Alopecia in preexisting autoimmune thyroid disease in family medicine practice: can hyperprolactinemia induce hair loss? A case report

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Abstract

Management of hair loss in women presents several challenges for general practitioners, who are the first in identifying its cause and consequences in everyday clinical practice. It is usually associated with multiple secondary factors, including endocrine disorders, drug side effects, and physical or emotional stress. We report a possible pathophysiological link between hyperprolactinemia and alopecia in a patient with preexisting autoimmune thyroid disease, which has not been documented in a significant number in the literature. A 27-year-old female patient with a previous history of an autoimmune thyroid disease on hormone substitution therapy presented to a family doctor with signs of frontal alopecia that had started several months previously. On examination, frontal alopecia was confirmed. Laboratory results and thyroid ultrasound confirmed autoimmune thyroid disease, with reduced parathyroid hormone and elevated prolactin. Her female sex hormones were in the normal range. Due to elevated prolactin levels, computed tomography of the pituitary gland was performed, which excluded any brain pathology. Based on available data, there is no published systematic analysis on hyperprolactinemia-induced alopecia in previous autoimmune diseases (large cohort studies). This report indicates that, due to moderately elevated prolactin values, consideration of alternative causes and further diagnostics are necessary to exclude a prolactin-producing tumor of the pituitary gland.

Keywords: alopecia, autoimmunity, hair loss, hyperprolactinemia

Introduction

It has previously been reported that alopecia affects both sexes at various ages, and it can appear anywhere on the body. It may affect body image and self-esteem and many patients experience the absence of positive psychological wellbeing (1). Androgenetic alopecia (AGA) is the most common presentation of hair loss, and epidemiological data indicate that around 50 million men and 30 million women in the United States suffer from AGA (2, 3). Even though the age of onset has been documented as usually around 40 years, there are reports indicating that alopecia may begin much earlier, at age 30 (4, 5). In the literature so far, many potential causes have been associated with hair loss in women. Patient history and clinical examination of hair loss are helpful in revealing the potential cause of alopecia, but, on the other hand, overlapping characteristics may appear and present severe difficulties in its management. Many different types of endocrine disorders may play a role in hair loss, including thyroid disorders. The majority of patients with severe hypothyroidism have an underlying autoimmune thyroid disease. Isolated or acquired hypoparathyroidism may occur as an autoimmune disorder, either alone or in association with other autoimmune diseases such as autoimmune polyendocrinopathy–candidiasis–ectodermal dystrophy (APECED) (6). Furthermore, there are other rare autoimmune conditions that may manifest with hair loss, such as systemic lupus erythematosus (SLE). Hyperprolactinemia presents a challenge in everyday clinical practice, considering the consequences of pharmacological alterations in the pathways that control prolactin secretion. Other medical conditions, such as metabolic or neoplastic disorders, should also be considered (7, 8). There are several other causes associated with hyperprolactinemia, including drug side effects and other pituitary disorders. Female androgenetic alopecia (FAGA) is still a common cause of non-scarring alopecia in women, with no clear role of androgens in a pathophysiological mechanism. Nevertheless, FAGA can be observed in women with hyperandrogenism due to endocrinological diseases, including polycystic ovarian syndrome (PCOS), hyperprolactinemia, adrenal hyperplasia, and ovarian and adrenal tumors (9). Furthermore, disruptions of immune regulation may result in autoimmune polyglanulad tumours syndrome (APS), and alopecia may be associated with this condition. However, there is no strong evidence that moderately elevated prolactin levels may promote hair loss, especially in women (10, 11). We present a case report that considers preexisting autoimmune thyroid disease associated with hyperprolactinemia as the cause of alopecia.

Case report

A 27-year-old female patient, working as a secretary, unmarried, and with no previous pregnancy, presented to a family doctor with signs of frontal alopecia (hair thinning in the frontal and parietal regions of the head, preserving the anterior hair implantation line) that had started several months previously. Her eyelashes and eyebrows were normal. According to Olsen’s classification, her condition belonged to Class 2 (12). The patient had a history of Hashimoto’s thyroiditis, was on levothyroxine substitution therapy of 75 μg per day, and had no history of similar illness in the family and no history suggestive
of systemic disorders. Her menstrual cycle was normal. On examination, the hair loss started as a small patch and gradually increased in length predominantly in the frontal part, with no sign of an annular pattern. Physical examination showed a slightly palpable thyroid gland, with palpable superficial cervical lymph nodes, less than 1 cm in size. Laboratory results showed a normal complete blood count, slightly elevated sedimentation rate (27 mm/hr, normal ranges 4–24 mm/hr), slightly elevated thyroid-stimulating hormone (TSH; 4.40 mU/l, normal ranges 0.27–4.20 mU/l), elevated antithyroglobulin antibodies (250 IU/ml, normal ranges < 40 IU/ml), elevated anti-thyroid peroxidase antibodies (TPO; 350 kIU/l, normal ranges < 34 kIU/l), normal values of free triiodothyronine and thyroxin, and elevated immunoglobulin E (144.4 kIU/l). Other laboratory results showed low parathyroid hormone (13.34 pg/ml, level ranges 16–65 pg/ml), elevated serum levels of vitamin D (93.5 nmol/l, normal ranges 50–75 nmol/l), slightly elevated serum calcium levels (2.57 mmol/l, normal ranges 2.14–2.43 mmol/l), normal phosphate levels, normal lactate dehydrogenase levels, and elevated prolactin levels (607.5 mIU/l, normal ranges 102–496 mIU/l). A rapid strep test was negative. Thyroid and neck ultrasound showed overactive thyroid tissue, with homogeneously hypoechoic parathyroid glands and with no signs of nodes resembling a tumor. Her female sex hormones were in the normal range. Due to elevated prolactin levels, computed tomography of the pituitary gland was performed and showed no signs of brain pathology. The goal of therapy was to increase hair regrowth and to stop the progression of hair loss. The patient was treated with topical minoxidil 5% foam daily for the next 3 months. After 3 months of therapy, there were no signs of recovery, and the laboratory results were similar to the previous ones. The long-term treatment plan for this patient was a multidisciplinary approach involving various specialists (dermatologists and endocrinologists) and follow-up every 3 to 6 months with a laboratory check.

**Discussion**

Hair loss is a frequently underestimated condition in general medical practice due to the complex and difficult understanding of overlapping characteristics. Because most women with frontal alopecia do not have hyperandrogenism, the European consensus held in 2011 recommended a free androgen index (FAI) and prolactin dosage as screening tests in management (13). Crucial factors in the management of alopecia also include the duration and pattern of hair loss, the patient’s present and past medical conditions, diet, medications, and family history. A systematic approach may be helpful in everyday clinical practice, but in some cases there are difficulties in the proper recognition of common causes, and hyperprolactinemia may be among them. Even though hyperprolactinemia is not a rare endocrine disorder of the hypothalamic–pituitary axis, there is some pathophysiological complexity, especially in underlying autoimmune diseases. Many autoimmune diseases, such as SLE, rheumatoid arthritis, Sjögren’s syndrome, Hashimoto’s thyroiditis, multiple sclerosis,
and psoriasis, are suggested to be associated with high prolactin levels and possible hair loss (14).

Previous studies have also confirmed the association between autoimmunity and hypoparathyroidism because antibodies against parathyroids were detected in isolated hypoparathyroidism (15). A study by Lyakhovitsky et al. also demonstrated a significant association between alopecia areata and thyroid abnormalities, and it proposed screening for thyroid function and anti-thyroid autoantibodies (16).

Previous reports on hormone analysis in androgenetic hair loss in woman showed somewhat contradictory results, which may favor interactions between hypothyroidism and androgen metabolism (17). However, pathophysiological complexity could be due to the agent triggering autoimmune thyroid disease, and so it is possible that hyperprolactinemia precipitates the onset of preexisting autoimmune thyroid disorder. Previous reports also showed that patients with hyperprolactinemia had a higher prevalence of autoantibodies, elevated serum levels of interleukin 4 and 6, and antibody-secreting B cells. These findings imply that hyperprolactinemia may be associated with autoimmune diseases (18).

Regular thyroid function testing and measurement of TPO antibodies should be performed in patients with a positive family history of autoimmune disorders. Schmidt et al. performed a study involving 31 female patients suffering from AGA and suggested that TSH and prolactin may interact with androgen metabolism (19).

Conclusions

Our report is highly specific because, based on available data, there are only five reports that studied an association between hair loss and hyperprolactinemia, and with no focus on the presence of underlying autoimmune diseases (11, 19, 20, 21). Furthermore, we express the need for a more comprehensive understanding of hair loss, which includes broad-range hormone analysis to elucidate all possible pathophysiological mechanisms. We also propose considering other causes (i.e., the agent triggering autoimmune disease), as shown in Figure 1. Finally, special attention should be paid to proper evaluation of prolactin levels in such patients in order to exclude underlying endocrinopathy or malignancy.

References