

LETTER TO THE EDITOR

Occipital short anagen syndrome: Epidemiological, clinical and trichoscopy features of a case series

Dear Editor,

Short anagen syndrome (SAS) presents with short and fine hairs that fail to grow long due to markedly shortened anagen phase and by an increased proportion of telogen hairs, usually demonstrated on pull test.¹ It typically manifests as a diffuse scalp disorder with early life onset, most commonly between 2 and 4 years of age, and shows higher prevalence in females.^{1,2} Reports on localized short hair growth remain scarce, particularly when confined to specific scalp regions. To date, no investigations have focused on cases of short anagen restricted to the central occipital area. This study aims to describe the epidemiological, clinical and trichoscopic features in a case series of patients with an occipital short anagen (OSA), an underrecognized localized phenotype of SAS.

This retrospective multicentre study analysed data from 14 female patients with congenital/early childhood OSA. Age of onset was determined retrospectively based on parental reports; therefore, a precise distinction between congenital and early childhood onset could not be established. [Table 1](#) summarizes the data. Mean age at evaluation was 26.9 years (adolescence to older adulthood), reflecting the age at the time of clinical assessment rather than the age of OSA onset. Most patients had Fitzpatrick phototypes II or III (78,6%) and the majority presented with Caucasian hair (92.9%), followed by African hair type (7.1%). One patient underwent chemical hair straightening before OSA onset. In all cases, slow-growing hair was restricted to the central occipital region and was noticeable upon central parting, as a zone of shorter, untrimmed hairs compared to surrounding strands ([Figure 1](#)). Trichoscopy revealed normal hair count and shaft thickness in the affected area in 12 cases. Short regrowing hairs were observed in two patients. Regarding treatment, low dose oral minoxidil (LDM) was widely used, with six patients receiving 1–3 mg/day for a mean duration of 24 months. All users reported clinical improvement, with most describing good/excellent results.

OSA is a localized variant of SAS and its aetiopathogenesis is not fully understood. Notably, OSA may be underrecognized in males, likely because the predominance of short hair makes reduced occipital hair length less perceptible. In this cohort, no specific trichoscopic alterations were identified that could be directly associated with OSA,

TABLE 1 Clinical and trichoscopic characteristics of study patients.

Characteristics	Category	n (%) or mean (min; max)	
Demographics	Current age, years (n = 14)	26.9 (15–65)	
	Fitzpatrick phototype	I	2 (14.3)
		II	5 (35.7)
		III	6 (42.9)
		IV	1 (7.1)
Medical history (n = 14)	Recurrent telogen effluvium	3 (21.4)	
Associated hair disorders (n = 14)	Short anagen hair syndrome	3 (21.4)	
	Anagen effluvium	1 (7.1)	
	Loose anagen hair syndrome (LAHS)	1 (7.1)	
	Female pattern hair loss (FPHL)	1 (7.1)	
	Short anagen	1 (7.1)	
Hair type (n = 14)	Caucasian	13 (92.9)	
	African descent	1 (7.1)	
Hair grooming practices	Chemical hair straightening	1 (7.1)	
Trichoscopic findings (n = 14)	Short regrowing hairs	2 (14.3)	
	Reduced follicular units	1 (7.1)	
	Shaft thickness reduction	1 (7.1)	
Treatments	Use of oral minoxidil (n = 14)	6 (42.9)	
	–Duration of oral minoxidil Use (months) (n = 6)	24.2 (6–48)	
	–Oral minoxidil dosage (mg) (n = 6)	1.6 (1.0–3.0)	
	–Observed improvement with oral minoxidil (n = 6)	6 (100.0)	
	–Good improvement (> 50% hair lengthening)/excellent improvement (> 70% hair lengthening)	4 (66.7)	
	–Time frame for visible results with oral minoxidil (months) (n = 12)	8.0 (6–12)	
Occipital growth pattern (n = 14)	Central	14 (100.0)	



FIGURE 1 (a–b) Clinical image showing a localized phenotype of short anagen syndrome, with markedly short hair restricted to the central occipital scalp. (c–d). Trichoscopic images of the occipital scalp showing no specific abnormalities; no characteristic trichoscopic features of OSA are observed.

underscoring the importance of clinical history and epidemiological context for diagnosis.

The predominance of Caucasian hair may reflect the greater ease of recognizing localized hair shortening in straight hair, as opposed to other types, particularly curly. Our patients consistently exhibited uniform hair texture across the scalp. In contrast to hair breakage from chemical damage, 92.9% of them reported no fragility-inducing grooming practices, and the short hair had pointed tips, indicating no cutting or breakage.

During foetal development, the occipital region follows distinct hair-cycle dynamics compared with other scalp areas, and in adulthood, it maintains a shorter cycle than the frontal or vertex regions.³ This may explain why some individuals naturally have shorter hair in the nape area, subtly resembling an inverted fringe, usually without clinical significance. This clinical finding contrasts with our unique phenotype, which confines OSA to the central occipital region.

LDOM, by prolonging the anagen phase, represents a useful and efficient therapeutic option to enhance hair regrowth in OSA.⁴ Clinical improvement was noted in all users, with most reporting good/excellent outcomes. Limitations include the retrospective design, which restricted determination of onset age; small sample size; lack of systematic documentation of telogen ratios; and absence of prospective hair length measurements. Additionally, without histopathology, phototrichograms or molecular markers, OSA remains a clinical phenotype rather than a distinct pathophysiologic entity. Genetic testing was not performed due to the retrospective, multicentre design, limiting the evaluation of genetic associations, which should be addressed in future studies.

This series highlighted a previous unrecognized presentation of SAS restricted to the central occiput, predominantly affecting females. Preserved hair texture and the absence of fragility-linked grooming practices help distinguish OSA from acquired chemical hair breakage. Diagnosis is

clinical and trichoscopy excludes hair breakage due to conditions such as trichorrhexis nodosa. LDOM showed positive responses for hair elongation. Recognizing this entity is important to avoid misdiagnosis and unnecessary interventions. Further prospective and histopathological studies are warranted to clarify its mechanisms and particularities.

KEY WORDS

Alopecia, dermoscopy, loose Anagen hair syndrome, phenotype

FUNDING INFORMATION

The authors have nothing to report.

CONFLICT OF INTEREST STATEMENT

Dr. Adriana Rakowska acts as speaker and/or consultant for L'Oréal, Pierre Fabre, Arristo Pharma, Egis and Phizer; Dr. Antonella Tosti acts as consultant for DS Laboratories, Almirall, Thirty Madison, Eli Lilly, Pfizer, Myovant, Bristol Myers Squibb, Ortho Dermatologics and Sun Pharma; Dr. Bianca Maria Piraccini acts as speaker and/or consultant for Almirall, Difa Cooper, Dercos-L'Oréal, Eli-Lilly, ISDIN, Legacy Healthcare, Pierre Fabre-Ducray, Giuliani, Olistic, Pfizer and Bionike. Dr. Natasha Atanaskova Mesinkovska acts as advisory board for AbbVie, Arcutis, Sun Pharma, Pfizer, Lilly, Merck, L'Oréal and Veradermics; speaker Bureau for Lilly, Pfizer and Sun Pharma; principal investigator for AbbVie, Sanofi, Regeneron, Arcutis, Sun Pharma, Pfizer and Lilly. The other authors have no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

ETHICAL APPROVAL

The study complies with the internationally accepted standards for research practice and reporting. Ethical approval was not required for this study as per national guidelines.

ETHICS STATEMENT

The patients in this manuscript have given written informed consent to publication of their case details.

Flavia Oliveira Xavier de Brito¹
 Rita Fernanda Cortez de Almeida¹
 Carla Machado²
 Violeta Tortelly¹
 Fernanda Torres¹
 Antonella Tosti³
 Michela Valeria Rita Starace^{4,5}
 Lidia Rudnicka⁶
 Ncoza Dlova⁷
 Claudia Montoya⁸
 Adriana Rakowska⁶
 Hoda Moneib⁹
 Alba Gómez-Zubiaur^{10,11}
 Aline Blanco¹²

Ana Lisia Giudice¹³
 Anna Waškiel-Burnat⁶
 Colombina Vincenzi¹⁴
 Danielle Claudino Avelar¹⁵
 Federico Quadrelli⁵
 Gisela D'Atri¹⁶
 Leticia Arsie Contin¹⁷
 Lizet Rojano¹⁸
 Maria Andrea Ocampo¹⁹
 Mariana Lavia²⁰
 Mariana Penha²¹
 Maryanne Makredes Senna^{22,23}
 Matilde Iorizzo²⁴
 Natasha Atanaskova Mesinkovska²⁵
 Ramon Grimalt²⁶
 Sheila Requena-López²⁷
 Taynara Barreto²⁸
 Thaíssa Oliveira de Almeida Coelho²⁹
 Bianca Maria Piraccini³⁰
 Daniel Fernandes Melo¹

¹Research Department, Advanced Institute of Trichology, Rio de Janeiro, Brazil

²Preventive and Social Medicine Department, Federal University of Minas Gerais, Belo Horizonte, Brazil

³Dr. Phillip Frost Department of Dermatology and Cutaneous Surgery, University of Miami Miller School of Medicine, Miami, Florida, USA

⁴Dermatology Unit, IRCCS Azienda Ospedaliero-Universitaria di Bologna, Bologna, Italy

⁵Department of Medical and Surgical Sciences, University of Bologna, Bologna, Italy

⁶Department of Dermatology, Medical University of Warsaw, Warszawa, Poland

⁷Department of Dermatology, Faculty of Health Sciences, University of KwaZulu-Natal, Durban, South Africa

⁸Department of Dermatology, Universidad del Norte, Barranquilla, Colombia

⁹Department Dermatology, Venereology and Andrology, Faculty of Medicine, Ain Shams University, Cairo, Egypt

¹⁰Trichology Unit, Instituto Médico Ricart, Hospital Ruber Internacional, Grupo Quirónsalud, Madrid, Spain

¹¹Department of Dermatology, Hospital Universitario Príncipe de Asturias, Madrid, Spain

¹²Department of Dermatology, Hospital Universitário Maria Aparecida Pedrossian, Campo Grande, Brazil

¹³Department of Dermatology, Universidade Federal da Bahia, Salvador, Brazil

¹⁴Private Dermatology Practice, Bologna, Italy

¹⁵Dermatology Department, Uninovafapi University Center, Brazil

¹⁶Dermatology, Grupo MCI, Buenos Aires, Argentina

¹⁷Department of Dermatology, Hospital Do Servidor Público Municipal de São Paulo, São Paulo, Brazil

- ¹⁸Private Practice, Barranquilla, Colombia
- ¹⁹Department of Dermatology, Universidad Javeriana,
Bogotá
- ²⁰Department of Dermatology, Hospital de Clínicas
José de San Martín, Universidad de Buenos Aires,
Buenos Aires, Argentina
- ²¹Department of Dermatology, Faculty of Medicine
of Botucatu, São Paulo State University (UNESP),
Botucatu, Brazil
- ²²Lahey Hospital & Medical Center, Department of
Dermatology, Burlington, Massachusetts, USA
- ²³Harvard Medical School, Boston, Massachusetts,
USA
- ²⁴Private Dermatology Practice, Lugano/Bellinzona,
Switzerland
- ²⁵University of California, Irvine, California, USA
- ²⁶Department of Dermatology, Faculty of Medicine
and Health Sciences, Universitat Internacional de
Catalunya, Barcelona, Spain
- ²⁷Department of Dermatology, Hospital Universitario
Central de Asturias, Oviedo, Spain
- ²⁸Private Practice, Rio de Janeiro, Brazil

- ²⁹Trichology Outpatient Clinic, Dermatology Service,
Hospital das Clínicas, Universidade Federal de Minas
Gerais, Belo Horizonte, MG, Brazil
- ³⁰Private Practice, Bologna, Italy

Correspondence

Flavia Oliveira Xavier de Brito, Rua Cruz Lima 8/702
CEP 22230-010, Rio de Janeiro, RJ—Brazil.
Email: flaviaoxb@gmail.com

REFERENCES

1. Starace M, Gurioli C, Carpanese MA, Bruni F, Piraccini BM, Patrizi A, et al. Short anagen syndrome: a case series and algorithm for diagnosis. *Pediatric Dermatology*. 2021;38(5):1157–61. <https://doi.org/10.1111/pde.14750>
2. Oberlin KE, Maddy AJ, Martínez-Velasco MA, Vázquez-Herrera NE, Schachner LA, Tosti A. Short anagen syndrome: case series and literature review. *Pediatric Dermatology*. 2018;35(3):388–91. <https://doi.org/10.1111/pde.13478>
3. Barth JH. Normal hair growth in children. *Pediatric Dermatology*. 1987;4(3):173–84. <https://doi.org/10.1111/j.1525-1470.1987.tb00775.x>
4. Sharma D, Morsia S, Ungar B. Low-dose oral minoxidil in a case of short anagen syndrome. *The Journal of Dermatological Treatment*. 2025;36(1):2460580. <https://doi.org/10.1080/09546634.2025.2460580>