Although acute kidney injury (AKI) is extremely common – with an incidence of about 2,100 per million population [1], similar to that of acute myocardial infarction – the condition remains difficult to treat and prognosis is poor. Indeed, early mortality associated with some forms of AKI such as neutrophil gelatinase-associated lipocalin and kidney injury molecule-1. Insulin-like growth factor-binding protein 7 and tissue inhibitor of metalloproteinases-2 performed better than other known markers and their combination provided additional information. These markers could be useful in clinical practice to uncover silent episodes of AKI or to make an early identification of patients at risk. Ultimately they could help to detect and possibly prevent episodes of acute injury to the kidney, sometimes referred to as kidney attack.

The concept of risk and the value of novel markers of acute kidney injury

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See related research by Kashani et al., http://ccforum.com/content/17/1/R25

Abstract

Kashani and colleagues studied two novel markers, insulin-like growth factor-binding protein 7 and tissue inhibitor of metalloproteinases-2, in the urine of patients at high risk of acute kidney injury (AKI). They validated these markers in a separate large multicenter study and compared them with known markers of AKI such as neutrophil gelatinase-associated lipocalin and kidney injury molecule-1. Insulin-like growth factor-binding protein 7 and tissue inhibitor of metalloproteinases-2 performed better than other known markers and their combination provided additional information. These markers could be useful in clinical practice to uncover silent episodes of AKI or to make an early identification of patients at risk. Ultimately they could help to detect and possibly prevent episodes of acute injury to the kidney, sometimes referred to as kidney attack.
example, compared with the lowest third, patients manifesting these new markers at concentrations in the middle third had a threefold increase in risk while the third with the highest levels had a 10-fold increase. These markers therefore appear to be the pain signal that is missing in this silent disease.

An intriguing implication of this study is that, because both of these new markers can be upregulated in response to a wide range of noxious stimuli, they have the potential to be a nonspecific alarm raised by the renal tubules in response to stress. Detecting this alarm will permit several things to happen, including appropriate triage of patients, more intensive monitoring, and perhaps early involvement from specialists in nephrology and critical care who can promptly evaluate these patients while they are still in the golden hours of this disease prior to irreversible damage to the kidneys.

Finally, as new therapies for AKI are being evaluated in the next few years, the use of biomarkers to help select which patients should be enrolled in trials will be an enormous advantage over current study designs [8].

In conclusion, every single insult to the kidney is potentially dangerous and deleterious for the clinical outcome and future progression of chronic kidney disease. Because early diagnosis and thus related interventions appear possible today thanks to the use of new biomarkers, we should echo the World Kidney Day 2013 claim to ‘Stop Kidney Attack’.

Abbreviations
AKI, acute kidney injury.

Competing interests
CR received honoraria for conferences from Alere, Abbvie, and Astute. ZR declares that he has no competing interests.

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