Actinic Granuloma: One of the Causes of Secondary Anetoderma

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Actinic granuloma (O'Brien) is an annular inflammatory reaction that develops in skin after long-term sun-exposure. Anetoderma is characterized by discrete skin lesions that appear to be loose and wrinkled. The primary type arises in clinically normal skin, and the secondary type appears in lesions of other disorders. We describe a case of anetoderma that developed on the neck, both arms and calves, and showed histopathological findings of actinic granuloma. (Ann Dermatol 11(2) 106-108, 1999).

Key Words: Actinic granuloma, Anetoderma

In 1975, O'Brien1 described actinic granuloma and indirectly proposed criteria for its diagnosis: (1) Annular, slowly enlarging plaques develop on sun-exposed, weather-beaten skin of patients at least 30 or 40 years of age. (2) Elastotic material is present within giant cells, and the inflammation is granulomatous. (3) Elastotic material and elastic fibers are absent from the postreactive central zone and are greatly reduced in number and density in the vicinity of giant cells in the annular zone. Reports of granuloma annulare on sun-exposed skin have appeared since 1961, and destruction of elastic tissue in granuloma annulare have been written about even earlier1.

We describe a case of anetoderma that developed on the neck, both arms and calves, and showed histopathological findings of actinic granuloma.

CASE REPORT

A 38-year-old Korean woman presented with multiple wrinkled macules. The skin lesions were seen 1 year ago and the size of the lesions gradually increased and became hypopigmented without increasing in number. A skin examination showed multiple, whitish to erythematous, ovoid, wrinkled macules on the right side of the neck, both arms and calves (Fig. 1). The lesions were slightly atrophic, soft and easily wrinkled when pinched between the fingers (Fig. 2). Otherwise, she was healthy. The lesions persisted despite treatment with topical corticosteroid.

A biopsy specimen taken from the macule of the left forearm revealed an atrophic epidermis. In addition histiocytic and multinucleated giant cell inflammatory reactions in the loosely edematous papillary and reticular dermis (Fig. 3). Some giant cells contained intracytoplasmic grayish fragmented elastotic materials. Diminished, fragmented black elastotic fibers in the entire dermis and relatively abundant elastic materials around the cellular infiltrates and in the multinucleated giant cells were seen on Verhoeff-van Gieson staining (Fig. 4). During 1-year of follow-up, the lesions gradually improved with sun-screen only.
Actinic granuloma is an annular inflammatory reaction that develops in skin after long-term sun-exposure. Actinic granuloma typically affects the exposed, weather-beaten skin of patients who are at least 30 and 40 years old or more. Favored sites are the neck, face, chest, and arms. Affected male and female patients are about equal in number. The lesions often begin as flesh-colored to pink papules and nodules that expand slowly to become annular to serpiginous plaques with normal to atrophic centers.

A biopsy specimen taken radially across the annulus shows three distinct zones in the dermis. At the periphery within normal skin, actinic elastosis is prominent. The annulus shows a histiocytic and giant cell inflammatory reaction in the papillary and mid-dermal region. The giant cells may contain intracytoplasmic degenerated elastic fibers or they may surround foci of elastosis. A central, relatively elastin-free zone is present within the annulus. O'Brien described four main histological patterns of actinic granuloma: giant cell, necrobiotic (vascular), histiocytic, and sarcoidal variants.

Our case showed interesting clinical findings of multiple, hypopigmented to erythematous wrinkled macules instead of typical annular or serpiginous plaques and characteristic clinical features of actinic granuloma. A histopathological examination showed many multinucleated giant cells containing fragmented elastic fibers in the entire dermis.
and diminished and fragmented elastic fibers, which explained the clinical presentation showing anetoderma. Our case did not show peripheral annular borders, but histopathologic findings showed those of the zone of annulus as described by O'Brien. We considered our case to have a giant cell variant of actinic granuloma. In Korea, giant cell and sarcoidal variants of actinic granuloma have been described.

Boreschi V et al. described “annular elastolytic giant cell granuloma” in a 13-year-old girl on sun-protected areas. Their case showed annular lesions with elevated edges and wrinkled centers, and combined features of actinic granuloma and anetoderma. They considered their case to be a variant of granuloma annulare. They excluded anetoderma in that anetoderma did not show annular borders clinically and scanty inflammatory cellular infiltrate were noticed in the initial stage of both inflammatory and non-inflammatory anetoderma histologically. Actinic granuloma was excluded in that their patient was so young and the lesions were distributed on sun-protected areas.

Anetoderma is characterized by discrete skin lesions that appear to be loose and wrinkled. The primary type arises in clinically normal skin, and the secondary type replaces the lesions of another disorder, such as acne, varicella, secondary syphilis, endocrine disorders, and cutaneous tumors. Among the causes of the secondary type of anetoderma, actinic granuloma have not been described. The pathogenesis of anetoderma remains unknown. The loss of elastic fibers may be due to either excessive degradation or to diminished fibers. In our case, the lesions were noticed more on both the forearms and calves, and more on the lateral side of the neck than the upper arms. We could not detect any lesions on the trunk and thighs. This suggests the role of actinic stimuli. In the pathogenesis of actinic granuloma, McGrae postulated that it represents a cell-mediated immune response to weakly antigenic determinants on actinically altered elastic fibers. We speculate that actinic stimuli may play a role in the pathogenesis of actinic granuloma, which leads to fragmented and diminished elastic fibers histopathologically and to macular atrophy (anetoderma) clinically. Moreover, our case showed a gradual improvement with sun-screen only. Actinic granulomas may persist for years but can spontaneously resolve. Actinic granuloma responds to topical corticosteroids and intralesional corticosteroids.

We describe a case of actinic granuloma presenting as anetoderma on sun-exposed areas. We suggest that actinic granuloma should be put on the list of causes of secondary anetoderma.

REFERENCES