Annular elastolytic giant cell granuloma (AEGCG) is a rare granulomatous skin disease characterised by small papules that evolve into annular/polycyclic plaques. Plaques typically have a slightly raised border; the centre may be hypopigmented and/or atrophic. Lesions are usually located on sun-exposed areas such as the face and the neck (1, 2). Histologically, AEGCG is associated with multinucleated giant cell infiltrate, elastolysis, and elastophagocytosis, localised mainly in the mid-dermis. Elastophagocytosis is considered to be brought on by one or more triggers, which leads to the loss of elastic fibres (3); in AEGCG, the possible triggers include ultraviolet radiation, heat, or other unknown factors that induce a cellular immune response (4). AEGCG is often refractory to any treatment, and no standard therapy has been established (5). Here, we report a case of AEGCG that improved after 11 weeks of treatment with oral minocycline hydrochloride.

CASE REPORT

A 46-year-old Japanese man was referred to our hospital. He had a well-demarcated, annular, erythematous plaque with a raised border and slightly atrophic centre (Fig. 1a, b) on his left temple. The patient first noticed the lesion one year prior to his first visit to our hospital. Although he had been treated with a 7-week course of topical beclomethasone dipropionate at a previous hospital, the lesion progressed in size, ultimately measuring 5 cm in diameter. The patient did not complain of any pain or pruritus. Histopathology of a punch biopsy, which was taken from the elevated border, revealed an infiltrate of lymphocytes and macrophages that formed non-palisading granulomas in the upper to mid-dermis (Fig. 2a). Elastica van Gieson stain of the tissue revealed a marked reduction in elastic fibres, and evidence of phagocytosis of elastic fibres by multinucleated giant cells and macrophages (Fig. 2b). Ziehl-Neelsen staining of the tissue was negative. Bacterial and fungal cultures of biopsy specimens obtained from the lesion were negative. Laboratory studies, including complete blood cell count, biochemical tests, and serum levels of angiotensin-converting enzyme were within normal limits. Enzyme-Linked ImmunoSpot assay, used for tuberculosis diagnosis, was negative. Serum levels of blood glucose and haemoglobin A1c were also normal. A diagnosis of AEGCG was made on the basis of clinical and histopathological findings.

Because the patient’s AEGCG was refractory to a potent glucocorticoid ointment, we obtained informed consent and administered oral minocycline hydrochloride at 200 mg/day for 2 weeks, followed by 100 mg/day for 9 weeks. Eleven weeks later, the active erythematous infiltration had gradually decreased, and the lesion had faded with pigmentation (Fig. 1c). No adverse effects were reported.

Fig. 1. Patient’s clinical features. Pre-treatment, a well-demarcated, annular, erythematous plaque is seen on the left temple (a, b). Post-treatment with systemic minocycline, erythema is decreased and pigmentation is observed (c).
DISCUSSION

AEGCG is a rare, reactive granulomatous dermatosis, usually associated with actinic damage. The pathogenesis of AEGCG is unidentified. Both normal and degenerated elastic fibres are phagocytosed by macrophages in AEGCG. Several treatments for AEGCG have previously been proposed, including topical or intralesional glucocorticoids, cyclosporine, topical calcineurin inhibitors, dapsone, hydroxychloroquine sulphate, clofazimine, cryotherapy, methotrexate, pсорalen plus ultraviolet A therapy, narrowband ultraviolet B therapy, retinoids, fumaric acid esters, and tranilast. However, most treatments are unsatisfactory, and there is no definitive therapy for AEGCG. A triple antibiotic therapy regimen, which included minocycline, has previously been reported to provide some beneficial effects for granuloma annulare, although not curing the disease. Minocycline has anti-inflammatory effects that interfere with lymphocyte proliferation, especially that of T cells, as well as immunomodulating and anti-granulomatous effects. We considered that these mechanisms of action of minocycline may have affected AEGCG in the present patient. However, minocycline should be used with care, as it may be associated with photosensitivity. This could potentially worsen, rather than improve AEGCG, as previously reported with doxycycline.

To our knowledge, this is the first time that AEGCG was successfully treated with minocycline. In conclusion, the present case suggests that minocycline may be a useful therapeutic option for AEGCG. We cannot, however, exclude a spontaneous resolution of the lesion, and further case reports and studies are needed to confirm our observation.

REFERENCES