

Letter

Acute kidney injury: time to shift from creatinine to the estimated glomerular filtration rate?

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Acute kidney injury (AKI) is a complex disorder for which currently there is no accepted definition. Although several groups are working on developing and validating biomarkers of kidney injury and the glomerular filtration rate (GFR), the proposed diagnostic criteria from the Acute Kidney Injury Network are based on an absolute increase in serum creatinine ≥ 0.3 mg/dl (≥ 26.4 μ mol/l), a percentage increase in serum creatinine $\geq 50\%$ (1.5-fold from baseline), or a reduction in urine output (documented oliguria < 0.5 ml/kg per hour for more than 6 hours) [1].

A recent report from the Laboratory Working Group of the National Kidney Disease Education Program, however, recommends that serum creatinine alone should not be used to assess the GFR or to detect the presence of kidney disease because it is affected by the GFR and by factors independent of the GFR, including age, sex, race, body size, diet, certain drugs, and laboratory analytical methods [2]. Rather, the Working Group suggests implementing the estimated GFR using the Modification of Diet in Renal Disease (MDRD) study [2]. In analogy with chronic kidney disease, implementation of the MDRD equation would probably grant more clinically useful information to assess AKI.

Authors' response

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Lippi and Guidi suggest using the MDRD estimated GFR as a criterion for diagnosing and staging AKI [1]. The MDRD estimated GFR was derived from patients with chronic kidney disease who were at steady state (age < 70 years; average GFR, 40 ml/min/1.73 m²) and had renal functional changes over several months and years [2].

In contrast to chronic kidney disease, patients with AKI have rapidly changing levels of serum creatinine over a period of days. Consequently, estimated GFR measurements do not represent the nonsteady-state conditions inherent in AKI and are not recommended in hospitalized patients [3]. Secondly, the estimated GFR is derived from serum creatinine, and changes in the opposite direction – that is, when serum

creatinine doubles, the estimated GFR is reduced by half. Using a relative change in serum creatinine is therefore easier, without the need for an additional step to compute the estimated GFR. Creatinine and urine output are markers of severity in AKI, and have been validated as important risk predictors for outcome [4,5], but do not directly correspond to kidney function at any given time point.

We concur with Lippi and Guidi that more sensitive, accurate and predictive indicators of the measured GFR, injury and prognosis are needed in AKI. Until such markers are validated and widely available, however, the use of the MDRD estimated GFR for the diagnosis and staging of AKI does not provide any additional information to measured changes in serum creatinine.

Competing interests

The authors declare that they have no competing interests.

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