DYNAMICS OF ULTRASTRUCTURAL CHANGES IN SMOOTH MUSCLE CELLS OF AORTIC COMPRESSION CHAMBER IN EXPERIMENTAL STENOSIS

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Smooth muscle cells are the only cell component in the medial tunic of the aortic wall producing the precursors of vascular extracellular matrix (7, 8). They play a basic role in the tissue transformation of the wall in response to traumatic influences. The altered hemodynamics in case of “critical” stenosis of the aortic lumen exerts similar influences on the wall. It means a reduction of the blood flow with about 70 per cent (3) without its complete stopping. Aortic part above the stenosis presents namely an aortic compression chamber (1). The long-lasting loading of chamber wall in these conditions diminishes sharply its elastic and volume capacities which cannot be then compensated even on the account of oncoming dilatation of the chamber (1). This is a result from the tissue alterations of the wall the intimate mechanisms of which are not clarified enough.

The aim of the present work is to study the age dynamics of the ultrastructural changes in smooth muscle cells in experimental stenosis that will enable to enlighten some aspects of the process of tissue transformation of the arterial wall.

Material and methods

9 one month old dogs were surgically implanted a metal clamp on the abdominal aorta 2 cm below right renal artery beginning. Aortic lumen stenosis about 70 per cent was achieved. The duration of the experiment was 1, 3, 5, 10, 20, 30, 50 and 60 days. 3 control animals were operated without any metal cramp implantation. The animals were killed on the 5th, 20th and 60th day. Material from the wall of the thoracic aorta (aortic compression chamber) was taken for electron microscope study. Fixation with glutaraldehyde 3 per cent solution in phosphate buffer with pH 7,4 for 2 hours at 4 °C and with osmium tetraoxide 1 per cent solution for 2 hours at 4 °C, too. Material was embedded in Durcopan/Fluka and investigated on a transmission electron microscope JEM-7A.

Results and discussion

In early experimental terms (1, 3, 5 and 10 days) we establish a strongly developed granulated endoplasmic reticulum with dilatation of its cisternae filled with non-homogenous electron light matrix widely communicating with perinuclear space when smooth muscle cells in the medial tunic of the aortic compression chamber are observed. The enlargement of some cisternae which are subsarcolemmally located takes a vacuolar character. Golgi complex is poorly developed. Mitochondriae are with intact mitochondriolemma, electron-dense matrix and well-visible cristae. Myofilaments are subsarcolemmally located. Basal membrane is unbroken (fig. 1). About the 10th day the dilatation of the cisternae of the granulated endoplasmic reticulum is very well-expressed.
With 30-day experiment the granulated endoplasmic reticulum in the smooth muscle cells is strongly developed, its cisternae are dilatated but vacuolary dilatations are absent. Their contents is homogenous with a mean electron densi-

Fig. 1. Electronogram of smooth muscle cell from the wall of aortic compression chamber. 1st experimental day. Massive dilatation of the cisternae of the granulated endoplasmic reticulum. Dilatated perinuclear space. M — mitochondria. N — nucleus. Magn. × 20 000

ty. A wealth of Golgi complex is established. A partial enlightening of the matrix, loss of cristae and lesion of mitochondriolemmal parts can be observed in mitochondria. Myelin-like figures occur, too.

In later experimental terms (50 and 60 days) granulated endoplasmic reticulum of smooth muscle cells is well-developed with single cisternae dilatations. The latter are also filled with an electron-dense matrix. Golgi complex is strongly developed. The alterations described in the mitochondrial structure occur more often. There is a wealth of myelin-like figures some of which are formed on mitochondrial basis (fig. 2). In certain regions a condensation and disorganization of the basal membrane is observed.

The increased pressure from the lumen exerts a main traumatic influence upon the wall of the aortic compression chamber in conditions of our experiment. In this case this pressure has a rather complex genesis — both mechanical and renal (3). The ultrastructural response of smooth muscle cells immediately on the first day of the trial consists in a hypertrophy of the cell organellae responsible for the synthesis of the precursors of vascular extracellular matrix. Here
smooth muscle cells of secretory type, strongly “modified” (4, 5) are considered. The absence of a well-developed Golgi complex in the same cells and subsarcolemmally located cisternae gives us reason to accept the concept about a direct precursor export (6). The hypertrophy of the granulated endoplasmic reticulum which persists in any experimental terms as well as the vacuolary dilatation in the early ones can be considered by us an ultrastructural expression of developing thickening of the medial tunic of aortic compression chamber in experimental conditions. Some changes of the mitochondrial ultrastructure can be seen in the intermediate terms of the experiment (on the 20th and 90th day). However, they occur considerably more frequently in later terms. This fact together with the myelin degeneration of the mitochondriae and basal membrane disorganization is an indication for certain pathological alterations in smooth muscle cells most probably due to hypoxia.

In conclusion it should be mentioned that ultrastructural changes in smooth muscle cells are with functional-adaptive character (2). On the other hand, data about mitochondrial lesion in late experimental terms as well as basal membrane disorganization can be an initial sign of smooth muscle cell damage.

Fig. 2. Electronogram of parts of smooth muscle cells at the 50th day of experiment. A developed granulated endoplasmic reticulum. Wealth of Golgi complex — G. Alterations in the mitochondriae — M. Left below — myelin-like figure on the basis of mitochondriae. Magn. × 20 000
Исследовались ультраструктурные изменения в гладкомышечных клетках аортной компрессионной камеры при экспериментальной стенозе брюшной аорты. Экспериментальный стеноз был проведен на 9 одноутробных щенках посредством наложения металлической скобы, сужающей просвет аорты приблизительно на 70 %. Длительность эксперимента — 1, 3, 5, 10, 20, 30, 50 и 60 дней.

В ранние экспериментальные сроки наблюдаются массивные расширения цистерн гранулированного эндоплазматического ретикулума при слабо развитом комплексе Гольджи. В поздние экспериментальные сроки гранулированный эндоплазматический ретикулум (с незначительными расширениями) и комплекс Гольджи хорошо развиты. Наблюдаются изменения и в митохондриях, а также дезорганизация базальной мембраны.

Находка обсуждается в связи с травматическим воздействием гемодинамики на стенку аортной компрессионной камеры в условиях эксперимента. Характер изменений определяется как функционально-адаптивный.