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Editorial

Volume Status, a Novel Marker of Contrast Induced Acute Kidney Injury in Acute Heart Failure Undergoing Coronary Angiography?

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▸ See the article "Incidence, Risk Factors and Prognosis of Contrast-Induced Acute Kidney Injury in Acute Heart Failure Patients Undergoing Coronary Angiography" in volume 1 on page 72.

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Heart and kidney orchestrate each other to maintain homeostasis in hemodynamics. If one is compromised, the other follows, which is now described as cardio-renal syndrome.[1] Diuretics are key medications in treating patients with heart failure (HF) because they are in danger of volume overload. However, too much diuretics increase the risk of renal hypoperfusion. In acute heart failure (AHF), ischemic heart disease is a leading cause and the recent guideline recommends prompt evaluation of coronary artery.[2] During coronary angiography (CAG), patients with AHF should be monitored cautiously because forward and/or backward failure and routine administration of diuretics may augment the risk of contrast induced acute kidney injury (CI-AKI). Although volume expansion dramatically reduces the risk of CI-AKI,[3] pulmonary congestion and peripheral edema in patients with AHF limit the use of volume expanders including saline or bicarbonate fluid. Therefore, factors associated with CI-AKI in patients with AHF undergoing CAG need to be evaluated.

Park et al.[4] analyzed 594 patients with AHF undergoing CAG who were enrolled from 2011 to 2013 in three tertiary hospitals. Mean age of the enrolled patients were 69.9±12.9 years, 40% had diabetes, 50% had chronic kidney disease, and 80% had pulmonary congestion. These were typical conditions where the risk of CI-AKI development was very high, but adequate hydration could be harmful to be provided. Authors reported that the overall incidence of CI-AKI in AHF was 24.7%. To the best knowledge, there have been no similar studies to be compared because most previous studies analyzed patients with stable HF undergoing CAG with adequate hydration, and the reported incidence of CI-AKI in these patients was 11–15%.[5-8] Unfortunately, authors did not provide results regarding on hydration during CAG. Therefore, we cannot conclude whether higher incidence of CI-AKI in the present study was due to decompensated background itself or paucity of hydration. Nonetheless, authors successfully confirmed that baseline kidney function and contrast dye volume were independently associated with CI-AKI in AHF. However, they failed to confirm the well-known risk factors including age, diabetes, anemia, and so forth were associated with CI-AKI development. Whether these discordant results were due to different clinical conditions or a simple coincidence needs to be re-evaluated in the next studies.

Park et al.[4] strongly suggested that the deviation of body weight from dry weight (ΔBWT) may be a novel marker of subsequent development of CI-AKI in patients with AHF. Authors identified that the relationship between ΔBWT and the risk of CI-AKI was U-shaped. According to the feature of ROC curve, they arbitrarily defined three distinct zones: weight excess zone, weight optimum zone, and weight deficiency zone. As expected, increased ΔBWT was not associated with CI-AKI in patients with weight optimum zone and decreased ΔBWT was associated with increased risk of CI-AKI in patients with weight deficiency zone. Interestingly, however, increased ΔBWT was independently associated with increased risk of CI-AKI in patients with weight excess zone. One possible explanation would be the paucity of hydration in patients with weight excess zone. If accept this hypothesis, meticulous hydration in AHF might attenuate the risk of CI-AKI. In weight excess zone, the risk of CI-AKI development was more evident in group with heart failure with preserved ejection fraction (HFpEF) than those with HFrEF, as ΔBWT increased (Supplementary Figure 4D). This might mean that decreased renal perfusion caused by diastolic HF might also be the other explanation of the risk of increased ΔBWT for CI-AKI in weight excess zone. If accept the hypothesis, aggressive weight squeezing with diuretics or delaying CAG procedure until the optimal weight is achieved might reduce the risk of CI-AKI. Therefore, I hope subsequent interventional studies are conducted to validate the hypotheses.

Park et al.[4] also explored the hazard of CI-AKI on all-cause mortality in patients with AHF. When compared to patients without CI-AKI, not only patients with persistent CI-AKI, but also patients with CI-AKI recovery were associated with increased risk of future all-cause death, accentuating the importance of prevention of CI-AKI development in patients with AHF. Authors reported that, when compared to patients with weight optimum zone, patients with weight deficiency zone were in danger with future all-cause death, suggesting the potential hazard of initial over-decongestion. These results remind me of a proverb "Mend the barn after the horse is stolen." Although a barn can be fixed and new horses may occupy the barn, a trace of CI-AKI in patients with AHF is not erasable. Therefore, extra-cautions are required to prevent the development of CI-AKI in patients with AHF. Meticulous assessment of initial volume status before CAG will help to reduce the risk of CI-AKI development in patients with AHF undergoing CAG.

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