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ORIGINAL ARTICLE

Factors Affecting Survival in Patients with Acute Kidney Injury and Receiving Renal Replacement Therapy

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Abstract

Introduction: Acute kidney injury (AKI) is a common problem. This study aimed to compare the results of complete recovery, chronic kidney disease (CKD), end-stage renal disease (ESRD), and death in patients receiving renal replacement therapy (RRT) by examining the factors determining mortality and morbidity in patients with AKI.

Methods: Patients who received RRT due to AKI in our hospital between 2012 and 2013 were analyzed retrospectively. Eighty-seven patients diagnosed with AKI according to RIFLE and KDIGO criteria and who received RRT were evaluated according to their demographic, laboratory, and recovery status.

Results: The number of patients undergoing hemodialysis due to AKI was 87. Forty-four of the cases were female, and 43 were male. The mean age was 67.6±17 years. Eighty-one of the patients had at least one co-morbid disease other than AKI. Thirty-one patients died before leaving the hospital. Nineteen patients recovered from AKI. Permanent kidney damage occurred in 37 patients. Two patients developed ESRD. The most common cause of death was sepsis (39%).

Discussion and Conclusion: According to RIFLE and KDIGO classification, in patients with AKI who underwent RRT, the presence of infection, concomitant diseases, and advanced age of the patient significantly contribute to determining the prognosis when examining the irreversible deterioration of kidney functions and the factors causing the death of the patient.

Keywords: Acute kidney injury; Mortality-morbidity; Renal replacement therapy.

Acute kidney injury (AKI) is a complex condition that may result in permanent kidney damage, end-stage renal disease (ESRD), and death if the underlying damage is not prevented in the short-term deterioration of kidney functions[1,2]. AKI is seen in 10-15% of patients admitted to the emergency department. This rate can be seen in more than 50% of intensive care units[3-5]. AKI that is not in the hospital setting is called community-acquired (CA)-AKI[3]. The incidence of AKI has been increasing over the years. The increase in the elderly population, the increase in comorbidities, the use of nephrotoxic agents, and the increase in invasive manipulations all contribute to the rising incidence of AKI[5].

AKI is defined as a decrease in creatinine (≥0.3 mg/dL or ≥1.5 times baseline within 48 hours) or urine volume (<0.5 mL/kg/hour within 6 hours)[6]. The onset of renal dysfunction is usually silent. With the increase of uremia, acidosis increases and causes decompensation in the

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patient. Its current diagnosis depends on the interpretation of changes in kidney function or decreased urine output. We assess kidney function by measuring solutes such as creatinine and cystatin C that are excreted by the kidney. However, we do not use cystatin C routinely. In addition, although both tests show changes in urine output, they are not sensitive and specific in AKI[7].

In AKI, the cause of the damage should be determined immediately, and treatments such as fluid replacement or antibiotherapy should be started in a short time. Due to the high morbidity and mortality associated with AKI, early initiation of RRT could be thought to be beneficial. Early initiation of RRT in severe AKI can prevent kidney-specific damage and distant organ damage caused by fluid overload, systemic inflammation, electrolyte, and metabolic imbalance[8,9]. The optimal timing of starting RRT in AKI patients is still unknown. In this study, we aimed to determine whether early initiation of RRT reduces all-cause mortality in patients who are critically ill due to AKI.

Materials and Methods

Study Population

Eighty-seven patients with AKI who needed RRT between October 2012 and October 2013 participated in this retrospective study. The patients participating in the study were non-intubated, hospitalized in the ward and/or non-intubated patients in the intensive care unit. Approval was obtained from the Ethics Committee of Haydarpaşa Numune Training and Research Hospital, where the study was conducted, with the decision dated 25.11.2013 and numbered 2969 (EC-2013/KK/84). There is no profit motive and no working cost in our work. The study followed the guidelines of the Declaration of Helsinki.

For patients with AKI and needing RRT, we examined demographic characteristics, blood tests, and how many sessions of hemodialysis were needed in our hospital's automation system. All patients had a hemodialysis catheter inserted into the left subclavian vein, and the patients required at least 3 sessions and a maximum of 6 sessions of standard continuous hemodialysis during the discharge period. Eighty-seven patients were evaluated for the development of AKI according to the RIFLE and KDIGO classification. Patients who were hospitalized for less than 24 hours, younger than 18 years of age, had chronic kidney disease (CKD) or had a previous kidney transplant, and had kidney pathology were excluded from the study. Signs of sepsis were detected in thirty-seven patients, and growth was also detected in their cultures. In addition to the systemic inflammatory response syndrome (SIRS) findings, the diagnosis of sepsis was also documented. For RIFLE and KDIGO classification, serum creatinine and hourly urine output according to body weight were used, and the worst value was included in the classification. Patients who received RRT were considered stage 3 regardless of other criteria. Demographic characteristics and hospitalization etiologies of all patients included in the evaluation were recorded. Stages of patients diagnosed with AKI according to the RIFLE and KDIGO system; septic, prerenal, postrenal, cardiorenal, acute tubular necrosis (ATN), acute interstitial nephritis (AIN), and glomerulonephritis were evaluated separately.

Statistical Analysis of Data

While evaluating the findings obtained in the study, the SPSS 21.0 Statistics package program was used for statistical analysis. Descriptive statistical methods (frequency, percentage, mean, standard deviation) were used when evaluating the study data. The Pearson Chi-Square test and Fisher Exact test were used to compare qualitative data. The t-test was used to compare the parameters between groups. Logistic regression analysis was used to examine multivariate risk factors that were significant in univariate analyses. In the logistic regression analysis, variables were selected with the enter method, and the odds ratio was calculated by taking the first categories as a reference. The results were evaluated at the 95% confidence interval, at the $p<0.05$ significance level.

Results

Eighty-seven patients who applied to the emergency department between 2012 and 2013 were included in our study. These patients were diagnosed with AKI and received RRT treatment. As a result of follow-up and treatment, the patients were discharged with recovery, CKD, or death. Forty-four of these patients (50.6%) were female and 43 were male (49.4%). The average age was 67.6±17 years (age range 21-93). Eighty-one (93.1%) of the patients had at least one co-morbid disease other than kidney disease (Table 1).

There was no significant difference in terms of gender between patients discharged with recovery and with CKD ($p=0.119>0.05$). It was observed that 9 (42.9%) of those discharged with recovery were women and 12 (57.1%) were men. Thirty-one of the women (55.4%) survived and 25 of the men (44.6%) survived. Of those whose blood cultures showed no growth, 20 (95.2%) were discharged with full recovery. Again, 34 (97.1%) of those without growth were

Table 1. Distributions by Mortality

Parameters		Alive (n=56)	Dead (n=31)	p
Age		68.8±15.6	65.5±19.3	0.411
Hemoglobin	gr/dL	9.9±1.9	9.9±2	0.994
Blood Urea Nitrogen (BUN)	mg/dL	80±39	83±34	0.696
Creatinine	mg/dL	6.3±3.9	5.2±2.3	0.114
Albumin	gr/dL	2.6±0.6	2.4±0.7	0.164
Potassium	mEq/L	5.0±1.26	4.7±1.1	0.274
Sodium	mEq/L	133±6.2	135±6.9	0.12
pH		7.32±0.1	7.35±0.09	0.262
HCO_3	mmol/L	18.5±7.23	20.5±7.6	0.26
C-reactive protein (CRP)	mg/dL	9.25±4	10.75±9.33	0.358
Gender	Female	31	13	0.165
	Male	25	18	
Hematuria	Absent	31	16	0.584
	Available	25	13	
Leukocyturia	Absent	23	16	0.157
	Available	33	13	
Proteinuria	Absent	34	18	0.547
	Available	22	11	
Growth in Blood Culture	Absent	54	25	0.022
	Available	2	6	
Growth in Urine Culture	Absent	47	22	0.125
	Available	9	9	
Reproduction in Catheter Culture	Absent	55	30	0.588
	Available	1	1	
Reproduction in Tracheal Culture	Absent	56	22	0.000
	Available	0	9	
Chronic heart failure	Absent	45	25	0.604
	Available	11	6	
Coronary artery disease	Absent	44	25	0.525
	Available	12	6	
Cerebrovascular diseases	Absent	50	22	0.033
	Available	6	9	
Hypertension	Absent	17	18	0.011
	Available	39	13	
Diabetes mellitus	Absent	29	21	0.112
	Available	27	10	
Oliguria	Absent	14	11	0.215
	Available	42	20	
Fever	Absent	48	18	0.009
	Available	8	12	
Discharged with CKD	Healing	21	31	0.000
	CKD	35	0	
End Stage Kidney Failure	Absent	54	31	0.412
	Available	2	0	
Discharged with healing	Unsuccessful	37	31	0.000
	Successful	19	0	

HCO3 : bicarbonate, pH: potential of hydrogen; CKD: chronic kidney disease.

discharged with CKD. However, a significant difference was found between living patients and deceased patients in terms of growth in blood culture (p=0.022<0.05). Of those with growth in blood culture, 2 (3.6%) survived and 6 (19.4%) died. A significant difference was found in terms of growth in the trachea culture of patients with CKD and those who were discharged with recovery (p=0.000<0.05). Of those with no growth in trachea culture, 21 (100%) were discharged with recovery and 35 (100%) were discharged with CKD. A significant difference was found in terms of reproduction in tracheal culture between living and deceased patients (p=0.000<0.05). It was observed that 9 (29.0%) patients had growth in trachea culture and these patients died. However, a significant difference was found between living and deceased patients in terms of cerebrovascular diseases (p=0.033<0.05). It was determined that 6 (10.7%) of the patients with cerebrovascular diseases survived and 9 (29.0%) died. However, a significant difference was found between living and deceased patients in terms of hypertension (HT) (p=0.011<0.05). It was observed that 39 (69.6%) of the patients with HT survived and 13 (41.9%) died. However, there was a significant difference in fever between living patients and deceased patients (p=0.009<0.05). Of the patients with fever, 8 (14.3%) survived and 12 (40.0%) died (Tables 1, 2).

As a result of the t-test performed to determine whether the glomerular filtration rate (GFR) averages of patients presenting with AKI show a significant difference according

Table 2. Distribution according to the status of chronic kidney disease in surviving patients

		Non-CKD	CKD	p
Gender	Female	9	22	0.119
	Male	12	13	
Hematuria	Absent	13	18	0.315
	Available	8	17	
Leukocyturia	Absent	12	11	0.054
	Available	9	24	
Proteinuria	Absent	12	22	0.442
	Available	9	13	
Growth in Blood Culture	Absent	20	34	0.614
	Available	1	1	
Growth in Urine Culture	Absent	20	27	0.075
	Available	1	8	
Reproduction in Catheter Culture	Absent	21	34	0.625
	Available	0	1	
Reproduction in Tracheal Culture	Absent	21	35	-
Chronic heart failure	Absent	20	25	0.029
	Available	1	10	
Coronary artery disease	Absent	18	26	0.254
	Available	3	9	
Cerebrovascular diseases	Absent	18	32	0.401
	Available	3	3	
Hypertension	Absent	7	10	0.466
	Available	14	25	
Diabetes mellitus	Absent	11	18	0.582
	Available	10	17	
Oliguria	Absent	5	9	0.568
	Available	16	26	
Fever	Absent	19	29	0.356
	Available	2	6	
Healing	Unsuccessful	2	35	0.000
	Successful	19	0	

CKD: chronic kidney disease.

to the status of having CKD after discharge, the difference between the group averages was found to be statistically significant (p=0.000<0.05). Again, when the GFR averages of the living and deceased patients were evaluated, a significant difference was found (p=0.000<0.05). The mean GFR (54.490) of the surviving patients was found to be higher than the mean GFR (24.790) of the patients who died. The mean GFR of those without CKD (77.630) was higher than the mean GFR (40.600) of those with CKD. As a result of the t-test performed to determine whether the calcium (Ca) averages of the patients presenting with AKI show a significant difference according to the death status, the difference between the group means was found to be statistically significant (p=0.048<0.05). The mean Ca of the surviving patients (8.660) was higher than the mean of the patients who died (8.210). As a result of the t-test performed to determine whether the pH averages of the patients presenting with AKI show a significant difference according to the outcome with CKD, the difference between the group averages was found to be statistically significant (p=0.006<0.05). The pH averages of those without CKD (7.370) were found to be higher than the pH averages of those with CKD (7.290). As a result of the t-test performed to determine whether the mean exit creatinine averages of patients presenting with AKI show a significant difference according to the death status, the difference between the group means was found to be statistically significant (p=0.000<0.05). The mean exit creatinine of the patients who died (3.960) was found to be higher than the mean of the exit creatinine (1.420) of the patients who survived. The rate of outcome with CKD was significantly higher in patients with low GFR and pH (p<0.05). Mortality was significantly higher in patients with high white blood cell (WBC) and exit creatinine (p<0.05). Mortality was significantly higher in patients with low GFR, Ca, and parathormone (PTH) (p<0.05) (Tables 3, 4).

Table 3. Distribution according to chronic kidney disease results in surviving patients

	Non-CKD (n=21)		CKD (n=35)		t	p
	Average	SD	Average	SD		
Age	64.100	19.099	71.690	12.616	-1.793	0.115
Hemoglobin, (gr/dL)	10.020	2.172	9.940	1.769	0.162	0.872
Hematocrit, (%)	29.810	6.644	29.790	5.318	0.018	0.986
White blood cell, (mcL)	13.030	5.499	11.480	5.944	0.975	0.334
BUN, (mg/dL)	73,190	45.631	83.970	34.752	-0.998	0.323
Creatinine, (mg/dL)	5.870	3.205	6.540	4.253	-0.618	0.539
Glomerular filtration rate (GFR)	77.630	34.844	40.600	12.785	5.708	0.000
Albumin, (gr/dL)	2.520	0.641	2.610	0.605	-0.542	0.590
Phosphorus (P), (mg/dL)	5.600	2.932	5.470	2.355	0.188	0.851
Sodium (Na), (mEq/L)	132.430	7.004	133.060	5.729	-0.365	0.716
Potassium (K), (mEq/L)	4.810	1.064	5.130	1.352	-0.917	0.363
Magnesium (Mg), (mg/dL)	2.100	0.515	2.040	0.466	0.466	0.643
Calcium (Ca), (mg/dL)	8.870	1.722	8.530	1.303	0.825	0.413
pH	7.370	0.101	7.290	0.096	2.888	0.006
pCO_2	40.370	23.853	36.910	11.056	0.740	0.462
HCO_3, (mmol/L)	19.540	6.700	17.850	7.539	0.850	0.399
Lactic acid, (mmol/L)	6.510	20.776	1.940	2.132	1.298	0.327
Chlorine (Cl), (mEq/L)	133.210	155.589	102.600	6.946	1.169	0.378
Parathormone (PTH), (pg/mL)	111.150	95.003	183.550	140.366	-1.660	0.106
CRP, (mg/dL)	105.040	434.268	7.020	7.687	1.343	0.313
Ferritin, (mg/L)	397.730	508.579	362.590	483.024	0.243	0.809
Protein, (gr/dL)	5.950	1.420	8.360	10.236	-1.070	0.290
Proteinuria, (gr/24h)	1.120	1.515	0.880	1.456	0.445	0.660
Exit Creatinine	1.320	1.551	1.480	0.379	-0.600	0.639

CKD: chronic kidney disease ,BUN: blood urea nitrogen, pH: potential of hydrogen, pCO2: partial pressure of carbon dioxide, HCO3 : bicarbonate, CRP: C-reactive protein.

Table 4. Averages by Mortality

	Alive (n=56)		Dead (n=31)		t	p
	Average	SD	Average	SD		
Age	68.840	15.646	65.480	19.303	0.880	0.381
Hemoglobin, (gr/dL)	9.970	1.911	9.970	2.069	0.007	0.994
Hematocrit	29.800	5.791	30.020	6.115	-0.169	0.867
White blood cell (mcL)	12.060	5.780	14.890	8.077	-1.889	0.062
BUN, (mg/dL)	79.930	39.134	83.100	34.208	-0.377	0.707
Creatinine, (mg/dL)	6.290	3.876	5.230	2.284	1.384	0.114
GFR, (ml/dk/1.73m2)	54.490	29.493	24.790	28.698	4.541	0.000
Albumin, (gr/dL)	2.580	0.615	2.370	0.682	1.453	0.150
P, (mg/dL)	5.520	2.561	4.950	2.216	1.016	0.312
Na, (mEq/L)	132.820	6.182	135.160	6.856	-1.626	0.108
K, (mEq/L)	5.010	1.251	4.730	1.064	1.053	0.295
Mg, (mg/dL)	2.060	0.481	2.200	0.533	-1.204	0.232
Ca, (mg/dL)	8.660	1.468	8.210	0.612	1.630	0.048
pH	7.320	0.104	7.340	0.089	-1.067	0.289
pCO_2	38.210	16.892	37.340	10.949	0.239	0.812
HCO_3, (mmol/L)	18.480	7.221	20.490	7.519	-1.158	0.250
Lactic acid (mmol/L)	3.660	12.835	1.520	0.923	0.845	0.401
Cl, (mEq/L)	114.080	95.165	103.290	11.133	0.627	0.532
PTH, (pg/mL)	158.110	129.708	68.260	71.611	1.994	0.052
CRP, (mg/dL)	43.780	266.283	10.730	9.327	0.689	0.493
Ferritin, (mg/L)	376.210	488.112	610.420	723.889	-1.519	0.134
Protein, (gr/dL)	7.440	8.135	6.350	1.033	0.731	0.467
Proteinuria, (gr/24h)	1.000	1.463	4.720	6.270	-2.949	0.256
Exit creatinine, (mg/dL)	1.420	0.985	3.960	2.379	-6.987	0.000

BUN: blood urea nitrogen, GFR:glomerular filtration rate, P:phosphorus , Na:sodium, K:potassium, Mg:magnesium (Mg), Ca:calcium, pH: potential of hydrogen, pCO2: partial pressure of carbon dioxide, HCO3 : bicarbonate, Cl:chlorine, PTH:parathormone CRP: C-reactive protein.

Generalization with multivariate analysis; urinary culture reproductive status and mean GFR were not found to be sufficiently significant risk factors for CKD (p>0.05). The risk of CKD was 3.591 times higher in patients with leukocyturia in urinalysis (p<0.05). The mortality risk was 3.125 times higher in patients with chronic heart failure (CHF) (p<0.05) (Table 5).

Generalization with multivariate analysis; it was seen that reproduction in blood culture, WBC, and Ca did not create enough significant risk factors for mortality (p>0.05). The risk of mortality was 7.726 times higher in patients with cerebrovascular diseases (p<0.05). The mortality risk was 3.383 times higher in patients with HT (p<0.05). The mortality risk was 1.052 times higher in patients with low GFR levels (p<0.05) (Table 6).

Table 5. Logistic Regression Analysis for Risk Factors Affecting Chronic Kidney Disease

	B	p	OR	95% C.I.for OR	
				Lower limit	Upper limit
Leukocyturia	1.279	0.013	3.591	1.313	9.823
Growth in urine culture	-0.270	0.643	1.310	0.418	4.099
Chronic heart failure	1.139	0.059	3.125	0.956	10.215
GFR (ml/dk/1.73m^2)	-0.006	0.465	1.006	0.991	1.021

GFR: Glomerular filtration rate.

Table 6. Logistic Regression Analysis for Risk Factors Affecting Mortality

	B	p	OR	95% C.I.for OR	
				Lower limit	Upper limit
Growth in blood culture	1.113	0.383	3.043	0.250	36.968
Cerebrovascular diseases	2.045	0.016	7.726	1.462	40.827
Hypertension	-1.219	0.049	3.383	1.008	11.352
Fever	1.269	0.079	3.556	0.863	14.643
White blood cell (mcL)	0.044	0.298	1.045	0.962	1.136
GFR, (ml/dk/1.73m^2)	-0.051	0.001	1.052	1.022	1.083
Ca, (mg/dL)	-0.196	0.561	1.217	0.627	2.360

GFR: Glomerular filtration rate, Ca: calcium.

Discussion

The development of AKI is multifactorial. The most common cause of AKI in outpatients is reduced renal perfusion. ATN is the most common cause of AKI in hospitalized patients. Not all clinical phenotypes of AKI are explained by a single pathophysiological pathway. AKI is usually asymptomatic until the late stages. The patient's medical history, time of onset of AKI, and detailed physical examination are important in determining the underlying etiology. Determining the causes is important for early intervention for the patient[10,11].

Many studies have reported that the most common cause of AKI is sepsis, the second most common is dehydration (e.g. due to vomiting and diarrhea), and the third is heart failure. Still, other studies have shown that the most common cause of death in AKI is sepsis[12]. In our study, RRT was applied to 87 patients due to the development of AKI. Among these patients, 31 female patients and 25 male patients were discharged with recovery and CKD, while 13 male patients and 18 male patients were discharged with death. The reason for our high mortality rate may be the high average age of our study group, infections, and the presence of more than one systemic disease in the patients. When the factors affecting CKD in living patients in our study were examined; the incidence of CKD in patients with leukocytosis in urinalysis (72.7%) was significantly higher than in patients without leukocysts (47.8%). The CKD outcome rate in patients with positive urine culture (88.9%) was significantly higher than in patients with no urine culture (57.4%). The rate of outcome with CKD in patients with CHF (90.9%) was significantly higher than in patients without CHF (55.6%). The mortality rate in patients with growth in blood culture (75%) was significantly higher than in patients with no growth in blood culture (31.6%). The mortality rate in patients with growth in tracheal culture (100%) was significantly higher than in patients without growth in tracheal culture (28.2%). The mortality rate in patients with cerebrovascular diseases (60%) was significantly higher than in patients without cerebrovascular diseases (30.6%). The mortality rate in patients with HT (25%) was significantly lower than in patients without HT (51.4%). The mortality rate in patients with fever (60%) was significantly higher than in patients without fever (27.3%).

Overall, 90% or more of our patients received RRT after reaching KDIGO stage 3. Although most non-randomized studies suggest that early onset of RRT may provide better outcomes, no significant association was found between mortality and morbidity between early-onset RRT and late-onset RRT[11-19]. Data from recent randomized trials showed that early RRT strategy had no benefit on mortality and morbidity[20]. In both the AKIKI and IDEAL-ICU studies, a significant proportion of AKI patients recovered spontaneously when RRT was discontinued, provided that classical indications for RRT did not occur[21,22]. The practice observed in our study is consistent with these more recent findings regarding the initiation of RRT in stage 3 AKI using predominantly classical indications.

Classical indications were the dominant trigger for initiating RRT in this study. This was consistent with the delayed series of the IDEAL-ICU and AKIKI studies[21,22]. The IDEAL-ICU delayed group was initiated on RRT for indications comparable to our study population. These were metabolic acidosis, hyperkalemia, fluid overload, and other causes. The ability to predict deterioration before it occurs can provide a therapeutic or preventive window.

Study limitations; this study was a single-center retrospective study with a small sample size using convenience sampling over a 1-year period. In addition, due to the high age of the patients, creatinine and GFR

values were not compatible with each other. All of the above can lead to a non-representative sample of the general population. In the study, information was obtained from patient charts, clinical notes, and a database created for each patient. This was done solely as information for the on-site data collection system. AKI patients not on dialysis were not evaluated further. This group of patients may represent a subgroup of patients who need dialysis but cannot afford it due to limited resources, are hemodynamically unstable, or have a poor prognosis. Although the study period may represent a study period (2012 to 2013), the absolute and relative indications for RRT remained unchanged. Expanding the study population to other centers and conducting a prospective study should also be considered.

Conclusion

In AKI patients who underwent RRT according to the RIFLE and KDIGO classification, the presence of infection, concomitant diseases, and the advanced age of the patient contribute significantly to the prognosis when examining the factors that cause irreversible deterioration in kidney functions and the death of the patient. Initiating effective treatment according to the patient's underlying diseases before RRT treatment is an effective and fundamental intervention. In KDIGO stage 3 patients, the clinician's early and/or late initiation of RRT does not have a significant impact on mortality. Additionally, the presence of infections in renal failure is very important in terms of mortality and morbidity. Early diagnosis and effective treatment of infections are required in AKI patients. This effective treatment can significantly reduce rates of both permanent kidney damage and death.

Ethics Committee Approval: This study was conducted with approval from the Haydarpaşa Numune Training and Research Hospital Ethics Committee with the decision dated 25.11.2013 and numbered 2969 (EC-2013/KK/84).

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