Austin Journal of Nephrology and Hypertension

Open Access

Austin Publishing Group

Editorial

Is Creatinine Always Reflecting Kidney Function in AKI?

Wael Lateef Jebur*
Department of Consultant Nephrologist, NMC Specialty Hospital, Dubai-UAE

***Corresponding author:** Wael Lateef Jebur, Department of Consultant Nephrologist, NMC Specialty Hospital, Dubai-UAE, Tel: 00971507758901; Email: drwaellatif@hotmail.com

Received: February 16, 2015; **Accepted:** February 20, 2015; **Published:** February 23, 2015

The reduction in the filtration function of the kidney is not always in parallel with the global renal function impairment encountered in Acute Kidney Injury (AKI). Nor it is an absolute surrogate marker for its ultimate function, giving the vast diversity of the functions allocated to the different parts of renal structures. Moreover the exact nature of the injury is yet to be contemplated in different stages of Acute Kidney Injury (AKI), whether tubular, glomerular, filtration related, hormonal homeostasis or all. In addition, the precise nature of Acute Tubular Necrosis (ATN) and its interplay to impair the glomerular filtration is yet to be elucidated. I would like to share with you this particular case of AKI, elaborating on its distinctive features, which highlight the concept we mentioned earlier.

A 25 year old patient with no past history of significance was admitted with AKI of unknown etiology. His main complaint was back pain of few days duration, which was of no surgical importance. Clinical examination proved unremarkable. Thorough history revision and direct questioning failed to declare any unforeseeable clue to a probable underlying pathology. On admission he was oliguric, not dehydrated, and not dyspneic. One liter saline challenge was ineffective in improving his urine output. Initial investigations showed advanced renal failure with creatinine of 2.9mg/dl that had steadily increased during his stay in the hospital to 4, 9 and lastly 15 mg/dl over seven days. Similarly his urea increased gradually to 166 over the same period. C-reactive protein was elevated to 65 initially, but no focus of infection was demonstrated, nor did blood cultures grow pathogens. His body weight was increased by 8 kilogram, with prominent facial and sacral edema. However no uremic manifestations of encephalopathy, metabolic acidosis, pericarditis, or resistant hypervolemia could be appreciated. The patient had been kept in the intensive care with close observation. Surprisingly In parallel to the deterioration of filtration function and increasing serum creatinine day by day, the urine output started increasing gradually, and urine output amounted to 1, 2.5, and 4 liters with maximum decline of glomerular filtration. Consequently he turned to polyuric phase with daily urine output of five to seven liters. His body weight reduced gradually reduced in parallel. Measured creatinine clearance initially was 3.5ml/min/1.73,which plummeted to 3 ml/min/1.73 after seven days. Na fractional excretion was more than 2%, denoting acute tubular necrosis. Uric acid, Phosphate and Calcium derangement was noticed. No urinary active sediment was detected, and proteinuria of 250 mg per day was shown. Markers of connective tissue diseases and vasculitis were negative. Ultra sound study revealed normal kidneys size and texture. The management was conservative with initial fluid therapy of one liter given over ten hours, calcium carbonate tablets with meals for hyperphosphatemia, Acetaminophen injections for his back pain two to three injections over the early few days, anti-emetics, and proton pump inhibitor. Despite the fact that his glomerular filtration was immensely compromised, with stage 3 or F according to AKIN and ADQI classification respectively, No hemodialysis was considered, and he was kept on close observation for any sign of uremia.

The intriguing point was the absence of systemic derangement suspected with advanced renal failure. Despite the tremendous decline in glomerular filtration; he never had metabolic acidosis, encephalopathy, hypertension, gastrointestinal manifestation, and neither uremic smell nor uremic taste. However, he was hypervolemic but without pulmonary edema, reflected clinically as orthopnea or dyspnea. Metabolically, mild hyperphosphatemia of 6 meq/l, hyperuricemia of 9 mg/dl, hyponatremia of 131 meq/l, and normal k and Chloride ions, were prevailing at the maximum deterioration of filtration rate of 3 ml/min/1.73. His hemoglobin was always within the normal range of 13-15 gm/dl with normal white cell count and platelets. Similarly Brain Natriuretic peptide was elevated at 500 at the peak of kidney function decline; however echo study of the heart revealed normal myocardium and normal systolic, and diastolic function with ejection fraction of 70%. Interestingly the pro-BNP level was declining with the improvement of glomerular filtration, when serum creatinine leveled from 15 to 6. After which BNP normalized to 50. In order to assess the hormonal activity of the kidney, blood level of Erythropoietin, and 1, 25(OH) 2 D3 were contemplated, and to address the effect of AKI on the parathyroid gland, Parathyroid Hormone (PTH) assay was done. To my surprise Erythropoietin hormone was elevated in comparison to the level of hemoglobin, and active Vitamin D3 was within normal level. Despite the fact that serum phosphorus was elevated, his PTH level fell within normal range. Blood PH and serum bicarbonate were normal throughout his illness. CRP dropped gradually with the improvement of glomerular filtration from 60 to 8 when creatinine was 1.5 after two weeks.

This case was explicitly featuring a differential response of the kidneys to the acute injury, which can be highlighted distinctively as hypo filtration related complications. All the abnormalities detected were secondary to the immense reduction of Glomerular Filtration Rate (GFR). Both water excretion and electrolytes derangement were exponentially related to GFR. With marked water retention of 8 liters that had gradually alleviated during the polyuric phase, similarly phosphate and uric acid retention had mounted up with the peak of GFR decline, and improved stepwise afterwards with the recovery of glomerular filtration. Likewise the BNP had risen up steadily with fluid retention to level with maximum decline of GFR, and consequently to improve and normalize gradually in parallel to fluid retention and overdiuresis thereafter. Conversely other functions were obviously unaffected by the massive defect in the

Citation: Jebur WL. Is Creatinine Always Reflecting Kidney Function in AKI?. Austin J Nephrol Hypertens. 2015;2(2): 1038.

GFR, thereby the patient was nonuremic throughout his illness, and non-acidotic with normal serum bicarbonate, similarly non anemic after two weeks of tremendous decline of GFR. Surprisingly serum Erythropoietin level was above the normal range despite having had normal hemoglobin level, meanwhile 1, 25(D3) was fallen within normal ranges. Both of the findings were apparently indicative of the vitality of renal tubular and peritubular structures, notwithstanding, the diagnosis of ATN was presumed.

Austin J Nephrol Hypertens - Volume 2 Issue 2 - 2015
ISSN : 2381-8964 | www.austinpublishinggroup.com

Citation: Jebur WL. Is Creatinine Always Reflecting Kidney Function in AKI?. Austin J Nephrol Hypertens. 2015;2(2): 1038.