JACC: ASIA VOL. 1, NO. 3, 2021

EDITORIAL COMMENT

Early Detection of Acute Kidney Injury Can Further Improve the Prognosis of Acute Myocardial Infarction*

Hiroshi Asanuma, MD, PhD

Acute kidney injury (AKI) is defined by a rapid increase in serum creatinine (sCr) or decrease in urine output that occurs in serious diseases. ST-segment elevation myocardial infarction (STEMI) is one of the serious diseases that causes AKI. It has been reported that the incidence of AKI in patients with STEMI ranges from 10% to 30% because it varies with the criteria used for diagnosing AKI (1). The mechanisms of AKI in patients with STEMI are multifactorial. Growing evidence indicates that primary percutaneous coronary intervention (PCI) is the most effective treatment of STEMI because this therapeutic approach preserves ventricular function and improves survival of STEMI. However, contrast-induced nephropathy is a possible complication of PCI. Direct toxic effects of contrast media, vasoconstriction, oxidative stress, and medullary ischemia are known as the mechanisms of contrast-induced nephropathy. Cardiologists tend not to take AKI seriously, especially when sCr level remains within the normal range or decreases rapidly; however, AKI is known to have a great impact on in-hospital and long-term prognosis. In fact, early development of AKI (within 48 hours after admission) is an independent predictor of in-hospital mortality in patients with acute myocardial infarction, even once renal function has returned to baseline level (2). Therefore, all patients with STEMI should be received an AKI risk assessment as soon as they arrive at the hospital for the purpose of improving prognosis. From that point of view, several risk score models have been developed to assess the incidence of AKI, and Mehran's score model has been the most widely used risk score for predicting the risk of AKI (3). However, Mehran's score model requires the volume of contrast medium administered and use of a hemodynamic support device, and those are unknown before the procedure. Furthermore, it has been reported that severity and hemodynamic impairment due to STEMI rather than contrast media–induced nephropathy is the key contributor for AKI in STEMI (4). In addition, there is a report that the total amount of contrast medium administered is not related to the development of contrast-induced AKI (5).

In this issue of *JACC: Asia*, Goriki et al (6) developed a risk score prediction model, based on a combination of parameters obtained on routine preprocedural blood tests, for in-hospital AKI in patients with STEMI who underwent primary PCI and to compare the predictive utility of that model with that of conventional Mehran model. To develop the predictive model, a total of 908 patients were ultimately included in this study, and based on the index date of hospital admission, they were divided into 2 groups, the derivation and validation sets, which consisted of 617 patients hospitalized from April 2013 to December 2017 and 291 patients hospitalized from January 2018 to January 2020, respectively. In this study, 7.9% and 9.6% of patients had AKI in the derivation and validation cohorts, respectively. Of these, 6.3% and 7.6%, respectively, had AKI stage 1; 0.8% and 0.7%, respectively, were AKI stage 2; and 0.8% and 1.3%, respectively, were AKI stage 3. First of all, the authors demonstrated that the following 4 laboratory parameters quantified on admission were independently associated with an increased risk of AKI in Japanese patients with STEMI who underwent primary PCI: 1) blood sugar ≥200 mg/dL;

*Editorials published in *JACC: Asia* reflect the views of the authors and do not necessarily represent the views of *JACC: Asia* or the American College of Cardiology.

From the Department of Internal Medicine, Meiji University of Integrative Medicine, Nantan, Japan.

The author attests they are in compliance with human studies committees and animal welfare regulations of the author's institution and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

ISSN 2772-3747 https://doi.org/10.1016/j.jacasi.2021.08.005

2) high-sensitivity troponin I >1.6 ng/mL; 3) albumin ≤3.5 mg/dL; and 4) estimated glomerular filtration rate <45 mL/min/1.73 m^2 in the derivation cohort. Second, when 0 to 4 points were given according to the number of these four laboratory parameters, incremental total risk scores were significantly associated with a higher incidence of AKI in both the derivation cohort and the validation cohort. Third, receiver-operating-characteristic curve analysis of risk models showed adequate discrimination between patients with and without AKI in both the derivation cohort and the validation cohort. When they classified the patients into 3 groups according to risk score to simplify its use in clinical settings: a low-risk group (0 or 1 point), a moderate-risk group (2 points), and a high-risk group (3 or 4 points); these subgroups also showed a significant trend for in-hospital incidence of AKI among the respective validation and derivation cohorts. Fourth, the area under the curve of the laboratory-based model and that applied to Mehran's model in all cohorts was 0.755 (95% confidence interval: 0.696-0.805) and 0.749 (95% confidence interval: 0.681-0.807), respectively, and the predictive ability was similar between models. Finally, when patients in these 3 laboratory risk score subgroups were subdivided into Mehran's 4 risk groups (low, moderate, high, very high) (3), their laboratory-based model could further stratify the risk of AKI in the low-, moderate-, and high-risk subgroups of Mehran's model.

Underlying renal dysfunction is known as the strongest risk factor of AKI in patients with STEMI, but it is more often influenced by multiple risk factors. Not to mention the negative impact of contrast agent used during PCI, the key mechanisms in AKI pathogenesis, including systemic and renal hemodynamic changes secondary to impaired cardiac output and increased venous congestion, lead to decreased glomerular filtration rate. Several neurohormonal systems have significant effects on renal blood flow and renal dysfunction. Changes in volume status, drugs, anemia owing to bleeding, and inflammatory activation also have a negative impact. In fact, several blood biomarkers such as B-type natriuretic peptide (5), admission hyperglycemia (7), C-reactive protein (8), and serum albumin (9) have been reported as potential predictors of AKI in patients with acute myocardial infarction. Several prediction scores of AKI after the PCI have been reported. However, all of those prediction scores require any information such as age, patient's medical history, volume of contrast medium, and use of intra-aortic balloon pump, and some of them are somewhat complicated to calculate in an emergency clinical setting. In this study, the authors succeeded in developing a risk score prediction model that can predict the subsequent AKI with only 4 laboratory parameters (blood sugar, high-sensitivity troponin I, albumin, and estimated glomerular filtration rate) quantified on admission in patients with STEMI. Very importantly and interestingly, all 4 of these laboratory parameters have been reported as predictors of the development of AKI after the PCI or are included in the risk score prediction model. Although the Kidney Disease: Improving Global Outcomes guidelines include urine volume as part of the diagnosis of AKI, urine volume was not taken into account in this study. Therefore, the incidence of AKI is somewhat lower than previously reported. This study is meaningful, and future prospective clinical studies with large sample sizes for patients with STEMI are largely warranted.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The author has reported that he has no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Hiroshi Asanuma, Department of Internal Medicine, Meiji University of Integrative Medicine, Hiyoshi-cho, Nantan 629-0392, Japan. E-mail: h_asanuma@meiji-u.ac.jp.

REFERENCES

1. Marenzi G, Cosentino N, Bartorelli AL. Acute kidney injury in patients with acute coronary syndromes. *Heart*. 2015;101(22):1778–1785.

2. Moriyama N, Ishihara M, Noguchi T, et al. Early development of acute kidney injury is an independent predictor of in-hospital mortality in patients with acute myocardial infarction. *J Cardiol*. 2017;69(1):79–83.

3. Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol*. 2004;44(7):1393–1399.

4. Schmucker J, Fach A, Becker M, et al. Predictors of acute kidney injury in patients admitted with ST-elevation myocardial infarction - results from the Bremen STEMI-Registry. *Eur Heart J Acute Cardiovasc Care*. 2018;7(8):710–722.

5. Jarai R, Dangas G, Huber K, et al. B-type natriuretic peptide and risk of contrast-induced acute kidney injury in acute ST-segment-elevation myocardial infarction: a substudy from the HORIZONS-AMI trial. *Circ Cardiovasc Interv*. 2012;5(6):813–820.

6. Goriki Y, Tanaka A, Nishihira K, Kuriyama N, Shibata Y, Node K. A novel prediction model of acute kidney injury based on combined-blood variables in STEMI. *JACC: Asia*. 2021;1:372–381.

7. Moriyama N, Ishihara M, Noguchi T, et al. Admission hyperglycemia is an independent predictor of acute kidney injury in patients with acute myocardial infarction. *Circ J*. 2014;78(6):1475–1480.

8. Gao F, Zhou YJ, Zhu X, et al. C-reactive protein and the risk of contrast-induced acute kidney injury in patients undergoing percutaneous coronary intervention. *Am J Nephrol*. 2011;34(3):203–210.

9. Murat SN, Kurtul A, Yarlioglues M. Impact of serum albumin levels on contrast-induced acute kidney injury in patients with acute coronary syndromes treated with percutaneous coronary intervention. *Angiology*. 2015;66(8):732–737.

KEY WORDS acute kidney injury, biomarker, myocardial infarction