

CONTRAST-ASSOCIATED ACUTE KIDNEY INJURY FOLLOWING PERCUTANEOUS CORONARY INTERVENTION: FROM EARLY RENAL INJURY TO LONG-TERM CHRONIC KIDNEY DISEASE

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Background: Contrast-associated acute kidney injury (CA-AKI) is one of the most important renal complications following percutaneous coronary intervention (PCI). Although traditionally considered a transient and reversible decline in renal function, increasing evidence suggests that CA-AKI may represent an early manifestation of persistent renal injury and may contribute to the development or progression of chronic kidney disease (CKD). The risk is particularly relevant in patients with diabetes mellitus, pre-existing renal dysfunction, advanced age, heart failure, anemia, and hemodynamic instability.

Objective: To evaluate the potential role of CA-AKI as an early predictor of long-term renal dysfunction after PCI and to summarize the current understanding of its pathophysiological mechanisms, early biomarkers, and preventive strategies.

Materials and Methods: A review of contemporary clinical and experimental studies concerning CA-AKI after coronary angiography and PCI was performed. Particular attention was paid to the mechanisms of renal tubular injury, including renal medullary hypoxia, oxidative stress, mitochondrial dysfunction, endothelial injury, inflammation, and apoptosis. The diagnostic and prognostic potential of serum creatinine, cystatin C, neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), and interleukin-18 (IL-18) was also assessed.

Results: The development of CA-AKI is considered to result from the interaction of contrast exposure with pre-existing renal and cardiovascular vulnerability. Contrast administration may induce renal vasoconstriction, impair medullary microcirculation, and aggravate tissue hypoxia. These changes are accompanied by increased production of reactive oxygen species, mitochondrial dysfunction, inflammatory activation, and tubular epithelial cell injury.

Serum creatinine remains the conventional marker for diagnosing AKI; however, its relatively delayed increase limits its ability to detect early structural renal injury. In contrast, biomarkers such as NGAL and KIM-1 may provide earlier information regarding tubular damage, while cystatin C may allow earlier detection of changes in glomerular filtration. A combined assessment of functional and structural biomarkers may therefore improve early identification of patients at increased risk of incomplete renal recovery.

An important clinical implication is the transition from **acute kidney injury to acute kidney disease and, in susceptible patients, chronic kidney disease**. Persistent renal dysfunction after CA-AKI may reflect incomplete recovery and loss of renal functional reserve. Therefore, the occurrence of CA-AKI should prompt not only short-term monitoring of serum creatinine but also subsequent assessment of estimated glomerular filtration rate and albuminuria.

Preventive management remains centered on individualized risk assessment, adequate volume management, minimization of contrast volume, maintenance of hemodynamic stability, and

avoidance of unnecessary nephrotoxic exposure. Particular attention should be given to high-risk patients undergoing complex or repeated PCI procedures.

Conclusion: CA-AKI following PCI should be regarded not merely as a short-term procedural complication but as a potential marker of long-term cardiorenal risk. The combination of renal hypoxia, oxidative stress, mitochondrial dysfunction, inflammation, and tubular injury may contribute to incomplete renal recovery and subsequent CKD progression. Early biomarkers such as NGAL, KIM-1, and cystatin C may complement serum creatinine in identifying subclinical renal injury, although their routine clinical implementation requires further validation. An integrated strategy combining pre-procedural risk stratification, contrast minimization, individualized hydration, hemodynamic optimization, and post-procedural renal follow-up may reduce the risk of persistent kidney dysfunction.