FEATURES OF CHANGES IN LIPID AND CARBOHYDRATE METABOLISM IN PATIENTS WITH CORONARY HEART DISEASE AND DIABETES MELLITUS RENAL DYSFUNCTION IN PATIENTS WITH ISCHEMIC HEART DISEASE: CURRENT PERSPECTIVES

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Abstract. Chronic kidney disease (CKD) is detected in 20–40% of patients with ischemic heart disease (IHD) and increases the risk of cardiovascular death by

1.5–2 times independently of traditional risk factors. Pathogenetic mechanisms of cardiorenal interactions include hemodynamic disturbances, endothelial dysfunction, systemic inflammation, oxidative stress, accumulation of uremic toxins (ADMA), and vascular calcification. The presence of CKD worsens prognosis in stable angina, acute coronary syndrome, and after revascularization. Modern therapy with RAAS inhibitors, statins, SGLT2 inhibitors, and mineralocorticoid receptor antagonists (finerenone) has been shown to slow CKD progression and reduce cardiovascular risk.

Key words: ischemic heart disease, chronic kidney disease, cardiorenal syndrome, eGFR, resistive index, SGLT2 inhibitors.

The purpose: To analyze current data on the role of renal dysfunction in patients with ischemic heart disease, elucidate the pathogenetic mechanisms of cardiorenal interactions, and evaluate diagnostic approaches and therapeutic strategies for improving prognosis.

Materials and methods. A systematic analysis of scientific literature published over the past five years was conducted using PubMed, Scopus, and Web of Science databases. The review included epidemiological studies, clinical trials, meta-analyses, and current clinical guidelines on cardiorenal interactions in patients with IHD.

Results. Chronic kidney disease affects 20–40% of IHD patients and independently increases cardiovascular mortality by 1.5–2 times. The key pathogenetic mechanisms include: (1) hemodynamic disturbances—reduced cardiac output, RAAS activation, and venous congestion leading to glomerular hypertension and sclerosis; (2) endothelial dysfunction—impaired NO bioavailability, elevated endothelin-1, and ADMA accumulation; (3) systemic inflammation and oxidative stress—elevated IL-6, TNF-α, and ROS production;

(4) mineral metabolism disorders—hyperphosphatemia, vitamin D deficiency, and vascular calcification; and (5) anemia and hemostatic disturbances.

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