

Selenium levels and functional state of liver in patients with hypertension and nonalcoholic fatty liver disease

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Introduction. Hypertension (HTN) is significant medical problem due to high levels of morbidity and mortality worldwide. Nonalcoholic fatty liver disease (NAFLD) is chronic disease, with gradual progression and is one of main causes of cirrhosis and hepatocellular carcinoma.

Researches show controversies in Selenium levels in NAFLD patients. According to data, lower Selenium levels can increase risks of development of hypertension, while its increase can promote protective effect. Research in this area is actual due to insufficient data on Selenium metabolism in patients with HTN and NAFLD comorbidity, especially depending on liver damage grade.

Aim was to investigate levels of Selenium in patients with hypertension and NAFLD depending on functional state of liver.

Materials and methods. We examined 100 patients. Main group: 49 hypertensive NAFLD patients (33 (67.3%) females and 16 (32.7%) males). Comparison group: 51 non-hypertensive NAFLD patients (30 (58.8%) females and 21 (41.2%) males). Control group: 20 relatively healthy individuals (11 (55.0%) females and 9 (45.0%) males). Median age in main group was 51.0 [45.0-56.0] years; comparison group: 52.0 [47.0-54.0] years ($p=0.564$); control group: 51.0 [45.0-55.5] years.

In the main group hypertension 1 stage was diagnosed in 14 (28.6%) patients, 2 stage : in 35 (71.4%) patients; 1 and 2 grade was diagnosed respectively in 16 (32.7%) and 33 patients (67.3%).

In the main group steatosis was found in 27 (55.1%) patients; steatohepatitis : in 22 (44.9%) patients; in comparison group 30 (58.8%) and 21 (41.2%) patients respectively ($p=0.707$).

Selenium levels were assessed using Humal Selenium ELISA kit. Ultrasound of liver was performed according standard procedure using Samsung (Medison) SonoAce X8. Statistical calculations were made using IBM SPSS 25.0.

Results. Median systolic and diastolic blood pressure in main group was 150.0 [145.0-158.0] mm Hg. and 90.0 [85.0-90.0] mm Hg. In comparison group: 125.0 [115.0-130.0] and 80.0 [70.0-80.0] mm Hg. Respectively

Selenium levels were significantly ($p<0.001$) increased in patients of comparison group: 67.2 [61.5-77.4] mkg/l vs. 43.5 [39.9-49.1] mkg/l in main group. In control group its levels were 108.0 [96.9-118.8] mkg/l, which was significantly ($p<0.001$) higher than in other groups.

Analysis of Selenium levels depending on liver damage grade did not show significant differences. However patients with hypertension and steatohepatitis had higher Selenium levels than patients with steatohepatitis, while non-hypertensive patients had reversed trend (table 1)

Conclusions. Hypertension is main trigger of decrease in Selenium levels in patients with comorbid NAFLD, which can be connected with its increased metabolism due to providing antihypertensive homeostasis and protection of vascular endothelium via antioxidant mechanisms.

Table 1

Indices of Selenium metabolism in hypertensive and non-hypertensive NAFLD patients depending on presence of steatosis and steatohepatitis, Me [Lq; Uq]

| Indices | HTN+NAFLD (n = 49) | | p ₁₋₂ | NAFLD (n = 51) | | p ₃₋₄ |
|-----------------|----------------------|--------------------------|------------------|----------------------|--------------------------|------------------|
| | Steatosis (n = 27) | Steatohepatitis (n = 22) | | Steatosis (n = 30) | Steatohepatitis (n = 21) | |
| Selenium, mkg/l | 42,4 [34,5; 49,5] | 46,0 [42,3; 49,5] | 0,169 | 69,9 [62,4; 77,5] | 66,4 [57,0; 78,1] | 0,394 |