Zinc Deficiency Associated with Hypothyroidism: An Overlooked Cause of Severe Alopecia

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Abstract

Hypothyroidism is a common and well recognized cause of diffuse hair loss. Zinc and other trace elements such as copper and selenium are required for the synthesis of thyroid hormones, and deficiency of these can result in hypothyroidism. Conversely, thyroid hormones are essential for the absorption of zinc, and hence hypothyroidism can result in acquired zinc deficiency. The hair loss attributed to hypothyroidism may not improve with thyroxine unless zinc supplements are added, as demonstrated in our case.

Keywords: Alopecia, hypothyroidism, zinc deficiency

INTRODUCTION

The metabolism of zinc and thyroid hormones are closely interlinked. We describe a patient with a concurrent onset of hypothyroidism and zinc deficiency who presented with severe diffuse hair loss, and briefly discuss the association.

CASE REPORT

A 28-year-old woman presented with weakness, lack of appetite, diffuse hair loss, and asymptomatic scaly skin lesions of 2 months duration. There was no history suggestive of connective tissue disease or malabsorption. On physical examination, the patient appeared depressed. She had pallor and puffiness of the upper part of the face. There was diffuse, dry scaling over the face and extremities, along with annular, erythematous, scaly patches over the nape of the neck. The notable finding on the scalp was profound diffuse alopecia [Figure 1] with marked hair loss over the occipital area [Figure 2]. There was also severe thinning of the hair of the eyebrows and eyelashes [Figure 3]. A hair pull test done from five different sites on the scalp was positive. Light microscopic examination of the hair revealed telogen type hair roots without any hair shaft abnormalities. Thyroid function tests were as follows: T3 70 ng/dl (normal 80-200), T4 2 mcg/dl (normal 4-12), and Thyroid Stimulating Hormone 64 IU/ml (normal 0.30 -4.5). All routine investigations including antinuclear antibody profile were within normal limits, except for a low hemoglobin level of 9 g/dl. Histopathological examination of an annular scaly lesion showed minimal epidermal hyperplasia with a mild perivascular inflammatory infiltrate.

Thyroid hormone supplements were started, but the scaly lesions persisted and there was no detectable improvement in the hair loss. Since a definite diagnosis of the annular scaly lesions could not be made, plasma zinc level was estimated, which was 62 mcg/dl (normal 66-144 mcg/dl and optimal levels 90-150...
Zinc monohydrate 140 mg capsules containing 50 mg of elemental zinc was given twice a day, along with other multivitamins. One month later, the skin lesions had cleared completely, and there was such a remarkable improvement in her mental depression and facial puffiness, that her facies was totally unrecognizable when compared to her previous appearance [Figure 4]. Subsequent follow-up demonstrated complete regrowth of hair over a course of 4 months [Figure 5].

DISCUSSION

Acrodermatitis enteropathica usually presents in children as a triad of dermatitis, diarrhea, and alopecia. Conditions that cause acquired zinc deficiency include inadequate intake, parenteral nutrition, pregnancy and lactation, extensive burns, exfoliative dermatitis, intestinal malabsorption syndromes, cystic fibrosis, alcoholism, HIV infection, malignancies, and chronic renal disease.[2] The formation of insoluble complexes with calcium, fiber, and phytates markedly decrease the intestinal absorption of zinc.

The diagnosis of acquired zinc deficiency is often missed. It may present as psoriasiform, annular or crusted plaques, with decreased hair and nail growth. Hair changes include alopecia, loss of pigment, dryness, brittleness, hair shaft, and structural abnormalities leading to fragility and breakage. The hair loss starts in the occipital region and may extend over the rest of the scalp. Eyebrows and eyelashes may also be involved. In severe cases, there may be total loss of hair and nails. Nail manifestations include Muehrcke’s nails, transverse leukonychia, brittle nails, onychorrhexis, and Beau’s lines.[3]

Levels of zinc either in plasma or serum are not reliable indicators for establishing a diagnosis of zinc deficiency. Normal values may be obtained in the presence of subclinical zinc deficiency. Therapeutic response in suspected cases remains the gold standard of diagnosis.[1] Decreased levels of zinc may indicate a shift of zinc from plasma to another body pool. Serum copper levels tend to fluctuate inversely with serum zinc levels, and it has been suggested that the zinc-copper ratio may be used as an index of zinc levels. The concentration of zinc in hair is a more reliable indicator of chronic zinc deficiency.[4] Hair is a readily accessible specimen as well. A sample of hair cut close to the scalp from the back of the head or the nape of the neck is required. Normal values range from 150 to 240 mcg/g of hair. Levels of lesser than 70 mcg/g are indicative of zinc deficiency. The concentration of zinc in melanosomes range from 641 to 687 mcg/g, or about five times the concentration for the hair shaft as a whole.[5] The concentration of zinc in hair depends not only on the delivery of zinc to the root, but also on the rate of hair growth. The fact that zinc deficiency itself may impair the growth of hair has to be kept in mind. Though susceptible to environmental contamination from exogenous sources, the concentrations of copper and zinc in hair have not been found to be altered significantly by exogenous processes, and serve as useful bio-indicators.[6]

Several reports suggest that zinc deficiency is a cause of subclinical hypothyroidism. In Down’s syndrome, early thymic involution is associated with low serum zinc levels, and thymic function has been shown to be in relation to the pituitary-thyroid axis. Zinc supplementation improved thyroid function in nine patients with low zinc levels.[7] In addition, zinc is required for the T3 receptor to adopt it’s biologically active confirmation. Some of the effects of zinc deficiency therefore, may be due to loss of zinc from the T3 receptor and impairment of T3 action.[8] The intracytoplasmic zinc levels, which are only partially represented by the total plasma levels, would be more relevant for this effect.[9] Conversely, hypothyroidism reduces intestinal zinc absorption in rats.[10] Hence, an attempt to link the low levels of thyroid hormones and zinc is worthwhile.

In our patient, zinc deficiency may have contributed significantly to the development of hypothyroidism. Her mental depression, taste acuity, appetite, skin lesions, and hair loss showed a dramatic improvement not on initiation of thyroxine treatment, but with zinc supplementation. The effect of hypothyroidism on the metabolism of zinc should also not be overlooked. An evaluation for features of zinc deficiency, which is often under-recognized, is warranted in all cases of hypothyroidism.
REFERENCES


Figures and Tables
Figure 1

Diffuse alopecia of the scalp
Figure 2

Annular, erythematous, and scaly patches over the nape of neck
Figure 3

Scaling of the face with loss of eyebrows and eyelashes
Complete alteration of the appearance following thyroxine and zinc supplementation
Figure 5

Subsidence of the skin lesions and regrowth of hair following zinc supplementation

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