EXCELLENCIA
INTERNATIONAL MULTI-DISCIPLINARY JOURNAL OF EDUCATION
Volume 02, Issue 06, 2024 ISSN (E): 2994-9521

Kidney Morphology and Biochemical Changes in Blood during Chemotherapy and Toxic Liver Damage

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Abstract:

With chemotherapy and 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 chemotherapy and 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 chemotherapy and liver damage.

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

Relevance. With chemotherapy and impaired liver activity in the body, a number of diverse 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. With chemotherapy and 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 chemotherapy and 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 chemotherapy and 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 chemotherapy and liver disease, the development of pathological changes in the kidneys is now beyond any doubt. A number of authors (Rufanov, Shoffar, etc.) described the so-called hepatic-renal syndrome, in which the primary factor causing kidney damage is liver disease. As for kidney damage during chemotherapy and 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 during chemotherapy and 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].

In addition, given the close relationship 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.

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].

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. Dyscrasic albuminuria has been known for a long time [5].

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 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].

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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