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Successful treatment of systemic cold contact urticaria with etanercept in a patient with psoriasis

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MADAM, We report a 24-year-old white man who presented with long-standing psoriasis and psoriatic arthritis (8 years) in progressive deterioration. On inquiry, the patient reported that he was also affected by recurrent episodes of cold contact urticaria (CCU) over the last 10 years. If a cold object was applied anywhere on the skin, the patient would experience swelling, itching and weals 10–20 min later at the pressure site and on the back (Fig. 1).

After the first year of symptoms, the patient was diagnosed with CCU and was prescribed cetirizine 20 mg daily, which



Fig 1. Development of an atypical urticarial reaction on the back of the patient after 10–20 min of application of an ice cube on the volar side of his arm.

was reduced to 10 mg daily after 8 years. After many years of cetirizine and avoiding contact with particularly cold objects, the symptoms were stable but flared again if cetirizine was discontinued. Up until the time of presentation, the patient was taking cetirizine 10 mg daily to minimize his CCU symptoms. None the less, a mild level of CCU remained.

Regarding the patient's psoriasis, many previous treatments had failed to make any tolerable improvement: narrowband ultraviolet B (which had to be interrupted because of the great distance between the patient's home and the phototherapy unit), acitretin, methotrexate (which gave no substantial improvement) and ciclosporin (which had to be stopped because of leukopenia).

Nonsteroidal anti-inflammatory drugs had to be interrupted after 5 years of cyclic intake because of the onset of gastritis despite the concomitant intake of esomeprazole 40 mg daily.

Considering the Psoriasis Area and Severity Index (PASI) value (which was 18) and joint involvement, etanercept was started at the recommended dose of 25 mg subcutaneously twice per week for 24 weeks then 50 mg twice a week for 4 weeks, after all contraindications were ruled out.

A few weeks after starting etanercept, the patient experienced a rapid resolution of psoriasis and urticaria manifestations too, and these results were maintained during the treatment period. Then upon the patient's request treatment with etanercept was interrupted, with subsequent and fast relapse of CCU, followed by a reappearance of psoriasis (PASI 20) and psoriatic arthritis. Therefore, a second cycle of etanercept treatment at 25 mg subcutaneously twice per week was indicated. A few weeks after restarting etanercept, the PASI decreased to 0 and the urticarial symptoms disappeared too.

At the last follow-up, 1 year after the disappearance of all CCU symptoms, the patient was still free of any CCU symptoms and the PASI has remained at < 2.

Among all physical urticarias, CCU represents the second most common subtype after symptomatic dermatographism.¹ It is characterized by the development of weals and/or angio-oedema within minutes after cold contact, including exposure to cold air, liquids or objects, and persists for up to 2 h. Extensive cold contact of large areas of skin may lead to systemic reactions such as generalized urticaria, dyspnoea, tachycardia, hypotension and loss of consciousness.^{2,3}

Cold urticarias may be classified according to two different criteria: (i) the presence or absence of a family history of the condition (familial or acquired cold urticaria), and (ii) the results of the cold contact challenge test (typical or atypical cold urticarias).²

The response to the cold contact challenge test is considered to be 'typical' when a weal is induced in < 20 min in the cold-stimulated area (80–90% of all these urticaria types); the result is termed 'atypical' when there is no response, when the response occurs in an area distant from the stimulated one, or when no immediate response occurs.

Etanercept is a recently developed competitive inhibitor of the proinflammatory cytokine tumour necrosis factor (TNF)- α . It binds with TNF- α at a high affinity, thus preventing TNF- α from binding to its cell surface receptors. In clinical trials, etanercept

has been shown to be effective against psoriasis, without notable side-effects, as well as in rheumatoid diseases.^{4,5}

It has been shown that TNF- α is one of the mediators released in patients with CCU at significantly elevated levels in the skin and blood in the course of mast cell degranulation, together with other mediators such as histamine, prostaglandin D₂ and platelet activation factor. Increased expression of TNF- α and of interleukin 3 in the endothelial cells of healthy and affected skin has been reported in patients with different types of urticaria, including cold urticaria.⁶

This is the first report of successful treatment of CCU with a TNF- α inhibitor. The rapid and complete improvement supports the pathogenic role of TNF- α in CCU. Obviously, the rarity of the disease and the variation in the disease activity makes the real efficacy of this treatment difficult to assess. Further clinical trials should be performed, better to evaluate the efficacy of this treatment in other patients with CCU.

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Scalp discoloration from selenium sulphide: revisiting a report of terra firma-forme dermatosis of the scalp

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MADAM, A distinctive pattern of red-brown scalp discoloration has recently been described in six children due to the usage of

various selenium sulphide-containing shampoos.¹ In light of this report, I believe that the interesting Clinical Cameo by Mallari and Sinclair² describing a 4-year-old child with striking red discoloration in a patch of alopecia areata represents this selenium sulphide staining phenomenon rather than the diagnosis of terra firma-forme dermatosis given by the authors. First, the red-brown discoloration in the article's figures matches very well with the colour I've seen due to selenium sulphide-containing shampoos and has never been convincingly shown to occur in any other entities. Secondly, the red-brown discoloration in the case described by Mallari and Sinclair has never been reported specifically in terra firma-forme dermatosis (even though it is thought to be a relatively common, under-reported dermatosis), including in a recent series of over 30 patients with terra firma-forme dermatosis from my institution.³ Furthermore, location on the scalp would be unusual for terra firma-forme dermatosis. Moreover, terra firma-forme dermatosis has otherwise never been reported in a patch of alopecia areata. Finally, the finding of (complete or partial) lesion removal with alcohol swabbing (but not soap and/or water) is not specific for terra firma-forme dermatosis, but instead has been described in other disorders of retention hyperkeratosis (such as confluent and reticulated papillomatosis and acanthosis nigricans)^{4,5} as well as selenium sulphide shampoo-associated discoloration.¹ In summary, I believe it is instructive to revisit the case of Mallari and Sinclair and that clinicians maintain selenium sulphide staining in the differential diagnosis of unusual scalp discoloration, especially in children.

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