Commentary

Granuloma faciale: A master masquerader?

Granuloma faciale (GF) is a rare disease of unknown etiology, described by John Edwin Mackonochie Wigley (1892-1962) in 1945, as Boeck's sarcoid, and Pinkus, in 1952, suggested the term granuloma faciale.[1] It is characterized by rather asymptomatic facial papules, plaques, and nodules of varying sizes and colors.[2] Rarely, lesions may be painful or itchy. Lesions usually involve sun-exposed areas such as cheeks, nose, forehead, chin, external ear, and preauricular areas. GF lesion mimicking rhinophyma has been reported.[3] Extrafacial lesions may develop occasionally on scalp, trunk, breast, and extremities.[4-6] Lesions have a smooth surface with follicular accentuation giving a peau de orange appearance and occasional telangiectasia. There are rare reports of keloidal variants of GF occurring on the face and extrafacial sites such as shoulder.[6,7] A mucosal variant of GF, eosinophilic angiocentric fibrosis (EAF), an unusual fibrotic condition affecting the mucosa of the upper respiratory tract, has been reported recently. This is histologically identical to GF and may be associated with cutaneous lesions of GF.[8] Histologically, GF shows evidence of leukocytoclastic vasculitis with extensive fibrin deposition.[1]

GF is a disease of the middle age and males are frequently affected. Although it is common in whites it has been reported rarely in Japanese and blacks. Sun exposure, polyclonal expansion of CD3+, CD4+, CD8+, and CD30+ T cells with upregulation of cytokines such as IL5[9] and gamma interferon[10] with subsequent inflammatory cell infiltration, have been thought to be etiological factors of GF.

GF is supposed to be a great mimicker clinically. It may resemble several dermatoses such as Sweet syndrome, Kimura's disease, cutaneous T-cell lymphoma, pseudolymphoma, lupus erythematosus, Jessner's lymphocytic infiltration, lymphocytoma cutis, sarcoidosis, and even insect bite reaction.[2]

Skin biopsy is often diagnostic even though there is no granuloma seen histologically (histologic misnomer). A grenz zone, polymorphic infiltrate composed of predominantly eosinophils, neutrophils, histiocytes, plasma cells and lymphocytes, perivascular inflammation with nuclear dust, extravasation of erythrocytes, and extensive deposition of fibrin and hemosiderin suggest that GF could be a subtype of leukocytoclastic vasculitis.[1]

Of late, dermoscopy has been found to be a novel diagnostic tool for GF. Translucent whitish-grayish structureless areas, intermingled with orthogonal whitish streaks, focused and elongated telangiectasias, or a pink background with whitish areas are the reported dermoscopic findings in GF.[11]

GF is usually resistant to therapy. Various treatment modes have been tried with variable effects such as topical tacrolimus,[12] topical and intralesional corticosteroids, dapsone, antimalarials, isoniazid, clofazimine,[3] and topical nitrogen mustard. A variety of surgical procedures such as surgical excision, dermabrasion, argon laser, pulsed dye laser, carbon dioxide laser, electrosurgery, and cryotherapy have been tried for the management of GF.[13]

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