ULTRASTRUCTURE OF THE ADRENAL CORTEX IN BURNS

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Submicroscopic changes in adrenocorticocytes and sinusoidal capillaries were studied in experiments on sexually mature male guinea pigs on the 1st-2nd, 7th, 14th, 21st, and 28th days after burns. During the first week the hormone-forming cells of the gland were dominated by hyperplasia, which took place against the background of functionally excited sinusoids. On the 14th day, besides hyperplasia and hypertrophy, destruction of organelles was considerably increased in parenchymatous and endothelial cells. Increasing disorganization and death of membranous components of the cells led to a subsequent sharp disturbance of vaso-parenchymatous relations and to the development of profound dystrophic changes and necrobiotic lesions of the adrenal cortex.

KEY WORDS: adrenals; ultrastructure; burns.

An important role in the complex pathogenesis of burns is played by the glands of internal secretion and, in particular, the adrenal cortex, which produces adaptive hormones. Many investigations have been undertaken in connection with the study of this important problem, mainly with the use of physiological and biochemical methods [1, 5, 6, 8, 14, 16]. There is much less information, and what there is is contradictory, on the character of morphological changes in the adrenal cortex in extensive and deep burns [2, 4, 9, 12, 13, 15, 17, 18]. In the accessible literature there was no information on changes in the adrenals during burns at the subcellular level. At the same time, there is convincing evidence that functional overstrain on organs and tissues during stress is accompanied by considerable disturbances of the ultrastructural components of the cells [3, 10, 11], which are manifested as a complex reaction described as the "standard form of intracellular dystrophic process" [7]. With these considerations in mind the investigation described below was undertaken.

EXPERIMENTAL METHOD

Experiments were carried out on 40 sexually mature male guinea pigs. A burn was inflicted by steam at a temperature of 96-97°C on the epilated surface of the back for 60 sec. The size of the burned area averaged 20% of the body surface.

Histological investigations of the skin confirmed the development of burns of the IIIA and IIIB degrees. The animals were decapitated after 1-2, 7, 14, 21, and 28 days. Material for electron microscopy (UÉMV-100 microscope) was processed in the usual way. The ultrastructure of the adrenal cortex of intact guinea pigs was the subject of a parallel study.

EXPERIMENTAL RESULTS

Ultrastructural changes 12-24 h after burning were most marked in cells of the zona fasciculata. They were manifested submicroscopically as a marked increase in the number of liposomes in the adrenocorticocytes, vacuolation of many of them, and appreciable dilatation of the tubules of the cytoplasmic reticulum. Most mitochondria were swollen and contained an unevenly condensed matrix and a few vesicular cristae. At the same time mitochondria with translucent matrix and with separated and sometimes destroyed membranes were seen.

The perinuclear zone was enlarged. The nucleolus lay directly against the karyolemma. Changes observed in the parenchymatous cells during the period of burn shock developed against the background of sharp

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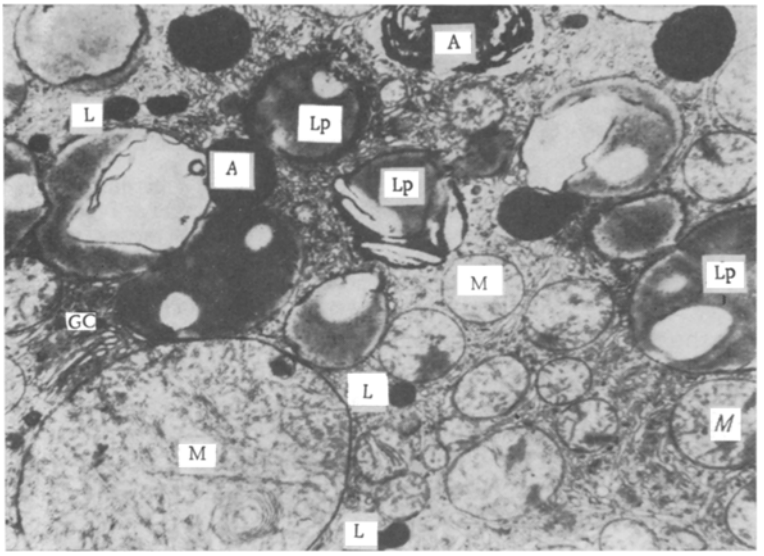

Fig. 1. Ultrastructure of an adrenocorticocyte from zona fasciculata on 7th day after burns: changes in mitochondria, vacuolation and hydrolysis of liposomes, enlargement of autophagosomes (20,000×). M) Mitochondria; L) lysosomes; A) autophagosomes; Lp) liposomes; GC) Golgi complex.

dilatation of the capillaries, periendothelial spaces, and intercellular spaces, which, taken together, resulted in maximal communication between hormone-forming cells and the blood stream.

On the 7th day after burns considerable changes also were observed in cells of the zona glomerulosa. There was an increase in the number of epithelial cells, which were dominated by mitochondria with vesicular cristae, i.e., many adrenocorticocytes had acquired morphological features of fascicular cells. Dark and pale cells also were seen in all zones of the adrenal glands. The latter contained large liposomes, with smooth outlines, and a few organelles. The dark adrenocorticocytes were characterized submicroscopically by the development of a cytoplasmic reticulum, few "motheaten" lipid inclusions, and the presence of elongated mitochondria with a dark matrix. No significant changes were found in the Golgi complex. At this stage of burns many of the hormone-forming cells contained fewer ribosomes, contact between the liposomes and mitochondria was modified, and considerable vacuolation of the lipids was discovered (Fig. 1). The number of secondary lysosomes in the adrenocorticocytes was sharply increased.

On the 14th day after burns the number of pale cells containing many very large liposomes and crystal-like structures, evidently of cholesterol, in the adrenal cortex was sharply increased. Only structures of the smooth cytoplasmic reticulum and a few deformed mitochondria, with separated membranes, could be detected in such cells. The shape of the mitochondria in the dark cells was altered. They became long and thin. Compared with the previous stage of burns there were many more swollen mitochondria with gross destruction of cristae, a vacuolated matrix, and locally disintegrated membranes. Everywhere in the adrenocorticocytes the content of granular material was reduced, the number of secondary lysosomes was increased, and zones of focal degradation of the cytoplasm and signs of necrobiosis of the parenchymatous cells were detected. Besides the adrenocorticocytes, the basic components of the blood capillary walls also showed considerable changes. Fenestration of the thinner regions of the endothelium was disturbed, few pinocytotic vesicles were discovered in the dilated zones, and microvilli on the luminal surface of the endothelial cells were virtually absent. Obliteration spots and marginal folds of the plasma membranes had disappeared in the region of junctions. Even more severe disturbances of sinusoid ultrastructure were found on the 21st and, in particular, on the 28th day after burns. At these times many swollen pale endothelial cells with destructive changes of their organoids and signs of desquamation could be seen, connections between the endothelial cells were grossly disturbed, and fragments of cytoplasmic reticulum, liposomes, mitochondria, and other components of disintegrated adrenocorticocytes could be seen in the lumen of the dilated capillaries (Fig. 2).

It is demonstrative that the contents of the disintegrated cells were observed not only in the lumen of the capillaries, but also in the dilated intercellular spaces and pericapillary spaces, whereas blood cells were frequently found among the damaged epithelial cells, especially of the zona fasciculata (Fig. 3).

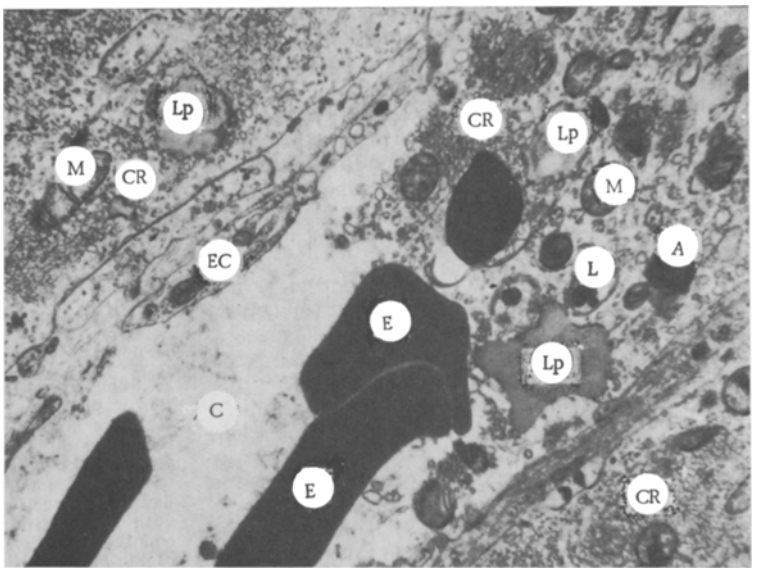

Fig. 2. Zona glomerulosa. Contents of disintegrated adrenocorticocytes in lumen of sinusoid with destructive changes on 21st day after burns (15,000×). E) Erythrocytes; Lp) liposomes; M) mitochondria; CR) cytoplasmic reticulum; EC) endothelial cells; C) capillary; A) autophagosome; L) lysosome.

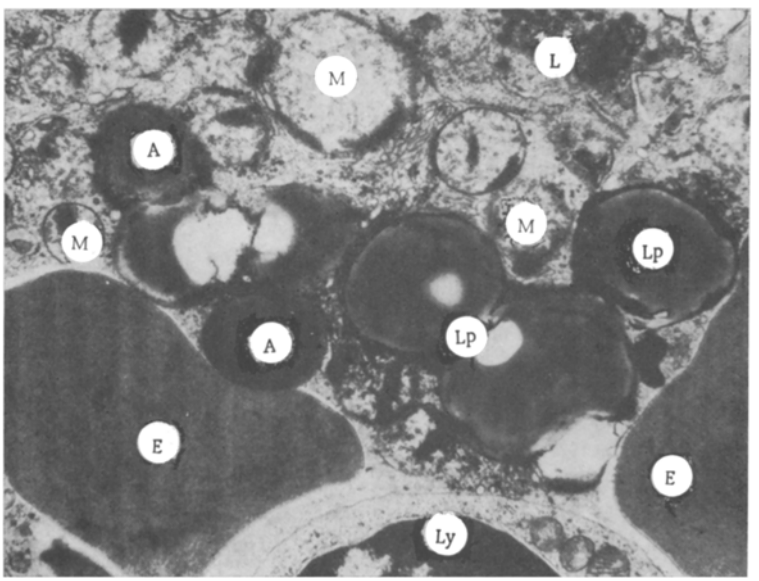

Fig. 3. Infiltration of necrobiotic regions of zona fasciculata of adrenal cortex by blood cells on 28th day after burns (20,000×). M) Mitochondria; Lp) liposomes; L) lysosomes; A) autophagosomes; E) erythrocytes; Ly) lymphocyte.

Disturbance of vaso-parenchymatous relations aggravated the morphological and functional state of the cells and contributed to the development of irreversible degenerative and necrobiotic lesions of the adrenal cortex.

The results are evidence that in burns of the IIIA and IIIB degree a combination of ultrastructural changes, manifested differently at different stages of the disease, develops in the adrenal cortex.

During the first week predominantly hyperplastic processes are observed in the adrenocorticocytes and endothelium of the functionally excited capillaries, evidence of increased functional activity of the adrenal cortex. However, the prolonged functional strain of the cells evidently interferes with reproduction of the

subcellular structures, so that the number of degeneratively changed organelles in the cytoplasm of the cells rises sharply, with the result that the compensatory and adaptive powers of the hormone-forming and endothelial cells are reduced.

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