Loose Anagen Syndrome: A Little Response to Minoxidil

Sir,

Loose anagen syndrome (LAS) is a rare congenital hair growth disorder that is caused by premature keratinization of the inner root sheath, which causes impaired adhesion between the cuticle, the sheath, and the hair shaft. It is characterized by short, fine hair of the entire scalp, but the other features of the hair, such as the structure and strength, are usually normal without evidence of increased fragility, breakage, or inflammation. We report a case of a healthy 5-year-old girl with congenital isolated hypotrichosis due to LAS, successfully controlled by topical minoxidil.

A healthy 5-year-old girl presented with a history of short and scattered hair since birth [Figure 1]. Her father was beardless since his teens. The parents reported that there was excessive shedding and poor hair growth and that the girl had never needed a haircut. Aggravating factors were denied, and the past medical history was uneventful. On examination, the patient was well-developed and did not present any abnormality except for short, dark, and thin scattered hair. The pull test was positive and painless. Body hair was normal, as was the rest of the physical examination. Trichoscopy shows a decreased hair density with most follicular units containing only one hair and some yellow dots [Figure 2]. Trichogram of pulled hairs showed a 90% rate of loose anagen hairs (LAH). Furthermore, the proximal segment of some hair units appears distorted and twisted with ruffling of the cuticles [Figure 3]. Thus, the diagnosis of LAS was retained. Routine laboratory tests and genetic screening
for related syndromes were negative. Treatment consists in topical 2% minoxidil associated with zinc supplementation which allowed a considerable slowing down of hair loss without improvement of capillary density or length [Figure 4] and a switch to a 5% topical minoxidil is considered. LAS is estimated to have an incidence of 2–2.25 cases per million per year. Cantatore-Francis and Orlow[1] in a retrospective survey, including 374 children with alopecia, finds an incidence of 10% (n = 37) among all the subjects. It has been reported to occur typically in young girls (ratio, 6:1) and in most cases, spontaneously improves clinically by adulthood or as early as adolescence.[2] The most probable cause of LAS is premature keratinization of the inner root sheath, which causes impaired adhesion between the cuticle of the inner root sheath and that of the hair shaft. Thus, hair is pulled out easily and painlessly with minimal mechanical force, and it regrows quickly. The fast turnover of short hairs gives the impression of “short hair that does not grow.”[1‑3] LAS is thought to be an inherited condition in an autosomal dominant pattern with incomplete penetrance, although sporadic cases have been reported.[3] Mutations in K6hf of the companion layer of the hair follicle have been found in only some patients with LAHS, suggesting that mutations of other keratins of the inner root sheath may play a role.[4] The familial history of our patient may comfort this hypothesis.

The most frequent complaint from the parents is that the child’s hair is lusterless and never needed a haircut, explaining why it is underdiagnosed in boys. Clinically, the presentation of LAS is heterogeneous with three different phenotypes identified, with each having in common the finding of easily and painlessly extracted LAH on gentle hair pull. These phenotypes are as follows:

1. Type A LAS, characterized by decreased hair density
2. Type B LAS, characterized by mainly unruly hair
3. Type C LAS, characterized by normal appearing hair with excessive shedding of LAH.[2]

The above phenotypes appear to be age-dependent, with Types A and B occurring in children, possibly evolving

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**Letters to Editor**

**Figure 1:** Short and spars hair in a 5-year-old girl

**Figure 2:** Trichoscopy showing low follicular density

**Figure 3:** Dystrophic hair sheet

**Figure 4:** Moderate control after 3 months treatment with minoxidil
into the Type C phenotype around the age of 8 years and occurring in adults.[5] According to this, our patient seems to be a Type A LAS.

Even though LAS is most often isolated, it can sometimes be related to a congenital condition or acquired as coloboma, Noonan syndrome, hypohidrotic ectodermal dysplasia, EEC (ectrodactyly–ectodermal dysplasia–clefting) syndrome, trichorhinophalangeal syndrome, nail-patella syndrome, neurofibromatosis, trichotillomania, woolly hair, and has been described with AIDS.[6] LAS can be a presenting feature in alopecia areata.[7,8]

The basis for the diagnosis is based on a positive pull test resulting in a painless extraction of at least 10 LAHs and the presence of >50%–70% LAH on trichogram.[1,2]

Although the clinical expression of LAH improves with age, it has been suggested that the phenomenon persists throughout the individual's lifetime.[9] Thus, the use of topical minoxidil, mainly by increasing local blood flow, is promising in anecdotal reports and has been suggested as first-line therapy for children with clinically severe LAH.[1,5] The most commonly used presentation is the 2% solution, but a recently reported case reported the successful use of a 5% over 28 months, resulting in quick, significant clinical improvement with no adverse effects.[4]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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