Commentary

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A Short Note on Biomarkers of Kidney Diseases

San Arrie*

Department of Anesthesiology, University of Gothenburg, Gothenburg, Sweden

*Corresponding author: San Arrie, Department of Anesthesiology, University of Gothenburg, Gothenburg, Sweden, E-mail: sanarrie225@odontologi.gu.se

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Description

Kidney disorders are complex and varied. However, clinical evaluation of kidney disease is heavily reliant on the glomerulus, the kidney's specialised filtering unit. This one-dimensional paradigm affects kidney disease diagnosis and therapy, with obvious consequences: Acute and chronic kidney illnesses continue to outstrip clinical care and are becoming recognised as major global health concerns. Because these abnormalities are discovered too late in the disease progress, no effective therapies have been found to prevent kidney impairment, change the course of disease, or limit the associated morbidity and death.

Biomarkers of tubular injury

Tubular epithelial cells undergo minor modifications in response to initial damage, releasing particular proteins into the urine and systemic circulation. Neutrophil Gelatinase-Associated Lipocalin (NGAL), also known as lipocalin 2 (LCN2), is a glycoprotein found in human neutrophils that is coupled to matrix metalloproteinase-9. It is one of the most extensively studied kidney biomarkers. It is a 25-kDa lipocalin superfamily protein involved in the transport of hydrophilic molecules across membranes to maintain cellular homeostasis. NGAL suppresses bacterial growth by binding to bacterial siderophores and sequestering iron, thus it is vital in innate immunity against infections that require iron acquisition for life.

NGAL is expressed in a variety of organs throughout the body, including the lung, gastrointestinal tract, liver, and kidney, and is significantly elevated in damaged epithelial cells in response to injury, inflammation, and neoplastic transformation. Thus, while both plasma and urine NGAL have been studied as potential indicators of renal damage, urinary NGAL is more selective to that generated by the kidney after insult. Transcriptome profiling studies in mouse models found NGAL as one of the most upregulated genes in the kidney very early after tubular damage, particularly in distal nephron segments, implying that it might be the first recognised indicator of kidney damage.

Urinary NGAL levels in mice models were significantly elevated after 2 hours of renal ischemia-reperfusion damage, and both urine and serum levels were elevated within 2 hours of heart surgery in children who had postoperative AKI. Furthermore, urine NGAL levels in patients with CKD have been shown to be negatively associated to estimated Glomerular Filtration Rate (eGFR) and directly connected to both interstitial fibrosis and tubular atrophy. Based on these positive findings, commercial NGAL tests have been approved for use in the diagnosis of AKI in Europe and Asia, with US FDA approval expected soon.

Tissue inhibitor of metalloproteinases-2 (TIMP-2) and Insulin-like Growth Factor-Binding Protein 7 (IGFBP-7) are both involved in cell-cycle arrest, which is a normal response to kidney damage. IGFBP-7 (through p523 and p21) and TIMP-2 (*via* p27) both inhibit the action of cyclin-dependent protein kinase complexes and produce brief periods of G1 cell-cycle arrest. These biomarkers were found in the clinical environment of critical illness and have been authorised by the FDA for use in combination with clinical evaluation in patients in intensive care units who have acute cardiovascular and/or respiratory failure.