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IMPACT OF TOXIC LIVER INJURY ON RENAL MORPHOLOGICAL AND FUNCTIONAL PARAMETERS IN RATS

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Abstract: Liver lesions produce a number of substances that are not indifferent to the body and must be removed from it. Therefore, the role of the kidneys in liver disease is of particular importance: timely elimination of toxic products depends on their normal functional ability. Metabolic disorders (carbohydrate, protein, lipid) observed in this kind of patients may be associated with liver damage.
Thus, with liver disease, the development of pathological changes in the kidneys is now beyond any doubt.

Keywords: toxic hepatitis, jaundice, hemodynamics, acid-base balance, morphology, nephron, glomerulus, proximal convoluted tubule, distal convoluted tubule.

Relevance. When liver activity is disrupted in the body, a number of various pathological phenomena occur due to the variety of functions of this organ. Such phenomena include changes in the daily protein metabolism and protein composition of the blood, a violation of sugar metabolism and acid-base balance, a disorder of water metabolism and, finally, a change in hemodynamics. In case of liver damage, a number of substances are formed that are not indifferent to the body and must be removed from it. Therefore, the role of the kidneys in liver disease is of particular importance: timely elimination of toxic products depends on their normal functional ability. Already in physiological conditions, there is a functional relationship between the liver and kidneys [3].

Asher and Mayer, using the isolated cardiopulmonary renal system according to Starling in a frog, showed that the inclusion of the liver in the blood circulation increases diuresis. Glaubach and Molitor received increased diuresis in renal patients by prescribing liver preparations. In anatomically healthy kidneys, impaired liver function can cause severe disruption of inter—daily nitrogen metabolism - hepatic uremia, which can even lead to death [2].

Folgard observed uremia phenomena in liver patients treated with urea as a diuretic. In renal patients, with the onset of liver failure, the circulation of toxic products of inter-daily metabolism increases. There are indications of the hormonal effect of the liver on kidney function, which is reflected in the fact that during autolysis of the liver, special nephrotropic substances are formed in it [8].

Thus, with liver disease, the development of pathological changes in the kidneys is now beyond any doubt. A number of authors (Rufanov, Shoffara, etc.) described the so-called hepatic-renal syndrome, in which the primary factor causing kidney damage is liver disease. As for kidney damage in parenchymal hepatitis, we have not found comprehensive descriptions of changes in kidney function in this disease in the literature available to us, nor have we found indications of the mechanism of kidney changes in parenchymal hepatitis. Kidney damage in jaundice is caused by bile acid salts. However, a number of data suggest that changes in the kidneys are caused not so much by bile acid salts in jaundice, but rather by the consequences of a profound violation of liver function. According to the clinical observations of a number of authors, kidney damage in jaundice is observed already in the first days of its appearance. The amount of urine decreases, diuresis becomes negative, cylinders, protein and red blood cells appear in the urine [1].

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In addition, taking into account the close connection of the excretory ability of the kidneys with the activity of the cardiovascular system, arterial and venous blood pressure and blood flow rate. Indeed, during the period of increasing jaundice, there is a drop in arterial and venous pressure and a slowdown in blood flow [9].

Changes in sugar metabolism, residual nitrogen and alkaline blood reserve. During severe jaundice, there is a violation of sugar metabolism; in some cases, characteristic hepatic sugar curves are observed.

All pathological phenomena in the kidneys, as the patient's general condition improves and jaundice converges, usually pass quickly, but sometimes, with an increase in liver damage, cases of death from uremia have been observed [4].

To study kidney function, diuresis is determined, urine tests are performed daily, the amount of chlorides in urine and blood is determined, Zimnitsky, Folgard and McClure samples are taken and the general metabolism in patients: residual nitrogen, reserve alkalinity, cholesterol and bilirubin in the blood.

An increase in residual blood nitrogen. Reduced reserve alkalinity of the blood, often a drop in the alkaline reserve of up to 20%. All these changes are certainly important for kidney function, as they largely determine the conditions in which the kidneys work.

During the period of severe jaundice, the daily amount of urine decreases. Lowering the amount of urine to 200-300 cm3 per day. There were often sharp discrepancies between the drunk liquid and the excreted urine, reaching up to 1500 cm3. At the same time, there may be delays in the release of water and its impregnation of tissues. Thus, the McClure test, which, with severe jaundice in patients, was on average equal to 38 minutes, after the onset of jaundice, lengthened to 55 minutes. To find out the causes of fluid retention, it is necessary to measure chlorides in the blood and urine. At the height of the disorder of water metabolism, in some cases we get a slight delay in urinary chlorides, in most cases the release of chlorides is not disturbed. Blood chlorides, both during the period of jaundice increase, its convergence, and after recovery, fluctuated within normal numbers [5].

Thus, fluid retention in the body does not occur due to the retention of chlorides, the release of which is most often normal; the decrease in chloride release observed in some patients does not correspond to the amount of fluid retention.

Albuminuria since the time of Soloni (1837), who first introduced this term, has been associated with functional or anatomical damage to the kidneys, mainly the tubular apparatus. However, Claude Bernard also pointed out the possibility of protein passing through the kidneys without damaging the latter. Non-renal or dyscrasic albuminuria has been known for a long time.

Many authors assign a significant role to the liver in the mechanism of albuminuria. With albuminuria, liver protein is primarily excreted, not serum protein. Hepatic albuminuria, without kidney damage, was also described by Mergison. There are even attempts to differentiate dyscrasic, hepatic albuminuria from renal albuminuria [7].

So, changes in kidney function in parenchymal hepatitis are expressed in a decrease in diuresis, nocturia, and a decrease in urine excretion during the Folgard test. These changes are not permanent, as jaundice converges, kidney function is restored.

The question of the mechanism of all these changes is very complicated; it consists of various pathological metabolic changes observed in parenchymal hepatitis. Among these changes, the first place is occupied by the phenomena of general toxicosis, consisting of azotemia, changes in the acid-base balance towards acidosis, changes in the protein economy (blood proteins and protein metabolism) towards the predominance of globulins. The elimination of these metabolic

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products, as well as the elimination of the so-called products of liver autolysis, apparently causes damage to the renal tubular system (cholemic nephrosis) [6].

As for the changes in the function of the glomeruli, they undoubtedly stand in connection with a violation of the cardiovascular system. Weakening of cardiac activity may lead to a decrease in filtration in the glomeruli during the period of jaundice. As jaundice converges and the heart is restored, filtration in the glomeruli improves at the same time. Minute filtration of the glomeruli is determined in a greater proportion by renal blood flow. With a significant slowdown in the blood flow rate, a drop in minute blood volume and venous pressure, conditions are created that reduce blood flow through the kidneys, and as a result, minute filtration in the kidneys decreases. In addition, during the period of increasing jaundice, a drop in blood pressure also leads to a decrease in glomerular filtration. Hemorrhages in the renal parenchyma also lead to a decrease in diuresis, due to the shutdown of some areas of the kidney.

Finally, biochemical changes in the blood, due to impaired liver and cardiovascular functions, leading to a violation of the acid-base balance - acidosis, do not remain indifferent to the kidneys. For example, in malignant anemia, with a significant drop in hemoglobin and erythrocytes, impaired renal function is noted. In malignant anemia, a change in kidney function is the result of oxygen starvation due to anoxemia. There is a change in the acid-base balance towards acidosis, which also seems to lower kidney function.

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