

# The Clinical Utility of Kinetic Glomerular Filtration Rate in the Assessment of Renal Function and Prediction of Outcomes Among Critically Ill Patients With Acute Kidney Injury: A Single-Center Retrospective Cohort Study



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## ABSTRACT

**Objective:** To determine the discriminatory ability of kinetic glomerular filtration rate (kGFR) to detect acute kidney injury (AKI) when compared with established GFR equations and criteria and relating it to mortality, renal replacement therapy initiation and renal recovery.

**Methods:** This was a retrospective analysis using data from chart review of 109 intensive care unit (ICU) patients at the University of Santo Tomas Hospital (USTH). The renal function estimates using Chronic Kidney Disease Epidemiology Collaboration (CKD-Epi), modification of diet in renal disease (MDRD), Kidney Disease Improving Global Outcomes Acute Kidney Injury (KDIGO AKI), as well as kinetic GFR equations were compared and correlated with renal and cardiovascular outcomes.

**Results:** The renal function assessed by kGFR, CKD-Epi, MDRD and KDIGO staging based on serum

creatinine (SCr) showed no significant association with mortality outcomes. However, AKI diagnosed based on urine output (UO), and combined SCr and urine output (KDIGO) showed association with all-cause mortality. The UO detected severe stages of AKI while SCr (based on KDIGO) better identified the earlier stages of AKI. The criteria for KDIGO AKI when combined also shows mortality prediction since it joins together the effects of SCr and UO. There was a remarkable 3.5 times increase in hemodialysis initiation ( $p=0.0001$ ) and 12.89 times increase in peritoneal dialysis initiation ( $p=0.01$ ) for every stage increase in the KDIGO classification. kGFR, CKD-Epi and MDRD have 5%, 6%, and 6% decrease, respectively in the odds of initiating hemodialysis. There was however, no association for peritoneal dialysis.

**Conclusion:** kGFR was the least able in detecting AKI and KDIGO AKI criteria remains to be the standard in identifying AKI in the critical care setting. Increase in SCr was a sensitive tool in diagnosing AKI due to its ability to detect AKI based on a small increase in SCr regardless of the baseline renal function. Decreasing UO, however, is the prognosticating variable in KDIGO AKI criteria,

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in that it portends higher probability of initiation of renal replacement therapy (RRT) and ultimately higher mortality when present.

## INTRODUCTION

Acute kidney injury (AKI) is a common cause of morbidity and mortality among critically ill patients with incidence varying from 36% to 67%. [1] It is shown that variation in serum creatinine (SCr) is associated with poor outcomes in hospitalized patients. [2] It is however challenging to assess an acutely unwell patient with rapidly changing kidney function.

The commonly used formulae such as the Modification of Diet in Renal Disease (MDRD) or chronic kidney disease epidemiology collaboration (CKD-Epi) are used in patients with stable renal function to compute the estimated glomerular filtration rate (eGFR). Unfortunately, these current formulae fall short in an acutely unwell patient with rapidly changing renal function.

The current approaches to classify AKI severity and identify patients at the highest risk for poor outcomes focus on the maximum change in SCr values. The non-steady-state kinetic eGFR could add clinical and prognostic information in critically ill patients beyond the current AKI classification system.

In critically ill patients with AKI, it is imperative that we have an estimate of their renal function for the proper adjustment of medication dosages, nutrition and need for renal replacement therapy (RRT). These measures must be instituted at the earliest possible time to minimize unwanted outcomes. kGFR takes into account these shortcomings and reinforces the relevance of timely detection of impending AKI. If proven to be accurate in unstable renal function estimation among critically ill patients, its clinical utility lies in secondary preventive measures that will subsequently limit AKI progression, thereby increasing the recovery chances of our patients.

There is no universally accepted and validated measure of renal function among patients with AKI, more so among critically ill patients. In this subset of patients, the estimates of kidney function are vital to adjustments in drug dosing, revision of nutritional therapy and decisions to provide dialytic support. However, estimating GFR is challenging due to fluctuations and instabilities in kidney function, creatinine production and fluid balance. The common

tools in estimating GFR (ie, CKD-Epi, MDRD) are only used when kidney function and SCr are stable. This led to recent efforts to quantify and classify AKI based on changes in SCr and diuresis. Kidney Disease Improving Global Outcomes (KDIGO) developed the AKI criteria in an attempt to standardize the definition and risk stratification of susceptible patients. [3] This widely used criteria categorized AKI in stages but failed to provide a precise measurement of the kidney function. Potential pitfalls of the criteria include the need for a minimum timeframe (48 h) to proceed with the classification and its inconsideration of baseline renal function level (history of chronic kidney disease) and rapidity of renal function deterioration. This inevitable time window can be a potential cause of delay in the detection and management of AKI.

Kinetic glomerular filtration rate (kGFR) was developed by Chen to analyze the renal function during AKI. [4] It is derived from the initial creatinine content, volume of distribution, creatinine production rate and the quantitative difference between consecutive SCrs over a given time. For that period, the computed creatinine excretion then yields the creatinine clearance rate. Additional formula variables needed in the computation are any steady-state plasma creatinine, the corresponding eGFR by an empirical formula, and the maximum increase in creatinine per day if the patient is anuric. The kinetic formula guides the nephrologists in estimating renal function and complements their clinical intuition by adding a quantitative value to rapidly changing SCr during AKI and in the recovery phase.

The calculation of kGFR also facilitates early prediction of delayed graft function among renal transplant patients within 4 hours of renal transplantation. [5] In an acute critical setting, kGFR was noted to be significantly associated with AKI and initiation of renal replacement therapy (RRT) compared to MDRD but failed to correlate with 30-day mortality and cardiovascular outcomes. [6] There have been no studies addressing the relationship of kGFR and other parameters to renal outcomes in terms of recovery and maintenance of RRT.

The general objective of the study was to determine the clinical utility of kGFR in assessing renal function and correlating outcomes of critically ill patients with AKI. Specifically, it compared the estimates of renal function during AKI using the different parameters of KDIGO-AKI criteria, CKD-Epi, MDRD formula

and kGFR and their correlation to the following endpoints: (a) all cause mortality, (b) initiation of RRT and (c) resolution of AKI. This study also determined the sensitivity of kGFR in assessing AKI among critically ill patients.

## METHODOLOGY

Patients admitted to the UST Hospital Cardiovascular Unit (CVU) and Critical Care Unit (CCU) from January 2017 to December 2017 were included in the study. A chart review of the admissions was done and appropriate data collected using an abstraction form (see appendix) and a retrospective cohort analysis design employed. For patients with multiple CCU/ CVU admissions for the same condition, we only included the initial critical admission and treated the subsequent admissions as outcomes.

Patient profiles included patients greater than 18 years of age, ICU stay of more than 48 h with at least two SCr measurements taken (prior to RRT for those who underwent dialysis) during admission. Patients excluded were known cases of end-stage renal disease, renal transplant patients, transferred patients from other institutions and those who underwent RRT before onset of critical condition.

A sample size of 109 patients yielded a power of 80.00% at a significance level of 5.00% (two-tailed). [9,10] Data extracted from chart review included demographic profile (age, gender), comorbidities, admission data and mortality outcomes. In addition, clinical data collected included but not limited to clinical diagnosis, clinical status (with requirements for vasoactive drugs or mechanical intubation, development of oliguria or anuria, decline in sensorium, etc.), surgical interventions, laboratory and ancillary results.

Patients' admission SCr was treated as the baseline, unless an accessible SCr within three months prior to admission was available. If during the course of admission SCr eventually went down, the nadir SCr was reconsidered as the baseline. Disease severity based on APACHE II score was also computed if available data permitted. Serial SCr measurements were used as a gauge for renal function when using kinetic eGFR formula. This was likewise the case for estimation of kidney function when using the KDIGO-AKI criteria together with the recorded urine output. The highest recorded SCr

was used to estimate renal function when using CKD-Epi and MDRD formula. The worst recorded KDIGO-AKI, CKD Epi, MDRD and kGFR levels during the course of admission or prior to initiation of RRT (for those who underwent dialysis) was used.

All-cause mortality was set as the primary endpoint of the study. Secondary endpoints included initiation of RRT (hemodialysis, peritoneal dialysis, continuous RRT). Those who underwent RRT were further evaluated in terms of AKI resolution. The following operational definitions of AKI resolution were used: (a) no resolution - if a patient was dialysis dependent upon discharge or mortality; (b) partial resolution - weaned off from RRT but SCr failed to go back to normal, nadir or baseline; (c) complete resolution - weaned off from RRT with SCr going back to normal, or SCr decreased within 25% of nadir or baseline.

Patients who developed AKI but were not initiated on RRT were also assessed on the same outcomes as those in the RRT group: (a) no resolution – discharge SCr is the same, higher or fails to decrease by >50% from zenith SCr (highest recorded SCr during admission); (b) partial resolution – discharge SCr decreased by >50% from zenith SCr but failed to go back to normal, nadir or baseline SCr; (c) complete resolution – discharge SCr decreased going back to normal, or SCr decreased within 25% of nadir or baseline SCr.

A Cox regression for survival analysis was performed to assess survival by AKI severity. Logistic regression analysis was used to assess the association and effect of renal parameters (kGFR, KDIGO, eGFR) in predicting prespecified outcomes. Receiver operating characteristic (ROC) analysis was used to find the optimal cut-point value for each assessment that best discriminates between selected prespecified outcomes. Each ROC analysis included an ROC curve, a table with the sensitivities and specificities at certain assessment cut-points, the area under the ROC curve (AUC) and 95% confidence intervals (CIs). AUC curves were compared using the DeLong method. The significance threshold was set at 0.05 for all calculations.

## RESULTS

The study population included 109 admitted critical care patients. Table 1 shows the demographic profile

**Table 1.** Demographic Profile of Patients (N = 109)

Characteristic	Frequency (f)	%	Mean (SD) or Median (IQR)
<b>Age</b>			68.62 (14.04)
<b>Sex</b>			
Male	63	57.80%	
Female	46	42.20%	
<b>Renal Diagnosis</b>			
AKI due to Sepsis	50	45.87%	
AKI due to ATN	1	0.92%	
AKI due to CRS	34	31.19%	
AKI due to Obstructive Uropathy	2	1.83%	
AKI due to Decreased ECV	15	13.76%	
AKI due to AIN	2	1.83%	
AKI due to Hepatorenal Syndrome	1	0.92%	
AKI due to Ischemic Nephropathy	1	0.92%	
AKI due to TTP	1	0.92%	
AKI due to DHN	2	1.83%	
<b>CV Outcome</b>			
Discharged	61	55.96%	
Expired	48	44.04%	
<b>Renal Outcome</b>			
No Resolution	57	52.29%	
Partial Resolution	3	2.75%	
Complete Resolution	49	44.95%	
<b>Initiation of Renal Replacement Therapy (RRT)</b>			
No RRT	60	55.05%	
Hemodialysis	41	37.61%	
Peritoneal Dialysis	8	7.34%	
<b>Underlying CKD</b>			
Yes	66	60.55%	
No	43	39.45%	
<b>Number of Inotropes</b>			1 (0 – 3)
<b>Invasive Ventilation</b>			
Yes	48	44.04%	
No	61	55.96%	

AKI - acute kidney injury

ATN - acute tubular necrosis

CRS - cardiorenal syndrome

ECV - effective circulating volume

AIN - acute interstitial nephritis

TTP - thrombotic thrombocytopenic purpura

DHN - dehydration

of patients with the mean age of 68.62 (14.04). Most of them were male (57.80%), had AKI due to sepsis (45.87%), were discharged (55.96%), had no renal resolution (52.29%), had no RRT (55.05%),

and had underlying CKD (60.55%). The median number of inotropes was 1 with an IQR of 0 – 3. Most respondents did not have invasive ventilation (59.96%).

**Table 2. Clinical and Renal Profiles of the Patients**

Descriptive Statistics (N = 109)

Characteristic	Frequency (f)	%	Mean (SD) or Median (IQR)
<b>APACHE Score</b>			19.16 (7.11)
<b>Kinetic GFR</b>			22.92 (18.20)
<b>CKD-Epi</b>			24.51 (14.29)
<b>MDRD Score</b>			24.69 (13.87)
<b>KDIGO Creatinine</b>			
No AKI	12	11.01%	
Stage 1 AKI	57	52.29%	
Stage 2 AKI	23	21.10%	
Stage 3 AKI	17	15.60%	
<b>KDIGO Urine Output</b>			
No AKI	49	44.95%	
Stage 1 AKI	8	7.34%	
Stage 2 AKI	18	16.51%	
Stage 3 AKI	34	31.19%	
<b>KDIGO</b>			
No AKI	3	2.75%	
Stage 1 AKI	32	29.36%	
Stage 2 AKI	29	26.61%	
Stage 3 AKI	45	41.28%	

The renal profile of these patients is presented in Table 2 showing the descriptive analysis for renal function tests. The mean APACHE score was 19.16 with approximate mortality of 24% for non-postoperative and 12% for postoperative patients, respectively.[7] The study population mortality was 44%, way above the expected mortality rate. The mean kGFR, CKD-Epi and MDRD scores were 22.92, 24.51 and 24.69, respectively. For the KDIGO creatinine, most had Stage 1 AKI (52.29%) while the KDIGO urine output mostly had no AKI (44.95%). The KDIGO AKI, with the combination of urine output and SCr as measures, mostly had Stage 3 AKI (41.28%). KDIGO criteria of AKI had been considered the standard definition and classification of AKI. The criteria using solely the changes in SCr seemed to catch the earlier stages of AKI while the urine output detected more severe stages of AKI but when combined together increased the detection of all stages of AKI.

The mortality outcomes had no significant association with renal function assessed by kGFR, CKD-Epi, MDRD, KDIGO staging based on SCr. However, AKI diagnosed based on urine output, and combined SCr and urine output (KDIGO) showed

association with all-cause mortality (see Table 3). The urine output detected severe stages of AKI while SCr (KDIGO) identified the early stage of AKI (see Table 2). Combining the criteria for KDIGO-AKI was also shown to be predictive of mortality since it combined together the effects of SCr and UO, increasing the recognition of AKI.

There was a remarkable 3.5 times increase of initiating hemodialysis ( $p=0.0001$ ), and 12.89 times increase of initiating peritoneal dialysis ( $p=0.01$ ) for every stage increase in the KDIGO classification as shown in Table 4. Using renal estimates by KDIGO, the UO yielded 4.18 times increase for peritoneal dialysis initiation for every stage increase in the UO criteria. Similarly, individual indices for KDIGO have 71% and 215% increases in hemodialysis initiation for SCr and UO criteria, respectively. For kGFR, CKD-Epi, MDRD have 5%, 6%, 6% decrease, respectively in the odds of initiating hemodialysis. There was, however, no association for peritoneal dialysis.

Renal recovery was not associated with renal function estimated by kGFR, CKD-Epi and MDRD formula. In contrary, there was a significant decrease in the odds of complete recovery for every

**Table 3.** Univariate Binary Logistic Regression of the Predictors of All-Cause Mortality (N = 109)

Predictors	All-Cause Mortality			
	Odds Ratio	Standard Error	p-values (Two-tailed)	95% CI, Odds Ratio
<b>Kinetic GFR</b>	1.00	0.01	0.655	0.97, 1.02
<b>CKD-Epi</b>	1.00	0.01	0.982	0.97, 1.03
<b>MDRD Score</b>	1.00	0.01	0.780	0.97, 1.03
<b>KDIGO Creatinine</b>	1.49	0.34	0.076	0.96, 2.32
<b>KDIGO UO</b>	2.21	0.38	0.0001 <sup>†</sup>	1.58, 3.09
<b>KDIGO</b>	2.83	0.73	0.0001 <sup>†</sup>	1.71, 4.70

<sup>†</sup>Significant at  $\leq 0.01$  level

**Table 4.** Polynomial Logistic Regression of the Predictors of Renal Replacement Therapy (N = 109)

Predictors	Renal Replacement Therapy			
	Odds Ratio	Standard Error	p-values (Two-tailed)	95% CI, Odds Ratio
<b>Kinetic GFR</b>				
No RRT	1.00 (Referent)	–	–	–
Hemodialysis	0.95 <sup>†</sup>	0.01	0.0001	0.92, 0.97
Peritoneal Dialysis	0.96	0.02	0.101	0.91, 1.01
<b>CKD-Epi</b>				
No RRT	1.00 (Referent)	–	–	–
Hemodialysis	0.94 <sup>†</sup>	0.02	0.001	0.91, 0.97
Peritoneal Dialysis	0.99	0.03	0.695	0.94, 1.04
<b>MDRD Score</b>				
No RRT	1.00 (Referent)	–	–	–
Hemodialysis	0.94 <sup>†</sup>	0.02	0.001	0.90, 0.97
Peritoneal Dialysis	0.99	0.03	0.702	0.94, 0.97
<b>KDIGO Creatinine</b>				
No RRT	1.00 (Referent)	–	–	–
Hemodialysis	1.71 <sup>*</sup>	0.41	0.027	1.06, 2.74
Peritoneal Dialysis	1.69	0.72	0.219	0.73, 3.88
<b>KDIGO UO</b>				
No RRT	1.00 (Referent)	–	–	–
Hemodialysis	2.15 <sup>†</sup>	0.38	0.0001	1.52, 3.05
Peritoneal Dialysis	4.18	2.03	0.003 <sup>†</sup>	1.61, 10.82
<b>KDIGO</b>				
No RRT	1.00 (Referent)	–	–	–
Hemodialysis	3.59 <sup>†</sup>	1.05	0.0001	2.03, 6.35
Peritoneal Dialysis	12.89 <sup>†</sup>	12.85	0.010	1.83, 90.98

<sup>\*</sup>Significant at  $\leq 0.05$  level

<sup>†</sup>Significant at  $\leq 0.01$  level

GFR - glomerular filtration rate

RRT - renal replacement therapy

stage increase in KDIGO (OR 0.26,  $p=0.0001$ ). Similarly, there was a significant reduction of complete recovery for every stage increase in SCr

and UO in the KDIGO criteria (OR 0.5,  $p=0.006$ ; OR 0.42,  $p=0.0001$ , respectively) as shown in Table 5.



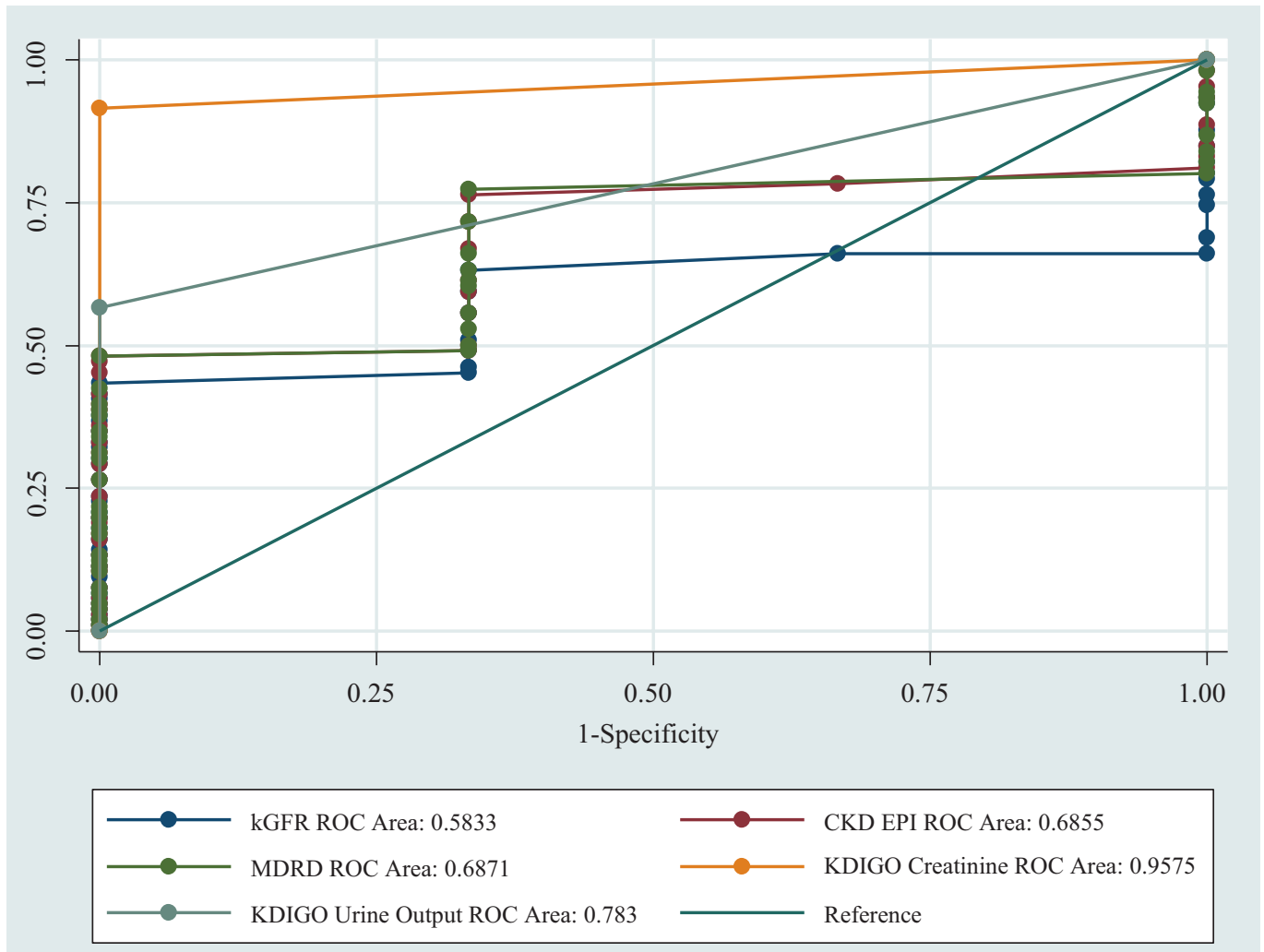
**Table 5.** Polynomial Logistic Regression of the Predictors of Renal Outcome (N = 109)

Predictors	Renal Replacement Therapy			
	Odds Ratio	Standard Error	p-values (Two-tailed)	95% CI, Odds Ratio
<b>Kinetic GFR</b>				
No Resolution	1.00 (Referent)	–	–	–
Partial Resolution	1.04	0.03	0.177	0.98, 1.10
Complete Resolution	1.02	0.01	0.115	1.00, 1.04
<b>CKD-Epi</b>				
No Resolution	1.00 (Referent)	–	–	–
Partial Resolution	1.07	0.04	0.089	0.99, 1.15
Complete Resolution	1.01	0.01	0.545	0.98, 1.04
<b>MDRD Score</b>				
No Resolution	1.00 (Referent)	–	–	–
Partial Resolution	1.07	0.043	0.107	0.99, 1.15
Complete Resolution	1.01	0.01	0.442	0.98, 1.04
<b>KDIGO Creatinine</b>				
No Resolution	1.00 (Referent)	–	–	–
Partial Resolution	1.04	0.69	0.947	0.29, 3.78
Complete Resolution	0.50	0.13	0.006 <sup>†</sup>	0.31, 0.82
<b>KDIGO UO</b>				
No Resolution	1.00 (Referent)	–	–	–
Partial Resolution	0.55	0.27	0.220	0.21, 1.42
Complete Resolution	0.42	0.08	0.0001 <sup>†</sup>	0.29, 0.60
<b>KDIGO</b>				
No Resolution	1.00 (Referent)	–	–	–
Partial Resolution	0.47	0.33	0.277	0.12, 1.83
Complete Resolution	0.26	0.07	0.0001 <sup>†</sup>	0.15, 0.45

\*Significant at  $\leq 0.05$  level†Significant at  $\leq 0.01$  level

Comparison of different renal function tests in diagnosing AKI compared to KDIGO criteria, the current standard for definition and classification of AKI showed kGFR to be the least able to diagnose AKI (See Figure 1 and Table 6). Increase in SCr

based on KDIGO criteria appeared to be the best diagnostic test due to its ability to detect early stages of AKI with small changes in SCr, making it a sensitive measure of AKI.



**Figure 1.** Receiver Operating Characteristics (ROC) of Renal Function against the Gold Standard (KDIGO)

**Table 6.** Receiver Operating Characteristics (ROC) of Renal Function Against the Gold Standard (KDIGO) (N = 109)

Renal Function	AUC	Standard Error	95% CI	p-value
<b>Kinetic GFR</b>	0.5833	0.08	0.42, 0.74	0.00001
<b>CKD-Epi</b>	0.6855	0.11	0.42, 0.89	
<b>MDRD Score</b>	0.6871	0.11	0.48, 0.90	
<b>KDIGO Creatinine</b>	0.9575	0.01	0.93, 0.98	
<b>KDIGO UO</b>	0.7830	0.02	0.74, 0.83	

**DISCUSSION**

Equations used to estimate GFR like CKD-Epi and MDRD are often used in acute critical settings to estimate renal function despite the lack of data from studies supporting its validity in the said setting. These formulae provide physicians with renal function estimate for use of initial drug dosing, prescribed nutrition and even decisions whether to

initiate dialysis. The problem with these equations is the tendency to overestimate GFR when the baseline SCr is low.[8] Small absolute changes in SCr will not be reflected as a significant relative change in GFR, particularly with a lower baseline SCr.

The above problem is taken into account by kGFR where it measures the change in SCr within a given time interval. Intuitively, the larger the excursion in the SCr, the greater the effect will be on the kidney



function. It is proportional to how much the GFR has improved or worsened in comparison with the steady-state GFR. The finding of kGFR inability to diagnose AKI is secondary to the fact that some patients in the study were caught in the resolution phase of the AKI rather than during the initial kidney insult, hence the computed kGFR is higher and ultimately, it decreased its ability to detect AKI during the time of computation. The kGFR formula also incorporates the steady-state SCr, the corresponding eGFR by an empirical formula and the maximum increase in creatinine per day if patient is anuric. If no steady-state SCr is available, the baseline SCr can be substituted as such. In this study, the admission SCr was taken as the steady-state SCr unless a pre-admission SCr done within the last three months was available. The computed change in SCr was then compared to the baseline SCr. If the admission SCr was high, then the change in SCr relative to the baseline was lower.

The KDIGO criteria combines both the increase in SCr and UO as parameters for AKI detection and classification. Increase in SCr based on KDIGO definition was a sensitive gauge in diagnosing AKI compared to the UO criteria due to minimal incremental increase in SCr (0.3 mg/dl within 48 h) required. It was able to detect AKI the most among the renal function tests mostly on the earlier stage

of AKI. UO was able to identify more severe stages of AKI, hence when UO became inadequate the tendency for initiation of RRT was higher with OR of 2.15 and 4.18 for HD and PD, respectively. Mortality also became significantly higher as UO became inadequate, whereas kGFR, CKD-Epi and MDRD did not reflect the same mortality and cardiovascular outcomes similar to the findings of O'Sullivan et al (2016). There was decreasing odds of complete renal recovery as the KDIGO stage became worse.

## CONCLUSION

The KDIGO AKI criterion remains the standard in identifying AKI in the critical care setting. Increase in SCr was a sensitive tool in diagnosing AKI due to its ability to detect AKI based on a small increase in SCr regardless of baseline renal function. Decreasing urine output, however, is the prognosticating variable in KDIGO AKI criteria, in that it portends higher probability of initiation of RRT and ultimately higher mortality when present.

The researcher recommends utilizing prospective studies in AKI among subjects exclusively presenting with increasing azotemia. This research design will better gauge the clinical utility of kGFR diagnosing and prognosticating AKI. It is also preferable that long-term outcomes be measured.

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The study was granted approval by the Ethics Committee of the University of Santo Tomas Institutional Review Board.



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