Case 16.1
A 67-year-old Thai man from Nontaburi

**Chief complaint:** Asymptomatic erythematous papules and plaques for 2 months

**Present illness:** Two months, he developed asymptomatic multiple erythematous papules and plaque at v-shape of neck, upper back, both arms and hands which did not improve after treat with moderate potency steroids. He had no other symptoms.

**Past history**
Recently diagnosed with diabetes mellitus treated with metformin (500 mg) 1 tab@bid.

**Skin examination**
Multiple erythematous papules coalescing into large annular plaques with raised erythematous borders and slightly atrophic, hypopigmented central regions distributed mainly on sun-exposed areas.

**Histopathology** (S11-010080A)
- Palisading inflammatory-cell infiltrate of histiocytes, and multinucleated giant cells some containing elastotic tissue, in the upper dermis
- Negative for mucin and slightly decrease density of elastic tissue

**Immunohistochemistry:** Verhoeff-van Gieson stain
**Investigation:** FBS 117 mg/dL  
**Diagnosis:** Actinic granuloma  
**Treatment:** Neotigason (10 mg) 1 tab @ od pc, clobetasol propionate ointment apply bid  
**Diagnosis:** actinic granuloma  
**Treatment:** clobetasol propionate cream apply bid

**Case 16.2**

A 63-year-old Thai woman from Bangkok

**Chief complaint:** Pink papules and plaques for 1 year

**Present illness:** The patient noticed pink papules with centrifugal enlargement on both cheeks, v-shape of neck, both hands and arms with mild pruritus. She did not have other systemic symptoms.

**Past history**
Diagnosed with diabetes mellitus and hypertension for 4 years, current treatments including quinaril 1tab@od, felodipine 1tab@od, metformin (500 mg) 1tab@bid. Recent diagnosis of myelodysplastic syndrome for 6 months.

**Skin examination**
Multiple groups of slightly raised borders of pink papules coalescing into serpigenous plaques with slightly atrophic centre on sun exposed area.

**Histopathology (54-165A-B)**
Palisading inflammatory-cell infiltrate of histiocytes and multinucleated giant cells, some containing elastotic fibers, in the upper dermis
Diagnosis: Actinic granuloma
Treatment: clobetasol propionate cream apply bid

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Consultant: Penpun Wattanakrai

Discussion:

We present two cases of actinic granuloma. Actinic granuloma is sometimes considered to be a type of GA due to the similarities between the two. But yet, there is still a debate in their relationship.

Actinic granuloma is an uncommon, idiopathic disorder of middle-aged adults, which occurs more commonly in women than men. Clinically, it is characterized by raised erythematous borders and slightly hypopigmented centre on sun-exposed areas. The head, neck and arms are commonly involved.

The histopathologic similarities to GA are granulomas, multinucleated giant cells as well as lymphocytes and histiocytes. A recent study stated that the histopathologic findings that help distinguish actinic granuloma from GA include the presence of elastophagocytosis by multi-nucleated giant cells and the absence of elastic fibers in the center of the actinic granuloma lesions. Multinucleated histiocytes are conspicuous, usually large and contain as many as a dozen of nuclei, mostly in haphazard arrangement. Additionally, there should not be any increase in mucin, which might be expected in GA (see Table I). A recent study stated that the histopathologic findings that help distinguish actinic granuloma from GA include the presence of elastophagocytosis by multi-nucleated giant cells and the absence of elastic fibers in the center of the actinic granuloma lesions. Multinucleated histiocytes are conspicuous, usually large and contain as many as a dozen of nuclei, mostly in haphazard arrangement. Additionally, there should not be any increase in mucin, which might be expected in GA. (see Table I)

Actinic granuloma specifically appears in sun-exposed skin. The postulated hypothesis behind the actinic damage is that solar-damaged elastic fibers are antigenic and result in a cell-mediated immune response with a predominance of CD4 lymphocytes and subsequence into granulomatous inflammation. The sun-exposed distribution of the disease, the presence of CD4 cells, and the presence of giant cells ingesting the elastic fiber supports this hypothesis.
Table I. Histologic features of the major granulomatous dermatitides

<table>
<thead>
<tr>
<th>Granuloma annulare</th>
<th>AEGCG*</th>
<th>Necrobiosis lipoidica</th>
<th>Rheumatoid nodule</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical location</td>
<td>Superficial and mid dermis</td>
<td>Superficial and mid dermis</td>
<td>Entire dermis, subcutis</td>
</tr>
<tr>
<td>Granuloma pattern</td>
<td>Palisading or interstitial</td>
<td>Palisading, irregular</td>
<td>Diffuse palisading and interstitial; horizontal ‘tiers’</td>
</tr>
<tr>
<td>Necrobiosis (altered collagen)</td>
<td>Yes (‘blue’)</td>
<td>No</td>
<td>Yes (‘red’)</td>
</tr>
<tr>
<td>Giant cells</td>
<td>Variable</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Elastolysis</td>
<td>Variable</td>
<td>Yes</td>
<td>Variable</td>
</tr>
<tr>
<td>Elastophagocytosis</td>
<td>No</td>
<td>Yes</td>
<td>Variable</td>
</tr>
<tr>
<td>Asteroid bodies</td>
<td>Variable</td>
<td>Yes</td>
<td>Variable</td>
</tr>
<tr>
<td>Mucin</td>
<td>Yes</td>
<td>No</td>
<td>Minimal</td>
</tr>
<tr>
<td>Extracellular lipid</td>
<td>Variable</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Vascular changes</td>
<td>Variable</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

* annular elastolytic giant cell granuloma

Some authors reported lesions resembling actinic granuloma in unexposed areas. In addition, one case report described an inability to provoke lesions with a 4-day photochallenge in a patient with pre-existing actinic granuloma. The term ‘annular elastolytic giant cell granuloma’ is used to describe these features of annular lesions with a major granulomatous elastolytic component which may occur anywhere in the skin.

Several different forms of treatment have been proposed with varying results: hydroxychloroquine, intralesional steroids, cyclosporine, methotrexate and cryotherapy. Case reports have shown positive results with the use of isotretinoin, acitretin and retinoid psoralen + UVA (RePUVA). The mechanism of action of systemic retinoids is not yet completely understood; it is believed to be associated with their effect on photo-aging and actinic elastosis, which are present in actinic granuloma.

Both of our patients presented with sun-exposed lesions and histopathologic findings of lymphohistiocytic granuloma including the presence of elastophagocytosis by multi-nucleated giant cells that points to the diagnosis of actinic granuloma. We could not see absence of elastic fibers which could possibly be due to the early time of biopsy.

Interestingly, both of our patients are associated with diabetes mellitus, a previously described co-existing disease in actinic granuloma and GA. Diabetes mellitus may have a contributive role in the pathogenesis of actinic granuloma as in GA.

General advice for both patients was sun avoidance. For treatment, the first case received systemic retinoid treatment and showed some improvement while the second patient applying only potent topical steroid had no improvement. The use of systemic acitretin is under our future plan.
References