Introduction

Acute kidney injury (AKI) is a common clinical problem encountered in critically ill patients and characteristically portends an increase in morbidity and mortality ***(Uchino et al, .2005)***.

Previous epidemiologic investigations describing the incidence and outcomes of AKI in critically ill patients have been limited due to the differences used in defining and classifying AKI ***(Bagshaw et al., 2005)***. This has been unfortunate and likely contributed to hindering scientific progress in the field of critical care nephrology ***(Kellum et al.,2002)***.

The Acute Dialysis Quality Initiative (ADQI) group, comprising experts in the fields of nephrology and critical care medicine, recently published the RIFLE classification, a new consensus and evidence-based definition for AKI. The RIFLE classification defines three grades of severity of AKI (Risk, Injury and Failure) based on changes to serum creatinine and urine output and two clinical outcomes (Loss, End-stage). The RIFLE classification has now been evaluated in a number of clinical studies of critically ill patients with AKI ***(Abosaif et al., 2005)***.

In general, these criteria have been found to have clinical relevance for the diagnosis of AKI, classifying the severity of AKI and for monitoring the progression of AKI, as well as having modest predictive ability for mortality.

More recently, the Acute Kidney Injury Network (AKIN) group, an international collaboration of nephrologists and intensivists, have proposed refinements to the RIFLE criteria ***(Mehta et al., 2007)***.

In particular, the AKIN group sought to increase the sensitivity of the RIFLE criteria by recommending that a smaller change in serum creatinine (≥ 26.2 μmol/L) can be used as a threshold to define the presence of AKI and identify patients with Stage 1 AKI (analogous to RIFLE-Risk). Second, a time constraint of 48 h for the diagnosis of AKI was proposed. Finally, any patients receiving renal replacement therapy (RRT) were to now be classified as Stage 3 AKI (RIFLE-Failure).It is currently unknown whether discernible advantages exist with one approach to definition and classification versus the other ***(Mehta et al., 2007)***.

Acute Kidney Injury (AKI) is a relatively common condition in the intensive care unit (ICU) and is associated with an increased risk of mortality despite the ability to support the functions of the kidneys by means of renal replacement therapy (RRT). Within the ICU it has been demonstrated that AKI is an independent risk factor for mortality, which contrasts with the previously held view that patients die with rather than because of AKI **(Schrier RW 2010).**

New high-throughput screening approaches are being applied to accelerate the discovery process. One hopes that in the next decade AKI will no longer be defined by changes in serum creatinine level, but rather by multiple biomarkers and clinical parameters that form etiology-specific signatures that will clearly discern subtypes of AKI and closely related disorders within minutes to hours, rather than days, after renal injury ***(Murray et al., 2008)***.

In the last decade, great progress has been made in dissecting the molecular mechanisms of acute kidney injury, however, translation of these findings to therapeutics of clinical utility has lagged figure. Development of therapeutics for AKI is slow and has garnered limited industry interest because AKI is poorly characterized and difficult to diagnose early. Additional challenges include an inability to predict severity, measure progression, or measure response to therapy, all of which add complexity and risk to clinical trials ***(Coca et al., 2008)***.

Serum creatinine in AKI has poor sensitivity and specificity: patients are not in steady state; hence serum creatinine lags behind both renal injury and renal recovery. Conventional urine markers (casts, fractional excretion of sodium) are nonspecific and insensitive. Reliance on traditional markers slows recognition of AKI and hence delays nephrologic consultation and discontinuation of nephrotoxic agents, as well as complicates drug development ***(Murray and Palevsky, 2009)***.

The evaluation and initial management of patients with acute kidney injury (AKI) should include: 1) an assessment of the contributing causes of the kidney injury, 2) an assessment of the clinical course including comorbidities, 3) a careful assessment of volume status, and 4) the institution of appropriate therapeutic measures designed to reverse or prevent worsening of functional or structural kidney abnormalities. The initial assessment of patients with AKI classically includes the differentiation between prerenal, renal, and postrenal causes ***(Fry and Farrington 2006)***.