MYOCARDIAL INFARCTION IN PATIENTS WITH CHRONIC KIDNEY DISEASE: CLINICAL, LABORATORY AND INSTRUMENTAL CORRELATIONS

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ABSTRACT

Myocardial infarction (MI) is one of the most severe and life-threatening forms of ischemic heart disease, caused by an acute and significant disruption of coronary blood flow that leads to irreversible necrosis of the myocardium. Despite notable advances in cardiology, including the development of modern reperfusion strategies, potent antithrombotic therapies, and improved intensive care management, MI remains a leading cause of mortality and long-term disability across the globe. The burden of MI is particularly critical in patients with concomitant systemic disorders such as chronic kidney disease (CKD), which considerably worsens the course of the disease and negatively affects both short-term and long-term outcomes.

Introduction: Myocardial infarction (MI) is one of the most severe and life-threatening forms of ischemic heart disease, caused by an acute and significant disruption of coronary blood flow that leads to irreversible necrosis of the myocardium. Despite notable advances in cardiology, including the development of modern reperfusion strategies, potent antithrombotic therapies, and improved intensive care management, MI remains a leading cause of mortality and long-term disability across the globe. The burden of MI is particularly critical in patients with concomitant systemic disorders such as **chronic kidney disease (CKD)**, which considerably worsens the course of the disease and negatively affects both short-term and long-term outcomes.

CKD is a progressive, multifactorial pathological process characterized by a sustained decline in glomerular filtration rate (GFR) and structural as well as functional abnormalities of the kidneys. The deterioration of renal function activates a complex cascade of neurohormonal and metabolic mechanisms — including the renin–angiotensin–aldosterone system (RAAS), sympathetic overactivity, endothelial dysfunction, and chronic inflammation — which collectively contribute to vascular injury, cardiac remodeling, and atherosclerosis. These mechanisms not only accelerate cardiovascular aging but also increase susceptibility to ischemic events. Epidemiological studies have established that patients with CKD are three to five times more likely to develop cardiovascular disease than individuals with normal renal function.

When myocardial infarction occurs in patients with impaired renal function, the clinical course tends to be more severe and refractory to standard therapy. Such patients frequently exhibit atypical or masked symptoms, delayed diagnosis, and limited tolerance to reperfusion and pharmacological treatments. The coexistence of MI and CKD results in higher rates of heart failure, arrhythmias, recurrent ischemia, and mortality. Furthermore, impaired renal clearance alters drug metabolism, often necessitating individualized treatment regimens and more careful hemodynamic monitoring.

In this context, a comprehensive understanding of the **pathophysiological interconnections between MI and CKD** is essential for developing more effective diagnostic, prognostic, and therapeutic strategies. Identifying early markers of cardiorenal interaction could enable timely intervention and improve both cardiac and renal outcomes.

Objective: To perform a comparative assessment of clinical, laboratory, and instrumental parameters in patients with acute myocardial infarction with and without concomitant chronic kidney disease. Specific attention was given to evaluating the frequency of heart failure, serum troponin I levels, glomerular filtration rate (GFR), and arterial blood pressure in order to detect early manifestations of the cardiorenal syndrome and enhance the efficiency of clinical monitoring and management.

Materials and Methods: A retrospective clinical-laboratory study was conducted, including **60 patients aged 30 to 75 years** with a confirmed diagnosis of acute myocardial infarction. Based on renal function, the patients were divided into two equal groups:

- **Group 1 (n = 30):** Patients with MI and preserved renal function (without CKD).
- **Group 2 (n = 30):** Patients with MI and previously diagnosed CKD.

All subjects underwent a standardized diagnostic protocol, which included:

- Complete blood count and urinalysis;
- Comprehensive biochemical testing (serum creatinine, uric acid, and troponin I);
- Calculation of estimated GFR using the **CKD-EPI formula**;
- Instrumental studies such as **electrocardiography (ECG)**, **echocardiography (EchoCG)**, and **renal ultrasound** to evaluate cardiac and renal morphology and function.

Statistical analysis was carried out to compare quantitative and categorical variables between groups. Mean values were expressed as mean ± standard deviation (SD)

Result:

Clinical and instrumental findings revealed a significantly more adverse course of myocardial infarction in patients with concomitant CKD.

- **Heart Failure:** Signs of congestive heart failure of functional class II–III (according to NYHA classification) were identified **2.5 times more frequently** in Group 2 compared to Group 1.
- **Blood Pressure:** The mean systolic arterial pressure in CKD patients was **145 ± 12 mm Hg**, whereas patients without CKD demonstrated an average of **132 ± 10 mm Hg**, indicating higher prevalence of hypertension and impaired hemodynamic adaptation.
- **Troponin I:** Serum troponin I concentration was markedly elevated in the CKD group (**3.8 ± 1.2 ng/mL**) relative to the non-CKD group, reflecting more extensive myocardial injury and prolonged ischemic insult.

• **Renal Function:** Mean GFR among CKD patients was **42 ± 9 mL/min**, corresponding to moderate or severe renal impairment.

• **Left Ventricular Function:** Echocardiographic evaluation demonstrated a reduction in left ventricular ejection fraction (LVEF) in CKD patients (**45 ± 7%**) compared to those without CKD (**52 ± 6%**), suggesting decreased myocardial contractility and impaired recovery post-MI.

The overall findings indicate that renal dysfunction aggravates myocardial damage, promotes hypertensive response, and increases the likelihood of post-infarction heart failure.

Discussion: The present study highlights the intricate bidirectional relationship between the heart and kidneys in the setting of acute coronary syndromes. Chronic kidney disease acts not only as a comorbidity but also as an independent risk factor that exacerbates ischemic myocardial injury through several mechanisms — including endothelial dysfunction, oxidative stress, volume overload, and increased myocardial fibrosis. Furthermore, the reduced renal clearance of cardiac biomarkers such as troponin can complicate diagnostic interpretation, necessitating integration of laboratory, clinical, and imaging data.

Patients with MI and CKD represent a particularly vulnerable population that requires **multidisciplinary management** involving cardiologists, nephrologists, and critical care specialists. Early recognition of cardiorenal syndrome, optimal control of hemodynamic parameters, and careful adjustment of pharmacotherapy (especially antiplatelet, anticoagulant, and renin-angiotensin system inhibitors) are essential to improving survival and reducing rehospitalization rates

Conclusion Myocardial infarction in patients with chronic kidney disease is characterized by a more severe clinical course, higher incidence of heart failure, increased troponin levels, and pronounced arterial hypertension. Decline in renal function significantly limits therapeutic options and adversely affects prognosis. Therefore, early detection of cardiorenal interactions and implementation of an **integrated, patient-centered approach** to management are imperative. Collaboration between cardiologists and nephrologists, coupled with continuous clinical and laboratory monitoring, can substantially enhance treatment outcomes and quality of life in this high-risk patient group.

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