Alopecia Areata Associated with Graves’ Disease

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Abstract

Alopecia areata (AA) is usually characterized by areas of patchy hair loss on the scalp, although in severe cases, the total loss of scalp or body hair may occur. While the association between AA and Graves’ disease is described in textbooks, few reports have been published. We herein report a case of AA associated with Graves’ disease, in which a skin biopsy revealed marked perifollicular infiltration of mononuclear cells and pigment incontinence in the dermis. Slight staining of IgG and IgM was observed in the follicle on a direct immunofluorescence test. Pulse corticosteroid therapy followed by contact immunotherapy with squaric acid dibutylester (SADBE) was effective.

Introduction

Alopecia areata (AA) is recognized to be sometimes associated with thyroid disease.¹−² However, there have been few reports of patients with AA associated with Graves’ disease have been reported,³ and the pathogenesis of AA in patients with Graves’ disease is unknown. We herein report a case of AA associated with Graves’ disease. This case was considered to belong to autoimmune polyglandular syndrome (APS) that encompasses multiple autoimmune diseases.⁴

Case report

A 34-year-old male noted areas of hair loss six months prior to his first visit, which had slowly increased in both size and number. He had also noticed general fatigue and a low-grade fever for 50 months. There was no significant family history, however, he had suffered from atopic dermatitis since the age of 8. A physical examination revealed multiple areas of AA (Fig. 1a) and a swollen thyroid gland (Fig. 1b). Easy hair loss, black dots and tapering hair were observed on dermoscopy. The results of laboratory examination were as follows: red blood cell count, 581×10⁶/ml (normal range: 420–570); white blood cell count, 3,900/ml (normal range: 4,000–9,600); TSH, <0.05 µU/ml (normal range: 0.35–4.94); freeT4, 3.38 ng/dl (normal range: 0.70–1.48); freeT3, >26.0 pg/ml (normal range: 1.71–3.71); antithyroid microsomal antibodies, ×6,400 (normal range: <100); anti-TSH receptor antibodies, 5.9 (normal range: 0–1.99). The levels of antithyroglobulin antibodies, antinuclear antibodies, HbA1c, RPR and TPHA were all within the normal limits or negative. We suspected a diagnosis of AA associated with Graves’ disease and performed a skin biopsy. A histopathological examination showed peribulbar lymphocytic infiltration with pigment incontinence within the follicular papilla (Fig. 2a). Clumps of pigment were also noted in the stelae (Fig. 2b).
2b), and faint staining of IgG and IgM was observed in the follicle on a direct immunofluorescence test. We referred the patient to an endocrinologist, and the patient was diagnosed as having Graves’ disease. Treatment with thiamazole (15 mg/day) improved his systemic symptoms. Furthermore, the free T3, T4, TSH and TSH receptor antibody titers returned to the normal limits after three months’ treatment. After 4 months of treatment with thiamazole, the lesions of AA unchanged with broken and tapering hairs. Therefore, we started one cycle of pulse corticosteroid therapy at a dose of 0.5 g for three days. As the lesion was partially improved, subsequent contact immunotherapy with SADBE resulted in the complete restoration.

Discussion

Thyroid disease is recognized to be most frequently associated with AA. Muller and Winkelmann found evidence of thyroid disease in 8% of 736 AA patients, compared with less than 2% in the control population in North America. However, Puavilai S. et al. reported that the prevalence of positive microsomal antibodies was 4.6% among AA patients, which was not statistically distinct from that in the control group, suggesting that AA and Graves’ disease may develop coincidentally.

The association between thyroid disease and AA is thought to be related to the onset of APS. Our patient was affected with Graves’ disease and AA; therefore, his condition can be categorized into APS type 3C (autoimmune thyroid disease and skin, neural or neuromuscular autoimmune disease). The direct immunofluorescence test exhibited slight staining of IgG and IgM in the matrix of our patient. The staining pattern resembled that of melanocytes in the hair matrix, which are postulated to be a target of AA.
Nevertheless, the pathogenicity of the deposited immunoglobulin is unknown.

AA of our patient did not respond to thiamazole, indicating that the elevated level of thyroid hormone itself did not play a pathogenic role in AA. Based on lymphocytic infiltration around the hair follicle, we started pulse steroid therapy and subsequent contact immunotherapy. Taking into account the patient’s response to the treatment, immunological abnormality was primarily responsible for the development of AA associated with Graves’ disease.

References