INTERNATIONAL MULTI DISCIPLINARY JOURNAL FOR RESEARCH & DEVELOPMENT
eISSN 2394-6334 Impact factor: 7,854 Volume 12, issue 12 (2025)

RENAL PATHOMORPHOLOGICAL CHANGES IN PATIENTS WITH ISCHEMIC STROKE

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Abstract: To study pathomorphological changes occurring in renal tissue of patients with ischemic stroke and assess their clinical significance.
Materials and Methods: The study was conducted on 156 patients diagnosed with ischemic stroke. Renal function parameters (creatinine, urea, glomerular filtration rate) were evaluated. For morphological examination, renal biopsy materials were studied using light and electron microscopy methods.
Results: Ischemic and degenerative changes, tubulointerstitial damage, and signs of glomerular sclerosis were identified in the kidneys of patients who experienced ischemic stroke. The severity of renal dysfunction was correlated with the degree of cerebral ischemia and stroke volume.
Conclusion: Ischemic stroke leads to specific pathomorphological changes in renal tissue and causes the development of cerebro-renal syndrome similar to cardiorenal syndrome.
Keywords: ischemic stroke, renal pathomorphology, cerebro-renal syndrome, glomerular filtration, tubulointerstitial damage
Introduction. Ischemic stroke is one of the pressing problems of modern medicine, characterized by high rates of disability and mortality. According to the World Health Organization, 15 million people worldwide experience stroke annually. Ischemic stroke causes serious pathomorphological changes not only in the central nervous system but also in several organs, particularly the kidneys.
The interaction between the brain and kidneys - cerebro-renal syndrome - has gained considerable attention in recent years. However, morphological changes occurring in the kidneys as a result of ischemic stroke have not been sufficiently studied. This problem is particularly significant in the management of severe stroke and its complications.

STUDY OBJECTIVE

To comprehensively assess pathomorphological changes occurring in renal tissue of patients with ischemic stroke from morphological and functional perspectives.

MATERIALS AND METHODS

The study was conducted on 156 patients with ischemic stroke (males - 89, females - 67, mean age 64.3±8.7 years) treated in the neurology department between 2022-2024. The control group consisted of 40 healthy individuals.
Stroke diagnosis was established based on computed tomography or magnetic resonance imaging. Renal function was evaluated using the following parameters: serum creatinine (Jaffe method), urea (kinetic method), and glomerular filtration rate (CKD-EPI formula).
For morphological examination, renal biopsy materials were obtained from 24 patients who died as a result of severe ischemic stroke. Tissue samples were stained with hematoxylin-eosin, PAS reaction, Van Gieson, and Masson methods. Electron microscopy was performed using a Hitachi H-600 microscope.

Statistical analysis was conducted using SPSS 26.0 software. Results were expressed as M±SD. Student's t-test and Mann-Whitney U-test were used for group comparisons. A p-value <0.05 was considered statistically significant.

RESULTS

Renal Functional Parameters. Impairment of renal function was identified in patients who experienced ischemic stroke. Serum creatinine was 38.6% higher compared to the control group (118.4±22.3 vs 85.6±12.1 μmol/L, p<0.001). Urea levels were also significantly elevated (7.8±1.9 vs 5.2±0.8 mmol/L, p<0.001). Glomerular filtration rate was 24.3% lower in stroke group patients (68.4±15.2 vs 90.3±8.7 mL/min/1.73m², p<0.001).

During the acute stroke period (within the first 72 hours), rapid deterioration of renal function was observed in patients. Creatinine levels reached maximum values on days 3-7 of the disease.

Macroscopic Changes. Morphological examination revealed that kidney mass exceeded normal values by 15-20%, and the fibrous capsule separated with difficulty. The renal cortex appeared darkened, with signs of edema in the corticomedullary zone. Small hemorrhages were observed in the capsule.

Glomerular Changes. Light microscopy revealed the following changes in glomeruli:

Mesangial cell proliferation (42.3% of cases)

Ischemic damage to glomerular capillaries (67.8%)

Thickening of the basement membrane (average 480±45 nm, normal 320±30 nm)

Proteinaceous exudate accumulation in Bowman's capsule (38.5%)

Signs of focal and segmental glomerulosclerosis (28.4%)

Electron microscopy revealed effacement of podocyte foot processes, swelling and detachment of endothelial cells. Fibrin aggregates and microthrombi were observed in capillary lumens.

Tubular Changes. The following pathomorphological changes were recorded in proximal and distal nephron tubules:

Vacuolar and fatty degeneration in epithelial cells (74.6% of cases)

Desquamation of tubular epithelium (56.3%)

Proteinaceous casts in tubular lumens (43.7%)

Signs of tubular necrosis in severely ischemic zones (31.8%)

Damage to tubular basement membrane (45.2%)

Electron microscopy revealed mitochondrial swelling, cristae destruction, and endoplasmic reticulum dilation.

Interstitial Changes. The following were observed in the renal interstitium:

Interstitial edema (89.4% of cases)

Lymphohistiocytic infiltration (52.7%)

Interstitial fibrosis (34.6%)

Microhemorrhages between peritubular capillaries (41.3%)

Vascular Changes. Significant changes were also identified in renal vessels:

Hyalinosis in arterioles (38.9% of cases)

Fibrinoid necrosis of arterial walls (23.5%)

Swelling and detachment of endothelial cells (67.4%)

Perivascular edema and fibrosis (44.8%)

Correlation Analysis. The degree of renal pathomorphological changes showed positive correlation with stroke severity (NIHSS scale, r=0.72, p<0.001), cerebral ischemic area volume (r=0.68, p<0.001), and systemic inflammation markers (CRP level, r=0.59, p<0.01).

DISCUSSION

The obtained results demonstrated that complex pathomorphological changes occur in the kidneys of patients with ischemic stroke. The development mechanisms of these changes are multifactorial.

First, the systemic inflammatory response activated as a result of ischemic stroke leads to oxidative stress and endothelial dysfunction in renal tissue. Increased proinflammatory cytokines in circulation (IL-1β, IL-6, TNF-α) exert direct toxic effects on renal glomerular and tubular structures.

Second, sympathetic nervous system hyperactivation observed during the acute stroke period leads to decreased renal perfusion, activation of the renin-angiotensin-aldosterone system, and consequently renal ischemia. This explains the ischemic damage to glomerular and tubular epithelium.

Third, disruption of the coagulation system and hypercoagulable state resulting from cerebral ischemia promote the development of thrombotic processes in renal microcirculation. The finding of fibrin aggregates and microthrombi in glomerular capillaries and perivascular zones in our study confirms this.

Fourth, medications administered to patients at high doses after stroke (diuretics, neuroprotectors) may exert additional toxic effects on the kidneys.

Our findings are consistent with results from foreign researchers. Zhang et al. (2021) demonstrated renal tubular necrosis and glomerular damage in mice with ischemic stroke models. Kumar et al. (2020) noted the development of acute kidney injury (AKI) in 40-45% of cases in patients with stroke.

However, our study is the first to comprehensively assess renal pathomorphology in patients with ischemic stroke in the Uzbek population and identify the morphological basis of cerebro-renal syndrome.

CONCLUSION. Ischemic stroke causes complex pathomorphological changes in the kidneys - glomerular, tubular, interstitial, and vascular damage.

The most characteristic morphological changes are: ischemic damage to glomerular capillaries, degeneration and necrosis of tubular epithelium, interstitial edema and inflammation, and microcirculatory disturbances.

The degree of renal pathomorphological changes is directly related to stroke severity and volume, indicating the development of cerebro-renal syndrome.

Regular monitoring of renal function and application of nephroprotective therapy is necessary in patients with ischemic stroke.

Further in-depth study of the pathogenesis of cerebro-renal syndrome and development of effective treatment strategies is warranted.

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