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Oral monteleukast in urticaria induced by nonsteroidal anti-inflammatory drugs

Editor

About 30% of patients with chronic urticaria experience flares of hives and/or angio-oedema after ingesting either aspirin or nonsteroidal anti-inflammatory drugs (NSAID).¹ In such patients, cross-reactivity to all NSAIDs seems to occur; moreover, their sensitivity is particularly high when urticaria is active and disappears when the underlying disease is in remission. The pathogenic basis of such reactions are still poorly defined; nonetheless, cross-reactivity between chemically efficient NSAIDs suggests a mechanism dependent on cyclooxygenase inhibition. This hypothesis is supported by recent reports

of effectiveness of leukotriene antagonists in some patients with chronic urticaria.^{2–5} We conducted an open study of monteleukast in NSAID-induced urticaria.

Twenty-five patients with NSAID-induced urticaria were recruited from our urticaria clinic to evaluate the efficacy of oral leukotriene antagonists (oral monteleukast, available as Tab Montair 10 mg; CIPLA Pharmaceuticals, Mumbai, India). The patients consist of six men and 19 women, aged between 20 and 65 years. The study was conducted for 6 weeks. All the patients were prescribed oral monteleukast 10 mg once daily to be taken for 6 weeks.

Pruritus severity was measured and graded on a scale of 0–4: 0 = none; 1 = mild; 2 = moderate, annoying and troublesome; 3 = severe, substantially interfering with sleep/daily activities; and 4 = very severe, intolerable. Similarly, number of weals were rated on scale of 0–4: 0 = none; 1 = 1–5 weals; 2 = 6–15 weals; 3 = 16–25 weals; and 4 ≥ 25 weals. Number of episodes were also taken into consideration for calculating urticaria severity as 0 = 0; 1 = 1 episode; 2 = 2 episode; and 3 ≥ 3 episodes per 24 h. A mark to complete improvement was defined as a decrease in score by 70% or more from baseline. The patients were re-evaluated weekly for 4 weeks and then at the end of 2 weeks (i.e. 6 weeks).

At the end of the study, 22 patients showed marked to complete improvement (> 70% reduction from the baseline score) at the end of 3 weeks treatment and no relapse thereafter up to 6 weeks of therapy. Two patients complained of relapse due to stoppage of therapy in between, but showed sustained relief with continuous treatment without any further relapse. Only one patient was lost for follow-up. During the 6-week treatment, all the patients were well and never took any antihistamine drugs.

The possibility that cyclooxygenase blockade plays a pivotal role in the pathogenesis of aspirin-induced asthma has been known for years and was recently confirmed by observation that leukotriene antagonists may prevent asthma following aspirin challenge. The multiple NSAIDs sensitivity, which characterizes a subset of patients with chronic urticaria, raised the possibility that similar mechanism might play a role in them as well. This hypothesis was recently supported by the study reporting a favourable effect of leukotriene antagonists in patients with chronic urticaria, particularly in those with NSAIDs intolerance.

In conclusion, our positive results with monteleukast support possible role of leukotrienes in the pathogenesis of chronic urticaria with intolerance to NSAIDs. A strong advantage of monteleukast is pharmacokinetic profile: a single dose may provide protection for 24 h.⁶ Additional studies are required to characterize further optimal conditions for the use of monteleukast in chronic urticaria.

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Confetti-like hypopigmentation: a rare complication of common phototherapeutic modality

Editor

Hypopigmentation and vitiligo occurring during psoralen and ultraviolet A (PUVA) therapy have been described in the literature.^{1,2} Leucoderma punctatum, a confetti-like hypopigmentation, as a distinct entity was first described by Falabella *et al.*³

A 21-year-old woman presented with multiple asymptomatic, punctiform, achromic spots of 1-month duration, distributed predominantly on extensor aspect of extremities. She had vitiligo vulgaris and was being treated with trimethylpsoralen 0.6 mg/kg, three times per week, followed by UVA exposure for 3 months before appearance of these lesions. Before starting PUVA treatment she was given 8 months' treatment of narrowband ultraviolet B (NBUVB), during which no similar lesion was observed. She was shifted to PUVA as there was minimal improvement of vitiligo with NBUVB. She had skin type V and had 40% repigmentation of the vitiliginous lesions over this period. Her disease was stable during therapy. On

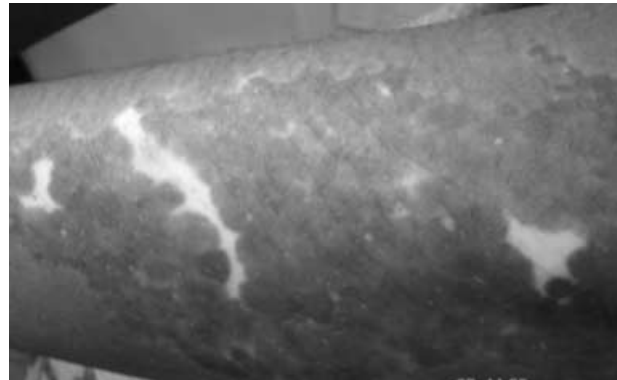


fig. 1 Multiple perifollicular hypopigmented macules on repigmented vitiligo lesions.

examination she had several small hypopigmented to depigmented macules, round to oval in shape, with well-defined margins and measuring about 1.5 mm, which were distributed over the repigmented areas (fig. 1). These lesions were distributed over extensor aspect of the extremities and were predominantly perifollicular. There was no history of phototoxic reaction or inflammatory lesions. Skin biopsy was taken from a representative lesion, which showed patchy loss of pigment in basal keratinocytes with mild hyperkeratosis of the epidermis. The photochemotherapy was subsequently discontinued.

Falabella *et al.*³ first described leucoderma punctatum in 1988, in 13 vitiligo patients during and after PUVASOL (oral psoralen followed by solar UV exposure) treatment as leucoderma distinct from idiopathic guttate hypomelanosis and vitiligo. Later, Park *et al.*⁴ reported four cases of generalized punctate leucoderma after UVB phototherapy for psoriasis. Most of the cases were females. Dogra *et al.*⁵ first reported a case of leucoderma punctatum appearing after systemic PUVA therapy. Recently, it has been reported after topical PUVA treatment for segmental vitiligo by Park *et al.*⁶ The typical skin lesions described are multiple discrete well-defined macules of size varying from 0.5 to 1.5 mm, most commonly located on the upper and lower extremities. Special stains for melanocytes show a decreased number of functional melanocytes. In ultrastructural study by Falabella *et al.*,³ cellular damage within keratinocytes and melanocytes was evident in form of intracellular oedema and intracytoplasmic vacuolar degeneration. Although pathogenesis is not well understood, the most likely cause is PUVA and UVB phototoxicity-induced damage to melanocytes. Park *et al.*⁴ also suggested that cumulative UVB phototoxicity may be the cause of leucoderma punctatum. In this case, perhaps the exposure to both NBUVB and PUVA was the cause of phototoxicity and appearance of these lesions. It is important to be aware of this rare