

**Abstract N°: 8349****Trauma-Induced Alopecia Areata with Poliosis: Insights into Hair Regrowth and Melanocyte Dysfunction**

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Introduction & Objectives:

Alopecia areata (AA) is an autoimmune condition characterized by non-scarring hair loss, most frequently involving the scalp and beard. The exact pathogenesis remains multifactorial, with both genetic and environmental triggers implicated. Poliosis, or localized depigmentation of hair, may rarely occur in the regrowth phase of AA due to melanocyte dysfunction. We present a case of trauma-induced AA with poliosis, highlighting an unusual regrowth pattern and a potential link between physical injury and autoimmune hair loss.

Materials & Methods:

A 47-year-old male presented to our dermatology clinic with a history of patchy hair loss involving the scalp and beard, beginning approximately one year prior. The onset of alopecia closely followed a physical trauma event in which the patient sustained a stab wound. He was previously evaluated in another dermatology service, where a diagnosis of alopecia areata was established, and oral systemic corticosteroids were prescribed. However, treatment was discontinued early due to elevated blood pressure values. The patient noted partial regrowth of hair several months later, but the regrown hair was white in color (poliosis). No family history of autoimmune or pigmentary disorders was reported. Clinical examination confirmed sharply demarcated, non-scarring alopecic patches with white terminal hairs. Dermoscopic evaluation was suggestive of regrowing white hair shafts, with few exclamation mark hairs. No histopathologic examination was performed, as the clinical diagnosis was clear.

Results:

The patient exhibited signs of active but regressing alopecia areata with poliosis. Regrowth occurred in affected areas despite incomplete systemic treatment, and was characterized by depigmented terminal hairs. No signs of other autoimmune comorbidities were identified. Blood pressure remained elevated and was managed by the primary care provider. At follow-up, no new alopecic patches were observed, and the white hair persisted, indicating stable remission with residual pigmentary alteration.

Conclusion:

This case illustrates a potential link between physical trauma and the onset of alopecia areata, likely through a localized immune-mediated response. Furthermore, the development of poliosis in regrowing hair highlights the impact of inflammatory processes on follicular melanocytes. Recognition of poliosis in the context of AA may prevent misdiagnosis and provide insight into underlying immune dysregulation. Further studies are needed to clarify the mechanisms behind trauma-induced AA and the pathophysiology of pigmentary changes in hair regrowth.

