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Chapter 14

Hair Loss in Children, Etiologies, and Treatment

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Abstract

Hair loss in children is a common and important complaint in dermatology and pediatric clinics and can be considered as a difficult and challenging problem in some cases. Early management is needed as this has its effect on development of normal mental and physical growth of children. Alopecia or hair loss is of particular concern in pediatric group, as it is associated with more significant psychological consequences in this growing age group and has patterns that are different from that seen in adults. There are common and uncommon causes for this complaint, and this usually covers a broad differential diagnosis. This chapter has been written in an attempt to distinguish the types of hair loss (acquired and congenital, common and uncommon), to facilitate the diagnostic causes of this problem, and finally to reach a proper treatment.

Keywords: children, hair loss, etiology

1. Introduction

Hair loss in children is a common and important complaint in dermatology and pediatric clinics and can be considered as a difficult and challenging problem in some cases. This problem of irreversible hair loss can be a worrying one for parents and kids. In addition, the diagnosis may be a challenge for the doctors to reach a proper diagnosis and therapy for their patients. Early management is needed as this has its effect on development of normal mental and physical growth of children.

Alopecia or hair loss is of particular concern in a pediatric group, as it is associated with more significant psychological consequences in this growing age group and has patterns that are different from that seen in adults.

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Similar to hair loss in adults [1], this problem can be caused by a number of conditions, but in patterns that are different in frequencies and presentations from those seen in adults. There are common and uncommon causes for this complaint, and this usually covers a broad differential diagnosis [2]. This chapter has been written in an attempt to distinguish the types of hair loss (acquired and congenital, common and uncommon), to facilitate the diagnostic causes of this problem, and finally to reach a proper treatment.

2. Normal hair growth

To fully understand hair loss during childhood, a basic comprehension of normal hair growth is necessary [3, 4]. Generally, at 22 weeks of intrauterine life, the developing fetus has all of its hair follicles formed. At this stage, there are nearly about one million of hair follicles on the head. Hair on the scalp grows about 0.3–0.4 mm/day or about 6 inches/year.

At any given time, a random number of hairs will be in one of three stages of growth and shedding: anagen, catagen, and telogen. Each phase has specific characteristics that determine the length of the hair. All three phases occur simultaneously; one strand of hair may be in the anagen phase, while another is in the telogen phase.

Essentially, there are three basic groups of hair based on hair follicle size [5].

(A) Lanugo hair is long, unpigmented, and very fine, and the very first hair fiber to be produced is by a hair follicle. As an embryo develops, the hair follicles form and begin to produce this type of hair. This first wave of growth is normally shed by the embryo at around 8 months gestation and replaced by terminal or vellus hair ready for birth. However, sometimes babies can be born with this coat of lanugo hair (called "congenital hypertrichosis lanuginosa").

(B) Vellus is short, fine, unpigmented hair. This type of hair is commonly seen on the nose and over the cheeks.

(C) Terminal, it is long, coarse, pigmented, and frequently contains a medulla. During puberty, many hair follicles around the genitals, armpits, beard, and chest in men transform from vellus hair to terminal hair under the direction of hormones. Equally, hormones can cause terminal hairs to revert to vellus hair production as in androgenetic alopecia.

3. Congenital causes of hair loss

Congenital hair loss is a loss of hair that is present at birth; the following conditions are some of possible causes of congenital hair loss.

3.1. Nevus sebaceous of Jadassohn

Nevus sebaceous of Jadassohn (**Figure 1**) (also known, as sebaceous nevus) is a yellow-orange, waxy, and hairless plaque that typically occurs on the scalp [6]. Such nevi are present

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Figure 1. This is a 5-year-old male patient presented with yellow plaque, waxy on the scalp without hair growth. This plaque was since birth.

at birth or early childhood as a congenital plaque without hair but at the age of puberty show remarkable overgrowth (due to the activity of sebaceous glands at this age). Hair follicles are not present within the lesion itself, but lesions on the scalp may be covered over by surrounding hair, so careful examination is important. Skin growths such as benign tumors and basal cell carcinoma can arise in sebaceous nevi, usually in adulthood. Rarely, sebaceous nevi can give rise to sebaceous carcinoma.

3.2. Aplasia cutis congenita

Aplasia cutis congenita (also known as "Cutis aplasia," "Congenital absence of skin," and "Congenital scars") is a heterogeneous group of disorders characterized by the absence of a portion of skin in a localized or widespread area at birth [7, 8]. The defect may involve only the epidermis and upper dermis (localized and noninflammatory defect) resulting in minimal alopecia, or it may extend into the deep dermis, subcutaneous tissue, or rarely periosteum, skull, and dura (especially in the deeper and lager one). This deep and severe form of aplasia cutis can be associated with a neural cranial tube defect (encephalocoele or meningocoele), which can be demonstrated by an ultrasound scan showing misplaced brain tissue outside the skull. It is the most common congenital cicatricial alopecia and manifests as a solitary defect on the scalp in 70% of cases, but it may sometimes occur as multiple lesions. Most lesions of aplasia cutis congenita occur on the scalp vertex just lateral to the midline, but defects may also occur on the face, trunk, or limbs, sometimes symmetrically.

3.3. Ectodermal dysplasia

Ectodermal dysplasias are described as "heritable conditions in which there are abnormalities of two or more ectodermal structures such as hair, teeth, nails, or sweat gland function, in addition to another abnormality in a tissue of ectodermal origin, e.g., ears, eyes, lips, mucous membranes of the mouth, or nose, central nervous system" [9]. It is not a single disorder, but a group of syndromes. The signs and symptoms of ectodermal dysplasia differ markedly

between the different types of the condition and depend on the structures that are affected. Signs and symptoms are not usually apparent in newborns and may not be picked up till infancy or childhood. The affected individuals have abnormalities of the hair follicles (the scalp and body hair may be thin, sparse, and very light in color). The hair may grow very slowly or sporadically, and it may be excessively fragile, curly, or even twisted (**Figure 2**).



Figure 2. A child presented with fragile, curly hair since birth; the main complaint was persistent short and fragile hair. This child had also cone-shaped teeth and scanty eyebrows.

3.4. Hair shaft defects

Hair shaft abnormalities are characterized by changes in color, density, length, and structure [10]. Hair shaft alterations often result from structural changes within the hair fibers and cuticles, which may lead to brittle and uncombable hair.



Figure 3. A female child with loose anagen hair syndrome. It is prominent that the hair was relatively sparse and does not grow long. Hair is of fair color, and hair shafts were of reduced caliber.

Hair shaft defects may result in dry and lusterless hair, coarse or fizzy hair, uncombable hair, and fragile hair. Hair shaft diseases may occur as localized or generalized disorders.

3.5. Loose anagen hair syndrome

Loose anagen syndrome is a benign, self-limiting condition where anagen hairs are easily and painlessly extracted and mainly reported in childhood [11]. It can be seen in normal population and in alopecia areata. The hair is relatively sparse and does not grow long. Hair is of fair color and hair shafts of reduced caliber, and an early age of onset are features. Usually, the hairs are not fragile, and there are no areas of breakage (**Figure 3**).

4. Acquired causes of hair loss

Common causes [12–15] of hair loss in children include telogen effluvium, tinea capitis, bacterial infections, traction alopecia, trichotillomania, and alopecia areata. In addition to the previous, other less common causes of hair loss can be seen including thyroid disorders and illnesses, such as systemic lupus erythematosus, diabetes mellitus, or iron deficiency anemia, malnutrition, structural abnormalities of the hair shaft that usually result in easy breakage and dry brittle hair. Hair types are influenced by ethnic groups that vary from region to region, and subsequently, this may reflect itself on the variation of common and uncommon causes of hair loss. This usually covers a broad differential diagnosis, and correct diagnosis, specific environmental and cultural factors may reflect itself on the prevalence of specific types of hair loss in children.

4.1. Common causes

There are five common types of hair loss in children: alopecia related to tinea capitis, alopecia areata, traction alopecia, telogen effluvium, and trichotillomania/trichotillosis.

4.1.1. Tinea capitis

Tinea capitis (ringworm of the scalp) is one of the more common causes of hair loss [16]. It is a disease caused by superficial fungal infection (superficial mycosis or dermatophytosis) of the skin of the scalp, with a propensity for attacking hair shafts and follicles. Tinea capitis is the most common pediatric dermatophyte infection worldwide [15].

Tinea capitis may present in several ways such as:

1. Dry scaling—resembling seborrheic dermatitis, but usually with moth-eaten hair loss (**Figure 4**).
2. Black dots—the hairs are broken off at the scalp surface, which is scaly, smooth areas of hair loss (**Figure 5**).

3. Kerion—severely inflamed deep abscesses.
4. Favus—yellow crusts and matted hair.
5. Carrier state with no symptoms and only mild scaling (*Trichophyton tonsurans*).

The potential of scarring and permanent alopecia is common in tinea capitis if left untreated (**Figure 6**).

Regarding the in vivo hair invasion in tinea capitis, there are three recognized types:

1. Ectothrix invasion is characterized by the development of arthroconidia on the exterior of the hair shaft and usually fluoresces a bright greenish-yellow color under a Wood lamp ultraviolet light. Common agents include *Microsporum canis*, *Microsporum gypseum*, *Trichophyton equinum*, and *Trichophyton verrucosum*.
2. Endothrix invasion results from infection with *T. tonsurans*, *Trichophyton violaceum* and *Trichophyton soudanense*. The hair shaft is filled with fungal branches (hyphae) and spores and usually does not fluoresce with Woods light. All endothrix-producing agents are anthropophilic (e.g., *T. tonsurans*, *T. violaceum*).
3. Favus, usually caused by *Trichophyton schoenleinii*, produces favus-like crusts or scutula and corresponding hair loss.

4.1.2. Alopecia areata

It is an autoimmune disease in which hair is lost from the scalp (**Figure 7**) or other hairy areas such as eyebrows, eyelashes, and other hairy areas in the body. It often results in bald spots on the scalp, especially in the first stages [17]. Rarely, the condition can spread to the entire scalp (alopecia totalis) or to the entire skin (alopecia universalis) (**Figure 8**). This type of alopecia is characterized by nonscarring alopecia (no fibrosis or inflammation), where the hair shafts are gone, but the hair follicles are preserved, making this type of alopecia reversible. Typically,



Figure 4. Tinea capitis in child presented as seborrheic dermatitis.

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Figure 5. Tinea capitis in child with black dots presentation. There is no inflammation or no scales.



Figure 6. A child with scarring alopecia secondary to untreatable tinea capitis.

the patient first presented with small bald patches. The underlying skin is unscarred and looks superficially normal. These patches can take many shapes but are most usually round or oval. The disease may also go into remission for a time or may be permanent. It is common in children. Exclamation point hairs, narrower along the length of the strand closer to the base, may present and represent an activity of the disease.

4.1.3. Traction alopecia

Traction alopecia is a gradual hair loss, caused primarily by frequent and chronic hair pulling (**Figure 9**), and this is usually due to habit of hair styling [18]. It is also seen occasionally in longhaired people who use barrettes to keep hair out of their faces. There is a large variation



Figure 7. Small patch of alopecia areata; the skin is normal with no scales or no erythema.

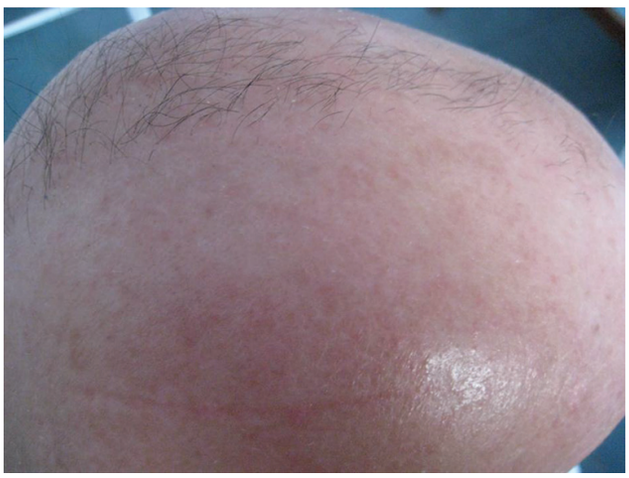


Figure 8. Alopecia totalis with prominent exclamation marks.

in the pattern of clinical presentation of traction alopecia. If there is no suspicion of traction, it can be difficult to diagnose. Patients may present with (itching, redness, scaling, folliculitis or pustules, multiple short broken hairs, thinning, and hair loss). At a later stage, vellus hairs (fine short hairs) develop and terminal hair follicles reduce and are replaced by fibrotic fibrous tracts (scars).

4.1.4. Telogen effluvium

This is not an uncommon cause of hair loss in children; it refers to an abnormality of the normal hair cycle leading to excessive loss of telogen hair [19]. In telogen effluvium, many factors happen to interrupt the normal life cycle of hair and to throw many or all of the hairs into the telogen phase. After few weeks of the insult, partial or complete baldness appears. Frequent

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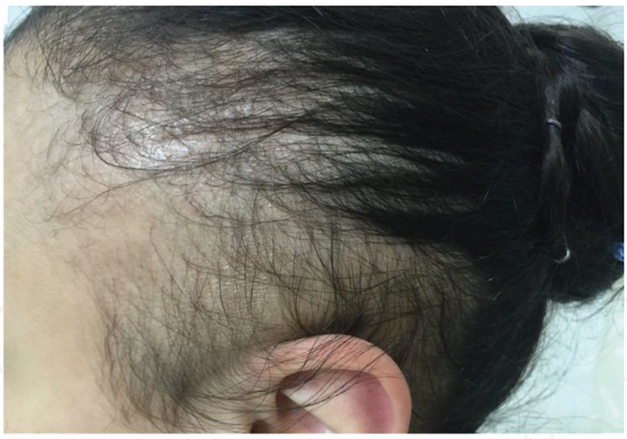


Figure 9. Traction alopecia.

triggers include physiologic effluvium of the newborn, in this type; babies often lose their hair during the first 6 months. Similar to adult type of telogen effluvium, many different events can cause telogen effluvium, including extremely high fevers, severe previous illnesses, surgery under general anesthesia, severe prolonged emotional stress, severe injuries, and the use of certain prescription medication.

4.1.5. Trichotollimenia

Trichotollimenia (**Figure 10**) is defined as a child or a teen that compulsively pulls out her hair and is thought to be related to obsessive-compulsive disorder [20]. These children have noticeable hair loss and often need treatment from a child psychiatrist and/or a child psychologist who specializes in trichotillomania. The hair loss is patchy and characterized by broken hairs of varying length. Patches are typically seen on the side of the child's dominant hand.



Figure 10. Trichotollimenia. The hair loss is patchy and characterized by broken hairs of varying length.

4.2. Uncommon causes

Rarer reasons for alopecia in children include pressure-induced alopecia, alopecia related to nutritional deficiency or toxic ingestion, and androgenetic alopecia. Other causes such as lichen planopliaris, chronic skin inflammation, universal pruritus, and severe dehydration.

5. Diagnosis of hair loss in children

In an attempt to facilitate the diagnosis of hair loss in children, it is helpful to have a proper history from the parents; the key points in patient's history are age of onset of the patient; onset of hair loss: sudden or gradual; extent of alopecia: patchy or diffuse; associated symptoms; mental development; emotional triggers in the previous few months; and any accompanying complaints (e.g., fatigue, weight changes, and nail or skin abnormalities); past medical history (including chronic illnesses, surgeries, medication, autoimmune); family history of alopecia, autoimmune disease, dermatologic or psychiatric disorders; hair-grooming practices (chemicals, tight braiding) [21, 22]. Thorough examination of scalp as well as other hair-bearing areas of the body is another key factor in diagnosis of hair loss. The examination should have the following components: type of hair loss: localized or diffuse; scarring or nonscarring; any hair shaft abnormalities; exclamation marks; hair texture and fragility; presence of pustules, scales, and erythema. Clinical examination of the entire body is necessary to evaluate hair loss, including teeth, skin, and mucous membranes.

The activity of hair shedding can be evaluated by hair pull test in which approximately 20 hairs are grasped and firmly tugged away from the scalp and then the number of extracted hairs is counted. Normally, fewer than three hairs per area should come out with each pull. If more than 10 hairs are obtained, the pull test is considered positive. The root of the plucked hair can be examined under a microscope to determine the phase of growth and is used to diagnose a defect of telogen, anagen, or systemic disease. Telogen hairs have tiny bulbs without sheaths at their roots. When the diagnosis of hair loss is unsure; a biopsy allows for differing between scarring and nonscarring forms. Skin biopsies are taken from areas of inflammation, usually around the border of the bald patch.

Further investigations are needed according to the suspected cases of tinea capitis, alopecia areata, or telogen effluvium. In tinea capitis, the diagnosis should be confirmed by microscopy and culture of skin scrapings (a potassium hydroxide preparation); woods lamp examination: as screening to detect flourescing species. When telogen effluvium is suspected, and there is no obvious trigger of telogen effluvium, blood tests are needed and include complete blood count; serum ferritin; serum zinc, antinuclear antibody; and thyroid function test.

Newly, dermoscope (trichoscope) [23, 24] is a noninvasive method of examining hair and scalp. It allows differential diagnosis of hair loss in most cases, especially in cases of hereditary hair shaft abnormalities. In the last few years, many studies have been published in this field. It may be performed with a manual dermoscope (×10 magnification) or a videodermoscope (up to ×1000 magnification). In particular, trichoscopy enhances the diagnosis of

androgenetic alopecia, alopecia areata, telogen effluvium, trichotillomania, and congenital triangular alopecia, scarring alopecia, tinea capitis, and hair shaft disorders.

6. Treatment

For the majority of the cases of hair loss in children, a dermatologist would be able to diagnose these conditions and prescribe the appropriate treatment. But some hair disturbances have no effective treatment, and for others, no single treatment is 100% successful. Congenital and hereditary hypotrichosis and hair shaft abnormalities often have no effective treatment.

For tinea capitis, treatment usually involves systemic antifungal therapy, such as griseofulvin, which is taken by mouth for 8 weeks. Tinea capitis is also treated with antifungal shampoo to decrease shedding of fungus, which is used to wash the scalp 2–3 times a week. It is very important to continue the use of the oral medication and shampoo for the entire 8 weeks. Children who have tinea capitis are not required to leave school if treatment is used as directed but should be careful not to share any objects that touch the heads such as hats and pillows. Most children are not contagious when using the oral medication and shampoo.

Alopecia areata is an unpredictable disease, and even with complete remission, it is possible for it to occur again throughout your child's lifetime. While there is no cure, and unfortunately since there is no FDA-approved drugs specifically designed to treat the disease in some children. Many have their hair back within a year, although regrowth is unpredictable and many will lose hair again. The treatment of alopecia areata [25] depends on the severity of involvement. If the disorder is mild and does not cause the patient very much distress, waiting for a spontaneous remission is a sensible option. Treatment with zinc as a putative immunemodulator generally has no side effects and is, therefore, suitable for use in children. Topical and systemic immunomodulators are currently being employed for treating alopecia areata, but their efficacy against alopecia areata has not been established. Children with permanent hair loss can be offered surgical hair transplantation or camouflage devices, such as wigs.

In trichotillominea [26], counseling and psychotropic drugs such as clomipramine or sertraline, N-acetyl cysteine, and behavior modification techniques (e.g., habit-reversal therapy) are effective treatment options. These are novel therapeutic agents found to be effective in trichotillomania.

Traction alopecia [27] is a reversible alopecia and cessation of the offending hair practice is the treatment. But if the traction is continued over years, mechanical damage to hair follicles may result in permanent hair loss.

In telogen effluvium, assuming there is no intervening pathological process, the loss is usually replaced in 6–12 months. Treatment revolves around addressing the underlying cause

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