Acute kidney injury (AKI) is a devastating clinical syndrome that occurs commonly in hospitalized patients, particularly in patients with chronic comorbidities (chronic kidney disease, congestive heart failure, etc) suffering from major ischemic, inflammatory, or nephrotoxic insults during acute illnesses. Early identification of patients at increased risk of AKI may improve the primary prevention, early detection, and prompt therapy of AKI. Most scores to predict AKI risk focus on critically ill patients in the intensive care unit (ICU) (who have the highest risk and the worst outcomes) or specific populations undergoing potentially nephrotoxic exposures (e.g., iodinated radiocontrast and cardiac surgery). Comparatively little attention has been paid to AKI risk factors and prediction tools for patients hospitalized outside the ICU.

In this issue of Mayo Clinic Proceedings, Safadi et al report the development and initial validation of a risk factor–based model to predict AKI in patients hospitalized outside the ICU. They retrospectively used the medical records of (local) Olmsted County residents who were admitted to Mayo Clinic in Rochester, Minnesota, in 2013 and 2014. They excluded patients who were initially admitted in an ICU; other exclusions included pediatric and psychiatric admissions, as well as patients with end-stage kidney disease (including kidney transplant recipients), those with AKI on admission, or those missing baseline or admission serum creatinine values. The primary end point was the development of hospital-acquired AKI, defined and staged by the Kidney Disease: Improving Global Outcomes classification system. Acute kidney injury cases were identified with a previously validated electronic alert system ("AKI sniffer"). The development cohort included 3816 patients who were admitted in 2014, and the validation cohort included 3232 patients admitted in 2013. Acute kidney injury developed in 10.3% of the development cohort, including moderate (stage 2) or severe (stage 3) AKI in 1.9% of the cohort. The mortality rate was 13.2%, higher in those with AKI (33%) than in those without AKI (11%). Acute dialysis initiation occurred in 0.4% of the development cohort. Characteristics and outcomes of the validation cohort were similar.

In the development cohort, univariate and multivariate logistic regression analyses were used to identify risk factors that were independently associated with any stage of AKI or with moderate-severe AKI. The final model, with an area under the receiver operating characteristic curve (AUC-ROC) of 0.72 for AKI prediction, included baseline serum creatinine value, admission to a medical service, and the presence of pulmonary disease, diabetes mellitus, kidney disease, cancer, hypertension, and vascular disease. In the validation cohort, the AUC-ROC of this model was 0.75, indicating similarly good discrimination to predict AKI in hospitalized, noncritically ill patients.

Thus, Safadi et al have successfully developed and validated a model that performs moderately well to predict AKI in adult residents of their region when they are hospitalized outside the ICUs. Strengths of this study include the availability of baseline serum creatinine values for the chosen study cohorts and the impressive ascertainment of clinical follow-up data and outcomes. The observed incidence, severity, and outcomes of AKI in these hospitalized patients are consistent with the AKI literature. Some of the limitations of this study are acknowledged by the authors. The homogeneity of the study population potentially limits the generalizability of their findings, and of course prospective local and multicenter
validation is required. Although it is unsurprising that the model consists of a combination of baseline kidney function with a number of chronic comorbidities, the lack of acute physiological data (pulse, blood pressure, respiratory rate, oxygen saturation, and urine output) may represent a missed opportunity to further calibrate and improve the model. Although the measurement of urine output is not immediately available on admission and its recording is notoriously inconsistent outside the ICU, the other vital signs are routinely recorded on admission in all hospitals and are actionable elements of early warning scores. It is likely that such parameters of acute clinical instability could provide additional discrimination to predict AKI in hospitalized patients.

Although the development of AKI risk scores for patients hospitalized outside the ICU has been a relatively neglected topic, Koyner et al recently developed and validated such a model at 5 Chicago hospitals. Their model included chronic comorbidities and also admission vital signs (not including urine output) and admission laboratory values. The AUC-ROC of their model to predict AKI developing within 24 hours of hospital admission was 0.74 (stage 1) and 0.83 (stage 3). Subsequently, they used artificial intelligence with machine learning from all their clinical data from hospitalized patients at a single center (including ICUs) to develop an excellent AKI prediction model. This model had an AUC-ROC of 0.9 to predict AKI stage 2 developing within the next 24 hours and 0.87 to predict AKI stage 2 developing within 48 hours. Furthermore, the model had an AUC-ROC of 0.96 to predict the initiation of acute renal replacement therapy within 48 hours. The growing emphasis on harm reduction through the identification of patients at increased risk of AKI, combined with careful monitoring of renal function and tailored management, is leading to improved renal outcomes and is becoming a measure of quality of care. The development, validation, and implementation of AKI risk prediction tools such as those proposed by Safadi et al, Koyner and colleagues, and others promises to yield more improvements in our efforts to prevent and treat AKI.

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REFERENCES