A Series of Patients with Renal Infarction: Presentation and Impact on Renal Function

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Abstract

Introduction: Renal infarction is a rare pathology that often has a delayed diagnosis because of its non-specific symptoms. The main objective of our study was to report the difficulties of obtaining positive and etiological diagnoses, and their impacts on renal function.

Patients and methods: This retrospective single-center study was conducted between January 1999 and August 2012. We compiled data from all cases of renal-artery thrombosis. Renal infarction was defined as poor vascularization leading to ischemic necrosis of a circumscribed territory of the renal parenchyma, as assessed by an angio-CT scan, an angio-MRI, or a renal arteriograph.

Results: During the study period, 49 cases of renal infarction were compiled. The patient’s median age was 50 years. More male patients were affected (67% of cases). The major risk factors were smoking (35%), high blood pressure (28.5%), peripheral vascular disease (26.5%), and emboligenic heart disorders (24%). At clinical presentation, renal-colic pain was the most frequent symptom (71.4% of cases). Complications, such as high blood pressure (40%) or acute renal failure (48.8%), helped formulate a diagnosis. Other factors that aided a diagnosis were C-reactive protein and lactate dehydrogenase levels, which were increased in >70% of patients. Etiologically, local renal and cardiac causes were the most frequent: respectively, 32.5% and 24%. However, no etiology was found in 37% of cases. Overall, 68% of patients received anti-clotting therapies and 27% received platelet anti aggregation therapy. Eighteen patients had a follow-up greater than 6 months: 5 had chronic renal insufficiency, of which two required supportive renal therapy.

Conclusion: Renal infarction remains a rare pathology. Its diagnosis is difficult because its clinical signs are non-specific. The major risk is progression towards renal failure; thus, it is important to initiate early and adaptive treatment.

INTRODUCTION

Renal infarction is defined as poor vascularization that leads to ischemic necrosis of a circumscribed territory of the renal parenchyma. This interrupted vascularization is most often caused by a thrombus localized in the renal artery or one of its ramifications [1]. This pathology affects 0.007% of the general population [1], with a prevalence of 1.4% according to a series of autopsies carried out by Foxie and Coggin [2]. This low incidence is because renal-artery thrombosis is an unrecognized pathology with a late diagnosis, despite that the first case was described in 1835. Thus, early diagnosis plus appropriate therapeutic treatment are necessary to preserve renal function.

Our study aimed to identify the different manifestations of renal infarction and to determine its impact on renal function. Herein, we report on one of the largest series published so far on renal infarction.

PATIENTS AND METHODS

This retrospective study was conducted at the Department of Nephrology and Organ Transplantation at CHU Toulouse (France) between January 1999 and August 2012. We compiled all cases of renal infarction using the patient’s computerized records.

Renal infarction was defined as poor vascularization that led to ischemic necrosis of a circumscribed territory of the renal parenchyma, as assessed by color Doppler sonography, renal angiography coupled to CT scanning, an angio-MRI, or renal arteriography.
We excluded cases of renal infarction that occurred after aortic/renal vascular surgery or after interventional radiology, as well as renal infarction of the transplanted kidney.

Our variables were cardiovascular-risk factors and those that favored thromboembolic diseases, such as high blood pressure, varuous artery, hypercholesterolemia, smoking, obesity, diabetes. The existence of embolicogenic heart disease and coagulation disorders were systematically sought. The clinical data chosen were those obtained between the initial symptoms and diagnosis, and included renal colic pain, fever, macroscopic hematuria, and high blood pressure. At presentation, a color Doppler sonograph of the renal arteries was performed when possible to search for any abnormalities in the renal vasculature. An angiography was performed to determine the uni/bilateral characteristics of the thrombosis and the types of lesions. Echocardiography was carried out to search for a thrombus or embolicigenic cardiopathy.

After renal infarction was Diagnosed, renal function was assessed at between 6 months and 1 year later.

RESULTS

There were 49 cases of renal infarction over the study period: 31 males and 18 females (gender ratio: male/female of 1.72). The median age of our population was 50 years, with extremes between 25 and 80 years.

In our series, the four major cardiovascular risk factors were smoking (35%), high blood pressure (28.5%), peripheral vascular disease (26.5%), and embolicigenic heart disorders (24%). The time between the first symptoms and a diagnosis was >7 days in 49% of cases.

Renal colic pain was the most frequent symptom in our series: it was reported in 35 cases (71.4%), followed by hypertension (40.8%) and fever (22.4%) (Table 1). With regards to biological parameters, serum creatinine was 107 (55–940) µmol/L, C-reactive protein was increased at 43.2 (2–335) mg/L, and serum lactate dehydrogenase was 1134 (274–9060) µmol/L, C-reactive protein was increased at 43.2 (2–335) mg/L, and serum lactate dehydrogenase was 1134 (274–9060) µmol/L.

We performed an angio-CT scan on 44 patients (89%), and it was always positive. For the other cases, a diagnosis was ascertained using either renal angio-MRI or renal arteriography.

A unilateral renal infarction was the most frequent event (88%). Most infarctions were commonly observed in the region of a main renal artery (53%). With regards to the cause of the infarction, a thrombus was found in 39% of cases, an artery dissection in 16.5% an artery dysplasia in 10%, and an artery stenosis in 6%. However, no lesion was found in 28.5% of cases.

Seven patients had extra-renal infarctions (spleen: 2, liver: 2; lungs: 2; brain: 2). Twelve patients (24.5%) had embolicigenic heart disorders, of which atrial fibrillation occurred in 50%.

Three cases of renal infarction were ascribed to either an antiphospholipid syndrome (n=2) or nephrotic syndrome (n=1) (Table 3).

Therapeutically, 68% of patients benefited from heparin treatment followed by anti-vitamin K; 27% of patients received a platelet anti aggregant at some time. Seven patients (14%) received no treatment. One patient underwent an endovascular procedure that involved inserting a stent.

Median follow-up was 6 months. For the 18 patients where a longer follow-up was possible, five had chronic renal insufficiency, of which two required supportive renal therapy.

DISCUSSION

Renal infarction is a rare pathology: 49 cases have been recorded in our department over the last 14 years. This pathology affects all ages (median age in our series was 50 years).

Our findings with regards to age are similar to those of Vaucher et al. [3], and Antopolsky et al. [4]. However, Korzets et al. [5], reported a median age of 63.4 years.

In our series, renal infarction affected more males (63%; gender ratio of 1.72). This predominance was also observed in the study by Vaucher et al. [3]. In contrast, Antopolsky et al. [4], reported a female predominance of 52% and Korzets et al. [5], reported equal numbers of males and females.

Among the cardiovascular-risk factors in our series, high blood pressure was found in 29%, an impaired vascular terrain

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>Number of cases</th>
<th>%</th>
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<tbody>
<tr>
<td>Renal colic</td>
<td>35</td>
<td>71.4</td>
</tr>
<tr>
<td>Macroscopic haematuria</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>Fever</td>
<td>11</td>
<td>22.4</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>20</td>
<td>40.8</td>
</tr>
<tr>
<td>Coincidental finding</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>Vomiting, nausea</td>
<td>9</td>
<td>18.3</td>
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<tr>
<th>Parameter</th>
<th>Median</th>
<th>Range</th>
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<tbody>
<tr>
<td>Serum creatinine (µmol/L)</td>
<td>107</td>
<td>55 – 940</td>
</tr>
<tr>
<td>Estimated glomerular filtration rate (mL/min; MDRD)</td>
<td>60</td>
<td>4 – 109</td>
</tr>
<tr>
<td>C-reactive protein (mg/L; N&lt;5)</td>
<td>43.2</td>
<td>2.1 – 335</td>
</tr>
<tr>
<td>Lactic dehydrogenase (IU/L; N&lt;333)</td>
<td>1134</td>
<td>274 – 9060</td>
</tr>
<tr>
<td>Renin (mIU/L; N&lt;17.5)</td>
<td>127</td>
<td>27.3 – 155.3</td>
</tr>
<tr>
<td>Serum aldosterone (µg/mL; N&lt;202)</td>
<td>729</td>
<td>19 – 1926</td>
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<tr>
<th>Etiologies of renal infarction.</th>
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<tr>
<td>Renal cause (n=16)</td>
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<tr>
<td>Cardiac cause (n=12)</td>
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<tr>
<td>Anti phospholipid syndrome (n=2)</td>
</tr>
<tr>
<td>Nephrotic syndrome (n=1)</td>
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<tr>
<td>No obvious aetiology (n=20)</td>
</tr>
</tbody>
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Table 1: Clinical presentation of renal-artery thrombosis.

Table 2: Major biological signs.

Table 3: Etiologies of renal infarction.
in 27%, and 35% of patients were smokers. A high proportion of smokers were also reported in the work by Elalouf et al., indeed, smoking is a risk factor associated with atherosclerosis and fibromuscular dysplasia.

The time between the first symptoms and a diagnosis was >7 days for 49% of our series. This duration is similar to the median time of 15 days reported by Rivalan et al. [6]. In contrast, a diagnosis was reached in 1.3 to 72 hours by Elalouf et al. [7], and within a median of 3 days for Cerba et al. [8]. Our longer duration until a diagnosis was because a renal angio-CT scan was not systematically conducted before any lower back pain, in contrast to Elalouf et al., series.

The clinical presentation of renal infarction was unspecific. Renal colic-type pain was the most frequent symptom (72% of cases). This finding is similar to that reported by Antropolski et al. [4], Korzets et al. [5], and Rivalan et al. [6], (86%, 90%, and 100%, respectively).

Fever constituted the second most common symptom in our study (22.4% of cases). This occurred less frequently compared to that reported by Korzets et al. [5], and Rivalan et al. [6], (45% and 62%, respectively). Macroscopic haematuria was rare: there were just three cases in our study. Rivalan et al. [6], reported two cases.

High blood pressure is found within the clinical presentation of renal infarction; in our series, 38 patients (i.e., 40.8%) had high blood pressure as a result of renal infarction. High blood pressure occurred in 48% of the cases reported by Bourgault et al. [9], and in 30% of the cases reported by Lee et al. [10]. The presence of high blood pressure in a patient that has abdominal pain should lead to a search for renal infarction. High blood pressure is secondary to the secretion of renin by the ischemic artery thrombosis. However, we rarely performed it. Arteriography was performed in only 18% of our series, either to look for small anomalies in intra-renal arteries that had not been visualized by the angi-o-scanner or as a therapeutic procedure.

Renal causes were the most frequent etiology in our series (16 patients, i.e., 33%). Similar results were reported by Bourgault et al. [9], (31%).

The presence of an arterial thrombus was the most frequently found lesion (39% of our cases). Fibro dysplasia or a dissection was found in only 26.5% of cases, and a stenosis (most often atheromatous) was found in only 6% of cases. Cardiac embolic causes occurred in 12 patients (24.5%), where as they occurred in 61% of Lee et al. [10], patients.

Non-determined causes were frequent in our series (36.5%), very close to what was found by Bourgault et al. [9], hence, it is important to carry out thorough and systematic etiological research using trans-thoracic or even trans-esophageal echocardiography. A fine-sectioned angio-scan can obtain a thorough picture of the trunks of the renal arteries, and especially the intra-renal branches, in order to detect a small renal infarction.

The most important factor was early diagnosis to determine possible in-situ thrombolysis if the thrombus was visible. Treatment with intravenous heparin was often given (68%) followed by anti vitamin K therapy. This needs to be implemented early to be effective. Only one patient received endovascular treatment, consisting of recanalization of a thrombosed artery with placement of a self-expanding stent in the trunk of the artery, which resulted in a good morphological result. Bourgault et al., series reported a higher number of endovascular treatments, i.e., 9.5% of patients in their series, including endovascular stenting (4.2%) and in-situ thrombolysis (5.3%) [9].

With regards to preventing relapsing infarction/thrombosis, some teams have administered platelet-anti aggregant agents, but there is no conclusive consensus on this approach in the literature. This therapeutic approach was used for >1 year in the studies by Vaucher et al. [3], and Lee et al. [10], and 27% of our patients benefited from this treatment.

Assessment of renal function in 18 of our patients at 6 months after a renal infarction showed chronic renal insufficiency in five cases, including two patients that required hemodialysis. These latter two patients had bilateral problems with the renal artery and >80% destruction of the renal parenchyma. In addition, rhabdomyolysis was found in one of our patients at presentation. In a recent retrospective single-center study Kagaya et al., assessed in 39 patients the volume of the infarction measured using reconstructed computed tomography data and renal function outcome. The volume of the infarction was significantly associated with the peak lactate dehydrogenase (LDH) level (median, 728 IU/L; interquartile range, 491-1227 IU/L) (r = 0.58, p < 0.01) and the degree of renal function decline in both acute and chronic phases (r = -0.44, -0.38, respectively, p < 0.05) [11]. In another recent study including 23 patients with acute renal infarction mean eGFR was impaired at admission (70.8 ± 23.2 mL/min/1.73 m²) but improved significantly to 82.3 ± 23.4 mL/
min/1.73 m² at 1 year follow up. However, at 1-year follow-up 5 patients (22%) had chronic impairment of renal function as defined by eGFR < 60 mL/min/1.73 m² [12].

Our study has limitations because it was a retrospective case-series observational study.

CONCLUSION
Renal infarction is a rare pathology and there is often a delayed diagnosis. Its clinical presentation is non-specific, but it is normally associated with renal-colic pain. The consequences of renal infarction can be acute renal failure and high blood pressure.

A diagnosis is based on anamnestic data, biological data, and especially the results from an angio-scan. In most cases, the etiology has a renal or a cardiac origin. Management is mostly medical. A rapid diagnosis is justified by the possibility, in rare cases, of endovascular revascularization or thrombolysis.

REFERENCES