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Prostaglandin E in Blistering Skin Diseases

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Abstract. Blister fluid from 3 patients with bullous pemphigoid and from one patient each with cicatricial pemphigoid, pemphigus vulgaris, burn blisters and pressure bullae due to intoxication showed considerable prostaglandin E activity as measured in radioimmunoassay. Prednisolone treatment, in doses of 80 to 25 mg/day seemed to reduce the prostaglandin synthesis.

Key words: Blister; Pemphigus; Prostaglandins; Steroids

Prostaglandins (PGs) are common denominators in various cutaneous inflammations. PGs have been recovered from such skin inflammations of toxic origin as burn blisters (1), sunburn (5), cantharidin blisters (4, 6) and primary irritant dermatitis (10), whereas generally no prostaglandin E₂ (PGE₂) activity was found in suction blister fluid from normal skin (4). Preliminary studies have demonstrated PGE₂ in blister fluid from patients with allergic contact dermatitis (4). Recently, prostaglandin F (PGF) was found in blister fluid from 2 of 3 patients with bullous pemphigoid (3). Since E and F prostaglandins have different and often opposing vascular and cellular effects on the tissues, a study of the PGE content of the blister fluid in blistering skin diseases seemed warranted.

MATERIAL AND METHODS

The series consisted of 7 patients. Three patients suffered from bullous pemphigoid and one patient each had benign mucous membrane (cicatricial) pemphigoid, pemphigus vulgaris, burn blisters and pressure bullae due to intoxication with alcohol and neuroleptic drugs. The diagnoses

Table 1. *Prostaglandin E activity in blister fluid as measured by RIA*

Case no.	Age, sex	Diagnosis	Treatment	PGE activity (pg/0.1 ml of fluid)
1	75 ♀	Bullous pemphigoid	Prednisolone 80 mg/day, 2 days	375
2	84 ♀	Bullous pemphigoid	—	3 000
3	74 ♀	Bullous pemphigoid	Prednisolone 70 mg/day, 14 days	75
4	71 ♂	Cicatricial pemphigoid	—	1 500
			Prednisolone 25 mg/day, 4 months Azathioprine 150 mg/day, 4 months	156
5	75 ♀	Pemphigus vulgaris	Prednisolone 10 mg/day, 6 months	1 200
6	48 ♂	Burn blister	—	2 500
7	41 ♂	Intoxication	Hydrocortisone inject., 100 mg	463

were verified by histopathology and immunohistopathology.

The fluid was collected by means of needle and syringe from blisters not older than 48 hours. The samples were stored at -70°C until radioimmunoassay for determination of PGE activity was performed.

Determination of PGE activity

Prostaglandins of the E group were determined by a sensitive radioimmunoassay technique described by Levine et al. (9) and Gutierrez-Cernosek et al. (7) and using a commercially available reagent kit (Clinical Assays, Inc., Cambridge, Mass., USA). Because of the low protein content of the blister fluid and its predilution (1:10), deproteinization of the specimen was not necessary. The PGE amount was determined as PGB equivalents after conversion by alkaline treatment. The antiserum cross-reacted to about 16% with PGA₁ and to 3% with PGA₂. There were no measurable cross-reactions with PGF₁ and PGF₂. The cross-reactions were measured at 50% displacement. The minimum detectable level of PGE activity was 15 pg.

RESULTS

The results are given in Table I, which also shows the steroid medication given to some of the patients prior to collection of the samples. PGE-like activity was recovered in the blister fluid from all the patients. Patients not yet treated with steroids showed higher PGE values than those treated. The highest value, 3000 pg/0.1 ml of fluid, occurred in a case of bullous pemphigoid not yet treated with steroids. The lowest value, 75 pg/0.1 ml of fluid, was encountered in another case with the same diagnosis treated with 70 mg of prednisolone daily for 2 weeks. In a case of cicatricial pemphigoid, the PGE content of the bullae dropped during treatment with prednisolone and azathioprine from 1500 to 156 pg/0.1 ml of fluid.

COMMENTS

The study demonstrates considerable PGE-like activity in the blister fluid from blistering dermatoses of apparently immunological origin as well as from burn blisters and pressure bullae due to intoxication. In a recent *in vitro* study, hydrocortisone and methylprednisolone were ineffective as inhibitors of PGE₂-synthesis in isolated human epidermal cells (2). In the present study, prednisolone treatment, 80 to 25 mg daily, seemed to have an inhibitory effect on the PG-synthesis, although measurable amounts of PGE were still present. The demonstration of PGs of the E group in the blister fluid may be more important than the demonstration of the F group, since the former is much more active than the latter when injected into human skin (8).

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Acute Tendovaginitis after Percutaneous Steroid Injection with Dermojet®

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Key words: Dermojet; Tendovaginitis; Corticosteroids

The mode of intralesional administration of steroids has been facilitated by devices utilizing high pressure injection of a crystal suspension of the steroid aided by a jet stream (1, 2). Dermojet® and Port-O-Jet® are two types of such devices on the market. Complications connected with this treatment are few (1). The present case report describes an uncommon side-effect following the percutaneous injection of triamcinolone acetonide (Kenacort-T, Squibb, 5 mg/ml) into a plaque of lichenified atopic dermatitis on the dorsal aspect of the hand.

CASE HISTORY

A 34-year-old woman, a member of an atopic family, had had atopic dermatitis since infancy and allergic rhinitis and asthma since early childhood. In recent years her main complaint has been a disseminated atopic dermatitis. She also has lichenified plaques on the backs of the hands and on the buttocks.