CARDIOVASCULAR SYSTEM IN WISTAR RATS RECEIVING RATION WITH HIGH SODIUM CHLORIDE CONTENT

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Objectives:

The aim of the research was to study the influence of diet with different contents of NaCl on the level of arterial blood pressure (BP) and processes of myocardial remodeling in association with changes of nuclear transcription factor κB (NFκB) expression in the myocardium in rats.

Methods:

Two groups of male Wistar rats were studied. Animals in experimental group (E; n=8) within two months received ration with high content of sodium chloride (8%). Rats in control group (C; n=8) for the same time kept on a normal salt diet (0.34%). Concentrations of urea, creatinine, sodium in blood serum; albumin and sodium in the urine were determined. Blood pressure (BP, mm Hg) was measure in awaked rats by tail cuff method. Left ventricular mass index (LVMI) was calculated as the ratio: ventricular mass/body mass (mg/g). Evaluation of the mRNA NFκB gene expression in myocardium was performed using RT-PCR in the presence of EvaGreen. GAPDH was used as the reference gene. The relative gene expression (RGE) values for NFκBp65 were calculated by $2^{-\Delta\Delta Ct}$ protocol. Morphological changes of the myocardium were evaluated by light microscopy in slices stained with H&E and van Gieson. Quantitative morphometric analysis was performed using the system VideoTest 5.2. Results are presented as mean $_{(SE)}$. Unpaired Student t-test was used.

Results:

Between E and C there were no significant differences in the serum levels of urea ($5.6_{(0.8)}$ *vs* $6.2_{(0.5)}$), creatinine ($0.038_{(0.04)}$ *vs* $0.044_{(0.01)}$) and sodium ($144.1_{(1.0)}$ *vs* $143.8_{(1.2)}$, mmol/l; P>0.05 in all cases). The high salt diet resulted in a significant increase of diuresis (E: $8.9_{(1.0)}$; C: $7.0_{(0.5)}$ ml/24h, P<0.05) urinary sodium concentration (E: $401.5_{(60.21)}$; C: $86.3_{(1.0)}$, mmol/l, P<0.001) and albumin excretion (E: $88.8_{(23.98)}$; C: $6.3_{(3.92)}$, mg/24h, P=0.0037). High dietary levels of NaCl did not affect significantly BP (E: $135.0_{(5.0)}$; C: $130.0_{(5.0)}$, P>0.05) and LVMI (E: $2.31_{(0.07)}$; C: 2.30(0.04), mg/g; P>0.05).
However, there were changes in the myocardium structure in the high-salt diet group, including myocardium hypertrophy and hyperplasia of cardiomyocytes (Fig. 1), perivascular fibrosis, angiospasm, increased thickness of the artery walls due to smooth muscle cells hypertrophy (Fig. 2), their vacuolization and vascular sclerosis. Quantitative morphometry showed that high salt diet led to the cardiomyocyte thickening (E: $14.1_{(0.3)}$; C: $11.07_{(0.4)}$; μm, P<0.001), the increment of length (E: $15.0_{(0.6)}$; C: $12.6_{(0.6)}$; μm, P<0.02) and area of cardiomyocyte nuclei (E: $44.3_{(1.7)}$; C: $36.2_{(2.3)}$; μm²; P< 0.02). Lastly, the level of gene expression of nuclear κB transcription factor in myocardial tissue was 3.4 times higher in high salt diet group than in rats fed on low salt diet.

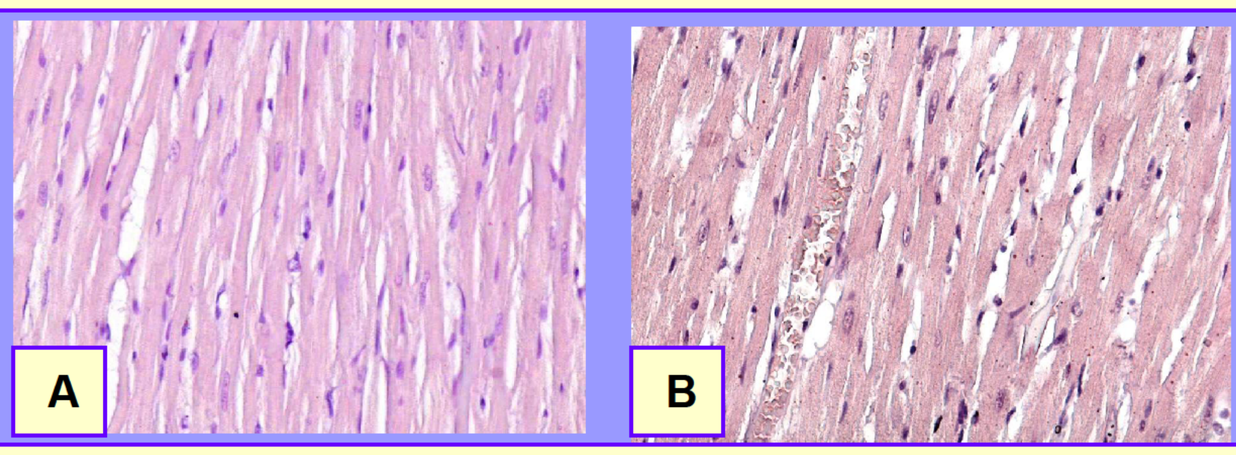

Fig. 1. Cardiomyocytes morphology on low (A) and high (B) salts diet. H&E; ×100

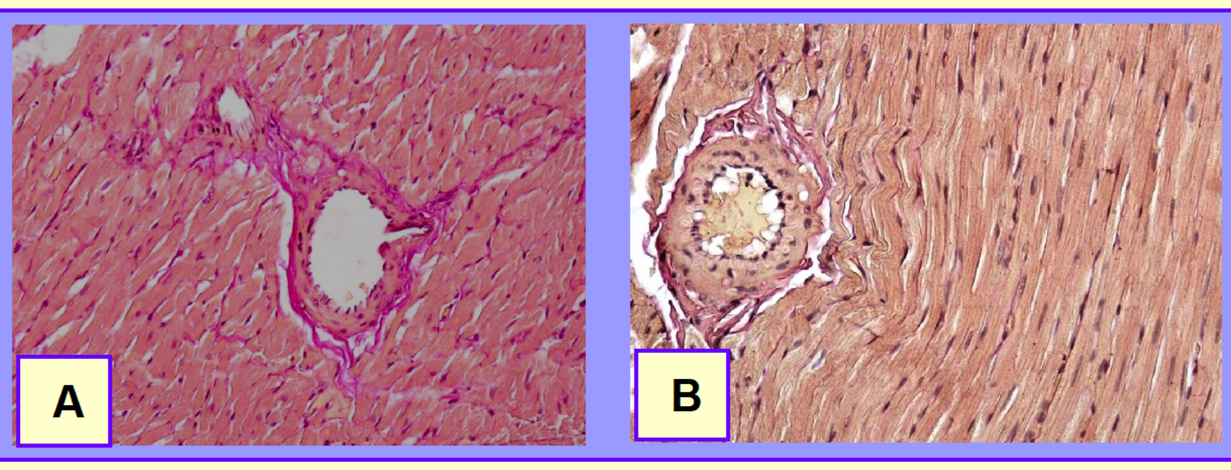

Fig. 2. Myocardial vessels morphology on low (A) and high (B) salts diet. Van Gieson's stain; ×200

Conclusions:

Our findings suggest that the high consumption of sodium chloride can cause myocardial remodeling regardless of changes in BP. These changes might be associated with NFκB associated signaling pathways activation. However, their role in this type of myocardial remodeling requires further investigation.

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