persons ($p = 0.033$). Eight of the 11 DLE patients sampled during the sunny season and 1 of 11 sampled during the winter were using antimalarial medication.

The forearm *cis*- and total UCA contents of the PLE patients did not differ significantly from those of control persons.

DISCUSSION

Cis-UCA has a suppressive effect on delayed hypersensitivity (1). Thus, the lowered epidermal *cis*-UCA content in UVR-protected buttock skin of DLE patients, demonstrated in our study, agrees with the proposal that DLE skin symptoms are caused by an augmented T-cell-mediated mechanism (2). Unexpectedly, contradictory results were found in PLE, which is also proposed to be mediated by a delayed hypersensitivity reaction (3).

We do not believe that sampling during different seasons skewed the results of non-protected buttock skin, since the *cis*- or total UCA values of control persons were unaffected by sun exposure of other skin sites (data not shown). In a recent Danish study, it was observed that the total UCA content in buttock skin was lower and the percentage of *cis*-UCA elevated during the summer compared with other seasons. Our differing result may be due to differing sampling period during the sunny season, i.e. before July in the present study vs. after July in the Danish study (7).

Unlike in UVR-protected buttock skin, there were no differences in *cis*-UCA contents between DLE patients and control persons in sun-exposed skin of the back of the hand. This may be due to the disease process itself. However, the possibility of a normalizing effect of antimalarial medication must also be considered, since the *cis*-UCA contents also tended to be higher in UVR-protected buttock skin of DLE patients on antimalarial medication than in non-medicated DLE patients.

ACKNOWLEDGEMENT

This study was supported by a grant from the Medical Research Fund of Tampere University Hospital, Tampere, Finland.

REFERENCES

1. Noonan FP, De Fabo EC. Immunosuppression by ultraviolet B radiation: initiation by urocanic acid. Immunol Today 1992; 13: 250–254.
2. Sontheimer RD. Photoimmunology of lupus erythematosus and dermatomyositis: a speculative review. Photochem Photobiol 1996; 63: 583–594.
3. Norris PG, Morris J, McGibbon DM, et al. Polymorphic light eruption: an immunopathological study of evolving lesions. Br J Dermatol 1989; 120: 173–183.
4. van Braag MCG, Boom BW, Vermeer BJ. Diagnosis and treatment of polymorphous light eruption. Int J Dermatol 1994; 33: 233–239.
5. Nyberg F, Hasan T, Puska P, Stephansson E, Häkkinen M, Ranki A, et al. Occurrence of polymorphous light eruption in lupus erythematosus. Br J Dermatol 1997; 136: 217–221.
6. Jansen CT, Lammintausta K, Pasanen P, Neuvonen K, Varjonen E, Kalimo K, et al. A non-invasive chamber sampling technique for HPLC analysis of human epidermal urocanic acid isomers. Acta Derm Venereol 1991; 71: 143–145.
7. de Fine Olivarius F, Wulf HC, Crosby J, Norval M. Seasonal variation in urocanic acid isomers in human skin. Photochem Photobiol 1997; 66: 119–123.

Accepted March 21, 1999.

Taina Hasan[1], Paavo Pasanen[2] and Christer T. Jansen[3]
[1]Department of Dermatology, University of Tampere and Tampere University Hospital, PO Box 2000, FIN-33521, Tampere, Finland (E-mail: thasan@tays.fi), [2]Department of Chemistry, University of Turku and [3]Department of Dermatology, Turku University Central Hospital, Turku, Finland.

Chloramphenicol Induced Acute Generalized Exanthematous Pustulosis Proved by Patch Test and Systemic Provocation

Sir,

Acute generalized exanthematous pustulosis (AGEP) is characterized by sudden onset of high fever, generalized scarlatiniform erythema covered by numerous non-follicular small superficial sterile pustules, blood leukocytosis with neutrophilia, and acute evolution (1–2). The main causative agent is drugs, but chloramphenicol has been rarely implicated (3).

Patch tests were performed in several cases of AGEP and results showing eczematous or pustular reaction were considered positive (2). Systemic provocation proved the cause in 1 case sensitive to isoniazid (4).

We here report a case of AGEP in which chloramphenicol was shown to be the cause by both patch test and oral provocation with a lowered dose.

CASE REPORT

A 36-year-old Korean woman had treated her rhinitis with acetaminophen and codeine for 2 days and with chloramphenicol for less than 1 day in June 1998. After ingestion of the former drugs 5 times and injection of the latter drug twice, pruritic, deeply erythematous and oedematous patches developed suddenly on almost her entire body, accompanied by mild fever (37.5°C). She also intermittently felt a burning sensation. The skin lesions became worse the next day, showing marked facial oedema and superimposed tiny superficial pustules. She had treated her rhinitis before, but had never had skin lesions. The laboratory findings were unremarkable, except for blood neutrophilia (8,500/μl) and glucosuria (more than 2g/dl), which normalized after 2 days. After administration of oral prednisolone, the skin lesions improved rapidly with disappearance of the facial oedema and fever the next day and of the pustules after a few days. Mild shallow desquamation followed. Patch tests were

performed 2 weeks after complete recovery with the ingested drugs, acetaminophen (10% in pet), codeine (0.5% in pet) and chloramphenicol (500 mg/ml, aq.). Eczematous reactions without any pustules developed at the sites patch-tested with codeine and chloramphenicol on days 2 and 4, respectively. Systemic provocation was performed to identify the causative drug and to confirm the results of patch testing after obtaining the patient's approval. Re-administration of acetaminophen and codeine caused no skin eruption, even at therapeutic doses, indicating that the patch test result for codeine was false. However, intravenous injection of 50 mg (1/20 of a therapeutic dose) of chloramphenicol produced deeply red, oedematous, pruritic patches within 5 h, similar to the original early skin lesion on the face, back, anterior chest, abdomen, neck and part of the extremities.

DISCUSSION

Skin tests, including patch testing, sometimes give false positive or false negative reactions, especially if the concentrations are not appropriate and the vehicles unknown. A control study and skin biopsy of the patch test may help to eliminate the false reactions (2). Re-administration of the suspected drug in 1/10 – 1/20 of the usual dose can help to confirm the diagnosis. The interval between the systemic re-administration and provocation of the skin lesions might also be of importance to protect the patients from a serious outcome.

REFERENCES

1. Roujeau J-C, Bioulac-Sage P, Bourseau C, et al. Acute generalized exanthematous pustulosis. Analysis of 63 cases. Arch Dermatol 1991; 127: 1333 – 1338.
2. Moreau A, Dompmartin A, Castel B, Remond B, Leroy DI. Drug-induced acute generalized exanthematous pustulosis with positive patch tests. Int J Dermatol 1995; 34: 263 – 266.
3. Macmillan AL. Generalized pustular drug rash. Dermatologica 1973; 146: 285 – 291.
4. Yamasaki R, Yamasaki M, Kawasaki Y, Nagasako R. Generalized pustular dermatosis caused by isoniazid. Br J Dermatol 1985; 112: 504 – 506.

Accepted April 8, 1999.

Ai-Young Lee and Sang-Hee Yoo
Department of Dermatology, Eulji Hospital College of Medicine, 280-1, Hagye-1-dong, Nowon-gu, Seoul 139-711, Korea. E-mail: lay5103@eulji.or.kr.