Tabel II.

Pat	Sex	Age	Causes of death
M. S.	m	57	Ictus cerebri
L. P.	m	70	Colonic cancer in ulcerative colitis
R. I.	f	70	Breast cancer, diabetes
F. D.	f	85	Heart failure
N. L.	f	88	Angiomyocardiac sclerosis, bladder cancer
G. M.	f	74	Hepatic coma
G. M.	f	61	Bronchopneumonia, heart failure, cirrhosis (ante-mortem bioptic CAH)

Table III. *Normal-values*

SGOT-SGPT	15–45 U/l
AP	70–210 U/l
GT	5–25 U/l
G	19–21%
IgG	800–1 500 mg%
IgM	80–170 mg%

Lichen planus is a chronic skin disease that has rarely been associated with internal disorders. Its association with CAH may be more than fortuitous, as both diseases share a similar histopathology reminiscent of graft-vs-host reaction and may have the same autoimmune pathogenesis.

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Balanitis circumscripta plasmacellularis: Case Report with Ultrastructural Study

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Abstract. A typical case of balanitis circumscripta plasmacellularis is described in a 74-year-old man. Electron microscopy revealed a dense infiltrate composed mainly of plasma cells and macrophages; the former showed considerable rough endoplasmic reticulum and phagolysosomes, while the latter contained variable amounts of sideromes. A few mast cells, eosinophils and extravasated erythrocytes were also present. Connective tissue was scarce; no elastic fibres and viral particles were seen.

Key words: Plasma cell; Rough endoplasmic reticulum; Phagolysosome; Macrophage; Siderome

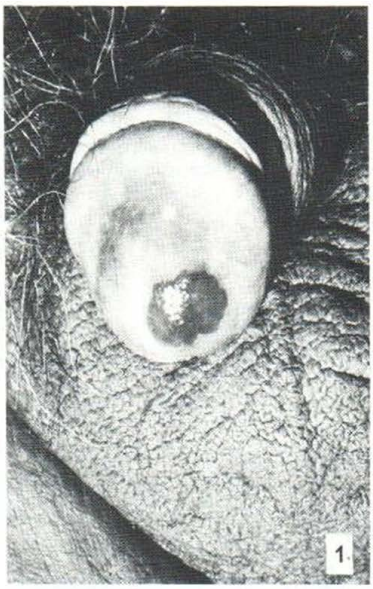
Balanitis circumscripta plasmacellularis (BCP) is a rare disorder which generally consists of a single red, shiny, smooth patch (3, 9, 12). It can also involve the prepuce, the vulva, the oral mucosa and the conjunctiva, for which reason it has also been named "plasmocytosis circumorificialis" (1, 4, 6, 11). Clinically, BCP should be distinguished from the erythroplasia of Queyrat and other erythroplasiform lesions (3, 9, 12). BCP is brown-red, purplish, and shows irregular borders and telangiectases (3, 4, 5, 9).

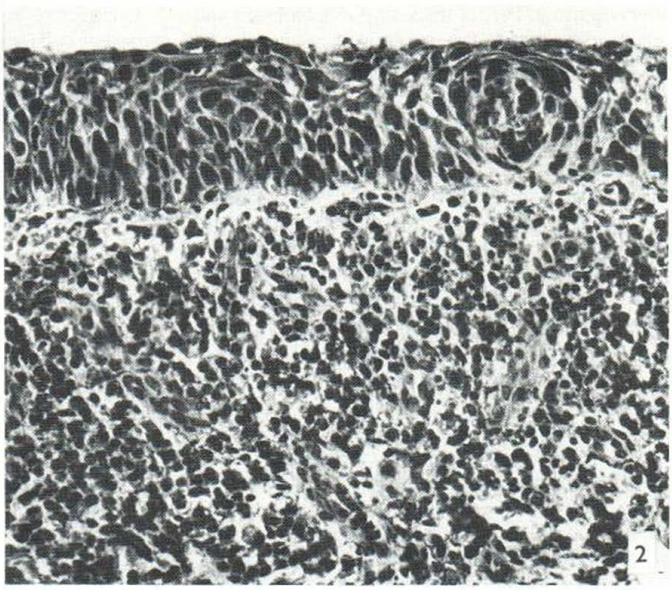
The histological features are also characteristic (3, 8, 9): a band-like inflammatory infiltrate of the upper dermis, mainly plasmocytic, dilated capillaries, and deposits of hemosiderin. The presence of lymphoid follicles is rare (4, 5).

This report describes the ultrastructural findings of a typical case of BCP. To the best of our knowledge, this is the first electron microscopic study of this disorder.

CASE REPORT

A 74-year-old man was seen for a 4-year-old asymptomatic lesion on the glans penis. Physical examination revealed a red, shiny, smooth, sharply-defined 15 mm patch, localized on the left side of the external urinary meatus (Fig. 1). There was no inguinal lymphadenopathy; on the right side there was a hernia. Routine blood and urine





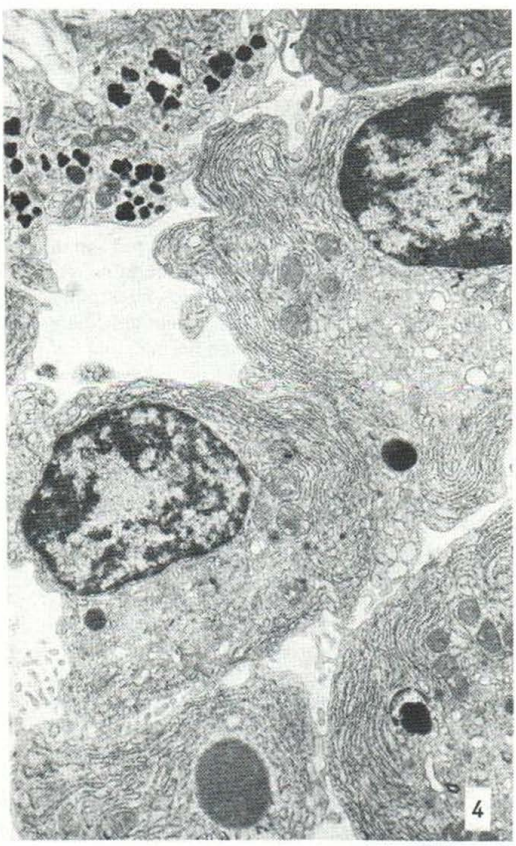


Fig. 1. Red, shiny, well-delimited patch on the glans penis.

Fig. 2. Bowenoid epithelium and band-like dense cellular infiltrate. Abundant hemosiderin deposits. HE, ×320.

Fig. 3. Enlarged intercellular spaces. The desmosomes are distended. ×8000.

Fig. 4. Plasma cells predominate in this area. A macrophage contains sideromes. ×7000.

analyses were normal. Part of the lesion was biopsied and the rest electrocoagulated. The patient was re-examined 2 years later; the disorder had not reccurred.

Light microscopy

Histopathologically the epithelium was of normal thickness, with an absence of granular and horny layers, and discrete generalized spongiosis (Fig. 2). The squamous cell layer showed a certain degree of disorganization and some hyperchromatic nuclei. Exocytosis was rare. The dermo-epidermal junction was flattened. The upper dermis showed a dense, band-like cellular infiltrate composed largely of plasma cells, dilated capillaries and abundant hemosiderin deposits. Extravasation of red cells was pronounced. A thin subepidermal band was devoid of inflammatory infiltrate.

Prussian blue staining revealed tightly packed blue granules located in the cytoplasm of macrophages. Furthermore, some plasma cells contained up to 10 isolated blue granules, near the nucleus.

In semithin sections of specimens for electron microscopy stained with toluidine blue the macrophages showed clustered iron granules. In rare cases the plasma cells contained these granules, but showed various larger blue granules.

Electron microscopy

Specimens were fixed in 5% glutaraldehyde following 1% osmium tetroxide in *s*-collidine buffer at pH 7.4 dehydrated in graded series of acetone, and embedded in Epon-Araldite. Ultrathin sections were stained with uranil acetate and lead citrate.

The epithelial intercellular spaces were enlarged and the desmosomes were distended (Fig. 3). The keratinocytes showed microvilli which traversed the intercellular spaces. Some inter- and intra-cellular myelinoid figures were present. The keratinocytes looked normal, and contained abundant glycogen granules and occasional tonofilaments according to the region. Some exocytotic cells were eosinophils. There was no sign of melanocytes, melanin granules, or Langerhans' cells.

The infiltrate was composed largely of plasma cells (Fig. 4) and macrophages (Fig. 5). Plasma cells were either clustered together or intermingled with other cells. They showed extensive rough endoplasmic reticulum and round dense bodies (Fig. 6). Sometimes the endoplasmic reticulum was transversely sectioned and appeared fenestrated. Spherical, dense bodies, sharply outlined from limiting membranes, measuring up to 3 μm in diameter and containing more electron-dense material were found near the Golgi area. Some plasma cells showed up to five such phagolysosome-like bodies. Nuclear filaments were also seen. Often the surface of the plasma cells emitted microvilli.

The macrophages contained numerous hemosiderin aggregates or sideromes. They were generally isolated, polygonal, measuring up to 0.5 μm, i and membrane-bound (Fig. 7). Sometimes they were grouped into the heterophagolysosomes.

Extravasated erythrocytes were frequently seen. Mast cells and eosinophils were also present. Lymphocytes and fibroblasts were rarely distinguishable.

Connective tissue was scarce, edematous, and composed of collagen fibres; elastic fibres were not observed. A small band of connective tissue separated the basal membrane from the cellular infiltrate. The congested capillaries contained erythrocytes and leukocytes (Fig. 8). Often they showed enlarged endothelial gaps. Sometimes the endothelial cells contained hemosiderin or microtubuli. Non-myelinated nerves appeared normal. No viral particles were found.

DISCUSSION

The etiopathogenesis of BCP is still unknown. Some have considered it to be an unspecific inflammatory process (3, 6, 9). Ferreira-Marques (1) thought it was a bacterial response, and Hornstein (4) suggested that it was a proliferating reticulohistiocytosis. Plasma cells are abundant in the periorificialis inflammatory process; they produce antibodies in the well developed rough endoplasmic reticulum. Morphologically the dense bodies revealed by electron microscopy were phagolysosomes. The phagocytosed material could be either endogenous or exogenous. The plasma cells seem to have an evolutional cycle. In the lamina propria of the vagina they display immature, mature and senescent stages (10). The cycle is hormone dependent. Small size, pyknotic nuclei, dilated endoplasmic reticulum and regular plasma membrane indicated involution. The plasma cells described here, having dense bodies, had a regular membrane, but the nucleus and the endoplasmic reticulum were normal. Thus, it is unlikely that they represented autophagolysosomes in involutive cells. It is probable that they were heterophagolysosomes containing iron, but the findings reported here are not conclusive. The existence of such metal in plasma cells had been demonstrated ultrastructurally in hematologic disorders (2). Plasma cells are capable of phagocytosis and, when overloaded with iron, they phagocytose it (7); this situation exists in BCP.

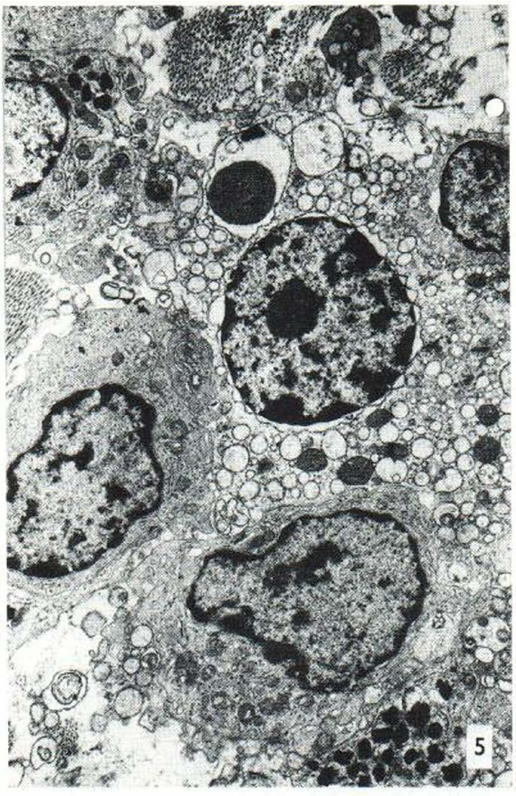
The present electron microscopic study confirms

Fig. 5. Macrophages predominate in this area, showing vacuoles or sideromes. ×7000.

Fig. 6. Plasma cell, showing phagolysosomes and rich developed endoplasmic reticulum. ×11 000.

Fig. 7. Macrophage, showing heterophagolysosomes containing single or multiple sideromes. ×9000.

Fig. 8. Congested vessel containing erythrocytes (E), lymphocytes (L), neutrophils (N) and monocytes (M). ×6000.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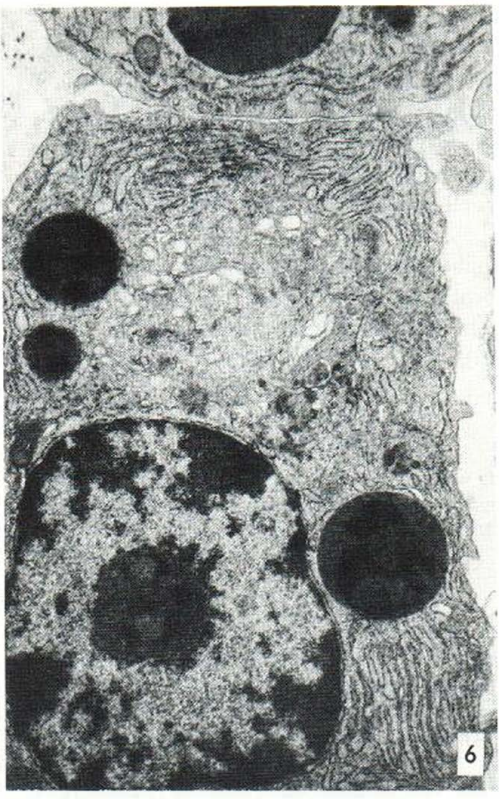
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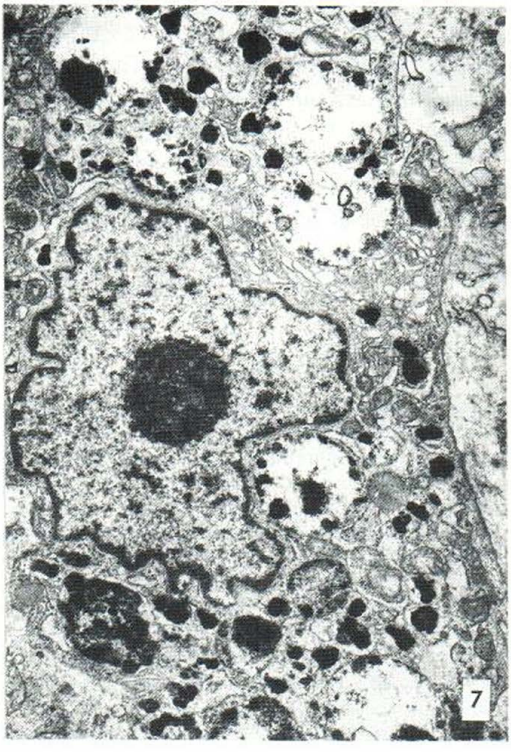
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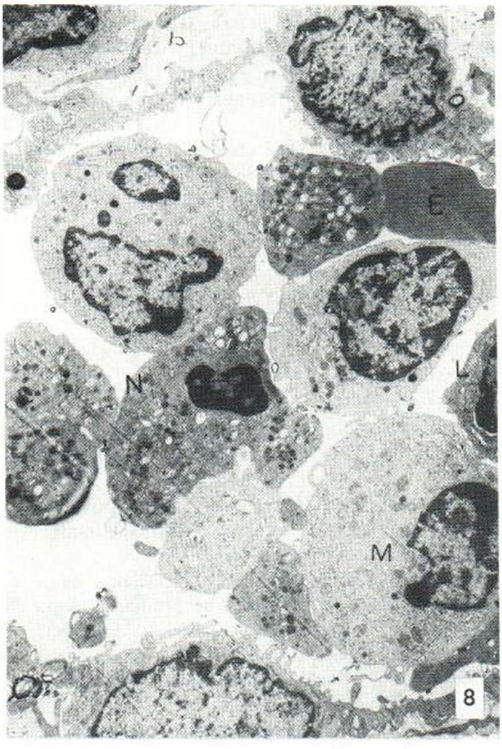
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the histologic concepts. The vascular changes are very important: the dilated vessels carry leukocytes and erythrocytes to the affected region. As a consequence of the repeated hemorrhages, hemosiderin is phagocytosed by the macrophages, to a lesser degree by endothelial cells, and probably also by plasma cells. Intercellular hemosiderin was not found.

The slightly bowenoid epithelial appearance seen histologically was not verified by electron microscopy. This study confirms the absence of elastic fibres in the infiltrated areas of BCP. A viral origin is rejected.

ACKNOWLEDGEMENTS

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Malignant Melanoma Caused by UV-A Suntan Bed?

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Abstract. A 31-year-old female hairdresser who had exposed herself regularly to UVA on a suntan bed for a year developed a malignant melanoma on the left wrist. For 6 years there had been a brown pigmented lesion there which was normally covered by her watch, except when she lay on suntan bed.

Key words: Malignant melanoma; UVA suntan bed

Skin malignancies (carciomas) in humans are clearly related to UV-light exposure (3) and the expanding business of sun-tan parlors (sun-lamps) has provoked a statement from the AAD (9). The Austrian Dermatological Society has recently published a warning against the use of UV-A light (6).

In Denmark and many other places in Europe tanning booths with relatively high intensity UV-A light have become increasingly popular during recent years. These sun-lamps have been installed in ladies' hairdressers and in special sun-tan saloons.

The light emitted from these lamps is almost entirely UV-A, and only 2–5 % or less of the emission is in the UV-B area, thus avoiding a sunburn reaction, irrespective of skin type.

The present case may raise a question of the safety of this unprofessional, cosmetic suntanning.

CASE HISTORY

A 31-year-old female ladies' hairdresser had 15 years earlier had a periocular eczema (patch tests: negative). In 1977, she underwent curettage of two compound nevi on the left side of the nose.

In 1967 she had a severe sunburn reaction with blister formation on the upper part of the back and breast which has left multiple lentigines. She has blue eyes, blond hair, and anamnestically normal tolerance to sunlight.

From December 1979 to April 1980 and from October 1980 to March 1981 she used the UV-A sunbeds installed at her place of work regularly at least once a week. No record was kept of the total exposure time, and the energy in joules cannot be calculated.

Six years earlier she had observed a pigmented lesion under the watch on her left wrist (documented by a color