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NEPHROLOGY - ORIGINAL PAPER

Acute renal infarction in Turkey: a review of 121 cases

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Abstract

Purpose Renal infarction is a clinical condition which is caused by renal artery occlusion and leads to permanent renal parenchymal damage. In the literature, there are generally case reports on this subject, and few studies that include a large group of patients. Therefore, we aimed to present the data of a large group of patients who were diagnosed with acute renal infarction in our country in this retrospective study.

Methods The data of patients who were diagnosed with acute renal infarction according to clinical and radiological findings in Turkey in the last 3 years were examined. For this purpose, we contacted with more than 40 centers in 7 regions and obtained support from clinically responsible persons. Demographic data of patients, laboratory data at the time of diagnosis, tests performed for etiologic evaluation, given medications, and patients' clinical status during follow-up were obtained from databases and statistical analysis was performed.

Results One-hundred and twenty-one patients were included in the study. The mean age was 53 ± 1.4 (19–91) years. Seventy-one (58.7%) patients were male, 18 (14.9%) had diabetes, 53 (43.8%) had hypertension, 36 (30%) had atrial fibrillation (AF), and 6 had a history of lupus + antiphospholipid syndrome (APS). Forty-five patients had right renal infarction, 50 patients had left renal infarction, and 26 (21.5%) patients had bilateral renal infarction. The examinations for the ethiologies revealed that, 36 patients had thromboemboli due to atrial fibrillation, 10 patients had genetic anomalies leading to thrombosis, 9 patients had trauma, 6 patients had lupus + APS, 2 patients had hematologic diseases, and 1 patient had a substance abuse problem. Fifty-seven (57%) patients had unknown. The mean follow-up period was 14 ± 2 months. The mean creatinine and glomerular filtration rate (GFR) values at 3 months were found to be 1.65 ± 0.16 mg/dl and 62 ± 3 ml/min, respectively. The final mean creatinine and GFR values were found to be 1.69 ± 0.16 mg/dl and 62 ± 3 ml/min, respectively.

Conclusions Our study is the second largest series published on renal infarction in the literature. More detailed studies are needed to determine the etiological causes of acute renal infarction occurring in patients.

Keywords Acute kidney injury · Etiology · Renal infarction · Turkey

Introduction

Renal infarction is a clinical condition which is caused by renal artery occlusion that leads to permanent renal parenchymal damage. Studies have shown that its incidence among all emergency admissions is as low as 0.007% [1]. Therefore, misdiagnosis or delayed diagnosis for renal infarctions are often encountered. Although the average age of onset varies according to etiology, it is usually seen in patients over 40 years of age. It often begins as a sudden onset of severe flank pain. Patients may also present with nonspecific symptoms, including nausea, vomiting, fever and sudden-onset high blood pressure. In laboratory data, elevated serum creatinine and lactate dehydrogenase (LDH) levels can be seen depending on the degree of renal parenchymal involvement [2]. Contrast-enhanced computer

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tomography (CECT) is used for the diagnosis of renal infarction in a radiological manner. CECT shows the existence of hypodense lesion shaped like a wedge in the peripheral area.

For the causality of renal infarctions, three etiologic groups, including cardioembolic diseases, renal artery injury and conditions of hypercoagulability are most frequently accused [3]. However, there is also a group of renal infarctions with unclassified etiologies except for these three groups. Atrial fibrillation constitutes a major part of cardioembolic diseases which are the most commonly seen etiologies. It was reported in previous studies that the following diseases were associated with renal infarctions; vascular inflammatory diseases (such as Polyarteritis Nodosa, Takayasu's Arteritis, Kawasaki's Disease, and Behcet's Disease); nephrotic syndrome; infectious diseases (like Syphilis); and Sickle Cell Anemia [2, 3]. The first case of renal embolic disease was reported in 1856. Since then, some more case studies have been published in the literature [2–5].

In this retrospective study, we aimed to present the demographic data, etiology, medications and outcomes of patients who were diagnosed with acute renal infarction in the last 3 years in our country.

Patients and methods

This study was planned to involve nephrology specialists from more than 40 universities and education-research hospitals in seven regions of our country. To achieve more accurate data, the discharge reports and laboratory data of the patients who were admitted to the hospital due to acute renal infarction in the last 3 years were retrospectively screened. According to this information, only the patients who were definitely diagnosed with acute renal infarction based on clinical, radiological and laboratory findings were included in the study. The diagnosis of the acute renal infarction was made in patients who applied to the hospital with suddenly starting abdominal pain or side pain and with high LDH levels in laboratory tests, with radiologic appearances in favor of renal infarction in contrasted and/or unenhanced abdominal CT, and in patients in whom other clinical diagnoses (especially urinary infections) were excluded. Diagnosis of acute kidney injury (AKI) was based on KDIGO 2012 (only the change in creatinine was considered (after 2 or 7 days)) criteria.

The age and gender of the patients, their additional diseases (diabetes, hypertension, coronary artery disease, rheumatological disease, etc.), localization (right-left kidney) and size (percentage covered) of the infarct, laboratory data on admission (creatinine, LDH, etc.), glomerular filtration rate (GFR) estimated by the Modification of Diet in Renal Disease (MDRD) equation, cardiac evaluation (echocardiography), immunological assays (antinuclear antibody (ANA), anti-dsDNA, C3, C4, anticardiolipin antibodies), hematological evaluation (protein C, protein S, antithrombin 3, homocysteine, active protein C resistance values), and genetic evaluation (methylenetetrahydrofolate reductase (MTHFR) mutation, Factor V Leiden mutation, etc.) were screened via the databases. The etiologies of renal infarctions in the patients were examined in detail through the information in the discharge reports. Medications given at the time of diagnosis and after discharge (thrombolytic therapy, heparin, etc.), average length of stay in hospital, renal functions (dialysis requirement, creatinine and eGFR values at 3 months and at the end of mean follow-up period) during follow-up, and outcomes were evaluated.

Results

One hundred and twenty-one patients were included in the study. All cases were diagnosed based on clinical findings and infarct areas on computerized tomography. The mean age was 53 ± 1.4 (19–91) years. Seventy-one (58.7%) patients were male. Of the patients, 18 (14.9%) had diabetes, 53 (43.8%) had hypertension, 36 (30%) had atrial fibrillation (AF), and 6 had a history of systemic lupus erythematosus and antiphospholipid antibody syndrome.

Forty-five patients had right renal infarction, 50 patients had left renal infarction, and 26 (21.5%) patients had bilateral renal infarction. The average infarct size was 44% in the right kidney and 47% in the left kidney, respectively. The mean serum creatinine and eGFR values at the time of admission were 1.5 ± 0.1 mg/dl (0.38–6.4) and 68 ± 3 ml/min (6–132), respectively. Forty-three (35.5%) patients had AKI and 5 patients required dialysis. The mean serum LDH value at the time of admission was 696 ± 93 U/l (172–8946). Demographic and laboratory data of the patients are presented in Table 1. The patients with and without AKI were compared among themselves and there were no significant differences in terms of comorbidities (Table 2).

Forty-three patients underwent renal angiography at the time of admission. Twenty-six patients had a thrombus-like appearance. Fourteen patients underwent stenting. Three patients underwent thrombolytic therapy.

When the etiology of the patients was examined, 36 patients were considered to have thromboembolism due to atrial fibrillation. However, only 6 of these patients had a thrombus-like appearance on echocardiography. Nine patients had a history of abdominal and renal trauma, 10 patients had genetic anomalies leading to thrombosis (six of them had MTHFR C677T mutation, three had Factor V Leiden mutation and one patient had documented PAI mutation), six patients had systemic lupus erythematosus (SLE) + antiphospholipid antibody syndrome (APS), two patients had hematologic diseases, and one patient had a

Table 1 Demographic and laboratory data of the patients

Number of patients	121
Age (years)	53 ± 1.4 (19–91)
Gender: male/female (%)	58/42
DM (%)	18.9
HT (%)	43.8
Renal infarct localization (%)	Right: 37% Left: 41.3% Bilateral: 21.5%
Admission serum creatinine (mg/dl)	1.5 ± 0.1
Admission eGFR (ml/dk)	68 ± 3
Acute kidney injury ratio (%)	35.5
Acute dailysis require (%)	4
Mean serum LDH (U/l)	696 ± 93
Mean hospitalization time (day)	11 ± 1
Etiology of the patients (%)	Atrial fibrilation: 29.7 Genetic anomalies: 8 Trauma: 7 SLE + APS: 5 Hematologic diseases: 2 Substance abuse: 1 Unknown: 47

eGFR estimated glomerular filtration rate, *SLE* systemic lupus erythematosus, *APS* antiphospholipid antibody syndrome, *LDH* lactate dehydrogenase

Table 2 Comparison of with or without acute kidney injury groups

	Acute kidney injury (+) (35%)	Acute kidney injury (−) (65%)	*p* value
Age (years)	55 ± 16	52 ± 15	0.32
Gender (M/F) (%)	4/25	27/14	0.23
HT (%)	20.7	23.1	0.07
DM (%)	6.6	8.3	0.39
Atrial fibrilation (%)	12.6	13.4	0.16
Bilateral involvement (%)	12.6	9.2	0.07
Admition LDH (U/l)	875 ± 135	575 ± 493	0.11
Creatinine (third month) (mg/dl)	2.4 ± 1.6	0.9 ± 0.5	0.000
eGFR (third month) (ml/min)	42 ± 21	79 ± 26	0.000
Last eGFR (ml/min)	41 ± 30	76 ± 28	0.000

substance abuse problem (ecstasy). Forty-seven (57%) patients had no revealed etiological cause. Of these patients, 44 (83%) were screened for SLE, 16 (30%) were screened for APS. 17 (32%) were evaluated for hematological etiologies and only 4 (7%) were evaluated for genetic predisposition.

The average length of stay in hospital was 11 ± 1 days. At the time of discharge, warfarin was prescribed for 50 patients (to all of the patients with atrial fibrillation), and low molecular weight heparin was prescribed for 38 patients. A total of 33 patients were not given anticoagulant treatment. These patients were those whose etiologies could not be determined. The mean follow-up period was 14 ± 2 months. The mean creatinine and eGFR values at 3 months were found to be 1.65 ± 0.16 mg/dl and 62 ± 3 ml/min, respectively. The final mean creatinine and GFR values were found to be 1.69 ± 0.16 mg/dl and 62 ± 3 ml/min, respectively. Chronic kidney disease (CKD) developed in 28.9% of patients during an average follow-up of 14 months. The risk of developing of CKD was also higher in patients with AKI than in patients without AKI. A total of 4 patients had stage 2, 12 patients had stage 3, 11 patients had stage 4 and 8 patients had stage 5 CKD. When the etiologies of the patients who developed CKD were examined, it was determined that there were thromboembolic events in 11 patients, SLE + APS in 1 patient, trauma in 2 patients, genetic disease in 5 patients; and no causes were determined in 16 patients. Only two of the patients who developed CKD recovered to initial renal functions, nonetheless CKD developed at follow-up. Four patients were included in the chronic dialysis program in the follow-up period. Six patients died during follow-up. While the etiology of renal infarction in two of these patients was atrial fibrillation, the other four patients had unknown etiological cause.

Discussion

In this retrospective study, we evaluated the patients who were diagnosed with acute renal infarction in the last 3 years in different regions of our country. Similar to the previous literature, cardioembolic diseases were the most common cause of renal infarction in our country. Interestingly, 47% of the patients had no defined etiological cause in our study. However, we found that further diagnostic tests of these patients were insufficient which might have affected this result.

Renal infarction is a very rare condition in nephrology practice. The causes of low incidence may be the fact that there are no specific clinical signs and symptoms of renal infarction [6]. Besides, its diagnosis may be confused with urinary system stone disease, lumbalgia or other intra-abdominal pathologies (which have similar symptomatology and are more common), and therefore, it is not considered in first order during clinical differential diagnosis [7].

Renal infarction can affect one or two kidneys at varying degrees. In a retrospective study of Yang et al., 19.1% of the patients had bilateral renal infarction [8] which was similar to our study results (21.5%). They reported that 34.8% of the patients with acute renal infarction had renal failure at the time of diagnosis. This rate was also similar in our study (35.5%). In our study, the patients with and without AKI were compared among themselves, there were

no significant differences in terms of comorbidities such as diabetes, hypertension, and atrial fibrillation. The prognosis could be worse in bilateral infarctions [8]. In our study, AKI developed in 58% of patients with bilateral involvement.

Unfortunately, patients with renal infarction are at risk for developing CKD. In the study of Yang et al., this rate was 27.4% [8]. In our study, CKD developed in 28.9% of patients during an average follow-up of 14 months. The risk of developing CKD was also higher in patients with AKI than in patients without AKI.

Acute renal infarction is usually seen in middle-aged patients with atherosclerotic cardiovascular disease and with atrial fibrillation in clinical practice [9]. This has also been shown in retrospective studies [8, 9]. In our study, 43.8% had hypertension and 30% had atrial fibrillation. When renal infarction occurs in patients with atrial fibrillation, the patient should be evaluated by echocardiography. The presence of thrombus in the heart wall or any of the valves should definitely be investigated. However, it may not be possible to demonstrate an intracardiac thrombus in every patient. Our patients were assessed by echocardiography and only six patients had a thrombus-like appearance on the heart valve. In cases with renal infarction and concurrent atrial fibrillation, it may not be necessary to investigate other etiological causes. However, a detailed examination may be required in the presence of systemic findings. In our study, we observed that most of the patients who were considered to have AF in etiology did not undergo additional clinical examination.

Renal damage can be seen in 8–10% of trauma patients [10]. The incidence of renal infarction due to blunt abdominal trauma is not fully known. There are many case reports in the literature. In a retrospective study of Nagasawa et al. [3], the etiology was attributed to trauma in 9% of patients with renal infarction. This rate was similar in our study (7%). The infarct can be unilateral or bilateral according to the size and localization of the trauma. Renal infarct related to trauma was unilateral in all of our patients. Traumatic renal artery injury can lead to renal infarction in the early period and later. To accuse trauma alone in etiology in these patients, it is necessary to conduct a detailed investigation of other causes that may lead to renal infarction. In all of the patients who were diagnosed with traumatic renal infarction in our series, the diagnosis was made after other causes were examined and excluded in detail.

Inherited predisposition to thrombosis is one of the rare causes of acute renal infarction. Case reports considered to develop acute renal infarction due to MTHFR C677T and Factor V Leiden mutations have been reported [11, 12]. Ten patients had the genetic background which may constitute susceptibility to develop renal infarction. Six of them had MTHFR C677T mutation, three had Factor V Leiden mutation and one patient had documented PAI mutation. AKI developed in two of these patients. The MTHFR C677T mutation can lead to homocysteinemia [13], facilitating the development of renal infarction. Homocysteine levels were examined and found high in only two of six patients. Primary or secondary antiphospholipid antibody syndrome (APS) can also result in renal infarctions [14]. Secondary forms are usually associated with SLE. In our study, six patients had SLE + APS.

Substance abuse is a very rare cause of renal infarction. There are two patients in the literature who developed renal infarction due to the use of ecstasy [15]. One of these patients is in our series. Although the exact mechanism is unfortunately unknown, 3,4-methylene dioxymethamphetamine-induced vasospasm and increased susceptibility to thrombosis may be responsible. Both patients in the literature were diagnosed after all the possible causes of renal infarction were excluded.

In our study, 57 (47%) patients had no defined etiological cause. This rate was 11.2% in the study by Yang et al. [8]. All of them were evaluated with echocardiograpy and none had cardiac thrombus and atrial fibrillation. Of these patients, 44 (83%) were screened for SLE (ANA, anti-dsDNA, C3, C4 levels), 16 (30%) were screened for antiphospholipid antibody syndrome (autoantibodies), 17 (32%) were evaluated for hematological etiologies (protein C, protein S, antithrombin III, activated protein C resistance, homocysteine etc.), and only 4 (7%) were evaluated for genetic predisposition. The rate of patients whose etiology could not be determined was high in our study. In our opinion, this result could be due to insufficient investigation of these patients in terms of immunologic, hematologic and genetic factors. Although atrial fibrillation and thromboembolism are the most common causes of acute renal infarction, patients with acute renal infarction without these risk factors should be evaluated in more detail.

In the literature, patients with acute renal infarction are generally presented as case reports, and there are few studies in which the data on a large number of patients have been examined [16]. Our data can be a source in terms of tests done to determine the etiology of patients presenting with acute renal infarction. Our retrospective study has examined the data of patients diagnosed with acute renal infarction in the last 3 years in our country.

Our study had some limitations. Because of the retrospective design of our study, we were only able to obtain the laboratory results, determined etiologic causes and documented medications of the patients that were recorded in the databases.

As a conclusion, acute renal infarction is a rare but very important nephrological problem. It must be kept in mind in the differential diagnosis of flank pain and AKI. Patients should be examined in detail in terms of etiological factors and treated properly.

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Compliance with ethical standards

Conflict of interest The authors declare no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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