

rounded by a zone of normal skin and a peripheral ring of macular hyperpigmentation.

Histopathological changes were similar, though not identical, in all these cases. The central lesion was either a compound or a junctional naevus and the halo was formed by collections of pigmented junctional naevus cells except in the first case reported by Warin, in which excess pigment was present in the epidermis without obvious increase in the melanocyte population. In the case reported here, and in the second case reported by Warin, junctional naevus cells could be traced from the central tumour to a point where the pigmented halo was formed.

The cause of this unusual morphological pattern is uncertain. The positive Dopa oxidase reaction within epidermal melanocytes indicates that these cells contain tyrosinase and are capable of synthesizing melanin (end-product of the reaction) (5). That these same cells do not stain for melanin suggests that melanin synthesis has been blocked. Melanin precursors produced by the epidermal melanocytes may inhibit melanin biosynthesis (1), or the dermal component of the naevus in some way may inhibit melanin synthesis in the overlying epidermal melanocytes. Away from the tumour where inhibition weakens, melanin can be synthesized, leading to a ring of pigmentation. Inhibitors of tyrosinase occur in certain amelanotic melanomas and are thought to account for the lack of melanin in these tumours (6).

The absence of a dense lympho-histiocytic infiltrate beneath the tumour and the presence of Dopa oxidase positive melanocytes in the overlying epidermis indicate that the absence of melanin in these tumours is unlikely to be due to an immunological mechanism. Such mechanisms are thought to account for the depigmentation which occurs in Sutton's halo naevus. However, in this tumour the presence of an infiltrate and loss of epidermal melanocyte Dopa oxidase reactivity are characteristic features (3).

In conclusion, this report documents a case of unusual target-like or cockade naevi arising on the trunk of a young girl. This type of naevus appears to be a benign but unusual variant of the cellular naevus and at least in some cases, the unusual morphology could be explained by the inhibition of melanin synthesis in epidermal melanocytes.

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Solar Urticaria: A Case with Increased Skin Mast Cells and Good Therapeutic Response to an Antihistamine

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Abstract. A 45-year-old woman with solar urticaria is described. She was found to have: (a) increase of skin mast cells; (b) a good therapeutic response to hydroxyzine hydrochloride; (c) frequent peptic ulcers of the stomach; (d) antinuclear IgM antibodies in a titre of 1:1000; (e) cold urticaria and dermographism; (f) anamnesticallly, suppression of the solar urticaria by a sequential oral contraceptive.

Key words: Urticaria; Mast cells

Solar urticaria is a fairly uncommon but well recognized clinical entity characterized by erythema, itching and wealing immediately after exposure to sunlight. The symptoms appear within a few minutes and usually do not persist for more than one hour.

The pathogenetic mechanisms are not clear. Immunoglobulins may be involved, according to suc-

cessful passive and reverse passive transfer tests. Histamine is assumed to play an important role as mediator (15), and recently two different research groups demonstrated elevated serum histamine levels in venous blood draining ultraviolet (UV) irradiated wealed skin (6, 12). Increase of skin mast cells has been reported (1). Stimulation of gastric secretion (7) and gastric ulcers (14) are also mentioned in the literature.

Treatment has been disappointing. Occasional therapeutic successes have been achieved with various treatment modalities (3).

CASE REPORT

A female laboratory assistant, born in 1933, had been in good health until summer 1961, when she noted typical symptoms of solar urticaria. They were provoked mostly by direct sunshine, but also through a glass window. In the latter case a much longer exposure was needed. Since 1967 the patient had had five episodes of peptic ulcer of the stomach. From 1970 to 1972 she used an oral sequential contraceptive containing ethinyloestradiol and lynestrenol. During that time she tolerated sunlight much better, only occasionally noticing slight symptoms. In 1972, because of a rise in her blood pressure, she discontinued taking the contraceptive. Instead, she was prescribed a 'minipill' containing only lynestrenol. Subsequently, her solar urticaria again became much worse and she began to get occasional symptoms of cold urticaria and dermographism.

Phototests

The light source was a 400 W dysprosium lamp (HQI-TS 400W, Osram GmbH, West Germany), which in addition to UVB has a high output of UV A rays (4). The tests were performed with unfiltered radiation from the lamp, and with a 5 mm thick plate of window glass to filter out the UV B rays. The antihistamine therapy was discontinued one week prior to testing. Wealing, confined to the irradiated area, was readily produced with a low dose of both filtered and unfiltered UV light. Hydroxyzine hydrochloride (Atarax®, UCB), 25 mg, given perorally 5 hours before testing, prevented the wealing, but not the immediate erythema.

Microscopy

Skin biopsies were obtained from the test site 5 min after the weal formation, and from non-irradiated skin. A 1% lidocaine solution was used as local anaesthetic. Formalin-fixed specimens were stained with haematoxylin + eosin and with toluidine blue for mast cells. All sections showed a rather sparse inflammatory infiltrate perivascularly and around the skin appendages. Half of the cells of the infiltrate were mast cells, up to 15 per microscope field (480×). No significant difference in the number of mast cells was found between irradiated and control skin. Deep-frozen skin sections were investigated by means of

immunofluorescence procedures for IgG, IgM, IgA, C₃ and fibrin (10). The results were negative.

Miscellaneous investigations

Blood, urine, bone marrow and X-ray investigations were performed. They revealed only a slight increase in the serum IgM level and antinuclear antibodies (ANA) of the IgM class in a titre of 1:1000. No other findings suggestive of a systemic lupus erythematosus (SLE) were found.

Therapy

Trials with a topical sunscreen preparation (Contralum®, Hermal Chemie), oral chloroquine and oral beta-carotene proved unsuccessful. Since 1975 the patient has been using hydroxyzine hydrochloride, 10–60 mg daily, with good response. This antihistamine allows her to go about her normal daily life, even to sunbathe, with minimal side effects. After discontinuation of the therapy the symptoms regularly reappear within a few days.

DISCUSSION

Skin sections showed mast cells in unusual numbers. Neither the histological nor the clinical picture was that of urticaria pigmentosa or systemic mastocytosis. The number of dermal mast cells can be increased in many inflammatory conditions, e.g. in the granulation tissue of healing wounds, in atopic dermatitis, lichen planus, pemphigus vulgaris, urticaria and lupus erythematosus (8). The mechanism and significance of this non-pathognomonic finding remain unsolved.

The frequent occurrence of the peptic ulcers may be connected with the skin condition. Stimulated gastric secretion in association with solar urticaria has been described (7), and a recent report presented a case with peptic ulcers (14). On the other hand, large numbers of mast cells have been found in biopsies of stomachs with peptic ulcers (11).

Occasional cold urticaria and dermographism were probably unspecific, reflecting the increased number of dermal mast cells, which, following minor irritations, could liberate histamine in sufficient amounts to cause clinical symptoms.

The good therapeutic response to an antihistamine favours the concept that histamine has some importance as mediator, at least in this case of solar urticaria. The negative results of the IF investigations do not exclude an antibody-mediated pathogenesis. Transfer tests were not considered justified. Our phototests suggest only that the UV A range is included in the action spectrum. A more accurate determination was not possible because the appropriate equipment was not available. This,

together with omission of passive transfer tests, makes it impossible to place our case in any group of the proposed classifications (3, 5).

Our patient showed ANA in a high titre, but no other signs of SLE or other collagen diseases. Nevertheless, cases of SLE with solar urticaria (13), even as presenting symptom (2), have been reported. In our case, however, SLE seems unlikely.

Hormones are generally known to be modulating factors in urticaria (9), and the anamnestic tolerance of our patient to sunlight when she was taking pills containing oestrogen is noteworthy. Oestrogens have been used in the treatment of solar urticaria (3) without any dramatic effect.

In further cases of solar urticaria attention should be paid to the number of dermal mast cells and to the symptoms of any gastric ulcer. Therapeutically, antihistamines (especially hydroxyzine hydrochloride) and oestrogens are worth trying.

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Circulating Anticoagulant and Serological Tests for Syphilis

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Abstract. Circulating anticoagulant (CA) and particularly lupus anticoagulant are commonly associated with biological false-positive tests for syphilis (BFP-STs) in patients with collagen diseases. CA was presently found in the sera of 8 out of 30 subjects with chronic BFP-STs without collagen or autoimmune diseases. It was not found in any of the 21 patients with various stages of syphilis. In 5 out of 21 elderly subjects (age >70 years) in whom a positive BFP-STs was detected, there was no CA. It is concluded that the association between CA and BFP-STs is found only in patients with collagen and autoimmune diseases and in some of the younger chronic BFP-STs reactors. It is not detected in syphilitic patients or elderly subjects in whom a high incidence of BFP-STs can be found. The difference in the incidence of this association is probably due to the differing biologic behaviour of these autoantibodies.

Key words: Circulating anticoagulant; Lupus anticoagulant; Serological tests for syphilis; Syphilis

The appearance of circulating anticoagulants (CA), directed against most of the clotting factors may