Letters to the Editor

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Fixed Drug Eruption to Paracetamol

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To the Editor

Fixed drug eruption (FDE) can be caused by many different drugs [1], but the list of chemical compounds that most frequently induce fixed eruption does not include paracetamol. In fact there are few reports in the literature of paracetamol causing an FDE [2–5].

A 45-year-old man presented to our Allergy Unit with a red-to-violet edematous infiltrative lesion surmounted by a large bulla on the glans penis; in addition he complained of pruritus (fig. 1). The patient gave a history of three episodes occurring in the same site at a time when he took a Zerinol tablet (paracetamol 300 mg and pheprazone 100 mg) for headache. In December 1987 the patient took the last Zerinol tablet because he was feverish and 12 h later he developed a round plaque of erythema and oedema with bulla on the glans penis. Three weeks later his practitioner, on account of headache, prescribed Tachipirina tablets (paracetamol 500 mg) and the patient experienced a flare of lesions in exactly the same spot. After this last eruption had subsided a patch test on normal skin with paracetamol 10% in ethanol appeared to give a negative result. A challenge oral test was performed and 200 mg of paracetamol were administered to the patient. During the following 24 h a renewal of the lesions in the same site as before was observed.

Sulphonamides, analgesics and tetracyclines are the most common causes of FDE according to Sehgal [6], tetracyclines, metamizole and oxyphenbutazone according to Pasricha [7]. Other authors [8–10] report that phenazones, barbiturates, several antibiotics, chemotherapeutic agents and psychotropic drugs are the most common medications causing fixed eruptions. Paracetamol appears to be an uncommon cause of FDE [2–5].

The medication used by our patient (Zerinol) is a combination of paracetamol and pheprazone and the latter drug has been initially suspected. In our country paracetamol is a commonly used drug and it is prescribed in aspirin- and phenazone-intolerant patients, when the oral provocation and tolerance tests are negative.

In our patient the causal relationship to paracetamol seems sure: patch testing gave a negative result, but an oral challenge test led to a renewal of the lesions in the same site. Some reports [5–11] assert that in fixed eruptions topical provocation can give positive results on sites of previous eruption; however previously involved skin may give positive or negative results.

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Fig. 1. Lesions present on the glans penis.

References


Nuchal Nevi flammei and Alopecia areata

Sir.

we read with interest the paper of Hatzis et al. [1] concerning nuchal nevi flammei (NNF) as a skin marker of prognosis in alopecia areata. The authors state that the prevalence (‘incidence’) of NNF in a group of alopecia areata totalis and alopecia areata universalis was significantly higher than in a control group. They found the prevalence in a group of alopecia areata totalis and universalis to be 58.2% as compared to 4.5% in controls. They postulate that NNF could be a valuable skin marker in the prediction of the course of alopecia areata.
We were surprised about the low prevalence of 4.5% nevi flammei in the control group, because in standard textbooks figures ranging up to 50% in normal populations are found. Therefore we looked for nevi flammei in outpatients (and almost everyone working in our department). As a result of this careful inspection we found that out of 120 controls 57.5% had an NNF.

We then screened a group of patients with alopecia areata (patchy type, totalis, universalis) whom we treated with topical immunotherapy. Interestingly, we found NNF in 40% of these patients. This is at variance with the findings of Hatzis et al. Our findings proved to be statistically significant (p < 0.05, chi-square test). We furthermore could not confirm a significant difference between the subgroups as reported. Our data are summarized in table 1.

In sum, we could neither find an increased prevalence of NNF in patients with alopecia areata, nor could we confirm the hypothesis of NNF as a prognostic marker. Possible explanations for this discrepancy

Table 1. NNF in patients with alopecia areata and controls: comparison of prevalence

<table>
<thead>
<tr>
<th>Cases</th>
<th>M/F Age</th>
<th>Prevalence examined</th>
<th>mean ± SEM of NNF, %*</th>
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</thead>
<tbody>
<tr>
<td>Alopecia areata</td>
<td>53 30/23 33.4 ± 1.7</td>
<td>39.62 (patchy type, (17–68) totalis, universalis)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>120 43/77 45.2 ± 1.8</td>
<td>57.50 (4–83)</td>
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R. Ranges in parentheses.
Difference statistically significant (chi-square test p < 0.05).