BRIEF REPORT



Annular Elastolytic Giant Cell Granuloma: Chronic Heat Exposure, an Underestimated Factor

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Dear Editor:

Annular elastolytic giant cell granuloma (AEGCG) is a rare skin disease presenting with annular lesions with erythematous borders and atrophic centers. Although its pathogenesis is elusive, AEGCG frequently appears on the supexposed area, suggesting that sunlight is an important predisposing factor. However, the specific mechanism of sunlight inducing AEGCG, and whether radiation of different wavelengths can have a similar effect is unknown. Herein, we report a case of AEGCG in a patient with minimal sunlight exposure but chronic exposure to high heat.

A 49-year-old female was referred to Chung-Ang University Hospital for multiple annular lesions on her forehead, neck, upper chest, forearms, and dorsal hands. The lesions had developed 1 year ago and had slowly progressed with peripheral growth. The brownish-red, annular plaques had slightly elevated borders with shiny atrophic centers (Fig. 1A). The patient did not complain of any discomfort on the lesion. She did not have any relevant past medical and familial history. She denied of chronic sun exposure reporting that she had grown up in a city and had usually spent her time indoors. Interestingly, she had worked at a barbeque restaurant for 20 years, where she was constantly exposed to high heat especially on face and

forearm.

Punch biopsy of the lesion showed granulomatous infiltration of histiocytes with multinucleated giant cells, but no necrobiotic collagen bundles (Fig. 2A). Verhoeff–Van Gieson elastin stain showed fragmented elastin fibers and elastophagocytosis by giant cells (Fig. 2B). Based on these findings, the patient was diagnosed with AEGCG. In spite of oral methylprednisolone with ultraviolet (UV)-A therapy, the lesions continued to progress (Fig. 1B). Treatment is ongoing with hydroxychloroquine and UV-A therapy. We received the patient's consent form about publishing all photographic materials.

AEGCG typically presents as large annular plaques with elevated borders and atrophic centers located on sun-exposed areas in middle-aged women. However, in a recent study, the location of the lesions showed weak association with sun exposure¹. These findings suggest that actinic damage may not be the sole factor in pathogenesis¹. Currently, the most acceptable hypothesis on the etiology of AEGCG is that unknown cellular immunological reactions affect the elastic fibers², and that UV radiation, heat, or nerve fiber impairment trigger granulomatous inflammation³. Rasmussen et al.⁴ have shown that the dermis undergoes physical changes in vitro when it reaches 60°C, leading to elastolysis which can progress to develop AEGCG. Furthermore, chronic heat damage may also change the antigenicity of elastic fibers⁵. Our patient seldom spent her time outside but spent most of her time inside barbecuing. We informed her to avoid heat exposure but she could not quit her job. Her skin lesions have shown no response to our treatment until now.

We have reported a rare case of AEGCG associated with chronic heat exposure. Actinic damage may be an important factor but not the sole factor in AEGCG pathogenesis. Thus, when encountering a patient suspicious for AEGCG, dermatologists should meticulously examine the

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Fig. 1. (A) Clinical image of multiple annular lesions during initial visit. The brownish-red, annular shaped plaques with a slightly elevated border and shiny atrophic center. (B) Progressive lesions despite treatment with steroids and ultraviolet A therapy.

Fig. 2. (A) Skin biopsy specimen showing dermal granulomatous infiltrates of histiocytes with multinucleated giant cells (H&E, \times 100). (B) Elastin stain showed fragmented elastin fibers and elastophagocytosis by giant cells (red arrowhead). The amount of elastic fiber is reduced around the granulomatous infiltrations (Verhoeff–Van Gieson stain, \times 200).

patient's history regarding any exposure to radiation of different wavelengths, including sunlight and heat.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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REFERENCES

- 1. Chen WT, Hsiao PF, Wu YH. Spectrum and clinical variants of giant cell elastolytic granuloma. Int J Dermatol 2017; 56:738-745.
- El-Khoury J, Kurban M, Abbas O. Elastophagocytosis: underlying mechanisms and associated cutaneous entities. J Am Acad Dermatol 2014;70:934-944.
- 3. Zacharias I, Nagy G, Orban I. [O'Brien's actinic granuloma]. Ann Dermatol Venereol 1992;119:137-138. French.
- Rasmussen DM, Wakim KG, Winkelmann RK. Isotonic and isometric thermal contraction of human dermis. II. Age-related changes. J Invest Dermatol 1964;43:341-348.
- Fujimoto N, Akagi A, Tajima S. Expression of 67-kDa elastin receptor in annular elastolytic giant cell granuloma: elastin peptides induce monocyte-derived dendritic cells or macrophages to form granuloma in vitro. Exp Dermatol 2004;13: 179-184.