Kidney & Blood Pressure Research

Kidney Blood Press Res 2016;41:865-872
DOI: 10.1159/000452589
Published online: November 21, 2016
Accepted: September 12, 2016

Original Paper

Renal Complications in Patients with Renal Infarction: Prevalence and Risk Factors

Jae Hyun Kwon[a] Bum Jin Oh[a] Sang Ook Ha[b] Dae Yong Kim[c] Han Ho Do[d]

[a]Department of Emergency Medicine, Asan Medical Center, University of Ulsan College of Medicine, Seoul; [b]Department of Emergency Medicine, Hallym University Sacred Heart Hospital, Hallym University Medical Center, Anyang-si, Gyeonggi-do; [c]Department of Emergency Medicine, Brain Allgood Army Community Hospital, Seoul; [d]Department of Emergency Medicine, Dongguk University Ilsan Hospital, Goyang-si, Gyeonggi-do, Republic of Korea

Key Words
Acute kidney injury • Chronic kidney disease • Prevalence • Renal infarction • Risk factor

Abstract
Background/Aims: This study aimed to investigate the incidence and risk factors for acute kidney injury (AKI) and chronic kidney disease (CKD) in patients with renal infarction. ***Methods:*** A single-center retrospective study was conducted from January 2005 to December 2013. Baseline and clinical characteristics of the enrolled patients with renal infarction were evaluated and analyzed according to the presence of AKI and CKD. In particular, predictors for AKI and CKD were determined using logistic regression analysis. ***Results:*** Of the 105 patients included in present study, 41 (39.0%) patients had AKI. A total of 80 patients were followed up for 2 years after hospital discharge. Among these patients, 27 (33.8%) patients had CKD. In the multivariate analysis, the predictors were mean blood pressure (odds ratio [OR] 1.062, 95% confidence interval [CI] 1.015-1.112, p = 0.009) and bilateral involvement (OR 4.396, 95% CI 1.096-17.632, p = 0.037) for AKI, and AKI (OR 14.799, 95% CI 4.173-52.490, p < 0.001) and old age (OR 1.065, 95% CI 1.016-1.116, p = 0.009) for CKD. ***Conclusions:*** Physicians should pay attention to the development of AKI and CKD after renal infarction and follow patients over a long term.

J.H. Kwon and B.J. Oh contributed equally to this work and therefore share first authorship.

Sang Ook Ha

Department of Emergency Medicine, Hallym University Sacred Heart Hospital, Hallym University Medical Center, 22, Gwanpyeong-ro 170 beon-gil, Donan-gu, Anyang-si, Gyeonggi-do, 431-070 (Republic of Korea), Tel. +82-31-380-4129, Fax +82-33-380-4130
E-Mail loneness@naver.com

Kidney & Blood Pressure Research

Kidney Blood Press Res 2016;41:865-872
DOI: 10.1159/000452589
Published online: November 21, 2016

Introduction

The diagnosis of renal infarction is frequently missed and requires high suspicion. Many other diseases (e.g., ureteric stone, acute pyelonephritis, aortic dissection, and renal cancer) mimic the abdominal or flank pain of renal infarction [1]. Hence, the practical incidence of renal infarction may be more than previously known incidence of 0.004-0.007% in the emergency department due to this underestimation [2-4]. However, research on patients with renal infarction has been scarce. Especially, only a few studies have evaluated the renal complications of acute kidney injury (AKI) and chronic kidney disease (CKD) in patients with renal infarction, and the results of these studies have been heterogeneous (AKI incidence of 8-64% and CKD incidence of 6-32.5%) [5-8]. A comprehensive evaluation of risk factors for AKI and CKD has not been performed. This study aimed to investigate the incidence and risk factors for AKI and CKD in patients with renal infarction.

Participants and Methods

Study design and patient selection

This study design was approved by the Institutional Review Board of the author's institution. Owing to the purely observational, retrospective, and non-interventional nature of this study, informed consent was deemed unnecessary and was not obtained. Patients with renal infarction confirmed by enhanced computed tomography (CT) from January 2005 to December 2013 were enrolled. Typical parenchymal finding of renal infarction appears as a single or multiple wedge shaped perfusion defect that involves both the cortex and medulla and extends to the capsular surface in the kidney (Figure 1). Patients with underlying CKD or those younger than 20 years were excluded. Figure 2 presets the flow chart of inclusion and exclusion of patients.

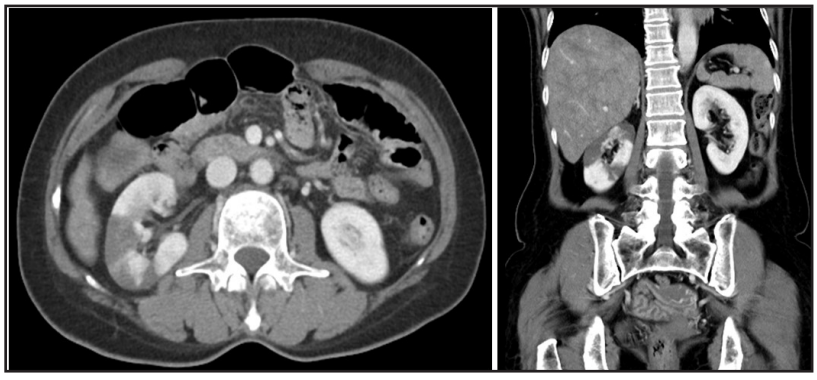

Fig. 1. Contrast-enhanced CT showing typical parenchymal finding of renal infarction. CT revealed a multiple wedge shaped perfusion defects in upper polar and posterior segments of the right kidney.
Abbr.: CT, computed tomography.

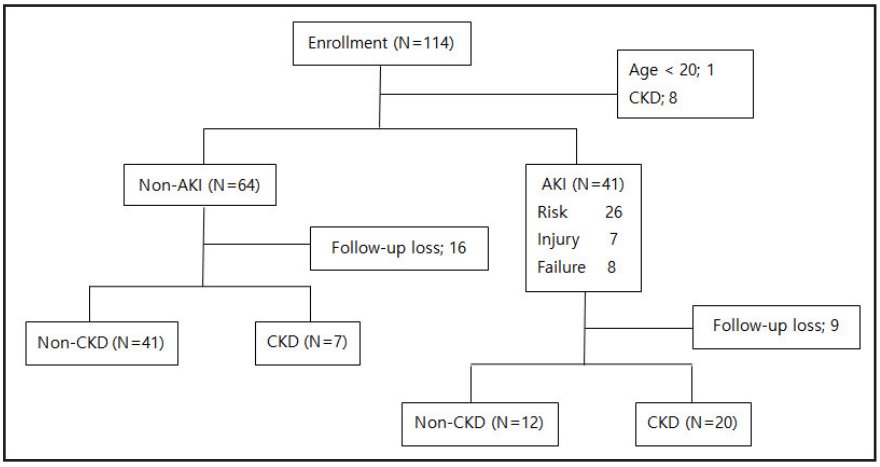

Fig. 2. Flow chart of inclusion and exclusion. Abbreviations: AKI, acute kidney injury; CKD, chronic kidney disease.

Data collection

Electronic medical records (EMR) of enrolled patients were reviewed. Baseline characteristics (sex, age, body mass index (BMI), smoking, Charlson comorbidity index [CCI], underlying disease, causes), clinical characteristics (initial vital signs, symptoms, signs, length of stay, intensive care unit [ICU] admission, treatment), CT findings and laboratory data, and data regarding renal complications (AKI and CKD) were obtained and evaluated retrospectively.

Definitions of AKI and CKD

AKI was defined by the KIDGO guidelines [9]. Increase in serum creatinine (SCr) level by ≥ 0.3 mg/dL within 48 hours; or increase in SCr level to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or Urine volume < 0.5 mL/kg/h for 6 hours. When the baseline SCr level was unknown, an estimated SCr value was obtained from the Modification of Diet in Renal Disease formula by using four variables (serum creatinine, age, sex, and trace). Given that it was not possible to obtain accurate records of urine output from patients admitted to the general ward, we could not use urine output as a criterion for classification. CKD was defined as an eGFR < 60 mL/min/1.73 m^2 that was noted for 3 or more months within 24 months after hospital discharge [10]. Hypertension was defined as systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg. Mean blood pressure (MBP) was estimated as the sum of the diastolic blood pressure and one-third of the pulse pressure.

Statistical Analyses

The normality of data distributions was evaluated using the Kolmogorov-Smirnov test to select appropriate parametric and nonparametric statistical methods. For the comparison of the AKI and non-AKI groups and the CKD and non-CKD groups, collected data were analyzed. Categorical variables were analyzed using the Chi-square or Fisher's exact tests. Continuous variables were expressed as the median (25-75th percentile) and analyzed using the Mann-Whitney U test. To examine the risk factors associated with AKI or CKD, a logistic regression model was used to test each variable. Thereafter, multivariate logistic regression using backward stepwise variable elimination was applied after adjustment for confounding factors, defined as all factors found to be significant in the univariate analysis based on a type I error of 0.1. For all comparisons, tests were 2-tailed and group differences were regarded as statistically significant when p values were less than 0.05. SPSS version 18.0.0 statistical software for Windows (SPSS, Inc., Chicago, IL) was used for all analyses.

Results

Comparison of the AKI and non-AKI groups

Of the 105 patients included in the present study, 41 (39.0%) patients had AKI. Between the AKI and non-AKI groups, there was no significant difference in sex, age, BMI, smoking status, CCI, and underlying disease. Although the most common cause of renal infarction was embolism in both groups, idiopathic renal infarction was more common in the non-AKI group, whereas sepsis was more common in the AKI group (Table 1). With respect to clinical characteristics and treatment, the AKI group showed significantly greater MBP, longer length of stay, more frequent ICU admission, and hemodialysis treatment than the non-AKI group (Table 2). With respect to the CT and laboratory findings, AKI was more common in bilateral involvement than unilateral involvement. AKI group had higher C-reactive protein (CRP) levels, lactate dehydrogenase (LDH) levels, urine red blood cell (RBC) positivity, and urine albumin positivity than the non-AKI group (Table 3).

Comparison of the CKD and non-CKD groups

A total of 80 patients were followed up for 2 years after hospital discharge. Among these patients, 27 (33.8%) patients had CKD. Comparison of the CKD and non-CKD groups revealed that the CKD group patients were older and had higher CCI scores. Although the most common cause of renal infarction was embolism in both groups, idiopathic renal

Table 1. Baseline characteristics of patients with renal infarction

Characteristics	Non-AKI N = 64	AKI N = 41	*P* value	Non-CKD N = 53	CKD N = 27	*P* value
Sex (male)	40 (62.5)	24 (58.5)	0.685	35 (66.0)	14 (51.9)	0.218
Age (years)	59.0 (42.5-68.5)	59.0 (53.0-70.0)	0.144	55.0 (38.0-64.0)	66.0 (57.0-73.0)	0.001
BMI (Kg/m^2)	24.0 (20.6-26.0)	24.2 (22.3-26.3)	0.119	24.7 (23.1-27.3)	23.8 (21.0-26.2)	0.156
Smoking	29 (45.3)	20 (48.8)	0.728	24 (45.3)	12 (44.4)	0.943
CCI	3.0 (1.0-4.0)	3.0 (2.0-4.0)	0.719	2.0 (1.0-4.0)	3.0 (2.5-4.0)	0.041
Underlying disease						
Hypertension	18 (28.1)	16 (39.0)	0.244	14 (26.4)	12 (44.4)	0.104
Diabetes mellitus	11 (17.2)	5 (12.2)	0.487	6 (11.3)	5 (18.5)	0.494
Atrial fibrillation	28 (43.8)	22 (53.7)	0.321	24 (43.4)	15 (55.6)	0.303
Liver cirrhosis	2 (3.1)	1 (2.4)	1.000	1 (1.9)	0 (0.0)	1.000
Cancer	8 (12.5)	4 (9.8)	0.761	5 (9.4)	2 (7.4)	1.000
ACS	6 (9.4)	7 (17.1)	0.243	5 (9.4)	6 (22.2)	0.169
Cerebral infarction	5 (7.8)	5 (12.2)	0.507	4 (7.5)	3 (11.1)	0.683
Cause						
Embolism	33 (51.6)	25 (61.0)	0.344	27 (50.9)	17 (63.0)	0.307
Renal artery injury	5 (7.8)	4 (9.8)	0.734	4 (7.5)	2 (7.4)	1.000
Hypercoagulation	3 (4.7)	3 (7.3)	0.676	3 (5.7)	2 (7.4)	1.000
Sepsis	0 (0.0)	3 (7.3)	0.057	0 (0.0)	3 (11.1)	0.036
Cancer	3 (4.7)	1 (2.4)	1.000	2 (3.8)	0 (0.0)	0.547
Idiopathic	20 (31.3)	5 (12.2)	0.025	17 (32.1)	3 (11.1)	0.041

Results are presented as the total number (percentage) or the median (25-75th percentile). Abbreviations: ACS, acute coronary syndrome; AKI, acute kidney injury; BMI, body mass index; CCI, Charlson comorbidity index, CKD, chronic kidney disease.

Table 2. Clinical characteristics and treatment of patients with renal infarction

Characteristics	Non-AKI N = 64	AKI N = 41	*P* value	Non-CKD N = 53	CKD N = 27	*P* value
Initial vital sign						
MBP (mmHg)	97.3 (89.0-110.2)	106.7 (93.3-118.0)	0.012	96.3 (89.7-113.3)	110.0 (97.7-118.3)	0.023
Heart rate (rate/min)	83.5 (68.0-98.0)	75.0 (65.0-90.0)	0.290	84.0 (67.0-96.0)	75.0 (66.0-87.5)	0.413
Respiratory rate (rate/min)	20.0 (18.0-20.0)	20.0 (18.0-20.0)	0.559	20.0 (18.0-20.0)	20.0 (20.0-20.0)	0.557
Fever (≥ 37.3°C)	9 (14.1)	4 (9.8)	0.513	5 (9.4)	4 (14.8)	0.477
Symptom						
Pain severity (NRS)	6.5 (5.0-7.0)	7.0 (5.0-8.0)	0.182	7.0 (5.0-8.0)	7.0 (5.5-8.0)	0.767
Interval time (≥ 1 day)	53 (82.8)	34 (82.9)	0.988	45 (84.9)	22 (81.5)	0.753
Flank pain	39 (60.9)	27 (65.9)	0.611	36 (67.9)	17 (63.0)	0.657
Abdominal pain	44 (68.8)	25 (61.0)	0.413	37 (69.8)	19 (70.4)	0.959
Nausea	26 (40.6)	18 (43.9)	0.740	23 (43.4)	10 (37.0)	0.585
Sign						
CVAT	34 (53.1)	26 (63.4)	0.299	30 (56.6)	17 (63.0)	0.585
Abdominal tenderness	27 (42.2)	15 (36.6)	0.568	25 (47.2)	10 (37.0)	0.388
Hospital, length of stay (days)	7.0 (5.0-11.0)	10.0 (7.0-20.0)	0.008	8.0 (5.0-12.0)	10.0 (6.0-18.0)	0.053
ICU admission (days)	3 (4.7)	9 (22.0)	0.010	3 (5.7)	5 (18.5)	0.112
Treatment						
Antiplatelet	1 (1.6)	1 (2.4)	1.000	0 (0.0)	1 (3.7)	0.337
Anticoagulation	53 (82.8)	37 (90.2)	0.288	46 (86.8)	25 (92.6)	0.710
Thrombolysis	1 (1.6)	0 (0.0)	1.000	0 (0.0)	0 (0.0)	1.000
Endovascular procedure	0 (0.0)	1 (2.4)	0.390	1 (1.9)	0 (0.0)	1.000
Surgical intervention	3 (4.7)	1 (2.4)	1.000	2 (3.8)	1 (3.7)	1.000
Hemodialysis	0 (0.0)	6 (14.6)	0.003	1 (1.9)	4 (14.8)	0.042

Results are presented as the total number (percentage) or the median (25-75th percentile). Abbreviations: AKI, acute kidney injury; CKD, chronic kidney disease; CVAT, costovertebral angle tenderness; ICU, intensive care unit; MBP, mean blood pressure.

infarction was more common in the non-CKD group, whereas sepsis was more common in the CKD group (Table 1). With respect to the clinical characteristics and treatment, the CKD group had significantly greater MBP and received hemodialysis treatment more frequently than the non-CKD group (Table 2). With respect to CT and laboratory findings, CKD was more common in bilateral involvement than unilateral involvement. CKD group had higher albumin levels and urine albumin positivity than the non-CKD group. There was no statistical significance between AKI stage and development of CKD (Table 3).

Risk factors for AKI and CKD

In the univariate logistic regression analysis, MBP, bilateral involvement, idiopathic and sepsis as the cause, CRP and LDH level, urine RBC, and urine albumin positivity all showed statistical significance. However, in the multivariate analysis, only MBP (odds ratio [OR] 1.062, 95% confidence interval [CI] 1.015-1.112, p = 0.009) and bilateral involvement

Table 3. CT finding, laboratory data and AKI stage of patients with renal infarction

Variables	Non-AKI N = 64	AKI N = 41	*P* value	Non-CKD N = 53	CKD N = 27	*P* value
CT finding						
Involvement			0.001			0.001
Bilateral	9 (33.3)	18 (66.7)		8 (38.1)	13 (61.9)	
Unilateral	55 (70.5)	23 (29.5)		45 (76.3)	14 (23.7)	
Extra-renal involvement	10 (15.6)	7 (17.1)	0.844	4 (7.5)	4 (14.8)	0.432
Laboratory data at admission						
WBC (103/uL)	10.1 (8.3-13.4)	12.1 (9.1-14.5)	0.191	11.6 (8.9-14.2)	10.3 (8.4-13.5)	0.843
CRP (mg/dL)	0.7 (0.2-5.0)	2.9 (0.9-18.0)	0.013	0.9 (0.2-5.0)	2.3 (0.4-16.6)	0.296
LDH (IU/L)	441.0 (349.0-564.0)	602.0 (369.0-1164.0)	0.027	474.0 (347.0-558.0)	427.5 (283.5-1252.5)	0.102
Albumin (mg/dL)	3.8 (3.3-4.1)	3.4 (3.0-3.9)	0.182	3.9 (3.4-4.1)	3.5 (3.1-3.9)	0.078
Total bilirubin (mg/dL)	1.1 (0.8-1.3)	1.1 (0.8-1.3)	0.148	1.1 (0.8-1.3)	1.0 (0.8-1.4)	0.774
AST (IU/L)	37.0 (27.0-59.0)	52.0 (34.0-89.0)	0.215	44.0 (29.0-62.0)	38.0 (25.5-72.0)	0.534
ALT (IU/L)	34.0 (21.5-52.0)	46.0 (26.0-68.0)	0.200	40.0 (26.0-58.0)	39.0 (15.0-65.5)	0.617
Total cholesterol (mg/dL)	167.5 (137.5-186.5)	146.0 (133.0-181.0)	0.230	175.0 (145.0-187.0)	150.0 (140.0-200.5)	0.601
Creatinine (mg/dL)	0.9 (0.8-0.9)	1.2 (1.0-1.9)	< 0.001	0.9 (0.8-1.0)	1.1 (0.8-1.9)	0.025
BUN (mg/dL)	14.0 (9.0-17.0)	18.0 (14.0-30.0)	< 0.001	14.0 (10.0-17.0)	16.0 (13.5-28.5)	0.013
GFR (mL/min/1.73 m2)	85.0 (73.5-90.0)	53.0 (37.0-67.0)	< 0.001	84.0 (69.0-90.0)	54.0 (37.5-76.5)	< 0.001
Urine RBC (Positive)	30 (48.4)	28 (70.0)	0.031	27 (51.9)	18 (69.2)	0.145
Urine WBC (Positive)	8 (12.9)	9 (22.5)	0.204	6 (11.5)	7 (26.9)	0.111
Urine Albumin (Positive)	15 (24.2)	19 (47.5)	0.015	14 (26.9)	12 (46.2)	0.089
AKI stage						0.791
Stage 1		26		7	10	
Stage 2		7		3	4	
Stage 3		8		2	6	

Results are presented as the total number (percentage) or the median (25-75th percentile). Abbreviations: AKI, acute kidney injury; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CKD, chronic kidney disease; BUN, blood urea nitrogen; CRP, C-reactive protein; GFR, glomerular filtration rate; LDH, lactate dehydrogenase; MBP, mean blood pressure; RBC, red blood cell; WBC, white blood cell .

(OR 4.396, 95% CI 1.096-17.632, p = 0.037) had sufficient statistical power to discriminate between the two groups after adjustment for all variables (Table 4).

In the univariate logistic regression analysis, AKI, age, hemodialysis treatment, MBP, CCI, albumin and urine albumin levels, and urine WBC positivity, except idiopathy and sepsis as the cause, were all statistically higher in the CKD group than in the non-CKD group. In the subsequent multivariate analysis, only AKI (OR 14.799, 95% CI 4.173-52.490, p < 0.001) and old age (OR 1.065, 95% CI 1.016-1.116, p = 0.009) showed statistical significance for discriminating between the two groups after adjustment for all variables (Table 4).

Table 4. Multivariate analysis of predicting factors for AKI and CKD

Variables	Odds ratio	95% Confidence Interval	*P* value
AKI			
MBP (mmHg)	1.062	1.015-1.112	0.009
Bilateral involvement	4.396	1.096-17.632	0.037
CRP (mg/dL)	1.059	0.996-1.126	0.067
LDH (mg/dL)	1.001	1.000-1.003	0.064
CKD			
AKI	14.799	4.173 – 52.490	< 0.001
Age (years)	1.065	1.016 – 1.116	0.009

Abbreviations: AKI, acute kidney injury; CKD, chronic kidney disease; CRP, C-reactive protein; LDH, lactate dehydrogenase; MBP, mean blood pressure.

Discussion

The present study demonstrated that the incidence of AKI of 39.0% and that of CKD of 33.8% in patients with renal infarction. Independent risk factors for AKI were high MBP and bilateral involvement and whereas those for CKD were old age and AKI.

Incidence of AKI and CKD after renal infarction

Our incidence of AKI (39.0%) was reasonable compared with previous study results in Table 5. However, in CKD defined variously according to each study, the incidence rates were heterogeneous and our result (33.8%) was relatively higher than that reported by other

studies. Even considering these results, this present study included patients with relatively severe renal infarction due to its retrospective design. Urine output could not be used as a criterion for AKI. Hence, a study estimating the actual incidence of AKI and CKD after renal infarction is needed in the future. In particular, clinicians pay attention to patients with flank pain and increased LDH level or atrial fibrillation, and consider a CT scan with enhancement when renal infarction is suspected. In the present study, only eight patients (7.6%) had LDH level in the normal range and 50 patients (47.6%) had atrial fibrillation.

Table 5. Literature review of AKI and chronic renal failure after acute renal infarction

References	Year	Number of patients	Acute kidney injury	Chronic kidney disease
Lessman et al. [11]	1978	17	64.7%	11.8%
Korzets et al. [12]	2002	11	27.3%	9.1%
Hananov et al. [13]	2004	44	39.5%	7.9%
Tsai et al. [14]	2007	18	11.1%	5.6%
Antopolsky et al. [15]	2011	38	7.9%	NA
Rhee et al. [5]	2012	67	40.7%	1.5%
Bourgault et al. [2]	2013	94	40.4%	NA
Lin et al. [6]	2014	40	NA	32.5%
Bae et al. [7]	2014	100	30.0%	7.0%
Oh et al. [8]	2015	422	20.9%	10.9%
Ha et al. [present study]	2016	105	39.9%	33.8%

High blood pressure and bilateral involvement in patients with AKI

In the present study, high blood pressure was a risk factor for AKI. Bourgault et al. demonstrated that an increase in blood pressure was frequently observed at the time of renal infarction diagnosis (48% of cases) [2]. In an animal model, activation of renin-angiotensin system plays a role in the onset of partial renal infarct hypertension [16]. Clinically, Paris reported that the ischemia induced activation of renin-angiotensin system resulted in hypertension in patients with renal infarction [17]. Subsequently, the elevated blood pressure leads to high intra-glomerular pressure and glomerular filtration impairment [18]. This pathophysiology may have contributed to our results. In the present study, optimal cutoff value of MBP was 105 mmHg (area under the curve 0.642, sensitivity 53.7%, and specificity 68.8%) for discriminating between the AKI and non-AKI groups in the receiver operating characteristic curve (data not shown). Furthermore, we suggest that bilateral involvement and large infarction size, which results in greater renin-angiotensin secretion than small involvement, may be associated with higher blood pressure. This suggestion was supported by the results of Bae et al., who showed that the renal infarct size was positively correlated with the occurrence of AKI [7]. In conclusion, we suggested that AKI was developed due to more extent of involvement itself and high blood pressure effect. Although there was no statistical significance, another considerable laboratory finding related with ischemia was CRP. CRP is a marker of systemic inflammation and promotes ischemia reperfusion kidney injury [19, 20].

AKI and old age for patients with CKD

Consistent with the findings of Goldstein and Coca [21, 22], we found that AKI was a major risk factor for CKD progression. Although the reasons why AKI would increase the risk of CKD remain unknown, Yang et al. suggested that inappropriate vascular, interstitial, and tubular regeneration after AKI may lead to renal fibrosis and progressive kidney failure [23]. Heung et al. and Bucaloiu et al. emphasized that acute kidney injury, even when mild or followed by full recovery, is associated with an increased risk for future development of CKD [24, 25]. In the present study, CKD occurred in 50% of patients with AKI. It is reasonable to pay attention and recommend long term follow up to the patients with an initial AKI episode.

Nephron compensatory adaptation initially allows maintaining normal GFR after renal infarction. However, relative small reservoir in old age may be a potential risk for CKD.

Goldstein et al. and Macedo et al. suggested that advanced age is the most significant risk factor for non-recovery of renal function [22, 26]. Lin et al. and Rhee et al. also showed that, using multivariate analysis, old age was found to be an independent risk factor for CKD and long-term mortality. In the present study, optimal cutoff value of age was 65 years (area under the curve 0.691, sensitivity 52.0%, and specificity 83.0%) for discriminating between the CKD and non-CKD groups in the receiver operating characteristic curve (data not shown).

There were several limitations to the present study. First, there could have been selection bias due to the study's retrospective design. Moreover, there was a possibility of not including patients with silent renal infarction without remarkable symptoms and signs. Second, although we evaluated the risk factors for AKI and CKD by using the multivariate analysis after adjusting the cause to be sepsis, there remained a doubt regarding the cause for AKI and CKD because of the small sample number. The dehydrated condition and the contrast media-induced AKI cannot be evaluated. Third, previous presence of CKD was uncertain when there was no objective record in the EMR; we relied on only the history taken. Fourth, too small a number of patients were included due to the rarity of renal infarction incidence. Fifth, this was a single center study; our results may not be generalizable. A more comprehensive and well-designed prospective study focused on this issue should be performed.

Conclusions

In conclusion, high mean blood pressure and bilateral involvement were independent risk factors for AKI, and old age and AKI for CKD. Physicians should pay attention to the development of AKI and CKD after renal infarction and follow patients over a long term.

Disclosure Statement

The authors declare that they have no competing interest.

References

1 Piccoli GB, Priola AM, Vigotti FN, Guzzo G, Veltri A: Renal infarction versus pyelonephritis in a woman presenting with fever and flank pain. Am J Kidney Dis 2014;64:311-314.
2 Bourgault M, Grimbert P, Verret C, Pourrat J, Herody M, Halimi JM, Karras A, Amoura Z, Jourde-Chiche N, Izzedine H, Francois H, Boffa JJ, Hummel A, Bernadet-Monrozies P, Fouque D, Canoui-Poitrine F, Lang P, Daugas E, Audard V: Acute renal infarction: a case series. Clin J Am Soc Nephrol 2013;8:392-398.
3 Domanovits H, Paulis M, Nikfardjam M, Meron G, Kurkciyan I, Bankier AA, Laggner AN: Acute renal infarction. Clinical characteristics of 17 patients. Medicine (Baltimore) 1999;78:386-394.
4 Huang CC, Lo HC, Huang HH, Kao WF, Yen DH, Wang LM, Huang CI, Lee CH: ED presentations of acute renal infarction. Am J Emerg Med 2007;25:164-169.
5 Rhee H, Song SH, Won Lee D, Lee SB, Kwak IS, Seong EY: The significance of clinical features in the prognosis of acute renal infarction: single center experience. Clin Exp Nephrol 2012;16:611-616.
6 Lin WL, Seak CJ, Wu JY, Weng YM, Chen HC: Risk factors for development of chronic kidney disease following renal infarction: retrospective evaluation of emergency room patients from a single center. PLoS One 2014;9:e98880.
7 Bae EJ, Hwang K, Jang HN, Kim MJ, Jeon DH, Kim HJ, Cho HS, Chang SH, Park DJ: A retrospective study of short- and long-term effects on renal function after acute renal infarction. Ren Fail 2014;36:1385-1389.
8 Oh YK, Yang CW, Kim YL, Kang SW, Park CW, Kim YS, Lee EY, Han BG, Lee SH, Kim SH, Lee H, Lim CS: Clinical Characteristics and Outcomes of Renal Infarction. Am J Kidney Dis 2016;67:243-250.

9 Group KDIGOKAKIW: KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2:19-22.
10 National Kidney F: K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 2002;39:S1-266.
11 Lessman RK, Johnson SF, Coburn JW, Kaufman JJ: Renal artery embolism: clinical features and long-term follow-up of 17 cases. Ann Intern Med 1978;89:477-482.
12 Korzets Z, Plotkin E, Bernheim J, Zissin R: The clinical spectrum of acute renal infarction. Isr Med Assoc J 2002;4:781-784.
13 Hazanov N, Somin M, Attali M, Beilinson N, Thaler M, Mouallem M, Maor Y, Zaks N, Malnick S: Acute renal embolism. Forty-four cases of renal infarction in patients with atrial fibrillation. Medicine (Baltimore) 2004;83:292-299.
14 Tsai SH, Chu SJ, Chen SJ, Fan YM, Chang WC, Wu CP, Hsu CW: Acute renal infarction: a 10-year experience. Int J Clin Pract 2007;61:62-67.
15 Antopolsky M, Simanovsky N, Stalnikowicz R, Salameh S, Hiller N: Renal infarction in the ED: 10-year experience and review of the literature. Am J Emerg Med 2012;30:1055-1060.
16 Norman RA, Jr., Galloway PG, Dzielak DJ, Huang M: Mechanisms of partial renal infarct hypertension. J Hypertens 1988;6:397-403.
17 Paris B, Bobrie G, Rossignol P, Le Coz S, Chedid A, Plouin PF: Blood pressure and renal outcomes in patients with kidney infarction and hypertension. J Hypertens 2006;24:1649-1654.
18 Yoshioka T, Rennke HG, Salant DJ, Deen WM, Ichikawa I: Role of abnormally high transmural pressure in the permselectivity defect of glomerular capillary wall: a study in early passive Heymann nephritis. Circ Res 1987;61:531-538.
19 Tang Y, Huang XR, Lv J, Chung AC, Zhang Y, Chen JZ, Szalai AJ, Xu A, Lan HY: C-reactive protein promotes acute kidney injury by impairing G1/S-dependent tubular epithelium cell regeneration. Clin Sci (Lond) 2014;126:645-659.
20 Pegues MA, McCrory MA, Zarjou A, Szalai AJ: C-reactive protein exacerbates renal ischemia-reperfusion injury. Am J Physiol Renal Physiol 2013;304:F1358-1365.
21 Coca SG, Singanamala S, Parikh CR: Chronic kidney disease after acute kidney injury: a systematic review and meta-analysis. Kidney Int 2012;81:442-448.
22 Goldstein SL, Jaber BL, Faubel S, Chawla LS, Acute Kidney Injury Advisory Group of American Society of N: AKI transition of care: a potential opportunity to detect and prevent CKD. Clin J Am Soc Nephrol 2013;8:476-483.
23 Yang L, Besschetnova TY, Brooks CR, Shah JV, Bonventre JV: Epithelial cell cycle arrest in G2/M mediates kidney fibrosis after injury. Nat Med 2010;16:535-543. 1p following 143.
24 Heung M, Chawla LS: Predicting progression to chronic kidney disease after recovery from acute kidney injury. Curr Opin Nephrol Hypertens 2012;21:628-634.
25 Bucaloiu ID, Kirchner HL, Norfolk ER, Hartle JE, 2nd, Perkins RM: Increased risk of death and de novo chronic kidney disease following reversible acute kidney injury. Kidney Int 2012;81:477-485.
26 Macedo E, Zanetta DM, Abdulkader RC: Long-term follow-up of patients after acute kidney injury: patterns of renal functional recovery. PLoS One 2012;7:e36388.