

Loose Anagen Syndrome: A New Case

Sir,

Loose anagen syndrome is a recently described disorder characterized by easily pluckable hair (1–3).

CASE REPORT

A 4-year-old girl who had never required hair cutting presented sparse hair, particularly in the right temporal zone. Occipital hair was matted and sticky. The condition had started at 1 year of age. No other defects were observed. There were no similar family antecedents. All analytical data were normal. Numerous hairs were easily and painlessly pulled out after exerting soft traction. Light microscopic examination of these hairs mounted in glycerin showed them to be in anagen with pigmented, twisted hair bulbs and no internal or external root sheaths. Ruffling of the cuticle was seen on a short part of the proximal hair shaft, the rest of the hair being normal in appearance. Following maternal informed consent, a 3-mm punch biopsy on the right parietal zone was performed, showing a fragmented inner root sheath with small notches complementary to the ruffled hair shaft cuticle, cleft formation in the outer root sheath, and absence of inflammatory infiltrates. Some changes were more evident at electron microscopy examination of the pulled hairs, showing rolled-back cuticle scales on a short segment of the proximal hair shaft with normal scales distal to this segment, and longitudinal grooving of the hair shaft (Fig. 1).

DISCUSSION

The percentage of hairs in telogen in children varies from 1 to 10% (4), versus 4–25% in adults (5). Under normal conditions, these are the hairs that pull out under soft traction.

Most cases of loose anagen syndrome are females with blond hair; they present diffuse or patchy alopecia without an increase

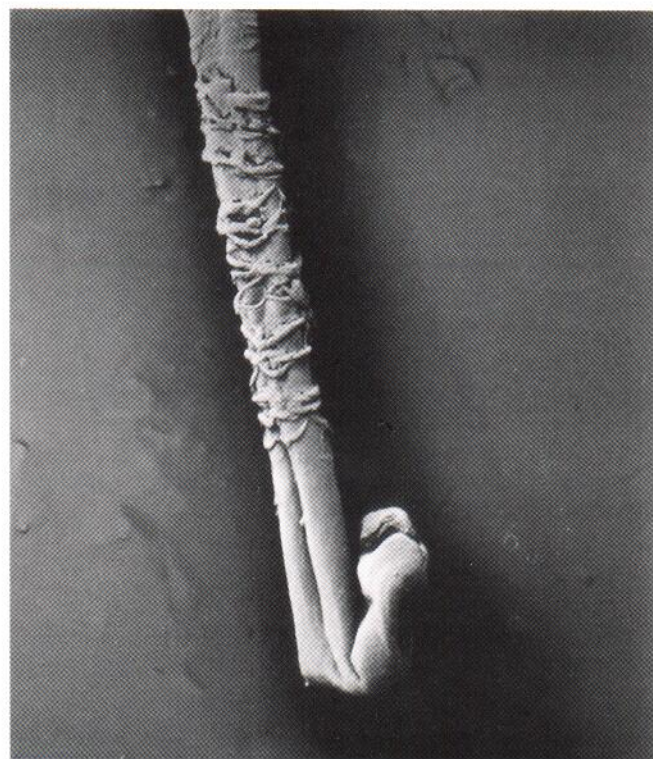


Fig. 1. Scanning electron micrograph of loose anagen hair ($\times 250$).

in hair fragility. Familial cases with dominant autosomal hereditary features have been reported (2, 6). Trichograms from clinically involved and uninvolved areas show a prevalence of anagen hairs (98–100%) and a complete absence of telogen hairs (3, 7). Possibly the defect is an early keratinization of the inner root sheath, interfering with normal interdigitation of the inner root sheath cuticle with hair cuticle, leading to an insufficient anchoring of the hair (2, 3). Baden et al. suggest that the disease may be due to an alteration in intercellular adhesion with altered desmosomal and/or cadherine components (6). No associated anomalies are normally found, though association with Noonan's syndrome has been reported (7).

Physiologically, there is a prepubertal increase in hair shaft calibre proportional to the sequential enlargement of follicles (8). This may explain the habitual improvement in the syndrome over time, though there is no complete resolution. The family of the patient should therefore be comforted in the knowledge that the syndrome evolves favourably and that no other treatment is available.

Differential diagnosis should mainly be established with telogenous effluvium, alopecia areata and trichotillomania. The intake of medication or toxic substances should be discarded, along with the theory of family antecedents and possible associated anomalies.

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