

CASE REPORT

A Case of Atypical Granuloma Annulare Involving Both Ears

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We report a rare case of granuloma annulare (GA), affecting both ear antihelices, in a 28-year old male patient that presented with a 1-year history of non-tender, firm, skin-colored, 1~5 mm papules on both ear antihelices. There was no history of trauma. An excisional biopsy specimen taken from one of the lesions of the right ear revealed infiltration of histiocytes and lymphocytes around a zone of collagen alteration in the dermis. Based on the clinical and pathological findings, the patient was diagnosed with a rare case of bilateral GA of both antihelices; this is the first report in the Korean dermatology literature. (**Ann Dermatol 21(4) 413~415, 2009**)

-Keywords-

Bilateral ear antihelical area, Granuloma annulare

INTRODUCTION

Granuloma annulare (GA) is a common, benign, inflammatory skin disease of unknown etiology that occurs in both adults and children¹. Typically it is clinically characterized by papular lesions, usually distributed in ring-shaped configurations. Histologically the findings correspond to a necrobiotic granuloma, surrounded commonly by a radial arrangement of infiltrated lymphocytes and histiocytes². The localized variant is the most common type and the acral sites are most commonly affected^{2,3}. The ear is an unusual site for GA. Herein we report a rare case of bilateral GA on both ear anti-helical

areas in a 28-year old man.

CASE REPORT

A healthy 28-year old man presented with multiple papules on both ear antihelical areas present for about 1 year. Physical examination revealed multiple, 1~5 mm, skin-colored, firm, non-tender papules on both ear antihelical areas (Fig. 1). The patient did not report subjective skin symptoms such as itching and pain. There was no previous history of trauma or chronic medical conditions, such as diabetes mellitus or rheumatoid arthritis. The patient was not routinely taking medication. The laboratory studies were within normal range except for the GPT (62 U/L); the tests included: a complete blood count, liver function, urine analysis and syphilis serology (VDRL). Excisional biopsy was performed of the right ear antihelical area. A biopsy specimen from the lesion of the right antihelical area showed a palisading infiltration of histiocytes, and lymphocytes around and between altered collagen fibers in the dermis (Fig. 2). Mucin was demonstrated within degenerative collagen bundles by the alcian blue stain (Fig. 3). After treatment with topical corticosteroid ointment and pimecrolimus cream (Elidel[®], Novartis Pharma, Basel, Switzerland) for one month, the size of the lesions was slightly decreased.

DISCUSSION

GA is a benign, granulomatous disease that presents clinically as papules, nodules and plaques, often in an annular configuration. It is a self-limited disease, first described by Colcott-Fox in 1895 and Radcliffe-Crocker in 1902⁴. There are at least four clinical forms: (1) localized with single or multiple rings, (2) generalized, (3) subcutaneous, and (4) perforating². Among the four clinical subtypes, the localized form is the most common type. It usually presents

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Fig. 1. Multiple skin-colored, firm, non-tender papules on the both ear antihelical areas.

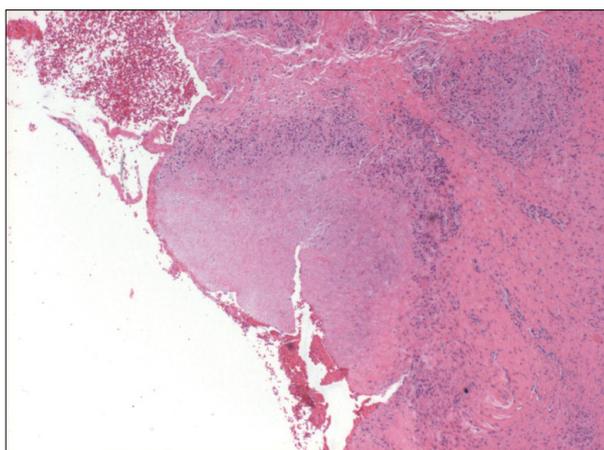


Fig. 2. An acellular zone of degenerated collagen is surrounded by palisading chronic inflammatory cells including histiocytes (H&E, $\times 40$).

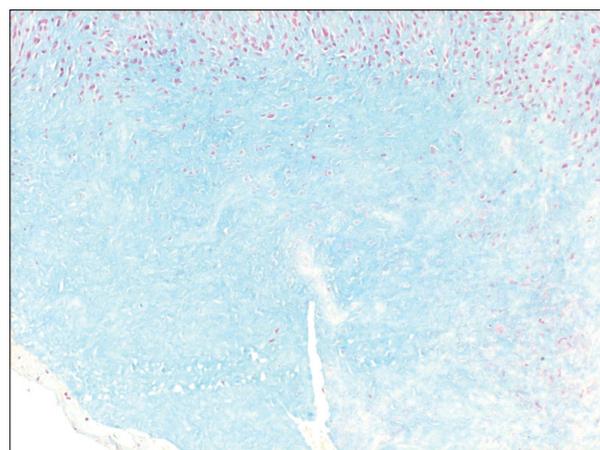


Fig. 3. Increasing mucin deposits in the central zone of a granuloma (Alcian blue stain, $\times 100$).

during the first three decades of life and has a female preference (2.25:1)³. About 50% of patients have solitary lesions. The most common affected areas are in the acral regions, especially the knuckles and dorsum of the fingers³. Facial involvement is rare³. Farrar et al.⁵ reported one case of perforating GA of both ears. In the Korean dermatology literature, there have been no reported cases of GA on both ears in the helical area.

The etiology of GA is unknown⁶. Some cases have been reported to follow an insect bite, trauma, sun exposure, tuberculin testing, PUVA therapy and viral infection including Epstein-Barr, HIV, and herpes zoster^{4,6}. In our case, there was no previous history of trauma, excess sun ex-

posure, insect bites or medication use.

The histopathological features of localized GA include necrobiotic collagen and mucin in the dermis with surrounding inflammatory cells such as histiocytes, multinucleated giant cells, and a few acute inflammatory cells^{6,7}. A palisading granuloma is the most characteristic finding of the histological lesion³. Considering the clinical and histological findings, the differential diagnosis of GA includes necrobiosis lipoidica, chondrodermatitis helicis nodularis and rheumatoid nodules³. Necrobiosis lipoidica shows dermal sclerosis and thickened subcutaneous septa and mucin deposits are uncommon. Chondrodermatitis helicis nodularis presents with painful nodules and has a

characteristic layering of fibrin. Rheumatoid nodules histologically have fibrin rather than mucin deposits⁷.

GA has a spontaneous remission in about 50% of patients within two years. Recurrence occurs at the same site in about 40% of patients^{4,7-9}. Reassurance and clinical observation may be the treatment of choice for localized, asymptomatic patients. Intralesion injections of triamcinolone and topical steroids are effective for individual lesions and are reasonable initial treatments⁸. Topical vitamin E, cryotherapy, intralesion interferon gamma, and surgical excision may be effective⁸. Other cutaneous treatment modalities include PUVA or UVA1 therapy, and CO₂ laser treatment⁷. For generalized GA, many treatment modalities have been tried; this form is a chronic disease with a relapsing course that has shown a poor therapeutic response^{10,11}. Systemic steroids may be very effective, but the side effects of high doses are dangerous. Dapsone, niacinamide, isotretinoin, sulfone, potassium iodide, chloroquine, alkylating agents, and electrodesiccation are used as additional options for treatment^{7,10}. The patient in this case was treated with a topical steroid and a calcineurin inhibitor and the size of the lesions decreased slightly after one month of treatment.

This case illustrates a rare presentation of GA on the ear antihelices with typical clinical and pathological findings.

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