Characteristics and Clinical Outcomes of Septic AKI Compared to Non-Septic AKI: A Hospital Based Prospective Cohort Study

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Abstract

Background: AKI is a common clinical condition and is associated with increased mortality and morbidity and sepsis is the most common contributing factor to AKI. There is lack of study focused on characteristics differences between septic and non-septic AKI patients and clinical outcome in our setting. The aim of the study is to describe and compare the characteristics and clinical outcomes of patients with septic and non-septic AKI.

Materials and methods: This was hospital based prospective cohort study carried out in the Department of Nephrology of CMCH, from March 2019 to February 2020. A total 94 patients of AKI diagnosed by The KDIGO AKI creatinine criteria, were enrolled in the study after following inclusion and exclusion criteria and divided into two groups- septic and non-septic AKI patients. Patients were followed till 90 days from admission. Demographics, clinical characteristics and outcome were analyzed.

Results: Septic AKI was associated with worse clinical (Decreased MAP, increased mean HR, RR,) hematological (Higher mean WBC count, lower mean Hb%) and biochemical disturbance (Lower mean P^H, HCO_3). Septic AKI had significant association with Diabetes and NonSeptic AKI patients had significant association with HTN. Septic AKI patients had longer mean duration of hospital stay and need more ICU admission. Septic AKI had more complete recovery of renal function than Non-septic AKI (p=.010). Septic AKI had higher 90 days' mortality rate compared with Non-Septic AKI (p=.047). Kaplan-Meier survival curves demonstrated reduced survival for patients with Septic AKI compared with Non-Septic AKI (p=.044).

Conclusion: Significant differences were found in the characteristics and clinical outcome of patients with Septic AKI compared to Non-Septic AKI. Septic AKI was associated with worse clinical, hematological and biochemical disturbance. Patients with Septic AKI had longer duration of hospitalization, an increased risk of death and and showed trends toward greater renal function complete recovery within 3 months of follow up.

Key words: Death; Non-septic AKI; Renal replacement therapy; Septic AKI.

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Introduction

Acute Kidney Injury (AKI) is commonly defined as an abrupt decline in renal function over the course of hours to weeks, manifesting as a reversible acute increase in nitrogen waste products-measured by blood urea and serum creatinine levels.[1] AKI is a syndrome with various causes, associated with different comorbidities, which makes estimating its outcome very complicated. AKI often occurs in the older patients with pre-existing Chronic Kidney Disease (CKD) and chance of risk for dialysis is increased.[2]

The primary diseases on top of which the patients develop AKI in different settings are also heterogeneous, depends on disease prevalence in community, as well as the susceptibility to AKI. Sepsis, volume depletion and hypotension, contrast induced nephropathy, nephrotoxic drugs are most common causes of AKI. A significant proportion of patients may have more than one cause of AKI.[3] Several researchers highlighted the risk factors of AKI as male gender, diabetes mellitus, hypertension, pre-existing CKD, cancer, COPD and tuberculosis.[3,4]

The aetiology of AKI is multi-factorial, but sepsis was leading contributing factor to AKI, approximately 45-70% of all cases, especially in the Intensive Care Units (ICU).[5] Suh et al. suggested that, AKI occur in more than 50% of patients with sepsis and septic shock. Old age positive blood culture results, presence of shock, pre-existing CKD, use of Angiotensin Converting Enzyme (ACE) inhibitors and Angiotensin Receptor Blockers (ARBs) and low WBC and platelet counts were associated with an intensify chance for the development of AKI in septic patient. The development of AKI in septic patient influence worsen in clinical outcomes.[6]

To find out incidence, characteristics and prognosis of AKI in tertiary care hospital of Cameroon, Marie Patrice et al. conducted a prospective study. They got a result of 536 developed AKI among of the 2402 patients. sepsis (50.4%) and volume depletion (31.6%) were main cause. Renal outcome in 34% of patients was unknown. Among 354 patients with known renal function at 3 months, 84.2% completely recovered, 14.7% partially and 1.1% progressed to CKD. Global mortality rate mainly due to sepsis was 36.9%.[7]

Moreover, the severity of AKI was associated with raised short-term mortality. Recent evidence suggests that septic AKI had a distinct pathophysiology.[8] Sepsis is a life-threatening organ dysfunction that happens when the body's response to an infectious agent, injures its own tissues and organs. The diagnosis of sepsis clinically requires finding a infection focus as well as organ dysfunction, identified as an acute change in the total Sequential Organ Failure Assessment (SOFA) score 2 points consequent to the infection.[9]

There were a few clinical studies findings that have investigated about comparison in between septic and non -septic AKI in relation of clinical characteristics, profile and renal function outcome. From these multicenter observational studies, it was found that septic AKI is common and is associated with greater abnormalities in clinical and laboratory parameters compared with either non-septic AKI or sepsis alone. Moreover, sepsis related AKI is associated with increased risk in hospital death that exceeds that of either non-septic AKI or sepsis alone. Among survivors in septic AKI, was also associated with prolonged ICU and hospital stays.[5,10]

Aggarwal et al., conducted a large prospective cohort study to search the spectrum and outcomes of acute kidney injury requiring hemodialysis in a tertiary care hospital of India. They find that, AKI was associated with high mortality and morbidity in the community if not managed early. The most common cause of AKI in the present study were Sepsis and hypovolemia.[11]

Another cohort study was designed with an aim to describe the clinical outcomes of septic associated AKI admitted in ICU from August 2009 to September 2010 in a Tertiary Care Center of Central Nepal. This prospective study showed that the major causes of AKI were medical illness of which pneumonia is most common reason and sepsis-induced AKI had significantly high mortality rate.[12]

At the 2013 world congress of Nephrology in Hong Kong, International Society of Nephrology (ISN), set out the organization's vision for a world in which no one dies of preventable and treatable acute kidney injury. To achieve the ambitious goal, the ISN launched '0by25' (Zero preventable deaths from acute kidney injury by 2025) initiative which aims to eliminate preventable death from AKI worldwide by 2025. '0by25' is a global initiative with a strong emphasis on developing countries like Bangladesh.[13]

Chittagong Medical College Hospital (CMCH) is a 1313 bedded hospital and the second largest tertiary level hospital in Bangladesh. Nephrology Department of this hospital is providing service to a large number of AKI patients routinely. There is lack of study that has searched clinical characteristics and renal outcome of septic AKI in comparison to non-septic AKI patients in our setting. So, Present study is aimed to recite and compare the characteristics and clinical outcomes of patients with septic and nonseptic AKI.

Materials and methods

This was prospective cohort study carried out in the Nephrology Department of CMCH, from March 2019 to February 2020 which was approved by Ethical committee of Chittagong Medical College. Every patient was given informed consent. According to The KDIGO AKI creatinine criteria, 94 AKI patients were diagnosed and enrolled in the study after following inclusion and exclusion criteria and

after that, patients were divided into two groups: patients with septic AKI and patients with non-septic AKI on depends on etiology.

Demographic information included age, gender, types of admission residence, monthly income, comorbidities and clinical, hematological and biochemical information were recorded. Data variable including aetiology of AKI, KDIGO AKI stage, and SOFA score were also recorded. Patients were followed up to 90 days from admission and recorded the patient's outcome. For renal recovery, serum creatinine was done on discharge, at days 30, 60 and 90. When serum creatinine will not find at pre-admission, it will be estimated by the Modification of Diet in Renal Disease equation, as recommended by the Acute Dialysis Quality Initiative Working Group. Data obtained were compiled in Microsoft Xcel sheet to produce a master sheet. Then they were fed into computer software package (SPSS, version 23) for processing and analysis. The qualitative variables were expressed as the absolute number (valid percentage), and the quantitative variables were expressed as the means and standard deviations. To compare the frequency for qualitative variables, Chi-square and Z test of proportion were used and to compare means for continuous variables, Student's t tests was used. To identify the independent predictors of death, Logistic regression analysis was performed. Statistical significance and confidence interval were set at $p<0.05$ and 95% level respectively. Kaplan-Meier survival curve for evaluate 90 days' mortality were plotted to see the survivorship in between septic and non-septic AKI patients. Equality in the estimated survivorship function determined by log-rank test.

Results

Prevalence of Diabetes and Hypertension were significantly more in Septic AKI and Non-Septic AKI group respectively. Septic AKI was associated with significantly higher mean SOFA Score, higher mean heart rates, respiratory rates and lower mean blood pressure and more from the rural area than urban area compared to Non-Septic AKI. Monthly income was significantly more among Non-septic AKI group than their counterpart (Table I). In Septic AKI groups, UTI was main cause (62.8%) and Non-Septic AKI groups, Hypovolemic/AGE was main cause (28.3%) (Table II). Septic AKI group was significantly associated with lower mean Hemoglobin percentage, higher mean white blood cell count, higher neutrophil percentage, lower lymphocyte percentage, lower mean p^H, lower mean HCO_3^- than Nonseptic AKI group (Table III). Septic AKI was significantly associated with higher mean length of hospital stay and higher percentage of need of ICU admission than Non-Septic AKI group. there was no significant difference in between Septic AKI and Non-Septic AKI in comparison of severity of AKI in terms of Stage 1,2 and 3 for RRT. Septic AKI had significantly more complete renal recovery but death occurred in Septic AKI than Non-Septic AKI (Table IV). Logistic regression analysis showed that Age >50yrs, Presence of DM, Presence of HTN, SOFA score >2-point, HR >90, RR >18, SBP 100 mmHg, Hb% <11 g/dl, WBC >10000 per mm3, Creatinine >4mg/dl, K^+>5.5 mmol/l, HCO_3^-<24mmol/l, p^H< 7.4, Urine<400ml/day expect Presence of IHD, RRT independently predict 90days mortality (Table V). Kaplan-Meier survival curves showed reduced survival among Septic AKI patients compared with Non-Septic AKI which was statistically significant (Fig-I).

Table I Baseline Characteristics between Septic AKI and Non-Septic AKI groups.

Variable (Unit)	Groups		
	Septic AKI (n=43)	Non-Septic AKI (n=46)	p-value
Age (Years)	43.14 ± 17.805	38.11 ± 15.38	.157*
Sex			
Male	25 (58.1%)	31 (67.4%)	.367†
Female	18 (41.9%)	15 (32.6%)	
Weight (Kg)	67.465±6.15	68.00±6.8	.700*
DM			
Present	24(55.8%)	16(34.8%)	.046†
Absent	19(44.2%)	30(65.2%)	
HTN			
Present	16(37.2%)	28(60.9%)	.026†
Absent	27(62.8%)	18(39.1%)	
IHD			
Present	13(30.2%)	11(23.9%)	.502†
Absent	30(69.8%)	35(76.1%)	
Creatinine (mg/dl)	6.37±3.1	5.6±2.7	.257*
SOFA score	5.23±1.6	3.22±0.86	<.0001*

Variable (Unit)	Groups		p-value
	Septic AKI (n=43)	Non-Septic AKI (n=46)	
Residence			
Rural	23(53.5%)	14(30.4%)	.027*
Urban	20(46.5%)	32(69.6%)	
Monthly income			
<10000Tk	22(51.1%)	11(23.9%)	.007†
10000-20000Tk	18(41.9%)	25(54.3%)	238†
>20000Tk	3(7%)	10(21.8%)	025†
RR (Per min)	19.86±2.484	17.65±1.66	<.0001*
MAP (mmHg)	80.04±10.65	91.51±14.98	<.0001*

Data are expressed as frequency and Mean (±SD). †p values are derived from Chi-square test, *p values are derived from independent sample t test.

Table II Aetiology of Septic AKI and Non-Septic AKI patients

Groups	Cause	Frequency	Percent
Septic AKI (n=43)	UTI	27	62.8
	Malaria	04	9.3
	Dengue	01	2.3
	Leptospirosis	02	4.7
	Unknown infection	03	7.0
	Pneumonia	02	4.7
	Cellulitis	04	9.3
	Total	43	100.0
Non-Septic AKI (n=46)	Hypovolemic / AGE	13	28.3
	Rhabdomyolysis	06	13.0
	GN	05	10.9
	Pregnancy	05	10.9
	Acute pancreatitis	04	8.7
	Poisoning	05	10.9
	Cardio renal syndrome	04	8.7
	TLS	01	2.2
	Contrast induced	01	2.2
	Drugs	02	4.3
	Gastrointestinal Hemorrhage	00	00
	Lower urinary tract obstruction	00	00
	Hepatorenal syndrome	00	00
	Total	46	100

Data are expressed as frequency (Percentage).

Table III Comparison of Haematological and biochemical parameters in between Septic AKI and Non- Septic AKI groups

Variable (Units)	Groups		p-value
	Septic AKI (n=43)	Non-Septic AKI (n=46)	
Hb% (gm/dl)	10.32±2.10	11.45±2.30	.018*
WBC (/mm^3)	17,786.05±7,075.90	12,143.48±5,000.69	<.0001*
Neutrophil (%)	84.23±5.145	63.85±3.818	<.0001*
Lymphocyte (%)	11.84±4.766	31.17±4.552	<.0001*
Platelet(/mm^3)	2,40,860.47±1,36,029.33	2,55,391.30±1,06,527.30	.575*
Na^+ (mmol/l)	133.03±5.7	134.54±6.8	.267*
K^+ (mmol/l)	4.95±1.29	4.56±0.84	.096*
HCO_3^-(mmol/l)	22.61±1.39	24.32±2.19	<.0001*
PH	7.29±0.09	7.33±0.08	.029*
Creatinine (mg/dl)	6.37±3.1	5.6±2.7	.257*

Data are expressed as Mean (±SD). * p values are derived from independent sample t test.

Table IV Comparison of severity and outcome of AKI in between Septic AKI and Non-Septic AKI groups

Variable	Groups		p-value
	Septic AKI (n=43)	Non-Septic AKI (n=46)	
AKI Stage			
Stage 1	1 (2.3%)	0 (0.0%)	.298#
Stage 2	9 (20.9%)	10 (21.7%)	.928#
Stage 3	33 (76.7%)	36 (78.3%)	.865#
RRT			
Yes	30 (69.8%)	29 (63.0%)	.502†
No	13(30.2%)	17(37.0%)	
Length of Hospital stay	10.23±1.9	8.76±3.8	.025*
Need of ICU admission			
Yes	6 (14%)	1 (2.2%)	
No	37(86.0%)	45(97.8%)	.039†
Renal recovery			
Complete recovery	32 (74.4%)	22 (47.8%)	.010†
Partial recovery & Non recovery	11(25.6%)	24 (52.2%)	
Death			
Yes	9 (20.9%)	3 (6.5%)	.047†
No	34 (79.1%)	43 (93.5%)	

Data are expressed as frequency (Percentage) and Mean (±SD). #p values are derived from Z test of proportion. †p values are derived from Chi-square test; *p values are derived from independent sample t test.

Table V Logistic regression analysis for independent predictors of 90-days mortality of Septic AKI patients

Factor	Odds ratio	95% CI	p value
Age >50yrs	3.64	2.65-4.99	<.0001
Presence of DM	9.60	1.08- 85.16	.042
Presence of HTN	13.00	2.28-74.09	.004
Presence of IHD	8.57	0.83-87.82	.070
SOFA score >2 point	4.062	2.04-8.05	<.0001
HR >90	2.131	1.846-2.459	<.0001
RR >18	2.32	1.40-3.84	<.0001
SBP 100 mmHg	6.787	5.381-8.561	<.0001
Hb% <11 g/dl	2.806	1.59-4.95	<.0001
WBC >10000 per mm^3	2.189	2.16-2.21	<.0001
Creatinine >4mg/dl	5.414	3.00-9.76	<.0001
K^+ >5.5 mmol/l	2.24	1.07-4.69	.033
HCO_3^- <24mmol/l	2.12	1.4-3.04	<.0001
P^H <7.4	5.31	2.92-9.67	<.0001
RRT (Present)	3.714	.891-15.438	.072
Urine<400ml/day	4.5	3.97-5.10	<.0001

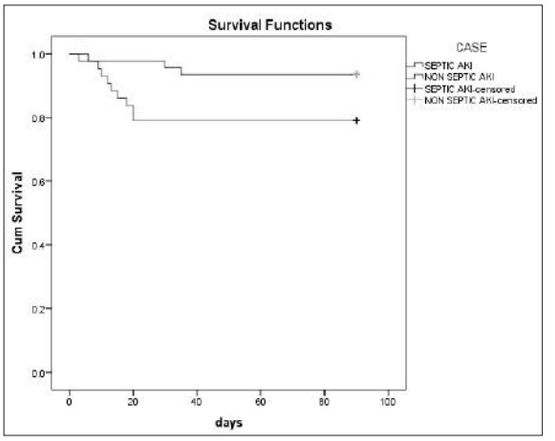

Figure I Comparison of Kaplan-Meier survival plot for 90-days mortality in between Septic AKI and Non-Septic AKI patients (Log rank, p value =0.044)

Discussion

The mean age was 43.14 (±17.81) years and 38.11 (±15.38) years respectively in Septic AKI and NonSeptic AKI group without statistically significant difference (p=.157) in the present study. There was male predominance in both groups without statistical significance (58.1% vs 67.4%, p=.367). Mean weight of the patients was similar in both groups (67.5 vs 68, p=.700). Patients of Septic AKI groups were significantly more belongs to low socioeconomic condition than Non-Septic AKI. Poverty and overcrowding, unhygienic living conditions and poor sanitation contribute to the source of infection. Bagshaw et al. conducted a study which showed that the mean age was 63.2(±16.2) years and 63.5 (±15.7) years respectively in Septic AKI and Non-Septic AKI group without statistically significant difference (p=.49). and also, male predominance in both groups.[5] This finding is almost compatible to the current study although age of both groups of current study was lower.

In the current study, among comorbidities Hypertension was more significantly present in Non-Septic AKI than Septic AKI (60.9% vs 37.2%, p=.026). Diabetes was significantly associated with Septic AKI than Non-Septic AKI (55.8% vs 34.8%, p=.046). Regarding IHD, no significant difference in between the groups (30.2%vs 23.9%, p=502). Shum et al. conducted a study which showed insignificant difference in between these two groups in terms of presence of DM, HTN and IHD.[14] Other study conducted by Cruz et al. showed there was significant difference in between Septic AKI and Non-Septic AKI groups in terms of presence of HTN and IHD except presence of DM.[15]

Regarding SOFA Score, Septic AKI had higher mean score than Non-Septic AKI which was statistically significant (5.23±1.6 vs 3.22±0.86, p= <.0001). Bagshaw et al. showed that there was significant difference in between Septic AKI and Non-Septic AKI group (11.5±3.4 vs 9.5±3.4, p<.001).[5] This finding is almost compatible to the current study although mean of SOFA Score in both groups of current study is lower.

In this study, main aetiology of Septic AKI was UTI (62.8%). The same result was found in study conducted by Tania et al. in Bangladesh and in other study conducted by Marie et al. although percentage of UTI was higher in this study.[16,7] Hypovolemia (28.3%) was main cause of Non-Septic AKI in present study which was consistent result in study conducted by Marie et al.[7]

Regarding clinical characteristics, all parameters were significant difference in between Septic AKI and Non-Septic AKI. Septic AKI patients had higher mean heart rates (101.84 vs 91.3) respiratory rate (19.86 vs 17.65) than Non-Septic AKI which was statistically significant (p<0.0001). Septic AKI group had significantly lower mean MAP than Non-Septic AKI (80.04 vs 91.51 p=<.0001). Bagshaw et al. conducted a large multicentre observational study included 1753 patients of AKI to describe the clinical characteristics in between Septic AKI and Non-Septic AKI.[5] Results of clinical parameter of this study were consistent with current study.

Regarding haematological parameters, Septic AKI group had lower mean Haemoglobin than Non Septic AKI which was statistically significant (10.32 v 11.45, p=.018). Higher mean WBC count/mm^3 (17,786 vs 12,143), higher percentage of Neutrophil (84.23% vs 63.85%) and lower percentage of Lymphocyte (11.84% vs 31.17 %) were significantly present in Septic AKI than Non-Septic AKI (p<0.0001). No significant difference was present in between the group in term of Platelet count/mm^3 (2,40,860 vs 2,55,391 p=.575). These haematological findings were consistent with result of study that conducted by Shum et al. except Haemoglobin which was insignificant difference in between the group.[14]

During sepsis, anaemia is a common sign that occurs due to depression of serum iron levels and erythropoietin production, iatrogenic blood loss and decreased life span of erythrocyte, which may explain the lower mean haemoglobin in Septic AKI in the current study.

In the present study, regarding biochemical parameter no significant difference between in Septic AKI and Non-Septic AKI group in terms of mean Sodium level (133 vs 134 mmol/l, p=.267), Potassium level (4.95 vs 4.56 mmol/l, p=.096) and creatinine level (6.37 vs 5.6 mg/dl, p=.257). Compare to Non-Septic AKI, patients of Septic AKI had lower mean Bio-carbonate level (22.6 vs 24.3, p=<.0001), lower mean P^H (7.29 vs 7.33, p=.029). Bagshaw et al. showed that no significant difference between in Septic AKI and Non-Septic AKI group in terms of Sodium level, Potassium level and creatinine level. Compare to Non-Septic AKI, patients of Septic AKI had lower mean Bio-carbonate level (18.9 vs 20.7), lower mean P^H (7.28 vs 7.34). The comparison of both studies had shown that there were similar findings.[5]

Regarding severity of AKI, no statistically significant difference was found in between Septic AKI and Non-Septic AKI group in terms of KDIGO Stage 1 (2.3% vs 0%, p=0.298), Stage2 (20.9% vs 21.7%, p=0.928) and Stage3 (76.7% vs 78.3%, p=0.865). Aida et al conducted a study in which severity of AKI was described in RIFLE criteria and showed that no statistically significant difference in between Septic AKI and Non-Septic AKI group for severity of AKI Risk/Injury/Failure (8.8/17.6/73.5 vs 7.6/12.1/80.3%).[17] Comparing both studies there was similar findings with more percentage in Stage 3 AKI.

In the present study, no significance difference was present in between Septic AKI and Non-Septic AKI group for receiving RRT (69% vs 63%, p=.502). Bagshaw et al. showed that receiving of RRT was similar in both group (72% vs 71%, p=.83).[5] This finding was compatible with the present study. The mean length of hospital stay of Septic AKI patients was more than Non-Septic AKI (10.23 vs 8.76, p=.025). Septic AKI patients needed more ICU admission than Non-Septic AKI (14% vs 2.2%, p=.039). Bagshaw et al. showed that there was significant difference regarding length of hospital stay in between the groups (25.5 vs 21.1, p=.032) which was similar with the current study.[5]

Regarding renal recovery, Septic AKI had significantly more complete renal recovery percentage (74.4 vs 47.8%, p=.010) than Non-Septic AKI. Shum et al showed that renal function partial recovery was more in Septic AKI (27.5 vs 21.4%, p=.002) and non- recovery was significantly more in Non-Septic AKI (2.5% vs 6.4%, p<.0001). For Full recovery, there was no significant difference (70% vs 72.1%, p=.303).[14] Regarding death, 90 days' mortality was significantly more in Septic AKI than Non-Septic AKI (20.9% vs 6.5%, p=.047) in the current study. Cruz et al and Aida et al showed that death was significantly more in Septic AKI than Non-Septic AKI (64% vs 14%, p=<.0001) and (14.7% vs 3.03%, p=.04) respectively which was similar to current study.[15,17] Though septic AKI patients had more complete renal function recovery due to early diagnosis and management, small sample size, but more death occurred due to multiorgan failure.

Kaplan-Meier survival curves indicate reduced survival for Septic AKI group compared with Non-Septic AKI group which was statistically significant (p=.044). Logistic regression analysis was performed to predict of 90 days' mortality in the current study. It showed that Age >50yrs, Presence of DM, Presence of HTN, SOFA score >2-point, HR >90, RR >18, SBP 100 mmHg, Hb% <11 g/dl, WBC >10000 per mm3, Creatinine >4mg/dl, K+ >5.5 mmol/l, HCO3 <24mmol/l, PH < 7.4, Urine<400ml/day expect Presence of IHD, RRT independently predict 90 days mortality.

Limitation

This was a single-centre small, short duration study. Patient's outcome was influenced by our clinical protocol which may vary from other centre, and incidence and severity of disease might be biased. RRT mode and intensity, initiation time, fluid status and appropriateness of antibiotic for septic patients were not included. Baseline creatinine was missing in some patients,

Conclusion

Rural residence and DM had significant association with septic AKI. Increased mean Heart Rate (HR) and respiratory rate (RR) and decreased Mean Arterial Pressure (MAP) were more common in septic AKI (p=<.0001). Septic AKI

had longer duration of hospitalization and required more ICU admission. Complete Renal function recovery was significant more in septic AKI but death was more in septic AKI.

Recommendations

Large multicentre study involving different ethnicities are required for validation of result and needed to get the national scenario. Long-term follow up is required to define progression of AKI to CKD.

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Contribution of authors

RKS-Conception, design, acquisition of data, analysis, interpretation of data, drafting and final approval.

QI-Analysis, interpretation of data, critical revision & final approval.

SH- Analysis, drafting & final approval.

TKD- Analysis, critical revision & final approval.

MSH-Analysis, interpretation of data, drafting & final approval.

RBK-Analysis, drafting & final approval.

NH-Interpretation of data, critical revision and final approval.

PKD-Conception, design, interpretation of data, critical revision and final approval.

Disclosure

All the authors declared no competing interests.

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