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INVESTIGATION OF MODELS OF UNILATERAL PATHOLOGICAL PROCESSES OF BREACH OF LOCAL RENAL BLOOD FLOW IN ORDER TO DETERMINE CHANGES IN THE STATUS OF THE CONTRALATERAL KIDNEY: IMMUNOMORPHOLOGICAL ASPECTS

The study was conducted as a part of research work “Impaired blood circulation and microcirculation in parenchyma of the remaining kidney after nephrectomy, and the methods for their correction”.

ABSTRACT. Background. Morphology of compensatory-adaptive changes and their dynamics in a single remained kidney are still not clarified. Therefore, the study of renal parenchyma lesions in rat models of the most common pathological states has a great practical importance. **Objective.** To investigate the models of unilateral pathological processes of breach of local renal blood flow (ischemia, infringement of urodynamics) in order to determine changes in the status of the contralateral kidney. **Methods.** In the experiment on white Wistar rats permanent ischemia was modeled by a fixed bending left kidney artery (15 individuals – group A), acute infringement of urodynamics was modeled by a fixed crossing of the left ureter kidney (4 individuals – group B) and both pathological conditions were simulated (5 individuals – group C). Kidneys of 10 relatively healthy rats were used as a control. The primary monoclonal antibodies for CD3 and α SMA (TermoScientific, USA) were used. **Results.** The main pathological manifestations in the contralateral right “healthy” kidneys was in degenerative changes in epithelial tubule cells, foci of edema and sclerosis around large vessels, interstitial and perivascular inflammatory infiltrates containing CD3⁺ T-cells (for A II – 10,3±1,2, for B II – 11,9±1,1, for C II – 10,9±0,8). It differs significantly from the control group ($p < 0,05$), where CD3⁺ T-lymphocytes occurred as single cells of interstitium. **Conclusions.** Unilateral damage of the kidney due to experimental ischemia, or infringement of urodynamics, or both pathological conditions, affecting local renal blood flow, results in pathological changes in the contralateral “healthy” kidney prior to the nephrectomy and suggests the importance of its medicament support.

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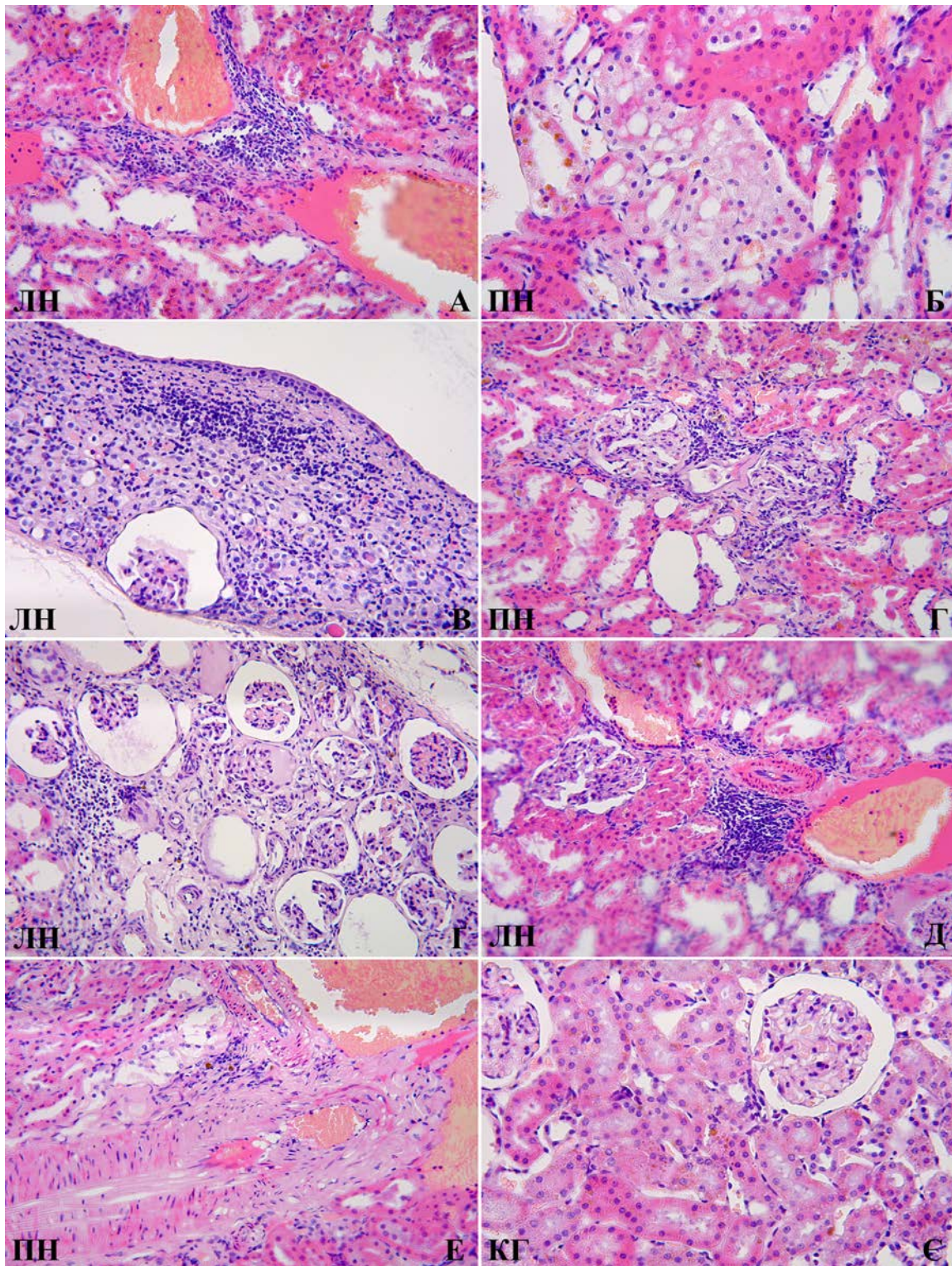


Fig.1. Pathomorphological changes of the rat kidneys. Experimental ischemia: A – full-blooded dilated vessels with inflammatory perivascular infiltrates, focal hemorrhages, Б – marked hydropic dystrophy of the tubular epithelium. Impaired urodynamics: B – hydronephrosis, atrophy of the renal parenchyma with decreased number of glomeruli, inflammatory infiltrates (×200), Г – perivascular thickening of connective tissue and foci of inflammation, dystrophic changes of the tubular epithelium. Impaired blood circulation and urodynamics: Г – sclerotic and atrophic changes of the cortical substance, aggregation of glomeruli, inflammatory infiltrates (×200), Д – perivascular infiltrates around the dilated and full-blooded vessels; E – thickening of the vascular wall – sclerosis, hyalinosis. Control group: € – normal relations of the number of glomeruli and tubuli without pathological changes. ЛН – left kidney, ПН – right kidney, КГ – control group. Hematoxylin&Eosin staining, ×400.

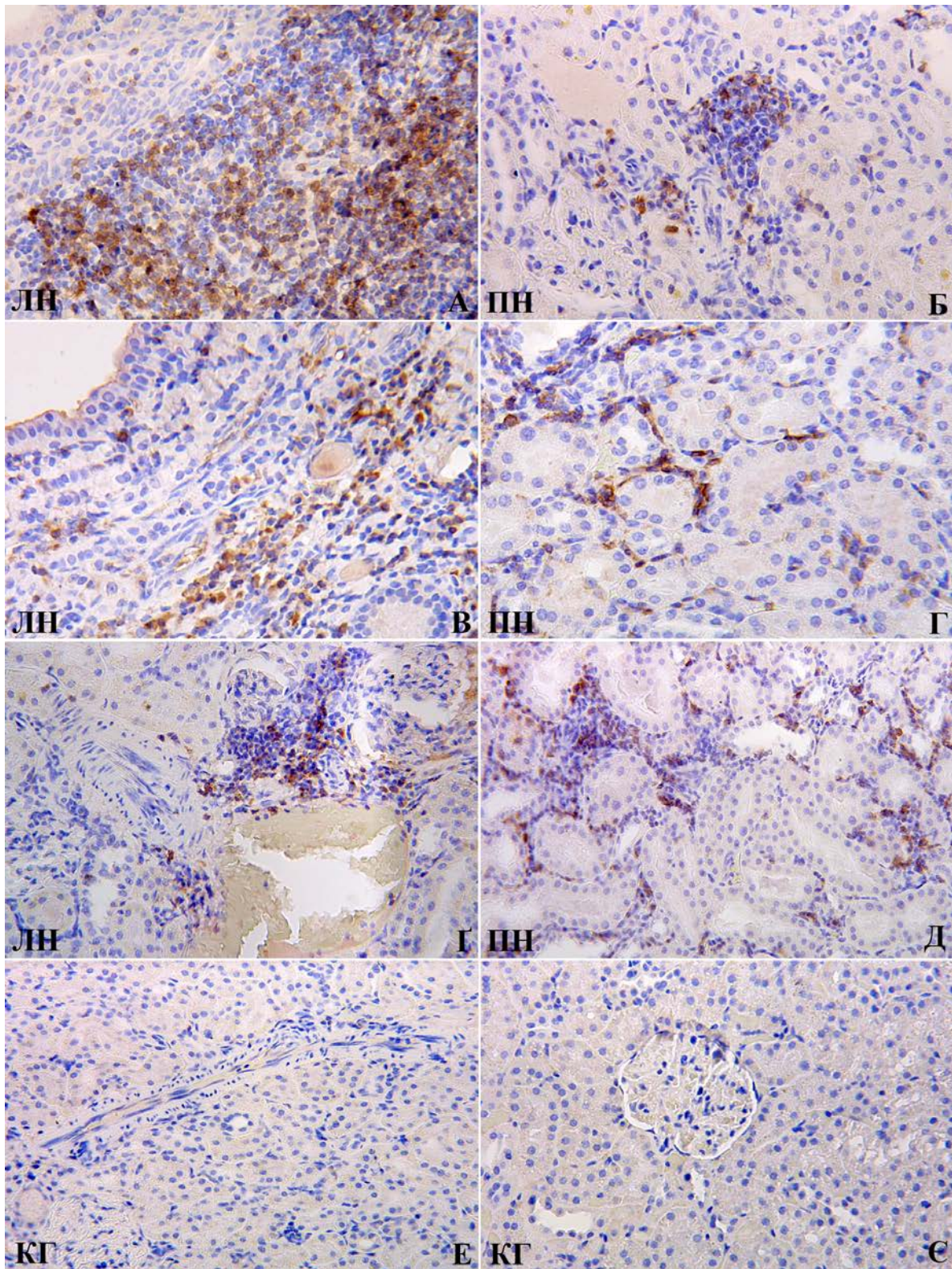


Fig. 2. Expression of CD3 marker on the membranes of T-lymphocytes. Experimental ischemia: А – massive paranephral inflammatory infiltrate, Б – aggregation of T-lymphocytes around vessels. Impaired urodynamics: В – diffuse inflammatory infiltrate in the atrophic parenchyma, Г – peritubular T-lymphocyte infiltrates. Impaired blood circulation and urodynamics: Г – inflammatory infiltrates around the dilated and full-blooded vessels, Д – peritubular T-lymphocyte infiltrates. Control group: Е – absence of the CD3+ T-lymphocytes around vessels, Е – absence of the CD3+ T-lymphocytes around the renal tubuli and glomeruli. ЛН – left kidney, ПН – right kidney, КГ – control group. Immunohistochemical method, additional staining with Mayer's hematoxylin, x400.

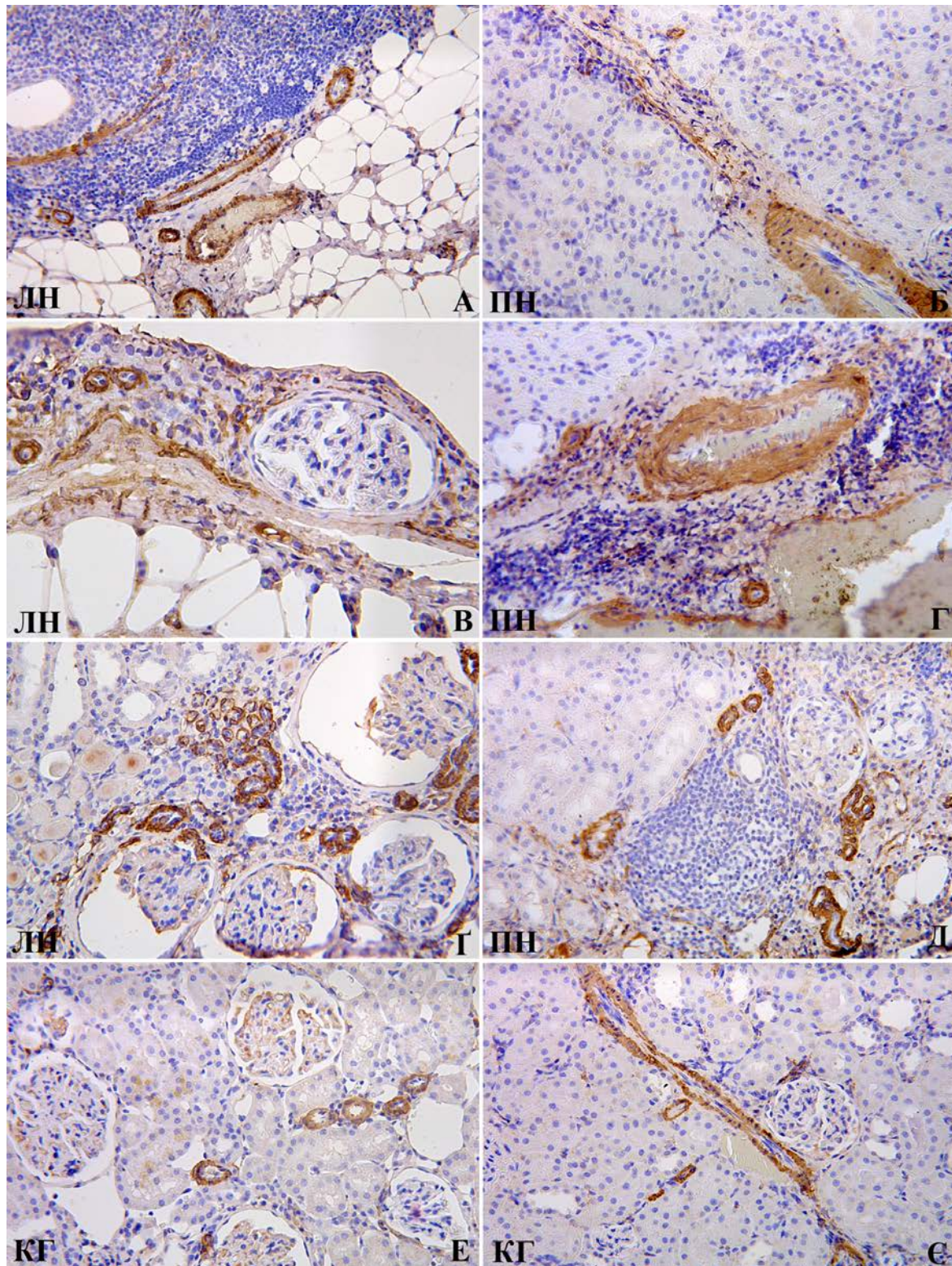


Fig. 3. Expression of α SMA marker in the cytoplasm of smooth muscle cells in the vascular wall. Experimental ischemia: A – full-blooded and dilated vessels around the inflammatory paranephral infiltrate, Б – thickened hypertrophic vascular wall. Impaired urodynamics: B – changes of angioarchitectonics in atrophic parenchyma, Г – thickened hypertrophic vascular wall, inflammatory infiltrate. Impaired blood circulation and urodynamics: Г – increased number of vessels around the aggregation of glomeruli, changes of angioarchitectonics, Д – perivascular infiltrates, dilated and full-blooded vessels. Control group: E – preserved parenchyma structures, regular disposition of vessels, E – absence of dilatation and thickening of vascular wall. ЛН – left kidney, ПН – right kidney, КГ – control group. Immunohistochemical method, additional staining with Mayer's hematoxylin, $\times 400$.

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