Archives of Nephrology

ISSN: 2639-3573

Volume 1, Issue 2, 2018, PP: 28-30

Can a Very High Creatinine Level Predict Kidneys Chronicity?

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Abstract

Isolated serum creatinine cannot differentiate acute from chronic kidney diseases and other clinical parameter that could assist in differentiating acute from chronic kidney disease at early stage is still missing. Renal sonography is mandatory for any patients presentwith any degree of renal impairment; accordingly further intervention will be decided.

We are presenting here unusual case of severe acute kidney injury where the clinical manifestations are not matching with his biochemical profile.

Introduction

Isolated serum creatinine cannot differentiate acute from chronic kidney failure and other clinical parameter that could assist in differentiating acute from chronic kidney disease at early stage is still missing. Renal sonography is compulsory for any patients present with any level of renal impairment; it is simple and non-invasive method in evaluating patients with various kidney diseases.

Case Report

24 years old male with no past medical history, had presented to ER with nausea and vomiting on and off of nine days duration, associated with abdominal pain. He denied any history of chest pain, fever or diarrhea and he was still passing good amount of urine. There was no history of NSAID intake; however, he gave history of having an electrical shock at upper back 10 days ago, while he was repairing his washing machine. There was no significant past medical history and no family history of renal disease.

Examination upon presentation revealed a well looking young man. He was afebrile, with BP of 138/80, pulse rate of 89b/min. System sexamination was unremarkable.

His laboratory investigations showed the followings: Hb 14.8g/dl, WBC 9.8 10^3/uL, Platelets count 326 10^3/uL. Urea 380 mg/dl(63270 µmol/l), Na 131 mmol/L, K 5.8 mmol/L, CO2 18.2 mmol/l, Creatinine 34.2 mg/dl (3023.2µmol/l), with eGFR (CKD-EPI) 1.5 mL/min/1.73m [(2)]

Amylase 66u/l.Lipase 33.7u/l.

Procalcitonin(PCT) 0.16 (reference <0.05 ng/mL), C-Reactive Protein 6.7 mg/L

Calcium 9.3mg/dL (8.9 - 10.2), Phosphate 7.2 mg/dL(2.7 - 4.5)

Parathyroid Hormone 134pg/ml (6.2 - 29.0)

Hepatitis B Surface Antigen, Hepatitis C Antibody and HIV 1 & 2 Ag & Ab all were negative.

24 hrs. Urine Protein 336 mg/24hrs with Urine Volume 1700mL

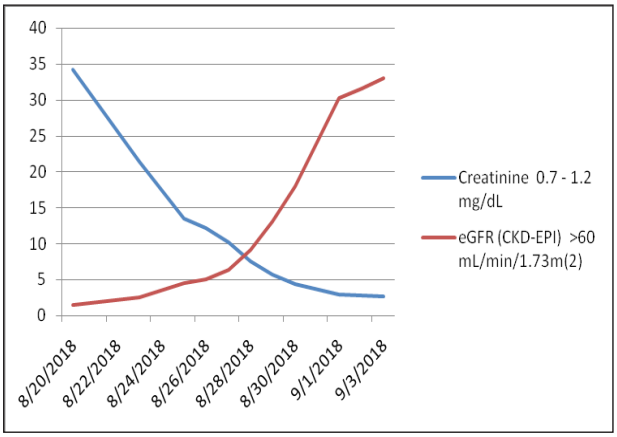
Urine analysis showed: Urine pH 6.0, Urine Protein negative, glucose negative, WBC/HPF 20-25, RBC/HPF 0 – 2. Urine culture was negative.

Table. *Other lab parameters*

Result	Value	Ref Range
Glucose, Random	97	65 - 140 mg/dL
Liver Function Test	Value	Ref Range
Bilirubin,Total	0.5	0 - 1.0 mg/dL
Alkaline Phosphatase	80	40 - 129 U/L
SGPT(ALT)	120 (HH)	0 - 41 U/L
Total Protein	7.5	6.6 - 8.7 g/dL
Albumin	3.5	3.4 - 4.8 g/dL
Globulin	4.0 (H)	2.8 - 3.4 g/dL
ABG Metabolic Panel		
Result	Value	Ref Range
PH	7.330 (L)	7.35 - 7.45
PCO2	38.6	35 - 45 mmHg
PO2	31.2 (L)	83 - 108 mmHg
HCO3 (P)	19.7 (L)	21 - 28 mmol/L
FHBF	3	%
K	5.5 (H)	3.4 - 5.0 mmol/L
NA	129 (L)	134 - 143 mmol/L
Ionised Calcium	1.11 (L)	1.15 - 1.29 mmol/L
CL	109 (H)	97 - 108 mmol/L
Lactic Acid	1.1	0.5 - 1.6 mmol/L
BASE (ECF) (-)	5.1	mmol/L
Result	Value	Ref Range
CPK(CK)	923 (H)	0 - 190 U/L
Result	Value	Ref Range
Lactic Acid	1.1	0.5 - 2.2 mmol/L
Result	Value	Ref Range
Calcium	9.3	8.9 - 10.2 mg/dL

With this advanced azotemia, patient was commenced on hemodialysis through a femoral catheter. An initial impression of ESRD was made keeping in mind the advanced degree of his uremic status with such astonishing high creatinine level in a relatively well looking young male. However, renal sonography was done and showed normal sizes kidneys with good cortical thickness.

Patient had received three hemodialysis sessions, after which he made a remarkable recovery. His creatinine had reduced gradually till it reached a nadir level of 2.6 mg/dl (229.8 µmol/l)at the time of discharge. Patient refused a kidney biopsy to determine the cause of his acute kidney injury.



Discussion

The first task in assessing patients with an elevated creatinine level is to categorize the patient's clinical pre increase in serum creatinine

values from previous baseline levels. History and physical examination are critical for recognizing pre-renal azotemia. Unfortunately this was lacking in our case. Serum creatinine is the most ordinarily utilized measure for renal function in clinical practice nowadays; however, isolated serum creatinine cannot differentiate acute from chronic kidney failure regardless the level and other clinical parameter that could assist us with differentiating acute from chronic kidney disease at early stage is still absent(1,2).

Our patient had severe kidney injury at the time of presentation. Usually with this degree of azotemia he should shows severe symptoms considering his acute status but very few have been reported with this drastic deterioration in renal function. Probably the cause in our scenario is a tubule-interstitial disease rather than a glomerular pathology, aggravated by mild degree of rhabdomyolysis. Unfortunately he refused a kidney biopsy to determine the exact etiology.

All patients with newly discovered renal insufficiency (as confirmed by serum creatinine elevated to a level above the upper limit of the normal range in that laboratory, adjusted for age and height) must investigated to determine the potential reversibility of disease, to evaluate the prognosis and plan patients therapy (3). Ultrasonography remained the simplest non-invasive method and the gold standard in evaluating patients with various kidney diseases. In conjunction to other clinical and laboratory parameters cortical echogenicity could be valuable to anticipate a clinical setting in which a renal biopsy will show acute or an advanced irreversible chronic lesions (2).

Conclusion

This case highlights the fact that clinicians should have a high index of suspicion to look for recoverable causes even if patient has advanced kidney injury with mild or no symptoms.

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Citation: **Fakhriya Alalawi, Hind AlNour, Amna Alhadari.** *Can a Very High Creatinine Level Predict Kidneys Chronicity?. Archives of Nephrology. 2018; 1(2): 28-30.*