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Skin, Hair, Nail and their Diseases

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Abstract

Full skin care requires special recognition of the natural course of the disease and distinguishing primary and secondary lesions. If the diagnosis is not definitive, aggressive treatment should not be performed. Even when the diagnosis is clear, acute dermatitis may require mild initial treatment and relief. Skin appendages are derived from the accumulation of epidermal cells that specialize early in embryonic development. Primary epithelial germ cells appear in the third embryonic month and are transformed into follicles of the gonads and apocrine glands and connective protrusions to adhere to the hair straightening muscles nail formation begins in the third month of intrauterine life. This article is about the morphology of skin, nail, hair and related disease.

Keywords: Skin; Nail; Hair; Disease

Introduction

Skin morphology

The adult epidermis is a stratified epithelial tissue consisting mainly of keratinocytes. The lower keratinocyte layer of the basal cell layer constantly renews its epidermis with its mitotic divisions. Keratinocyte stem cells come from hair follicles. Individual keratinocytes mature during the differentiation process of the epidermis and from the stratum corneum of the epidermis defense barrier. When the stratum corneum is composed of mature and differentiated keratinocytes, it is 10 µm-50 µm thick. Damage to the stratum corneum increases skin permeability and it may increase the susceptibility to skin and systemic infections or systemic poisoning caused by topical medicines and chemicals. Continuous renewal of epidermis superficial keratinocytes occurs naturally in neat pattern during which cells of the basal layer move upward to the stratum corneum. Cell life is approximately 28 days from basal cell mitotic division until disappearance in the stratum corneum. In highly proliferative diseases such as psoriasis, the cells move faster. Excluding keratinocytes, epidermis contains three other cell types. Melanocytes are pigment producing cells responsible for the color of the skin. Melanocytes produce melanosomes containing melanin. Epidermal melanocytes originate from the neural crest and they migrate to the skin during the embryonic period. They are located in the epidermis between the follicles and the hair follicles and by dividing mitosis or migrating extra cells to the epidermis, their numbers increase. Merkel cells are nerve related epidermal cells that may be important in the sense of touch and development of the skin. Langerhans cells are dendritic cells of the mononuclear phagocyte system. They contain a particular organ called the birbeck granule. These cells originate from the bone marrow and participate in skin immune responses and play an active role as antigen presenters and antigen processors [1].

Hair follicle morphology

The hair follicle is the most dominant structure in the hair collection in the hair area these include sebaceous glands and musculus arrector pili and in the armhole include the apocrine gland. Hair follicles are distributed throughout the surface of the skin except thenar, lips and glans penis and if destroyed, they cannot be rebuilt. Each hair follicle is stretched from the epidermis to the local dermis where the matrix cells with the papillary dermis form onions are pulled by the hair roots to create. The growing hair is made of two parts: Onion and matrix which produce keratinized hair stems. The hair stem is made of the medulla inner, cortex and cuticular layer are made. Hair growth in humans is periodically associated with alternating phases of anagen growth and telogen relaxation. The anagen phase varies from month to year. At birth all hairs are in the anagen phase. Subsequent production activities are not coordinated as such, a pattern of random growth and fall occurs everywhere. Scalp hair typically grows about 1 cm per month. Hair types include: Fetal lango hair, terminal hair, vellus (fluffy) hair. Lango hair is short and thin and falls before birth and fluffy hair replaces it at 36 weeks-40 weeks of gestation. Terminal hair is long and rough and is found on the bearded area, eyebrows, eyelashes, privy parts and axillae. Short fluffy hair is soft and often colorless and diffuses to other parts of the body. During puberty stimulation of androgen hormones causes fluffy hair to become terminal in the areas of pubic symphysis, axilla and beard.

Nails morphology

Nails are special epidermal protective structures that make a transparent, convex and firm plate at the end of the fingers and toes. The nail plate originates from the matrix which is

metabolically active and it is made of proliferating cells below the posterior nail flod and grow at a rate of 1 cm within three months. The nail plate is surrounded by posterior and lateral nail folds and the thin cuticle extends from the posterior nail to the white crescent region called the lunula. The pink color of the nail is a reflection of the blood under it [2].

Literature Review

Diseases skin

Ectodermal dysplasia: It is a rare hereditary disorder with a characteristic physiognomy. It is a genetic disorder affecting the development or function of the teeth, hair, nails and sweat glands. Depending on the particular syndrome ectodermal dysplasia can also affect the skin, the lens or retina of the eye, parts of the inner ear, the development of fingers and toes, the nerves and other parts of the body.

Hypohidrotic ectodermal dysplasia: There are three main features that are displayed in diagnosing hypohidrotic ectodermal dysplasia. They are hypotrichosis, hypohidrosis, and hypodontia. Hypotrichosis is the sparseness of scalp and body hair. The scalp hair has thin shafts and is lightly pigmented, while sexual hair is normal. Hypohidrosis is the reduced ability to sweat and this can lead to hyperthermia. Hypodontia is a congenital absence of teeth. On average, nine permanent teeth develop. Usually the canines form and then the first molars. These teeth are smaller than average and often have conical crowns (Figure 1).

Figure1: Hypohidrotic ectodermal dysplasia.

Hypo pigmented lesions

Albinism: There are several types of congenital ocular albinism, including partial or complete melanin deficiency in the skin of the hair and eyes, despite the number of structures and the normal distribution of melanocytes. Albinism is considered a rare inherited disorder. According to the National Organization for Albinism and Hypopigmentation (NHOAH), about 1 in 20,000 people have some type of albinism in the United States. Albinism occurs because of a defect in the gene that affects melanin production. The result is a reduction in melanin. Since people with albinism cannot produce melanin, they have a lack of skin pigmentation. Their skin and hair appear white, and they may have less pigment in the irises of their eyes [3].

Partial albinism: Partial albinism is inherited as a dominant mendelian characteristic. It consists of patches of depigmentation and usually a white forelock. There is confusion about the disorder and its relation to vitiligo. Dermatologic texts discuss this clinical picture with universal albinism and fail to draw a clear distinction between the two. The latter is usually a recessive trait in which there is a melanin deficit in the retina, iris, and hair, in addition to the skin. There are frequently other associated congenital abnormalities in total albinism but not in partial albinism. Universal albinism is much more common than the partial variety. The term vitiligo should not be applied to partial albinism although microscopically they cannot be differentiated (Figure 2).

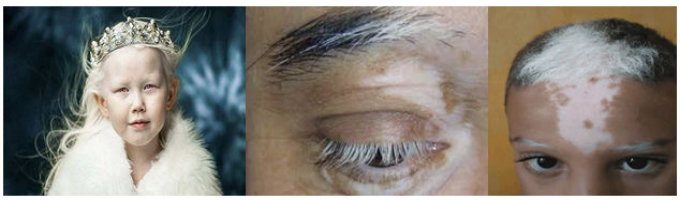

Figure2: Partial albinism condition person inherit because of error for genes code for melanin production inside our body.

Tuberous sclerosis: This disorder is a multi-system disorder that primarily affects ectoderm derived tissue but organs of mesodermal and endodermal origin, especially the eyes, also affect the kidneys and heart. Initially named for the characteristic tuber or potato like growths seen in the brain. Common manifestations include seizures, developmental delay, behavior problems, skin abnormalities, kidney disease. Prevalence of 25,000 to 40,000 patients in USA, and 1 million to 2 million worldwide. Clinical features associated with TSC usually show within the first year of life; however, some may be subtle and insidious in nature, delaying diagnosis until much later. Mutations in TSC1 or TSC2 (chromosome 9) are known causes for TSC. Hamartin (TSC1) and tuberin (TSC2) are proteins that are missing in the disease leading to unregulated loss of tumor suppression. Hamartin and tuberin can be found in the cells of many different organs, thus explaining tumor presentation all over the body (Figure 3).

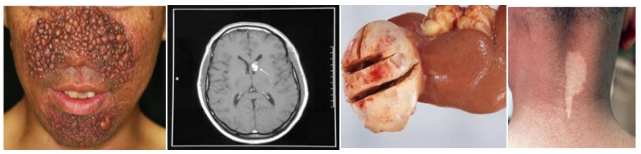

Figure3: Multi system disorder of tuberous sclerosis.

Hypomelanosis of Ito: It is also called incontinentia pigmenti achromians, is a rare birth defect that causes streaked, whirled, or mottled patches of light colored skin. These skin changes often develop within the first two years of life. Other symptoms may include varying degrees of learning disability, seizures, increased body hair, scoliosis, and strabismus. While the exact cause is not known, hypomelanosis of Ito syndrome is strongly linked to its genetics and many patients have chormosomal abnormalities. The disease may be caused by abnormal nerve termination in the involved areas of the skin. Girls tend to be

affected more commonly than boys. Treatment depends on the problems that are presented (Figure 4).

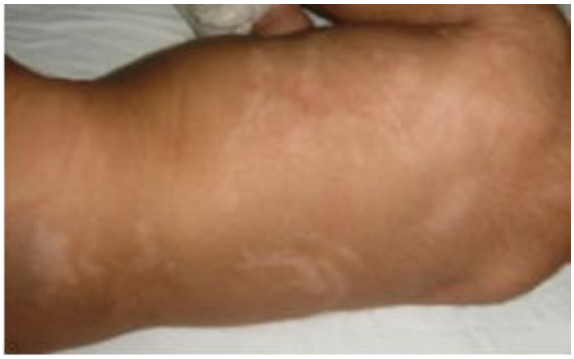

Figure 4: Hypomelanosis of Ito.

Vitiligo: This (vit-ih-LIE-go) is a disease that causes the loss of skin color in blotches. The extent and rate of color loss from vitiligo is unpredictable. It can affect the skin on any part of your body. It may also affect hair and the inside of the mouth. Normally, the color of hair and skin is determined by melanin. Vitiligo occurs when the cells that produce melanin die or stop functioning. Vitiligo affects people of all skin types, but it may be more noticeable in people with darker skin. The condition is not life threatening or contagious. It can be stressful or make you feel bad about yourself (Figure 5) [4].

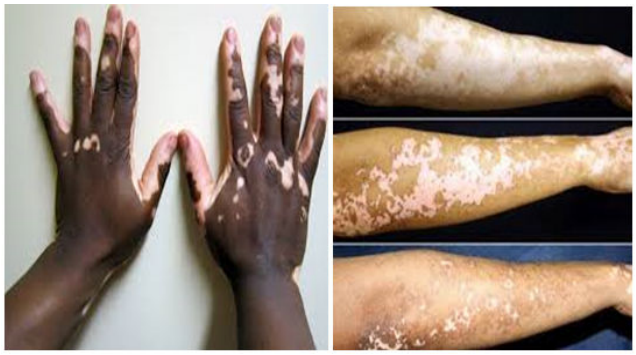

Figure 5: Condition of vitiligo.

Diseases hair

Hair disorders in infants and children may be caused by intrinsic hair growth disorders, underlying biochemical or metabolic defects of inflammatory dermatitis or structural abnormalities of the hair. Hair growth abnormal is called hypertrichosis. True alopecia is rarely congenital, often inflammatory dermatosis is associated with mechanical factors of eating medication endocrine infection, nutritional disorders or hair loss disorder.

Telogen effluvium: Telogen Effluvium (TE) is probably the second most common form of hair loss dermatologists see. It is a poorly defined condition; very little research has been done to understand TE. In essence though, TE happens when there is a change in the number of hair follicles growing hair. If the number of hair follicles producing hair drops significantly for any reason during the resting, or telogen phase, there will be a significant increase in dormant, telogen stage hair follicles. The result is shedding, or TE hair loss (Figure 6).

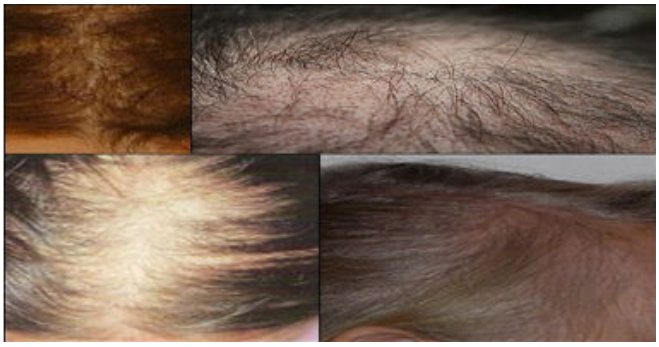

Figure 6: Telogen effluvium condition.

Trichotillomania: Trichotillomania (TTM) is characterized as an impulse control disorder in which individuals fail to resist urges to pull out their own hair, and is associated with significant functional impairment and psychiatric comorbidity across the developmental spectrum. Onset in childhood or adolescence appears to be the norm, yet the research literature involving pediatric samples is particularly sparse. Efficacious treatments have been developed, in particular cognitive behavioral interventions involving procedures collectively known as habit reversal training, yet relapse in adults appears to be common. Recent developments in pharmacotherapies for TTM and in combining cognitive behavioral therapy approaches with medication hold promise, and efforts to examine their relative and combined efficacy are needed. Dissemination of information about TTM and its treatment is a critical next step in the field, since many affected individuals and families cannot find local treatment providers with sufficient knowledge to deliver interventions known to reduce hair pulling behavior (Figure 7).

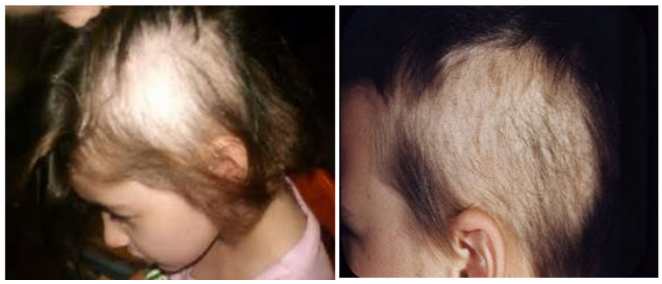

Figure 7: A pattern of incomplete hair loss on the scalp of a person with trichotillomania.

Pili torti: Pili Torti also known as 'twisted hairs' (Latin: Pili=Hair; Torti=Twisted) is a rare, pili torti is a hair shaft disorder characterized by hair that does not grow long and is easily broken; the hair often has a coarse or spangled appearance. A diagnosis is made by light microscopy of flattened hair twisted 1800 along its axis. Although pili torti may be isolated, it is commonly associated with other congenital defects and therefore, if identified, further evaluation for possible neurologic deficits and ectodermal disorders is an important part of the

clinical evaluation. Alterations of the inner root sheath likely lead to the abnormal molding and twisting of the hair shaft. More recent research suggests that these alterations may occur in the face of mitochondrial dysfunction and may be influenced by the presence of reactive oxygen species (Figure 8) [5].

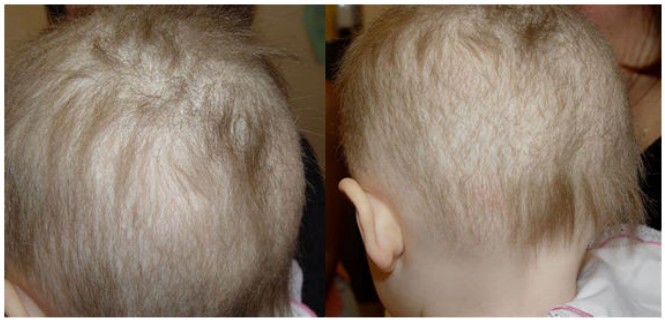

Figure 8: Condition of pili torti.

Diseases nail

Nail disorders in children may be a manifestation of a generalized disease of a skin condition confined to the side of the nail of a systemic disease.

Onycholysis: Onycholysis occurs when the nail separates from the nail bed or the skin directly beneath the nail. There are several possible causes for this common condition. Onycholysis can last for several months and will typically correct itself when the nail fully grows out. Until then, the nail will not reattach to the skin beneath it. Recovery time varies for onycholysis as it is largely dependent on nail growth. It is not uncommon for fingernails to take about 4 months to 6 months to grow to a full size. Toe nails may take up to 8 months. Injury to the nail can cause onycholysis. Wearing tight shoes can cause injury. The condition can also result from an allergy to products used on the nail, like chemical nail polish remover or artificial nail tips (Figure 9) [6].

Figure 9: Condition of onycholysis.

Yellow nail syndrome: Yellow nail syndrome is a rare condition that affects the fingernails and toenails. People who develop this condition also have respiratory problems and lymphatic system problems with swelling in the lower parts of their body. Swelling is caused by a buildup of lymph under the soft tissue of the skin. Lymph is a colorless fluid that circulates throughout your body and helps cleanse it. Yellow nail syndrome can occur in anyone, but usually occurs in adults over the age of 50. This condition can start sporadically for no apparent reason, which occurs in most cases. Even so, in rare cases, it's believed it may run in families. A mutation of the *FOXC2* gene which causes a disorder called lymphedema distichiasis syndrome may play a role in developing yellow nail syndrome. More research is needed to confirm this, as other literature currently reports that there is no known genetic factor for yellow nail syndrome. Yellow nail syndrome can also develop on its own or occur with certain types of cancers, autoimmune diseases like rheumatoid arthritis, and immunodeficiency's. Yellow nail syndrome is when nails gradually turn yellow and thicken. Symptoms also include: The loss of the cuticle, which is part of the protective skin covering the Nail:

- Nails that curve
- Nails that stop growing
- Nails that separate from the nail bed
- Nail loss

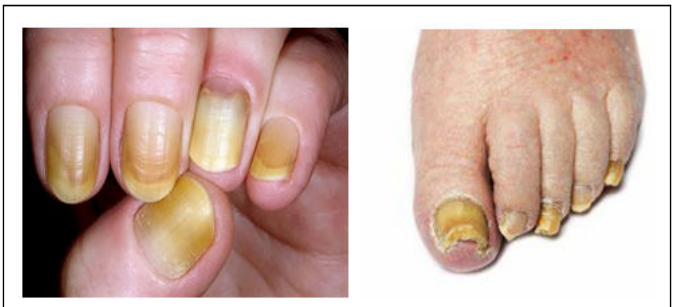

Figure10: Yellow nail syndrome.

Acrodermatitis enteropathica: Epidermolysis Bullosa (EB) (ep-ih-dur-MOL-uh-sis buhl-LOE-sah) is a group of rare diseases that cause fragile, blistering skin. The blisters may appear in response to minor injury, even from heat, rubbing, scratching or adhesive tape. In severe cases, the blisters may occur inside the body, such as the lining of the mouth or the stomach. Most types of epidermolysis bullosa are inherited. The condition usually shows up in infancy or early childhood. Some people don't develop signs and symptoms until adolescence or early adulthood. Epidermolysis bullosa has no cure, though mild forms may improve with age. Treatment focuses on caring for blisters and preventing new ones [7]. The hallmark cutaneous features of inherited EB are: Mechanically fragile skin and easy inducibility of blisters or erosions some or all of the following:

- Milia
- Nail dystrophy or absence
- Dental problems such as tooth decay
- Scarring (usually atrophic)

Discussion

Additionally useful findings, if present, include exuberant granulation tissue, localized or confluent keratoderma of the palms and soles, and altered pigmentation [8]. Infrequently seen and extremely non-specific cutaneous findings include decreased or absent hair, albopapuloid lesions (flesh colored or hypo pigmented papules, usually arising on the lower trunk), and hypo/hyperhidrosis (Figure 11) [9,10].

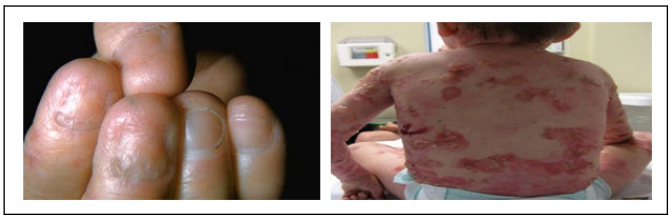

Figure 11: Epidermolysis Bullosa (EB) (ep-ih-dur-MOL-uh-sis buhl-LOE-sah).

Conclusion

Dermatologic manifestations of systemic diseases produce distinctive and reliable diagnostic clues that hasten time to intervention and improve overall outcomes. Notably, nutritional deficiencies are a class of diseases with representative and well established dermatologic associations. Nutritional deficiencies can be primary or secondary and genetic or acquired; however, mucocutaneous findings remain characteristic irrespective of analysis.

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Conflict of Interests

The author declares that they have no competing interests.

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