World Kidney Day 2013: Acute Kidney Injury; a Public Health Aware

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Once again we reached to March 14, the World Kidney Day. The 8th World Kidney Day (WKD) on March 14, 2013, will be celebrated (1-3). This day is a yearly event rearranged both by the International Society of Nephrology (ISN) and the International Federation of Kidney Foundations (1,2). In this year, the WKD committee aimed to aware the worldwide increase in acute kidney injury (AKI) in both developed and developing countries (4). AKI is characterized by sudden decrease in renal function by decrease in glomerular filtration rate (GFR), followed by accumulation of nitrogenous waste products and the inability to maintain fluid and electrolyte homeostasis (5-7), which usually accompanied by decrease in urine output and various clinical presentations, that is highly linked to increased early and long term mortality and morbidity of the patients. Importantly also, there is a risk of the development of chronic renal failure subsequently (5-7). Despite progress in the understanding of pathogenesis of acute renal dysfunction, we only have a blurred opinion as to why renal function deteriorates so dramatically in many patients with acute illness or injury, or why, despite kidney replacement therapy, mortality is so high (8,9). Since the incidence of AKI has been rising over time, alongside, the prevalence of chronic renal failure has also been increasing. While AKI has long been considered of as a completely reversible disease, however, over the past several years, a bulk of data from experimental animals and humans have been published and pointed out that, AKI more than likely leads to permanent renal damage as chronic renal failure (10,11). On the other hand, the proportion of patients existing after AKI has also been increasing over time (4,10,11). Thus, if AKI really increase the risk for chronic renal failure, then it could imply significant public health concerns with regard to the proportion of persons developing incident chronic renal failure, progressive chronic renal failure, end-stage renal disease (ESRD) (4,10,11). The reasons why AKI would increase the risk of chronic renal failure, end-stage renal disease, and other adverse outcomes not yet fully understood. Various animal investigations suggest that AKI can induce glomerular and interstitial fibrosis (12-17). Hence, despite the fact that AKI is typically reversible in nature, however there may be subclinical kidney damage that persists and mediates this outcome (12-17). Hence, there an international health strategy is necessary to reduce the huge growing load of AKI and its complications. Indeed efforts should focused on preventing AKI accompanied by early detection and treatment, and enough follow up to decrease the mortality and the long term incidence of post-AKI chronic renal failure (4,18,19). AKI is described by one of the followings: increase in serum creatinine to ≥1.5 times baseline or increase in serum creatinine by ≥0.3 mg/dl during 48 hours; or, which is known or presumed to have occurred during the prior 7 days; or urine volume <0.5 ml/kg/h for 6 hours (4,6).
should consisted differentiating, prerenal and postrenal components from intrinsic kidney disease. Biological markers may give early caution of AKI and can help out the differential diagnosis and consideration of prognosis (4,20). Deficiencies in managing have been found as contributing factors in the death of many patients with AKI (19,20). Despite advances in the understanding of the pathogenesis of human AKI, our ability to assess kidney function is limited and functional impairment poorly correlates with structural injury to the kidneys (12-18).

Results from a number of studies have shown that AKI is common, increasing in incidence, and is associated with considerable morbidity and mortality. In the recent study conducted by Aitken E et al. on the demographic data of 1577 patients admitted to a teaching hospital during a one month period in UK, found the incidence of AKI at the time of admission was 4.6%. An additional 10.3% developed AKI during the hospital admission. All cause mortality was 4-fold higher among patients with AKI compared with those without. Mortality was significantly higher in those patients who developed AKI while an in-patient compared with those with AKI on admission. AKI was unrecognized in 23.5% of patients, two-thirds of whom were discharged without resolving of kidney function. They concluded that AKI is common in hospitalized patients and is associated with a significant increase in hospital admission and morbidity and mortality (21).

Many common causes of AKI in critically ill patients exist (17,19). Studies showed that sepsis remains the leading cause of AKI among the critically ill patients accounting for nearly 50% of cases (17-20). Several studies have reported that sepsis-induced AKI is associated with short and long-term risk of death (6,19-21). Indeed recent findings into the pathogenesis of AKI in sepsis are beginning to shift attention from renal blood flow to inflammation-mediated organ injury (20-23). A diagnostic assessment can be used to classify acute kidney injury as prerenal, intrinsic kidney, or postrenal (6, 19-22). The initial workup consists the patient history to find the use of nephrotoxic medications or systemic disease that might cause poor kidney perfusion or directly impair kidney function (20, 22).

Protective substances such as allopurinol, N-acetyl-L-cysteine, prostaglandins and various antioxidants can be used. Treatment modalities consists the elimination of postrenal and prerenal causes of AKI, adjustment of doses of drugs according to kidney status, avoidance of both low arterial pressure and overhydration, preservation of electrolytic balance, avoiding hyperkalemia and correcting hyperglycemia and nutritional support, assuring adequate protein intake (4,6, 22-27).

Ethical considerations

Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely observed by the authors.

References


