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Asmaa Mohamed Qandil
Department of Dermatology and Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Esraa El-Sayed El-Hawary
Department of Dermatology and Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Ghada Fawzy Rezk
Department of Dermatology and Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Naeim Mohamed Abd El-Naby
Department of Dermatology and Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Corresponding Author:
Asmaa Mohamed Qandil
Department of Dermatology and Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Minoxidil in the treatment of alopecia areata

Asmaa Mohamed Qandil, Esraa El-Sayed El-Hawary, Ghada Fawzy Rezk and Naeim Mohamed Abd El-Naby

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Abstract
Alopecia areata (AA) refers to a chronic inflammatory condition that causes non-scarring hair loss. It could impact hair on the scalp, beard, as well as eyebrows/eyelashes, or it can manifest as a more widespread condition. Using minoxidil in treating AA is biologically justified due to its capacity to reduce DNA synthesis along with leukocyte inhibitory factor within lymphocytes, without impacting their viability or migratory behavior. As a result, they inferred that minoxidil could exhibit a localized immunosuppressive impact that stimulates hair regrowth among individuals developing AA when administered topically. Utilizing a combination treatment with substances like minoxidil may result in a synergistic impact on promoting hair growth.

Keywords: Alopecia areata, treatment, minoxidil

Introduction
Alopecia areata (AA) represents a prevalent autoimmune disorder, affecting around 1.5%-2% of the general population, without any preference for gender or race [1]. AA may manifest at any age, however it commonly occurs during the second or third decades of life [2].
Minoxidil is often utilized as an off-label treatment for several hair problems, involving AA along with hair shaft disorders. Additionally, it is utilized to enhance the development of body hair in many regions, like the eyebrows as well as beard [3, 4].

Minoxidil in AA
An in vitro research was conducted to investigate the minoxidil's impact on human lymphocytes, providing a scientific rationale for utilizing minoxidil while treating AA. The researchers discovered that minoxidil inhibited DNA synthesis as well as leukocyte inhibitory factor in lymphocytes, while without impacting their viability or migratory behavior. As a result, concluding that minoxidil could exhibit a localized immunosuppressive impact, thus stimulating hair regrowth among individuals having alopecia areata when administered topically [3, 5].
Minoxidil inhibits calcium absorption through cell membranes in vascular smooth muscle cells, making it a powerful vasodilator. It predominantly acts on systemic arterioles, with minimal impact on veins. This results in a reduction in resistance in the small arteries along with a decrease in the cardiac afterload, leading to an increased blood flow through the body. While it may seem reasonable that the hair regrowth caused by topical minoxidil is a result of vasodilation, control studies employing topical glyceryl trinitrate have shown no hair regrowth. This suggests that dilatation of arterioles, rather than venules, is necessary for hair regeneration. Regrowth of hair in areas such as eyebrows as well as eyelashes in three individuals may possibly be attributed to a systemic impact of minoxidil that has been absorbed. The hypertrichosis does not seem to be caused by hormones, since the levels of plasma testosterone, urine hydroxysteroid along with ketosteroid levels remain normal following minoxidil therapy. This indicates that androgenic stimulation is not involved [6].
Typically, individuals who present with acute appendicitis for the first time tend to exhibit a more favorable and prompt response in comparison to those experiencing a recurrence. It is crucial to inform cases that this medication stimulates hair regrowth but does not alter the progression of the condition or prevent future occurrences [7].

Adverse effects of minoxidil

Using minoxidil topically has generally been regarded as safe; yet, several individuals have reported experiencing negative effects following its use. Irritant contact dermatitis, characterized by itching as well as scaling, is the most prevalent negative consequence of minoxidil. Such a complication could be reduced when utilizing 2% instead of 5%. Allergic contact dermatitis may also arise due to the presence of PG or minoxidil itself. Conducting a patch test is necessary to identify the substances responsible for causing the reaction [7, 8].

The study found that weight gain, headache, pruritus, as well as nasal and upper respiratory tract infections were the most frequently reported adverse effects, occurring in at least 2% of the individuals. Such consequences observed by certain authors, involving dermatitis, dandruff, erythema, along with burning/stinging, were identical within both 5% and 2% minoxidil treatment groups [9, 10].

Hypertrichosis is influenced by minoxidi concentrationl, and those treated with 5% minoxidil exhibited the greatest occurrence of undesired hair growth. The prevalence of this condition is higher among female cases in comparison to males. Although the exact cause remains unknown, it is possible that certain female cases exhibit a greater number of minoxidil-sensitive follicles than others. The process of spontaneous resolution first took place on the face as well as arms during a period of 1 to 3 months, and subsequently on the legs within a period of 4 to 5 months after discontinuing minoxidil. There was a belief that applying excessive amounts of minoxidil topically may cause it to be absorbed into the body, thus resulting in excessive hair growth in places that were not treated. Topical minoxidil used twice daily does not cause any systemic adverse effects, involving low blood pressure, abnormal heart rate, or weight gain. It is regarded as a safe treatment option that yields favorable results for a range of hair issues [7, 11].

The primary route of metabolism for oral minoxidil is hepatic conjugation with glucuronic acid. Minoxidil is then eliminated from the body via the kidneys around 3-4 hours after it is administered. However, the ability of minoxidil to induce vasodilation could continue for as long as 72 hours. Systemically-administered minoxidil has been associated with serious side effects, involving salt and fluid retention, as well as cardiovascular consequences, such as ischaemic heart disease, pericardial effusion, along with pulmonary hypertension [12].

Obesity and, in more severe cases, congestive heart failure may be attributed to the retention of sodium and fluids. This phenomenon occurs as a result of the shifting of blood flow from the outer to the inner cortex of the kidney, together with changes in plasma-renin activity. The case presented with ischemic heart disease, perhaps resulting from an elevated oxygen demand caused by a rise in heart rate and cardiac output. Approximately 5% of people get pericardial effusion as a result of minoxidil, however the exact mechanism is uncertain. There have been reports of pulmonary hypertension caused by elevated pulmonary artery pressure along with increased cardiac output resulting from utilizing minoxidil [13]. Additional adverse consequences could involve sporadic throbbing headache, irritated eyes, skin rashes including bullous eruptions, as well as excessive postmenstrual bleeding [14].

Conflict of Interest

Not available

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