pigment migration, the differentiation of cells in the neural crest is completed and no nerve tissue is involved.

The cause of SPD appearance is so far unknown. Lerner and others (3, 6) suggested the existence of an enzymatic stimulant resulting from a neurogenic provocation, that regulates the pigment production or destruction. Such a simulant may explain the appearance of vitiligo with a dermatomal distribution or a zosteriform lentigenous nevus (9). If such a neurogenic enzymatic stimulant is really active—the SPD too might be affected by it. An even less plausible explanation is the occurrence of human chimaera (2) as a cause for the relatively common SPD. When presenting in the midline, the pigmentation disorder is characterized by a sharply demarcated borderline. This is due to a mediolateral pigment migration, namely from the back midline to both sides toward the abdominal midline in the linea alba region, not traversing the linea. A defect in pigment distribution will result in excess or lack of pigment, usually on one side only. Such defects have been described elsewhere (1, 4, 7, 8) as sporadic descriptions not as a defined pathological entity. Such a state of SPD as presented here, has been overlooked, possibly because the child affected was developing nicely and was not bothered by the disorder which appears to fade away within a few years.

We may assume that alertness to this disorder in the future will show that it is quite common; with a better knowledge of SPD we might discover the pattern of its natural course. With a large series of cases, it will be possible to establish, without any doubt, that SPD is not related to any neurological or other pathology.

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Purpura with a Linear Epidermo-dermal Deposition of IgA

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Received June 8, 1982

Abstract. A patient suffering from purpura is reported, having persistent linear deposition of IgA along the basement membrane zone in both lesional and healthy skin. The ordinary histopathology showed the picture of purpura pigmentosa with perivascular lymphocytes, polymorphonuclear cells and extravasated erythrocytes. No blisters have been observed.

Non-thrombocytopenic purpura is a heterogeneous entity with or without simultaneous systemic disease. Circulating IgA complexes in blood have been found in Henoch-Schönlein purpura (7), along with IgA deposits in cutaneous blood vessel walls and mesangium (1). In both polymorphonuclear vasculitis and lymphocytic perivasculitis there can be immunoglobulins (IgG, IgM, IgA) in the vessel walls (9). As far as we know, there are no reports of immunoglobulin deposits in the epidermal–dermal junction in purpura. A case report is presented with purpura and persistent linear IgA deposition on the basement membrane.

CASE REPORT

A 60-year-old woman developed large purpuric lesions predominantly on her legs and arms and to a lesser ex-

tent on the other parts of her body. There were no blisters or ulcers on her skin. The patient was diabetic and had hypertension and she used Aptin® (alprenolol) and Euglucon® (glibenclamid) for medication. After stopping the Euglucon® therapy the lesions disappeared almost completely in 6 weeks and after re-exposure to the same drug, purpura reappeared slowly to its full extent in 6 weeks. After renewed withdrawal of the drug, symptoms disappeared slowly.

The biopsies showed perivasculitis of the upper dermis. The infiltrate consisted predominantly of lymphocytes and to some extent of histiocytes and polymorphonuclear cells; in some areas basal cells of epidermis were degenerated. A direct immunofluorescence (with purified antisera to IgG, IgM, IgA, and C_3) revealed pure linear IgA along the basement membrane zone in both lesional and healthy skin in non-sunexposed areas. This finding was seen similarly after the disappearance of purpura for 6 months. The duodenal biopsy specimen did not show any immunoglobulins. Moreover, the iodine patch test and Trafuril® test made on her arms proved negative. To exclude the possibility of immune-complex vasculitis, histamine was injected into unaffected skin and 10 minutes later a skin biopsy was taken. There were not detectable immunoglobulins in dermal vessels. This method has previously been used to detect early events of immune complex vasculitis (3, 4, 10, 11). Furthermore there were no immune complexes detectable in her peripheral blood.

DISCUSSION

Clinically and histologically the present case resembles an extensive purpura pigmentosa chronica. This seems to be aggravated by an antidiabetic drug, glibenclamid (Euglucon®).

The finding of IgA in the present case along the basement membrane, but not in dermal vessels, is interesting. Besides dermatitis herpetiformis and linear IgA dermatitis there are findings of IgA depositions on the basement membrane in bullous pemphigoid-like diseases (5). In one study, antibody of the IgA class was highly specific for the patient's own skin and it was debated that IgA might play an indirect pathogenic role causing induction of the alternate complement activation pathway (6). Both granular IgA deposits along the basement membrane (2) and band-like linear IgA deposits of the adnexal structures (8) can be detected in normal patients, especially on sun-exposed areas. There were no detectable immune complexes containing IgA circulating in the blood of this patient. The role of persistent linear IgA deposits along the basement membrane zone in non-sun-exposed skin in this patient suffering from purpura remains open and merits further study.

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Nail Bed Immunofluorescence in Pemphigus vulgaris

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Received July 10, 1982

Abstract. A 65-year-old man developed simultaneously pemphigus vulgaris and onychomadesis of his thumb nails. Nail bed biopsy demonstrated supra-basilar acantholysis and intercellular epidermal deposition of IgG and C_3.