

LICHEN SIMPLEX CHRONICUS VIDAL: COMPARATIVE SUBMICROSCOPIC ASPECTS OF ACANTHOTIC DISORDERS

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Abstract. Electron microscopic analyses of lichen simplex chronicus Vidal (LSC) are reported. The submicroscopic organization is described. The frequent occurrence of collagen fibres directly juxtaposed to and contiguous with the lamina basalis seems to be a distinguishing feature of the LSC. Discontinuities in the lamina basalis are rarely indicated. A ubiquitous fragmentation and a certain paucity of tonofilamentous structure are present in cells preceding parakeratosis. There is an indubitable paucity of tonofilament-keratohyalin association. Mitochondria, endoplasmic reticulum and ribosomes are abundant. Odland bodies of type II are completely dominant. Parakeratosis and observed submicroscopically deficient or incomplete orthokeratosis are related to the numbers of defective Odland bodies. The keratinization of some acanthotic disorders is discussed.

Key words: Lichen simplex chronicus Vidal; Neurodermatitis; Keratinization; Odland bodies

Lichen simplex chronicus (LSC) was originally described by Vidal & Leloir in 1883 (9). This disorder begins with intermittent itching but without visible changes in the skin. Through scratching and rubbing the characteristic skin lesions develop later. The exact position occupied by this condition has long been a vexed question.

The histological epidermal changes of LSC can be summarized as generalized hyperkeratosis with small areas of parakeratosis overlying a regular acanthotic elongation of rete ridges and with occasional presence of spongiosis. A scanty and diffuse dermal infiltrate dominated by mononuclear cells is present. The irregularly acanthotic epidermis dominates the micromorphology.

Acanthosis is a most prominent light microscopical feature of certain skin disorders accompanied by different keratinization. When represented by a fully developed lesion, the acanthotic

disease psoriasis is characterized by the absence of keratohyalin (3). The horny layer is completely parakeratotic. On the other hand, the acanthotic tumour of seborrhoeic keratosis always has an overlying orthohyperkeratosis, irrespective of the presence or absence of keratohyalin (5). The different aberrant submicroscopical changes in these two diseases, preceding the disturbed formation of the stratum corneum, have been analysed (5).

Another dermatosis characterized by acanthosis and focal parakeratosis, seborrhoeic dermatitis, has recently been investigated (7). In this paper it is concluded that the ultrastructure of the epidermis is not comparable to that in psoriasis. It seems conspicuous that parakeratosis and orthokeratosis are synthesized independently of the observed but diminished presence of keratohyalin.

To investigate the submicroscopical organization of epidermal cells preceding the formation of ortho- and parakeratosis of LSC, the present analyses were performed to further explore the keratogenous function of keratohyalin.

MATERIAL AND METHODS

Skin punch biopsies were obtained from 7 male patients aged 35-58 years, free from other skin disease and with macromorphologically typical LSC on upper arms and thighs. The diagnosis was histopathologically confirmed in each case. The duration of the skin lesions was 6-18 months. The specimens were fixed by immersion in 4% glutaraldehyde buffered with a phosphate solution at pH 7 for 6 hours at 4°C. Post-fixation was performed in 2% osmium tetroxide buffered with the phosphate solution for 2 hours at 18°C and the specimens were rinsed in the phosphate buffer solution.

Dehydration was carried out in increasing concentrations of acetone. The specimens were embedded in Ves-

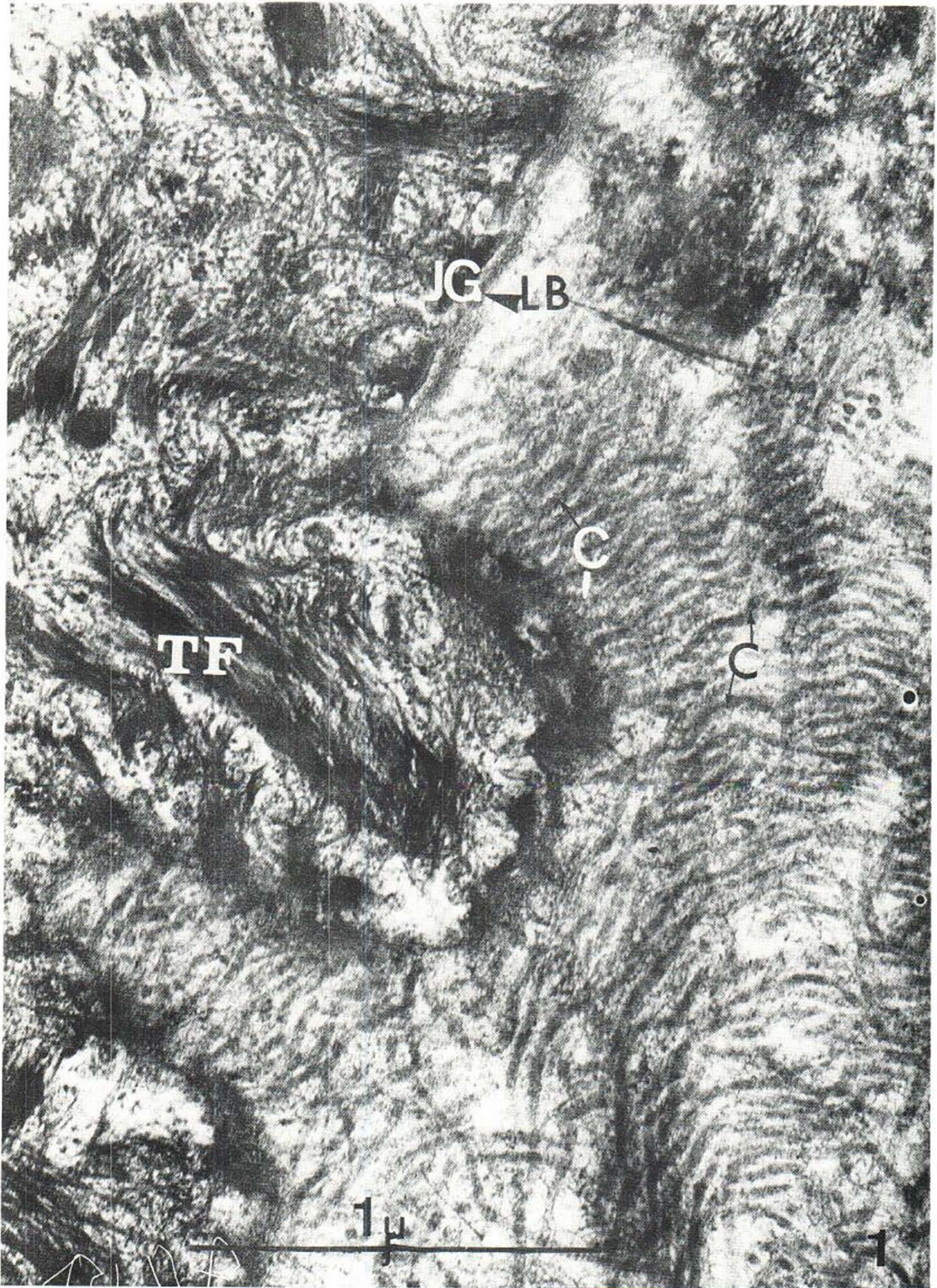


Fig. 1. Part of the dermis and a basal cell especially presenting the dermo-epidermal junction with collagen fibres often in perpendicular association with the lamina basalis. $\times 62\,000$.

total W. The ultrathin sections cut with the LKB-ultrathome III were double-stained with uranyl acetate and lead citrate.

RESULTS

In the dermo-epidermal junctional area there is an abundance of collagen fibres showing the typical periodicity. The collagen fibres seem to be localized in the utmost juxtaposition to the lamina basalis (Fig. 1). The collagen fibres appear to be partly organized in bundle-like formations and indicatively parallel to the skin surface. The supraposed fibres seem to be more tortuous and randomly dispersed. The outermost localized collagen fibres are indubitably perpendicularly orientated towards the lamina basalis. Furthermore the collagen fibres seem to be contiguous with the lamina basalis, and it is impossible to separate the two structures at high resolution. The collagen fibres seem to have the same electron scattering properties as the lamina basalis, especially as seen between the half-desmosomes. Only exceptionally does a collagen-free zone beneath the lamina basalis occur.

The epidermis of LSC is submicroscopically delineated by a lamina basalis (Fig. 1) which rarely shows indicated discontinuities. The half-desmosomes and their association with tonofilament bundles appear normal. The intermembranaceous space delineated by the plasma membrane and the outface of the lamina basalis is not enlarged. No small vesicles are found attached to or in juxtaposition to the plasma membrane of the basal cells which would be indicative of micropinocytotic activity at the interface between epidermis and dermis.

In the basal cells the tonofilament density appears normal and the filaments are usually organized in loosely aggregated bundles which course in different directions and apparently attach to the cytoplasmic aspect of the half-desmosomes (Fig. 1). No differences were found between LSC with ortho- or parakeratosis. In the cytoplasm of the

upper spinous cells, ubiquitous fragmentation and paucity of tonofilamentous structure are found in cells preceding parakeratosis (Fig. 2). The association of tonofilament bundles with the desmosomes is unaffected, however. In spinous cells with supraposed orthokeratosis, fragmentation is scarcely visible but the density appears slightly reduced. Tonofilaments associated with keratohyalin are rarely observed and are found only prior to orthokeratosis. Among cell organelles the mitochondria appear in normal numbers in the basal cells but in inevitably increased numbers in the supraposed cells. In the cytoplasm of spinous and supraposed cells the abundant mitochondria are mostly round or ovoid, presumably dependent on the orientation of the organelles within the section, thus causing a great range in diameter and also irregular shape. The internal structure of the mitochondria is very varied, presenting preserved cristae, cristolysis, or a more or less pronounced amorphism (Fig. 3).

The endoplasmic reticulum, mainly of the rough or granular type, is well developed. The free ribosomes are abundant and infrequently in polysomal configuration (Fig. 3). Odland bodies or keratinosomes are abundant, and the type II (8) without internal lamellar substructure is prominently observed (Fig. 4). Odland bodies of type II in the cytoplasm of spinous cells with subsequent parakeratosis, however, have most frequently a central cave or cleft of poor electron scattering properties. Only indicatively association of the Odland bodies with tonofilament bundles occurs rarely and then always with Odland bodies having a homogeneous internal structure. Odland bodies of type II with internal, even dense, electron scattering properties seem to be associated with orthokeratosis.

In the upper part of the spinous layer, cells with large intracytoplasmic vacuole-like structures occurs occasionally.

Throughout the epidermis the intercellular spaces appear normal. The granular layer is poorly developed and consists of only a single cell layer, with cells containing little or no keratohyalin. Keratohyalin-containing cells are succeeded by completely keratinized corneal cells of type A and B. Keratohyalin-deficient cells precede parakeratosis. In many places the differentiation of A-cells seems to be altered or incomplete with regard to the formation of a uniform keratin pattern.

Abbreviations used:

A=A cell, B=B cell, C=collagen, D=desmosomes, ER=endoplasmic reticulum, G=Golgi apparatus, IG=half-desmosomes, LB=lamina basalis, M=mitochondria, N=nucleus, O=Odland bodies, R=ribosomes, TF=tonofilaments, Z=defective keratinized zones.

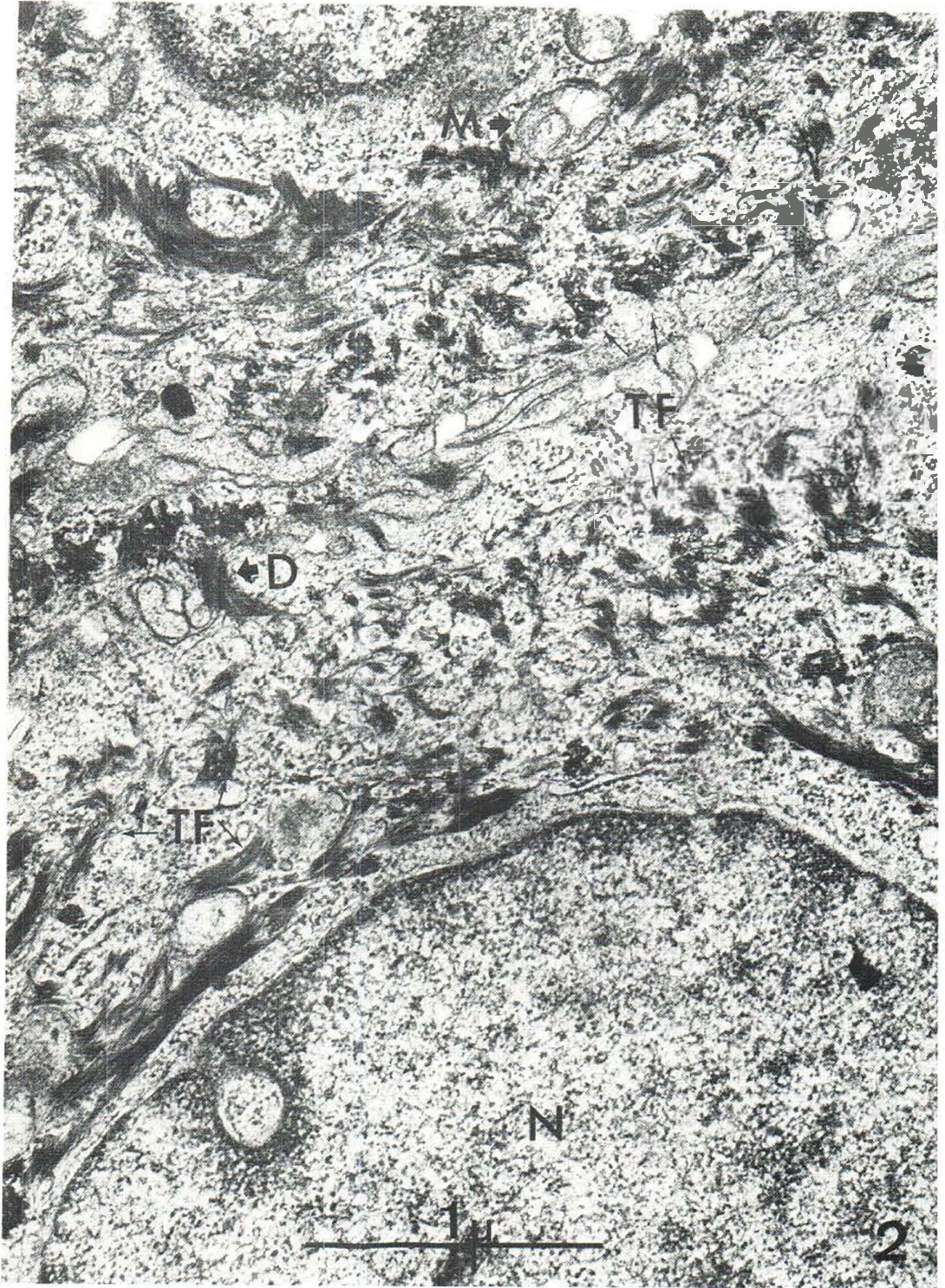


Fig. 2. Micrograph of upper spinous cells depicting fragmentation of the tonofilament bundles. $\times 45\,000$.

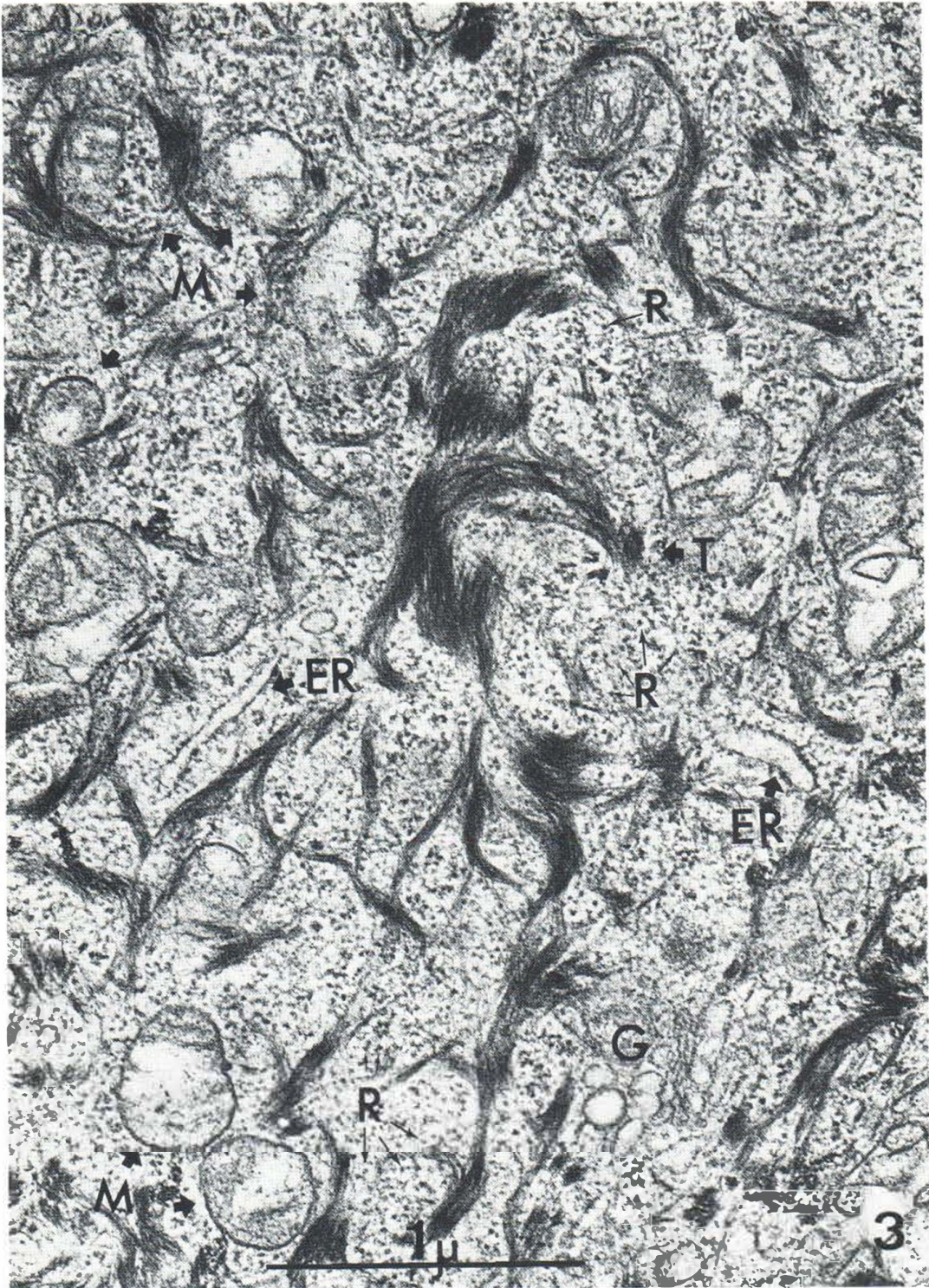


Fig. 3. Detail of cytoplasm of an epidermal cell from a LSC-lesion showing mitochondria of varying internal structure, numerous ribosomes and endoplasmic reticulum. $\times 58\,000$.

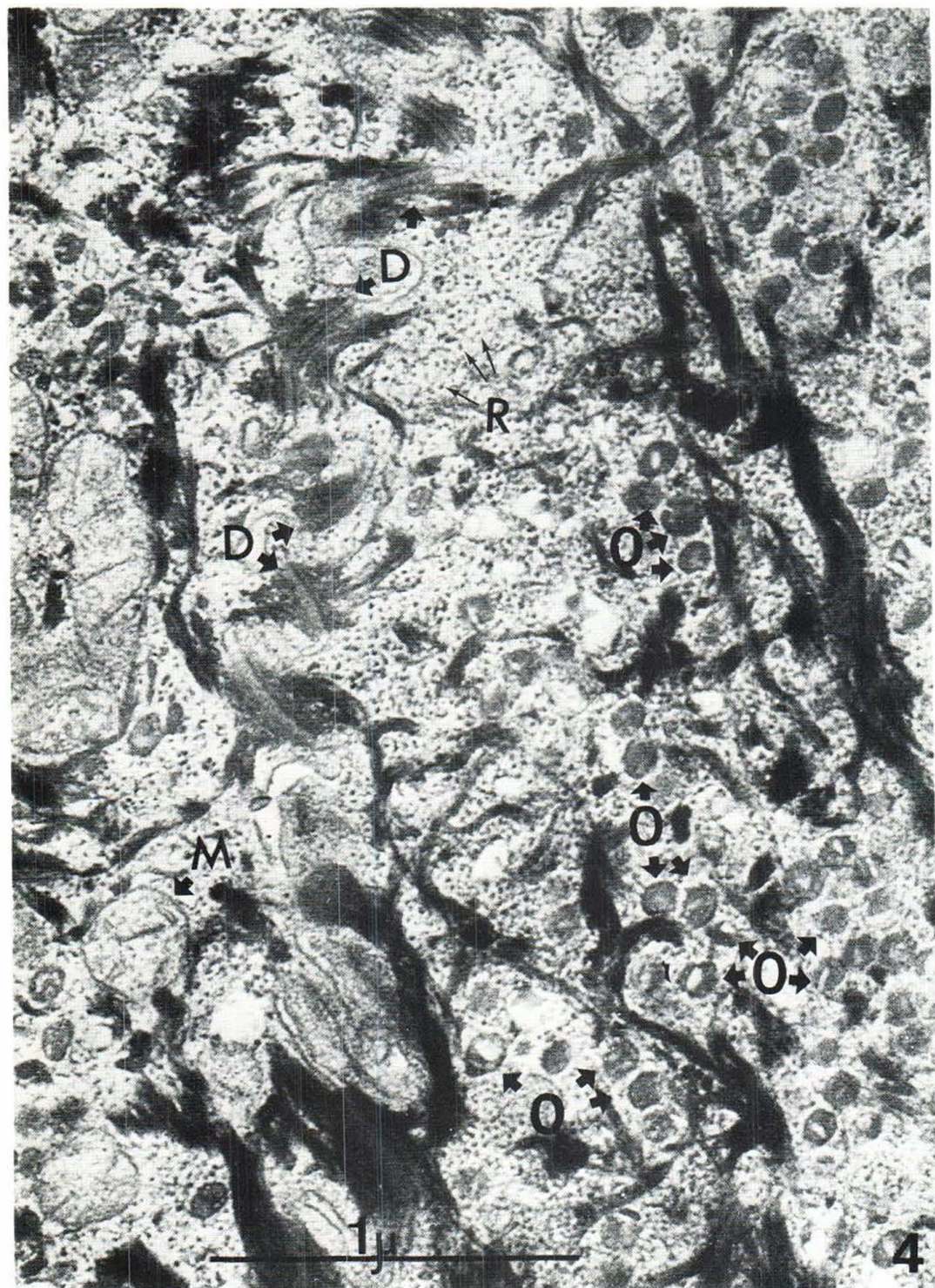


Fig. 4. Section of spinous cells with preserved desmosomes and numerous Odland bodies. $\times 56\,000$.

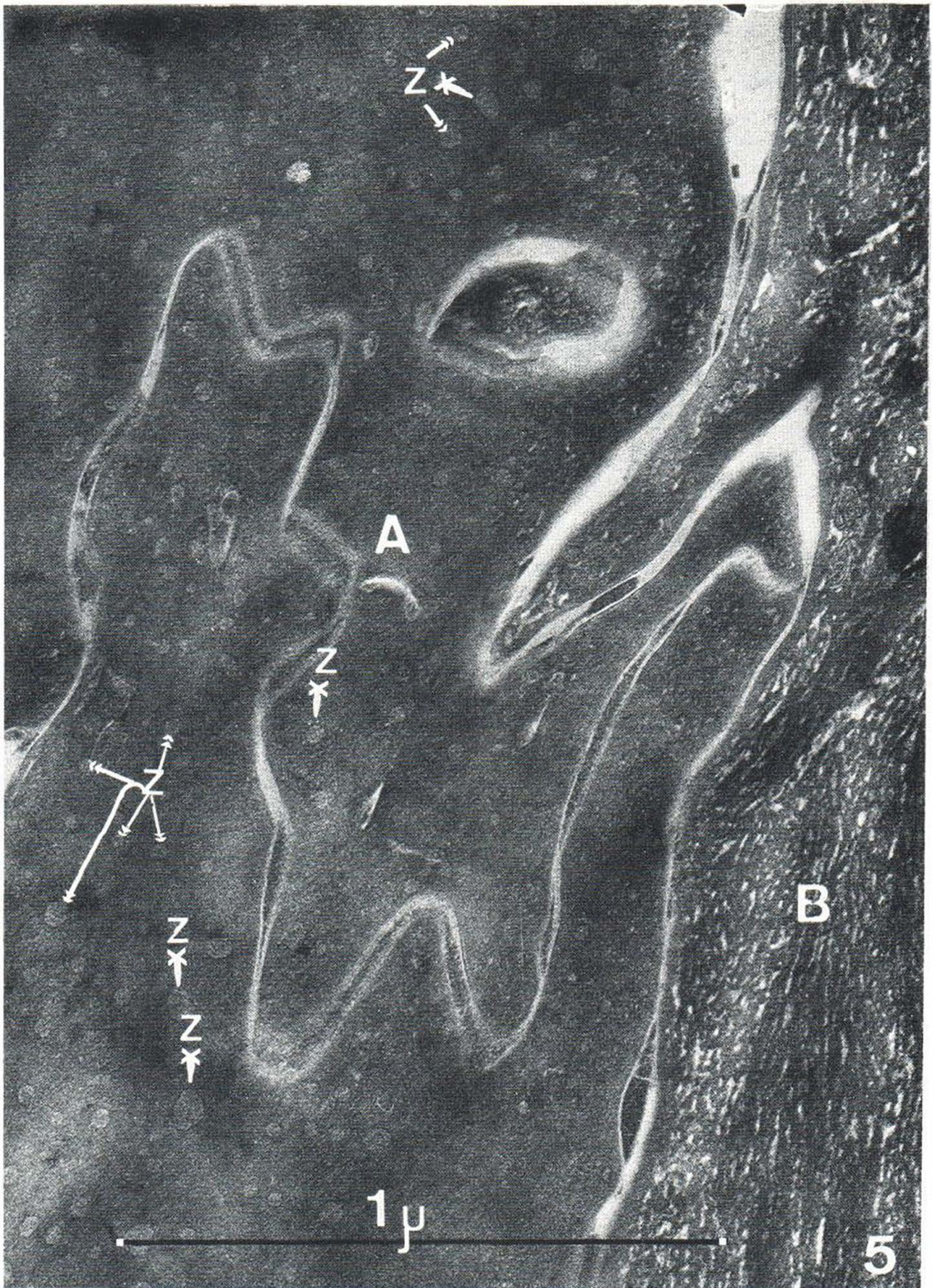


Fig. 5. Detail of the corneal layer representing light microscopical orthokeratosis showing part of a B cell and

parts of two A cells. The A cells display a special form of defective keratinization (Z). $\times 94\ 000$.

Haphazardly distributed in the cytoplasm of the A-cells are numerous round or ovoid zones containing an amorphous or fine granular material with less electron scattering properties than the surrounding keratinized cytoplasm as seen in the sectioned material. A limiting membrane is indicatively observed (Fig. 5).

DISCUSSION

Beneath the lamina basalis in the LSC-lesions it is sometimes possible to identify anchoring fibrils dispersed in a less dense area. The submicroscopic organization is conspicuously characterized by the juxtaposition of collagen fibres to the lamina basalis. The collagen fibres are often evincively observed in direct contact with the lamina basalis but without any visible discontinuity in the present material. This association might be considered as amphibological but seems to represent an ultrastructural phenomenon appurtenant to LSC. This conspicuous organization might indicate a participation of the collagen fibres in the formation of the collagenous component of the lamina basalis. It is impossible, however, to elaborate on the functional significance of this organization as observed in the electron micrographs.

Discontinuities of the lamina basalis of LSC lesions occur, and also comparatively often in seborrhoeic dermatitis, both acanthotic disorders of epidermo-dermitis, e.g. inflammatory cutaneous reaction. In uncomplicated psoriatic lesions, discontinuities are not observable but occur in eczematous or irritated psoriasis, i.e. psoriasis with dermatitis. Discontinuities of the lamina basalis are also absent in the acanthotic tumour basaloid cell papilloma.

No significant properties can be ascribed to the absence of multilayering of the lamina basalis in LSC, compared with the reduplication of lamina basalis ambiguously occurring in psoriasis, seborrhoeic dermatitis and basaloid cell papilloma and several other dermatoses (3, 5, 7).

The half-desmosomes appear normally organized as in psoriasis, basaloid cell papilloma and, presumably, seborrhoeic dermatitis. The intermembranaceous space appears, as in psoriasis, not dilatated and no voluminous invaginations are found as in the acanthotic tumour basaloid cell papilloma. As far as can be seen from the micrographs from lesions of seborrhoeic dermatitis pub-

lished by Metz & Metz (7) no dilatation of the intermembranaceous space seems to be present in spite of the demonstrable presence of spongiosis. In contrast to the eminent presence of micropinocytotic vesicles or psoriatic epidermal basal cells found as invaginations of the basal plasma membrane peripherally limiting the intermembranaceous space, the occurrence of micropinocytotic vesicles is remarkably exiguous in the LSC lesions. The basaloid cell papilloma and seborrhoeic dermatitis also seem to be characterized by a paucity of micropinocytotic vesicles. The rate of epidermal cell proliferation as examined by autoradiographic methods shows a corrected labelling index of the LSC similar to that of psoriasis (6). However, it was found that the rate of travel of labelled LSC cells was not so rapid as in psoriasis. There seems to be incontrovertible submicroscopical support for these findings. The abundant ribosomes in the psoriatic cells (2) are found to be slightly surpassed by the number of ribosomes in LSC cells (201 ± 4.06 per μm^2 cell section). The ribosomes in LSC cells, however, are defective in their functional organization. The rich formation of ribosomes in polysomes and helical arrangements are highly characteristic of psoriasis (1) but comparatively rarely observed in LSC. The numerous mitochondria of the LSC-cells are not as ubiquitous as in psoriatic cells and cristolysis and other signs of mitochondrial degeneration or altered mitochondrial differentiation are not as conspicuous in LSC cells as in psoriatic cells. This implies a higher degree of mitochondrial dysfunction in psoriatic cells.

The decided aberrant differentiation of psoriatic epidermal cells partly reflected by a pronounced macrocytosis is also in contrast to the normocytosis of the LSC cells ($69.2 \pm 13.1 \mu\text{m}^2$ versus $29.0 \pm 5.1 \mu\text{m}^2$) (cf. 2).

Orthokeratosis is preceded by the formation of keratohyalin. This formation is conditioned by an association of Odland bodies mainly of the homogenous type II and tonofilaments in the presence of preserved tonofilament-desmosome complexes (4). The abundant Odland bodies mainly of type II but with a central cleft, conceivably representing less differentiated Odland bodies, seem unable to accomplish the mentioned association with tonofilaments in spite of the preserved tonofilament-desmosome complexes, the final outcome of which is parakeratosis. In fully developed psoriatic lesions the paucity of tonofilaments and of pre-

served tonofilament-desmosome complexes and the superabundance of defective Odland bodies with central clefts all participate in a pervasive parakeratosis.

The light microscopical areas of orthokeratosis in LSC lesions reveal a peculiar deficiency of keratinization when analysed submicroscopically. The simultaneous occurrence of Odland bodies type II and Odland bodies with dominant central clefts seems to pertain to the observed round or ovoid less electron scattering structures in the cytoplasm of keratinized A-cells, conceivably representing areas of defective keratinization lacking osmiophilic components.

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