Telogen Effluvium Presenting as Frontotemporal and Central Hair Loss in a Nigerian Infant

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ABSTRACT

Telogen effluvium (TE) usually presents as a diffuse non-scarring hair loss affecting the scalp. A compatible clinical history may be suggestive. However, other hair loss types like androgenetic alopecia and diffuse alopecia areata with similar clinical appearance may mimic TE.

Furthermore, relying on known clinical patterns may lead to underdiagnosis as atypical patterns may be missed. Focal non-scarring hair loss in infants in our environment is usually attributed to pressure alopecia, especially if the location is central or occipital.

The gold standard of making a definitive diagnosis in most forms of alopecia is scalp biopsy. In settings where biopsy is not feasible or practicable, trichoscopy serves a useful purpose in diagnosing hair loss types, especially those presenting with unusual clinical patterns, as was the case in this 8-month old infant with telogen effluvium from nutritional deficiency presenting with multifocal hair loss.

Keywords: Telogen effluvium. Hair loss. Infants

Effluvium Télogène Se Présentant Comme une Perte de Cheveux Frontotemporale et Centrale Chez un Nourrisson Nigérian

ABSTRAIT

L'effluvium télogène (ET) se présente généralement comme une perte de cheveux diffuse non cicatricielle affectant le cuir chevelu. Une histoire clinique compatible peut être évocatrice. Cependant, d'autres types de perte de cheveux comme l'alopécie androgénétique et la pelade diffuse avec une apparence clinique similaire peuvent imiter l'ET.

De plus, s'appuyer sur des schémas cliniques connus peut conduire à un sous-diagnostic, car des schémas atypiques peuvent être manqués. La perte de cheveux focale non cicatricielle chez les nourrissons de notre environnement est généralement attribuée à une alopécie de pression, en particulier si la localisation est centrale ou occipitale.

L'étalon-or pour établir un diagnostic définitif dans la plupart des formes d'alopécie est la biopsie du cuir chevelu. Dans les contextes où la biopsie n'est pas réalisable ou praticable, la trichoscopie sert à diagnostiquer les types de perte de cheveux, en particulier ceux présentant des schémas cliniques inhabituels, comme ce fut le cas chez ce nourrisson de 8 mois avec un effluvium télogène dû à une carence nutritionnelle présentant une multifocale perte de cheveux.

Mots clés : Effluvium télogène. Perte de cheveux. Nourrissons

Background

Telogen effluvium is a common cause of diffuse non-scarring alopecia. It commonly presents as sudden onset of diffuse hair loss with identifiable triggers (acute TE) or follows a more insidious course without obvious triggers (chronic TE). It occurs due to increased shedding of normal club hairs from normal hair follicles seen 2-3 months after a triggering event.[1]

It can occur during the first 6months of life, leading to a diffuse non-scarring hair loss which is considered physiologic.[2] However, when focal hair loss occurs in infancy, especially in the occipital region, it is often attributed to other common causes of focal hair loss in infancy, like pressure alopecia.[3] However, trichoscopy is useful, especially where biopsy is not feasible in making distinctions between hair loss types as the

clinical examination may miss atypical patterns.[4,5] Trichoscopic findings in telogen effluvium include decreased hair density with empty hair follicles, multiple single hair follicular units and regrowing hairs(including pigtail hairs), especially in the recovery stage.

Telogen effluvium usually causes diffuse thinning of the hair.[1] There is currently no report of TE presenting with focal patches of hair loss in the literature.

We describe a case of telogen effluvium presenting as multifocal areas of hair loss in an 8-month-old female infant.

Case Report

An 8-month-old girl being managed for anal stenosis by the Pediatric surgeons was referred to the dermatology clinic on account of a 4-month history of gradual onset asymptomatic multifocal hair loss. The hair loss started as a single patch of hair loss on the occipital region, with subsequent development of other patches on the frontal and vertex regions. Her mother denied a history of her resting on the affected areas while sleeping. There was no history of skin rashes, hair pulling, or scales on the scalp. Pregnancy and birth history were unremarkable. She had poor weight gain despite a good appetite and being fed on demand by her mother (at least five times/day). She had hematochezia at birth and a reduced frequency of defecation. She had normal developmental milestones for her age. Examination findings were a small for age child, not pale and anicteric, but had multiple patches of hair loss located on the vertex, occipital and frontal areas (Fig. 1a and b). The hair pull test was negative. Full blood count results were normal, and abdominal ultrasound was unremarkable. Serum iron studies done were normal. A clinical assessment of pressure alopecia was made. However, trichoscopy showed decreased hair density with multiple empty follicles and predominance of hair follicles with only one emerging hair on all the affected areas (Fig. 2). A diagnosis of telogen effluvium was made based on the clinical history and compatible trichoscopy features.

Her hair density improved clinically (Fig. 3a, b and 4) and trichoscopically (Fig. 5) after iron and protein supplementation therapy.

Discussion

There is a paucity of studies on alopecia in infants in Nigeria. One study in South-East Nigeria listed telogen effluvium as one of the most common causes of diffuse hair loss in infants and children up to 3 years.[6] Although there is a high prevalence of pressure alopecia among infants, physiologic TE and other congenital forms of alopecia can also occur in children and infants.[6,7]

The triggering causes of TE could be physiological or pathological. Physiological causes include the physiological effluvium of the newborn. This often affects the fronto-temporal area, occurring within the first four weeks of life (neonatal type) or at the second to the third month of life (occipital type) and is characterized clinically by diffuse hair loss and trichoscopically by widespread thinning of the hair.[2] Pathological causes include malnutrition, nutritional deficiencies of iron and zinc, infections and stress.[8] The trigger in the index patient might have been a combination of physiological factors, nutritional deficiency from the hematochezia and stress from the anal stenosis.

Although TE usually presents with diffuse non-scarring alopecia, bitemporal recession can also occur, closely resembling that of the index patient.[9] The index patient presented with asymptomatic multifocal areas of hair loss affecting the occipital and frontal areas, which prompted the initial suspicion of occipital alopecia of the newborn (OAN). However, the involvement of other scalp areas, including the frontotemporal and central scalp areas, made this diagnosis unlikely. Although occipital alopecia of newborns presents as a focal patch of hair loss (localized alopecia), it usually affects the occipital region only due to the pressure of the pillow on that area as the baby sleeps.[3] The patient's mother said the infant often slept on her sides which excluded OAN clinically.

Alopecia areata (AA) can also present with asymptomatic multifocal patches of hair loss. However, key distinguishing trichoscopic features of AA like multiple broken hairs, black dots and exclamation mark hairs were absent in this patient.[10-12] (Table 1). There was also no involvement of other body hairs.

Female pattern hair loss (FPHL) may also present as diffuse hair thinning affecting the frontal, vertex and occipital areas. In addition to not being common in infants, the absence of peripilar sign and lack of significant hair shaft diameter variation, which are characteristic features of FPHL, makes the diagnosis unlikely.[4-6,13]

Aplasia cutis congenital (ACC), temporal triangular alopecia (TTE) and sebaceous nevus all would present with congenital areas of hair loss and distinguished by the absence of skin with or without underlying bony structures in ACC, the temporal location of TTE with the area of hair loss being triangular or spear-shaped with the tip directed up and back and the associated orange-red plaques of sebaceous nevus.[14,15,16]

Diagnosis of TE is established by a combination of a compatible clinical history, in this case, background organic gastrointestinal condition (anal stenosis) associated with poor nutritional status (small for age) and occasional hematochezia; and the corresponding trichoscopic features.[4,5,9,17] Scalp biopsy is only required for imprecise cases. Trichoscopic findings of TE are the predominance of hair follicles opening with a single emerging hair shaft and the presence of upward regrowing hair.[4,5,9,17]

This patient's negative hair pull test is in keeping with what is seen in chronic TE.[18] Supporting investigations in patients with chronic TE are based on clinical suspicion and may include thyroid function tests, iron studies and full blood count, among others. However, although these are important, normal levels do not exclude chronic TE.[19,20]

Due to the patient's age and the distinctive clinical and trichoscopic features, a scalp biopsy was not done.

Finally, the regrowth of the patient's hair and improvement of her anthropometric parameters following protein, zinc and iron supplementation further confirms the diagnosis of chronic TE.

There are no studies in the literature documenting the multifocal pattern of hair loss associated with telogen effluvium as was seen in the index patient.

Conclusion

Telogen effluvium may present with atypical patterns, especially in infants. Trichoscopy is an important tool in the diagnosis of hair disorders, especially in those presenting with atypical patterns and where scalp biopsy may be impracticable or not feasible.

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References

1. Kligman AM. Pathologic Dynamics of Human Hair Loss. Arch Dermatol. 1961;83(2):175–98.
2. Neri, I., Piccolo, V., Cocchi, G., Starace, M., Patrizi, A., Dika, E. and Piraccini, B. (2013), Hair in newborns and infants: clinical and dermoscopic evaluation of 45 cases. Br J Dermatol, 169: 896-900. https://doi.org/10.1111/bjd.12459
3. Chang MW, Orlow SJ. Neonatal, pediatric and adolescent Dermatology. In:Freedberg IM,, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI eds. Fitzpatricks's Dermatology in General Medicine. 7th ed. NewYork: McGraw-Hill;2008.pp.935-955.
4. Rudnicka L, Olszewska M, Rakowska A et al. Trichoscopy: A new method for diagnosing Hair loss. J Drugs Dermatol. 2008;7(7):651-654.
5. Rudnicka L, Olszewska M, Rakowska A, Slowinska M. Trichoscopy update 2011. J Dermatol Case Rep [Internet]. 2011 [cited 2021 Apr 22];5(4):82–8
6. Nnoruka EN, Obiagboso I, Maduechesi C. Hair loss in children in South-East Nigeria: Common and uncommon cases. Int J Dermatol. 2007;46(SUPPL. 1):18–22.
7. Al-Refu K. Hair loss in children: Common and uncommon causes; Clinical and epidemiological study in Jordan. Int J Trichology [Internet]. 2013 [cited 2021 Apr 22];5(4):185–9. Available from: /pmc/articles/PMC3999647/
8. Belgaumkar VA, Chavan RB, Deshmukh NS et al. Evaluation of serum ferritin, vitamin B12 and vitamin D levels as biochemical markers of chronic telogen effluvium in women. Int J Res Dermatol. 2021;7:407-12.
9. Malkud S. Telogen effluvium: A review. J Clin Diagnostic Res. 2015;9(9): WE01–3. Available from: /pmc/articles/PMC3241952/
10. Enechukwu NA, Ogunbiyi AO. Hair disorders. 2021.In Ericchetti E. Lallas A Eds. Dermoscopy in General Dermatology for Skin of Color. https://www.taylorfrancis.com/chapters/edit/10.1201/9780367816483-16/hair-disorders-trichoscopy-nkechi-enechukwu-adebola-ogunbiyi.
11. Waśkiel-Burnat A, Rakowska A, Sikora M, Olszewska M, Rudnicka L. Trichoscopy of alopecia areata in children. A retrospective comparative analysis of 50 children and 50 adults. Pediatr Dermatol. 2019 Sep;36(5):640-645. DOI: 10.1111/pde.13912. Epub 2019 Jul 11. PMID: 31294493.
12. Waśkiel A, Rakowska A, Sikora M, Olszewska M,

Rudnicka L. Trichoscopy of alopecia areata: An update. J Dermatol. 2018 Jun;45(6):692-700. doi: 10.1111/1346-8138.14283. Epub 2018 Mar 22. PMID: 29569271.

13. Cranwell W, Sinclair R. Common causes of pediatric alopecias. Australian J Gen Practitioners. 2018;47(10):692-696.
14. Schierz, I.A.M., Giuffrè, M., Del Vecchio, A. et al. Recognizable neonatal clinical features of aplasia cutis congenita. Ital J Pediatr 46, 25 (2020). https://doi.org/10.1186/s13052-020-0789-5.
15. Yin Li VC, Yesudian PD. Congenital Triangular Alopecia. Int J Trichol. 2015;7(2): 48-53.
16. Saedi T, Cetas J, Chang R, Krol A, Selden NR. Newborn with sebaceous nevus of jadassohn presenting as exophytic scalp lesion. Pediatr Neurosurg. 2008;44(2):144-7. DOI: 10.1159/000113118. Epub 2008 Jan 24. PMID: 18230930.
17. Jain N, Doshi B, Khopkar U. Trichoscopy in alopecias: diagnosis simplified. Int J Trichol. 2013;5:170-8.
18. Harrison S, Sinclair R. Telogen effluvium. Clinical and Experimental Dermatology: Clinical dermatology: Clinical dermatology. 2002;27(5): 389-95.
19. Chartier MB, Hoss DM, Grant-Kels JM. Approach to the adult female patient with diffuse nonscarring alopecia. J Am Acad Dermatol. 2002;47(6):809–18.
20. Olsen EA, Reed KB, Cacchio PB, Caudill L. Iron deficiency in female pattern hair loss, chronic telogen effluvium, and control groups. J Am Acad Dermatol. 2010;63:991-999.

Table 1: showing the key differences between telogen effluvium and close differential diagnoses

Parameters	Telogen effluvium	Alopecia areata	Androgenetic alopecia
History	Acute or chronic onset diffuse loss of hair associated with physiologic triggers (postpartum, neonatal) or pathologic conditions (iron deficiency, chronic diseases etc.)	Single or multiple patchy hair loss can affect any hair-bearing area.	Hair thinning often affecting the frontal hair areas and of insidious onset. Usually from the third decade of life.
Clinical features	Diffuse hair thinning often occurring 2-3 months after a precipitating event (fever, chronic disease, nutritional deficiency, during the first 2-3 months of life etc.) with increased hair shedding.	Focal or diffuse areas of non-scarring hair loss sometimes affecting other hair-bearing areas with or without other associated autoimmune diseases. There may be associated pruritus or burning sensation.	Progressive hair loss affecting the frontotemporal and vertex hair areas in males and the frontal scalp posterior to the frontal hairline in females. There is a transition from thick terminal hairs to non-pigmented vellus hairs.
Trichoscopy	Predominance of single hair follicular units, empty hair follicles, reduced hair density and upright regrowing hairs. No variation in hair diameter (hair diameter heterogeneity)	Broken hairs-micro exclamation, tapered hairs and black dots; yellow dots,	Hair diameter variability (heterogeneity) more on the frontal areas when compared with the occipital region, peripilar brown pigmentation.
Biopsy	Normal number and density of follicles with an increased percentage in the catagen or telogen phase. There are no miniaturized follicles. Terminal:vellus ratio is often greater than 8:1	In acute AA, Peri- and Intra bulbar inflammatory infiltrate comprising activated lymphocytes, plasma cells, histiocytes and eosinophils (swarm of bees appearance). In chronic stages, miniaturized follicles with follicular dropout may be seen.	Mildly increased telogen to anagen ratio, superficial perifollicular inflammatory infiltrate may be seen. Miniaturization of hair follicles with sebaceous pseudohyperplasia.

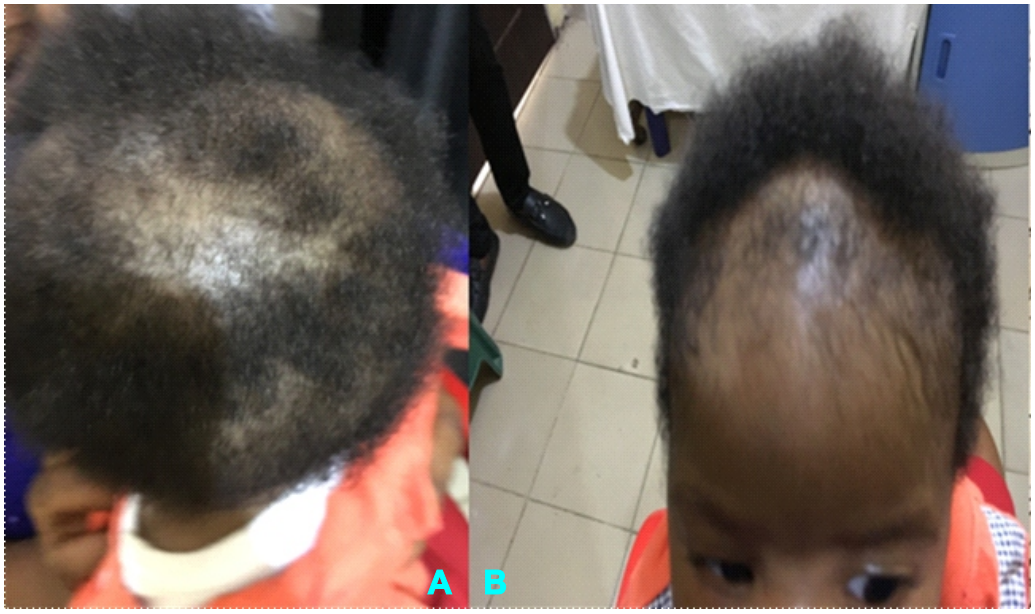

Fig. 1 a and b: Multiple focal hair loss at the vertex, occipital and frontal areas.

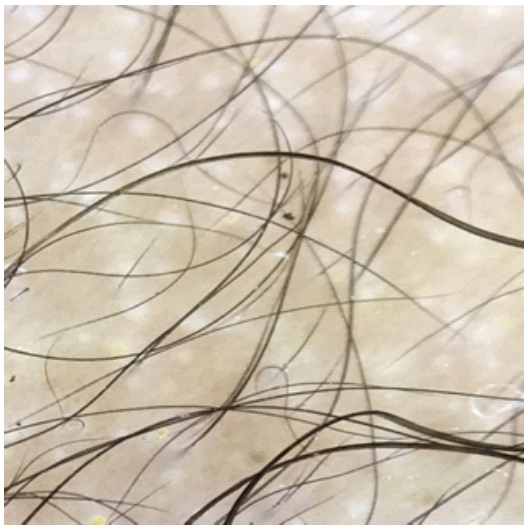

Fig 2: Multiple empty hair follicles with single hair emerging from each hair follicle

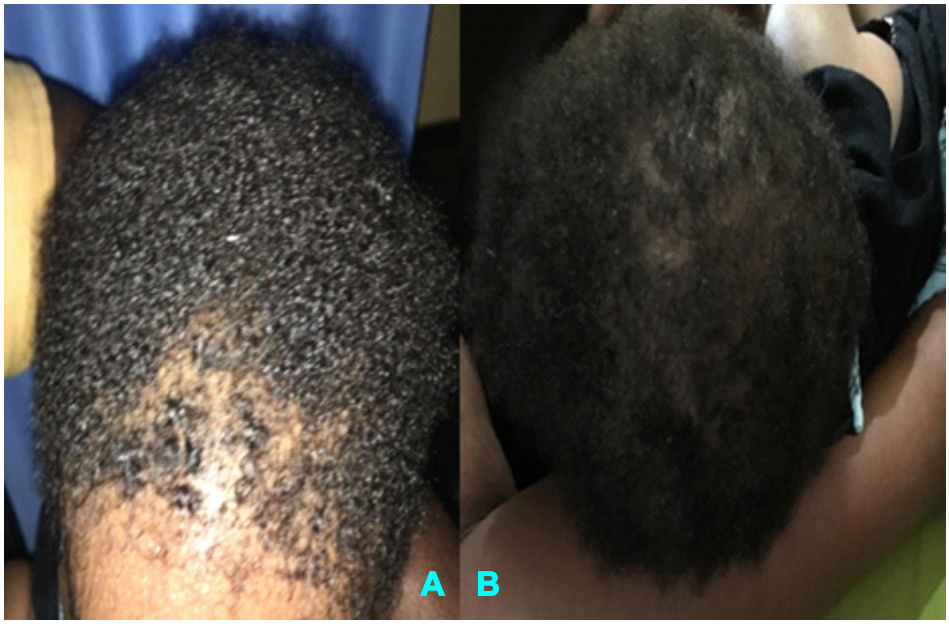

Fig. 3 a and b: Improved hair growth at the frontal, occipital and frontal areas after one month of treatment

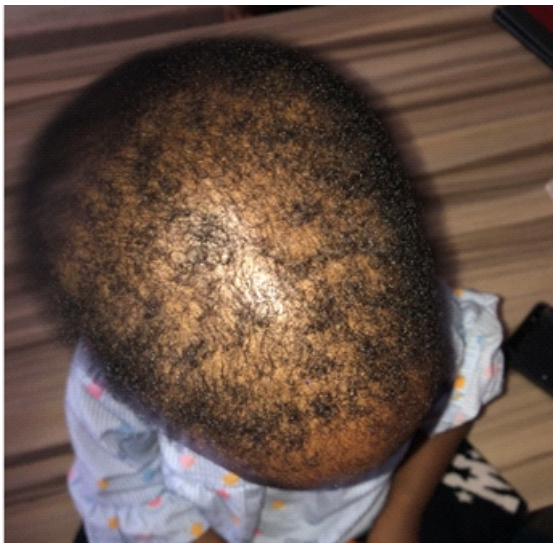

Fig. 4: Improved hair growth at the frontal, occipital and frontal areas after 4months of treatment

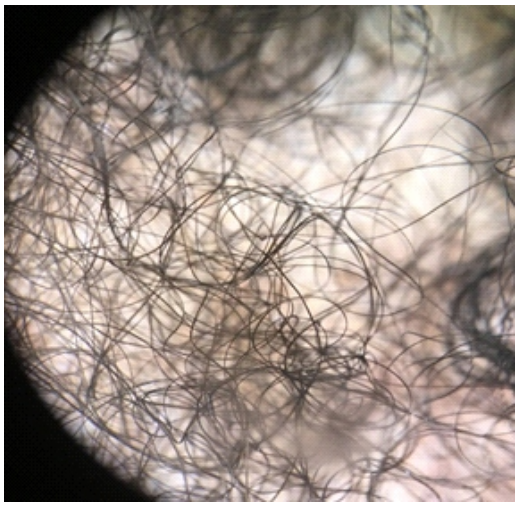

Fig. 5: Trichoscopic evidence of increased hair growth