Research article

V.P.Stus
K.S.Barannik
I.S.Shponka
O.V.Poslavska

State institution "Dnipropetrovsk medical academy of the Ministry of Health of Ukraine"

Key words: blood circulation of kidneys parenchyma, experimental ischemia, infringement of urodynamics, CD3, αSMA.

Received: 14.11.2014
Accepted: 22.12.2014

UDC 616.61-005-089.168-085

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 αSMA (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.

✉ **kostdok@ukr.net**

Citation:
Stus VP, Barannik KS, Shponka IS, Poslavska OV. [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]. Morphologia. 2014;8(4):43-50. Ukrainian.

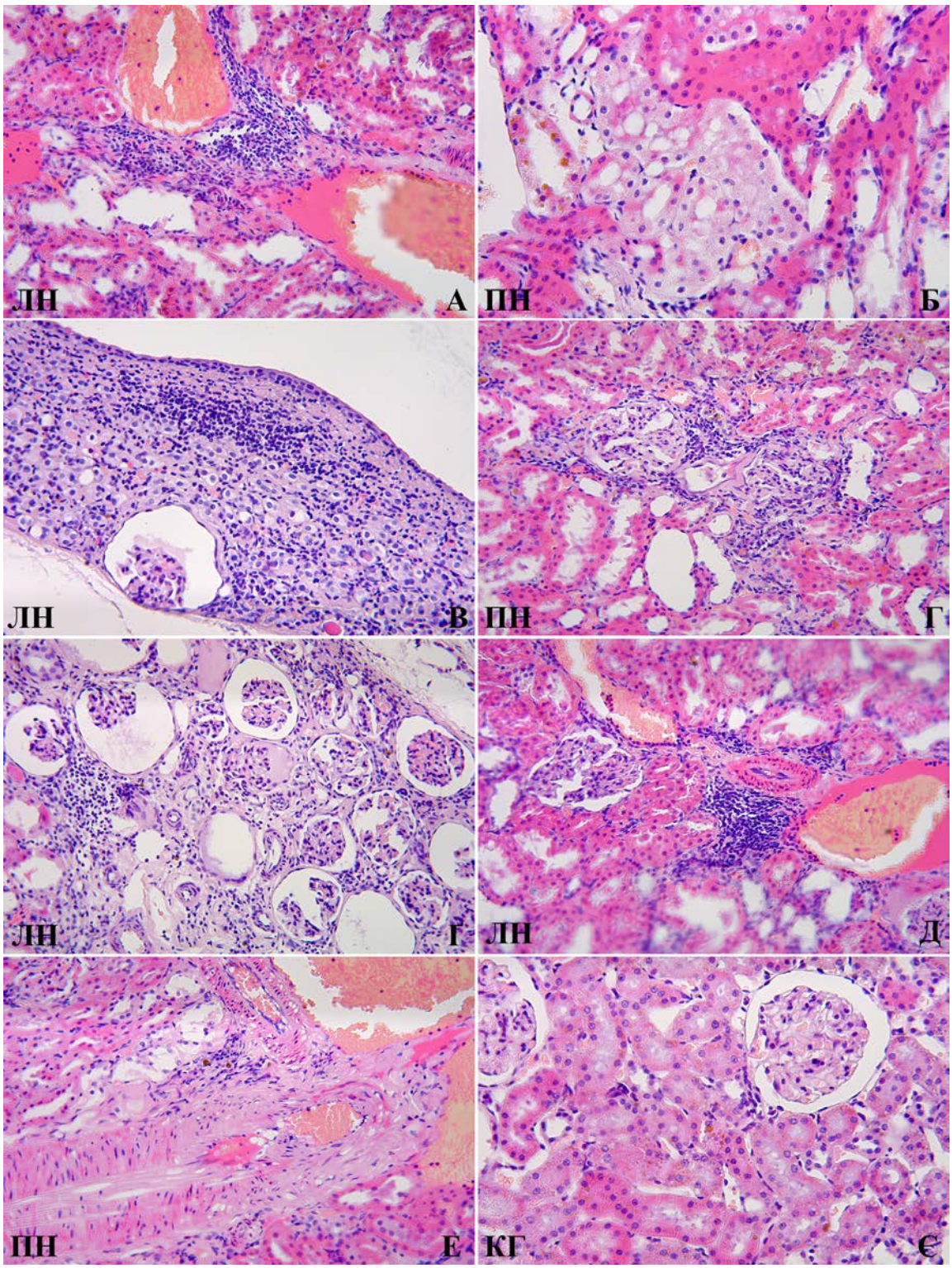

Fig.1. Pathomorhological changes of the rat kidneys. Experimental ischemia: А – full-blooded dilated vessels with inflammatory perivascular infiltrates, focal hemorrhages, Б – marked hydropic dystrophy of the tubular epithelium. Impaired urodynamics: В – hydronephrosis, atrophy of the renal parenchyma 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: Ґ – sclerotic and atrophic changes of the cortical substance, aggregation of glomeruli, inflammatory infiltrates (×200), Д – perivascular infiltrates around the dilated and full-blooded vessels; Е – thickening of the vascular wall – sclerosis, hyalinosis. Control group: Є – 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.

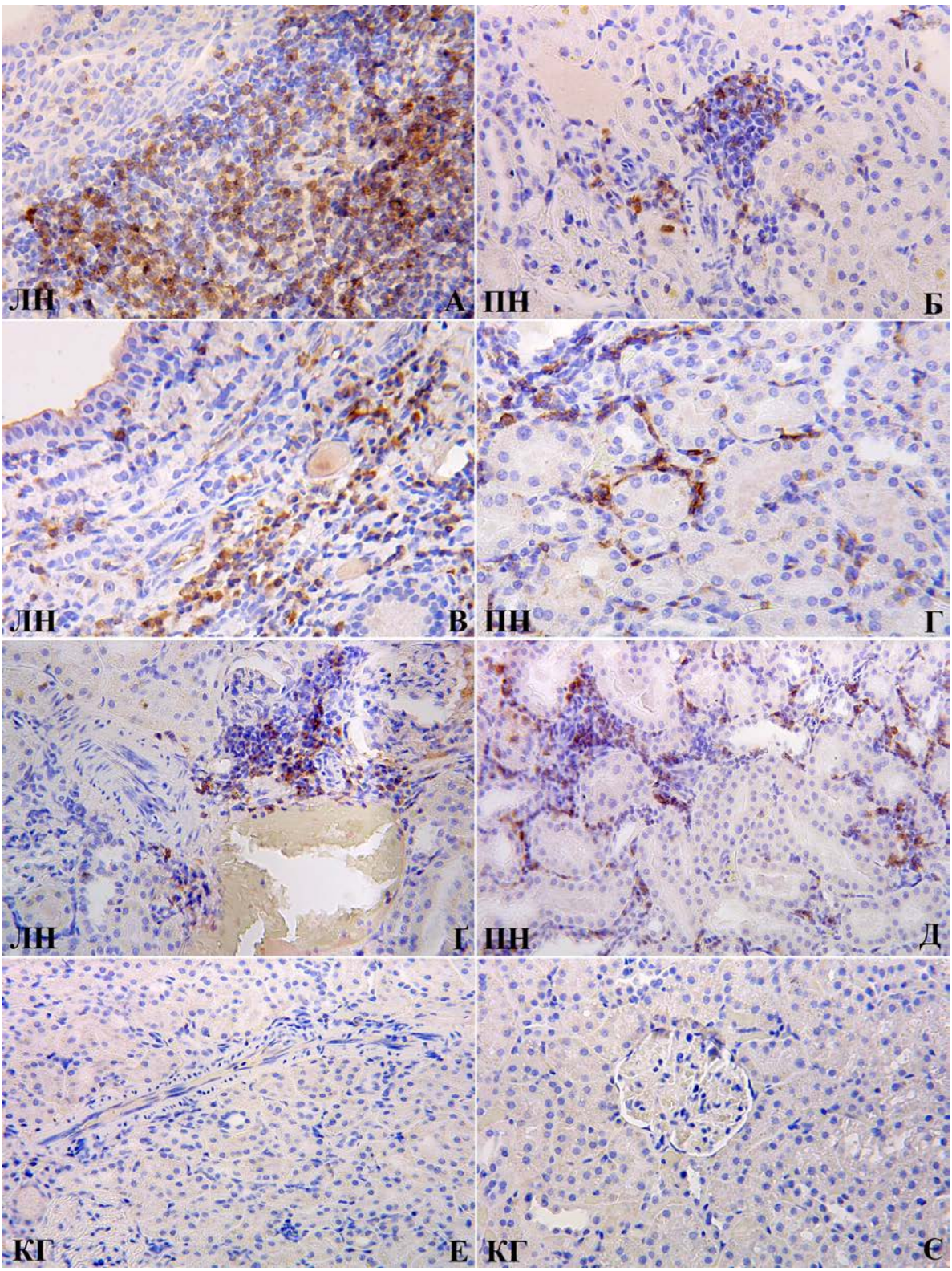

Fig. 2. Expression of CD3 marker on the membranes of T-lymphocytes. Experimental ischemia: А – massive paranephral inflammatory infiltrate, Б – aggregation of T-lymphocytes around vessels. Impaired urodynamics: В – diffuse inflammatory infiltrate in the atrophic parenchyma, Г – peritubular T-lymphocyte infiltrates. Impaired blood circulation and urodynamics: Ґ – 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, Є – 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, ×400.

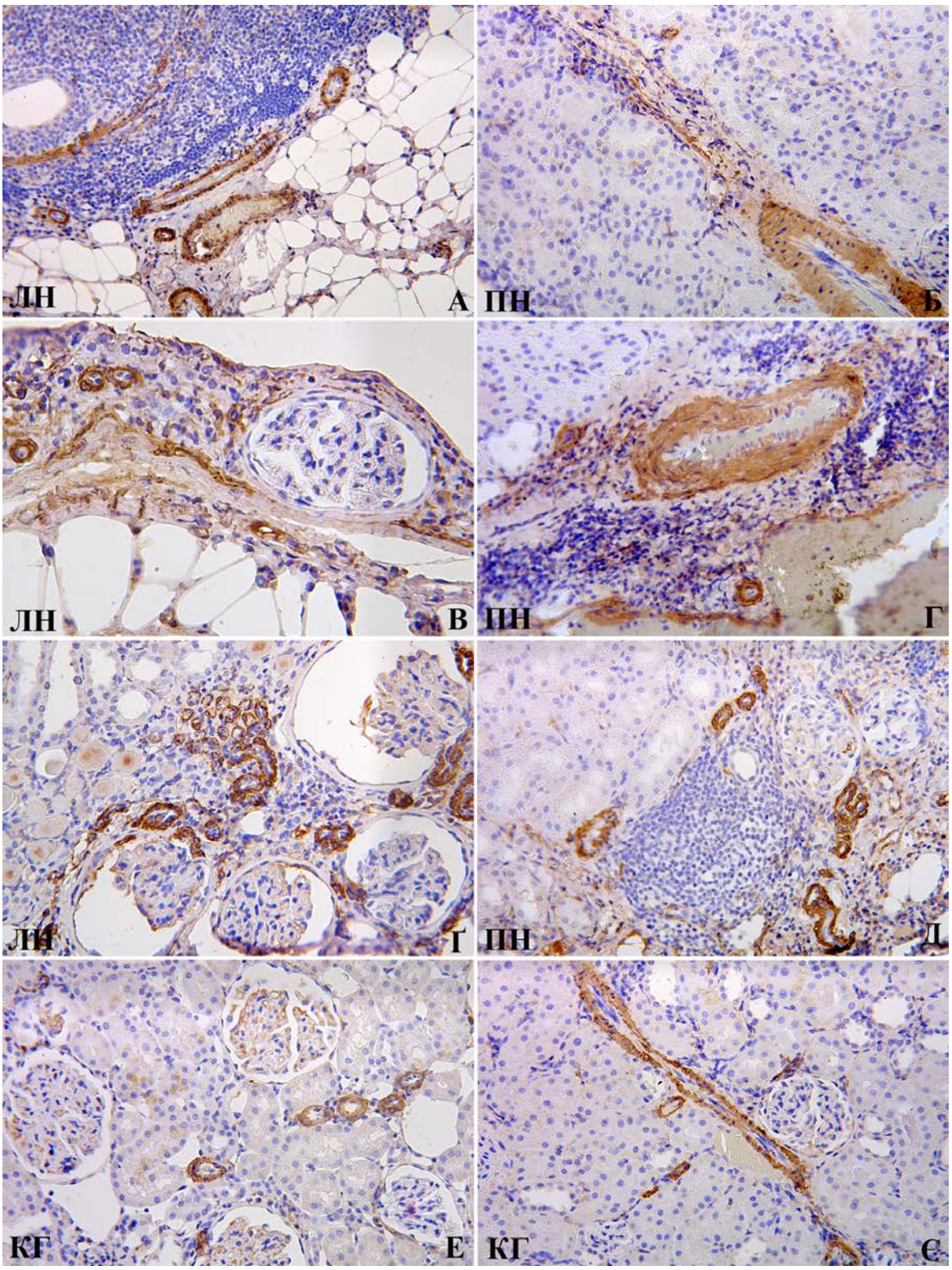

Fig. 3. Expression of αSMA marker in the cytoplasm of smooth muscle cellsin the vascular wall. Experimental ischemia: А –full-blooded and dilated vessels around the inflammatory paranephral infiltrate, Б – thickened hypertrophic vascular wall. Impaired urodynamic: В – changes of angioarchitectonics in atrophic parenchyma, Г – thickened hypertrophic vascular wall, inflammatory infiltrate. Impaired blood circulation and urodynamics: Ґ – increased number of vessels around the aggregation of glomeruli, changes of angioarchitectonics, Д – perivascular infiltrates, dilated and full-blooded vessels. Control group: Е – preserved parenchyma structures, regular disposition of vessels, Є – 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, ×400.

References:

1. Lyulko OV, Pidhirny YaM, Barannik SI. [Pathomorphologic changes of kidneys during ischemia]. Medical perspectives. 2006;11(3):16-25. Ukrainian.

2. Lul'ko OV, Barannik SI, authors. Zmini mikrocirkulatornogo rusla ta ATF-asnoi aktivnosti parenhimi nirok pri experementalniy ishemii [Changes in microcirculation and ATPase activity in experimental ischemia of the one kidney parenchyma] Urology. 2005;4:22-7. Ukrainian.

3. Marchenko TV, Morozov YA, Doletskaya LG, authors. [Functional status of solitary kidney after nephrectomy in living kidney donors of related kidney: is all that simple?] Pochki. 2014;(1):14-7. Russian.

4. Petersen RO, Sesterhenn IA, Davis ChJ. Urologic Pathology. 3rd ed. Washington, USA: Lippincott Williams & Wilkins; 2009. 612 p.

5. Bostwick DG, Cheng L. Urologic Surgical Pathology. 2nd ed. Washington, USA: MOSBE El-sevier; 2008. 1026 p.

6. Fogo AB, Cohen AH, Jennette JCh, Bruijn JA, Colvin RB. Fundamentals of Renal Pathology. New York, USA: Springer; 2006. 221 p.

7. Howie AJ. Handbook of Renal Biopsy Pathology. 2nd ed. New York, USA: Springer; 2008. 337 p.