





# Impact of SGLT2 inhibitors on contrast-induced acute kidney injury in patients undergoing percutaneous coronary interventions

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**KEYWORDS:** SGLT2 inhibitors, contrast-induced acute kidney injury, percutaneous coronary intervention.

**CITATION:** *Cardiol Croat.* 2024;19(3-4):118. | <https://doi.org/10.15836/ccar2024.118>

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Sodium-glucose cotransporter 2 inhibitors (SGLT2-I) are antihyperglycemic drugs that improve cardiovascular and renal outcomes in patients with or without type 2 diabetes.<sup>1,2</sup> Under certain circumstances, SGLT2 inhibition could potentially lead to significant renal impairment, like dehydration or renal parenchymal hypoxia and hypoxic kidney injury. The former is caused by osmotic diuresis and natriuresis mostly in patients on diuretics and the latter could be induced by SGLT2-I-mediated medullary hypoxia and might be clinically significant under specific conditions such as the use of non-steroidal anti-inflammatory drugs (NSAIDs) or radiocontrast agents.<sup>3</sup> Contrast-induced acute kidney injury (CI-AKI) is a possible complication of patients undergoing percutaneous coronary intervention (PCI).<sup>1,2</sup> Several clinical trials showed the effect of SGLT2-I on the development of contrast-induced nephropathy. Retrospective study (N = 1,510) showed that the incidence of CI-AKI in patients with type 2 diabetes (T2D) with coronary artery disease undergoing percutaneous coronary interventions (PCI) is lower in SGLT2-I users.<sup>1</sup> Another observational study enrolled patients from the SGLT2-I AMI PROTECT Registry (N=646), patients with T2D admitted with acute myocardial infarction (AMI) on chronic SGLT2-I therapy versus non-SGLT2-I users treated with PCI, with or without chronic kidney disease (CKD). The main finding was that in T2D patients with AMI, the use of SGLT2-I was associated with a lower risk of CI-AKI during hospitalization, mostly in patients without CKD.<sup>2</sup> Both studies identified the use of SGLT2-I as an independent predictor of reduced rate of CI-AKI. One smaller study that included patients with T2D on SGLT2-I therapy with non-ST segment elevation myocardial infarction underwent coronary angiography (CAG) and/or PCI also showed a significantly lower risk of CK-AKI in T2D patients on SGLT2-I therapy.<sup>4</sup> In these studies T2D patients had been treated with SGLT2-I for at least 3-6 months before PCI. To examine a possible use of these drugs as a preventive strategy, further studies should focus on the acute use of SGLT2-I in patients undergoing percutaneous coronary interventions, with or without T2D, considering indication of SGLT2-I for the treatment of chronic heart failure and chronic kidney disease.

**RECEIVED:**  
October 22, 2023

**ACCEPTED:**  
October 27, 2023



## LITERATURE

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