Thus, in animals of the intact and control groups it was found that during the first month of life there is insignificant content of the VSA receptors in the meniscus structures with a slight increase in the outer zone at the end of the second month. Intrafetal antigens injection results in a relative increase in the content of α-D-mannose residues in the outer zone on the 7th day and in the inner zone on the 21st day. The revealed changes between the observation groups are leveled after the 30th day.

References:

Key words: meniscus, rat, antigen, Vicia sativa agglutinin (VSA).

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ROLE OF ADIPOKINES AND INTERLEUKIN-17 IN THE REGULATORY MECHANISM OF BONE REMODELING IN RAT MODEL OF KIDNEY FUNCTION DISORDER

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Cytokines and adipokines take an active part in regulatory mechanisms of bone remodeling which is involved in the higher level mechanism of regulation uniting bone with the kidney. Role of these substances in the mechanisms is not clearly understood. Increased adiponectin levels are characteristic of the chronic kidney disease and associated with its progression. It promotes renal fibrosis through inducing monocyte-to-fibroblast transition [1]. In normal kidney it has anti-inflammatory properties and supposed to have renal protecting effect. Adiponectin signaling plays an important role in endocrine mineral regulation. Vistatin level is also increased in kidney disease and it was supposed that vistatin may mediate inflammatory responses inducing pro-inflammatory cytokines [2]. IL-17 is considered as proinflammatory cytokine which augment renal injury [3]. The aim of the study was to investigate the effects and interrelations of adiponectin, vistatin and IL-17 in bone remodeling regulation in kidney function disorder model in rats.

Materials and Methods In this study two groups of 9-month-old female rats (control (20 intact animals) and experimental (20 animals)) weighing 210 ± 30 g were used. Renal excretory function impairment was created by a single intramuscular 50% glycerol injection in dose of 1.0 ml / 100 g weight of the animal. Experiment was carried out in 12 weeks after glycerol injection. Blood samples were collected through heart puncture of the anesthetized with chloroform rats. Serum adiponectin, vistatin and interleukin-17 (IL-17) levels were measured using ELISA. The impairment of bone remodeling was controlled by direct measurements of the bone density which was calculated as a ratio between the bone mass (g) and the bone volume (cm³) was measured by the liquid replaced. The statistical analyses were performed by Statistica 6.0 programmes. The significance was considered at p<0.05.

Results. In rats with kidney function disorder model all the cytokines have elevated levels (p<0.05). These data correlate with literature [4-7]. Analysis of their interrelations showed that in the intact rats there was no correlation between adiponectin and vistatin levels, weak positive correlation between adiponectin and IL-17 and weak negative correlation between vistatin and IL-17 was shown. This may be the evidence of the same direction of adiponectin and IL-17 regulating action and opposite one of vistatin and IL-17 in the intact animals. In kidney function disorder model these cytokines change their interrelations. It was high negative correlation (r=−0.79) of adiponectin with vistatin levels, medium strength negative correlation (r=−0.43) of adiponectin with IL-17 levels and high positive correlation (r=0.84) of vistatin with IL-17 levels, demonstrating unidirectional regulatory effects of vistatin and IL-17 and opposite adiponectin with vistatin and adiponectin with IL-17 directions of their effects. This may give evidence of these three cytokines compensatory effects in impaired functional regulatory cytokine network in rats with kidney function disorder. Kidney function disorder model results in inflammatory processes and this involve certain links of integrative regulatory mechanisms which unite regulation in bone and kidney. Increased levels of proinflammatory vistatin and IL-17 may demonstrate their role in bone and kidney integration.

Conclusion. Adiponectin, vistatin and IL-17 can be identified as a link in regulatory mechanisms of bone remodeling in model of kidney function disorder.

Perspectives These adipokines and IL-17 may serve as a promising therapeutic target for correction of kidney disease.
TIBIA FRACTURE-INDUCED OXIDATIVE STRESS IN MEN IS ABLE TO UP-REGULATE METALLOTHIONEINS AND TRIGGER CASPASE-3-MEDIATED APOPTOSIS

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Trauma is avowed as an increasingly portion of the global burden of injuries and disorders. It displays a dominant clinical and socioeconomic issue especially in the context of osteoporosis which is constantly increasing nowadays and is able to cause higher rate of bone fragility and can enhance a risk of fractures. Nevertheless the risk of tibia fracture is relatively high all over the world, particularly in male (21.5 incidence/100,000/year) its pathogenesis is studied sporadic and not clear (Mills et al., 2017).

Material and methods. In regards of abovementioned we evaluated the parameters of oxidative stress which plays a major role in the development and progress of number of pathologies, and metabolic changes in men who had tibia fracture due to trauma. Also, metallothioneins (MTs) as the metal-buffering proteins with putative antioxidant function were estimated. The cytotoxicity markers namely caspase-3, DNA strand breaks and lactate dehydrogenase (LDH) were detected to evaluate the severity of lesions. About 15 men from each of control (C) and fracture (F) groups were screened. The panel of markers was applied due to the guidelines.

Results. The lower activity of catalase (by the 29%) and glutathione-S-transferase (by the 23%) as well as the level of glutathione (by the 83%) in the injured fellows were observed when compared to the control. Patients with bone fracture had several signs of oxidative stress namely TBARS and protein carbonyls. Meanwhile F-group was characterized by the higher concentration of MTs. Obviously MTs have been involving in scavenging of reactive oxygen species which are overexpressed when antioxidants are down-regulated and attenuate the fracture-induced oxidative stress. Tibia fracture provoked a cytotoxicity which was manifested by increasing LDH, DNA fragmentations and caspase-3 activity, the key effector of apoptosis in osteoclasts. The greatest variability was shown for glutathione (IV=5.8), protein carbonyls (IV=3.3) and LDH (IV=2.4).

To sum up when tibia fracture occurs in men, a remarkable outflow of oxidative injury products have been generating by the damaged tissue and the caspase-3 mediated apoptosis in cells was triggered. Continuous oxidative stress could impair healing of fractured bones, but antioxidant supplementation in post-traumatic rehabilitation of patients is able to ameliorate cell redox-state and accelerate remodelling of fractured bones.

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References:

Key words: Tibia fracture, metallothioneins, apoptosis, oxidative stress