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Literature Review
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Diagnosis and Management of Hair Loss in Pediatric

Fitriani Fitriani, Cayadi Sidarta Antonius, Soenarto Kartowigno, Sarah Diba, Fifa Argentina

Department of Dermatology and Venereology, Faculty of Medicine Universitas Sriwijaya/Dr. Mohammad Hoesin General Hospital, Palembang – Indonesia

ABSTRACT

Background: Hair loss can occur in pediatric and adult populations and may have different patterns. The causes of hair loss in pediatric patients include tinea capitis, alopecia areata, telogen effluvium, traction alopecia, anagen effluvium, androgenetic alopecia, loose anagen syndrome, short anagen syndrome, congenital aplasia cutis, congenital triangular alopecia, atrichia congenita, congenital hypotrichosis, and transient neonatal hair loss.Purpose**:** to understand etiologies of hair loss in pediatrics, and to determine the appropriate examinations for diagnosing and managing hair loss in children.**Review**: Hair loss in children can be categorized as congenital or acquired. Congenital hair loss is classified based on distribution, while acquired hair loss is classified as either scarring or non-scarring. Hair loss complications in children can be irreversible. The diagnosis of hair loss in pediatric patients includes anamnesis, physical examination, and supporting examinations. The supporting examinations used for diagnosis include fungal cultures, hair pull tests, hair tug tests, light microscopy, and trichoscopy. Conclusion: There are numerous causes of hair loss in pediatric patients that need to be identified before appropriate management can be implemented. The management of hair loss requires a holistic approach, including psychosocial support. Correct diagnosis and treatment of pediatric hair loss can prevent a decrease in the patient's quality of life.

Keywords: pediatric, diagnosis, hair loss, management.

Correspondence: Fitriani Fitriani, Department Dermatology and Venereology Universitas Sriwijaya, Jl. Jendral Sudirman Km. 3.5, Sekip Jaya, Kecamatan Kemuning, Palembang Indonesia, Telephone: +62 81994935475, Email: fitrianihasan@gmail.com

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BACKGROUND

Hair loss is a common disease that can occur due to various factors, including hormonal imbalances, age, autoimmune conditions, drug use, and genetics.[1] On average, people lose 100-200 strands of hair per day.[2] Children may experience different patterns of hair loss compared to adults, such as androgenetic alopecia (AGA).[2–4] Studies have shown that hair and scalp disorders in pediatric dermatology account for 3-7.5% of cases.[2–4] A study conducted at Sardjito National Hospital from 2015 to 2019 reported that18.4% of alopecia areata patients were under 18 years old.[5]

In children, hair loss can be classified as congenital or acquired, abnormalities in the hair shaft and follicles can cause it. Congenital hair loss can be present in healthy children or be associated with ssystemic disorders. Acquired hair loss can also result from infections. Both congenital and acquired hair loss can lead to irreversible damage to the hair follicles and the formation of fibrous tissue.[6]

Furthermore, hair loss in children can have a significant psychological and emotional impact on both the child and their parents. It can cause feelings of inferiority and difficulties in conforming to socio-cultural norms.[7] Timely diagnosis and appropriate treatment of hair loss in children can help prevent irreversible alopecia and mitigate its psychological effects.[2]

The purpose of writing a literature review is to discuss hair loss in children, especially in making a diagnosis. Diagnosis and proper treatment are expected

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to prevent complications and psychological effects that can occur due to hair loss in children.

REVIEW

The clinical approach to diagnosing hair loss in children is based on its underlying causes. Congenital hair loss can be categorized according to distribution, while acquired hair loss can be classified as either scarring or non-scarring.[6] Non-scarring alopecia results from damage to the hair follicles and can cause permanent hair loss if left untreated.[8] Common types of hair loss in children include tinea capitis, alopecia areata (AA), trichotillomania, traction alopecia, anagen effluvium, and androgenetic alopecia (AGA). [6]

One of the most prevalent causes of hair loss in children is tinea capitis, which is a scalp infection caused by dermatophytes. The incidence of tinea capitis in school-age children is 19.4%. Clinical manifestations range from diffuse scalp hyperkeratosis without hair change to circular patches of alopecia with hyperkeratosis, erythema, and itching. Tinea capitis with black dots appears as small black dots in the area of hair loss, indicating hair shaft damage.[2,9] If left undiagnosed and untreated, tinea capitis can progress to a kerion, which presents as plaques or nodules accompanied by pustules and crusts. Kerion often causes severe pain in children.[2]

Treatment for tinea capitis involves systemic antifungals such as griseofulvin, terbinafine, fluconazole, or itraconazole. In cases of kerion, short-term systemic corticosteroids are added for 2-4 weeks. The use of oral steroids reduces the risk of permanent alopecia.[2] To prevent reinfection, combs, hairbrushes, and hats should be disinfected or replaced, and family members should be examined and treated as necessary.[10] Alopecia areata (AA) is a non-scarring type of hair loss caused by autoimmune and inflammatory factors. The incidence of AA is 0.7-4%, with a relatively higher prevalence in individuals under 30 years old.[11] Approximately one-third of all AA cases are seen in children. While alopecia areata in infants is rare, research indicates that it can affect 1-2% of patients under 2 years old and 21-24% of children under 16 years old.[9] In children, AA can present as patchy, reticular, ophiasis, sisaihpo, diffuse, totalis, or universalis patterns.[12]

The management of AA depends on its severity and the patient's age. The treatment goal is to control hair loss and prevent its progression, usually requiring 3-4 months of treatment.[2] In children with patchy AA, hair typically regrows within 1 year even without treatment, so periodic observation is recommended.[9] For more extensive forms of AA, such as alopecia totalis or alopecia universalis, topical corticosteroids are generally ineffective. Systemic methotrexate and glucocorticoid therapy may be considered for patients with severe symptoms and significant psychological distress.[2] Anthralin 0.5-1% topical treatment can be used once daily to twice weekly in children with AA, with a response rate of 42%. However, this treatment has a high recurrence rate of over 50%. Significant hair regrowth usually requires a minimum of 3 months of therapy, with the maximum effect seen after 12 months of treatment. Anthralin is recommended for children under 3 years old and is considered a good alternative to topical steroids for chronic AA.[13]

Telogen effluvium (TE) is a non-scarring form of hair loss that can occur in individuals of all ages, both males and females. Hair loss due to TE typically occurs 3-5 months after a triggering event, such as physical or emotional stress.[14] In children, clinical manifestations of TE include hair loss throughout the entire scalp. Although hair thinning may not be evident upon physical examination, there is a history of increasing intensity of hair loss. The hair pull test is positive for hair loss across the entire scalp, not limited to specific areas. Assessing the amount of hair lost each day, such as by collecting hair on a pillow or in the shower, can help estimate the percentage of hair in the telogen phase. Normally, about 15% of scalp hair is in the telogen phase, but this can increase to over 25% in TE cases. Currently, there is no specific treatment for TE, and addressing the underlying cause is the main approach. Improvement in TE usually occurs after 3 months.[2]

Trichotillomania is a disorder where individuals can't resist the urge to pull or twist their hair. It typically starts around age 13 and affects approximately 4% of the population, with the highest incidence in childhood and adolescence. Diagnosing trichotillomania can be challenging if patients are not honest about their hair-pulling behavior.[14] Clinical features include focal hair loss on the frontoparietal or other areas of the head and uneven, twisted, or broken hair. Complications may include infection, skin damage, and permanent scarring.[14] Trichotillomania can sometimes be difficult to distinguish from alopecia areata (AA), as their clinical presentations may overlap. AA can be the initial trigger for trichotillomania, and both conditions can coexist.[15] Further observation is necessary for diagnosing trichotillomania. In children, it often improves on its

own, while treatment for adolescents is more challenging, requiring cognitive-behavioral therapy and pharmacological treatment under the care of a psychologist/psychiatrist. Combining these therapies is expected to be more effective in reducing the likelihood of relapse.[10]

Traction alopecia occurs due to physical trauma, often resulting from prolonged tension on the hair caused by styling practices like tight ponytails or the use of hair rollers. It is more common in black women and children, particularly those with afro-textured or finely curly hair. Clinical manifestations include hair thinning, reduced hair density, perifollicular erythema, short hairs, hair breakage, folliculitis, and follicular papules. Chronic traction can lead to perifollicular scarring, resulting in scarring alopecia commonly found along the hairline. Early stages of traction alopecia can be managed by changing hairstyles.[9] A characteristic sign of traction alopecia is the fringe sign, which indicates hair retention along the frontal or temporal edges. Excessive tension can cause hair follicles to be pulled out of the scalp.[6]

Anagen effluvium is severe hair loss that occurs during the anagen phase, which accounts for approximately 90% of normal scalp hair. Hair loss typically happens within 1-2 weeks after exposure to triggers.[9] Common causes of anagen effluvium include radiotherapy to the head and neck area, systemic chemotherapy (e.g., doxorubicin, cyclophosphamide, vincristine, bleomycin), certain medications, and exposure to toxic substances.[9,16] The diagnosis is based on the patient's clinical history and the presence of anagen-phase hair dystrophy. Avoiding the triggering factors allows for normal hair regrowth, as the disruption to the hair growth cycle is temporary.[9] New hair that grows after resolving the causative factors may have different characteristics, such as graying, increased curliness, or straightening, due to changes in the hair follicle melanocytes or inner root sheath epithelium.[16]

Androgenic alopecia is a non-scarring alopecia with a characteristic distribution pattern and a genetic influence. It commonly occurs in adolescents, developing gradually over several years. Androgenetic alopecia does not occur in children with normal androgen levels, so if a prepubertal child displays typical features of androgenetic alopecia, it is recommended to examine their androgen levels. Trichoscopy for androgenetic alopecia may reveal more than 20% hair diameter variability in miniaturized hairs.[9] Biopsy and histopathological examination are helpful when the clinical presentation is atypical or when trichoscopy is not available. In AGA, hair density is usually normal, but there is an increase in the number of vellus and telogen hairs and a decrease in the ratio of normal hair to vellus hairs from the normal value of 7:1 to <3:1.[17]

In some cases of pediatric AGA, a complete hormonal examination may be necessary to investigate underlying endocrine disorders, particularly PCOS in adolescent girls, as it can be the sole dermatological manifestation of PCOS. Diagnosing PCOS is crucial due to its association with increased risks of infertility, diabetes, endometrial carcinoma, obesity, and heart disease.[17] Topical minoxidil is an effective and well-tolerated therapy for AGA in adolescents.[9] In a study, 95% of patients treated with topical minoxidil for 18 months showed increased hair coverage on the scalp or reduced hair thinning, with similar efficacy in both boys and girls. However, it's important to note that children are more susceptible to systemic absorption and related side effects due to their lower body weight and larger scalp surface area relative to body weight.[17]

Loose anagen syndrome (LAS) is a disorder where anagen hair, which grows in the follicle, lacks anchoring strength, resulting in easily removable hair shafts. This syndrome is most commonly observed in children aged 2-7 years. Hair tends to become unruly between the ages of 2 and 3, but typically returns to normal by the age of 5-7. Hair length often does not increase during this period.[9,18] Gentle hair care during childhood can help minimize hair loss, and spontaneous recovery is common during puberty. Some studies have reported that the use of 5% minoxidil can reduce the severity of symptoms.[9]

Short anagen syndrome (SAS) is a rare condition in children characterized by a shorter hair growth phase compared to the normal hair cycle.[6] Clinical manifestations of SAS include scattered or diffuse short hair, low hair density, and no alopecia patches. In SAS, the anagen phase lasts from 4 to 10 months, leading to synchronization of the hair cycle and repeated episodes of heavy hair loss. To diagnose SAS, a hair pull test must be performed on at least five areas of the scalp, and it is considered positive if five or more hairs are loose.[19] Unlike LAS, in SAS, hair loss occurs in the telogen phase rather than the anagen phase, as observed in a gentle hair pull test.[6] The hair shaft's structure and strength are typically normal, without brittleness, hair breakage, unruly hair, or signs of inflammation. In a case report, topical application of 2% minoxidil twice daily for 7 months resulted in a significant 26% increase in hair density, although hair length did not improve.[19]

Aplasia cutis congenita (ACC) is a rare group of diseases characterized by the absence of skin tissue, resulting in scarring alopecia at one or multiple locations from birth.[6] The incidence of ACC is approximately 0.03% of all live births, but it is often underreported, especially when mild lesions are present in newborns. ACC has 25 clinical subtypes based on the location and pattern of the absence of skin tissue, as well as the presence or absence of accompanying malformations and genetic hereditary factors such as trisomy 13 or 4p syndrome and cleft lip and palate.[6] Most patients present with midline scalp lesions, although lesions can also occur on the trunk and extremities. Typical lesions are well-defined and may exhibit ulceration or be covered by a rigid epidermal membrane.[6] Most lesions spontaneously reepithelialize over several months, leaving hypertrophic or atrophic scars. Supportive wound care is recommended, while lesions larger than 3 cm in diameter may require skin grafting.[6] Conservative treatments include hydrogel, emollient, or silver dressings. In cases where the defect size exceeds 15 cm^2, emergency surgical options such as closure with allogeneic dermis, skin grafts, keratinocyte grafts, skin flaps, or cranioplasty reconstruction may be considered. Multiple surgical interventions may be necessary for cosmetic reasons.[20]

Congenital triangular alopecia, also known as temporal triangular alopecia or Brauer's nevus, is a localized, non-scarring form of alopecia present at birth or develops within the first 10 years of life. The lesions are typically triangular, unilateral or bilateral, several centimeters wide, and confined to the anterior temporal region. While the lesion area appears hairless, fine vellus hairs can be found within the affected area.[9,10] Topical administration of 3% minoxidil helps regrow hair in children with congenital triangular alopecia, but relapse can occur when minoxidil usage is discontinued.[21]

Atrichia congenita is a rare form of irreversible alopecia inherited in an autosomal recessive manner, associated with mutations on chromosome 8. It is characterized by follicular agenesis or damage, resulting in very little or no hair at birth, which persists for the first five years of life. Atrichia congenita can occur as a standalone condition or as part of a syndrome. To differentiate it from alopecia areata totalis, a scalp biopsy can be performed.[9] Atrichia congenita is often accompanied by papular lesions, known as atrichia congenita with papular lesions (APL). These diseases comprise a heterogeneous group of genodermatoses characterized by total hair loss soon after birth, along with the development of keratin-filled cysts throughout the body. The scalp, face, neck, trunk, and limbs are commonly affected. Other syndromes associated with atrichia congenita include progeria, Moynahan's syndrome, and hidrotic ectodermal dysplasia.[22,23] Initially, atrichia congenita is often misdiagnosed as alopecia universalis until it is confirmed that the patient does not respond to steroid therapy.[6]

Congenital hypotrichosis is a milder form of atrichia congenita characterized by patchy thinning of the hair. Few hair follicles are present on the scalp at birth, but the condition may not be recognized until the age of two since the hair quality and quantity at birth are considered normal. It is a standalone condition, not associated with any syndrome.[9]

Marie Unna hereditary hypotrichosis is an autosomal dominant form of congenital hypotrichosis, with progressive hair loss starting at puberty. Congenital hypotrichosis can also be associated with inherited metabolic disorders, epilepsy, Ehlers-Danlos syndrome, juvenile macular degeneration, bone and dental disorders, and other chromosomal abnormalities.[22] Topical minoxidil may be beneficial for treating congenital hypotrichosis caused by LIPH mutations. Genetic counseling can be offered to patients who do not respond to any treatment.[22]

Transient neonatal hair loss (TNHL) is a temporary type of localized non-scarring alopecia that occurs on the occiput of infants aged 2-3 months. First reported by Brocq in 1907, TNHL has a prevalence ranging from 9-12% and is more common in Caucasian infants. It presents as oval patches of alopecia, particularly on the occiput. Friction on the occiput, mainly due to the supine sleeping position, is believed to be the cause of TNHL. However, recent retrospective studies have found no association between TNHL and sleeping position. Hair can spontaneously regrow in cases of TNHL.[6]

DISCUSSION

Abnormal hair growth can be difficult to detect at birth but can be identified during infancy. To diagnose hair loss in children, it is crucial to obtain patient and family history, conduct a comprehensive clinical examination, and perform investigations for accurate diagnosis and early treatment.[9] A detailed patient history is essential, including information about the age of onset (congenital or acquired), hair loss onset (sudden or gradual), type of alopecia (localized or diffuse), physical and mental development related to an underlying syndrome, previous medical history (such

as surgery or autoimmune disease), medication use, psychiatric disorders, and family history of alopecia.[9]

Certain medications, including anti-cholesterol drugs, anticoagulants, hormone therapy, and isotretinoin, can potentially cause iatrogenic hair loss. Anamnesis should also inquire about the family history of hair, nail, and tooth disorders, as alopecia often accompanies ectodermal genetic diseases. Additionally, information about hair care products used, such as shampoo, conditioner, and styling techniques, can be valuable in determining the cause of hair loss.[2]

A comprehensive physical examination should be conducted to assess the type of alopecia (localized or diffuse, with or without scarring), hypotrichosis or alopecia, hair shaft abnormalities, hair quality, and hair color. The examination of the scalp should evaluate the presence of erythema, edema, pustules, hyperkeratosis, atrophy, or scarring. In cases of patchy alopecia, it is important to determine the presence of patent follicular ostia (hair exit holes) or the absence of visible follicular ostia in scar alopecia. Perifollicular erythema, follicular hyperkeratosis, pustules, or edema may indicate an inflammatory process.[9,10]

A general physical examination should be performed to identify signs of short stature, abnormal bone development, abnormal hearing, dysmorphia, visual disturbances, metabolic or autoimmune diseases, and inflammation that may underlie alopecia in children. Based on the pattern of hair loss, a widespread or generalized pattern may suggest a systemic cause. Examining the hair fiber can provide information about pigmentation, texture, and potential nutritional deficiencies or underlying diseases such as Cushing's or anorexia nervosa.[24]

The hair pull test is the most common technique used to examine hair loss in children. It involves pinching the hair with an outstretched finger and pulling it gently to observe if any hair falls out. Another technique involves grasping a section of hair (30-60 strands) from a 1 cm x 1 cm area, twisting it loosely, and gently pulling it at a 90° angle from the scalp. The amount and type of hair loss obtained from these tests can provide clues about the underlying diagnosis. The tug test is conducted if hair shaft damage is suspected. This test involves grasping the hair near the scalp and pulling the distal part of the hair. A hair shaft fracture indicates brittleness caused by disruption of the hair shaft.[9,10] Anagen hair has a pigmented bulb enclosed within its root sheath, while telogen hair has a club-shaped, unpigmented bulb.[10]

The investigations for diagnosing hair loss in children vary depending on the underlying etiology, and there is no standard set of tests. If the patient's history or physical examination suggests a specific underlying condition, laboratory tests or other investigations may be necessary to determine the cause of hair loss. Trichoscopy, scalp biopsy, and laboratory tests, including antinuclear antibody (ANA) testing and blood sedimentation rate, can be performed to diagnose autoimmune diseases. Testing the levels of zinc, selenium, vitamins, minerals, prolactin, and sex hormone-binding globulin can help assess nutrient deficiencies, hormonal imbalances, and potential malignancies related to hair loss.[14,24,25] Trichoscopy, a non-invasive examination of the skin and hair structure, is particularly useful for diagnosing conditions like tinea capitis, alopecia areata (AA), trichotillomania, androgenetic alopecia (AGA), and telogen effluvium (ET).[25]

Autoimmune disease is a known cause of hair loss, and its diagnosis can be confirmed through tests for antinuclear antibodies (ANA) and the blood sedimentation rate. Testing levels of zinc, selenium, vitamins, and minerals can investigate deficiencies or excesses of specific nutrients. Endocrine and imaging laboratory investigations are valuable for detecting hair loss associated with androgen imbalance, thyroid issues, or ovarian malignancy. Additionally, evaluating prolactin levels and sex hormone-binding globulin is necessary to assess androgen levels in the body.[24]

The prognosis for hair loss in children depends on the type of alopecia, whether it is scarring or non-scarring. In scarring alopecia, like traction alopecia, early diagnosis and treatment can promote regrowth within a few months. However, chronic traction alopecia may result in irreversible scarring even if traction is ceased. Tinea capitis patients can experience permanent hair loss if inadequately treated, leading to hypertrophic scars.[22]

Non-scarring alopecia, such as telogen effluvium, generally has a good prognosis, with most hair regrowing within a year at a rate of approximately 1 cm/month. Patients with short-anagen syndrome typically see improvement during puberty.[6] On the other hand, the prognosis for patients with alopecia areata can be worse and often recurrent, particularly in children younger than 6 years old, those with a family history of AA, a disease duration exceeding 1 year, multiple affected areas, more than 50% of scalp involvement, ophiasis-type alopecia, comorbid nail disease, trisomy 21, or a history of atopy.[11]

Alopecia areata significantly impacts the psychosocial well-being of patients, often leading to social isolation and a decline in their quality of life. Delayed treatment of trichotillomania can cause psychological disorders and social avoidance.[14] Therefore, psychological counseling and support for patients and their families are essential in managing hair loss. Additionally, styling the hair, wearing hats, or using wigs can be beneficial in concealing the affected area.[2]

Hair loss is a common issue among children and can cause significant psychological distress for both the affected individuals and their parents. An effective approach to diagnosing hair loss involves utilizing a diagnostic algorithm that identifies the underlying cause early on. Congenital hair loss can be attributed to conditions such as atrichia congenita, loose anagen syndrome, short anagen syndrome, aplasia cutis congenita, and congenital triangular alopecia. On the other hand, acquired hair loss is categorized into scarring and non-scarring types. Scarring hair loss encompasses conditions like trichotillomania, tinea capitis, and traction alopecia. Non-scarring hair loss includes telogen effluvium, anagen effluvium, alopecia areata, androgenetic alopecia, loose anagen syndrome, and transient neonatal hair loss. By accurately diagnosing and treating hair loss in children, we can minimize the associated morbidity and enhance outcomes, ultimately preventing a decline in the affected individual's quality of life.

REFERENCES

1. Houschyar KS, Borrelli MR, Tapking C, Popp D, Puladi B, Ooms M, et al. Molecular mechanisms of hair growth and regeneration: current understanding and novel paradigms. Dermatology 2020;236:271–80.
2. Koblinski JE, O'Haver JA, Andrews ID. An Approach to Hair Loss in Pediatric Primary Care. Journal of Pediatric Health Care 2021;35:651–61.
3. Al-Refu K. Hair loss in children: Common and uncommon causes; Clinical and epidemiological study in Jordan. Int J Trichology 2013;5:185–9.
4. Shetty VM, Shanmukhappa AG, Nataraj H V, Aradhya SS. Hair loss in children: A clinicoetiological study from South India. Int J Trichology 2021;13:17–25.
5. Maan MA, Abrar A, Zahoor H, Hussain F, Akhtar SJ. Clinical Profile of Pediatric Alopecia Areata in Faisalabad City. Annals of Punjab Medical College (APMC) 2020;14:233–6.
6. Xu L, Liu KX, Senna MM. A practical approach to the diagnosis and management of hair loss in children and adolescents. Front Med (Lausanne) 2017;4:1–13.
7. Gurusamy U, Venkataswamy C. Hair loss in pediatric and adolescent age group: A clinico-pathological analysis in a tertiary health care centre. Journal of Clinical and Diagnostic Research 2017;11:EC01–5.
8. Ahanogbe I, Gavino ACP. Evaluation and Management of the Hair Loss Patient in the Primary Care Setting. Primary Care - Clinics in Office Practice 2015;42:569–89.
9. Alves R, Grimalt R. Platelet-rich plasma and its use for cicatricial and non-cicatricial alopecias: a narrative review. Dermatol Ther (Heidelb) 2020;10:623–33.
10. Champagne C, Alwash N, Patel M, Arujuna N, Farrant P. Hair loss in infancy and childhood. Paediatr Child Health 2019;29:66–73.
11. Premkumar M, Thomas J. Successful Management of Alopecia Areata in Children With Oral Tofacitinib. Spec Educ 2022;1:516–22.
12. Nageswaramma S, Sarojini Vl, Vani T, Madhuri S. A clinico-epidemiological study of pediatric hair disorders. Indian Journal of Paediatric Dermatology 2017;18:100–3.
13. Manchanda Y, Ramamoorthy R. Revisiting pediatric alopecia areata: Newer insights. Indian Journal of Paediatric Dermatology 2021;22:301–5.
14. Phillips TG, Slomiany WP, Robert Allison II. Hair loss: common causes and treatment. Am Fam Physician 2017;96:371–8.
15. Rudnicka L, Rakowska A, Olszewska M. Trichoscopy: how it may help the clinician. Dermatol Clin 2013;31:29–41.
16. Kanwar AJ, Narang T. Anagen effluvium. Indian J Dermatol Venereol Leprol 2013;79:604-12.
17. Griggs J, Burroway B, Tosti A. Pediatric androgenetic alopecia: A review. J Am Acad Dermatol 2021;85:1267–73.

18. Swink SM, Castelo-Soccio L. Loose anagen syndrome: a retrospective chart review of 37 cases. Pediatr Dermatol 2016;33:507–10.
19. Oberlin KE, Maddy AJ, Martínez-Velasco MA, Vázquez-Herrera NE, Schachner LA, Tosti A. Short anagen syndrome: Case series and literature review Pediatr Dermatol. 2018;35:388–91.
20. Schierz IAM, Giuffrè M, Del Vecchio A, Antona V, Corsello G, Piro E. Recognizable neonatal clinical features of aplasia cutis congenita. Ital J Pediatr 2020;46:1–6.
21. Bang CY, Byun JW, Kang MJ, Yang BH, Song HJ, Shin J, et al. Successful treatment of temporal triangular alopecia with topical minoxidil. Ann Dermatol 2013;25:387–8.
22. Imhof RL, Davis DMR, Tollefson MM. Hair loss. Pediatr Rev 2020;41:570–84.
23. Verma R, Vasudevan B, Pragasam V, Badad A, Mitra D, Neema S. Congenital atrichia with papular lesions. Indian Journal of Paediatric Dermatology 2014;15:94–5.
24. Lin RL, Garibyan L, Kimball AB, Drake LA. Systemic causes of hair loss. Ann Med 2016;48:393–402.
25. Castelo-Soccio L. Diagnosis and management of hair loss in children. Curr Opin Pediatr 2016;28:483–9.