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ELECTRON MICROSCOPIC STUDY OF THE AORTIC INTIMA OF DIABETIC RATS

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Diabetes is a syndrome of abnormal energetic homeostasis (caused by relative or absolute insulin deficiency) causing a significant dissociation in a short- or long-term period. There is, however, some selectivity of engagement of vessels of the lower limbs, the retina, heart, pulp, etc. (3). Ultrastructural investigations on the early diabetic changes in major blood vessels are rare. The close relation between diabetic changes in the aorta and the fact that this big vessel is the preferable locus for atherosclerosis attracts our interest towards this question, i.e. the aorta under conditions of stable diabetes.

Diabetes was induced in male Wistar rats (400-450 g) by i.p. administration of buffered streptozotocin (65 mg/kg) (donated by the Division of Cancer Treatment, National Institute of Health, Bethesda, MD, USA). The presence of diabetes was proved by the rapid weight loss, polyuria and glucosuria. Controls consisted of two groups of rats of the same initial group. Samples from the aorta were processed for electron microscopy at the 10th week after the streptozotocin injection. Observations were made by JEM 7A electron microscope.

Our data show two types of changes in the intima of the vessel wall: in the subendothelium (SE) and the endothelial cells (EC). SE shows signs of oedema. In most cases it is characterized by solidity, focus of manifestation and predilection for invaginations in basal surface of EC. Sometimes EC are completely separated from the elastic membrane, but at other places they are insignificantly separated or connected with it by digitiformic tentacles. A fibrillar substance like the basal membrane is seen in the oedematose regions of the subendothelial space. This substance has a medium electron microscopical density and it is distributed evenly in the whole circumference of the vessel. There are young elastic fibres. Manifestation of cells (in the most cases myocytes) producing filamentous structures is established. However, they show signs of depletion and degeneration. The same changes can be seen in the elastic fibres. Numerous matrix vesicles have a connection with these reorganizing processes. The changes in the myocytes and the presence of matrix vesicles are one of the morphologic manifestations of initial atherosclerosis. The

changes in EC are expressed mostly by the abnormally increased micropinocytotic vesiculation on the background of the light matrix. We established vesicles even in the basal digitiformic tentacles of EC. Coated vesicles in a different stage of evolution and with different localization (luminal or basal) were seen. A common feature in EC, in comparison with the controls was the increase of Weibel-Palade bodies (WP). In addition, destructive forms of mitochondria, lysosomes, filamental and tubular structures with middle electronmicroscopic density were established in some EC.

Our data show morphological changes in aortic wall after streptozotozin-induced diabetes in rats expressed by the manifestation of oedema in the subendothelial space. We connected this fact with the primary changes in EC mostly related with permeability functions (1,2). The presence of increased micropynocytotic vesiculation and intactness of cell junctions turns our attention to the role of the transcellular transport in the genesis of endothelial oedema (3). The barrier function of EC is not only by the abnormal vesiculation. The presence of intracellular oedema, manifested by the rare matrix, as well as the increased amount of WP bodies are the other changes. The reaction of EC in this case is identical but not specific towards different causes. It can be speculated that diabetes of that type uses the same unique mechanism for the initiation of changes.

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