Kidney injury rose along with for contrast-induced acute kidney injury, according to a large, retrospective analysis study.

“Hyperglycemic patients without known diabetes should be recognized as a high-risk group for contrast-induced acute kidney injury and should be considered for prophylactic measures similar to those used in other high-risk patients,” wrote Dr. Joshua M. Stolker of the Mid American Heart Institute of Saint Luke’s Hospital, Kansas City, Mo., and colleagues (J. Am. Coll. Cardiol. 2010;55:1433-40).

The study is the first to document an increasing risk of CI-AKI with progressive blood glucose elevations in patients who do not have diabetes, Dr. Martin A. Alpert and Dr. Carl Carlson of the division of cardiovascular medicine, University of Missouri, Columbia, noted in an editorial (J. Am. Coll. Cardiol. 2010;55:1441-3).

“Hyperglycemia occurs in more than 40% of patients without diabetes with acute myocardial infarction. In the critically ill population, hyperglycemia may be seen as a ‘stress test’ denoting the failure of endogenous insulin reserves to adequately control blood glucose.”

The study analyzed 6,358 consecutive patients from the Health Facts database who underwent coronary angiography after acute MI. Of them, 1,929 (30%) had known diabetes. Preprocedural hyperglycemia (blood glucose at least 140 mg/dL) was present in 42% of the entire cohort, of whom 48% were nondiabetic. All patients were stratified according to their preprocedural blood glucose level: less than 110 mg/dL; 110 to less than 140 mg/dL; 140 to less than 170 mg/dL; 170 to less than 200 mg/dL; and 200 mg/dL or more.

After coronary angiography, 823 patients (13%) developed CI-AKI (an absolute serum creatinine increase of 0.3 mg/dL or more, or a relative increase in serum creatinine of 50% or more within 48 hours of the procedure), the primary study end point. After adjustment for confounders, there was a strong association between preprocedural glucose levels and CI-AKI risk in patients without diabetes, but not in patients with established diabetes—regardless of their glucose levels, reported the authors.

Among the nondiabetic patients, the risk for CI-AKI increased with increasing glucose levels. Compared with patients with blood glucose levels below 100 mg/dL (reference), those in the higher glucose categories had increasingly higher risks for CI-AKI, with odds ratios of 1.31, 1.51, 1.58, and 2.14, all significant differences. This pattern was not seen in diabetic patients (OR 0.71, 0.82, 0.73, 0.94).

Nondiabetic, hyperglycemic acute MI patients may receive less aggressive glucose control than their diabetic counterparts, and may also receive less aggressive CI-AKI prophylaxis, the authors said. Additionally, some hyperglycemic patients may have undiagnosed and untreated diabetes, putting them at higher risk. Also, nondiabetic patients who become hyperglycemic may be experiencing more severe illness compared with diabetes patients who become hyperglycemic.

The results identify a new risk marker and “raise the question of whether interventions such as intensive insulin therapy might reduce risk in this population,” noted the editorialists.

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