Hair pigmentation is a complex phenomenon that involves many hormones, neurotransmitters, cytokines, growth factors, eicosanoids, cyclic nucleotides, nutrients, and a physicochemical milieu. Repigmentation of gray hair has been associated with herpes zoster infection, use of systemic corticosteroids, thyroid hormone therapy, or treatment with interferon and ribavirin. We report a case of repigmentation of gray hairs in lesions of annular elastolytic giant cell granuloma (AEGCG) on the scalp of a 67-year-old man.

**Case Report**

A 67-year-old man presented to the dermatology department for evaluation of pruritic lesions on the face and scalp of 1 year’s duration. The patient reported that hairs in the involved areas of the scalp had turned from gray to a dark color since the appearance of the lesions. The patient had a history of hypertension and type 2 diabetes mellitus. His current medications included irbesartan, atorvastatin, metformin, acetylsalicylic acid, omeprazole, and repaglinide.

Physical examination revealed plaques on the scalp and cheeks that were 2 to 10 mm in diameter. Some of the plaques had an atrophic center and a desquamative peripheral border. The patient had androgenetic alopecia. The remaining hair was dark in the areas affected by the inflammatory plaques while it remained white-gray in the uninvolved areas (Figure 1).

A biopsy of one of the lesions was performed. Histopathology revealed a granulomatous dermatitis involving mostly the upper and mid dermis (Figure 2). Granulomas were epithelioid with many giant cells, some of which contained many nuclei. A ringed array of nuclei was noted in some histiocytes. Elastic fibers were absent in the central zone of the granulomas, a finding that was better evidenced on orcein staining (Figure 3). On the contrary, the peripheral zone of the granulomas showed an increased amount of thick elastotic material. Elastophagocytosis was observed, but no asteroid bodies, Schaumann bodies, or mucin deposits were noted. Histochemistry for microorganisms with

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The authors report no conflict of interest.

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Ziehl-Neelsen and periodic acid–Schiff staining was negative. Other findings included a mild infiltrate of melanophages in the papillary dermis as well as a mild superficial dermal inflammatory infiltrate that was rich in plasma cells. Immunostaining for Treponema pallidum was negative. The lymphocytic infiltrate was CD4+ predominant. A prominent dermal elastosis also was noted. Hair follicles within the plaques were small in size, penetrating just the dermis. Immunostaining for HMB-45, melan-A, and S-100 demonstrated preserved melanocytes in the hair bulbs (Figure 4). CD68 immunostaining made the infiltrate of macrophages stand out. Based on the results of the histopathologic evaluation, a diagnosis of AEGCG was made.

**Comment**

Annular elastolytic giant cell granuloma is a controversial entity that was first described by O’Brien in 1975 as actinic granuloma. Hanke et al proposed the term...
Hair Repigmentation in AEGCG

Hair pigmentation involves many hormones, neurotransmitters, cytokines, growth factors, eicosanoids, cyclic nucleotides, nutrients, and a physicochemical milieu\(^1,\text{21-23}\) (Table), and it is regulated by autocrines, paracrines, or intracrines.\(^\text{21}\) Therefore, it is likely that many environmental factors may affect hair pigmentation, which may explain why repigmentation of the hair has been seen in the setting of herpes zoster infection,\(^2\) use of systemic corticosteroids in the treatment of bullous pemphigoid,\(^4\) treatment with interferon and ribavirin,\(^5\) porphyria cutanea tarda,\(^26\) or lentigo maligna.\(^27\) In our patient, AEGCG might have induced some changes in the dermal environment that were responsible for the repigmentation of the patient’s gray hair. It is

Melanin units from the proximal hair bulb are responsible for pigmentation in adult hair follicles and are integrated by the hair matrix, melanocytes, keratinocytes, and fibroblasts.\(^\text{21}\) Hair bulb melanocytes are larger and more dendritic than epidermal melanocytes (Figure 5). The hair only

**Primary Regulators in Melanogenesis**

<table>
<thead>
<tr>
<th>Positive Regulators (Melanogenesis Stimulators)</th>
<th>Negative Regulators</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenocorticotropic hormone</td>
<td>Agouti protein</td>
</tr>
<tr>
<td>Androgens</td>
<td>(in animals, not in humans)</td>
</tr>
<tr>
<td>Bone morphogenetic proteins</td>
<td>Cholinergic agonists</td>
</tr>
<tr>
<td>β-endorphin</td>
<td>Dopaminergics</td>
</tr>
<tr>
<td>Endothelin-1</td>
<td>Glucocorticoids</td>
</tr>
<tr>
<td>Endothelin-3</td>
<td>IFN-γ</td>
</tr>
<tr>
<td>Estrogens</td>
<td>IL-1</td>
</tr>
<tr>
<td>Histamine</td>
<td>IL-6</td>
</tr>
<tr>
<td>L-3,4-dihydroxyphenylalanine</td>
<td>Melatonin</td>
</tr>
<tr>
<td>Leukotrienes</td>
<td>Tumor necrosis factor α</td>
</tr>
<tr>
<td>L-tyrosine</td>
<td>Transforming growth factor β</td>
</tr>
<tr>
<td>α-Melanocyte-stimulating hormone</td>
<td>Triiodothyronine</td>
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<tr>
<td>Prostaglandins</td>
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<tr>
<td>Stem cell factor</td>
<td></td>
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<tr>
<td>Vitamin D(_3)</td>
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</table>

**Figure 5.** A hair bulb of a normal follicle showing the morphology of melanocytes, which are dendritic and large (H&E, original magnification ×100).
speculated that solar radiation and other factors can transform the antigenicity of elastic fibers and induce an immune response in AEGCG.\textsuperscript{12,13} The lymphocytic infiltrate in these lesions is predominantly CD4\textsuperscript{+}, as seen in our patient, which is consistent with an autoimmune hypothesis.\textsuperscript{13} Nevertheless, it most likely is too simplistic to attribute the repigmentation to the influence of just these cells.

REFERENCES