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Renal Necrosis Encountered in Forensic Autopsies

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Annotation. Kidney damage consists of the involvement in the pathological process of three main structural elements of the kidney - the glomeruli, the tubulointerstitial apparatus and blood vessels. Regardless of which parts are affected, this is a single structure; all parts of the nephron are involved in the process, so there is no specific picture. The review is devoted to acute renal failure and the causes of this pathology, which led to death.

Key words: necrosis, damage, acute renal failure, nephrotoxicity, ischemia.

Relevance. AKI is a relatively new concept in nephrology, replacing the previously used "acute kidney failure" and means a potentially reversible sudden loss of kidney function. Morphological features of AKI include erasure and loss of the border of the proximal tubules, spotting of tubular cells, focal expansion of the proximal and distal tubules, and areas of cellular regeneration. Zones of tubular necrosis are practically not defined or are found only in the outer region of the medullary layer of the kidneys. **Glomerular necrosis** can be recorded only if the underlying disease of the glomerular apparatus has caused AKI. Stagnation, damage to endothelial cells, and leukocyte infiltration are detected in peritubular capillaries [18]. The traditional division of AKI remains into prerenal causes caused by hypoperfusion, or direct damage to the renal artery, renal (interstitial and glomerular lesions) and postrenal. Prerenal AKI is most often recorded (55-60%), less often - renal (35-40%) and even less often - postrenal (<5%). Prerenal AKI is caused by renal hypoperfusion. With timely restoration of normal renal perfusion, rapid normalization of renal function is observed. The causes of prerenal AKI may be due to a decrease in effective arterial volume: damage to the myocardium, valves, pericardium, rhythm and conduction disturbances; pulmonary hypertension, pulmonary thromboembolism, mechanical ventilation; systemic vasodilation; sepsis, liver failure, anaphylaxis; renal vasoconstriction: norepinephrine, ergotamine, liver disease; under the influence of pharmacological drugs: angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor blockers, non-steroidal anti-inflammatory drugs (NSAIDs). The development of AKI can be observed with general hypervolemia, but with a decrease in arterial blood volume, which is observed in chronic heart failure, nephrotic syndrome, liver cirrhosis and sepsis. Renal kidney damage includes glomerular, interstitial and tubular damage. In addition, there are two additional variants of AKI: renal vascular injury and intratubular obstruction.

Acute tubular necrosis (ATN) is the most common cause of renal AKI, accounting for 70%. There are ischemic and toxic ATN [13]. The share of ischemic ATN in the structure of causes is 50-60%, and in 20-45% of cases it is caused by sepsis [14]. Toxic tubulonecrosis accounts for 20% of AKI cases. Typically, ATN (ischemic and nephrotoxic) occurs under the influence of various factors (eg, sepsis, hypotension, and nephrotoxic medications). The initial

oliguric phase develops within 24 hours from the moment of exposure to the etiological factor and lasts 1-3 weeks, followed by a phase characterized by an increase in diuresis. Many patients with ATN do not have an oliguric phase. The prognosis for patients with ATN is quite unfavorable. Mortality reaches 50-70%, which is explained by severe concomitant diseases. In surviving patients, renal function improves without complete normalization.

Ischemic tubular necrosis. Ischemic kidney injury and prerenal AKI represent two stages of the pathological process. In severe hypoperfusion, tubular cells are damaged and renal dysfunction persists. Risk factors for the development of ischemic ATN include the presence of pre-existing kidney disease - CKD, atherosclerosis, diabetes mellitus; cardiac surgery. Ischemic ATN can develop in the absence of hypotension in cases of impaired renal autoregulation: in the elderly, with severe atherosclerosis, arterial hypertension and renovascular lesions, or in the presence of previous CKD. Nephrotoxic ATN develops under the influence of endogenous toxins (hemo- and myoglobulinuria with massive hemolysis and rhabdomyolysis, respectively) or exogenous toxins. The spectrum of exogenous agents has changed significantly in recent years; antimicrobial drugs, radiocontrast agents, and chemotherapeutic agents predominate.

The main causes of the development of renal AKI: damage to large renal vessels: thrombosis, atheroembolism, thromboembolism, dissection, vasculitis (Takayasu disease); classic polyarteritis nodosa; thrombosis, compression; damage to the glomerular apparatus: primary glomerulonephritis (GN), GN in systemic diseases and vasculitis (systemic lupus erythematosus, Wegener's granulomatosis, microscopic polyangiitis, Churg-Strauss syndrome, hemorrhagic vasculitis - Henoch-Schönlein purpura, cryoglobulinemic vasculitis); GN in infective endocarditis; malignant hypertension; gestosis in pregnant women; scleroderma kidney; hypercalcemia; medications; radiocontrast agents; hematological: hemolytic-uremic syndrome (thrombotic thrombocytopenic purpura), disseminated intravascular coagulation, hyperviscosity syndrome. Pathological conditions characterized by predominant tubular damage (often with the development of ATN): ischemia caused by renal hypoperfusion; exogenous toxins: antibiotics, antitumor drugs, radiocontrast agents, NSAIDs, diuretics, α-methyldopa, allopurinol, azathioprine, etc.; endogenous toxins (myoglobin, hemoglobin, uric acid, myeloma light chains). Acute lesions of the interstitial apparatus: interstitial nephritis (antibiotics, NSAIDs, etc.); infections (viruses, bacteria, fungi); acute cellular reaction of kidney transplant rejection; infiltrative processes (lymphomas, leukemia, sarcoidosis).

NSAIDs (salicylates, indomethacin, naproxen, diclofenac, etc.) have the most severe damaging effect on tubulointerstitial tissue [2]. The development of toxic nephropathy when exposed to NSAIDs depends on the specific drug, dose, duration of use and the patient's health status [3]. The literature describes the development of AKI in children with the use of cyclooxygenase-1 inhibitors: naproxen, dipirone, diclofenac, ibuprofen, paracetamol, ketorolac [4, 5]. In addition, there are reports of the development of AKI in children receiving the cyclooxygenase-2 inhibitor rofecoxib as therapy [46]. Currently, several severe cases of irreversible renal failure have been described in newborns who received indomethacin prenatally or in the first days of life for closure of the ductus arteriosus [47]. The main mechanism of action of NSAIDs is inhibition of the synthesis of prostaglandins from arachidonic acid by inhibiting the enzyme cyclooxygenase [48]. In euvolemia, prostaglandins have little effect on renal hemodynamics. Hypovolemia stimulates the production of prostaglandins in the kidneys, which counteract vasoconstriction by reducing preglomerular resistance and thus maintaining renal perfusion and glomerular filtration [49]. This protective mechanism is disrupted when NSAIDs

inhibit the synthesis of prostaglandins, which leads to a decrease in renal blood flow, the development of acute renal vasoconstriction, medullary ischemia and acute renal failure [50].

Postrenal AKI results from urinary system obstruction (USO), which may occur at the level of the bladder or urethra (lower UROS obstruction) or at the level of the ureters and kidneys (upper UROS obstruction). With unilateral obstruction, AKI syndrome usually does not develop if the function of the contralateral kidney is preserved. The main reasons for the development of postrenal AKI: obstruction of the upper parts of the MVS (bilateral obstruction or obstruction of a single kidney, papillary necrosis, thrombi, invasive renal carcinoma, retroperitoneal tumor, retroperitoneal fibrosis, endometriosis); lower parts of the bladder (neurogenic bladder, bladder carcinoma, blood clots, stones); prostate gland (prostate cancer, prostate adenoma); urethra (strictures, phimosis, calculi). Postrenal AKI can occur with either complete or partial obstruction. In the first case, anuria is observed, in the second - dysuric phenomena (frequent urination, false urges, nocturia, a feeling of incomplete emptying of the bladder). Partial obstruction can occur with oliguria, sometimes without it. **Papilonecrosis** is necrosis of the medulla and renal papillae (epithelium).

Renal papillary necrosis is a complication of underlying kidney diseases in which all or part of the renal papillae dies. The renal papillae are the areas where the collecting duct openings enter the kidney and where urine enters the ureters. Renal papillary necrosis often occurs with analgesic nephropathy. This damage is caused by overexposure to pain medications. Other causes also contribute: diabetic nephropathy, kidney infection (pyelonephritis), kidney transplant rejection, sickle cell anemia (especially in children), urinary tract blockage.

Cortical necrosis, (sublimate kidney) is the death of the tissue of the outer part of the kidney cortex caused by blockage of small arteries delivering blood to the cortex and leading to acute kidney damage.

Etiological factors of cortical necrosis: - bacterial infection (sepsis) dehydration shock hemolytic-uremic syndrome premature placental abruption or abnormal location of the placenta uterine bleeding infections after childbirth, blockage of arteries with amniotic fluid death of the fetus in the uterus preeclampsia rejection of a transplanted kidney pancreatitis snake bite poisoning by drugs and chemicals . Tubular necrosis is a kidney disease characterized by acute damage and dysfunction of tubular cells. Necrosis of the epithelium of the renal tubules. Renal hypoperfusion, which is caused by hypotension or sepsis, nephrotoxins, extensive surgery, burns, pigments heme-myoglobin and hemoglobin, rhabdomyolysis or massive hemolysis. **Nephrotoxicity** is the property of chemical substances, acting on the body in a non-mechanical way, to cause structural and functional disorders of the kidneys [11]. Nephrotoxicity can manifest itself both as a result of the direct effect of chemicals (or their metabolites) on the kidney parenchyma, and indirectly, mainly through changes in hemodynamics, acid-base balance of the internal environment, massive formation in the body of products of toxic destruction of cellular elements that are to be excreted through the kidneys (hemolysis, rhabdomyolysis) [11]. There are two main types of exotoxic kidney damage: specific, reflecting the direct damaging effect of a number of nephrotropic chemicals on the renal epithelium, and nonspecific, constituting the general pathology of the kidney response to “chemical injury” [8]. Common nephrotoxins: aminoglycoside, amphotericin B, cystiotin and other chemotherapy, X-ray contrast agents, NSAIDs, colistimethate, calcineurin inhibitors, cyclosporines, tacrolimus, vancomycin. Toxic exposure causes focal segmental occlusion of the tubular lumen by casts and dead cells or segmental tubular necrosis. Precursors of tubular necrosis: existing chronic kidney disease, diabetes mellitus, pre-existing hypovolemia and poor renal perfusion, old age.

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