*Iranian J Publ Health, Vol. 42, No.3, Mar 2013, pp.338-340*

**Letter to the Editor**

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

***Hamid NASRI***

*Dept. of Nephrology, Division of Nephropathology, Isfahan University of Medical Sciences, Isfahan, Iran*

***Corresponding Author:** Email: hamidnasri@med.mui.ac.ir

**(Received 18 Jan 2012; accepted 15 Feb 2013)**

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 prearranged 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 outcomem(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). Early examination

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, prostagiandins and various antioxidants can be used. Treatmental 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


1. Hostetter TH, Kochis DJ, Shaffer RN, Chertow G, Harmon WE, Klotman PE et al. (2011). World Kidney Day 2011. *J Am Soc Nephrol*, 22(3):397-8.
2. Tayebi Khosroshahi H (2012). Short history about renal transplantation program in Iran and the world: Special focus on world kidney day 2012. *J Nephropathology*, 1(1): 5-10.
3. Garcia Garcia G, Harden P, Jeremy Chapman J (2012). The Global Role of Kidney Transplantation. *J Nephropathology*, 1(2): 69-76.
4. Kam-Tao Li PK, Burdmann EA, Mehta RL (2013).Acute kidney injury: global health alert. *JNephropathology,* 2(2): 90-97.
5. Monedero P, García-Fernández N, Pérez-Valdivieso JR, Vives M, Lavilla J(2011). Acute kidney injury. *Rev Esp Anestesiol Reanim*, 58(6):365-74.
6. Gheissari A, Mehrasa P, Merrikhi A, Madihi Y (2012). Acute kidney injury: A pediatric experience over 10 years at a tertiary care center. *J Nephropathology*, 1(2): 101-108.
7. Nasri H (2012). Sudden onset of acute renal failure requiring dialysis associated with large B-cell lymphoma of colon. *J Nephropathology*, 1(3): 202-206.

8. Murugan R, Kellum JA (2011). Acute kidney injury: what's the prognosis? *Nat Rev Nephrol,*7(4):209-17.
9. Tian J, Barrantes F, Amoateng-Adjepong Y, Manthous CA (2009).Rapid reversal of acute kidney injury and hospital outcomes: a retrospective cohort study. *Am J Kidney Dis*, 53(6):974-81.
10. Waikar SS, Liu KD, Chertow GM (2008). Diagnosis, epidemiology and outcomes of acute kidney injury. *Clin J Am Soc Nephrol*, 3(3):844-61.
11. Waikar SS, Liu KD, Chertow GM (2007). The incidence and prognostic significance of acute kidney injury. *Curr Opin Nephrol Hypertens*, 16(3):227-36.
12. Assadi F(2012). The epidemic of pediatric chronic kidney disease: the danger of skepticism. *J Nephropathology*, 1(2): 61-64.
13. Kari J (2012). Epidemiology of chronic kidney disease in children. *J Nephropathology*, 1(3): 162-163.
14. Solati M, Mahboobi HR (2012). Paraoxonase enzyme activity and dyslipidemia in chronic kidney disease patients. *J Nephropathology*, 1(3): 123-125.
15. Sahni N, Gupta KL (2012). Dietary antioxidents and oxidative stress in predialysis chronic kidney disease patients. *J Nephropathology,* 1(3): 134-142.
16. Gheissari A, Hemmatzadeh S, Merrikhi A, Fadaei Tehrani S, Madihi Y (2012). Chronic Kidney Disease in Children: A report from a tertiary care center over 11 years. *J Nephropathology*, 1(3): 177-182.
17. Rahimi Z (2012). ACE insertion/deletion (I/D) polymorphism and diabetic nephropathy. *J Nephropathology,* 1(3): 143-151.
18. Murugan R, Kellum JA (2011).Acute kidney injury: what's the prognosis? Nat Rev Nephrol, 7(4):209-17.
19. Bagshaw SM, George C, Bellomo R (2008). Early acute kidney injury and sepsis: a multicentre evaluation. *Crit Care,* 12(2):R47.
20. Monedero P, García-Fernández N, Pérez-Valdivieso JR, Vives M, Lavilla J (2011).Acute kidney injury. *Rev Esp Anestesiol Reanim*, 58(6):365-74.
21. Aitken E, Carruthers C, Gall L, Kerr L, Geddes C, Kingsmore D (2013). Acute kidney injury: outcomes and quality of care. *QJM*. Jan 22.
22. Alhamad T, Blandon J, Meza AT, Bilbao JE, Hernandez GT. Acute kidney injury with oxalate deposition in a patient with a high anion gap metabolic acidosis and a normal osmolal gap. *J Nephropathology*, 2(2): 139-143.
23. Rahman M, Shad F, Smith MC(2012).Acute kidney injury: a guide to diagnosis and management. *Am Fam Physician*, 1;86(7):631-9.
24. Tavafi M. Diabetic nephropathy and antioxidants. *J Nephropathology*, 2(1): 20-27.
25. Tavafi M(2012). Inhibition of gentamicin – induced renal tubular cell necrosis. *J Nephropathology,* 1(2): 83-86.
26. Khajehdehi P (2012). Turmeric: Reemerging of a neglected Asian traditional remedy. *J Nephropathology*. 2012; 1(1):17-22.
27. Kadkhodaee M (2012). Erythropoietin; bright future and new hopes for an old drug. *J Nephropathology*. 2012; 1(2): 81-82.