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Commentary

Modern Detection Techniques in the Laboratory for Acute Kidney Injury

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ABOUT THE STUDY

It has been a decade since the Kidney Disease Improving Global Outcomes (KDIGO) guideline on Acute Kidney Injury (AKI) was submitted in 2012, which established a concept for the disease and provided useful guidance to practitioners caring for adults and children at risk of or with AKI. Since that seminal publication, significant advances in how we think about and detect AKI have occurred, including the emergence of novel structural and functional AKI biomarkers, the development of automated alerts to aid in disease detection, a new creatinine-based definition proposed by leading laboratory organizations such as the AACC Academy, and the evolution of machine learning tools for AKI prediction.

My aim in this special topic was to summarize the current state of these advances and highlight the most considerable developments in this field that can help improve our ability to detect this silent and deadly disease. Acute kidney injury is defined as a sudden deterioration in kidney function that occurs in about 15% of hospitalized patients and more than 50% of those in the intensive care unit and can lead to serious complications, including irreversible kidney damage and death. The Kidney Disease Improving Global Outcomes (KDIGO) 2012 guidelines define it as an increase in serum creatinine or a decrease in urine output. However, that definition has been called into question in recent years because it failed to account for the analytical and biological variability of creatinine, and it needed to be updated to account for the changes in structural biomarkers that have emerged since. Hasson continue to debate about what we know about these emerging diagnostic markers, which include Cystatin C, urine Neutrophil Gelatinase Associated Lipocalin (NGAL), and the urinary product of Tissue Inhibitor Metalloproteinase (TIMP-2) and Insulin Growth Factor Binding Protein-7 (IGFBP7), as well as how they have been used to delineate AKI phenotypes.

However, other unapproved biomarkers, such as proenkephalin or interleukins 6 (IL-6), IL-8, and IL-18, are being studied for their ability to detect AKI. The necessary to understand how well these are performing, and if they hold any clinical potential. In the case of urinary IL-18 and serum IL-6 and IL-8, their meta-analysis shows that, while levels of these markers are higher in AKI patients than in the control group, their sensitivity and specificity are low, calling their diagnostic value into question. We're turning our attention to creatinine, an old marker that keeps proving the worth of understanding the AKI evolves.

In this study, we demonstrate how monitoring day-to-day changes in creatinine among inpatients can help identify radiocontrast-induced nephropathy using large-data analysis. While examine the effectiveness of using electronic alerts (e-alerts) based on changes in serum creatinine measured in the clinical laboratory, as well as their ability to improve clinical outcomes.

The concerns with current approaches used by institutions that have attempted to implement e-alerts but have had unfavorable results, and then provide key recommendations to improve their performance. Tran demonstrated the importance of measuring urine albumin, dipstick blood, and urine uromodulin to differentiate glomerular from tubulointerstitial diseases. Used a case-based approach to emphasize the importance of using manual urine microscopy in AKI patients to improve clinical care.

Other noteworthy developments reported that these include, the potential association of high vancomycin trough concentration with AKI during piperacillin/tazobactam and vancomycin combination therapy, which would necessitate careful monitoring of vancomycin concentrations to prevent AKI progression and merits confirmation in larger trials, as reported. and the effect of acute changes in glomerular filtration rate, as occurs during AKI, Jones and Chung show that seven of the 28 common biochemistry tests they investigated had significant changes, while the remaining 21 did not.

Serum urea, phosphate, urate, parathyroid hormone, troponin T, B-Natriuretic Peptide (BNP), and NT-proBNP were all measured. The significant implications for clinical teams interpreting results involving AKI patients, ensuring that blood test results involving these analytes are not misinterpreted.

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