Herpes Zoster

Sir,

We read with interest the article entitled "Histopathological findings, viral DNA distribution and lymphocytic immunophenotypes in vesicular and papular types of herpes zoster", recently published in the journal (1). We are pleased to see that the authors confirm several of our previous findings, despite the fact that they do not quote these works.

In fact, in various stages of herpes zoster, including the erythematous, papular, vesicular and crusted stages, we compared by immunohistochemistry (IHC) the distribution of Herpes Simplex Virus (HSV) I and II and Varicella Zoster Virus (VZV) specific antigens (2). It was reported that HSV could only be detected in epithelial structures, whereas VZV was often present in both epidermal and dermal cell populations, including type I dendrocytes. Such findings shed some light on the occurrence of vasculitis, granulomas and scars at the site of former acute herpes zoster (2).

Furthermore, we performed a study using IHC and *in situ* hybridization (ISH), detecting the VZV envelope glycoproteins gE and gB and their corresponding nucleic acid sequences in various stages of varicella and herpes zoster skin lesions (3). Comparing the VZV/host cell relationship in epidermal and dermal cells we demonstrated a cell type-dependent permissiveness to VZV infection. The immunophenotype of the inflammatory infiltrate during herpes zoster and varicella skin rash was also discussed in these papers (2, 3).

The importance of the pilosebaceous structure in the pathogenic pathway of herpes zoster skin lesions was underlined in these studies (2, 3) and by Muraki and coworkers (4). Our observations also led to the individualization of follicular herpes zoster, characterized by viral replication restricted to the follicular keratinocytes. In a conceptual point of view, follicular herpes zoster might be situated between zoster sine herpete and regular herpes zoster (5). Recently, this concept was beautifully illustrated in a case report (case 4) by Weinberg and coworkers (6).

Our findings also shed new light on the pathogenesis of post zosterian granulomatous reactions most probably related to a delayed type hypersensitivity reaction to persisting VZV envelope glycoproteins in the deep dermis while unrelated to the presence of viral DNA (7).

More recently, an altered gene expression pattern of VZV was shown during chronic hyperkeratotic skin infections in AIDS patients (8). The astounding reduced expression of gE and gB glycoproteins leads to a non-cytolytic virus/keratinocyte relationship resulting in chronic infection. A similar type of low-productive virus/host cell relationship was also detected in keratinocytes of chronic HSV and VZV lichenoid dermatosis (9).

Our results underline the importance of pushing investigations further on *in vivo* viral gene expression in order to disclose possible altered patterns that may be related to the pathomechanisms of atypical clinical presentations.

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Response to the Letter by Nikkels & Piérard

It is my clinical opinion that herpes zoster in patients with persistent papules without vesicular change are found fairly frequently in Korea. In our study to elucidate differences between the classical vesicular and papular types of herpes zoster, it was found that the appearance of clinical types of herpes zoster depends on the distribution of varicella zoster virus in the tissue. Nikkels & Piérard indicated that there were some rare atypical clinical presentations in herpes zoster, and in previous studies the varicella zoster virus could have involved a pilosebaceous unit. But no mention was made of a precise correlation between virological and clinical findings.

We believe that it is necessary to distinguish between major clinical types (vesicular or papular types) when studying the pathogenesis and clinical efficacy of therapeutic agents in herpes zoster.

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Focal Dermal Hypoplasia: A Case with Minor Clinical Manifestations

Sir,

Since Goltz (1) first coined the term "focal dermal hypoplasia" in the 1960s, over 200 cases have been described worldwide. The syndrome may involve the skin, the skeleton, the ocular system, or, less frequently, the renal, gastroesophageal, auditory and nervous systems. Here we report the case of a patient who presented with less definite clinical features than commonly described.

CASE REPORT

Our patient, a 5-year-old girl in good general condition, was a first child, and her mother had not had any miscarriages. Family history was negative for skin diseases. In the third month of life she had shown mild papulocrusty lesions on the lower limbs that worsened during the summer and became itchy. The lesions were regarded as atopic dermatitis. When first observed by us, the lesions were affecting the flexor surface of the right lower limb and partially of the left one, up to the root of both limbs. They involved a band of skin appearing as atrophic-hypochromic, partly reticulated areas interspersed with reddish papules and small squamocrusts (Fig. 1). The nail plate of the right hallux showed a longitudinal dystrophic fissure in the middle. The lower lateral incisor teeth were cone-shaped and hypoplastic. Clinical chemistry tests (in particular total IgE, ANA, antiENA antibodies, total urinary porphyrins and blood protoporphyrins) were normal. Histologic examination of a papule from an atrophic patch revealed mild irregular acanthosis with focal parakeratosis in the central portion of the lesion. The dermis was thinned and edematous. The fat was located abnormally high (Fig. 2).

X-rays of the upper limbs showed small calcareous deposits and radiopaque symmetrical, horizontal striae in correspondence with the distal metaphyses of humerus. A phototest revealed a higher than normal and long-lasting UV-B skin reactivity. Eye and neurological examinations were negative. The chromosome map showed a 46 XX karyotype with no structural changes of the chromosomes.

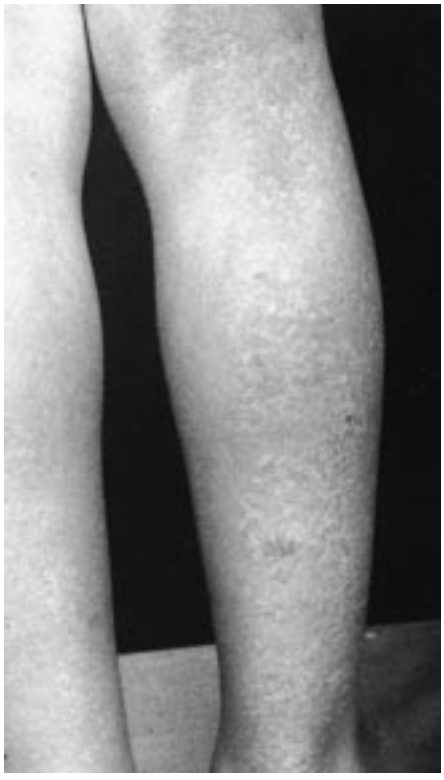

Fig. 1. Band of poikilodermatous skin with papules and squamocrusts lesions.

DISCUSSION

Our patient failed to show the most typical features of the Goltz syndrome, i.e. extroversion of the adipose tissue and eye involvement. Histology and bone X-rays were therefore conclusive for a correct diagnosis. Histological examination revealed a typical picture, but the radiological picture of our patient lacked the classic longitudinal distribution. Normally, the striated osteopathy appears as a net of condensed striae parallel to the longitudinal axis of the bone. The striae usually originate at metaphysis level in correspondence with the osteogenesis areas, where they look thicker and then gradually thin down as they go up along the diaphysis. They are usually bilateral and symmetrical, mainly involving the long bones and the sacral bone, while the vertebrae and the iliac bones are spared (2). Cases showing a less evident

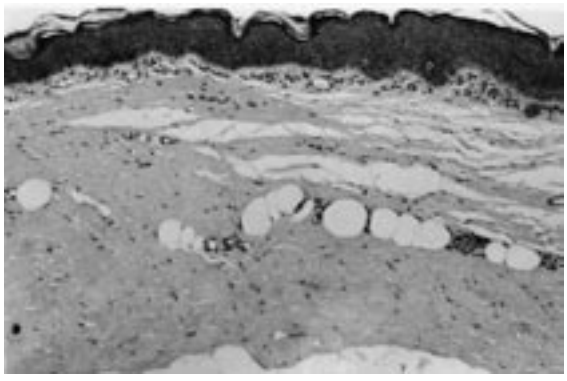

Fig. 2. Dermis thinned an edematous; fat located abnormally high (hematoxylin-eosin stain, 25×).