Telogen effluvium x female pattern hair loss: is there correlation?*

Fabiane Mulinari Brenner[1]
Carolina Oldoni[2]

DOI: http://dx.doi.org/10.1590/abd1806-4841.20198427

Dear Editor,

Initially described in 1961, Telogen Effluvium (TE) and its variants are still unclear with respect to classification, diagnosis and treatment. A new phase of the hair cycle was described by Stenn in 2002, when recognizing a proteolytic phase at the end of telogen, when shaft release occurs: the "exogen" phase.[1] Hypoxia was recognized—among other several well-documented factors, such as cytokines, hormones and nutrients—as impacting the follicle's reentry into a new hair cycle, which was termed by Rathman-Josserand *et al*. as the "neogen" phase.[2] In further development, a 2010 study by Gilmore and Sinclair suggested that chronic (TE) may be secondary to a reduction in anagen-phase variance, which would represent a new functional type of recurrent hair loss.[3]

Between 2015 and 2018, 42 women were treated for recent onset diffuse thinning (6 to 12 weeks) associated with a certain history event with a shedding of more than 100 shafts a day. Follow-up showed a reduction in fall with or without treatment in 6 to 12 weeks in all cases. These cases were initially considered as acute telogen effluvium, since they did not present previous complaints. The most frequent causes identified in the patients' history were divided into three groups: postpartum (n = 16); initiation of new drugs (n = 12), especially antidepressants and anticonvulsants (7 out of these 12); and recent medical events (n = 14) such as a diagnosis of other diseases (11 out of 14), specially thyroid diseases (6 out of 14) and surgical procedures (4 out of 14). Interestingly, 28 of the patients re-evaluated 12 to 18 weeks after diagnosis had dermatoscopic criteria for female pattern hair loss (FPHL). Reduction in density, variation in shaft diameter, and increase of perifollicular dyschromia or yellow dots in the central region (when compared to occipital region) were identified in this group. One year after diagnosis, 19 of them continued with follow-up and remained with clinical and dermatoscopic (n = 13) or histopathological (n = 6) diagnosis of FPHL.

Complete blood count, thyroid stimulating hormone (TSH) and ferritin were evaluated in all patients. Three had microcytic hypochromic anemia, four showed abnormalities in TSH (two others had started treatment and had normal levels), and 31 had ferritin below 40mg/L. Low levels of ferritin have been associated with telogen effluvium. However it may be a confounding factor in this group, since only cases with obvious causes were included and some were postpartum (nine other cases of clinical diagnosis with no obvious cause were discarded).

Of the 16 postpartum patients identified in our group, 12 were in the group that remained with a diagnosis of FPHL after one year of follow-up, which may suggest postpartum effluvium as a sign of possible FPHL. In addition, these findings are consistent with the lack of evidence for a postpartum physiological telogen effluvium already discussed in the literature.[4]

Knowledge about TE has progressed, but questions remain to be answered through studies with reliable methodologies. In addition, as suggested in this sample of patients, TE may be the initial manifestation in patients with FPHL. Rebora[5] emphasized that alopecia areata incognita occurs more frequently in patients with FPHL. Alopecia areata incognita is a diffuse loss of telogen hairs, usually without glabrous areas, that mimics severe TE; moreover, this entity involves a loss of more than 350 strands per day, with rare dystrophic anagen strands. Perhaps some of our cases were misdiagnosed, and dystrophic anagen bulbs may not have been identified among the fallen hair.❑

Received 27 February 2018.
Accepted 26 July 2018.
* Study conducted at the Hospital de Clínicas, Universidade Federal do Paraná, Curitiba, PR, Brazil.
Financial support: None.
Conflict of interest: None.

[1] Dermatology Service, Hospital de Clínicas, Universidade Federal do Paraná, Curitiba, PR, Brazil.
[2] Medical Student, Universidade Federal do Paraná, Curitiba, PR, Brazil.

Mailing Address:
Fabiane Mulinari Brenner
E-mail: fmbrenner@ufpr.br

REFERENCES

1. Stenn K. Exogen is an active, separately controlled phase of the hair growth cycle. J Am Acad Dermatol. 2005;52:374-5.
2. Rathman-Josserand M, Genty G, Lecardonnel J, Chabane S, Cousson A, François Michelet J, et al. Human hair follicle stem/progenitor cells express hypoxia markers. J Invest Dermatol. 2013;133:2094-7.
3. Gilmore S, Sinclair R. Chronic telogen effluvium is due to a reduction in the variance of anagen duration. Australas J Dermatol. 2010;51:163-7.
4. Mirallas O, Grimalt R. The Postpartum Telogen Effluvium Fallacy. Skin Appendage Disord. 2016;1:198-201.
5. Rebora A. Telogen Effluvium: Is There a Need for a New Classification? A Reply. Skin Appendage Disord. 2018;4:29-30.

AUTHORS' CONTRIBUTIONS

Fabiane Mulinari Brenner ORCID 0000-0001-7970-522X
Approval of the final version of the manuscript; Conception and planning of the study; Elaboration and writing of the manuscript; Obtaining, analyzing and interpreting the data; Effective participation in research orientation; Intellectual participation in propaedeutic and/or therapeutic conduct of the cases studied; Critical review of the literature; Critical review of the manuscript.

Carolina Oldoni ORCID 0000-0003-1649-3076
Elaboration and writing of the manuscript; Obtaining, analyzing and interpreting the data; Critical review of the literature.

How to cite this article: *Brenner FM, Oldoni C. Telogen effluvium x female pattern hair loss: is there correlation? An Bras Dermatol. 2019;94(4):*486-7.

CASE LETTERS

Novel mutation in PTCH1 gene in a patient with basal cell nevus syndrome and uterus bicornis*

Denis Miyashiro[1]
Luis Antonio Torezan[1]
Beni Moreinas Grinblat[1]
Cyro Festa Neto[1]

DOI: http://dx.doi.org/10.1590/abd1806-4841.20197449

Dear Editor,

A 31-year-old white female was evaluated due to a history of multiple basal cell carcinomas (BCCs) since childhood. Examination revealed multiple papules and erythematous nodules on the face, back, and thorax, palmar pits, hypertelorism, frontal bossing, and increased volume in the left maxillary region (Figure 1). Basal cell nevus syndrome (BCNS) was diagnosed and a search was performed for involvement of other organs. Radiography of the jaw revealed an odontogenic keratocyst, confirmed by histology; computed tomography revealed calcifications of the falx cerebri and cerebellar tentorium; pelvic ultrasound diagnosed uterus bicornis. The patient's parents had no clinical signs of BCNS and are not consanguineous. Genetic testing directed at BCNS was performed. A peripheral venous blood sample was collected for DNA amplification by polymerase chain reaction and sequencing of all coding exons and 20 base pairs from the non-coding regions of the PTCH1 gene. Microarray-based comparative genomic hybridization was performed to detect large deletions and duplications involving all exons of the PTCH1 gene. Two heterozygous variants in the PTCH1 gene were detected: c.3487G> A, which predicts the substitution of glycine (Gly) by serine (Ser) at codon 1163; and c.2778G> T, which predicts the substitution of tryptophan (Trp) by cysteine (Cys) at codon 926. No deletions or duplications were detected in the PTCH1 gene. The patient's mother tested negative for both variants, but the father tested heterozygous for the variant c.3487G> A. The variant c.3487G> A was inherited from the father, suggesting a benign mutation. Meanwhile, the variant c.2778G> T is a *de novo* mutation, and according to amino acid prediction programs (PolyPhen, SIFT, MutationTaster) this variant is probably pathogenic (Figure 2).[1] To our knowledge, this variant had not been described previously. The patient has been followed-up for 12 years. Surgical excision, imiquimod, and photodynamic therapy with methyl aminolevulinate were the treatments performed.

Basal cell nevus syndrome (BCNS) or Gorlin-Goltz syndrome is an autosomal dominant disease. It is characterized by multiple BCCs, benign odontogenic keratocysts in the jaw, palmoplantar pits, defects of the skeletal and central nervous system (spina bifida and bifid ribs, calcification of the falx cerebri, agenesis of the corpus callosum) and facial dysmorphisms. Aberrant activation of the Hedgehog signaling pathway is associated with sporadic and hereditary BCC (BCNS) as well as other developmental defects.[2] The Hedgehog pathway plays an important role in embryonic development. Its aberrant activation is involved in the development of many malignancies, including virtually all BCCs, whether sporadic or involved in the BCNS.[2] Sonic Hedgehog binds to PTCH-

Received 10 July 2017.
Accepted 13 August 2018.
* Study conducted at the Department of Dermatology, Faculdade de Medicina, Universidade de São Paulo, São Paulo, SP, Brazil.
Financial support: None.
Conflict of interest: None.

[1] Department of Dermatology, Hospital das Clínicas, Faculdade de Medicina, Universidade de São Paulo, São Paulo, SP, Brazil.

Mailing Address:
Denis Ricardo Miyashiro
E-mail: denisrmiyashiro@gmail.com

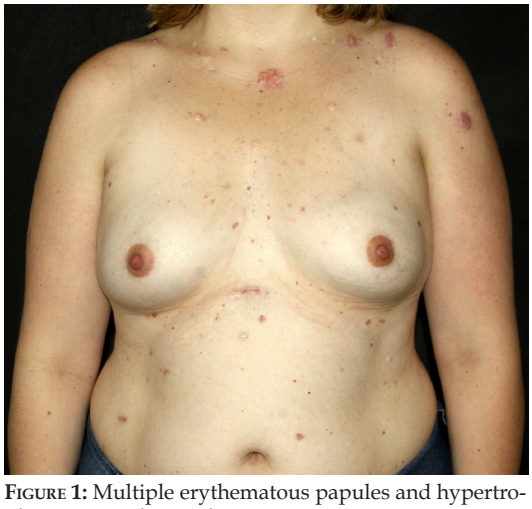

Figure 1: Multiple erythematous papules and hypertrophic scars on the trunk