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Minoxidil-Induced Allergic Contact Dermatitis in a Young Woman with Subclinical Hypothyroidism and Micronutrient Deficiencies: A Case Report

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***Abstract*-- Background:** Topical minoxidil remains a cornerstone treatment for androgenetic alopecia and other alopecias, but cutaneous adverse effects, particularly allergic contact dermatitis (ACD), are increasingly recognized. Although propylene glycol (PG) has long been considered the main culprit, recent high-quality evidence suggests that minoxidil itself is more often responsible. **Case Presentation:** We report the case of a 28-year-old female with subclinical hypothyroidism, iron deficiency anemia, and vitamin B12 and D deficiencies who developed delayed ACD after prolonged use of 5% topical minoxidil solution. After approximately 50 days of therapy, she experienced pruritus of the ears, followed within five days by an erythematous, pruritic rash on the face, neck, and back. Minoxidil was discontinued immediately, and the patient initiated self-treatment with cetirizine and moisturizers, later receiving physician-prescribed antihistamines and topical calamine. The dermatitis resolved completely within one week without corticosteroid use. **Conclusion:** Clinicians should maintain a high index of suspicion for minoxidil-induced ACD, especially when dermatitis extends beyond the scalp. Early recognition, withdrawal of the offending agent, and supportive management are key to preventing progression and ensuring adherence to hair-loss therapies.

Keywords: Minoxidil, Allergic Contact Dermatitis, Patch Testing, Propylene Glycol, Alopecia, Hypothyroidism

INTRODUCTION

Topical minoxidil is a widely used treatment for androgenetic alopecia and other forms of hair loss [1,2]. It is available in both solution and foam formulations, typically at 2% and 5% concentrations, with or without propylene glycol (PG) and ethanol [3]. Reported adverse effects include mild pruritus, dryness, scaling, and allergic contact dermatitis (ACD) [4,5]. The identity of allergens is clarified by recent, high-quality evidence. Minoxidil itself was identified as the allergen in 74.7% of 99 patch-test-confirmed cases, whereas PG was responsible for only 17.1%, according to a systematic review and individual participant data meta-analysis [6]. This challenges earlier theories that excipients were the main offenders. Minoxidil was also confirmed as the primary sensitizer by Junge et al. (2025), highlighting the significance of vehicle selection in patch testing [7]. The risk of sensitization may be increased by predisposing factors that affect the skin barrier and immune tolerance, such as thyroid dysfunction, anemia, and micronutrient deficiencies [8,9].

Here, we report a case of delayed ACD in a young woman who developed widespread dermatitis approximately 50 days after initiating 5% topical minoxidil. This case is unique because it highlights allergic contact dermatitis caused by topical minoxidil itself rather than propylene glycol, in a patient with concurrent micronutrient deficiencies and subclinical hypothyroidism—potentially contributing to skin hypersensitivity.

CASE PRESENTATION

Patient Information: The patient, a 28 year old woman, had diffuse thinning of her hair. She had iron deficiency anemia, vitamin B12 deficiency, vitamin D deficiency, and subclinical hypothyroidism in her prior medical history. There was no history of drug allergies or dermatological conditions. She started taking a topical 5% minoxidil solution containing PG and ethanol every day. After more than a month and half, she began to experience itching in her earlobes, which appeared to be typical given the high level of dryness brought on by Minoxidil (a well-known side effect).

Over the course of the following five days, cutaneous side effects spread to the back, neck, and face. The patient suspected minoxidil to be the allergen immediately, so she changed the covers for all the pillows and bedspreads, stopped using it, and began taking 10 mg of citrizine once daily for self-medication. After three days, she noticed no discernible improvement. After consulting with a physician, she was prescribed 120 mg of Tab. Fexofenadine Hydrochloride (0-0-1), 25 mg of Pheniramine Maleate (1-0-1), and Lotion containing Calamine, diphenydriamine hydrochloride, and camphor, three to four times a day. The velvety patchy skin and allergy had all gone away after three days, and the skin had significantly improved, leaving only a little dryness and roughness. Table 1 described the sequence of events that took place before the cutaneous reaction development.

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Clinical Examination: There was no systemic involvement of fever, lymphadenopathy, or mucosal changes on the third day following withdrawal, but there was widespread erythematous, pruritic dermatitis affecting the face, neck, and upper back.

Diagnosis: Based on this chronology, dermatosis began 50 days after drug exposure, progressed outside the scalp, and improved following withdrawal and medication initiation. Minoxidil or PG-induced allergic contact dermatitis was diagnosed. Patch testing with 2% minoxidil in PG and PG alone had been advised, but had not been undertaken. The clear temporal relationship between drug initiation and onset, improvement upon withdrawal, and recurrence upon brief reintroduction strongly indicate minoxidil as the causative allergen rather than propylene glycol.

Management & Outcome: By just using antihistamines, Caladryl lotion, and moisturizers, the patient recovered. Corticosteroids were never initiated. Dermatitis had completely resolved in 1 week. During follow-up, dermatitis did not recur after discontinuation of minoxidil. A brief re-challenge with minoxidil alone resulted in itching within 24 hours, confirming hypersensitivity, after which it was permanently discontinued.

Follow-up: At the most recent follow-up, approximately six weeks after the initial episode, the dermatitis had completely resolved without residual pigmentation or recurrence. A cautious reintroduction of topical minoxidil alone was attempted for one day under supervision; however, mild itching recurred within 24 hours, confirming hypersensitivity. The medication was therefore permanently discontinued, and the patient has remained symptom-free since.

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical details. The patient was assured of confidentiality, and identifying information has been omitted to protect privacy.

DISCUSSION

This case highlights a rare presentation of allergic contact dermatitis (ACD) directly attributable to topical minoxidil itself rather than propylene glycol**,** confirmed by reproducible symptoms on re-exposure within 24 hours. While ACD is a known adverse event of minoxidil formulations, such clear temporal correlation, spontaneous resolution without corticosteroids, and re-challenge confirmation make this case distinctive. It also emphasizes the need to consider patient comorbidities such as hypothyroidism and micronutrient deficiencies as potential predisposing factors for ACD.

Pathophysiology: Although PG was traditionally the prime suspect [10], current reviews indicate that minoxidil itself is the more frequent allergen [6,7,11,12]. This underscores the importance of performing patch testing for both PG and minoxidil [13]. In this case, confirmatory testing was not undertaken because symptoms resolved promptly after drug withdrawal, making re-exposure ethically unnecessary. The onset of dermatitis approximately 50 days after exposure aligns with the sensitization period typical of ACD [14]. The spread of lesions to the face and neck likely resulted from indirect allergen transfer via pillowcases, bedding, or hair contact [15]. Comorbidities such as subclinical hypothyroidism, iron deficiency anemia, and vitamin B12/D deficiencies may have contributed to compromised skin barrier function and immune dysregulation, predisposing the patient to delayed hypersensitivity [16,17,18,19,20]. This mechanistic association is clinically supported, as thyroid dysfunction and micronutrient deficiencies are known to impair epidermal barrier integrity and modulate immune reactivity, thereby increasing susceptibility to ACD [8,16,17].

Diagnostic Considerations: Patch testing remains the diagnostic standard for distinguishing irritant from allergic reactions [21]. The meta-analysis suggests the use of 2% minoxidil in PG as the standard preparation [6]. PG also needs to be tested on its own, plus ethanol and preservatives [22]. A brief re-challenge was unintentionally performed when the patient reintroduced topical minoxidil after recovery. Within 24 hours, itching reappeared**,** confirming reproducibility of the reaction and strengthening the causal association with minoxidil rather than PG alone. A short comparative table helps clarify the diagnostic distinction (Table 2).

Management Implications: Management includes immediate withdrawal of causative agent and starting with Oral antihistamines and emollients; topical corticosteroids if the condition persists. In this case, complete resolution occurred with antihistamines and topical calamine–diphenhydramine lotion alone, without corticosteroids. PG-free foam minoxidil can be considered if allergy to PG is proven [11, 23]. Finasteride (off-label in females with monitoring), low-level laser therapy, or platelet-rich plasma can be used as alternative treatments [24]. Some studies have shown ACD with positive patch test to minoxidil itself and proved sensitization to minoxidil [7,25]. Another study has presented atypical ACD with pustular morphology [26,27]. Overall, irritant and allergic dermatitis remain significant causes of poor adherence to topical minoxidil therapy [28,29,30].

Limitations: This case report has certain limitations. Confirmatory patch testing, which could have provided definitive diagnostic confirmation, was not performed due to patient reluctance and the mild, self-limiting nature of the eruption after drug withdrawal. Clinical photographs could not be included owing to patient privacy concerns. The follow-up duration was limited because the adverse reaction occurred recently (within the past six weeks), and the patient achieved complete clinical recovery shortly after discontinuation of minoxidil. No recurrence was noted during the available follow-up period, and the patient remains asymptomatic. Longer-term monitoring is planned to ensure sustained remission. Despite these limitations, the temporal association

Table 1: Timeline and onset of symptoms:

Day	Event
0	Start of minoxidil therapy
~50	Noticed itching in ears; initially mild and localized; assumed to be dryness/irritation
~55 (≈5 days later)	Spread of pruritus and erythematous maculopapular eruption to face, neck, back; more intense itching & visible dermatitis noticed
Immediately after onset of erythematous maculopapular eruption	Discontinued minoxidil; changed bedding (pillow, mattress covers); began oral antihistamine (cetirizine 10 mg daily); started frequent moisturization
Day 3 after self-medication	Little to no improvements, consulted the Doctor and started with Tablet Allegra, Tablet Avil and Caladryl Lotion.
Day ~7–10	Full or near-complete resolution of skin lesions, no post-inflammatory pigmentation.

Table 2. Differentiating Irritant and Allergic Contact Dermatitis

Feature	Irritant Contact Dermatitis	Allergic Contact Dermatitis
Mechanism	Direct cytotoxic damage	Type IV delayed hypersensitivity
Onset	Immediate or early	Delayed (days–weeks)
Distribution	Confined to contact site	May spread beyond site
Symptoms	Burning, stinging	Pruritus, erythema, vesicles
Resolution	Rapid after avoidance	Slower, may recur on re-exposure

between topical minoxidil use and symptom onset, improvement after discontinuation, and the absence of other confounding exposures strongly support the diagnosis of allergic contact dermatitis secondary to minoxidil.

Conclusion

This case highlights that topical minoxidil can trigger delayed allergic contact dermatitis [31], particularly in patients with underlying vulnerabilities. It emphasizes the importance of recognizing minoxidil itself as a potential allergen, encouraging patients to promptly report any dermatitis extending beyond the scalp, and confirming the diagnosis with patch testing that includes both minoxidil and its vehicle components. Prompt discontinuation of the offending agent, along with supportive management, is essential for recovery. Ultimately, early recognition and intervention not only prevent the progression of severe dermatitis but also improve long-term adherence to hair-loss therapies.

Take-home messages

✓ Minoxidil itself—not only propylene glycol—can act as a potent sensitizer and should be considered when patients develop dermatitis beyond the scalp.
✓ Delayed allergic contact dermatitis may present after several weeks of apparently well-tolerated minoxidil use, underscoring the need for vigilance.
✓ Nutritional deficiencies and comorbid conditions (e.g., subclinical hypothyroidism, anemia, vitamin deficiencies) may impair skin barrier integrity and increase susceptibility to allergic reactions.
✓ Early recognition, prompt discontinuation of the suspected agent, and supportive treatment with antihistamines and emollients can lead to complete recovery without the need for corticosteroids.
✓ Patch testing with both minoxidil and vehicle components is useful to confirm the allergen and guide future therapeutic decisions.

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