МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ

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106-ї підсумкової науково-практичної конференції
з міжнародною участю
професорсько-викладацького колективу
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AMELIORATION OF KIDNEY INJURY INDUCED BY AN ALKYLATING CHEMOTHERAPEUTIC AGENT

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Introduction. Cisplatin is an alkylating chemotherapeutic agent that is used in the treatment of solid tumors and hematological malignancies. A frequent adverse effect of cisplatin is nephrotoxicity, which arises from its accumulation in renal tubular cells, resulting in oxidative stress, inflammation, cell apoptosis and an enhanced risk of acute kidney injury (AKI) and a rapid decline in kidney function. The existing literature and the results of our previous research indicate that the pineal hormone melatonin has a nephroprotective effect in a number of experimental models of AKI. A series of studies have demonstrated that melatonin exerts a range of beneficial effects, including antioxidant, anti-inflammatory, anti-apoptotic, immunomodulatory and cytoprotective properties.

The aim of the study was to evaluate the effects of exogenous melatonin on the animal model of cisplatin-induced AKI.

Material and methods. Cisplatin-induced AKI was induced in rats by a single intraperitoneal injection of cisplatin (cis-diamminedichloroplatinum(II)) at a dose of 6 mg/kg 72 h prior to the withdrawal of the animals from the experiment. Melatonin (N-Acetyl-5-methoxy tryptamine) was administered at a dose of 5 mg/kg in a prophylactic and therapeutic regimen for a period of four days prior to and three days following the modelling of AKI. Biochemical and histopathological assessments were conducted on blood, urine, and kidneys samples. The statistical analysis of the data was conducted using the SPSS Statistics software.

Results. In the model of cisplatin-induced renal damage, a course of melatonin administration resulted in an increase in diuresis due to an increase in glomerular filtration rate (GFR) (2.6-fold, $p < 0.01$), as well as a decrease in the severity of hyperazotemia and proteinuria (1.7-fold, $p < 0.01$), and normalization of potassium levels in blood plasma. The cytoprotective effect was evidenced by a reduction in fractional sodium excretion and an accompanying increase in absolute sodium reabsorption. The data obtained are in accordance with the results of a morphological study of histopathological changes in renal tissue 72 hours after cisplatin administration. The administration of melatonin resulted in a reduction in the number of epithelial cells exhibiting irreversible damage, including necrosis (4.5% vs. 26%) and hydropic vacuolation (1% vs. 64% in the AKI group), while preserving the glomerular structure. Given that the primary mechanism of damage to proximal tubular cells following cisplatin administration is mitochondrial dysfunction, which in turn induces apoptosis, oxidative stress and energy collapse in cells, the antioxidant effect of melatonin and its impact on the energy supply of kidney cells in the context of cisplatin-induced AKI were investigated. It was observed that the activity of succinate dehydrogenase in rat kidney homogenate was elevated by 42.6% ($p < 0.01$) in the presence of melatonin, in comparison to the untreated control group. This increase correlated with an enhanced activity of glutathione peroxidase and catalase in the kidneys, as well as an elevated level of absolute sodium ion reabsorption. A correlation was identified between creatinine clearance and the activity of catalase and glutathione peroxidase in the kidneys, and between fractional excretion of sodium ions and the content of malondialdehyde and catalase activity.

Conclusions. The findings of the study demonstrate that melatonin exerts a complex nephroprotective effect in cisplatin toxicity, as evidenced by the preservation of the morphological and functional state of the kidneys.