A Case of Hair Structure Abnormality Associated with Iron Deficiency Anaemia

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Iron deficiency has been discussed as an etiologic factor in diffuse alopecia. Although the mechanism of alopecia related to iron deficiency remains unknown, the diagnosis is confirmed if hair loss ceases with iron administration. However, hair structure abnormalities due to iron deficiency are less known. We report a case of hair structure abnormality associated with iron deficiency anaemia. The hair structure abnormality was a fusiform and expanded node of the hair shaft. The hair loss ceased and the abnormality was corrected with iron administration. (Ann Dermatol 15(2) 89-91, 2003).

Key Words : Iron deficiency anaemia, Hair structure abnormality, Hair loss

Diffuse alopecia is a common condition for which no fully convincing cause can be established in many cases. Close evaluation of its cause must be carried out including the investigation of androgen/estrogen metabolism, thyroid disease and the nutritional states including iron deficiency anaemia. Iron deficiency, even in the absence of anaemia, has been discussed previously as an etiologic factor in diffuse alopecia such as telogen effluvium, especially in women. However, hair structure abnormality due to iron deficiency is uncommon and not well acknowledged.

CASE REPORT

A thirty-year-old Korean woman presented with an eight-month-history of alopecia on the scalp to the Department of Dermatology, Kyung Hee University, Korea (Fig. 1). She had no family history of alopecia. She did not take any medication before the onset of alopecia, had no history of contact with chemicals such as hair dyes or permanent agents, or application of any other traumatic procedures such as excessive combing or brushing. On physical examination, her skin and conjunctiva were pale and diffuse hair loss was found on the scalp. Broken hairs in the alopecic region were noted (Fig. 2). Hair samples were taken by cutting near the proximal end from an alopecic region. Microscopic examination revealed broken or slightly angulated, fusiform, expanded nodes on the proximal portion of the hair (Fig. 3). On scanning electron microscopic examination, the node had a smooth surface covered with normally imbricated scales of hair cuticle cells, and no intermodal twisting or grooving was found (Fig. 4). These findings were revealed in almost all of the shafts of sampled hairs. And so we considered that the hair loss resulted from the breakage of the expanded nodes on the proximal portion of the hair.

On the laboratory test, thyroid and sex hormone levels were within normal limits. Significant laboratory values were 5.6 g/dL hemoglobin, 19.5% hematocrit, 11.80/dL serum ferritin (normal value 50-150 μg/dL) and 414 μg/dL TIBC(total iron binding capacity) (normal value 200-400 μg/dL), which suggested iron deficiency anaemia. We sus-
Fig. 1. Diffuse alopecia appearing androgenetic alopecia or telogen effluvim at the first glance.

Fig. 2. The hairs in alopecic region were broken near proximal end.

Fig. 3. Microscopic examination revealed broken, fusiform and expanded nodes on the proximal portion of the hair (Original magnification ×100).

Fig. 4. On scanning electron microscopic examination, the node had a smooth surface covered with normally imbricated scales of hair cuticle cells, and no internodal twisting or grooving was found.

Expected that this hair structure abnormality might be associated with iron deficiency anaemia.

After taking oral iron supplement for 4 months, her hemoglobin value increased to 10.3 g/dL.

Interestingly, at the same time, the structural abnormality of the proximal hair segment was corrected, and excessive hair loss was no longer found.

**DISCUSSION**

Iron deficiency as an etiologic factor in diffuse alopecia has been discussed previously. But, hair structure abnormalities associated with iron deficiency remain not well acknowledged. So far several structural abnormalities of anagen hair have been reported in iron deficiency, including brittle, lusterless and dry hair, focal narrowing or splitting of the hair shaft, and changes in the distribution of pigment. These alterations of the hair shaft have been implicated on the basis of impaired keratin production resulting from iron deficiency. Recently, Karaman et al. reported localized monilethrix with improvement after treatment of iron deficiency anaemia.

Our patient had a structural defect of the hair shaft, which showed expanded and fusiform nodes proximally. The node had a similar appearance to that seen in monilethrix. Monilethrix is a rare autosomal dominant disorder, although autosomal recessive cases have been reported, in which hairs
show elliptical nodes with regular periodicity of 0.7-1 mm separated by tapered constrictions. Our patient had no positive family history and the morphology of her hair was different from that of monilethrix. Bentley-Phillips and Bayles described the term pseudomonilethrix, which is now believed to be artefactual in nature. In pseudomonilethrix, microscopic changes can be produced in normal hairs by trauma from tweezers or forceps, or from compressing overlapping hairs between two slide glasses. The same change can occur by pulling out the hair while taking samples for microscopic examination. Our patient denied any physical trauma such as excessive combing or brushing, the samples examined were taken by cutting gently without forceps to avoid artifact, and examined individually to avoid the indentation in one hair shaft caused by another overlying hair.

Although at first glance our patient appeared to have androgenetic alopecia or telogen effluvium, we could find neither family history nor signs of hormonal influence. We could not be convinced that hair structure abnormalities in our patient resulted from iron deficiency anaemia. However, the interesting point is that alopecia and hair structure abnormality had developed simultaneously with the occurrence of iron deficiency anaemia, and that they improved simultaneously with the correction of anaemia after iron administration. Therefore, we believe that this hair structural abnormality was associated with iron deficiency anaemia, although its mechanism cannot be described definitively.

REFERENCES