of gentamicin nephropathy, it's advisable to use medicines with antioxidant properties for the prophylactic and correction of this pathology.Glutathione functionates as a hydrogen donor and a cofactor of principal antioxidant enzymes – glutathione peroxidase and glutathione reductase [AoyamaK.,2015].

The aim of research was to estimate a nephroprotective potential of glutathione on a model of gentamicin-induced acute kidney injury.

The research was conducted on 21 non-linear white rats weighting 140-180 g, maintained under the standard vivarium conditions with free access to water and food. Animals were divided into three groups (n=7): 1st group – control, 2nd – animals with gentamicin nephropathy, induced by the dailyinjectionof 4% gentamicin solution in dose 80 mg/kg during 6 days, animals of the 3rd group were dailyinjected by glutathione preparation (TAD 600, "BiomedicaFoscama", Italy) in dose 30 mg/kg 40 min after gentamicin administration. Kidney function was assessed 24 h after the last gentamicin injection by the indices of diuresis, creatinine plasma concentration(Pcr), glomerular filtration rate (GFR) and protein concentration in urine(Uprot).

Progression of severe kidney injury after gentamicin administration resulted in the reduction of diuresis by 62% ($p<0.01$), decrease of GFR by 2.4 times ($p<0.01$) with the development of retentional azotemia,confirmed by an increase of Pcr by 72% ($p<0.01$) comparing to control group. Uprot concentration increased by 3.2 times ($p<0.01$),indicating the critical proteinuria caused by tubular damage.

Administration of glutathione significantly improved the excretory kidney function. Diuresis increased by 1.7 times ($p<0.01$),GFR – by 1.8 times ($p<0.01$), what was accompanied by the reduction of Pcr by 52%. Additionally, proteinuria decreased by 2.3 times ($p<0.01$), protein excretion – by 1.5 times($p<0.01$)in comparison to the untreated animals. Obtained results testify the ability of glutathione to mitigate toxic effects of gentamicin, extending the spectrum of its clinical use.

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THE CHANGES OF ENERGY METABOLISM IN THE TISSUE OF KIDNEYS AFTER SUBLIMATE DAMAGE AND MODULATION OF POTASSIUM FLOW WITH FLOCALIN

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A wide range of kidney diseases are caused by toxicants including heavy metals that determine violation of homeostatic kidney function. Nephrotoxicity is caused by the complexes of divalent metals with proteins. These complexes damage epithelium of tubulocytes during reabsorption with selective lesion of cellular membranes, mitochondria, and inhibit energy dependent transport processes in the tubular part of nephron. It is well known that potassium channels are a potent endogenous system of defense of the organism when energy resources of cells (including ATP) decrease. The aim of research was to study the changes of energy metabolism in the tissue of kidneys under the conditions of acute sublimate kidney injury after administration of flocalin which is ATP-dependent potassium channel activator of sarcolemmal and mitochondrial cell membranes.

The experiments were held on nonlinear laboratory white rats of both sexes 150-170 g of weight after a single and multiple (7 days) intraventricular administration of flocalin 5 mg/kg. Sublimate nephropathy was modeled by subcutaneous injection of 0.1% mercury dichloride 5 mg/kg. The activity of alkaline phosphatase (AP) in the cortical layer of kidney was measured on photocolorimeter KFK-2 according to instruction due to the ability of the enzyme to slit phenilphospate with production of phenol. The activity of succinate dehydrogenase (SDH) in cortical and medullary layers of kidney was measured on spectrophotometer according to contents of restored potassium ferricyanide (Prokhorova M.I., 1982). Biochemical research at the day of modeling of renal pathology has shown the decrease of AP activity by 20.3% in comparison to healthy rats. Activity of SDH decreased by 41.9% in the cortical and by 40% in the medullar layers of kidneys. After a single administration of flocalin to the rats with sublimate nephropathy the activity of AP increased by 12.5%. The activity of SDH in the medullary layer of kidney did not reach the level of intact rats. At the same time, the elevation of this enzyme in the cortical layer comprised 29.6%. On the 7^{th} day of acute sublimate nephropathy a decrease of both AP (by 67.7%) and SDH (by 30.8% and 45% in cortical and medullary layers respectively) was observed. After 7 days of flocalin administration AP increased by 41.3% and SDH increased by 19.6% in the cortical layer of kidneys. Therefore under the conditions of toxic kidney injury to prevent excessive accumulation of cytoplasmic calcium ions, pharmacological modulation of K_{ATP} channels is very important. The increase of AP activity which is donator of phosphor for ATP, as well as the increase of SDH activity which is marker of functional state of mitochondria, show the ability of flocalin to improve energy supplement to the nephrocytes.

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STUDY OF THE EFFECT OF DIFFERENT PIRACETAM DOSAGES ON THE CONDITION OF PROOXIDANT-ANTIOXIDANT SYSTEM OF CERTAIN BRAIN STRUCTURES IN CASE OF ACUTE HYPOXIA

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Hypoxia is the basic condition in pathogenesis of numerous neurologic diseases. According to the data of publications, pharmaco-therapeutic issues of cerebral pathology, being the third in the list of general mortality rate in