vernix include antioxidant activity, facilitates wound healing and water-resistant effect [6].

The color of vernix can reflect intrauterine problems. Yellow discoloration reflects hemolytic disease, meanwhile yellow-brownish color is frequently seen in post-term infants with bile staining from meconium passage. The odor of vernix is significant, since it can be an early sign of neonatal sepsis [3, 4, 5, 6, 7].

Vernix should be differentiated from the diseases which includes membranes covering the skin like collodion baby [7]. There is no need to treat vernix. It sheds during the first week of life [5].

Cutis Marmorata

During the neonatal period, a transient, benign cutaneous vascular phenomenon occurs, known as cutis marmorata. It can be defined as symmetrical, blanchable, red-blue reticulated mottling of trunk and extremities. It is most common in preterm infants but also affects full-term newborns [4, 5].

Cutis marmorata is thought to be an exaggerated vasomotor response to hypothermia and immaturity of autonomic nervous system facilitates its occurrence [7]. It is a normal physiologic response to ambient temperature changes; accentuates with decreased temperature and improves with re-warming. It is postulated to be caused by increased sympathetic tone with delayed vasodilatation in response to a flux in temperature resulting in dilatation of capillaries and small venules [5].

Histologic findings are normal; therefore, biopsy is not indicated. The differential diagnosis is limited, in cases of prolonged cutis marmorata, several diseases should be considered including trisomy 18-21, hypothyroidism and the *Cornelia de Lange* syndrome [4, 6, 7].

Cutis marmorata telangiectatica congenita (CMTC) should be considered in the differential diagnosis, especially if the mottling is particularly prominent and persistent. The mottling of the skin in CMTC is darker and

more pronounced than cutis marmorata. It does not resolve with warming of the skin and newborns with CMTC usually have telangiectasia, atrophy and ulcerations. Other causes of mottled appearance like livedo reticularis is seen in collagen vascular disease such as neonatal lupus.

In cutis marmorata no specific therapy is required. The aim of the treatment of cutis marmorata is to maintain even temperature of infant and surroundings. It generally resolves spontaneously as vasomotor responses mature by 6 months of age [1, 4, 5].

Acrocyanosis and Erythema Neonatorum

In the first hours of life a central erythema is present in the neonatal skin. This can be in striking contrast to the acrocyanosis that is also seen. Both of acrocyanosis and erythema neonatorum is physiologic skin finding of neonatal period.

Acrocyanosis is characterized by symmetric bluish discoloration of hands and feet. It is intermittent and has no pathological significance. It is usually pronounced on palms, soles and perioral region. Acrocyanosis is more common in term infants than preterm infants.

Acrocyanosis is more pronounced in hypothermia, polycythemia, and other hyperviscosity syndromes. The coloration is ameliorated by warming of the extremities. Acrocyanosis reflects the vasomotor instability related to immaturity.

Cardiovascular and respiratory system diseases associated cyanosis should be distinguished. Central cyanosis seen in the oral cavity is an expected sign in these conditions. Treatment for acrocyanosis is not required. It resolves in the first week of life.

Erythema neonatorum reflects the reflex vasodilatation of cutaneous capillaries related to decreased sympathetic tone. It resolves in 24-48 hours [3, 4, 7].

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 prominent demarcation

on the midline. It was first described by *Neligan* and *Strang* in 1952.

Harlequin color change is unusual vascular phenomenon occurring in low-birth-weight infants. It is more common in preterm (15%) than term infants. The abnormality in the nervous control of the vascular tone is thought to be located at the hypothalamic level. The exact mechanism of this phenomenon is not known but it may be due to autonomic vasomotor control.

Harlequin color change usually occurs on the third or fourth day of life and it can be seen until the 21th day of life. When the newborn is placed on one side, an erythematous color change occurs on the dependent site meanwhile the upper side becomes pale and there is a pronounced demarcation line between them. The location of the color changes with the turns of the baby. The head and the genitalia may be spared.

This phenomenon is believed to be benign and should subside after the third week. If the color change is persistent, a large capillary malformation may be suspected. No treatment is required [1, 2, 3, 4, 7].

Neonatal Desquamation

Desquamation of neonatal skin is the most common finding of neonatal period. It has been reported in up to 65-75% of normal newborns. Post maturity in the neonate leads to increased desquamation.

The pathophysiology is unknown. Scaling is most prominent on the hands, ankles and feet in term infants. In post-terms it may be more generalized and accompanied by other cutaneous signs of postterm including absence of vernix, long nails and hair and decreased subcutaneous fat.

Physiologic desquamation should be differentiated from ichthyosis vulgaris and continual peeling skin syndrome. Persistence of scaling beyond the first few weeks, combined with family history, distribution and appearance of scale lead to the correct diagnosis. Sometimes biopsy is required [2, 3, 4, 6, 7].

Sucking Blisters and Erosions

Sucking blisters are usually solitary, intact blisters or erosions on non-inflamed skin on

the dorsal aspect of forearms, wrists or fingers. These lesions are apparent at birth and believed to result from vigorous sucking *in utero*. They spontaneously resolve without scar formation, usually by the two weeks of life.

To recognize these lesions is crucial since they can be misdiagnosed with more serious diseases like herpes virus infection, bullous impetigo or bullous mastocytosis. Localization, characteristic morphology, failure to develop new blisters helps for correct diagnosis [1, 3, 4, 6, 7].

Lanugo

Newborns are covered with fine unmedullated vellus hairs called lanugo. It is most prominent in preterm infants since the first coat of lanugo hairs normally shed in utero during the last trimester.

Lanugo hairs are more tend to locate on shoulders, posterior trunk and cheeks. They are usually non-pigmented. During the first few months of life they are shed and replaced by vellus hairs.

Congenital hypertrichosis lanuginosa can be mistaken for normal lanugo hair growth in the neonate. The differential diagnosis of increased neonatal hair should include congenital lipodystrophy, *Cornelia de Lange* syndrome and mucopolysaccharidoses [3, 4, 6, 7].

Sebaceous Gland Hyperplasia

Approximately 50% of newborns have sebaceous gland hyperplasia. It is less common in preterm. Multiple, tiny, yellowish papules are seen at the opening of each pilosebaceous follicle in areas in which sebaceous glands are abundant such as nose, cheeks, upper lip and forehead.

Sebaceous hyperplasia results from the influences of maternal androgens on the pilosebaceous follicle. This is a benign condition and resolves spontaneously by 4-6 months of age [1, 3, 4, 7].

Milia

Milia are another common finding on the facial skin of newborns. Milia are multiple, pinpoint white papules representing benign

superficial keratin cysts. They originate from pilosebaceous unit. Milia are caused by retention of keratin within the superficial dermis.

These papules occur prominently on the nose, cheeks and forehead of 40% of newborns. They are expected findings and resolve within a few weeks of life. Differential diagnosis includes neonatal acne, sebaceous hyperplasia and molluscum contagiosum. Large numbers of milia, prolong persistence of milia may be associated with oral-facial-digital syndrome and *Marie-Unna* hypotrichosis.

Milia which are located on oral mucosa along the median palatal raphe, are named "*Ebstein's Pearls*" In a study from India *Ebstein* pearls is found to be the most common skin finding 61%. When they occur on the alveolar margins, they are termed *Bohn's* cysts [1, 2, 3, 4, 5, 7, 8].

Miliaria

The term miliaria is used to describe a group of transient eccrine disorders. These common dermatoses are due to occlusion of the eccrine ducts at various levels, resulting in rupture of the ducts and leakage of sweat in the epidermis and papillary dermis. The level of the obstruction determines the clinical manifestations. Miliaria crystallina, rubra and profunda.

Miliaria crystallina is the most common type in the newborn period. It is characterized by 1-2 mm superficial clear non-inflammatory vesicles and reflects superficial obstruction of the eccrine duct at the level of the stratum corneum. Lesions are most common on forehead and upper trunk. They rupture and desquamate in a few days.

Miliaria rubra (prickly heat) is due to intra-epidermal obstruction due to of the sweat duct. A secondary local inflammatory reaction is responsible for the erythema and papules. Miliaria rubra occurs later than miliaria crystallina. In type I pseudo-hypoadosteronism, pustular miliaria rubra is considered to be a specific cutaneous finding.

Miliaria profunda is rare in newborns. In miliaria profunda eccrine ducts are obstructed at the dermo-epidermal junction.

The differential diagnosis includes erythema toxicum, transient neonatal pustular melanosis candidiasis, neonatal acne and bacterial folliculitis. Minimizing overheating of the newborn is the only treatment. Cool bathing and air conditioning are the best therapeutic measures. In case of superinfection topical antibiotics can be used [2, 7, 9, 10].

Neonatal Acne

Neonatal acne has been described as lesions beginning within the first 30 days of life and manifested by multiple, inflammatory, erythematous papules, comedones, and pustules. It is estimated that 50 percent of newborns may experience neonatal acne.

Neonatal acne may be hormonally mediated because newborns with neonatal acne presents transient increases in circulating androgens. Meanwhile in the neonatal period sebaceous glands are hyperplastic and increased androgenic activity of the glands maybe responsible of neonatal acne development.

However recent studies suggest that many cases may be triggered by infection or colonization of *Malassezia* spp. and the term neonatal cephalic pustulosis has been proposed for this condition. Most cases resolve spontaneously, but severe cases can be treated with topical benzoyl peroxide gel.

It is suggested that newborns with widespread acne lesions may tend to have severe acne problems in adolescent [1, 7, 9].

Erythema Neonatorum Toxicum

Erythema neonatorum toxicum is an idiopathic, common condition seen in up to 75 percent of term newborns. It is rarely seen in pre-terms and low-birth-weight newborns. It is characterized by small erythematous macules with or without a central papule or pustule. It generally occurs in the second day of life. The lesions are tend to locate face, trunk and extremities but spare the palms and soles. The lesions may contain eosinophils and peripheral blood eosinophilia may be associated.

The etiology of erythema neonatorum toxicum remains unknown, intestinal tox-

ins, allergic reactions, mechanical or chemical irritation, hormonal influences on the extracellular matrix and a transient graft-versus-host reaction due to maternal lymphocyte is suggested to be a possible cause.

The diagnosis is usually made by clinical findings but sometimes biopsy may require for the correct diagnosis. The characterization of histologic examination reveals perivascular accumulation of eosinophils in the upper dermis.

The differentiated diagnosis should include transient neonatal pustular melanosis, congenital candidiasis, Staphylococcal impetigo, neonatal herpes simplex. As erythema neonatorum toxicum is self-limited, no treatment is required. It clears spontaneously in up to 4 week of life [1, 2, 5, 7, 9, 10].

Transient Neonatal Pustular Dermatitis

It is a self-limited, benign dermatosis of the newborn and occurs in approximately 0.2-4% of all term newborns. It is more commonly seen in black infants.

Lesions may be present in utero and are almost always present at birth. It is characterized by neutrophil containing pustules or vesicles without surrounding erythema. Lesions can occur anywhere but are common on the forehead and mandibular area.

The lesions have three stages;

1. Fragile 1-5 mm pustules present at birth
2. Resolution of pustules with surrounding fine white collarettes of scale.
3. Hyperpigmented macules represent post inflammatory hyperpigmentation; this stage may not be present in light-skinned infants.

The pathogenesis of transient neonatal pustular dermatosis is unknown. It can be a possible variant of erythema toxicum. Maternal infection, drug use and primary fetal infection have no influence on its occurrence.

The diagnosis is made by clinical findings. Scraping pustule and staining with *Wright* stain reveals many neutrophils and rare eosinophils. Histology of hyperpigmented macules demonstrates basilar hyperpig-

mentation and no dermal melanin; histology of pustules shows intra or subcorneal collections of neutrophils.

The differential diagnosis includes erythema neonatorum toxicum, miliaria, acropustulosis of infancy, candidiasis and Staphylococcal impetigo. No treatment is necessary. Pustules resolve over a few days. Hyperpigmented macules resolve over several weeks or months [1, 5, 9].

Mongolian Spot

The Mongolian spot, a blue-gray or blue-green irregular patch over the sacrogluteal region, is the most common of all birthmarks in pigmented races. Histologically, these spots demonstrate deep dermal melanocytes scattered between collagen bundles.

The pathogenesis of Mongolian spot is not well understood. During the fetal life melanocytes migrate from neural crest to the skin. It is thought that these cells have been arrested in their migration towards the epidermis from the neural crest.

Typically the irregular patch increases in size and intensity over the first year of life and then fades in early childhood [1, 4, 7].

Salmon Patch

Salmon patch is the most common capillary vascular malformation of the neonatal period. It is presented as a pale pink patch on the forehead or nape. It is termed as "angel kiss" when its located on the forehead and named as "stork bite" if it is located on the nape. The majority of these lesions disappear in infancy or early childhood. Some lesions may persist into adulthood. It may cause a cosmetic problem and can be treated with laser therapy [4, 7].

Miniature Puberty

A number of phenomena occur during the newborn period as a result of the influence of placental and maternal hormones. Hyperpigmentation or the linea alba and scrotum and external genitalia is frequent.

After a few days of birth hyperplastic vaginal mucosa desquamates and a whitish, creamy vaginal discharge can be seen. Oc-

asionally on the 3rd-4th days of life with-
drawal bleedings may occur. The engorged
breast tissue can secrete a claustrum-like
substance termed “*witches milk*” [3, 4, 6,
7].

Physiologic Jaundice

In addition to the other color changes of
newborns, neonatal skin frequently ac-
quires a yellow pigmentation usually on the
second day of life. This is common in pre-
term infants up to 80%. The immaturity of
the liver and bilirubin transport mechanism
results in excess unconjugated bilirubin
that accumulates in the skin and produces
physiologic jaundice.

Early onset jaundice may be an early sign of
neonatal sepsis and prolonged jaundice
more than two weeks suggests bilier atresia
and other liver diseases [3, 4, 6].

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