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LICHEN NITIDUS: ELECTRON MICROSCOPIC AND IMMUNOFLUORESCENCE STUDIES

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Abstract. Skin biopsies from a patient suffering from lichen nitidus were studied by electron microscopy and immunofluorescence studies. Complete disintegration of the stratum basale in the central area of the lesion and signs of acantholysis in the border area were found. In the central part of the lesion the dermo-epidermal junction was severely damaged in most places. In the dermal infiltrate, macrophages and lymphocytes were found with a considerable representation of Sézary cells, not previously reported in lichen nitidus. The ultrastructural changes are identical with those found in lichen planus. No *in vivo* bound immunoglobulins, fibrinogen, or complement C_3 were found.

Key words: Lichen nitidus; Lichen planus; Ultrastructure; Immunofluorescence; Sézary cell

Lichen nitidus is clinically characterized by minute, pin-point to pin-head sized papules, which are asymptomatic, and flesh-coloured with a flat, shiny surface (13). In contrast to lichen planus they are non-pruritic and do not coalesce, but may be widespread, with a predilection for the penis, the arms and the abdomen.

The view that lichen nitidus represents a variant of lichen planus has been supported by the fact that early, tiny lichen planus papules may be clinically and histopathologically indistinguishable from lichen nitidus (6, 17), but minor histopathological differences and differences in the development of the lesions seem to favour a separation into two dermatoses (11, 12). Furthermore, immunofluorescence studies have shown some differences, no gammaglobulin deposits being found in lichen nitidus in contrast to lichen planus (16).

The ultrastructure in lichen nitidus has been reported to be indistinguishable from that of lichen planus in one patient so far studied (1). In order to obtain more information about the relationship between lichen nitidus and lichen planus we wish to present an electron microscopic and immunofluorescence study of lichen nitidus in relation to a series of lichen planus cases published previously (3).

MATERIAL AND METHODS

Biopsies were obtained from an 18-year-old man who, from the age of 17, had developed a widespread asymptomatic eruption of uniform, pin-point sized, flesh-coloured, shiny, flat papules on the volar aspects of the arms, on the penis, the lower trunk, and scattered over the face and the legs. The eruption was symmetrical, with some grouping but without coalescence except in a scratch mark on the arm (Koebner phenomenon). The patient had previously been healthy apart from an allergic rhinitis in the summer period due to pollen allergy.

Routine skin biopsy revealed histopathological changes typical of lichen nitidus (Fig. 1).

Punch-biopsies for electron microscopy were taken from papules and clinically normal skin on the abdomen and were fixed in 2.5% glutaraldehyde in 0.1 M cacodylate buffer, pH 7.2, for 9 days at 4°C. They were post-fixed in 1% osmium tetroxide in the same buffer for one hour at room temperature. The tissue blocks were treated according to the method of Simionescu & Simionescu (14) with low molecular weight galloylglucoses (tannic acid) in order to produce a distinct contrast, dehydrated in graded series of ethanol and embedded in Araldite. Semithin sections were stained with basic toluidine blue and used for orientation. Ultrathin sections were double-contrasted with zinc uranyl acetate and lead citrate and studied in a JEM 100 CX electron microscope at 80 kV.

Punch-biopsies for immunofluorescence investigations were taken from papules and unaffected skin on the abdomen. The biopsies were frozen instantaneously in liquid nitrogen and stored at −40°C until investigation. The immunofluorescence study was made using a method described previously (8). Cryostat-cut 6 μm thick sections were incubated with fluorescein-conjugated rabbit anti-human IgG (F/P ratio: 2.2; final dilution 1 : 64), IgA (F/P ratio: 2.2; final dilution 1 : 64), IgM (F/P ratio: 2.2; final dilution 1 : 40), anti-complement C_3 (F/P ratio: 1.9; final dilution 1 : 40) and anti-fibrinogen (F/P ratio: 2.2; final dilution 1 : 20). All conjugates were obtained from Behringwerke AG.

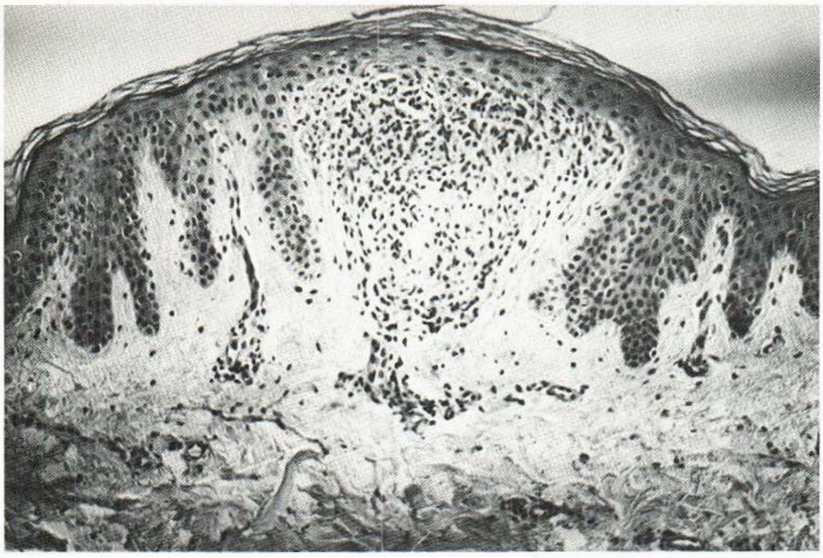

Fig. 1. Lichen nitidus papule. A well-circumscribed intense infiltrate below the epidermis formed by lymphocytes and histiocytes, but no giant-cells. Liquefaction degeneration of the basal-cell layer with elongated rete pegs at the margin of the infiltrate. Hem-Eos. ×215.

RESULTS

Electron microscopy

Central part of the lesion. The basal cell layer was severely damaged and had been infiltrated by macrophages from the dermis to such an extent that normal orientation of the basal cells was completely lost. Basal cells exhibited varying degrees of karyolysis and many of the cells had dissolved into

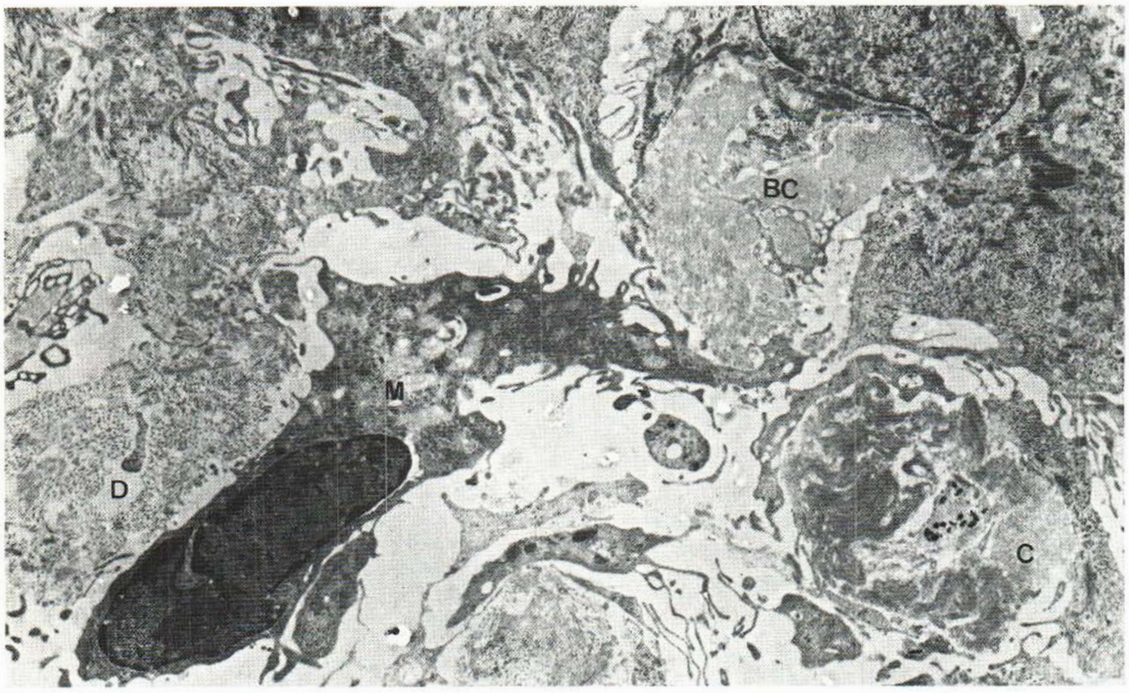

Fig. 2. A macrophage (*M*) is migrating from the dermis (*D*) to the epidermis, phagocytosing a necrotic basal cell (*BC*). Colloid body (*C*). ×5 000.

Fig. 3. Macrophages (*M*) in the dermal cellular infiltrate. Melanin-containing lysosomes (*arrow*). Pseudopodia (*arrowheads*). Dermis (*D*). ×5 500.

irregular cell debris surrounded by macrophages showing phagocytosis. Only a few preserved basal cells were noticed, but signs of degeneration such as swelling of the mitochondria and disorganization of tonofilaments in the cytoplasm were seen. Isolated structures resembling colloid bodies were seen.

In most areas the dermo-epidermal junction was no longer discernible, being infiltrated by macrophages. Locally, however, a junction between a degenerated basal cell and the basal lamina was still present. In such places the number of half-desmosomes was reduced, and only a few tonofilaments were inserting into them. The thickness of the basal lamina was irregular, and separation from the basal cells occurred. Duplication of the basal lamina was occasionally observed. Macrophages were migrating through breaks in the basal lamina from the dermis to the epidermis (Fig. 2).

The collagen fibrils in the dermis were scattered because of oedema and considerable infiltration of various types of cells. Most of the cells in the infiltrate were macrophages, with many vacuoles and lysosome-like structures containing melanin in their cytoplasm (Fig. 3). Long pseudopodia extended from the periphery of the cells, which sometimes lay close to each other. Many of the cells in the dermis showed morphological characteristics similar to those of Sézary cells (Fig. 4). The nuclei appeared lobulated and indented, and the chromatin granules showed an irregular distribution at the nuclear membrane, the pores of which were very distinct. No lysosomes or granules could be demonstrated in the cytoplasm, which contained many ribosomes. The cell surface had a few pseudopodia. In the dermal infiltrate epithelioid cells were also found, the cytoplasm of which contained large bundles of filaments surrounding dense mitochondria and lysosomes. Long thin microvilli projected from the cellular surface. Besides lymphocytes, monocytes and fibrocytes were present, whereas no multinucleated giant cells were observed. Only a few dermal vessels were found, none of which revealed abnormalities.

The border of the lesion. The basal layer of epidermal cells was preserved, their longitudinal axes being oriented almost parallel to the dermo-epidermal junction in contrast to the palisade arrangement of the basal cells in the control biopsy. The intercellular spaces were enlarged (Fig. 5). Long cytoplasmic processes extended into the intercellular spaces, and the desmosomes were rarified. The number of tonofilaments was reduced, the filaments further being irregularly distributed. Many did not terminate into the remaining desmosomes. The mitochondria were swollen, and no signs of cristae were present. As for the rest of the organelles in the basal cells, no abnormalities were demonstrated.

At the dermo-epidermal junction a few tonofilaments were inserting into the half-desmosomes, whose number was normal compared with that of the control biopsy. The ultrastructure of the half-desmosomes appeared normal, and the basal lamina was intact.

At some distance from the basal lamina a small infiltrate was found in the dermis, consisting of cells of the same type as already described in the central part of the lesion.

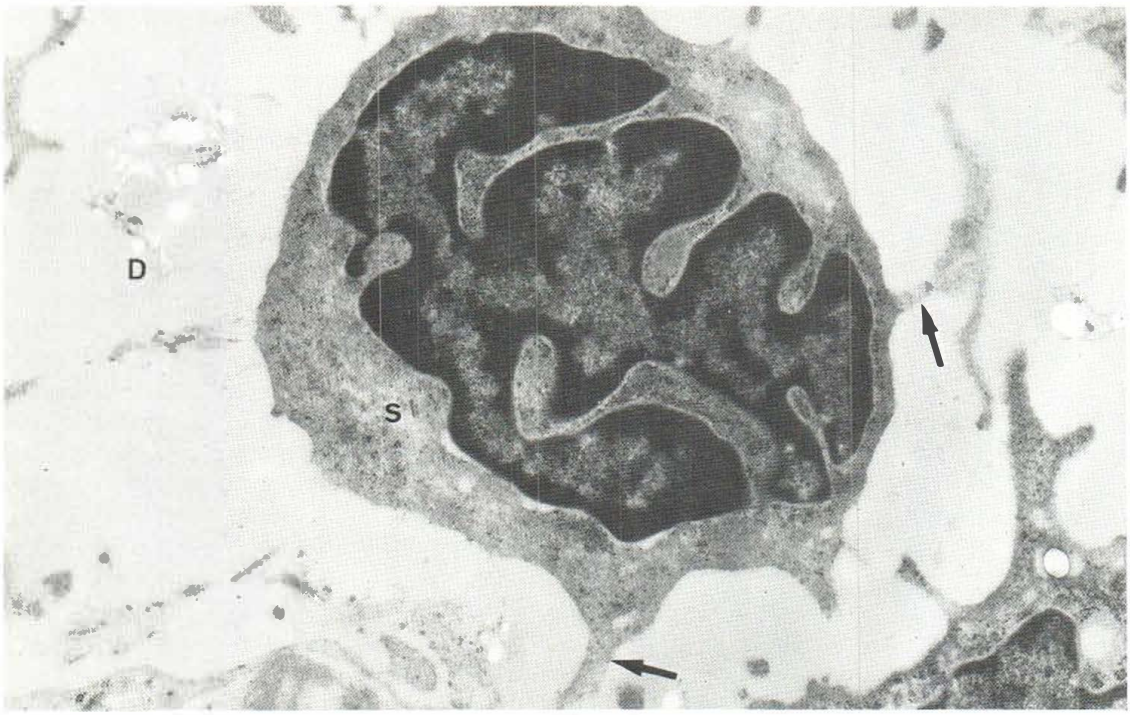

Fig. 4. Sézary cell (*S*). The nucleus is irregular and indented. Pseudopodia (*arrows*). Dermis (*D*). ×20 000.

Immunofluorescence

No *in vivo* bound immunoglobulins, fibrinogen or complement C_3 were found in the lesions or unaffected skin.

DISCUSSION

The present study showed ultrastructural changes of varying degrees reflected by complete disintegration of the stratum basale in the central area of the lesion and signs of acantholysis in the border areas. These findings are in agreement with findings in some of the previous ultrastructural studies on lichen planus, including the occasional appearance of colloid bodies (2, 3, 9). The generally held view of a primary process, affecting the basal cells in lichen planus, might therefore also be true of lichen nitidus because of the signs of degeneration presented by the basal epidermocytes.

In the central part of the lesion the dermo-epidermal junction was severely damaged in most places. The basal lamina had generally disappeared, but when present showed gaps and separation from the basal cell. The reason for this change might be the disintegration of the basal cells, rendering them

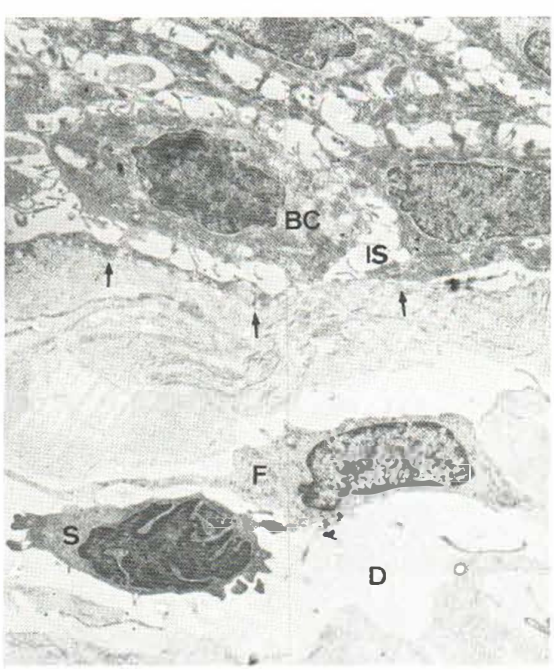

Fig. 5. Border of the lesion. Enlarged intercellular spaces (*IS*) between the basal cells (*BC*). Lamina basalis (*arrows*). Sézary cell (*S*). Fibrocyte (*F*). Dermis (*D*). ×4 000.

unable to produce a normal basal lamina (10). In the periphery of the lesion, however, the basal cells showed only minor changes of degeneration and thus still showed a capacity to maintain a normal basal lamina. Similar changes have been observed in lichen planus (3).

In the dermal infiltrate a variety of cells was present, most of which were macrophages migrating from the dermis into the epidermis, probably a secondary reaction provoked by the pathological basal cells (15). Some of the macrophages contained melanin, which might originate from melanocytes dropping through the gaps in the basal lamina. A similar finding is reported in lichen planus as a possible mechanism underlying pigment incontinence (3).

Many of the cells in the dermal infiltrate were indistinguishable from Sézary cells due to their cerebriform nucleus. They have not previously been reported in lichen nitidus, whereas their presence in lichen planus has been found in some studies (4, 7). Thus, Sézary cells are not specific for mycosis fungoides or the Sézary syndrome, but seem to represent stimulated T-lymphocytes (5, 18).

In agreement with a previous study of lichen nitidus (16) no immunoglobulins were found in our patient, in contrast to lichen planus, where globular deposits of immunoglobulins were found in papillary dermis (16). However, the distinguishing value of this finding has not been established.

In conclusion, the present study has shown that the ultrastructural changes in lichen nitidus and lichen planus are identical. This may indicate a close pathogenic relationship between the two diseases, although immunofluorescence studies differ.

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