Scalp Hair Loss Caused by Octreotide in a Patient with Acromegaly: A Case Report

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Abstract. We used octreotide to treat a woman with acromegaly and observed pituitary adenoma shrinkage after 5 months. Diffuse scalp hair loss occurred after 5 months, resulting in the discontinuation of treatment. After the cessation of octreotide, the hair loss stopped and hair growth resumed. Since bromocriptine did not effectively decrease the GH level of the patient, we decided to perform transsphenoidal surgery. After resection of the pituitary adenoma, her GH and IGF-1 levels were normalized. Although octreotide-induced scalp hair loss has not been well recognized, we should pay more attention to this side effect.

Key words: Acromegaly, Octreotide, Hair loss

THE USE of octreotide, a somatostatin analog, for the treatment of acromegaly is widely established [1]. The most common adverse effect of octreotide is pain at the site of injection, cholecystolithiasis and gastrointestinal symptoms such as abdominal cramps, nausea, bloating, flatulence, diarrhea and steatorrhea [2, 3]. Hair loss caused by octreotide administration has rarely been reported [4]. We describe a patient with acromegaly who was successfully treated with octreotide, achieving a reduction in pituitary adenoma size. However, after five months of octreotide administration, hair loss occurred and we had to discontinue the drug. After the cessation of octreotide, hair growth resumed.

Case Report

A 71-year-old woman with acromegaly was admitted to our hospital for further examination in March, 1993. She had spondylosis deformance that had been conservatively treated for 20 years. Although she had noticed that she had to wear larger shoes than before, she thought it was caused by weight gain (45 to 68 kg). When she visited a physician because of a cold, acromegalic facial features, and high serum levels of GH (5.7 ng/ml) and IGF-1(6.8 U/ml) were pointed out. She was suspected of having acromegaly and was referred to our clinic. On physical examination, she was obese (157.4 cm in height, 80.0 kg in weight, with a body mass index of 32.3 kg/m²) and her blood pressure was 106/60 mmHg. Her face appeared to be acromegalic, with protrusion of the jaw and eyebrows, and acrodactyly was noted. She had goiter, and her chest and abdomen were normal. There were no abnormal findings on neurological examination. No visual field defects were detected. No hair loss was seen on admission. With respect to laboratory findings, urinalysis revealed no abnormalities. Peripheral blood examination showed mild anemia with an Hb level of 11.5 g/dl. The fasting blood sugar level of the patient was 114 mg/dl. Serological results included a mi-
crosome hemagglutinin test of × 400, a thyroid hemagglutinin test of × 800 and a thyroglobulin level of 180 ng/ml, which suggested that her goiter was caused by chronic thyroiditis. Heel pad thickness was 33 mm.

Hormonal examination showed a GH level of 6.9 ng/ml, a IGF-1 level of 554 ng/ml and a urine-GH level of 25.8 pg/ml (44.6 pg/mg cre.), all of which had increased. Thyroid hormone examination disclosed an F-T3 level of 5.4 pg/ml and an F-T4 level of 1.0 ng/dl. The plasma cortisol level, urinary 17-KS and urinary 17-OHCS levels were 8.8 µg/dl, 2.7 mg/day and 5.5 mg/day, respectively. Pituitary function tests revealed that GH showed an exaggerated response to GRH loading, increasing from 6.1 to 31.0 ng/ml. GH paradoxically responded to TRH and LH-RH. ACTH increased markedly in the CRF test, while TSH and prolactin responses were normal in the TRH test. The LH and FSH levels of the patient were normal in the LH-RH test, considering her age of 71 years. In a 75-g oral glucose tolerance test, her GH level paradoxically rose from 5.3 to 9.9 ng/ml. Her blood sugar level revealed glucose intolerance, and her plasma insulin level showed a delayed but very strong response. Diagnostic brain magnetic resonance imaging was subsequently performed, and a pituitary tumor of 2 cm diameter was demonstrated (Fig. 1A). From these results, a definite diagnosis of acromegaly due to a GH-producing pituitary adenoma was made.

Since our patient requested conservative therapy, we performed an octreotide loading test and a bromocriptine loading test to determine a therapeutic plan. Subcutaneous injection of octreotide 100 µg suppressed GH, and the suppressive effect continued for 12 h. In contrast, oral administration of 2.5 mg bromocriptine failed to suppress GH (Fig. 2A, B). We therefore prescribed a self-injection therapy with octreotide of 200 µg/day (100 µg, every 12 h).

After the administration of octreotide, serum GH, plasma IGF-1 and 24-h urinary GH levels decreased to <0.2 ng/ml, 95 ng/ml and <3.0 pg/ml (<2.9 pg/mg cre.), respectively (Fig. 3). Magnetic resonance imaging of the pituitary gland showed that the adenoma was significantly smaller 6 months after the initiation of octreotide (Fig. 1B), but hair loss developed. The patient was admitted for treatment for the hair loss in December, 1993. Since we suspected that the hair loss was caused by octreotide, we discontinued octreotide and started bromocriptine therapy (2.5 mg/day). During bromocriptine administration, serum GH, plasma IGF-1 and 24-h urinary GH levels increased to 8.2 ng/ml, 508 ng/ml and 21.0 pg/ml (41.1 pg/mg cre.), respectively (Fig. 3) and the size of the pituitary adenoma increased on brain magnetic resonance imaging (Fig. 1C). Transsphenoidal pituitary adenectomy was subsequently performed on February 2, 1994. After resection of the adenoma, serum GH, plasma IGF-1 and 24-h urinary GH decreased to 1.0 ng/ml, 205 ng/ml and 3.7 pg/ml (5.3 pg/mg cre.), respectively (Fig. 3). One
Fig. 2. Serum GH responses to octreotide (A) and bromocriptine (B) loading tests. A: Injection of octreotide 100 μg produced complete suppression of GH. B: Oral administration of bromocriptine 2.5 mg produced a slight decrease in GH.

Fig. 3. The figure shows the clinical courses of serum GH, plasma IGF-1 and 24-h urinary GH levels.
week after the operation, pollakisuria appeared and the antidiuretic hormone level of the patient was 0.8 pg/ml. Urinary volume increased to 4500 ml. DDAVP administration was effective for her partial diabetes incipidus, which spontaneously recovered one month later. After the discontinuation of octreotide, hair loss stopped and hair growth resumed six months later (Fig. 4A, B).

Discussion

Hair loss caused by octreotide administration has rarely been reported. In 1992 Jónsson et al. described hair loss as a possible new side effect of octreotide [4]. All four reported cases occurred in women. After octreotide was withdrawn hair loss diminished in three of the four patients. Four months later complete recovery of scalp hair was observed. The fourth patient, who did not stop octreotide treatment, continued to show diffuse scalp hair loss.

Of six patients (four women and two men) given octreotide at our clinic, two women (this case and another woman) and one man experienced scalp hair loss during treatment. The two patients other than this case had slight hair loss and did not need to discontinue octreotide. The female patient was administered octreotide 200 µg daily. Her GH level recovered to 3–4 ng/ml and her IGF-1 level decreased to the upper limit of the normal range. Her hair loss remained slight during treatment.

Since hair loss is a less recognized side effect of octreotide, we must be aware of it during the administration of the drug. The etiology of this scalp hair loss has not been reported. Acromegaly is usually associated with hypertrichosis caused by an increase in IGF-1. During octreotide treatment serum GH, plasma IGF-1 and 24-h urinary GH decreased to very low levels. And after resection of the tumor, these hormonal data had levels within the normal ranges (Fig. 3). Therefore, acute and complete suppression of GH and IGF-1 might cause hair loss. And another possible cause of scalp hair loss is the direct action of this drug on the hair follicle cells. Our patient was in a euthyroid state and all of the other reported cases of hair loss occurred under euthyroid conditions. LH and FSH levels did not change during octreotide treatment. Although testosterone and other androgen levels were not measured in our patient, it is unlikely that changes in gonadal and thyroid function were involved in her hair loss.

Although hair loss caused by octreotide has rarely been reported, we should pay more attention to this side effect, especially in women.

Fig. 4. Scalp hair (clinical course) (A, B). A: After 5 months of treatment with octreotide, diffuse scalp hair loss occurred. B: Five months after the cessation of octreotide, hair growth resumed.
References


