

Topical glucocorticoid augments IgE-mediated passive cutaneous anaphylaxis in Balb/C mice and mast cell deficient WBB6F1 v/v mice

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Summary

Background In a last decade, new types of skin manifestations have been recognized in atopic dermatitis especially in Japan. They are frequently observed in adult patients with atopic dermatitis after a long-standing steroid ointment and termed adult type-atopic dermatitis.

Objective To clarify whether topical glucocorticoid (GC) modulates cutaneous inflammatory reactions in addition to known anti-inflammatory effect, we have examined the effect of long-term application of topical GC on IgE-mediated murine cutaneous reactions.

Methods Fifty microlitres of diflucortolone valerate (1 mg/mL), prednisolone valerate-acetate (3 mg/mL), or triamcinolone acetonide (1 mg/mL) were applied seven times on alternate day, to the flank skin of mice. On day 12 when mice received the seventh application of GC, each mouse was given an intravenous application of IgE anti-DNP antibody (PCA titre $\times 2560$) 1 h before the skin test with 0.15% DNFB in acetone:olive oil (4 : 1) on the right pinna. The left pinna was painted with a vehicle as a control. Increased ear thickness was measured at 1, 4, 24, 48 and 72 h to assess the augmentary effect of GC.

Results Topical application of GC (50 μ g diflucortolone valerate in ethanol) on the flank skin seven times on alternate days, augmented expression of passive cutaneous anaphylaxis reaction on the ear skin induced by intravenous applications of monoclonal IgE anti-DNP antibody and following the challenge test. In contrast, topical application of GC inhibited the reactions when applied on the challenged sites. Several types of GC, but not vitamin D₃, augmented the skin reactions and these augmented reactions persisted for 72 h when control skin reactions subsided. GC induced a late phase but not an early phase cutaneous reaction in mast cell deficient WBB6F1 v/v mice by IgE anti-DNP antibody.

Conclusion Long-term application of topical GC might modulate local cutaneous immune response and augment IgE-mediated cutaneous reactions. Fc ϵ R(+) cells other than mast cell might be involved in the IgE-mediated late-phase reaction.

Keywords: atopic dermatitis, steroid, mast cell, Fc ϵ receptor, late-phase reaction

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Introduction

In the last decade, new types of skin manifestations have been recognized in patients with atopic dermatitis (AD), especially in Japan [1–3]. These include acute onset of

facial and/or generalized swelling of the skin followed by oozing eczematous change with occasional superficial bacterial infection (rubor of the skin), persistent erythema of the face (atopic red face) and reticular poikilodermic pigmentation of the neck (dirty neck) [4]. They are frequently observed in adult patients with atopic dermatitis after a long-standing steroid ointment and termed adult type-atopic dermatitis. The former is thought to be induced by the

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suggests that stress-induced GC augments DNFB contact sensitivity reaction in rat at a physiological concentration but not at large concentration used as immunosuppressant [24]. Refractory adult patients with atopic dermatitis occasionally showed generalized erythematous rash after long-standing use of topical steroid and reduction or stop using topical steroid improved the exacerbated skin reaction. GC might be attributable to induce this type of skin reaction in a similar manner to this mouse model. This possibility should be clarified in a future work and newly coming topical immunosuppressants such as cyclosporin or FK506 also should be examined for the augmentative effect on cutaneous inflammation.

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