Letters and Replies

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Baseline creatinine: where to from here?

Sir,
The study of Závada et al. [1] and commentary of Gaião and Cruz [2] have highlighted once more the shortcomings of formula-based estimations of baseline creatinine to define acute kidney injury (AKI). The individual patient approach of Závada is an important consideration, especially for AKI biomarker studies and clinical trial outcomes where individual misclassification will add noise to the data. The age-specific variation in performance is interesting, but perhaps in hindsight inevitable when one considers that the modification of diet in renal disease (MDRD) formula performs worse at higher glomerular filtration rates, which are more likely in the younger age groups. Back-calculating with MDRD has been shown to perform poorly compared to actual baseline for the estimation of AKI incidence with Risk, Injury, Failure, Loss, End stage (RIFLE) now in three studies [1,3,4] and with AKIN in two [4,5]. Závada has evaluated one alternative formula, and in our own study, we evaluated the new Chronic Kidney Disease Epidemiology Collaboration (CKD)-EPI formula and showed that even more accurate estimation performed poorly (Supplementary data online at http://cjasn.asnjournals.org/content/5/7/1165/supp/DC1). Indeed, we showed that baselines obtained by distributing random numbers over a lognormal distribution performed better than back-calculating with MDRD.

An important question remains: Where to from here? We agree entirely with Gaião and Cruz [2] that ‘we do not want to find ourselves in 2015 with everyone using RIFLE/AKIN, but having 30 more different definitions of baseline creatinine’. For studies where baseline can be determined retrospectively, we suggest an adjudicated hierarchical approach that makes use of measured creatinine wherever possible. Siew et al. [5] demonstrated in a hospital cohort that on admission creatinine resulted in an underestimation of AKI incidence, and the lowest creatinine over 7 days resulted in an overestimation. In our intensive care unit cohort, the lowest in 7 days resulted in a 0% error (RIFLE), but with only 0.58 sensitivity and 0.80 specificity [4]. This is perhaps not surprising, as many patients are likely to have elevated creatinine on admission, whereas fluids following on admission are likely to artificially lower creatinine below true baseline. An adjudicated hierarchical approach would consider first non-emergency, preferably outpatient, preadmission creatinine (up to, say, 12 months). A final hospital or follow-up creatinine (if within 3 months, to avoid CKD on AKI) is likely to be the next best estimate. On admission, may be considered if it either precedes the insult (such as in elective surgery), or, perhaps, is shortly after insult (i.e. before creatinine had time to increase) if, prior to the event, (e.g. community cardiac arrest) the patient had been otherwise well. For patients who die in hospital, perhaps then the lowest of the final creatinine before death of on admission creatinine should be used. For prevention trials relying on a diagnosis of AKI prior to initiation of therapy, the problem remains for those patients without preadmission baseline. Other means of determining renal injury (e.g. urine output or injury biomarkers) should be considered in this situation.

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Reply

Sir,
Operating mainly with the creatinine criteria, RIFLE severity grades are based on the multiplication of the baseline creatinine value. That is why baseline creatinine becomes a point of Archimedes that moves the RIFLE. This works perfectly well when we have such a value readily at hand,