Early detection of urinary NGAL and plasma CysC may prevent progression to overt acute renal failure

Hakan Sarlak, Mustafa Dinc, Sevket Balta, Mustafa Cakar, Erol Arslan, Seref Demirbas

We read with interest the article “Cystatin C and neutrophil gelatinase-associated lipocalin: biomarkers for acute kidney injury after congenital heart surgery” written by Stefanie Seitz et al. [1]. They concluded that, after congenital heart surgery, urine neutrophil gelatinase-associated lipocalin (NGAL) indicated the damaging effects of cardiopulmonary bypass and that serum cystatin C was a valuable predictive biomarker for the resulting acute kidney injury (AKI) [1].

In current practice, the standard method for renal function monitoring remains serum creatinine (SCr) measurement, although it is late and insensitive as there is a 24- to 48-hour delay between renal insult and SCr changes [2]. Furthermore, creatinine starts increasing only when more than 50% of the glomerular filtration rate (GFR) is lost, cannot reflect GFR trends and may be diluted after aggressive fluid resuscitation [3]. NGAL has emerged as one of the most promising biomarkers for the early diagnosis of AKI, and a significant correlation has been found between acute renal injury and serum NGAL concentration 2 hours after cardiopulmonary bypass [4]. Recent investigations have validated the reliability of NGAL as a specific, sensitive and early predictor of AKI after cardiac surgery, contrast administration, septic shock and even renal transplantation [5]. Plasma NGAL (pNGAL) is less specific than its urinary counterpart [2]. In fact, pNGAL is already increased in patients with chronic kidney disease or systemic illnesses, and increases six times less than urinary NGAL in cases of renal injury [2]. Urinary NGAL was evident even after very mild “subclinical” renal ischaemia, despite normal serum creatinine levels [3]. In intensive care settings, plasma cystatin C (pCysC) was able to detect AKI earlier and was more sensitive than SCr in detecting minor GFR reductions [2]. Unfortunately, pCysC levels may also be influenced by several nonrenal factors including corticosteroid administration, thyroid dysfunction, systemic inflammation, neoplasia, age and muscular mass [2]. There is an urgent need for tools allowing an early and accurate detection of AKI [2]. In this regard, urinary NGAL and pCysC seem to be the most promising [2]. These early biomarkers will also help to develop new preventive and/or therapeutic methods for the management of AKI [2].

We think that such early, rapid and simple detection of urinary NGAL and pCysC in patients with subtle, subclinical ischaemic renal injury or subclinical nephrototoxic damage will alert the clinician to introduce manoeuvres aimed at preventing progression to overt acute renal failure.

Note from the publisher: The authors of the original article do not wish to comment on this contribution.

Funding / potential competing interests: No financial support and no other potential conflict of interest relevant to this article was reported.

Correspondence: Hakan Sarlak, MD, Department of Internal Medicine, Gulhane School of Medicine, Tavşik Saglam St., TR-06010 Etlik-Ankara, Turkey. Hakansarlak(at)gmail.com

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