

# A special case of annular elastolytic giant cell granuloma: case report and mini-review

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Dear Editor,

Annular elastolytic giant cell granuloma (AEGCG) is a rare and variable dermatological condition. It typically presents as annular plaques with raised edges and an atrophic center, and it usually affects fair-skinned, middle-aged to older Caucasian women in sun-exposed areas [1, 2, 3]. Due to its rarity and the numerous clinical variants ranging from papular to reticular to ring-shaped lesions, this disease is not always easy to recognize. Furthermore, these skin lesions can occur in areas not exposed to the sun [4]. In a retrospective study of 105 patients, Qian et al. classified AEGCG into annular, papular and mixed forms; the giant type, which has annular lesion diameters greater than 10 cm; and the generalized form, which affects more than 30% of the body's surface area [5]. Since not all forms present with the annular variant, the term elastolytic giant cell granuloma (EGCG) was introduced [5]. However, the histopathological features are constant and are characterized by elastolysis and elastophagocytosis in all clinical variants.

We present the case of a 92-year-old male patient with nonpalpable figured erythema on the back (Figure 1). The patient's history revealed that the general practitioner had made this incidental finding during auscultation. After the application of methylprednisolone aceponate was unsuccessful, laboratory parameters, including ANA/ENA, cardiolipin antibodies, cytoplasmic antibodies,  $\beta$ 2-glycoprotein antibodies, and standard blood test, were examined and found to be unremarkable. A hematological examination of the blood revealed an unremarkable cell distribution in the peripheral smear. A punch biopsy and Orcein staining revealed an annular elastophagic giant cell granuloma with granulomatous inflammatory infiltrates in the upper reticular dermis, accompanied by elastophagocytosis and loss of elastic fibers. Considering the clinical, dermoscopic, and histologic findings of our patient, we were able to diagnose EGCG. After discussing the diagnosis and treatment options, the patient declined treatment due to his advanced age and lack of symptoms.

Histologically, EGCG is characterized by granulomatous infiltrates in the mid-dermis consisting of multinucleated giant cells, histiocytes, and lymphocytes [6]. According to Qian et al., the most important features are elastolysis and elastophagocytosis [5], which were also present in this case (Figures 2, 3). These features are detected by elastic staining and play a crucial role in diagnosis [7].



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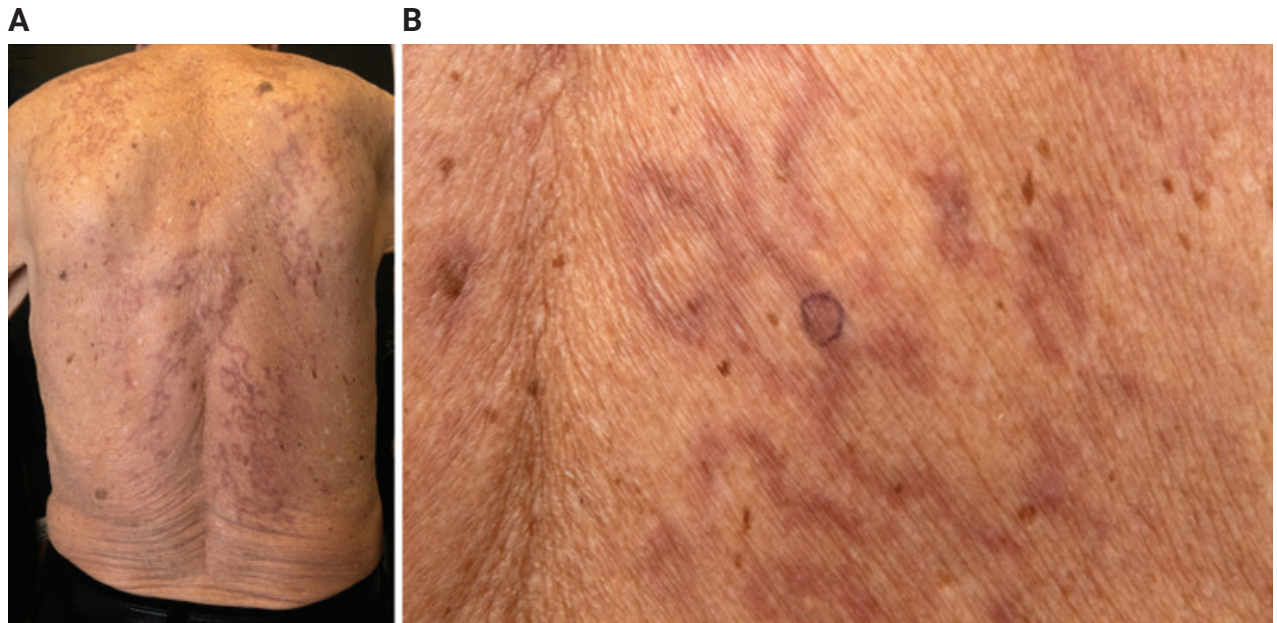
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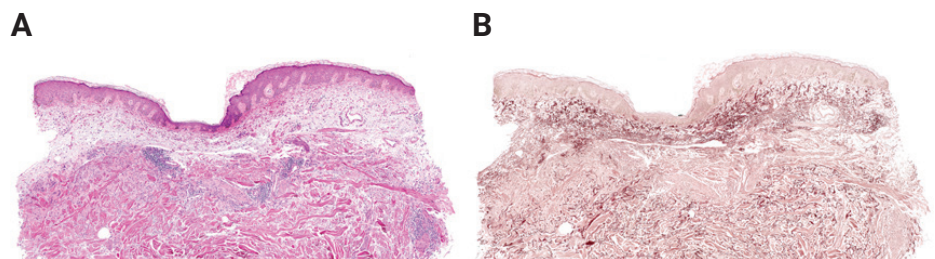
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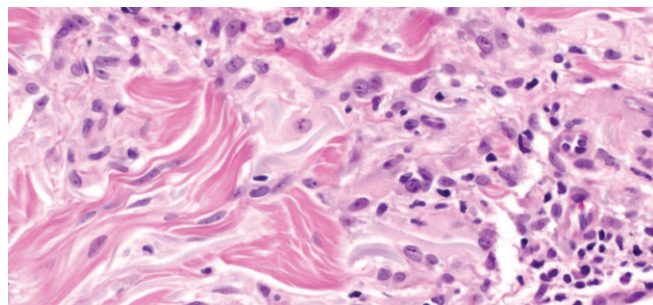
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**Figure 1.** Non-palpable, figured erythema on the patient's back (A). Close-up of the erythema. Marking of the skin punch removal site (B).



**Figure 2.** The most important features of EGCG: elastophagocytosis and loss of elastic fibres. (Haematoxylin-eosin staining (A), orcein staining (B), original magnification  $\times 100$ ).



**Figure 3.** Close – up showing elastophagocytosis/ fibres in the cytoplasm of giant cells. (Haematoxylin-eosin staining).

However, the diagnosis can be challenging due to similarities in clinical features with other granulomatous dermatoses, such as sarcoidosis, granuloma annulare, and necrobiosis lipoidica [7]. The most important differential diagnosis, both clinically and histopathologically, is granuloma annulare. This benign chronic inflammatory skin disease presents with arcuate or ring-shaped plaques but usually does not exhibit elastophagocytosis with diffuse loss of elastic tissue on histology [5].

Dermoscopy can help narrow down the clinical differential diagnosis more precisely. Granulomatous skin disease presents with a featureless yellowish-orange area that may be focal or diffusely distributed. This appearance is due to the presence of granulomas in the dermis, which is characteristic of granulomatous inflammation [8]. In AEGCG, Errichetti et al. described homogeneous, reticular, well-focused vessels in the center of the lesions, which they attributed to central epidermal atrophy. This dermoscopic aspect is not found in similar granulomatous dermatoses, facilitating differentiation [9].

In addition to having numerous cardiovascular diseases, the patient had a history of non-insulin-dependent diabetes mellitus. According to previous publications, diabetes mellitus is the systemic disease most frequently associated with AEGCG [10]. It is believed that the high serum glucose concentrations can cause structural damage to elastic fibers, resulting in inflammatory reactions that trigger EGCG [5]. EGCG lesions associated with certain malignancies, such as acute myeloid leukemia, adult T-cell leukemia, prostate carcinoma, gastric carcinoma, esophageal carcinoma, and primary cutaneous T-cell lymphoma, have been reported to decrease during treatment of these malignancies. This suggests that the coexistence of EGCG and malignancies may not be coincidental [5]. It has been hypothesized that these skin lesions may represent an immunological response of the body against tumor antigens as a possible paraneoplastic phenomenon [7].

Ultraviolet radiation, heat, and nerve fiber damage impair elastic fibers. As a result, previously unknown cellular immunological reactions occur, triggering granulomatous inflammation [4]. Macrophages or dendritic cells then phagocytose elastin fragments, forming granulomas or multinucleated giant cells. This assumption is confirmed by immunohistochemical studies showing that CD4+ cells predominate over CD8+ cells in inflammatory infiltrates [11]. The molecular pathogenesis mechanism is not fully understood, but the 67-kDa elastin receptor and human macrophage metalloelastase-12 are believed to be influential factors [4]. The binding of degraded elastin fragments to the 67-kDa elastin receptor (67LR) on macrophages and dendritic cells is thought to activate these immune cells, promote giant cell formation, and lead to the expression of human macrophage metalloelastase-12 (MMP-12), an elastolytic enzyme. The resulting elastolysis and elastophagocytosis processes are central histopathological hallmarks of the disease [4]. As mentioned above, environmental factors can alter the structure of the elastic fibers, further intensifying the immune response.

The possible chronic course of this disease contrasts with possible spontaneous remission without scarring within a few months to years. Many different therapeutic approaches exist, but no general standard therapy has been established, resulting in a variety of options that lead to different reactions in each patient. These options range from light therapies, such as psoralen-UV-A and narrow-band UV-B, to systemic therapies, including methotrexate, hydroxychloroquine, minocycline, acitretin, dapsone, ciclosporin and clofazimine, as well as physical options, such as cryotherapy, and glucocorticoids, which have been applied topically, intralesionally and systemically. Topical calcineurin inhibitors have also been used. Systemic corticosteroid therapy has been the most successful, although there is also a certain recurrence rate (19.4 %). Due to the possible spontaneous regression of the disease, it is difficult to determine the extent to which the response is drug-induced [5].

Early recognition of elastolytic giant cell granuloma is crucial, as it may be associated with systemic disease. Due to changes in the elastic fibers combined with elastophagocytosis, various clinical manifestations may occur, complicating the diagnosis. This is another reason why understanding the detailed histopathology is essential for diagnosis. Prompt detection of EGCG and an associated search for underlying causes can identify pre-existing malignancies and minimize their secondary effects, thereby improving the prognosis.

## Additional information

### Conflict of interest

The authors have declared that no competing interests exist.

### Ethical statements

The authors declared that no clinical trials were used in the present study.

The authors declared that no experiments on humans or human tissues were performed for the present study.

The authors declared that no informed consent was obtained from the humans, donors or donors' representatives participating in the study.

The authors declared that no experiments on animals were performed for the present study.

The authors declared that no commercially available immortalised human and animal cell lines were used in the present study.

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### Author contributions

Eva Teschl: conceptualization, data curation, and original draft writing. Regina Fink-Puches: conceptualization, review, editing and supervision. Rainer Hofmann-Wellenhof: supervision. Lorenzo Cerroni: Provision of histopathological images.

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### Data availability

All of the data that support the findings of this study are available in the main text.

## References

1. Ma DL, Vano-Galvan S. Actinic Granuloma. *N Engl J Med*. 2017;376(5):475. <https://doi.org/10.1056/NEJMicm1600384>
2. Hanke CW, Bailin PL, Roenigk HH, Jr. Annular elastolytic giant cell granuloma. A clinicopathologic study of five cases and a review of similar entities. *J Am Acad Dermatol*. 1979;1(5):413-21. [https://doi.org/10.1016/S0190-9622\(79\)70033-8](https://doi.org/10.1016/S0190-9622(79)70033-8)
3. Parikh SA, Que SKT, Holmes WD, Ferenczi K, Grant-Kels JM, Rothe MJ. Infiltrated papules on the trunk and headaches: A case of actinic granuloma and a review of the literature. *Int J Womens Dermatol*. 2015;1(3):131-5. <https://doi.org/10.1016/j.ijwd.2015.06.001>

4. Chen WT, Hsiao PF, Wu YH. Spectrum and clinical variants of giant cell elastolytic granuloma. *Int J Dermatol*. 2017;56(7):738-45. <https://doi.org/10.1111/ijd.13502>
5. Qian YT, Liu JW, Liu W, Chen T, Tan Y, Ma DL. A Retrospective Study of 105 Patients with Elastolytic Giant Cell Granuloma and a Proposal for a New Clinical Classification. *Acta Derm Venereol*. 2022;102:adv00684. <https://doi.org/10.2340/actadv.v102.1985>
6. Gutierrez-Gonzalez E, Gomez-Bernal S, Alvarez-Perez A, Sanchez-Aguilar D, Toribio J. Elastolytic giant cell granuloma: clinic-pathologic review of twenty cases. *Dermatol Online J*. 2013;19(10):20019. <https://doi.org/10.5070/D31910020019>
7. Gutiérrez-González E, Pereiro M, Jr., Toribio J. Elastolytic Actinic Giant Cell Granuloma. *Dermatol Clin*. 2015;33(3):331-41. <https://doi.org/10.1016/j.det.2015.03.002>
8. Errichetti E, Stinco G. Dermatoscopy of Granulomatous Disorders. *Dermatol Clin*. 2018;36(4):369-75. <https://doi.org/10.1016/j.det.2018.05.004>
9. Errichetti E, Cataldi P, Stinco G. Dermoscopy in annular elastolytic giant cell granuloma. *J Dermatol*. 2019;46(2):e66-e7. <https://doi.org/10.1111/1346-8138.14539>
10. Aso Y, Izaki S, Teraki Y. Annular elastolytic giant cell granuloma associated with diabetes mellitus: a case report and review of the Japanese literature. *Clin Exp Dermatol*. 2011;36(8):917-9. <https://doi.org/10.1111/j.1365-2230.2011.04094.x>
11. Stefanaki C, Panagiotopoulos A, Kostakis P, Stefanaki K, Petridis A. Actinic granuloma successfully treated with acitretin. *Int J Dermatol*. 2005;44(2):163-6. <https://doi.org/10.1111/j.1365-4632.2005.02043.x>