**INFLUENCE OF SODIUM POLY-(2,5-DYHIDROKSYFENILEN)-4-THIOSULFATE ACID (PDT-NA) ON LIPID PEROXIDES PROCESSES IN ETHYLENEGLYCOL’S MODEL ACUTE KIDNEY INJURY OF RATS**

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Acute kidney injury (AKI) is the leading cause of nephrology consultation and is associated with high mortality rates by dates of some scientists [1,2]. As mentioned previously, AKI has primarily become a nosocomial disease in the developed world. AKI has an incidence of 5–7% in hospitalized patients [2]. Nephrotoxins contribute to 80–90% of the renal etiologies [1]. The disease burden of AKI results in an estimated $10 billion in additional costs to the health care system in the Unites States [2] and is associated with a mortality of 45–70% [1]. These data indicate that the efficacy of AКI treatment remains a worldwide acute problem.

Today it is proved that one of the main pathogenesis’ links of acute kidney injury is a disruption in the activity of lipid peroxidation processes of cell membranes and is considered one of the leading links in the cellular mechanism of damage of kidneys local hemodynamic and glomerular filtration [1]. Therefore, for the treatment of acute kidney injury, the nephroprotector with membranestabilizing, antiinflammatory, antihypoxic activity in the mechanism of action should have an antioxidant effect. Such a nephroprotective agent can be the sodium poly (2,5-dihydroxyphenylene)-4-thiosulfate acid (PDT-Na) with proven antihypoxic activity. Because in our previous studies it was established that the antihypoxicant PDT-Na showed a nephroprotective effect in acute renal failure of various genesis, not inferior to the effects of reference drugs mexidol, hofitol and tiotriazolin [3].

So, based on the above the aim of our investigation is study of sodium poly-(2,5-dyhidroksyfenylen)-4-thiosulfate acid’s influence on lipid peroxides processes in ethylene glycol’s model acute kidney injury of rats in comparison with the antihypoxant mexidol, antioxidant tiotriazoline and plant nephroprotective drug hofitol. This model reflects the basic pathogenesis of AKI, affordable and easy reproduced in laboratory, suitable for screening and for in-depth studies of potential nephroprotective agents [4].

The research performed with 36 white nonlinear albino rats weighing 200-220 g according to the European convention for the protection of vertebrate animals used for experimental and other scientific purposes (Strasbourg, 1986) and according to the guidelines of the State Expert Center Ministry of Health of Ukraine.

According to the design study, the animals were divided into 6 groups, one of which is intact control (1) and 5 groups that were given ethylene glycol AКI: control pathology (2), and four treatment groups with PDT-Na (3), mexidol (4) ,thiotriazoline (5) and hofitol (6). At the end of the study, the animals were taken out of the experiment according to bioethical principles. Animals were collected blood and kidneys were removed to prepare biological substrates for further biochemical studies [4, 5]. The state of the LPO process was evaluated by the content of dienic conjugates (DC) and TBA-active products (TBA-AP) in the blood and kidney homogenate by conventional methods [4, 5, 6].

It has been shown that ethylene glycol is reliably related to intact control and increases the level of primary products of lipid peroxidation dienic conjugates in rats blood by 1.46 times (p <0.05) and in the kidney homogenate by 1.78 times (p<0.05). Ethylene glycol promotes the accumulation of malonic aldehydes, which is lipids peroxidation end-products, as evidenced by a significant increase in the level of TBK-AP in the rats blood by 1.79 times (p <0.05) and in the kidney homogenate by 1.74 times (p <0.05). Ethylene glycol causes accumulation of primary and products of lipids peroxidation in blood and in the kidneys tissue, which testifies to the development of the pathological process in the kidneys and in the body as a whole.

The use of antihypoxants of the new PDT-Na and reference mexidol leads to the normalization of the functional state of the kidneys and the activity of lipid peroxidation. Under the influence of PDT-Na in comparison with control, the level of DK and TBK-AP in the blood decreases by 1,3 times and by1,5 times (p <0,05), respectively, in the kidney homogenate by1,4 times and by 1,8 times (p <0,05), respectively. A similar dynamics is observed after influence of mexidol and antioxidant tiotriazolinum. Reference plant nephroprotector hofitol showed antioxidant activity which is lower than synthetic PDT-Na, meksidol and thiotriazolin.

Thus, it has been proved that antihypoxant PDT-Na and reference drugs meksidol, plant nephroprotectorhofitol and antioxidant thiotriazolin in conditions of ethylene glycol acute kidney injury show significant antioxidant properties. Antihypoxants PDT-Na and meksidol and antioxidant tiotriazolin exhibit antioxidant activity at the same level and have antioxidant action which prevails hofitol.

Treatment of ethyleneglycol’s AKI with PDT-Na causes a therapeutic effect, which include of antioxidant effect, as evidenced by it sability to inhibit the accumulation of primaryandend-products of LPO in the blood and the kidneys homogenate DK and TBK-AP, respectively. The above data allow recommending antihypoxant with nephroprotective action PDT-Na for the treatment of acute kidney injury.

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