Hair Loss in Children on Long-acting Gonadotropin-releasing Hormone Agonist Triptorelin Treatment

Sir,

Long-acting gonadotropin-releasing hormone agonist (GnRH-a) treatment of idiopathic central precocious puberty decreases gonadotropin and sex steroid levels and improves predicted final height (1). Chronic administration of GnRH-a induces rapid desensitization of pituitary GnRH receptors, and secretion is blocked after a short phase of physiological luteinizing hormone (LH) release. With adequate suppression, there should be no progression of secondary sexual maturation. The suppressive effects of GnRH-a are reversible 3 to 12 months after discontinuation of therapy (2). Side-effects have included occasional rash, exanthema, urticaria and androgen-dependent manifestations such as acne (3), but there have been no reports of hair loss.

Review of the recent literature did not reveal any report of hair loss. To the best of our knowledge, this is the first report of alopecia in children following administration of depot GnRH-a.

PATIENTS AND METHODS

Twenty-two children (20 girls and 2 boys), aged 6.5–11.0 years, with idiopathic central precocious puberty were treated monthly with the depot long-acting GnRH analogy, Decapeptyl® (D-Trp6, Ferring, Malmö, Sweden) 3.75 mg IM, for a period of 6–30 months. Prior to initiation of treatment, no hair loss was noted, either by the patients, their parents, or the pediatrician. The diagnosis of idiopathic central precocious puberty was based on the appearance of secondary isosexual characteristics before the age of 8 years in the girls and 9 years in the boys. All patients showed a pubertal response to GnRH stimulation, advanced bone age and accelerated growth on tests. All had normal sonography of the adrenals and of the gonads, and a normal brain CT scan. No patient with McCune-Albright syndrome, familial male precocious puberty or congenital adrenal hyperplasia was included in the study. Late onset adrenal hyperplasia was excluded by normal androgen response to ACTH test and normal adrenal ultrasound.

After at least 1 month of treatment, all the children were referred to the Department of Dermatology, Beilinson Medical Center, for clinical and microscopic examinations by two independent dermatologists (R.L. and A.I.). The Norwood classification for male pattern baldness was used to reveal the hair patterns (4).

RESULTS

Seventeen patients showed normal hair patterns. Five patients complained of moderate to massive hair loss. On clinical examination we observed diminished hair density of the vertex and frontal scalp.

Two of the children had type I and 3 showed type II hair loss, according to the Norwood classification (4). Microscopic findings were typical of androgenetic alopecia. The hairs become miniaturized, narrow and short, 0.5–1 cm in length, with tapered tips. Telogen hair loss was excluded by absence of diffuse alopecia and by the typical microscopic presentation of androgenetic alopecia.

DISCUSSION

Our data documented 5 patients with androgenic type hair loss following treatment with GnRH-a. Androgen-dependent skin changes, development of acne and mild gynecomastia have already been reported (5). Studies of patients with androgenic alopecia have reported variable results as to whether hyperandrogenism was present. Most patients showed normal blood testosterone levels (6). Hyperprolactinemia may be another cause of hyperandrogenism. Our patients showed hyperprolactinemia during treatment with GnRH-a, as previously reported by our group (7).

In summary, long-acting GnRH-a may lead to androgenic-type hair loss in children. Dermatologists, pediatricians and endocrinologists should be aware of this side-effect.

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Accepted February 3, 1997.

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