deposits of immunoglobulins in 3 out of the 5 DLE (cases 1, 2 and 5), whereas all biopsies from the other groups showed no deposits. These results have been included in another study (6).

DISCUSSION

The present study has shown that presence of CTS in vascular endothelium is a consistent finding in oral lesions of DLE, using a standardized examination procedure. CTS occur significantly more frequently in oral lesions of DLE than in LP or LEUK. The occurrence of CTS in DLE was not related to age of oral lesions or previous topical or general treatment, as discussed earlier (5). In DLE of the skin, CTS occurs more frequently in active lesions than in inactive ones (3). The present DLE lesions were all clinically active, i.e. showing central erythema with white spots and a border zone of irradiating white striae. It remains for further studies to show if the frequency of CTS in oral discoid lesions is related to clinical activity. The time required, 65 min on average for the non-DLE biopsies compared with 79 min for the DLE biopsies, can be attributed to difficulties in identifying the capillaries in the DLE due to the intense inflammatory infiltrate. The time needed for screening each capillary was fairly uniform in the various biopsies.

The diagnosis of oral lesions of DLE is usually based on clinical and histopathologic examination. In clinically and histologically atypical cases, direct IF staining for demonstration of deposits of immunoglobulins is often of value (6). However, the 3 DLE?, LP? were negative on IF-staining. The one DLE?, LP? patient showing CTS had increased DNA-antibody in serum and positive antinuclear factor indicating that this patient may possibly develop SLE later on. The two DLE biopsies in this study, which were negative on IF-staining (cases 3 and 4) both showed CTS.

To conclude, CTS occur in endothelial cells of active lesions of oral DLE, a CTS-positive biopsy can be identified by using a reasonable time and CTS seem to be absent from oral lesions of LP and LEUK, which are the most important differential diagnoses for oral DLE. Although clinical, histopathological and direct IF examination are the primary diagnostic procedures, the demonstration of CTS by using electron microscopy may be a helpful auxiliary diagnostic procedure in oral lesions of DLE.

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Drug-induced Bullous Dermatosis with Linear IgA Deposits along the Basement Membrane

T. Ø. Gabrielsen, F. Stærfelt and P. O. Thune

Department of Dermatology, Ullevål Hospital, Oslo, Norway

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Abstract. A 63-year-old woman presented with a drug-induced (diclophenac) bullous dermatosis. Direct immunofluorescence showed linear deposition of IgA along the basal membrane in both lesional and perilesional skin.

Key words: Drug-induced eruption; Linear IgA bullous dermatosis; Diclophenac

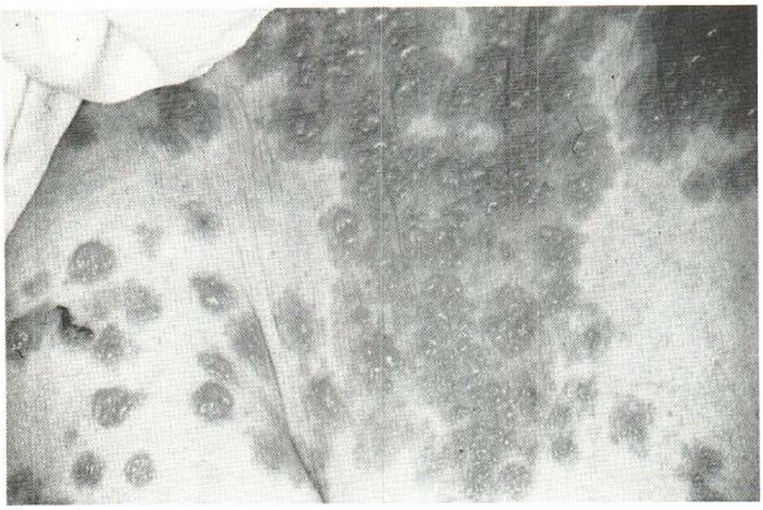

Fig. 1. The patient in the acute stage of the illness, showing numerous bullae on the left flank.

It is well known that immunological mechanisms play a major role in drug eruptions. Nevertheless, there are few reports in the literature, of immunopathological findings in drug-induced skin reactions. We present here a case of a drug-induced (diclophenac) bullous disorder with linear IgA deposits along the basal membrane.

CASE REPORT

A 63-year-old woman was admitted to hospital with an acute bullous dermatosis. She had a history of sero-positive rheumatoid arthritis for the previous 3 years and had previously been treated with naproxen and acetylsalicylic acid, though the former had been discontinued because of pruritus. At the time of admission she was participating in a multicentre trial of diclophenac (Voltaren®) and had received a daily dose of 50 mg b.i.d. for the last year. Diclophenac is a relatively new anti-inflammatory and analgesic agent presently under clinical trial in arthritic patients. About 9 days prior to her admission to hospital, and after taking a dose of 4 g acetylsalicylic acid during one day, she developed fever and symptoms of tinnitus and vertigo. The fever continued for a few days and was followed by a generalized rash 2 days before hospitalization. Blisters occurred first on the lips, later involving large areas of the body surface (Fig. 1). The areas involved were sharply demarcated by local erythema and oedema and contained several hemorrhagic bullae. Several of the latter had ruptured, giving rise to 'weeping' superficial erosions. She complained of a severe generalized itching and burning sensation of the skin. Although there was crusting of the lips, the mucous membranes were not involved. The temperature was elevated to 38.5°C.

The therapy with diclophenac was discontinued. New lesions continued to appear for 1–2 weeks after admission. The eruptions then subsided gradually and disappeared completely within 5 weeks. The patient did not receive any steroid medication. The patient's arthritic symptoms increased after the discontinuation of diclophenac. When her skin symptoms had completely disappeared, diclophenac was reintroduced and resulted in a recurrence of the rash, with severe itching and erythema within 4 hours of ingestion of the drug, thus indicating a clear relationship between her skin eruption and diclophenac. Her rheumatic condition was later adequately brought under control with a combination of indomethacin and acetylsalicyclic acid.

Laboratory investigations

Histopathological studies on lesional skin revealed formation of subepidermal bullae, with infiltration of polymorphonuclear leukocytes, mainly eosinophils, in the upper regions of the dermis and in the bullous liquid. There was some perivascular infiltration of lymphocytes in the upper dermis. The histopathological findings were compatible with bullous pemphigoid (BP), but the differentiation from erythema multiforme (EM) was difficult in this biopsy. Direct immunofluorescence studies (DIF) of lesional skin showed formation of subepidermal bullae with linear deposits of IgA along the basal membrane. Biopsy of clinically normal skin showed a similar arrangement with mainly IgA, but also IgM and IgG. The IgA depositions were of a monomere type. Indirect immunofluorescence studies did not reveal any circulating antibodies. Circulating immunocomplexes were present in the initial stages of the disease (PEG precipitate test).

Laboratory investigations showed an elevated IgA: 8.8 g/l, otherwise normal Ig and complement levels, positive RF/Waaler; HbG.: 10.0 g%; ESR: 70 mm/h (on admission) to 125 mm/h (after 2 weeks); WBC: $9\times10^3/mm^3$; platelets: $400\times10^3/mm^3$. Differential WBC and tests for liver function and renal function were normal. No growth of virus occurred in cultures (specimen from lip), and no serological evidence of a virus infection was found.

DISCUSSION

The introduction of immunofluorescence techniques in dermatology has contributed favourably to the exact diagnosis of bulloud disorders. It has also caused a certain amount of confusion in the old classification of these disorders. An example of this is the finding of linear IgA in dermatitis herpetiformis (DH), BP and chronic bullous dermatosis in childhood (1).

In our case the morphology of the eruption resembled BP, but the acute onset of the disease with high fever and its self-limiting nature was more compatible with a drug eruption or erythema multiforme. We have avoided the latter diagnosis because of the absence of typical lesions and the inconclusive histopathological findings. The immunopathological findings would strongly favour a diagnosis of DH, but the clinical course excludes this diagnosis. Linear IgA bullous dermatosis is considered by some authors to be a separate disease entity (1). It is, however, a clinically heterogeneous group of disorders, ranging from cases of typical DH to those of typical BP, including a spectrum of intermediate varieties, none of them resembling our case.

A definite relationship between the drug and the skin eruption was proven by re-introduction of the drug. Diclophenac has been reported to be well tolerated and to have a wide safety margin. In one report (2) only 2 out of 200 patients developed a rash which was considered to be related to the medication. There are few reports on the immunopathological findings in drug-induced dermatosis. Using the IF technique, van Joost (3) demonstrated circulating immunoglobulins with an affinity for basal cells. In one of his cases, IgG and C were detected by the DIF technique. Other authors have reported intercellular immunoglobulin deposition (4) and immunoglobulins bound to basal cells in lesional skin in toxic epidermal necrolysis (5). The exact mechanisms in these drug-induced disorders are not fully known at present. It is possible that more than one hypersensitivity mechanism is involved. The striking feature in our case is the finding of linear IgA along the basement membrane. This initially caused some confusion in the diagnosis and treatment of the illness, before the condition was subsequently shown to be a drug-induced bullous dermatosis with unusual immunopathological findings.

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Physicochemical Properties of "Spun Glass" Hair

Howard P. Baden, Robert J. Schoenfeld,[1] James D. Stroud,[1] and Rudolf Happle[2]

Department of Dermatology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts 02114, and [1]Wayne State University, Detroit, Michigan, USA; [2]Münster University, Münster, Federal Republic of Germany

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Abstract. The amount of protein solubilized with a Tris–urea–mercaptoethanol buffer was markedly reduced in a patient with "spun glass" hair but normal in 3 other patients with similar appearing hair. Polyacrylamide gel electrophoresis showed that it was mainly the matrix component that was solubilized. X-ray diffraction and stress strain tests on the "spun glass" hair proved normal, as was the amino acid analysis. The cause of the insolubility has not been established.

A large number of hereditary disorders of the hair have been described, but in only a few have chemical abnormalities been identified in the structural proteins (2, 10). Stroud & Mehregan (12) reported on a patient with "Spun-glass" hair that tended to stick out and they noted that the hairs were crimped and had a triangular or kidney shaped cross-section. The hair in this patient would not lie flat when combed.