Acquired Progressive Kinking of the Hair

Clinical Features, Pathological Study, and Follow-up of 7 Patients

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Background: Acquired progressive kinking of the hair (APKH) is a relatively rare condition, with fewer than 20 cases reported in the literature. Whether APKH is a separate entity or a variety of androgenetic alopecia is still controversial. This study reviews the clinical and pathological features and long-term follow-up of 7 patients with APKH.

Observations: Since January 1989, we have diagnosed APKH in 7 males aged 15 to 22 years. All patients had strong family history for androgenetic alopecia. Hair kinking affected the frontotemporal region and/or the vertex where the hair appeared curly, frizzy, and lusterless. The pathological features of the affected scalp were consistent with the diagnosis of the early stages of androgenetic alopecia. In all patients, APKH evolved into androgenetic alopecia during the follow-up period. Mean follow-up was 4.5 years (range, 2-9 years). Treatment with topical minoxidil did not prevent development of hair thinning in the scalp areas affected by hair kinking.

Conclusions: The term acquired progressive kinking of the hair encompasses a number of conditions characterized by acquired curling of the scalp hair. Acquired hair kinking on the androgen-dependent areas of the scalp represents a modality of onset of androgenetic alopecia associated with poor prognosis.

Arch Dermatol. 1999;135:1223-1226
distinguishable from the surrounding scalp, which presented straight or lightly curled shiny hair. The color of the hair in the affected area was darker than the surrounding hair in 1 patient and lighter than the surrounding hair in 2 patients. In the remaining patients, the hair with acquired kinking showed no color changes as compared with the surrounding scalp. Three of the 7 patients showed a mild hair thinning of the frontal area as compared with the occipital scalp. Examination of eyebrows, eyelashes, and axillary hair did not reveal any abnormality.

 LABORATORY INVESTIGATIONS

A pull test and a trichogram were performed from the affected and nonaffected scalp. The hair samples were taken 5 days after shampooing. A 5-mm punch biopsy sample was obtained, after informed consent from the patient, from the affected areas in all cases. A 5-mm punch biopsy sample from the occipital scalp was also obtained in 2 patients. Biopsy samples were processed for both longitudinal and transverse sections. The diameter of the hair shaft and the hair follicles was measured using a calibrated micrometer. Sections cut at the level of the isthmus were used for counting the total follicular units, including the terminal, vellus, and telogen follicles.

### RESULTS

#### PULL TEST

Five patients had positive results from the pull test in the frontotemporal area (extraction of 5-12 telogen hairs; mean, 8 hairs).

#### TRICHOGRAM

**Affected Scalp**

The trichogram revealed a decreased anagen to telogen ratio in all patients. Hair shaft examination did not show signs of hair weathering but only irregular torsions of the hair shaft along its major axis. In these areas, the hair shaft gave the impression of being thinned, but this was only a consequence of torsion.

**Unaffected Scalp**

A decreased anagen-telogen ratio was present in 3 cases.

#### PATHOLOGICAL FEATURES

The quantitative data from the measurements of follicular structures are reported in Table 2. The average total number of hairs in horizontal sections of the 5-mm punch biopsy specimens taken from the affected scalp was 40. Mean terminal to vellus ratio was 3.4:1, with 85.5% anagen and 14.5% telogen. Sections taken from the occipital scalp in 2 patients showed 45 hairs in one patient and 42 hairs in the other. Terminal to vellus ratio was 12.1 and 18.5:1, respectively. Two specimens showed a slight superficial perivascular lymphocytic infiltrate in the papillary

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**Table 1. Clinical and Follow-up Data of 7 Patients With Acquired Progressive Kinking of the Hair**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y</th>
<th>Age at Onset, y</th>
<th>Increased Hair Shedding</th>
<th>Family History of Androgenetic Alopecia</th>
<th>Area of Involvement</th>
<th>Hair Thinning</th>
<th>Follow-up</th>
<th>Evolution Condition</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>16</td>
<td>16</td>
<td>Yes</td>
<td>H5</td>
<td>Fr + T + V</td>
<td>No</td>
<td>9†</td>
<td>CW</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>14</td>
<td>No</td>
<td>H4</td>
<td>Fr + T</td>
<td>No</td>
<td>7</td>
<td>CW</td>
</tr>
<tr>
<td>3</td>
<td>19</td>
<td>18</td>
<td>Yes</td>
<td>H3</td>
<td>Fr + T + V</td>
<td>Yes</td>
<td>5†</td>
<td>W</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
<td>18</td>
<td>Yes</td>
<td>H3</td>
<td>Fr + T</td>
<td>Yes</td>
<td>5†</td>
<td>CW</td>
</tr>
<tr>
<td>5</td>
<td>22</td>
<td>21</td>
<td>No</td>
<td>H4</td>
<td>Fr + T + V</td>
<td>Yes</td>
<td>2†</td>
<td>ST</td>
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<tr>
<td>6</td>
<td>17</td>
<td>17</td>
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<td>Fr + T</td>
<td>No</td>
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<td>W</td>
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<tr>
<td>7</td>
<td>14</td>
<td>14</td>
<td>Yes</td>
<td>H3</td>
<td>Fr + T</td>
<td>No</td>
<td>2</td>
<td>W</td>
</tr>
</tbody>
</table>

*H3, H4, and H5 indicate Hamilton-type androgenetic alopecia; L1 and L2, Ludwig-type androgenetic alopecia; Fr, frontal area of the scalp; T, temporal area of the scalp; V, scalp vertex; CW, considerable worsening of the alopecia; W, worsening of alopecia; and ST, androgenetic alopecia remained unchanged. Ellipses indicate data not applicable.

†Patients applied topical minoxidil on a regular basis.

**Table 2. Follicular Structures Identified in Punch Biopsy Specimens**

<table>
<thead>
<tr>
<th>Follicular Structures, No.</th>
<th>Total No. of Follicles</th>
<th>Terminal</th>
<th>Vellus</th>
<th>Telogen</th>
<th>Stelae</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Affected Scalp</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
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<td>2</td>
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<tr>
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<td>8</td>
<td>4</td>
<td>6</td>
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<tr>
<td>3</td>
<td>36</td>
<td>14</td>
<td>15</td>
<td>7</td>
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</tr>
<tr>
<td>4</td>
<td>46</td>
<td>36</td>
<td>4</td>
<td>6</td>
<td></td>
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<tr>
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<td>7</td>
<td>3</td>
<td>2</td>
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<tr>
<td>6</td>
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<td>21</td>
<td>5</td>
<td>4</td>
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<td>3</td>
<td>7</td>
</tr>
<tr>
<td><strong>Occipital Scalp</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
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<td>36</td>
<td>3</td>
<td>6</td>
<td>0</td>
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<td>37</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

*Ellipses indicate data not applicable.*
dermis with a moderate amount of mucin between collagen bundles. Infundibular plugging and bacteria were also evident.

**TREATMENT**

Patients were instructed to apply topical minoxidil lotion at the dosage of 1 mL twice a day. The 2% minoxidil lotion was prescribed until 1995, when 5% minoxidil became available in Italy. At that time, all patients were switched to 5% minoxidil. Patients 5, 6, and 7 used 5% minoxidil from the first consultation.

**FOLLOW-UP**

All patients were examined every 6 to 12 months. Follow-up duration varied from 2 to 9 years (mean, 4.5 years). Only 5 of the 7 patients regularly applied topical minoxidil during the follow-up period. The other 2 patients discontinued minoxidil application due to poor compliance. This occurred after 9 to 36 months of treatment. In all patients, APKH evolved into androgenetic alopecia during the follow-up period. This also occurred in patients who regularly applied topical minoxidil. In particular, 3 of our 7 patients developed severe hair thinning that was especially evident on the crown region. The progression of APKH to androgenetic alopecia was quite rapid in all cases. All of the patients exhibited a mild-to-moderate androgenetic alopecia within 2 years of our first examination (Figure).

**COMMENT**

The term *acquired progressive kinking of the hair* has been used in the literature to describe rather different conditions. These include the following:

1. Kinking of the hair over the periauricular areas of the scalp (whisker hair).\(^5,6\) Whisker hair is short and curly and resembles a continuation of the beard. According to Norwood,\(^7\) whisker hair is strongly associated with severe androgenetic alopecia and typically affects young men (aged 18-25 years) who will rapidly develop extensive baldness.

2. Acquired progressive kinking of androgen-dependent hair associated with thinning. This type of APKH has until now been reported in 16 patients, including 9 patients in the literature\(^1-4,7\) and our 7 patients. All reported patients were postpuberal males and developed androgenetic alopecia of the areas affected by hair kinking.

3. Rapidly progressing kinking or most of all the scalp hair without associated hair thinning. This variety has been reported in 2 patients: a 16-year-old boy and a 14-year-old girl.\(^8,9\) In these cases, kinking had a rapid onset, quickly progressed to involve the entire scalp, and persisted unchanged during the years. This variety of acquired hair kinking affects both androgen-dependent and non–androgen-dependent scalp follicles and is not associated with hair thinning.

4. Acquired reversible hair kinking before or after puberty. This type has been reported in 2 females, including a child.\(^10,11\) In both these patients, hair kinking completely regressed with time and was not associated with hair thinning.

5. Acquired hair kinking involving a localized non–androgen-dependent area of the scalp. This variety has been reported in 3 patients, including 2 females.\(^12-14\) In all patients, the condition remained stable in the follow-up period, affected non–androgen-dependent follicles, and was not associated with hair thinning. This variety of APKH may clinically resemble woolly hair nevus, but it is acquired and not congenital.

In addition, a few patients described in the literature who were diagnosed as having APKH\(^13,10\) were actually affected by diffuse partial woolly hair. This condition is characterized by the presence of 2 distinct hair populations (straight hair and curly hair) that are contemporarily present and intermingled. Diffuse partial woolly hair probably results from hair miniaturization involving a few scattered hair follicles.\(^15\)

In types 3, 4, and 5 APKH, hair kinking is not restricted to androgen-dependent areas and does not lead to hair thinning. These types of hair kinking have been observed in women as well as in prepuberal patients.

In types 1 and 2 APKH, development of curly hair characteristically heralds the onset of androgenetic
alopecia. These types of APKH typically affect young males with a strong family history of androgenetic alopecia.

The pathological features of the frontotemporal area of our 7 patients with type 2 APKH were consistent with a diagnosis of male androgenetic alopecia, due to the presence of hair miniaturization, with a terminal to vellus ratio of 3.4:1.

Why hair thinning may cause kinking is not known, since factors determining the different hair shapes are not completely understood. Factors that may be implicated include (1) change in the shape of the hair follicle during miniaturization, (2) irregularities of mitosis within the hair bulb, and (3) changes in the arrangement of the keratin filaments within the hair shaft.

Treatment of APKH in our patients was scarcely successful and only 1 of them (patient 5) did not show progression of androgenetic alopecia during the follow-up period. Development of androgenetic alopecia was scarcely prevented by regular treatment with topical minoxidil. There are no data about the effects of finasteride in APKH, although increased scalp and serum levels of dihydrotestosterone have been reported in 1 patient with this condition.

Accepted for publication August 28, 1998.

This study was partially supported by the University of Bologna through its funds for selected research topics.

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REFERENCES