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# Localized alopecic myxedema of the scalp

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## Abstract

Myxedema is a rare, cutaneous complication of autoimmune thyroid diseases that most often affects the anterior shins. Herein, we report a patient with a history of Graves disease and Hashimoto thyroiditis who presented with boggy, alopecic patches associated with scalp pruritus. Punch biopsies from these lesions showed increased interstitial mucin in the reticular dermis, consistent with localized myxedema. This report showcases a rare presentation of localized myxedema of the scalp, highlighting the diverse cutaneous manifestations of autoimmune thyroid diseases.

*Keywords: alopecia, Graves disease, Hashimoto thyroiditis, myxedema, scalp pruritus*

## Introduction

Localized myxedema (also known as thyroid dermopathy) is a rare complication of autoimmune thyroid disease that can occur in patients with hypothyroidism, hyperthyroidism, and euthyroidism including Hashimoto thyroiditis and Graves disease. However, localized myxedema is most commonly found in patients with hyperthyroidism, affecting 0.5-4.3% of patients with a history of Graves disease [1]. Localized myxedema results from the accumulation of glycosaminoglycans in the dermis and subcutaneous layer of the skin [2]. Fibroblast-produced hyaluronic acid is the main glycosaminoglycan in localized myxedema.

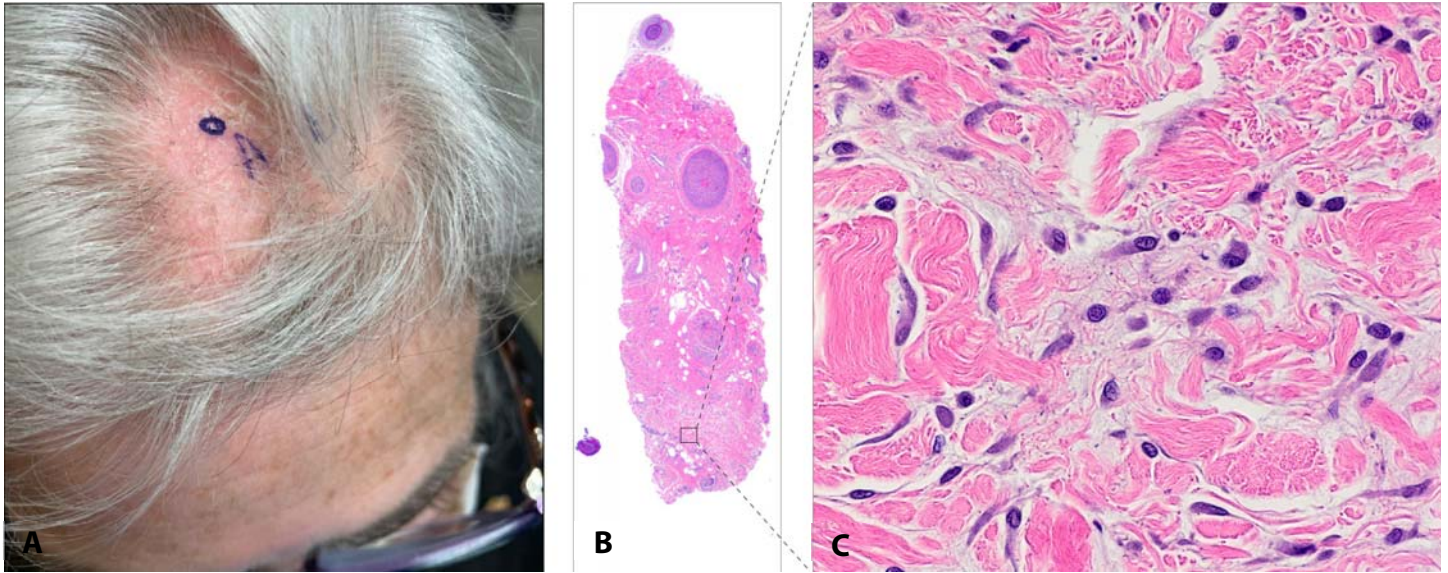
The underlying pathogenesis of localized myxedema is not well-understood. One theory is that

autoantibodies may activate fibroblasts, thereby stimulating the production of glycosaminoglycans [2]. Clinically, localized myxedema presents bilaterally with a “boggy” thickening of the skin as well as nodule and plaque formation; lesions classically have “waxy” swelling and induration [3].

Myxedematous lesions of the skin most often appear on the anterior aspects of the legs and dorsum of the feet [4]. They are often asymptomatic; targeted treatment to cutaneous lesions is usually reserved for symptomatic cases. Outside of the lower extremities, however, myxedema is comparatively rare. Herein, we report a 66-year-old woman with a history of both Graves disease and Hashimoto thyroiditis who developed chronic, pruritic plaques on the scalp that showed histologic features consistent with myxedema.

## Case Synopsis

A 66-year-old woman presented to our dermatology clinic with a one-year history of localized scalp pruritus associated with progressive hair loss. Her past medical history was significant for a diagnosis of Graves disease 12 years prior, which was managed with methimazole. Two years after this diagnosis, she developed Hashimoto thyroiditis, which had since been treated with a consistent dosage of levothyroxine (88mcg each morning). Her scalp pruritus had been unresponsive to high potency topical corticosteroids. Physical examination revealed two boggy, alopecic plaques with overlying lichenification on the anterior scalp (**Figure 1A**). Her lower extremities were unremarkable.



**Figure 1.** Clinical and histopathological findings. **A)** Anterior scalp with boggy, alopecic plaque with overlying lichenification. The blue circle in the alopecic plaque represents the punch biopsy site, which was labeled as A. H&E histopathology: **B)** horizontal section of the punch biopsy from **A)** is shown. The square area in **B)** is expanded in panel **C)**, 20x; **C)** close-up of highlighted area shown in **B)**, which features reticular dermis with increased interstitial mucin, .200x.

Punch biopsy of the anterior scalp plaque was performed. Horizontal and vertical sections revealed increased interstitial mucin in the reticular dermis, visible using hematoxylin and eosin staining (**Figure 1C**). Her clinical history and histopathological features of the scalp biopsy supported the diagnosis of localized myxedema.

At her follow-up visit, the plaques were injected with intralesional triamcinolone (1ml, 40mg/ml) in an attempt to relieve her symptoms of pruritus. The patient underwent monthly injections for three months and noted significantly decreased bogginess, edema, and scalp pruritus. She also endorsed increased hair regrowth in the injected areas that were previously alopecic.

## Case Discussion

Localized myxedema most often appears in the pretibial area and dorsum of the feet [4]. It has also been reported to occur rarely in other anatomic regions, including the fingers and upper extremities [4,5]. Localized myxedema of the scalp is less common and has been only reported in two patients (**Table 1**), [6,7]. Both these previously reported patients were female and in their 6<sup>th</sup> decade of life at the time of diagnosis. The ethnicity of the first

patient was reported as Hispanic, whereas that of the second patient was not reported.

Amongst the previously reported patients, one was reported to have a history of Graves disease whereas a past medical history of thyroid disease was not identified in the second patient. In our report, we have described a woman with both a history of Graves disease and Hashimoto thyroiditis.

Notably, localized myxedema has been mostly reported in patients with Graves disease; however, it can also occur in Hashimoto thyroiditis [4]. In our case, the patient had been diagnosed with both conditions, although was exhibiting hypothyroidism during the onset of her myxedema. This is consistent with previous reports that suggest the development of localized myxedema is not necessarily related to the thyroid status of the patient; it can develop in patients with hypothyroidism, euthyroidism, and hyperthyroidism [8].

The two previously reported patients were found to have scalp thickening for years as well as scalp tenderness. However, they did not have alopecia. In contrast, our patient developed alopecic and pruritic plaques on the scalp associated with scalp myxedema highlighting the unique features of our case.

**Table 1.** Characteristics of previous patients with myxedema of the scalp.

|                             | Patient 1 (Reference 7)   | Patient 2 (Reference 8)   | Our Patient  |
|-----------------------------|---|---|--|
| <b>Demographics</b>         |   |   |  |
| Age                         | 59  | 51  | 66   |
| Gender                      | Female  | Female  | Female   |
| Race                        | Hispanic  | Not reported  | White  |
| <b>Past medical history</b> |   |   |  |
| Thyroid disease             | None identified   | Graves' disease   | Graves' disease, Hashimoto thyroiditis   |
| Duration of thyroid disease | None identified   | Not reported  | 12 years   |
| <b>Present Illness</b>      |   |   |  |
| Symptoms                    | Scalp thickening since childhood. Scalp tenderness for 4 years. Physical examination: enlarged and multinodular thyroid gland | Scalp thickening for years. Sudden onset pain and swelling in scalp   | Localized scalp pruritus, progressive hair loss  |
| Duration of alopecia        | No alopecia   | No alopecia   | One year   |
| Location of alopecia        | None  | None  | Scalp  |
| Pathology                   | Increased fibroblasts. New collagen formation. Increased separation of collagen fibers  | Interstitial mucin deposition in the dermis and superficial subcutaneous layer. Clear spaces surrounding the collagen bundles in the dermis   | Increased interstitial mucin in the reticular dermis.                                      |
| Lab studies                 | Negative for thyroid-disease related auto-antibodies  | Normal thyroid stimulating hormone (TSH) levels. Elevated titers of anti-TSH receptor, antithyropoxidase and antithyroglobulin autoantibodies | Most recent TSH was within normal range while patient was on 88 mcg of levothyroxine daily |
| Imaging                     | X-rays: Scalp thickening  | Ultrasound and MRI: increased thickness of scalp cutaneous and subcutaneous tissue  | None   |
| Treatment                   | Desiccated thyroid  | hyaluronidase injection   | Intralesional triamcinolone  |
| Response to treatment       | Protein-bound iodine of 7 $\mu$ g/100 cc maintained after 1 year  | Decreased dermal-epidermal thickness and decreased scalp pain   | A decrease in boggy, edema, and scalp pruritus. Increased hair regrowth                    |
| Complications               | Not reported  | None  | None   |

The histopathological findings of the previously reported cases showed increased fibroblast and new collagen formation (Patient 1) and increased mucin deposition in the reticular dermis (Patient 2). Indeed, we found similar histopathological findings in our patient (increased interstitial mucin in the reticular dermis).

Amongst the previously reported patients, one showed elevated anti-TSH receptor, antithyropoxidase, and antithyroglobulin autoantibodies whereas no thyroid-disease related auto-antibodies were found in the other patient. Imaging studies of both reported patients show scalp thickening.

In terms of therapy, the first reported patient was treated with desiccated thyroid extracts, resulting in a maintenance of normal protein-bound iodine levels for one year. The second reported patient was treated with hyaluronidase injection, which reduced both dermal-epidermal thickness and scalp pain. As discussed above, our patient was treated with intralesional triamcinolone with improvement.

Topical and intralesional corticosteroids are recommended as the first line treatments for cutaneous involvement due to their presumptive ability to reduce both inflammation and ectopic collagen formation [9]. Recalcitrant cases have been

approached using modalities such as pentoxifylline, rituximab, and intravenous immunoglobulin [10,11].

## Conclusion

We report a rare occurrence of localized, pruritic myxedema on the scalp in a patient with a history of

both Graves disease and Hashimoto thyroiditis. This case contributes to the diversity of the cutaneous manifestations of autoimmune thyroid diseases.

## Potential conflicts of interest

The authors declare no conflicts of interest.

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