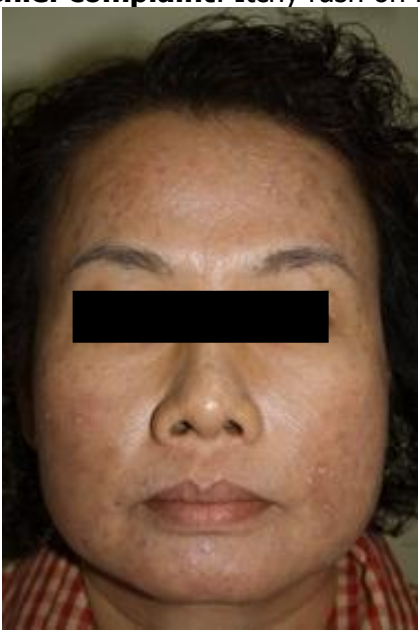
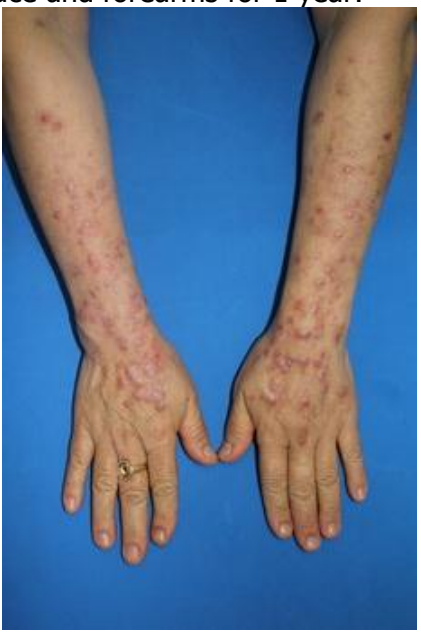
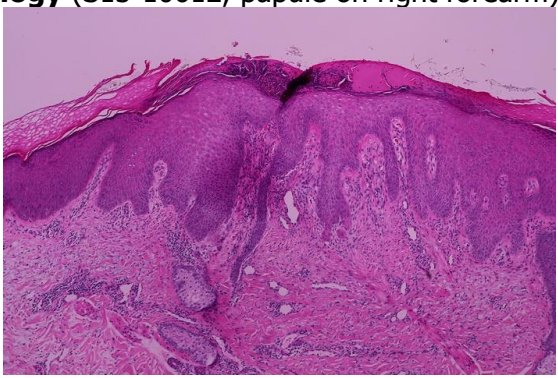
Case 19
A 48-year-old housewife from Ratchaburi.
Chief complaint: Itchy rash on face and forearms for 1 year.

Present illness: The patient has developed multiple red bumps on her face and extensor aspect of both forearms and both hands for 1 year. The lesions were accompanied by intense itching, from which she often had to scratch them to find some relief. They gradually increased in number. She has never had any blistering skin lesions. She could not relate the onset of her symptoms with any activity, exposure to sunlight or chemicals, or use of any oral or topical medication. She denies history of prolonged fever, weight loss, joint pain or swelling, or oral ulcers.
Past history: She has had hypertension and hyperlipidemia for 10 years. She is currently on hydrochlorothiazide 25 mg/day, olmesartan 20 mg/day, and simvastatin 10 mg/day, which have

been unchanged for many years.
Family history: None of other family members have similar skin lesions.
Physical examination: Her vital signs, HEENT, cardiovascular, respiratory, and abdominal examinations are normal.
Skin examination: Multiple, well-defined, excoriated, round, erythematous papules on face and extensor aspect of both forearms and both hands.
Investigation: CBC, fasting blood sugar, as well as liver, renal, and thyroid function tests are within normal limits. ANA and anti-HIV are negative. Minimal erythema doses (MED) of UVA and UVB are normal, measuring over 18 J/cm^2 and over 90 mJ/cm^2, respectively. Photoprovocation test with UVA at 1.5 MED (40 J/cm^2) for 3 consecutive days is negative, whereas the test with UVB produces a few small erythematous papules after 2 consecutive days of irradiation with 1.5 MED (140 mJ/cm^2). Patch and photopatch tests are negative.

Histopathology (S13-10612, papule on right forearm)



• Hyperkeratosis, parakeratosis with serum, and marked epidermal hyperplasia with focal spongiosis
• Superficial perivascular lymphocytic infiltrate in the thickened papillary dermis

Diagnosis: Actinic prurigo

Treatment: 0.05% clobetasol propionate cream applied twice daily and 10% salicylic acid cream applied nightly to lesions on forearms and hands, 0.02% triamcinolone acetonide cream applied twice daily to lesions on face, oral antihistamines, sun-protection measures including regular sunscreen use, and vitamin D supplement

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Discussion

Actinic prurigo (AP) is a rare acquired idiopathic photodermatosis characterized by persistent, pruritic, excoriated, papular or nodular eruption, predominantly in sun-exposed areas. The differential diagnosis may include polymorphic light eruption, hydroa vacciniforme, photoaggravated atopic dermatitis, photoallergic dermatitis, cutaneous lupus erythematosus, and prurigo nodularis.[1] The diagnosis of AP may be assisted by phototest and photoprovocation test, which can give positive results to variable action spectra, i.e. either UVA, UVB, or both.[2] AP is associated with HLA-DR4, especially the HLA-DRB1*0407 subtype, which may help to distinguish AP from polymorphic light eruption when the clinical features are not distinctive.[3]

AP most commonly affects American Indians and mestizos (people of mixed Caucasian and American Indian ancestry).[4] The clinical features of AP in American Indians and mestizos include early onset typically at 4-5 years of age, female-to-male ratio of 2:1,[4] possible conjunctivitis and/or cheilitis, and a hereditary tendency.[5] Many published AP cases from other populations, including Caucasian (British,[3, 6] French,[7] Albanian,[8] Australian[9]) and

Asian (Chinese,[10] Thai[11]), seem to share the same clinical features, and HLA associations when applicable, as the American Indians and mestizos.

However, there have been a few reports of a seemingly different subset of AP patients from Thailand[2] and Singapore,[12, 13] which was named "actinic prurigo, tropical (Southeast Asian) variant" by Tham and Tay.[12] Onset in adulthood (with mean age of onset as high as 36 years in Thais), male predominance, absence of family history, absence of conjunctivitis, and rarity of cheilitis are the characteristics different from those in American Indians and mestizos according to their case series.[2, 13] Nevertheless, a number of cases from some of the above-mentioned reports from American Indians[5] and other populations[3, 7, 9] also had onset in adulthood. This group of patients tends to have milder but more persistent disease, compared to their juvenile-onset counterpart.[1, 5]

Our patient presents with adult-onset pruriginous lesions in sun-exposed areas, without conjunctivitis or cheilitis. The differential diagnosis in our case may include hypertrophic discoid lupus erythematosus, photoallergic dermatitis, polymorphic light eruption, acquired perforating dermatosis, and prurigo nodularis. The clinical appearance, pathological result, positive photo-provocation test, as well as negative patch, photopatch, and other laboratory tests warrant the diagnosis of AP. She should fit into the Southeast Asian variant, if appropriate.

AP is generally recalcitrant to therapy, with frequent recurrence despite appropriate treatment.[2] The mainstay treatment is avoidance of sunlight and regular sunscreen use. Other treatment modalities include topical corticosteroids, keratolytics, emollients oral antihistamines, short courses of oral corticosteroids, photo-therapy, and thalidomide[1, 4, 9] In our case, only antihistamine and topical therapy were given. If the current treatment gives unsatisfactory results, hardening phototherapy may be considered in the future.

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