—Case Reports—

Graves' Disease Associated with Alopecia Areata Developing after Hashimoto's Thyroiditis

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Abstract

Graves' disease and Hashimoto's thyroiditis are the most common autoimmune thyroid diseases. Hypothyroidism can develop in patients with Graves' disease, either spontaneously or as a result of radioactive iodine therapy or surgery. However, it is rare for patients with Hashimoto's thyroiditis to subsequently develop Graves' disease. We report a case of alopecia areata associated with Graves' disease in a 41-year-old woman who had previously been diagnosed with Hashimoto's disease. Alopecia areata is an autoimmune disease associated with other autoimmune diseases such as thyroid disorders, anemia, and other skin disorders.
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Key words: alopecia areata, Graves' disease, Hashimoto's thyroiditis

Introduction

Autoimmune thyroid diseases are characterized by lymphocytic infiltration of the thyroid gland. The most common autoimmune thyroid diseases are Graves' disease and Hashimoto's thyroiditis. In patients with Graves' disease hypothyroidism can develop spontaneously or as a result of medical treatment, such as radioactive iodine therapy or surgery. However Graves' disease rarely develops after Hashimoto's thyroiditis. Alopecia areata (AA), which is characterized by hair loss without scarring, is most often associated with other autoimmune diseases, such as thyroid disorders, anemias, and other skin disorders with autoimmune etiology (Champion et al.[1]-Takasu et al.[2]-Yamasaki et al.[3]). We present a 41-years-old woman with AA associated with Graves' disease who was previously found to have Hashimoto's thyroiditis.

Case Report

A 41 year-old woman was referred to our clinic 6 year ago because of weight gain, constipation, weakness and somnolence. Results of laboratory studies were as follows: thyroid-stimulating hormone (TSH) 7.95 IU/mL (normal range: 0.2–4.2 IU/mL), free-T4 0.97 pmol/L (normal range 0.93–1.7 pmol/L), anti-thyroid peroxidase (TPO): 551 IU/mL (0–34 IU/mL), anti-thyroglobulin: 332 IU/mL (0–115 IU/mL). Ultrasonograpy of the thyroid demonstrated a

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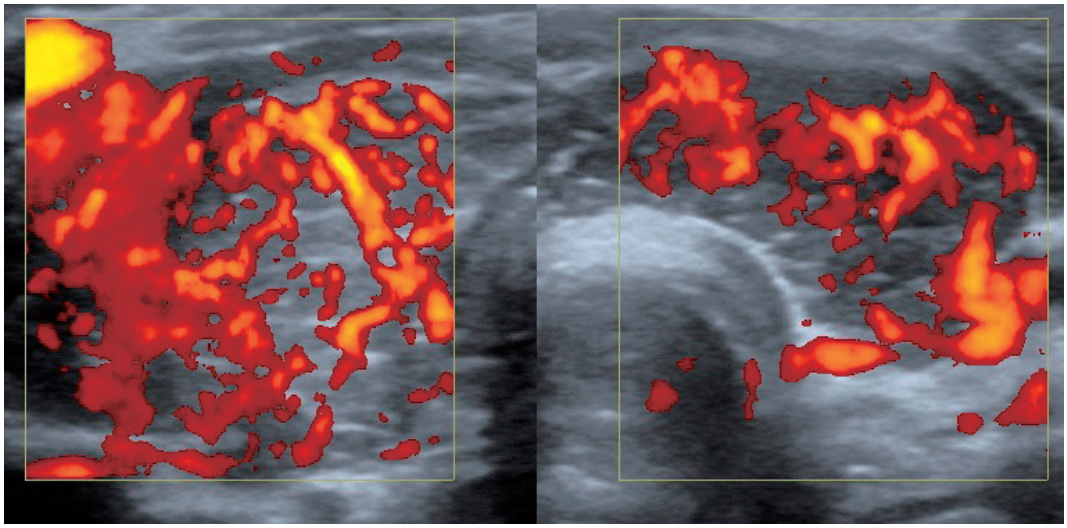

Fig. 1 Power doppler ultrasonograpy showing thyroid inferno.

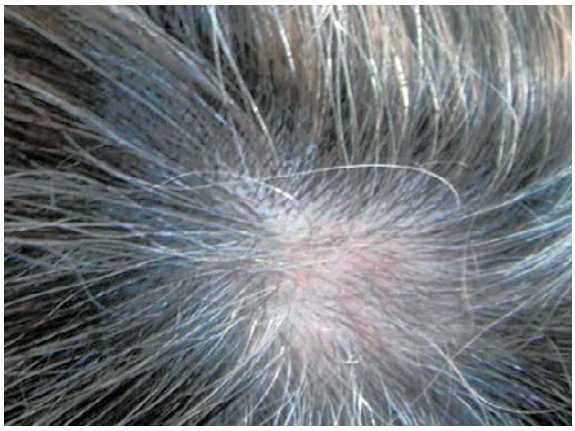

Fig. 2 Alopecia areata of the scalp.

diffuse hypoechoic pattern and a volume within the normal ranges (17 mm^3). After Hashimoto's throiditis was diagnosed, treatment with L-thyroxine (75 μg) was started and continued the same dose. Thyroid hormone levels were within normal limits for the subsequent 6 years.

However 4 months ago, the patients was admitted to our clinic with complaints of diaphoresis, palpitations, discomfort, weight loss, trembling of the hands, and diarrhea. Furthermore the pulse rate was 115/beats per minute. The levels of thyroid hormones were as follows: TSH: <0.005 IU/mL, free T3: 17.01 pmol/L, free T4: 4.35 pmol/L, anti-TPO: 381 IU/mL, antithyroglobulin : 84 IU/mL. Ultrasonograpy of the thyroid showed a diffuse hypoechoic pattern and a volume (8.78 mm^3) less than that 6 years earlier. Treatment with L-thyroxine was stopped and propranolol was started. After 1 week, TSH was <0.005 IU/mL, free T3 was 14.34 pmol/L and free T4 was 4.06 pmol/L. Thyroid scintigraphy showed increased diffuse ly increased uptake of radioactive iodine. Power Dopple ultrasonograpy r of the thyroid gland showed a pattern of thyroid inferno **(Fig. 1)**. The level of TSH receptor antibodies was >50 U/L (0–10 U/L). Treatment was propylthiouracil was started. Also, AA was observed during this period **(Fig. 2)**. Topical corticosteroids were applied. The AA improved with treatment and finally resolved completely after anti-thyroid treatment. The patient is still being followed up as an outpatient and being treated with anti-thyroid drugs.

Discussion

Graves' disease and Hashimoto's have many common immunologic features. The underlying pathogenesis of these conditions have not been clarifiedt. TSH receptor antibodies play an important role in the pathogenesis of Grave' disease. Two types of TSH antibody have been defined in Graves' disease stimulating TSH receptor antibodies (TSabs) and blocking TSH receptor antibodies (TBabs) (Morgenthaler et al.[4]). In Graves' disease, TSabs are dominant, but TBabs may also occur. Yamasaki et al., have demonstrated both TBabs and TSabs in a

patient with Hashimoto's thyroiditis. Although TSabs can be detected in Hashimoto's thyroiditis, their effects may be masked because of the destructive process in autoimmune hypothyroidism. This situation has previously been reported in several cases of Hashimoto's disease that converted to Graves' disease.

Even if Hashimoto's thyroiditis presents with an enlarged thyroid gland, diffuse atrophy can gradually occur. However, in the conversion to graves' disease, a few residual healthy thyroid cells may be found that can respond to TSH receptor antibodies. However, thyroid ultrasonograpy in our patient showed a diffuse hypoechoic pattern and a normal size. After the conversion to Graves disease, thyroid ultrasound showed an echoic pattern with diffusely changes and diffuse increased vascularity without enlargement of the gland. Therefore, the mentioned hypothesis for our case will be invalid.

Although AA leads to recurrent non-scarring hair loss, that can affect any area of the body, its etiology is unclear. However, AA has been suggested to be an auto-immune disease, caused by an abnormal T-cell response against hair follicle self-antigens (Tobin et al.[5]). The process is similar to T-cell mediated diseases such as Graves's disease and Hashimoto's thyroiditis (Barahmani et al.[6], Puavilai et al.[7]). The relationship between thyroid diseases and AA have been demonstrated by several studies Also, the presence of antithyroglobulin and anti-TPO antibodies is associated with AA. However, there is not enough information about TSH receptor antibodies. In previous studies, AA was found to associated with hypothyroidism rather than hyperthyroidism (Barahmani et al.[6], Thomas et al[8]). Interestingly, our patient did not show any sign of AA during the time of Hashimoto's thyroiditis. However, AA developed after the conversion to Graves' disease. Also, AA was started to resolve as hyperthyroidism resolved. Therefore we believe that TSabs might be associated with AA.

Conclusion

Although AA has been seen reported in concomitant Hashimoto's thyroiditis and Graves' disease, this case is, to our knowledge, the first of AA associated with Graves' disease developing after Hashimoto's thyroiditis.

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