Pruritic Erythematous Plaques Over the Face, Neck, and Extremities

Hsiao-Feng Liao¹ Chia-Yu Chu²

CASE REPORT

A 61 years old healthy man presented with pruritic eruption over his face, neck, chest, and upper extremities for 4 years. He denied chronic drug history but reported contact history with formaldehyde, wood, and melamine (1,3,5-triazine-2,4,6- triamine). Photoaggravation was also noted.

On examination, there were some erythematous eczematous plaques over the forehead, nose, neck, V-chest, nape, and upper extremities especially the extensor side and dorsal hands (Fig. 1). Skin biopsy from the forehead showed mild acanthosis with spongiosis and superficial perivascular lymphocytic and eosinophilic infiltration (Fig. 2). Results of anti-extractable nuclear antibody (Jo-1, scl-70, ribonucleoprotein, Sm, SSA, SSb) and anti-nuclear antibody (ANA) were negative. C3 and C4 quantitation were within normal limit. The patient’s skin phototype was type IV. Photo test at 24 hours and 48 hours disclosed that his minimal erythema dose (MED) of UVB was 60 mJ/cm². Patch testing of the European standard and photoallergen series were positive for potassium dichromate 0.5% and nickel sulphate 5%, which were not relevant to current exposure.
DIAGNOSIS: Chronic actinic dermatitis (CAD)

DISCUSSION

CAD is the unified denomination defined by Hawk and Magnus, including a series of clinical pictures described separately but with the common features of a chronic progressively aggravating photosensitivity. Persistent light reactivity, actinic reticuloid, photosensitive eczema, and photosensitivity dermatitis are all now considered variants of this syndrome. CAD occurs predominately in middle-aged or elderly males. The eruption affects mostly sun-exposed sites, notably the face, scalp, nape and sides of the neck, upper chest, and dorsa of hands and forearms, and is worse in summer. Onset of the eruption following ultraviolet radiation exposure may be insidious, delayed by hours to days. The pathogenesis is not completely understood, but any explanation must take into account the broad-spectrum abnormal photosensitivity. It has been suggested that the persistence of photoallergens in the skin, exogenous chemically induced photosensitivity, endogenous photosensitizer, cellular defect in the repair or prevention of photoproduction-induced damage, and failure in the normal suppression of delayed-type hypersensitivity, may be involved. Photo-testing is essential to confirm the diagnosis of CAD, and is characterized by abnormally low erythema thresholds with eczematous or pseudolymphomatous responses following UVB, and, in the majority of patients, also UVA. Some patients also react to visible light. Histological examinations, if performed, showed nonspecific eczema. CAD must be distinguished from allergic contact dermatitis, especially airborne allergens, from oral drug photosensitivity, from light-exacerbated endogenous eczemas, and from photoallergic contact dermatitis to sunscreens. CAD is a disabling and persistent disease, and treatment is often partially effective. Avoidance of UVR is necessary for all patients. Other treatment includes topical tacrolimus or pimecrolimus, azathioprine, cyclosporine, mycophenolate mofetil, and low dose PUVA.

REFERENCES