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Diagnosis of Acute Kidney Injury: Current Controversies

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Acute kidney injury (AKI) has been a global epidemic in the modern society, with amassing evidence showing that AKI occurs with increased frequency and brings about short-term/long-term complications [1-3]. AKI estimately occurs in 0.2-0.5% of community-indwelling adults per year, 5-20% of hospitalized patients, and 30-60 of critically ill patients in different healthcare settings [2,4,5]. However, as we gain further knowledge with the ever-growing numbers of publications, more controversies seem to appear. In the following section, we list some of the most debated aspects in the research of AKI, especially pertaining to its diagnosis.

First, the determination of baseline serum creatinine (SCr) is one major issue. Most clinicians now recognize AKI based upon the new classification schemes, risk-injury-failure-loss-end stage (RIFLE) and acute kidney injury network (AKIN) [1-6]. Both systems place emphasis on the use of SCr as a diagnostic criterion, though with different threshold. Since the change of SCr requires baseline levels for calibration, the determination of patients' original renal status becomes important. However, it is common that baseline values of SCr are missing or not available [7]. Consequently, researchers devise several methods to circumvent this issue, and these corrections heretofore introduce biases. The ADQI group initially proposed a simple method, that is, to assume a baseline estimated glomerular filtration rate (GFR) of 75 ml/min/1.73m^2, and calculate SCr accordingly (with MDRD (Modification of Diet in Renal Disease) formula) [6]. This method is touted for its easy-applicability and convenience, since no other personal information is required. However, subsequent reports identified that such assumption potentially over-estimates the incidence of AKI, since patients with AKI more often have lower eGFR rather than "normal" level [7,8]. This inflation of AKI incidence might also compromise our ability to interpret any associations between AKI and subsequent events. Others propose substitution with the admission SCr values [9] or minimum SCr values during hospitalization [5,7], or choose to use the lowest out-patient values [4]. However, all these estimates potentially create errors during the diagnosis and, also, the classification and staging of AKI [8]. Recently, Siew et al. utilized multiple imputation methods, comprised of different sets of covariates, to estimate baseline GFR and SCr [7]. Using a real world validation dataset (with available baseline SCr), they found that multiple imputation methods are significantly more accurate than assuming a baseline eGFR 75 ml/min/1.73m^2. This method could be a promising new way for estimating a baseline SCr value.

The other controversial issue in the diagnosis AKI is the reliance of SCr on diagnosis and the discovery of next-generation biomarkers. First, SCr is notorious for its poor sensitivity in detecting renal injury [1,6]. The minimal time required for SCr elevation after AKI onset is 48-72 hours, and creatinine kinetics are altered in many populations, such as the elderly, the muscular male, and patients with malnutrition [10]. Common disease statuses and pathophysiologic changes could also affect the SCr values, including volume dysregulation, use of medications that interfere renal hemodynamics (like angiotensin-converting enzymes inhibitors) and sepsis [11]. This is the reason why clinicians and scientists alike are eagerly pursuing novel biomarkers for diagnosis of AKI. Currently available AKI biomarkers include plasma, serum or urine neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule (KIM)-1, L type fatty acid binding protein (L-FABP), interleukin (IL)-18, netrin-1, and sodium hydrogen exchanger type 3 (NHE3) [12]. They all rise hours after renal insult occurs, and demonstrate prominent elevation compared with their original level. Except NGAL, which is well validated, other markers are still being actively tested for their role in diagnosing AKI. However, all these molecules are limited one way or not, and most important of all, the detection kits for these molecules are expensive and not amenable to large-scale testing. Despite these restrains and economical issues, the future of biomarkers for trailing paths of AKI is still promising. It is expected that we can someday recognize AKI in all populations earlier, and effectively interfere with its natural course so as to reduce the burden and sequels it carry.

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