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Novel Biomarkers of Acute Kidney Injury

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Acute kidney injury (AKI) represents a common and potentially devastating problem in clinical medicine. Many advances in basic research have illuminated the pathogenesis of AKI and have identified successful therapeutic approaches in animal models. However, translational research efforts in humans have yielded disappointing results. In clinical practice, AKI is typically diagnosed by measuring serum creatinine, which is an unreliable indicator during acute changes in kidney function. The lack of early biomarkers of AKI in humans has crippled our ability to launch potentially effective therapies within a narrow window of opportunity.

Extensive effort has been expended over the past 15 years to identify and validate novel biomarkers that are more sensitive to the onset of injury, specific for prediction and have greater discrimination for injury severity. Essentially, three types of novel biomarkers have been identified in the field of AKI. The first group is inflammatory biomarkers, including neutrophil gelatinase-associated lipocalin (NGAL) and pro-inflammatory cytokines such as interleukin-6 (IL-6) and IL-18. The second group includes cell injury biomarkers such as kidney injury molecule-1 (KIM-1), liver fatty acid-binding protein (L-FABP), sodium/hydrogen exchanger 3 (NHE-3) and netrin 1. The third recently identified group consists of cell cycle markers, such as urinary tissue inhibitor of metalloproteinase 2 (TIMP-2) and insulin-like growth factor-binding protein 7 (IGFBP-7).

Many recent in-depth reviews have summarized the characteristics, advantages and limitations of the first two groups of biomarkers. This presentation focuses on the most recent scientific and clinical information on the development and clinical applicability of cell cycle biomarkers, TIMP-2 and IGFBP-7, in the diagnosis and prognosis of patients at risk for AKI and those suffering from AKI. A number of evaluation studies have demonstrated that compared with existing biomarkers, urinary excretion of the product of both biomarkers, [TIMP-2].[IGFBP-7], improved diagnostic performance in assessing the risk for AKI, predicting the need for renal replacement therapy, AKI-related complications and short- and long-term prognoses. The combination of these two cell cycle arrest markers for the early detection of AKI is promising but concludes that its clinical impact is still unproved. Clinicians should understand the utility and limitations of this test before deciding whether to make it available at their institution.