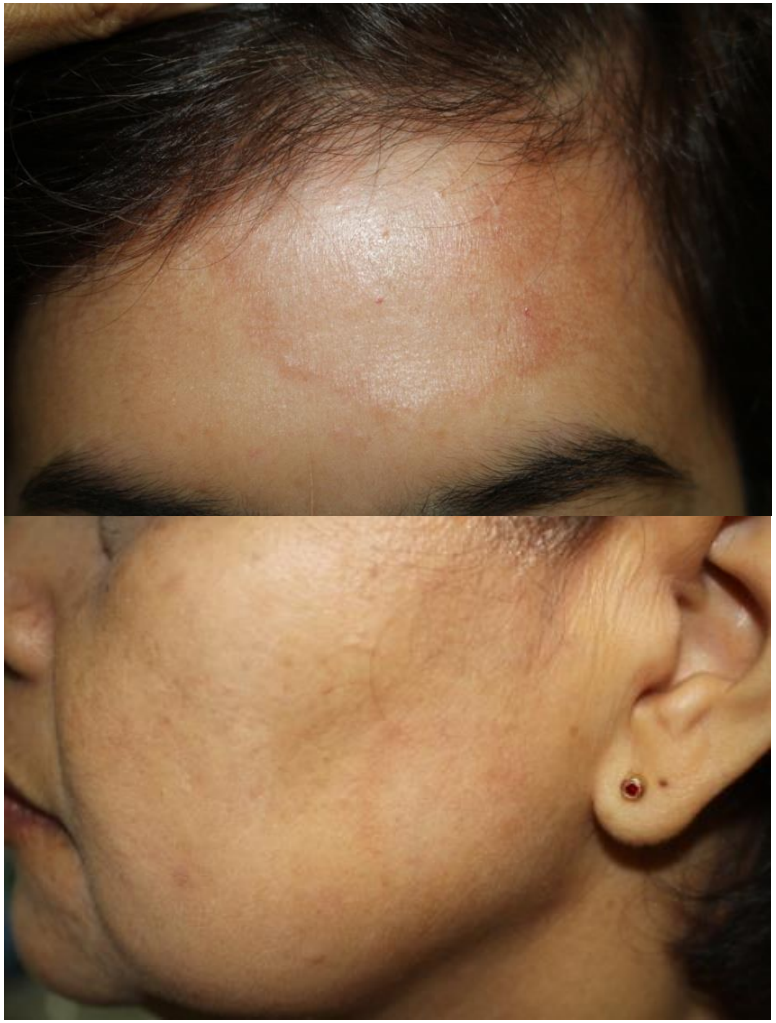
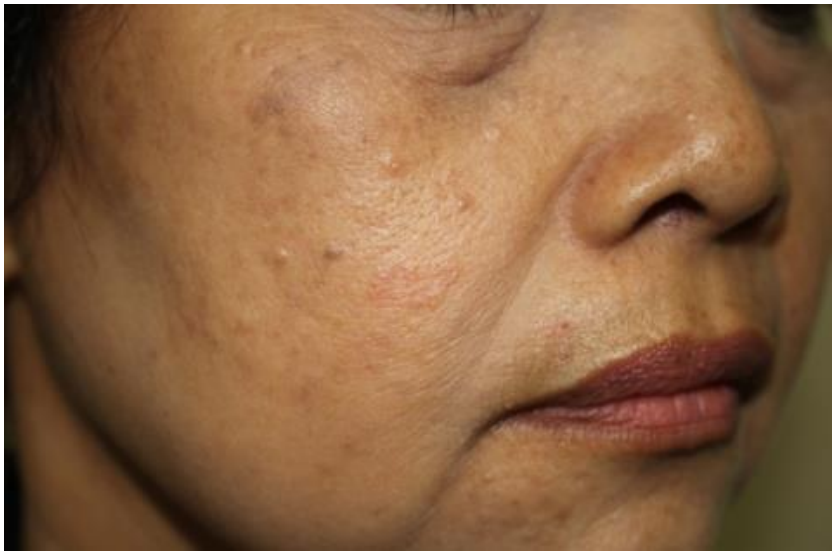
Case24
A 52-year-old Thai woman from Bangkok
Chief complaint: Annular erythematous plaques on face for 2 years

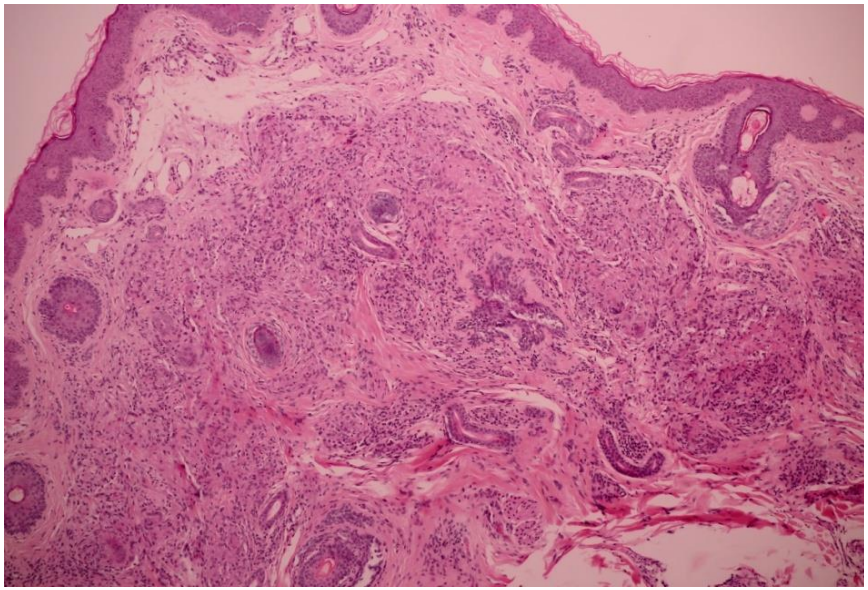




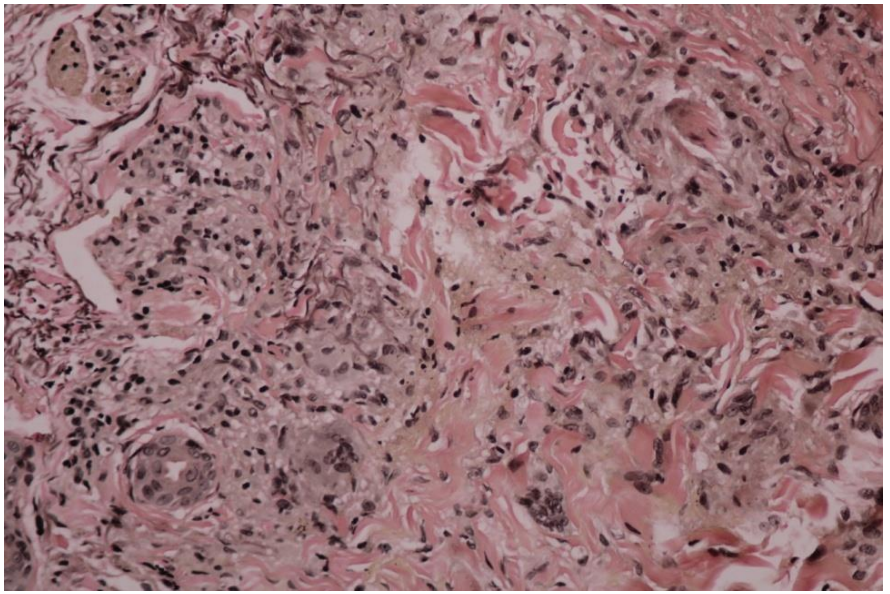
Present illness: The patient developed asymptomatic annular erythematous plaques on forehead and both cheeks for 2 years. The lesions gradually expand peripherally. She has no history of previous trauma or foreign material injection. She is a government officer and regularly works indoor.
Past history
Hypertension, dyslipidemia, chronic kidney disease stage II, and unspecified acute nephritic syndrome. Current medications include manidipine 20 mg/day, furosemide 40 mg/day, and simvastatin 10 mg/day.
Physical examination
HEENT: Not pale, no jaundice
Lymph node: Not palpable
Heart and lung: WNL
Abdomen: No hepatosplenomegaly
Neurological: No peripheral nerve enlargement

Skin examination
Few discrete annular erythematous plaques on forehead and both cheeks, size 1-5 cm. in diameter. Some lesions showed central hypopigmentation. There are solar lentigines and telangiectasias on malar area, nose, and forehead.

Histopathology: (S15-11976A, Left cheek) Nodular and interstitial inflammatory cell infiltrate of histiocytes intermingled with some lymphocytes in the dermis



Special stain: (S15-24592A, Left cheek) Some elastotic material phagoticized by multinucleated and marked decrease to absence of elastic tissue in some foci of affected dermis



Microscopic diagnosis: Actinic granuloma
Investigation
CBC and LFT: WNL
FBS: 90 mg/dL
BUN/Cr: 30/1.26 mg/dL
UA: Protein 4+
LDH: 255 U/L
Viral hepatitis profile, anti HIV: Negative
CXR: No pulmonary infiltration

Diagnosis: Actinic granuloma

Treatment
Sun avoidance
Short course systemic steroids
Hydroxychloroquine 200mg/day
0.1% Mometasone furoate cream apply twice daily

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Consultant: Vasanop Vachiramon, MD

Discussion:

Actinic granuloma (AG), also termed annular elastolytic giant cell granuloma, atypical necrobiosis lipoidica of the face and scalp, Miescher's granuloma of the face, and possibly granuloma multiformewas first described by O'Brien in 1975.[1-4] He postulated that actinically degenerated elastotic tissue could be the direct stimulus for the development of granulomas in this condition. The pathogenesis of AG is still not well understood. McGrae suggested an immune response mediated by cells to degenerated elastic tissue, with a predominance of helper T cells in the lymphocytic infiltrate.[5] Ultraviolet (UV) radiation, especially UVA and heat are recognized as causal factors, by changing the antigenicity of elastic fibers and producing an immune response.

AG is rare disease found equally regardless of gender. The common age of onset is between 40 and 70 years old. The typical cutaneous lesion of AG is initially smooth, elevated, nonscaly, erythematous papule which centrifugally extends to an annular plaque with central clearing, and sometimes atrophies or hypopigmentation. The lesions are usually distributed on chronically sun-exposed areas such as face, neck, dorsum of the hands, forearms, and upper back. Apart from the skin, conjunctival involvement has been reported for a few cases.[6, 7]

There are some reports association between AG and internal diseases such as hematologic and solid malignancy, monocloncal gammopathy, temporal arteritis, erythema nodosum, and x-linked dominant protoporphyria.[8-11] Diabetes mellitus has been found about 37-40% in patient with AG, may caused by injury of elastic fiber from hyperglycemic state.[12]

The differential diagnoses of AG are broad. These include granuloma annulare, erythema annulare centrifugum, annular lichen planus, secondary syphilis, necrobiosis lipoidica, tinea corporis, and tuberculoid leprosy. Therefore, histopathology is essential for diagnosis of AG. The best method to obtain an accurate histopathology is an elliptical biopsy across the annular rim and stained with elastic van Gieson to demonstrate the three zones of elastic tissue change. First zone, solar elastosis was identified in the surrounding unaffected skin. Second zone, granulomatous reaction consisting of histiocytes and foreign-body type multinucleated cells, with engulfment of elastotic fibers, representing the annular rim. Third zone, an absence of elastic tissue in the superficial dermis is found in center of the plaque.[13] Due to esthetic concern in our case, we decided to perform punch biopsy on her left cheek. The histopathology also show tuberculoid granuloma with elastophagocytosis, which is compatible with AG.

The treatment of AG is often unsuccessful. Topical, intralesional and systemic corticosteroids, topical pimecrolimus and tacrolimus, and phototherapy (narrow band UVB, PUVA, Re-PUVA) have been used with some benefit.[14-16] Cyclosporine A, dapsone, pentoxifylline, isotretinoin, and acitretin have been reported to be effective in some cases.[17-19] There are a few case reports with positive results from hydroxychloroquine and chloroquine therapy.[20] For preventing the new lesions, patients should also be educated to avoid sun exposure and regularly use sunscreen.

In our patient, she has good response to short course systemic steroids (prednisolone 10-15mg/day) with rapid relapse of the lesions after the treatment is discontinued.