EFFECT OF CHEMOTHERAPY ON RENAL TISSUE AND ITS MORPHOLOGICAL CHANGES

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Relevance: Chemotherapy, despite being an effective and widely used method in the treatment of oncological diseases, is characterized by systemic toxic effects that lead to profound morphofunctional changes in various organs, particularly in renal tissue [1]. Since the kidneys play a key role in the excretion of cytostatic drugs, nephrotoxic complications are considered among the most frequent and severe adverse effects of chemotherapy [2]. Numerous studies have investigated the pathogenetic mechanisms of chemotherapy-induced nephrotoxicity, emphasizing that toxic damage to renal tissue occurs through several interrelated pathways [1,4]. These include increased oxidative stress, excessive production of reactive oxygen species, mitochondrial dysfunction, activation of apoptosis and necrosis in tubular epithelial cells, as well as impairment of renal microcirculation.

According to data from scientific literature, chemotherapy induces glomerular morphological alterations such as endothelial swelling of glomerular capillaries, thickening and irregularity of the basement membrane, proliferation of mesangial cells, and reduction of the glomerular filtration surface area. These structural changes result in the development of proteinuria and a decrease in the glomerular filtration rate [2,5].

Foreign researchers note that the epithelial cells of renal tubules are the most sensitive structures to the toxic effects of chemotherapeutic agents. Morphologically, this is manifested by hydropic and vacuolar degeneration of epithelial cells, nuclear changes including pyknosis, karyorrhexis, and karyolysis, formation of detritus and casts within tubular lumens, and epithelial desquamation. In particular, under the influence of cisplatin and ifosfamide, necrotic changes develop predominantly in the proximal tubules, which may lead to acute tubular necrosis [3,6].

In the interstitial tissue of the kidney, chemotherapy exposure results in serous or serous-cellular edema, infiltration by lymphocytes and macrophages, increased fibroblast activity, and progression of interstitial fibrosis. These processes ultimately contribute to sclerotic changes in the renal parenchyma [7,10].

Chronic exposure to chemotherapeutic agents in oncology is also associated with vascular alterations, including arteriolar spasm, endothelial dysfunction, hyalinosis, thrombosis, and ischemic changes. These vascular disorders exacerbate renal tissue hypoxia and intensify necrotic processes [8,9].

Conclusion. The nephrotoxic effect of chemotherapy is characterized by a complex of morphological changes involving glomerular, tubular, interstitial, and vascular structures of the kidney. These alterations lead to a decline in nephron functional capacity and clinically contribute to the development of renal failure.

Therefore, regular monitoring of renal function using morphological and biochemical parameters, along with the implementation of nephroprotective strategies, remains a highly relevant issue in patients receiving chemotherapy.

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