

LOOSE ANAGEN HAIR SYNDROME, TYPE B

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Abbreviation LAS = loose anagen syndrome.

Case report. A 5-year-old girl was urgently examined because 4 days before the mother, grabbing her by the hair, found herself with a voluminous lock of hair in her hands (Fig. 2) without the girl having complained. Family history was negative for hair problems and autoimmunity. The remote personal medical history did not report relevant episodes; however, her mother reported that her hair was thin, always unruly, difficult to comb and that she did not need to cut it.

Physical examination showed fair, irregular hair and in the right parietal-occipital region a 6 x 3 cm, not completely alopecic patch (Fig. 1). Trichoscopy of the alopecic patch showed rarefaction of the hair and excluded signs of inflammation, the presence of exclamation mark hairs or hairs of different length. The pull test was positive with hair that had little resistance to traction. 60% of the hair extracted under an optical microscope was dystrophic in anagen phase, without sheath, with a pleated cuticle and with a hockey stick-like bulb (Fig. 3). Physical examination of the remaining skin, nails and teeth was within normal limits. These data led us to diagnose loose anagen hair syndrome, type B.

2% topical minoxidil was recommended and parents were reassured about the benignity of the disease and its likely improvement over time.

Discussion. Loose anagen syndrome (LAS), is a condition in which the hair has little resistance to traction and can be easily plucked out without pain, due to a faulty anchoring of the hair to the sheaths of the hair follicle. Preschool age and female sex are most affected. However, it is possible that these data are influenced by the progressive spontaneous improvement with time regarding the age and by the greater evidence in female sex due to the greater tendency to wear long hair that makes the problem more visible. The belief that people with fair hair are mainly affected has been unfounded (1).

The syndrome is often inherited with an autosomal dominant trait and in some cases mutations have been demonstrated in the *K6HF* and *K6IRS* genes that code for keratins expressed in the hair sheaths (3, 6). These mutations would be responsible for premature keratinization of the hair sheaths and therefore for their lower adhesion to the cuticle of the hair shaft and easier fall. LAS can also be associated with various hereditary diseases, more frequently with Noonan syndrome (4). However, there are also sporadic cases of LAS.

There are three clinical types of LAS: type A is characterized by decreased hair density, type B by unruly and difficult to comb hair, type C by apparently normal hair; type A and B are characteristic of preschool age, type C of adults.



Fig. 1



Fig. 2



Fig. 3

Fig. 1, 2, 3: Partially alopecic patch in loose anagen hair syndrome, type B (Fig. 1). Lock of teased hair exhibited by the mother (Fig. 2). Microscopic examination shows 60% of hair in dystrophic anagen, without sheath, with pleated cuticle and hockey stick-like bulb (Fig. 3).

The clinical symptoms of LAS are mainly influenced by the adhesion defect, so the reason why the patient comes to the doctor more often is the lack of hair growth and abundant fall; characteristic of LAS type B are thin, unruly, and difficult to comb hair.

The anamnestic suspicion of LAS is confirmed by the positivity of the pull test and above all by the presence on microscopic examination of numerous dystrophic anagen hairs, that is, hair without sheath, with an irregular, pleated cuticle, reminiscent of a soft sock, and with an acute angle of the bulb to the shaft to simulate a hockey stick or a mouse tail.

The number of dystrophic anagen hairs is important because dystrophic anagen hairs can be found in a modest percentage even in the normal subject and on the progression margin of alopecia areata (1). Dystrophic anagen hairs should be at least three in the pull test (5) and in the trichogram they should represent at least 50-70% (2, 7).

LAS usually has a good prognosis and tends to improve over the years. In severe cases, minoxidil is useful according to some Authors (2), perhaps prolonging the anagen phase.

Conclusion. The current case was presented for its rarity and to remember a condition whose recognition allows the doctor to reassure the parents and spare the child unnecessary tests.

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Conflicts of interest

The Author declares that he has no conflicts of interest.

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References

- 1) Abdel-Raouf H., El-Din W.H., Awad S.S. et Al. 2009. Loose anagen hair syndrome in children of upper Egypt. *J. Cosmet. Dermatol.* 8 (2): 103-7.
- 2) Cantatore-Francis J.L., Orlow S.J. 2009. Practical guidelines for evaluation of Loose Anagen Hair syndrome. *Arch Dermatol.* 145 (10): 1123-8.
- 3) Chapalain V., Winter H., Langbein L. et Al. 2002. Is the loose anagen hair syndrome a keratin disorder? A clinical and molecular study. *Arch. Dermatol.* 138 (4): 501-6.
- 4) Mazzanti L., Cacciari E., Cicognani A. et Al. 2003. *Am J. Med. Genet. Am.* 2003;118A (3): 279-86.
- 5) Olsen E.A., Bettencourt M.S., Coté NL. 1999. The presence of Loose Anagen Hairs obtained by hair pull in the normal population. *J. Invest. Dermatol. Symp. Proceed.* 4 (3): 258-60.
- 6) Porter R.M., Corden L.D., Lunny D.P. et Al. 2001. Keratin K6IRS is specific to the inner root sheath of hair follicle in mice and humans. *Br. J. Dermatol.* 145 (4): 558-68.
- 7) Tosti A., Piraccini B.M. 2002. Loose anagen hair syndrome and loose anagen hair. *Arch. Dermatol.* 138 (4): 521-2.