Nuchal Nevi flammei and Alopecia areata

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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

Cases examined	M/F	Age	Prevalence mean $\pm$ SEM of NNF, %*	
Alopecia areata (patchy type, totalis, universalis)	53	30/23	33.4 $\pm$ 1.7	39.62
Controls	120	43/77	45.2 $\pm$ 1.8	57.50
		(4-83)		

Ranges in parentheses.

Difference statistically significant (chi-square test, $p < 0.05$).

Letters to the Editor

could be: (1) Racial influences. We are not aware of comparative studies concerning racial differences in the expression of NNF. (2) Skin color. The expression of NNF is very variable. In our group, we noticed that 15% of patients and controls had a very faint NNF which could easily be overlooked in darker skin. (3) Hair color and density. In patients with dark-pigmented, curly and dense hair, the inspection of the nuchal skin can be very difficult. For the inspection of these patients, a good light source is especially important. To be quite sure, shaving or inducing emotional stress would be helpful, but is, of course, not feasible.

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Reference

1 Hatzis J, Kostakis P, Tosca A, et al: Nuchal nevus flammeus as a skin marker of prognosis in alopecia areata. *Dermatologica* 1988; 177:149–151.

Somatomedin, Somatostatin and Psoriasis

Sir.

I read with great interest the paper by Nickoloff et al. [1] dealing with somatomedin C receptors on keratinocytes. In their discussion the authors quote our previous work on somatostatin treatment of psoriasis [2] arguing that the role of somatomedin C in psoriatic keratinocyte proliferation could be sustained by ‘the therapeutic efficacy of somatostatin infusion in psoriasis’.

I would emphasize that, in our hands, somatostatin was efficient only in 30% of the patients. The improvement was very slight or nil in the others. Therefore the therapeutic efficacy of this drug was not clearly demonstrated by our study.

Even if a role for fibroblast-produced somatomedin C can be discussed in psoriasis, this hypothesis is not sustained by our rather disappointing results of somatostatin treatment in this disease.

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References

Nickoloff BJ. Misra P, Morhenn VB, et al: Further characterization of the keratinocyte somatomedin-C/insulin-like growth factor I (SM-C/IGF-I) receptor and the biological responsiveness of cultured keratinocytes to SM-C/IGF-I. *Dermatologica* 1988;177:265–273.

Guilhou JJ. Boulanger A. Barnéon G, et al: Somatostatin treatment of psoriasis. *Arch Dermatol Res* 1982;274:249–257.

Permanent Renal Impairment after Treatment with Ciclosporin

The effectiveness of ciclosporin (CsA) in severe plaque psoriasis is established [1. 2]. However. CsA may not be randomly used in clinical practice because of its side effects, which include hepatotoxicity. acute and chronic nephrotoxicity, hirsutism. hypertension and toxic effects on the central nervous system [3]. We here present a case of permanent renal impairment after treatment with CsA in a patient with pustular psoriasis and acrodermatitis continua (Hallopeau). The effectiveness of CsA treatment in this patient has been published previously [4].

Case Report. A 61-year-old healthy man, without a medical history, had therapy-resistant generalized pustular psoriasis and acrodermatitis continua (Hallopeau) since 1985. In 1988 oral therapy with CsA 8.0 mg/ kg/day (CsA trough level 600 ng/ml) was started in combination with

prednisone 10 mg/day. Within 3 weeks the skin was completely clear except for persistent severe nail lesions. Tapering off CsA or prednisone resulted in an exacerbation of acrodermatitis continua and pustular psoriasis. Therefore CsA and prednisone therapy was continued with unchanged dose. No concomitant medication was used.

Within a month serum creatinine started to rise to 157 $\mu\text{mol/l}$. In cooperation with the department of internal medicine, therapy was continued. After 3 months hypertension developed which could be controlled by atenolol 100 mg/day and furosemide 40 mg/day. Other side effects did not occur except for a slight increase in gammaGT, ALAT and LDH.

Within 12 months of CsA treatment, serum creatinine rose from 84 to 344 $\mu\text{mol/l}$. After CsA was lowered to 5.0 mg/kg/day (CsA trough level 290 ng/ml), gammaGT, ALAT and LDH normalized while serum creatinine decreased slowly in the following months. Hypertension however was persistent. Unfortunately the patient's pustular psoriasis and acrodermatitis continua could not be controlled with this dose. Even a combination therapy of CsA with etretinate and local therapy was not effective. Therefore CsA was discontinued and the patient was subsequently treated with methotrexate. He received CsA treatment during 18 months. When CsA treatment was discontinued, serum creatinine was 196 $\mu\text{mol/l}$. In the following 6 months blood pressure normalized and serum creatinine decreased to 138 $\mu\text{mol/l}$. Six months later serum creatinine was unchanged with a glomerular filtration rate of 53 ml/min and an extra renal plasma flow of 200 ml/min.

Discussion. Monotherapy CsA for generalized pustular psoriasis and acrodermatitis continua requires a high CsA dose [4, 5]. This can cause side effects as renal impairment within a short time. Long-term CsA treatment for pustular psoriasis or acrodermatitis continua is therefore not possible.

Serum creatinine improved until 6 months after stopping CsA therapy. Serum creatinine did not change in the following 6 months. Therefore it is likely the patient has irreversible renal impairment due to chronic nephrotoxicity in CsA treatment.

A. After CsA treatment permanent renal impairment may occur especially when CsA is administered for a long time [6, 7]. Morphological changes in kidneys associated with CsA treatment suggest the probability of chronic and irreversible renal injury [7, 8]. This case report clearly shows that CsA should be used with restraint and caution until ways are found to mitigate its nephrotoxicity.

CsA has been demonstrated to be nephrotoxic if the concentration is maintained at more than 300 ng/ml for extended periods [7]. Some correlation exists between a rise in serum creatinine and morphological