The problem of whether this band-like structure and dense granular bodies are incidental findings or of diagnostic or etiologic significance had not been settled. We assume that these structures may be of significance in these tumor cells; and this will be demonstrated in future investigative studies.

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Histocompatibility Antigens in Viral Warts

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Abstract. One hundred patients with viral warts and 108 apparently healthy matched controls were typed for HLA-A and B antigens. No significant difference in the frequencies of antigens was revealed, which suggested that a possible genetic trend in warts is at least not related to A and B loci of the histocompatibility system.

Key words: Viral warts; Histocompatibility antigens in; Genetic trend of; Human papilloma virus

Viral warts caused by different types of human papilloma virus (HPV) are a common disease. The association between viral infection and the major histocompatibility system is well documented (1). Some predisposition to HPV infection is a feature of persistent warts and in epidermodysplasia verruciformis, a genodermatosis, HPV has been demonstrated as a factor (4, 5). This prompted us to search for a possible particular pattern of the HLA antigens in viral warts.

MATERIAL AND METHOD

One hundred patients with viral warts, 18 females and 82 males, aged 7 to 33 years were investigated. Forty-seven were of European origin and the remaining 53 of Afro-Asian origin. The control group consisted of 108 matched apparently healthy subjects. Both patients and controls were typed for 14 HLA-A and 18 HLA-B antigens. The results were statistically evaluated utilizing the χ^2-test (2).

RESULTS

The results reveal that HLA-Bw35 is somewhat less common in patients of Afro-Asian origin than in controls ($p<0.05$). However, after correction with the number of antigens, no significant statistical differences were noted between patients and controls. Therefore, no difference in relative risks on the basis of HLA frequencies could be expected.

DISCUSSION

Evidence exists, pointing to a relationship between viral infection and histocompatibility antigens (3, 6). However, our data fail to demonstrake any such association. We may therefore speculate that the genetic trend of epidermodysplasia verruciformis —and in some instances of wart infection—is at least not related to A and B loci of this system.

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Immunoglobulin and Complement Deposits in the Skin and Circulating Immune Complexes in Scabies

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Abstract. Sixteen patients with papulovesicular, 6 with nodular and one with a Norwegian scabies were studied. Direct immunofluorescence (IF) examination revealed C_3 deposits in the skin lesions of 13 of the 18 patients. Among them were all 6 cases with nodular scabies. C_3 was found mostly in dermal vessel walls and 3 of the patients also showed IgM and 2 IgA deposits at the same site. No circulating immune complexes were found, with a solid-phase C_{1q} radioimmunoassay (RIA), but HSV- and RSV-RIA methods detected IgM antibodies of rheumatoid factor type in 5 of the 15 sera examined. These results suggest that local complement activation and perhaps also immune complex deposition may be important in the pathogenesis of the papular and nodular skin lesions of human scabies.

Key words: Scabies; Complement deposits; Immune complexes

The clinical symptoms of scabies only develop after a certain period of incubation. Mellanby (8) showed that after a primary scabietic inoculation it takes about one month before the itching and papulovesicles appear, but in a reinfection the symptoms develop within 24 hours. The development of immediate skin test reactivity and the occurrence of circulating IgE antibodies to scabies mite indicate that IgE-mediated hypersensitivity plays a role in the manifestation of human scabies (3, 10). Recent reports of immunoglobulin and complement deposits in the skin lesions suggest that mechanisms other than IgE-mediated are also involved (4, 5).

In a preliminary immunofluorescence (IF) study (11) we found deposits of the third component of complement (C_3) in dermal vessel walls of scabietic lesions. In this study we report IF findings in skin lesions and the occurrence of circulating immune complexes and IgM antibodies of rheumatoid factor (RF) type in papulovesicular, nodular and Norwegian scabies.

PATIENTS AND METHODS

Twenty-three patients, 18 males and 5 females, with scabies were examined. The diagnosis was confirmed by demonstrating a live mite in every patient. The mean age of the patients was 30 years and the mean duration of the clinical symptoms 3 months. Sixteen patients had a papulovesicular scabies with varying degrees of small vesicles and papules and 6 patients had a nodular form of scabies with many persistent nodules located mostly on genitals, groins and axillae. One patient had been treated for 2 months with peroral and topical steroids and he had developed crusted lesions with many mites i.e. the clinical picture of Norwegian scabies.

Skin biopsies were taken from 18 patients, from 16 of them before any treatment with a scabicide. The specimens were divided for routine histology (haematoxylin and eosin) and IF-examinations. In the latter, the specimens were examined for IgG, IgM, IgA and C_3 with commercial FIC-conjugated antisera (Behringwerke, Marburg, F.R.G.) using ordinary methods for direct IF. At least three tissue sections per conjugate were examined and the degree of fluorescence was graded as strong (+++), moderate (++), weak (+) and no (−) fluorescence.

Serum specimens from 15 patients were taken before any treatment and were stored at −50°C. For the presence of anti-immunoglobulins, the RFs were demonstrated by two methods. In these, herpes simplex virus (HSV) or respiratory syncytial virus (RSV) were used as antigens to which human IgG was bound. This antigen–antibody complex was fixed to a solid phase as reported earlier (6). The RF in the serum samples attached to these complexes was detected by radiolabelled antihuman IgM. In the third method, C_{1q} was attached to a solid phase and complexes bound from the serum were demonstrated by radiolabelled antihuman IgG (1).

Serum IgG, IgM, IgA and complement (C_3, C_4) levels were determined with a laser nephelometer method (Behring Institute, Marburg, F.R.G.). Normal limits for IgG were 8–18 g/l, for IgM 0.6–2.5 g/l, for IgA 0.9–4.5 g/l for C_3 0.45–1.1 g/l and for C_4 0.2–0.5 g/l.