TO THE ORIGINS OF RENOPROTECTIVE IN CHRONIC KIDNEY DISEASE: PAST AND PRESENT OF THE RENIN-ANGIOTENSIN SYSTEM

Authors: [1]*M. Ivanova,* [2]*O. Vetchinnikova,* [2]*A. Zulkarnaev*

Hospital: [1] First Moscow State Medical University by I.M. Sechenov.
[2] Moscow Regional Research and Clinical Institute, Russian Federation. Surgical department of transplantation and dialysis

The history of the renin-angiotensin system (RAS) has for 120 years. In 1897 the Scandinavian physiologies from the University of Stockholm R. Tigerstedt and his assistant P. G. Bergmann, found that intravenous administration of the renal extract causes an increase in blood pressure in rabbits. They proved that the kidneys produce soluble protein – they called it renin – which is responsible for the increase in pressure. R. Tigerstedt, reported on their summer opening at an international conference in Moscow, but the report has not caused interest. The Professor stopped his studies, and P.G. Bergman began to practice medicine. The scientific world forgot about the opening of the first element of the RAS - renin almost 40 years.

In 1934, a Canadian scientist who has worked in California, H. Goldblatt, published their findings in the Journal of Experimental Medicine. He caused hypertension in dogs by clamping the renal artery, but the clamping of the splenic and femoral arteries did not give such effect. Like their Scandinavian predecessors H. Goldblatt concluded: ischemia of the kidneys stimulates the secretion of "special" substances with vasoconstrictor action, but this idea, as in the case of R. Tigerstedt and P. G. Bergman, aroused only skepticism.

After a year, two independent groups of researchers – first under E. Braun-Menendez in the Medical School of the University Buenos Aires (Argentina), the other under the leadership of I. H. Page from Eli-Lilly Laboratories in Indianapolis (USA) repeated the experiment G. Goldblatt and demonstrated renal secretion of a pressor agent similar to renin. In the following years, both groups of researchers isolated a new substance from the venous blood of the ischemic kidney. It was a peptide of eight amino acids, which Argentinean researchers called it angiotonin, American researcher's – hypertensin. Twenty years later, scientists came to the conclusion about the uniform nature of the obtained substance and called it angiotensin.

Later L. Skeggs and his colleagues established the existence of two forms of angiotensin, and S. H. Ferreira highlighted the angiotensin-converting enzyme. So was open RAS, which are known at the present time, not only modulates arterial hypertension, but also affects the target organs, including the kidneys.

The next step in the development of the doctrine of the RAS was the creation of the means of its pharmacological blockade. There were modern drugs that inhibit various parts of the RAS. The first representative was saralasin – receptor blocker to angiotensin II intravenous not found wide clinical application. A few years later developed the first drug class of angiotensin-converting enzyme inhibitors (captopril), after a decade and a half – non-peptide blocker of the receptors to angiotensin II (losartan), and later – indirect renin inhibitor (aliskiren).

RAS plays an important role in the progression of chronic kidney disease, including renal transplant recipients, and its pharmacological blockade exerts renal protection effect and prevents lethal complications.

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