Manifestation of pityriasis amiantacea following initiation of minoxidil

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INTRODUCTION

Pityriasis amiantacea (PA) is a uncommon inflammatory condition of the scalp, characterized by hyperkeratotic, thick, adherent scales that bind to hair follicles and can lead to hair matting and alopecia.[1] It predominantly affects females (60%-70%) and has a variable age of onset[2]; however, it is commonly observed during the teenage years. Although its etiology remains unknown, it is hypothesized to be an autoinflammatory reaction pattern to a multitude of scalp diseases, including seborrheic dermatitis, psoriasis, and lichen planus.[3] Genetic and environmental factors also appear to play a role in PA.[4] In addition, there have been reports of PA development following the initiation of certain drugs, such as tumor necrosis factor α inhibitors.[5] However, this is, to our knowledge, the first known case of PA manifesting after the use of topical minoxidil.

CASE REPORT

A 47-year−old woman presented with a 3-month history of irritation and yellow scaling on her bitemporal scalp. After initiating topical minoxidil 5% foam, she developed irritation in the form of thick, dry, yellow, asbestos-like scales circumscribing the hair shafts as well as oozing plaques, which subsequently crusted (Fig 1). These lesions persisted despite stopping the minoxidil; moreover, they did not respond to topical treatments for scalp infection. The patient had never had previous issues with seborrheic dermatitis or flaking in the past and had no personal or family history of psoriasis. PA was diagnosed on the basis of clinical examination and history, and the patient was prescribed ketoconazole 2% shampoo and clobetasol solution. The patient rapidly improved under this regimen and reported complete resolution 1 month after initiation.

Abbreviation used:

PA: pityriasis amiantacea

DISCUSSION

PA (also known as tinea amiantacea, asbestos scalp, tinea asbestina, or keratosis follicularis amiantacea) is a reactive scaling of the scalp with a multitude of etiologies. It is often diagnosed clinically because of its distinct appearance, classically appearing as hyperkeratotic, silvery-to-yellow scales that circumscribe and bind to hair shafts and follicles to mat the hair and can lead to temporary or cicatricial alopecia (estimated at 25%).[2] Ancillary dermatoscopy and histopathology may be used to confirm the diagnosis; however, this is rarely necessary. It is associated with several inflammatory and infectious skin conditions, the most common being seborrheic dermatitis, psoriasis, and tinea capitis,[6] and has been reported as both an initial clinical sign[5] and later manifestation of these diseases. However, its etiology and pathogenesis remain challenging because it maintains a similar reaction pattern across multiple dermatoses. Some studies have suggested that microbial infection plays a role in the development and continuation of PAbecause *Staphylococcal aureus* was often isolated from lesions. However, such isolates are now believed to represent either a secondary, concomitant infection or normal skin flora. Instead, these microorganisms are theorized

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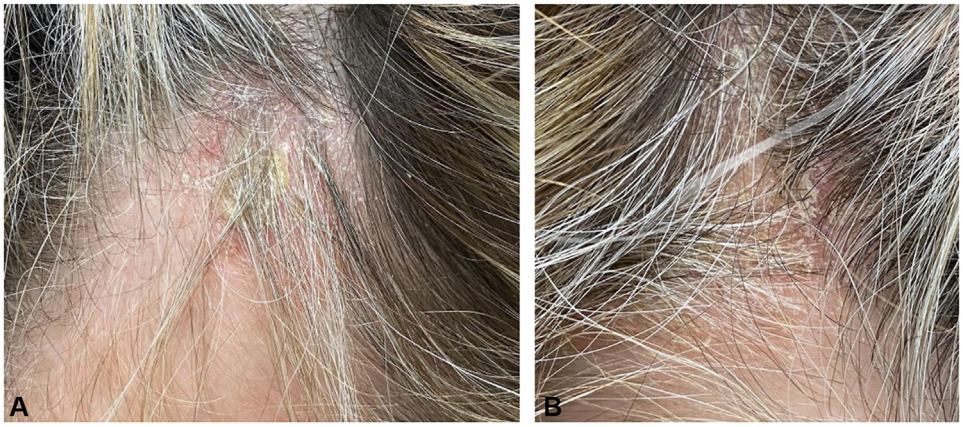

Fig 1. A, Pityriasis amiantacea present with thick, adherent scales that surround the follicle. **B,** These scales progressively build up and can cause hair matting and alopecia.

to play a role in disease maintenance through formation of biofilms and inhibiting cell differentiation.[7] Therefore, antibiotic use, when appropriate, is important in preventing the recurrence of PA.

Although no standard of care for PA exists, it is generally managed with topical medications, such as antifungal shampoo and oil-based coal tar or salicylic acid washes, which aid in the removal of the scales and crusts.[8] In severe cases, potent topical or systemic corticosteroids, retinoids, and tumor necrosis factor α blockers,[2] all of which reduce inflammation, can be considered; however, these have variable results. Additional antifungals or antimicrobials should be added if a concomitant or secondary infection is suspected, and previous case reports have generally documented successful treatment, if a microbe is isolated.

PA pathogenesis is multifactorial because variables such as stress, changes in environment, and genetic predisposition have been identified as contributors to the condition.[4] Although rare, the development of PA after the initiation of a drug has been reported in the literature. Tumor necrosis factor α inhibitors have been purported to cause a paradoxical development of psoriasis and PA, speculatively through multiple cellular alterations, such as increased production of interferon α, stimulation of other proinflammatory pathways such as interleukin 1, and blockade of autoreactive T-cell apoptosis.[5] PA was also observed in a case after the initiation of valproic acid, a glycogen synthase kinase-3β inhibitor,[8] as well as after the use of vemurafenib, a BRAF inhibitor.[1] These reports suggest that multiple cellular inflammatory and signaling pathways could be implicated in the disease. In these instances, researchers have speculated that specific keratinocyte proliferation effects of the drugs led to the development of PA. Minoxidil is a ubiquitous stimulator of hair growth, and although its mechanism of action is not completely understood, it is thought to promote the entry of follicles into the anagen phase, indirectly increasing the time for keratinocyte proliferation while also stimulating dermal papilla and epithelial cells.[9] This prokeratinocyte effect could play a role in the emergence of PA after treatment. In vitro studies have shown that low doses of minoxidil have proliferative effects on normal human keratinocytes,[10] supporting this hypothesis.

In conclusion, we present a unique case of PA developing after the initiation of topical minoxidil and propose a mechanism of development. This case emphasizes the multifactorial pathogenesis of PA and its capacity to appear as a sequela of multiple conditions, treatments, and environmental factors. Timely recognition and early treatment of PA should prevent serious alopecic complications of the condition.

Conflicts of interest

None disclosed.

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