THE ROLE OF ANGIOTENSIN II RECEPTOR ANTAGONIST IN INHIBITING OF MYOCARDIUM REMODELLING

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Angiotensin II (Ang-II) receptor antagonists, preparations acting on AT1 receptors which block the receptor for angiotensin II in the event cascade renin-angiotensin (RAS), are a promising group of drugs [1]. The were registered as hypotensive drugs, however, they are used in the treatment of chronic circulatory insufficiency, atherosclerosis, in patients after cardiac infarct and with chronic renal failure, among others they can retard the progression of diabetic nephropathy [2, 3]. Drugs from this group prevent the hypertrophy of the muscle of the left ventricle of the heart in reaction to pressure overload [4].

Losartan – the most popular drug blocking AT1 receptor – is characterized by high power of action, availability in the oral administration form and the lack of antagonistic properties.

White rats (20) of Wistar strain, females with body mass of 250 g, received losartan (Xartan®, Adamed, Poland) in water suspension, through a tube, into the stomach, in the equivalent dose of 0,28 mg/day during 8 weeks. For examinations using a transmission electron microscope there were collected sections of the left ventricle of the heart, which were prepared according to the standard method for the purpose of the study.

Inside the cardiomyocytes there were observed numerous large mitochondria with extended, long cristae and dense matrix (Fig. 1). Between mitochondria there were small clusters of glycogen. Sarcoplasmatic reticulum cisterns were dilated. The nuclear membrane demonstrated numerous invaginations. In accordance with the role attributed to sartans in the inhibiting of myocardium remodelling processes [3], the study results confirm the influence of angiotensin II receptor antagonists on the ultrastructural image of the myocardium, they also demonstrate the activity of losartan on the cellular level, and moreover, are another reason for further analysis of the preparation pharmacodynamics.

References