Chao, Gen Med (Los Angel) 2013, 1:1
DOI: 10.4172/2327-5146.1000e102

General Medicine: Open Access

Editorial Open Access

Risk Factors for Acute Kidney Injury in Intensive Care Units

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Keywords: Acute Kidney Injury (AKI); Creatinine; Intensive Care Unit (ICU)

In critical care settings, patients with acute kidney injury (AKI) constitute an important subgroup, in that they have higher short- and long-term mortality, prolonged hospital length of stay, and more resource consumption [1,2]. Incidence of AKI in intensive care unit (ICU) patients ranges between 20-70% according to settings, and, among these, patients who undergo renal replacement therapy (RRT) portend even worse outcome [3]. RRT-treated AKI patients have on average 50-70% in-hospital mortality (depending on AKI etiologies), and 25-50% patients develop chronic kidney disease (CKD) thereafter without complete recovery of renal function [1,3,4]. Consequently, better understanding of the precipitating factors of AKI in these critically ill patients is of paramount importance for clinicians to reduce the incidence of AKI in ICUs.

Risk factors for AKI in patients with severe illnesses are often multiple rather than single. These features can be grouped into several categories: first, certain underlying background predisposes patients to the development of AKI. Aged patients tend to acquire AKI more frequently than their younger counterparts, owing to the physiologic ageing of kidneys, multiple morbidities, and impaired renal recoverability [5]. Comorbidities include those with underlying diabetes mellitus (DM), hypertension, chronic kidney disease (CKD), and heart failure all reportedly set the backstage of subsequent renal injury, through the interplay of disrupted renal auto-regulation, pre-existing renal damage, and concomitant use of nephrotoxic medications [6,7]. Interestingly, as CKD often leads to AKI, AKI at its end begets CKD [8]. Also, "cardiorenal syndrome" has been another heated topic in the research field, after the introduction of the conceptually divided five cardiorenal-syndrome subtypes [9]. To complicate the situations, these morbidities usually come hand-in-hand in the modern society, since metabolic syndrome prevails with Westernization of lifestyles globally. It is then difficult to disentangle the complex of "which should be responsible for AKI" most of the time, but it could be helpful for clinicians to recognize these factors and modify them if feasible.

Second, the insult *per se* can be the precipitant of AKI through different mechanisms. For example, sepsis or systemic inflammatory response syndrome (SIRS) contributes to AKI development, by means of its glomerular hemodynamic alterations, induction of reactive oxygen species and oxidative stresses, and tubular ischemic injury (which is now considered a secondary event) [10]. Hypotension, shock at presentation, and use of vasopressors/inotropes, also account for part of the clinical settings that subsequently spawn AKI [6,7]. Intuitively, these presentations or medical maneuvers reduce renal perfusion and lead to renal ischemia, but the effect of overwhelmed renal auto-regulation by renal vessel selectivity could, in fact, be more important. Several high-risk procedures or operations, such as cardiac surgeries (with cardio-pulmonary bypass), emergent surgeries, or lengthy surgery period, serve as a predisposing factor for AKI after operations [11]. Transfusion with packed red blood cells or use of furosemide peri-operatively could also be associated with AKI [11,12]. The effect of peri-operative transfusion is especially worth mentioning, since transfused erythrocytes may already carry structural or functional changes that occur during prolonged storage, and lead to premature clearance from circulation, with release of unbound hemoglobin and paradoxically stimulation of inflammatory cascade [12]. Iron overload could also play a role in the pathophysiology.

Third, medications are often the one neglected component of the preludes for AKI. Angiotensin-converting enzyme inhibitors or angiotensin receptor blockers constitute one important example. Their use during coronary angiography is reported to increase risk of subsequent contrast induced nephropathy by nearly 50% [13]. Prolonged diuretics use in patients with heart failure or CKD can cause volume depletion and secondary renal ischemia, with resultant AKI susceptibility. Non-steroidal anti-inflammatory agents also predispose one to AKI through their selective renal hemodynamic changes, especially for hypoalbuminemic and anemic patients [14]. Combination of the above agents further raises the AKI risk up to 30%, especially within the initial one month of starting these medications [15]. Consequently, patients in current use of any combination of the above drugs should be attended to, especially during periods of potential renal insults, in order to reduce the incidence of subsequent AKI.

In conclusion, patients with AKI before and during their ICU stay carry a significantly worse outcome than their non-AKI counterparts. Factors including patients' demographic profile, comorbidities, clinical events occurred and medications they use can all take part in these patients' susceptibility to subsequent AKI. It is prudent for us to be cognizant of these factors and aim at preventing or modifying these factors well before the onset of AKI.

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Received January 02, 2013; **Accepted** January 05, 2013; **Published** January 25, 2013

Citation: Chao CT (2013) Risk Factors for Acute Kidney Injury in Intensive Care Units. Gen Med (Los Angel) 1: e102. doi: 10.4172/2327-5146.1000e102

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