Kidney Res Clin Pract 35 (2016) 90–95

Kidney Research and Clinical Practice

journal homepage: http://www.krcp-ksn.com
Contents lists available at ScienceDirect

Original Article

Risk factors and outcomes of acute renal infarction

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Article history:
Received 16 December 2015
Accepted 23 April 2016
Available online 11 May 2016

Keywords:
Acute kidney injury
Chronic kidney disease
Infarction
Renal artery

Abstract

Background: Renal infarction (RI) is an uncommon disease that is difficult to diagnose. As little is known about clinical characteristics of this disease, we investigated its underlying risk factors and outcomes.
Methods: We performed a retrospective single-center study of 89 patients newly diagnosed with acute RI between January 2002 and March 2015 using imaging modalities. Clinical features, possible etiologies, and long-term renal outcome data were reviewed.
Results: The patients' mean age was 63.5 ± 15.42 years; 23.6% had diabetes and 56.2% had hypertension. Unilateral and bilateral involvements were shown in 80.9% and 19.1% of patients, respectively; proteinuria and hematuria were reported in 40.4% and 41.6%, respectively. Cardiovascular disease was the most common underlying disease, followed by renal vascular injury and hypercoagulability disorder. Fourteen patients had no specific underlying disease. At the time of diagnosis, acute kidney injury (AKI) was found in 34.8% of patients. Univariate analysis revealed diabetes mellitus (DM), leukocytosis, and high C-reactive protein (CRP) as significant risk factors for the development of AKI. On multivariate analysis, DM and high CRP levels were independent predictors for AKI. During follow-up, chronic kidney disease developed in 27.4% of patients. Univariate and multivariate Cox regression analyses showed old age to be an independent risk factor for this disease, whereas AKI history was a negative risk factor.
Conclusion: DM patients or those with high CRP levels should be observed for renal function deterioration. Clinicians should also monitor for RI in elderly patients.

Introduction

Renal infarction (RI) is an infrequent condition that is related to atheroembolic or thromboembolic disease [1]. The first case of renal embolic disease was reported in 1856 [2], and several more cases and clinical analyses have been published since. Most reported cases have underlying causes, such as hypercoagulability, cardiac problems, trauma history, or previous cerebral infarction [2–4]. Various comorbidities, such as infection, malignancy, cocaine abuse, and autoimmune disease, may also contribute to RI [5–9]. Abdominal or flank pain is a common clinical manifestation, although some patients do not experience any subjective symptoms. Therefore, RI is difficult to detect unless a clinician specifically checks for its presence and is thus likely to be underdiagnosed [1,3,4,10–12]. Postmortem

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http://dx.doi.org/10.1016/j.krcp.2016.04.001

examinations in 1 study revealed that the incidence of RI was 1.4% (205 of 14,411) with only 2 of these cases having been clinically diagnosed antemortem [13]. Another study reported an RI incidence of 0.007% during a 36-month observation period [14].

With the advent of innovative imaging technologies, incidental RI cases are detected more frequently [4]. Improved accessibility to contrast-enhanced computed tomography can also reveal more RI cases. Nowadays, RI can be detected in patients who visit hospitals with nonspecific symptoms and no demonstrable underlying causes.

A partial RI animal model induced by renal artery ligation revealed increased plasma renin activity and development of hypertension [15], but there are no firm data regarding long-term follow-up results in patients after RI. Reduced kidney blood flow and mass reduction can cause deterioration of renal function, although hyperperfusion of the viable renal glomeruli can compensate for this [1]. In this context, RI may be thought of as a completely reversible disease. However, there are also patients who progress to a state where they require dialysis [8].

The purpose of this study was 2-fold. First, we aimed to reveal the clinical etiology and characteristics of RI. Second, we investigated the acute and long-term changes in renal function due to RI and determined the factors associated with renal functional deterioration.

Methods

Patients

This was a retrospective, single-center study of 89 patients newly diagnosed with acute RI between January 2002 and March 2015. Diagnosis was based on a radiologist's report of imaging modalities, such as computed tomography, sonography, and angiography. Patients who visited the emergency department or underwent imaging studies during hospitalization were included. The creatinine level measured 7–365 days before the study was considered as the baseline value [16]. If there were no preexisting creatinine data, baseline creatinine was regarded as the healthiest creatinine level measured. Patients younger than 18 years were excluded. Clinical manifestations and possible etiologies were extracted from initial emergency room medical records or admission notes. We used the Risk, Injury, Failure, Loss, and End-stage kidney disease criteria to define acute kidney injury (AKI) as measured by a change in serum creatinine levels within 1–7 days from the initial renal insult [17]. Development of chronic kidney disease (CKD) was defined as a creatinine-based estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m^2 over 3 months using the Modification of Diet in Renal Disease equation [18]. For long-term outcome analysis, we excluded patients whose eGFRs were < 60 mL/min/1.73 m^2 at baseline. Patients who have more than 1 underlying medical condition were assessed by using Charlson comorbidity index (CCI) score [19]. This study was approved by the Korea University Anam Hospital Institutional Review Board (2015-04-0345).

Statistical analysis

The IBM SPSS statistics software, version 20 (IBM analytics, Chicago, IL, USA) was used for statistical analyses. Comparison between 2 groups was performed using the *t* test for numerical data and the chi-square test or Fisher exact test for categorical data. Univariate logistic tests were conducted for AKI and RI. Results that followed normal distribution were presented as mean values. In contrast, if the results did not follow normal distribution, the median value was used. We used multivariate Cox regression tests to identify predictors of long-term decreased renal function because of RI diagnosis. All demographic factors, comorbidities, laboratory data, treatment options, and first manifestations of symptoms were included in the list of possible risk factors for decreasing renal function.

Results

A total of 89 acute RI patients were identified during the 13 years. The mean follow-up period was 33.45 ± 29.04 months with a maximum of 119 months. Six patients had no medical record because of discharge or follow-up loss (Fig. 1). Four patients transferred to another hospital before 3 months had died, whereas 3 patients were excluded from the analysis of long-term renal function because of self-discharge. Five patients died of pancreatic cancer, breast cancer, bladder cancer, myelodysplastic syndrome, and intracranial hemorrhage, respectively, whereas 3 patients died of sepsis. Nine patients exhibited reduced renal function at the first follow-up time point with an eGFR below 60 mL/min/1.73 m^2. Hence, 59 patients were ultimately included for analysis of the long-term change in renal function at 1 year or more after RI diagnosis.

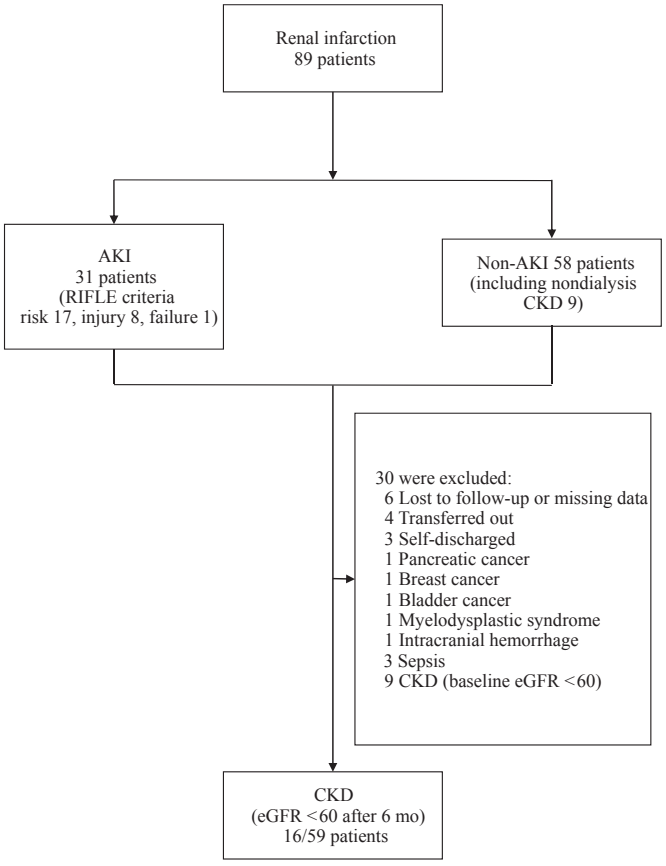

Figure 1. Flowchart of patient outcome.
AKI, acute kidney injury; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; RIFLE, Risk, Injury, Failure, Loss, and End-stage kidney disease.