Reversible Overcurvature of the Nails after Treatment with Practolol

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Abstract. A patient developed psoriasis-like skin eruptions, ocular reactions and nail changes during therapy with the beta-blocking agent practolol. The nail changes were characterized by an extremely painful narrowing of the distal nail bed and a transverse overcurvature of all the nails. The nail changes as well as the cutaneous and ocular manifestations disappeared when practolol was discontinued.

Key words: Nails; Practolol; Beta-blocking drugs, complications

The etiology in cases of transverse overcurvature of the nails is usually unknown. It has been considered to be an anomaly of developmental origin, but some cases have been attributed to psoriasis and to wearing of ill-fitting shoes (1, 6). The present report describes a woman who developed this type of onychodystrophy during treatment with practolol.

CASE REPORT

A 48-year-old woman who had suffered from prurigo Besnier, asthma bronchiale and hay-fever up to the age of 20 was given practolol 300 mg daily, beginning July 1974, for hypertensive heart disease. The patient developed cutaneous, ungual and ocular reactions in December 1974, necessitating discontinuation of the practolol therapy in January 1975. At that time she had eye symptoms, with dryness and itching, but the ophthalmological examination disclosed no abnormality. Skin reactions with psoriasis-like plaques over the elbows and on the backs of the hands, together with fissuring, scaling and thickening of the palmar skin and the fingertips were also present. At discontinuation of the practolol treatment the nails were reminiscent of psoriasis, with subungual hyperkeratosis, onycholysis and a brownish colour proximally. There was, however, no pitting of the nails (Fig. 1a, b). The eye symptoms disappeared within a week after the drug was withdrawn. The cutaneous reactions disappeared within a month of discontinuation of practolol therapy and progressive nail changes appeared. During the following 3 months the patient developed an extremely painful narrowing of the nail bed, most pronounced distally, combined with an excessive transverse overcurvature (Fig. 2a, b). The pain was most pronounced at the lateral borders of the nails. After a further 5 weeks the proximal half of the finger nails appeared normal with a sharp border to the affected distal parts of the nails. Seven months after withdrawal of practolol therapy, most of the finger nail plates were normal and the distal overcurvature was much less pronounced. The distal narrowing of the nail bed had disappeared and pain had ceased (Fig. 3a, b). Some months later the finger nails appeared normal, though the toe nails still showed slight distal overcurvature.

DISCUSSION

Psoriasis-like skin reactions, sometimes together with ocular reactions, have been noted in conjunction with the use of practolol (2, 4, 5, 7) as well as with other beta-blocking drugs (3). Psoriasis-like changes have also been reported (4, 5, 7) in nails, but we have found no reports on overcurvature of nails noted with the use of these drugs.

![Fig. 1a, b. Psoriasiform nail changes after treatment with practolol for 6 months, at the time of discontinuing the drug.](image-url)
The patient described here had been treated with practolol for 5 months prior to the onset of a psoriasiform eruption, nail changes and subjective eye symptoms. The nails had a highly characteristic and unusual appearance; all the finger- and toe-nails developed an excessive transverse overcurvature combined with an extremely painful narrowing of the nail bed. After treatment with practolol was stopped the cutaneous reactions disappeared after about a month and the onychia regressed during 7 months. The time connection between the practolol therapy and the development of cutaneous and ocular symptoms simultaneous with the nail changes indicates a causal relationship between practolol and the nail dystrophy, further strengthened by the healing of the nails when the drug therapy was discontinued. The nail changes differed from those of psoriasis vulgaris. Similar nail dystrophies caused by drugs do not seem to have been reported before.

REFERENCES

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 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 radioimmunotechnique 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.

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

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age, sex</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>PGE activity (pg/0.1 ml of fluid)</th>
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<tbody>
<tr>
<td>1</td>
<td>75</td>
<td>Bullous pemphigoid</td>
<td>Prednisolone 80 mg/day, 2 days</td>
<td>375</td>
</tr>
<tr>
<td>2</td>
<td>84</td>
<td>Bullous pemphigoid</td>
<td>Prednisolone 70 mg/day, 14 days</td>
<td>75</td>
</tr>
<tr>
<td>3</td>
<td>74</td>
<td>Bullous pemphigoid</td>
<td>Prednisolone 25 mg/day, 4 months</td>
<td>156</td>
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<tr>
<td>4</td>
<td>71</td>
<td>Cicatricial pemphigoid</td>
<td>Prednisolone 10 mg/day, 6 months</td>
<td>1200</td>
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<tr>
<td>5</td>
<td>75</td>
<td>Pemphigus vulgaris</td>
<td>Azathioprine 150 mg/day, 4 months</td>
<td>2500</td>
</tr>
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<td>6</td>
<td>48</td>
<td>Burn blister</td>
<td>Hydrocortisone inject., 100 mg</td>
<td>463</td>
</tr>
<tr>
<td>7</td>
<td>41</td>
<td>Intoxication</td>
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