Original Research Article

Differentiate Study on Urine Creatinine and Plasma Creatinine of Acute Kidney Injury in Eastern State of Jharkhand, Ranchi, India

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Abstract
Background: *Acute kidney injury is the sudden impairment of kidney function, resulting in the retention of urea and other nitrogenous waste products normally cleared by the kidney. Main objective of the present study is to compare the urine creatinine and plasma creatinine differential diagnosis of Acute Kidney Injury.*
Research Design: *Clinical observation and experimental design were done for the purpose of present study.*
Method: *We are selected in participants for the present study, these consecutive randomly admitted patients in the department of medicine in Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India.*
Sample: *Total 75 Patients were selected in the present study.*
Result: *Observation of the result in present study in no-diuretic group, it was found that the urine creatinine /plasma creatinine (UCr/PCr) area of ROC curve is (0.745), standard error (0.079), 95% of confidence interval (0.578-0.87) and the level of significant in p-value (0.5) is 0.0020; while the with-diuretic group of has found the urine creatinine/plasma creatinine area ROC curve is 0.801, standard error (0.072), 95% of confidence interval (0.637-0.913) and the level of significant in p value (0.5) is 0.0001.*
Conclusion: *Finding of the result concluded that the finding from our study is that Ucr/Pcr was more sensitive but less specific in differentiating prerenal from intrinsic AKI. The sensitivity is more in diuretic exposed group when cutoff value was 43.6364 (p=0.0001). based on these finding it can be good screening test.*
Keywords: *Urine Creatinine, Plasma Creatinine, Acute Kidney Injury.*

Introduction

Acute kidney injury is the sudden impairment of kidney function, resulting in the retention of urea and other nitrogenous waste products normally cleared by the kidney (Harrisons principle of internal medicine18th ed.)[1] Acute Kidney Injury (AKI) complicates 5-7% of acute care hospital admission and up to 30% of admission to the intensive care unit. [2] In severe cases mortality remains as high as 50% particularly in those admitted to the ICU. The causes of AKI other than urinary tract obstruction are usually divided into 2

categories prerenal and intrinsic causes of AKI. [3]Although pathological studies are lacking, the leading cause of persistent AKI in critically ill patients is believed to be acute tubular necrosis. It is usually assumed that it is a spectrum that leads from prerenal AKI to intrinsic AKI. Many publications advocate use of urine indices to differentiate. [4] However diuretic therapy or sepsis may affect these indices. Since urea reabsorption takes place mainly at proximal convoluted tubule and is unaffected by use of diuretics and so fractional excretion of urea may be more reliable than fractional excretion of sodium. [5] However, distinguishing prerenal AKI from intrinsic AKI is needed because it helps to choose treatment for critically ill patients.

The term AKI has largely replaced acute renal failure (ARF), reflecting the recognition that smaller decrements in kidney function that do not result in overt organ failure are of substantial clinical relevance and are associated with increased morbidity and mortality.[6]AKI (acute kidney injury) is common and it is associated with high morbidity and mortality.[7] The loss of kidney function that defines AKI is most easily detected by measurement of the serum creatinine, which is used to estimate the glomerular filtration rate (GFR).[8]

Prior lack of consensus in the quantitative definition of AKI, in particular, has hindered clinical research since it confounds comparisons between studies. Some definition employed in clinical studies have been extremely complex, with graded increments in serum creatinine for different baseline serum creatinine Values. [9] [10]

Objective
The main objective of current study is to compare the urine creatinine and plasma creatinine in differential diagnosis of Acute Kidney Injury.

Research design
Clinical observation and experimental design were done for the purpose of present study.

Method
Sources of data
We are selected in participants for the present study, these consecutive randomly admitted patients in the department of medicine in Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India.

Sample exclusion
Sample selection process we are excluded in some circumstances and illnesses such as patients below age group of 14 years, known case of chronic kidney disease, contrast nephropathy, rhabdomyolysis, acute glomerulonephritis, patient not giving consent, receiving osmotic diuretics eg. Mannitol, end stage of kidney disease receiving renal replacement therapy, obstructive nephropathy, renal transplantation and diabetic nephropathy.

Sample inclusion criteria and sample selection procedure
Total 75 Patients were selected in the present study. The criteria follow below e.g. decreased urine output (urine output < 0.5ml/kg/h) and the raised blood urea and serum creatinine.

Sample were fulfilling above criteria included and divided in two group- Prerenal AKI and Intrinsic AKI. These group were again divided in two sub-group depending upon whether they had received diuretics or not. Sub-group of division were based on history, physical examination, investigation, response to intravenous fluid reversibility of function, recovery of renal functions test, need for renal replacement therapy and staged according to recent KDIGO criteria, then fractional excretion of sodium and fractional excretion of urea were calculated in each patient.

Statistical analyses
Data were treated in the purpose of study, the logistic regression, ROC curve and descriptive analyses were done by the help of SPSS 16.

Result and Discussion
Acute kidney injury is the abrupt loss of kidney function, resulting in the retention of urea and other nitrogenous waste products and

abnormalities in regulation of extracellular fluid volume and electrolytes. Because pre-renal acute kidney injury and acute tubular necrosis (intrinsic AKI) both may need intravenous fluid administration but the risk of volume overload is more in case of later, so as a careful differentiation of AKI is helpful in deciding treatment strategy of patients.

Urine creatinine (UCR) and plasma creatinine (PCR) are useful in such situation to differentiate between prerenal and intrinsic causes of AKI. We studied the over a period of one year. In our study total 75 patients with acute kidney injury were included considering inclusion and exclusion criteria. They were divided in prerenal and intrinsic AKI and were again subdivided on the basis of diuretic exposure and no diuretic exposure and the data were analyzed.

The serum creatinine is widely used in diagnosing the presence of acute kidney injury. However, since it is a sub-optimal biomarker for this process, different urinary and serum proteins have been intensively investigated. Although there are promising candidate biomarkers, none are currently approved for use widely.

Table 1 shows the patients with no diuretic exposure (gold std-type of AKI) type of AKI 1=intrinsic, 0=pre-renal ROC curve analyses (for urine creatinine/plasma creatinine)

Variable	Ucr/Pcr
Classification variable	type of AKI
Select	Exposure Diu. =0

Positive group	
type of AKI	= 1
Sample size	15
Negative group	
type of AKI	= 0
Sample size	23

Area under the ROC curve	0.745
Standard error	0.079
95% Confidence interval	0.578 to 0.872
Significance level P (Area=0.5)	0.0020

Table 2 shows the patient with no diuretic exposure (gold standard-type of AKI) type of AKI 1=intrinsic, 0=pre-renal ROC curve (for urine creatinine/serum creatinine)

Criterion	Sensitivity	95% CI	Specificity	95% CI	+LR	-LR
<7.0625	0.00	0.0-22.0	100.00	85.0-100.0		1.00
<=9.5	0.00	0.0-22.0	91.30	71.9-98.7	0.00	1.10
<=11.6923	6.67	1.1-32.0	91.30	71.9-98.7	0.77	1.02
<=13.8437	6.67	1.1-32.0	86.96	66.4-97.1	0.51	1.07
<=17.9444	26.67	8.0-55.1	86.96	66.4-97.1	2.04	0.84
<=26	26.67	8.0-55.1	78.26	56.3-92.5	1.23	0.94
<=26.1538	33.33	11.9-61.6	78.26	56.3-92.5	1.53	0.85
<=26.6667	53.33	26.6-78.7	73.91	51.6-89.7	2.04	0.63
<=28.3333	60.00	32.3-83.6	73.91	51.6-89.7	2.30	0.54
<=30	66.67	38.4-88.1	69.57	47.1-86.7	2.19	0.48
<=30.5556	66.67	38.4-88.1	65.22	42.7-83.6	1.92	0.51
<=36.6667*	93.33	68.0-98.9	65.22	42.7-83.6	2.68	0.10
<=47.3684	93.33	68.0-98.9	52.17	30.6-73.2	1.95	0.13
<=49.6875	100.00	78.0-100.0	52.17	30.6-73.2	2.09	0.00
<=100	100.00	78.0-100.0	0.00	0.00-15.0	1.00	

+LR: Positive likelihood ratio
-LR: Negative likelihood ratio

Table 3 shows the Patients with diuretic exposure (gold standard-type of AKI) type of AKI 1=intrinsic, 0=pre-renal ROC curve (for urine creatinine/plasma creatinine)

Variable	Ucr/Pcr
Classification variable	type of AKI
Select	Exposure of Diuretic =1

Positive group	
type of AKI	= 1
Sample size	16
Negative group	
type of AKI	= 0
Sample size	21

Area under the ROC curve	0.801
Standard error	0.072
95% Confidence interval	0.637 to 0.913
Significance level P (Area=0.5)	0.0001

Table 4 shows the patient with diuretic exposure (gold standard-type of AKI) type of AKI 1=intrinsic, 0=pre-renal ROC curve (for urine creatinine/serum creatinine)

Criterion	Sensitivity	95% CI	Specificity	95% CI	+LR	-LR
<3.6842	0.00	0.0-20.8	100.00	83.7-100.0		1.00
<=7.1905	31.25	11.1-58.6	100.00	83.7-100.0		0.69
<=15.2941	31.25	11.1-58.6	80.95	58.1-94.4	1.64	0.85
<=20	43.75	19.8-70.01	80.95	58.1-94.4	2.30	0.65
<=20.4375	43.75	19.8-70.01	71.43	47.8-88.6	1.53	0.79
<=33.0769	81.25	54.3-95.7	71.43	47.8-88.6	2.84	0.26
<=33.3333	81.25	54.3-95.7	66.67	43.0-85.4	2.44	0.28
<=39	87.50	61.6-98.1	66.67	43.0-85.4	2.62	0.19
<=40.5385	87.50	61.6-98.1	61.90	38.5-81.8	2.30	0.20
<=43.6364*	100.00	79.2-100.0	61.90	38.5-81.8	2.62	0.00
<=92.6875	100.00	79.2-100.0	0.00	0.0-16.3	1.00	

+LR: Positive likelihood ratio
-LR: Negative likelihood ratio

Table 5 shows the overall value of the urine creatinine and plasma creatinine in no diuretic and with diuretic patients.

Variables	Diuretic exposure	No. of sample		Best cut of value	Sensitivity	Specificity	Level of sig.
		Pre-renal	Intrinsic				
Ucr/Pcr	No	23	15	36.6667	93.33	65.22	0.0020
Ucr/Pcr	Yes	21	16	43.6364	100	61.90	0.0001

Observation of the result in present study in no-diuretic group, it was found that the urine creatinine /plasma creatinine (UCr/PCr) area of ROC curve is (0.745), standard error (0.079), 95% of confidence interval (0.578-0.87) and the level of significant in p-value (0.5) is 0.0020; while the with-diuretic group of has found the urine creatinine/plasma creatinine area ROC curve is 0.801, standard error (0.072), 95% of confidence interval (0.637-0.913) and the level of significant in p value (0.5) is 0.0001.

Very few study were supported in our findings because current scenario of the AKI is different biomarker were widely used, urine creatinine/ plasma creatinine was not existent in widely to biomarker of acute kidney injury. other lateral comparable studies in serum creatinine and plasma creatinine in renal function were done and support the present findings. Studies finding and suggested that the under stable of kidney function sCr concentration can also reflect skeletal muscle mass if its non-muscle-mass-dependent variations (such as due to renal function or meat intake) can be accurately accounted for.[11,12] In people with stable kidney function and UO, a 24 h urine creatinine (uCr) is usually a constant number based on skeletal muscle mass and any variation observed is due to changes in meat consumption.[11,12] Given the fact that sCr co-varies closely with skeletal muscle mass, its utility

in estimating GFR using equations such as MDRD or CKD-EPI may not be appropriate when subjects exhibit weight variations during follow up as in the case of critically ill patients. Muscle loss might be misinterpreted as improvement of renal function.[11,13] In a recent study Hoste et al. studied critically ill patients admitted to ICU with sCr levels within the normal range and found that 25% of these patients had CrCl<60 ml/min/1.73m2. UCr excretion was low in patients with low CrCl, suggesting a pronounced muscle loss and depressed production of creatinine. [14]

Another studies have suggested that the urine or plasma biomarkers such as neutrophilgelatinase-associated lipocalin and IL-18 have beenevaluated as tools for distinguishing between transientAKI and persistent AKI [15-18] but produced conflicting results and therefore cannot be recommended for widespread use at present[16,17-21]. The determination of serum or plasma creatinine has been one cornerstone of diagnosis, as well as follow-up of chronic kidney disease(CKD). Recently, several different creatinine-based equations for the estimation of GFR have been validated and widely adopted for the staging of CKD as well as for follow-up, and planning of dialysis initiation[22]. In the present study by Wilson et al.[23], the creatinine generation rate in AKI patients treated with continuous renal replacement therapy was investigated. The study comprised 103 an uric patients who received stable continuous veno-venous haemo-dialysis and who were in a steady state in regard to plasma creatinine levels.

The finding from our study is that Ucr/Pcr was more sensitive but less specific in differentiating prerenal from intrinsic AKI. The sensitivity is more in diuretic exposed group when cutoff value was 43.6364 (p=0.0001). Based on these finding it can be good screening test.

Conclusion

Finding of the result concluded that the FE Urea showed higher sensitivity and specificity in differentiating prerenal from intrinsic AKI in patients irrespective of diuretic exposure.

Conflict of interest- authors are declaring that no any conflict of interest.

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