The impact of acute kidney injury in cirrhosis: does definition matter?

In their important study, Tsien et al demonstrated for the first time that minor increases of serum creatinine have major clinical impact in outpatients with cirrhosis, ascites and normal serum creatinine. Patients with acute kidney injury (AKI) had a slow constant increase of serum creatinine over time and, interestingly, a reduced survival probability. These data support the concept that not only hepatorenal syndrome, but also minor changes of normal serum creatinine may have clinical importance. However, no predictors of reversal of AKI could be identified. Possibly, more sensitive markers of reduced glomerular filtration rate, such as cystatin C or NGAL, may be suitable predictors. Another aspect of AKI remains to be elucidated: AKI stage 1 is defined by an increase of serum creatinine of at least 50% or 0.3 mg/dl over serum baseline. These changes, however, may denote different changes of creatinine, as illustrated in table 1, and consequently, even more marked differences of creatinine clearance. Patients with a serum creatinine >0.6 mg/dl do exhibit a more marked increase of creatinine when AKI is defined by a 50% rise. For instance, with a baseline creatinine of 1.0 mg/dl AKI may be reached when creatinine rises to 1.3 mg/dl or to 1.5 mg/dl depending on the definition used. Thus, it may be worthwhile to analyse the data of Wong et al with the hypothesis, that AKI defined by a 50% increase discriminates even more clearly between survivors and non-survivors, and may be a predictor of non-reversal of AKI.

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Table 1  Definition of AKI based on either a 50% rise OR an increase of 0.3 mg/dl over serum baseline creatinine levels

<table>
<thead>
<tr>
<th>Serum creatinine (mg/dl) and AKI</th>
<th>Baseline</th>
<th>Rise by 50%</th>
<th>Rise by 0.3</th>
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<tr>
<td>0.4</td>
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As the baseline serum creatinine increases, the discrepancy between the two definitions increases significantly.

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