- MacDonald, W. C., Dobbins, W. O. & Rubin, C. E.: Studies of the familial nature of sprue using biopsy of the small intestine. N Engl J Med 272: 448, 1965.
- Stokes, P. L., Asquith, P. & Cooke, W. T.: Genetics of coeliac disease. *In Clinics in Gastroenterology*, vol. 2, pp. 547–556. W. B. Saunders, London, 1973.
- Marks, J., May, S. B. & Roberts, D. F.: Dermatitis herpetiformis occurring in monozygotic twins. Br J Dermatol 84: 417, 1971.
- Björnberg, A. & Hellgren, L.: Dermatitis herpetiformis, a laboratory and clinical investigation based on a numerical study of 53 patients and matched controls. Dermatologica 125: 205, 1962.
- Reunala, T., Salo, O. P., Tilikainen, A., Selroos, O. & Kuitunen, P.: Family studies in dermatitis herpetiformis. Ann Clin Res 8: 254, 1976.
- Marks, J., Birkett, D., Shuster, S. & Roberts, D. F.: Small intestinal mucosal abnormalities in relatives of patients with dermatitis herpetiformis. Gut 11: 493, 1970.
- Doherty, M. & Berry, R. E.: Gluten-induced mucosal changes in subjects without overt small bowel disease. Lancet i: 517, 1981.
- Seah, P. P., Fry, L., Stewart, J. S., Chapman, B. L., Hoffbrand, A. V. & Holborow, E. J.: Immunoglobulins in the skin in dermatitis herpetiformis and coeliac disease. Lancet i: 611, 1972.
- Fry, L., Haffenden, G. P., Wojnarowska, F., Thompson, B. R. & Seah, P. P.: IgA and C3 component of complement in the uninvolved skin in dermatitis herpetiformis after gluten withdrawal. Br J Dermatol 99: 31, 1978.
- Terasaki, P. I. & McClelland, J. D.: Microdroplet assay of human cytotoxins. Nature 204: 998, 1964.
- Unsworth, D. J., Leonard, J. N., McMinn, R. M. H., Swain, A. F., Holborow, E. J. & Fry, L.: Antigliadin antibodies and small intestinal mucosal damage in dermatitis herpetiformis. Br J Dermatol 105: 653, 1981.

HLA Studies in Chronic Dermatophytosis Caused by *Trichophyton rubrum*

Else Svejgaard, Bodil Jakobsen and Arne Svejgaard

Departments of Dermatology and Tissue Typing Laboratory of the Blood Grouping Department, State University Hospital (Rigshospital). Copenhagen, Denmark

Received September 29, 1982

Abstract. The HLA-ABC antigens were investigated in 34 and DR antigens in 28 patients with chronic dermatophytosis caused by *Trichophyton rubrum*. The distribu-

tion of the antigens did not differ from that of the controls. Abnormal immune response may be of importance in the susceptibility to chronic dermatophytosis, but HLA-controlled immune mechanisms seem unlikely.

Dermatophytosis due to *Trichophyton rubrum* is a common skin disorder and usually of minor importance. In some cases, however, the disease is resistant to treatment and spreads from the usual location in the lateral toe clefts to involve the foot soles, the palms and/or a varying number of finger and toe nails. The clinical response to the invasion of the dermatophyte in such cases is non-inflammatory, showing slight erythema but pronounced desquamation and keratinization, followed by fissuring. The nails display subungual keratosis, onycholysis, brittleness and discolouration.

This pathological picture is found mainly in males and has been observed to occur in several members of a family. A genetic disorder might therefore be a factor in the development of the symptoms. The lack of response to treatment might indicate a possible immunological disorder. Chronic dermatophytosis (CD) is occasionally associated with atopy (1, 2) and specific IgE to *T. rubrum* has been demonstrated in these patients (4). To investigate the possibility that CD may have a genetic and possibly an immunological background, we found it worthwhile to study the HLA antigens in this disorder, since the HLA system is involved in a variety of immune responses. To our knowledge, such studies have not been reported previously.

PATIENTS

The study involved 34 patients with CD of hands, feet and nails persisting for 3-45 years (mean 16.7 years). All had undergone long periods of medication with griseofulvin and 26 of them also with Ketoconazole, with varying clinical response but without mycological cure in any case.

METHODS

Tissue typing was performed by using the NIH technique for the HLA-ABC antigens and with the 7th workshop technique for the DR antigens, as described elsewhere (3). Lymphocytes from the patients were typed with antisera defining 11 HLA-A, 20 HLA-B, 5 HLA-C and the following HLA-DR antigens: DR 1, 2, 3, 4, 5, 7, w8 and 9 (we do not consider DRw6 sufficiently well defined). All patients were typed for ABC, while only 28 were typed for HLA-DR. The control groups included 426–3301

Table 1. Result of tissue typing (DR antigens) in 28 patients with chronic dermatophytosis

Antigen	Patients		Controls	
	Pos. n	%	Pos. n	%
DR1	3/28	10	136/704	19.3
DR2	6/28	21	197/704	28.3
DR3	10/28	36	182/704	25.9
DR4	8/28	20	244/704	34.7
DR5	3/28	10	64/704	9.1
DR7	5/28	18	147/704	20.9
DR8	3/28	10	6/704	8.7
DR9	0/28	0	1/58	1.7
DRI0	1/28	4	5/704	0.7

unrelated healthy individuals typed for HLA-ABC, and 58-704 unrelated individuals typed for HLA-DR. Statistical comparison were made with Fisher's exact test.

RESULTS AND COMMENTS

In patients with CD the distribution of HLA-ABC antigens did not differ significantly from that of the controls. The antigen showing the 'most significant' deviation was HLA-Cw5, which was found in only one of 34 patients (3%), while 17.1% of the controls had this antigen. The difference is significant (p=0.016), but this significance disappears when corrected for number of antigens investigated. The results of DR typing are given in Table I. No significant difference could be demonstrated between patients and controls.

In conclusion, this study did not reveal any association between chronic dermatophytosis and the HLA system. However, this negative finding clearly does not exclude the possibility that abnormal immune responses may be responsible for the susceptibility to CD, but it seems unlikely that HLA-controlled immune responses are involved.

REFERENCES

- Hanifin, J. M., Ray, L. F. & Lobitz, W. C.: Immunological reactivity in dermatophytosis. Br J Dermatol 90: 1, 1974.
- Jones, H. E., Reinhardt, J. H. & Rinaldi, M. G.: Immunological susceptibility to chronic dermatophytosis. Arch Dermatol 110: 213, 1974.
- 3. Jakobsen, B. K., Morling, N., Platz, P., Ryder, L. P., Thomsen, M. & Svejgaard. A.: HLA-DR phenotype and HLA-B, DR haptotype frequences in 704 unrelated Danes. Tissue Antigens 18: 270, 1981.
- 4. Svejgaard, E. & Löwenstein, H.: Unpublished data.

Results of Lymphography in Early Mycosis fungoides

Hugh Zachariae, Peter Bjerring, Eva Grunnet, K. Thestrup-Petersen and Dagmar Davidsen¹

Department of Dermatology, Marselishorg Hospital and ¹Department of Radiology, Aarhus Kommunehospital, University of Aarhus, DK-8000 Aarhus, Denmark

Received October 6, 1982

Abstract. Lymphography was performed in 28 patients with mycosis fungoides. In 22 of the patients, the investigation took place prior to 2 months after the diagnosis was established, and in 7 of these lymphography was made before the histological verification of mycosis fungoides was possible. Five patients with widespread. persistent and severe atopic dermatitis served as controls. Eighteen patients with mycosis fungoides (64%) had abnormal lymphograms, while all 5 controls had normal lymphograms. Abnormal findings were diagnosed in 12 of 22 patients at the earliest time possible during the course of their disease and even found in 5 of 7 patients who only had premycotic lesions at the time of investigation. These results may have some bearing on therapy, suggesting that systemic treatment could possibly be introduced at a far earlier disease stage than is the custom at present.

Key words: Lymphography; Mycosis fungoides

Although a number of papers have appeared lately concerning lymphography (LG) in mycosis fungoides (MF) (2, 4, 5, 7, 10), the total number of patients studied hitherto is only small, and supplementary information may therefore be of value. Early data (4, 5, 10) suggested that LG might be helpful in staging MF, when modifying treatment planning, and when determining a prognosis, whereas the latest reports (2, 7) deny this and declare that there is little correlation between abnormalities in LG and the extent and the clinical course of MF.

This report presents our experience with LG in 28 patients affected by MF, almost all in the early stages of the disease. LG was performed when there was strong suspicion of MF, when the diagnosis was established, or on the earliest possible occasion following diagnosis. Only 2 of the 28 patients had their LG's performed more than 12 months after diagnosis. As a control, LG was also performed in 5 patients admitted to our ward suf-