Acute Renal Infarction: A Case Series

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Summary

Background and objectives Renal infarction is an arterial vascular event that may cause irreversible damage to kidney tissues. This study describes the clinical characteristics of patients with renal infarction according to underlying mechanism of vascular injury.

Design, setting, participants, & measurements This study retrospectively identified 94 patients with renal infarction diagnosed between 1989 and 2011 with the aim of highlighting potential correlations between demographic, clinical, and biologic characteristics and the etiology of renal infarction. Four groups were identified: renal infarction of cardiac origin (cardiac group, *n*=23), renal infarction associated with renal artery injury (renal injury group, *n*=29), renal infarction associated with hypercoagulability disorders (hypercoagulable group, *n*=15), and apparently idiopathic renal infarction (idiopathic group, *n*=27).

Results Clinical symptoms included abdominal and/or flank pain in 96.8% of cases; 46 patients had uncontrolled hypertension at diagnosis. Laboratory findings included increase of lactate dehydrogenase level (90.5%), increase in C-reactive protein level (77.6%), and renal impairment (40.4%). Compared with renal injury group patients, this study found that cardiac group patients were older (relative risk for 1 year increase=1.21, *P*=0.001) and displayed a lower diastolic BP (relative risk per 1 mmHg=0.94, *P*=0.05). Patients in the hypercoagulable group had a significantly lower diastolic BP (relative risk=0.86, *P*=0.005). Patients in the idiopathic group were older (relative risk=1.13, *P*=0.01) and less frequently men (relative risk=0.11, *P*=0.02). Seven patients required hemodialysis at the first evaluation, and zero patients died during the first 30 days.

Conclusions This study suggests that the clinical and biologic characteristics of patients can provide valuable information about the causal mechanism involved in renal infarction occurrence.

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Introduction

Renal infarction (RI) is an uncommon condition resulting from a sudden disruption of blood flow in the renal artery. RI is frequently misdiagnosed or diagnosed late because of its rarity and frequently nonspecific clinical presentation, which may result in irreversible damage to the renal parenchyma or an increase in the risk of other embolic events affecting other organs. The prevalence of RI has been estimated from autopsy studies at 14 per 1000 (1). Recent studies based on admissions to the emergency department have reported estimated incidences of 0.004%–0.007% (2,3). Many etiologic factors are known to favor the occurrence of RI, but atrial fibrillation (AF) is currently considered the most important risk factor for this condition. AF was recently identified as the main causal factor in 64% of published cases of RI (4), and an incidence of renal thromboembolism of 2% was reported in a series of almost 30,000 patients with AF followed for up to 13 years (5). Other risk factors for RI include valvular or ischemic heart disease, endocarditis, hypercoagulation disorders, hematologic disease, and spontaneous renal artery dissection, reflecting the multiplicity of underlying causal mechanisms (4,6). However, despite extensive investigations, the cause of RI remains undetermined in some cases. The work by Bolderman *et al.* (7) studied 27 patients with RI documented by computed tomography (CT) and found that 16 of these patients (59%) were classified as idiopathic. Several retrospective studies, including a limited number of cases (7–44), have reported demographic data, clinical and biologic features, and radiologic findings for patients with RI (2–4,6,8,9). Only one of these studies, based on the analysis of 27 cases, focused on whether medical history and clinical presentation of the patients at the time of RI diagnosis could differentiate between patients with cardiogenic RI and patients with idiopathic RI (7). We report here the largest series of patients with acute RI ever studied, including 94 patients over a 22-year period. Four groups of patients were identified on the basis of the cause of RI, and we carried out statistical analysis of the demographic data, clinical characteristics, and laboratory findings for these

groups of patients, with the aim of highlighting potential clinical and/or biologic correlations with the cause of RI.

Materials and Methods

Patient Population

This multicenter retrospective cohort survey was conducted in 14 French nephrology and internal medicine departments. We sent a questionnaire to French internal medicine and nephrology centers involved in the management of acute RI. At each hospital, patients were identified from clinical diagnosis databases. Ninety-four adult patients (>18 years) with radiologic evidence of acute RI, diagnosed between 1989 and 2011, were included in this study. RI diagnosis was confirmed by CT scan in 83 patients. Typical CT scan features of RI included single or multiple triangular defects in the renal parenchyma. Seven patients underwent magnetic resonance imaging to confirm RI diagnosis. In the remaining four patients, RI diagnosis was based exclusively on renal angiography, which showed an occlusion or a filling defect in the renal artery. We did not include patients presenting RI after recent vascular surgery or percutaneous transluminal angioplasty. We also excluded cases of RI in renal transplant recipients. Demographic data were collected for each patient. Clinical and laboratory data and therapeutic management were analyzed for each patient at the time of initial clinical presentation of RI. Hypertension was defined as an arterial systolic pressure greater than 140 mmHg, a diastolic pressure greater than 90 mmHg, or both (10). Renal impairment was defined as a decrease in estimated GFR of less than 60 ml/min per 1.73 m^2 according to the Modification of Diet in Renal Disease formula. Proteinuria was defined as an albumin excretion rate of more than 0.3 g/d. Increase in C-reactive protein (CRP) level was defined as CRP level more than 5 mg/L. We evaluated changes for laboratory tests (including CRP and lactate dehydrogenase [LDH] concentrations and white blood cell [WBC] counts) for patients with one or more test results since the onset of clinical symptoms. Patients were assigned to four groups on the basis of the underlying disorder responsible for RI. The cardiac group contained patients with RI of cardiac origin, including AF, valvular heart disease, cardiomyopathy with cardiac thrombus, and endocarditis. This group also included cases of mural thrombi of the suprarenal aorta. The renal injury group consisted of patients with RI directly related to renal artery injury (primary or secondary renal artery dissection). The hypercoagulable group included patients with hereditary thrombophilia or hypercoagulability disorders. The idiopathic group contained all the patients for whom extensive cardiac investigations and/or wide thrombophilia panel screening failed to identify the primary cause of RI.

Statistical Analyses

Data were analyzed with Stata V.9 software. Quantitative variables are expressed as means with 95% confidence intervals, and categorical variables are expressed as proportions. Quantitative variables were compared between groups or by ANOVA or the Kruskal–Wallis test if the conditions for ANOVA were not satisfied. Chi-squared tests were used for the comparison of categorical variables to assess their independence. We performed a multinomial logistic regression model (11) using renal injury as the reference group to assess factors associated with each RI cause in an overall model. Variables associated with at least one RI cause with $P<0.20$ were included in the multivariate model using descending stepwise procedure. A P value<0.05 was considered significant in these analyses. However, because samples sizes were small in some groups, we also retained P values from 0.05 to 0.10 in some cases, with these values being considered borderline significance.

Results

Demographic Data and Subgroups of Patients

Patients (94 total; 61 men and 33 women) who met the inclusion criteria were included in this study; 23 patients were assigned to the cardiac group (24.5% of the total population; atrial fibrillation in 17 cases, thrombi from atheroma of the suprarenal aorta in 4 patients, and endocarditis in 2 patients). Among patients with AF, RI occurred in one patient 3 days after electric cardioversion despite prophylactic anticoagulation. The renal injury group included 29 patients (30.8%). The main cause of RI in this group was spontaneous renal artery dissection in 17 cases, type B renal artery dissection in 2 cases, fibromuscular dysplasia in 8 cases, and Ehlers–Danlos syndrome with thrombotic aneurisms of the renal artery in 2 cases. The hypercoagulable group contained 15 patients (16%) with hereditary thrombophilia (n=6), hyperhomocysteinemia (n=4), antiphospholipid syndrome (n=4), and nephrotic syndrome secondary to AL amyloidosis (n=1). The idiopathic group contained 27 patients (28.7%) for whom the cause of RI remained unidentified. The demographic data for these patients are summarized in Table 1. Mean age differed significantly between groups, the oldest being cardiac group patients (65.1±15 years). Proportion of men was also different between groups, with the highest proportion of men in the renal injury group (86.2%). Previous history of arterial hypertension was different between groups and most frequently reported (in 69.6% of cases) in the cardiac group. Unsurprisingly, past medical history of cardiac disorders, including ischemic and arrhythmic heart disease, was most frequently found in the patients of the cardiac group; 9 patients (9.6%) had a history of embolic events, and 11 patients were already on anticoagulant treatment (11.7%) before RI occurred.

Clinical Presentation and Laboratory Findings

The mean time between the onset of clinical symptoms and radiologic findings confirming RI diagnosis was 5.4±6.5 days. The mean delay for definitive RI diagnosis was not significantly influenced by the period of RI occurrence. Clinical characteristics and laboratory parameters at the time of RI diagnosis are summarized in Table 2. RI involved the right kidney in 41 patients (43%) and the left kidney in 38 patients (41%). Bilateral renal involvement was more frequent in patients with coagulation dysfunction (P=0.04). Among patients with unilateral renal involvement (n=79), 65 patients (82.3%) exhibited one single defect into renal parenchyma, whereas 14 patients presented two or more lesions. All patients with bilateral involvement (n=15) had multiple (two or more) defects

Table 1. Demographic data of patients at the time of renal infarction diagnosis

	Overall (*n*=94)	Cardiac Group (*n*=23)	Renal Injury Group (*n*=29)	Hypercoagulable Group (*n*=15)	Idiopathic Group (*n*=27)	*P* Value
Age (yr; mean ± SD)	52.9±16.6	65.1±15	43.7±8,5	46.1±17.5	56.1±16.7	<0.001
Men (%)	61 (64.9)	12 (52.2)	25 (86.2)	10 (66.7)	14 (51.8)	0.02
Previous arterial hypertension (%)	37 (39.4)	16 (69.6)	7 (24.1)	3 (20)	11 (40.7)	0.003
Previous diabetes mellitus (%)	9 (7.4)	4 (17.4)	1 (3.4)	1 (6.6)	1 (3.7)	0.27
Smoking exposure (%)	31 (32.9)	4 (17.4)	14 (48.3)	5 (33.3)	8 (29.6)	0.13
Hyperlipidemia (%)	20 (21.3)	9 (39.1)	3 (10.3)	2 (13.3)	6 (22.2)	0.09
Previous ischemic heart disease (%)	9 (9.4)	7 (30.4)	0 (0)	1 (6.7)	1 (3.7)	0.001
Previous arrhythmic heart diseases (%)	20 (21.3)	16 (69.6)	0 (0)	1 (6.7)	3 (11.1)	0.001
Previous history of embolic event (%)	9 (9.6)	3 (13)	2 (6.9)	2 (13.3)	2 (7.4)	0.78
Time to diagnosis (d)	5.4±6.5	6.2±6.3	5.7±6.6	4.2±4.2	4.6±8.2	0.35
Antiplatelet therapy (%)[a]	12 (12.7)	7 (30.4)	2 (6.9)	0 (0)	3 (11.1)	0.04
Anticoagulant therapy (%)[a]	11 (11.7)	2 (8.7)	2 (6.9)	4 (26.6)	3 (11.1)	0.31

[a]Before renal infarction diagnosis.

in each kidney. Clinical symptoms at the first visit included persistent abdominal and/or flank pain in 91 patients (96.8%), nausea in 26 patients (27.6%), vomiting in 19 patients (20.2%), and fever in 19 patients (20.2%). Macroscopic hematuria was observed in only six patients, but all these patients belonged to either the cardiac (*n*=3) or hypercoagulable (*n*=3; *P*=0.005) group. In total, 46 patients (48%) presented marked hypertension (inaugural hypertension or uncontrolled hypertension in a patient on long-term antihypertensive treatment) at initial presentation of RI. Median diastolic BP (DBP) differed between groups; the highest was observed, at 90 mmHg (interquartile range [IQR]=80–100 mmHg) in the patients of the renal injury group. High leukocyte counts were recorded in 68 patients (72.3% of cases). Increase in CRP levels was found in 73 patients (77.6%). Median CRP concentration (60 mg/L; IQR=6–119 mg/L) was the highest in the cardiac and hypercoagulable groups. High LDH concentration was frequently associated with RI (85 patients; 90.5%). Increases in LDH concentration were greatest in the hypercoagulable group, but we did not consider this marker to be useful for differentiating between different causes of RI (*P*=0.08). An impairment of kidney function at admission was recorded for 38 patients (40.4% of the total population). Renal function at the time of RI management did not differ significantly between the groups, although GFR tended to be slightly, but not significantly, lower in cardiac and hypercoagulable groups than other groups (*P*=0.09). We next investigated changes in WBC count and LDH and CRP concentrations since the onset of clinical symptoms. Nine patients for whom a definitive diagnosis of RI was made 30 days or more after the first clinical presentation were excluded from this analysis. WBC counts peaked soon after first clinical symptoms (Figure 1), and leukocyte counts gradually normalized after day 15. LDH concentrations remained above the upper limit of the normal range on the 15th day of first clinical presentation (Figure 2). By contrast, the increase in CRP concentration was maximal between the fourth and fifth days after the occurrence of the first clinical symptoms (Figure 3).

Using a multivariate analysis, we found that three factors (age, sex, and DBP) remained independently associated with at least one RI cause. Compared with renal injury group patients, taken as reference group, we found that the cardiac group patients were older (adjusted relative risk [RR] for a 1-year increase=1.21, 95% confidence interval [CI]=1.08–1.34, *P*=0.001) and displayed a lower DBP (adjusted RR per 1 mmHg=0.94, 0.88–1.00, *P*=0.05). Patients of the hypercoagulable group had a significantly lower DBP compared with renal injury group (adjusted RR per 1 mmHg=0.86, 0.78–0.96, *P*=0.005). Patients of the idiopathic group were older (adjusted RR for a 1-year increase=1.13, 95% CI=1.03–1.25, *P*=0.01) and less frequently men (adjusted RR=0.11, 95% CI=0.02–0.68, *P*=0.02) than the renal injury group.

Treatment and Outcome

The therapeutic management of RI involved intravenous heparin administration in 42 patients. Oral anticoagulant maintenance therapy was initiated in 36 patients (14 patients in the cardiac group, 6 patients in the renal injury group, 8 patients in the hypercoagulable group, and 8 patients in the idiopathic group), and exclusive antiplatelet treatment was administered in 33 patients; 33 patients underwent supplementary endovascular radiologic procedures after diagnosis by CT scan, but curative treatment (intra-arterial urokinase infusion, thromboaspiration, and renal artery stenting) during angiography was performed in only 5 patients (the cardiac group had 2 patients, the renal injury group had 2 patients, and the idiopathic group had 1 patient). In one patient of the renal injury group with

Table 2. Clinical characteristics and laboratory parameters at the time of renal infarction diagnosis

	Total Population (*n*=94)	Cardiac Group (*n*=23)	Renal Injury Group (*n*=29)	Hypercoagulable Group (*n*=15)	Idiopathic Group (*n*=27)	*P* Value
Abdominal pain (%)	52 (50.9)	15 (65)	17 (58.6)	7 (46.6)	11 (40.7)	0.31
Flank pain (%)	46 (48.9)	11 (47.8)	14 (48.3)	6 (40)	15 (55.6)	0.07
Nausea (%)	26 (27.6)	8 (34.8)	8 (27.6)	2 (13.3)	8 (29.6)	0.55
Vomiting (%)	19 (20.2)	7 (30.4)	5 (17.2)	2 (13.3)	5 (18.5)	0.61
Fever (%)	19 (20.2)	6 (26.1)	6 (20.7)	0 (0)	7 (25.9)	0.14
SBP (mmHg)	140	140	149	130	142.5	0.12
IQR=25%–75%	127–155	113–148	132–160	118–140	130–150	
DBP (mmHg)	80	80	90	71	80	0.001
IQR=25%–75%	73–90	70–88	80–100	68–80	79.5–90	
Bilateral renal involvement (%)	15 (16)	1 (4)	6 (20)	5 (33)	3 (11)	0.04
Extra renal infarction (%)	9 (10)	3 (13)	0 (0)	2 (13)	4 (15)	0.09
Hemoglobin levels (g/dl; *n*=56)	14	14	14.3	13.6	13.6	0.46
IQR=25%–75%	12.8–15	11.6–15.2	13.6–15	11.4–14.3	12.5–14.9	
LDH concentration (UI/L; *n*=76)	660	836	537	935	458	0.08
IQR=25%–75%	380–1417	580–2017	363–1137	479–4131	260–1209	
CRP levels (mg/L; *n*=81)	60	96	33	95	16.5	0.005
IQR=25%–75%	6–119	59–226	4–76	89–119	5–104	
WBC count (/mm^3; *n*=81)	11,000	12,140	10,450	13,265	9227	0.12
IQR=25%–75%	8100–13,480	10,500–14,100	7540–13,050	10,550–15,000	6920–12,690	
Creatinine concentration (μmol/L; *n*=85)	112	126	103	197	94	0.36
IQR=25%–75%	87–148	103–148	87–124	80–332	80–131	
Estimated GFR (ml/min per 1.73 m^2)	64.1	46.2	73.7	35	66.1	0.09
IQR=25%–75%	38.1–86	36.9–68.5	55.5–88.9	18.6–95.0	49.5–78.2	
Proteinuria levels (g/d; *n*=36)	0.13	0.3	0.15	0.5	0.05	0.39
IQR=25%–75%	0–0.07	0.03–0.8	0–0.55	0–1.2	0–0.17	
Microscopic hematuria (%; *n*=33)	14 (42.4)	2 (29.2)	7 (43.7)	2 (50)	2 (50)	0.85
Macroscopic hematuria (%)	6 (5.9)	3 (13)	0 (0)	3 (20)	0 (0)	0.005

SBP, systolic BP; IQR, interquartile range; DBP, diastolic BP; LDH, lactate dehydrogenase; CRP, C-reactive protein; WBC, white blood cell count.

bilateral renal involvement, this therapeutic management failed to improve renal function, leading to ESRD. In four cases, endovascular curative procedures were associated with significant improvement in renal function (decrease in mean creatinine levels from 185 to 123.5 μmol/L before and 1 month after radiologic procedure). Surgical interventions were performed in four patients. Severe renal impairment necessitating hemodialysis occurred in seven patients at initial presentation, and five of these patients definitively required chronic intermittent hemodialysis. ESRD occurred in three patients with bilateral involvement (two patients in the renal injury group and one patient in the hypercoagulable group) and two patients of the cardiac group with one affected kidney but a pre-existing known chronic renal failure. None of the patients died in the first 30 days after the onset of RI, and only one patient died during the follow-up period (median follow-up=9.5 months, IQR=2.2–36.1).

Discussion

We report here the largest study investigating clinical characteristics and initial outcome as a function of the underlying mechanism in patients with RI. We identified local renal artery involvement (30.8%) as the most frequent cause of RI, with only 24.5% of patients experiencing RI of cardiac origin. In the recent review of 165 cases by Antopolsky *et al.* (4), 37% of RI cases were found to be

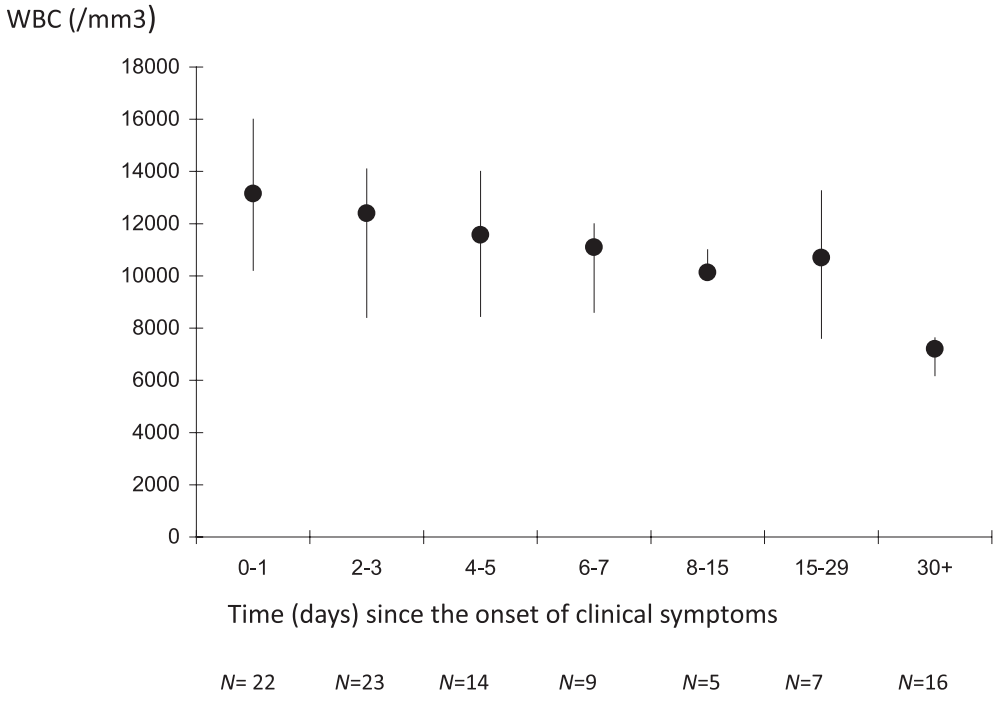

Figure 1. | **White blood cell count (96 values collected in 81 patients) according to the delay between the onset of clinical symptoms and blood samples collection.** The error line represents values between the 25th and 75th percentiles.

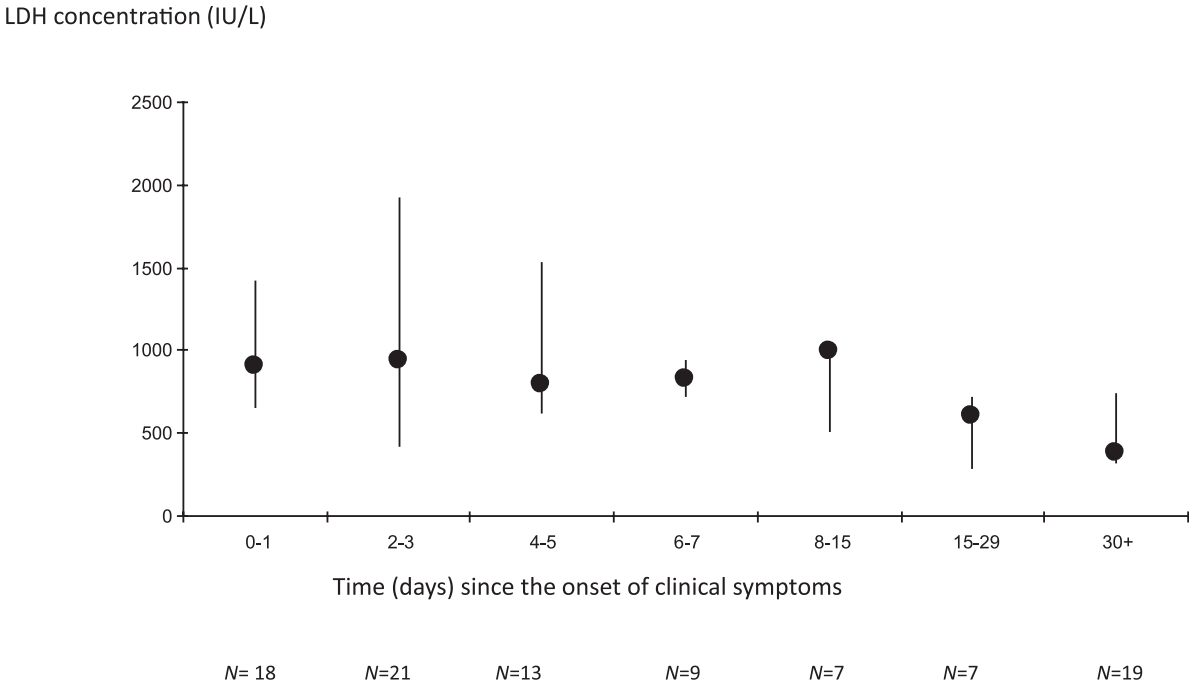

Figure 2. | **Lactate dehydrogenase levels (94 values collected in 76 patients) according to the delay between the onset of clinical symptoms and blood samples collection.** The error line represents values between the 25th and 75th percentiles.

related to AF (4). However, 44 of 165 patients originated from the study by Hazanov *et al.* (8), which included only cases of RI occurring in a context of AF. These results emphasize the need to perform large investigations in the context of RI to identify the etiologic factor for directing therapeutic management. Despite exhaustive investigations, the etiology of RI remained unknown in 28.7% of our cases, highlighting the lack of knowledge about the precise nature of the causal factor in a substantial number of cases.

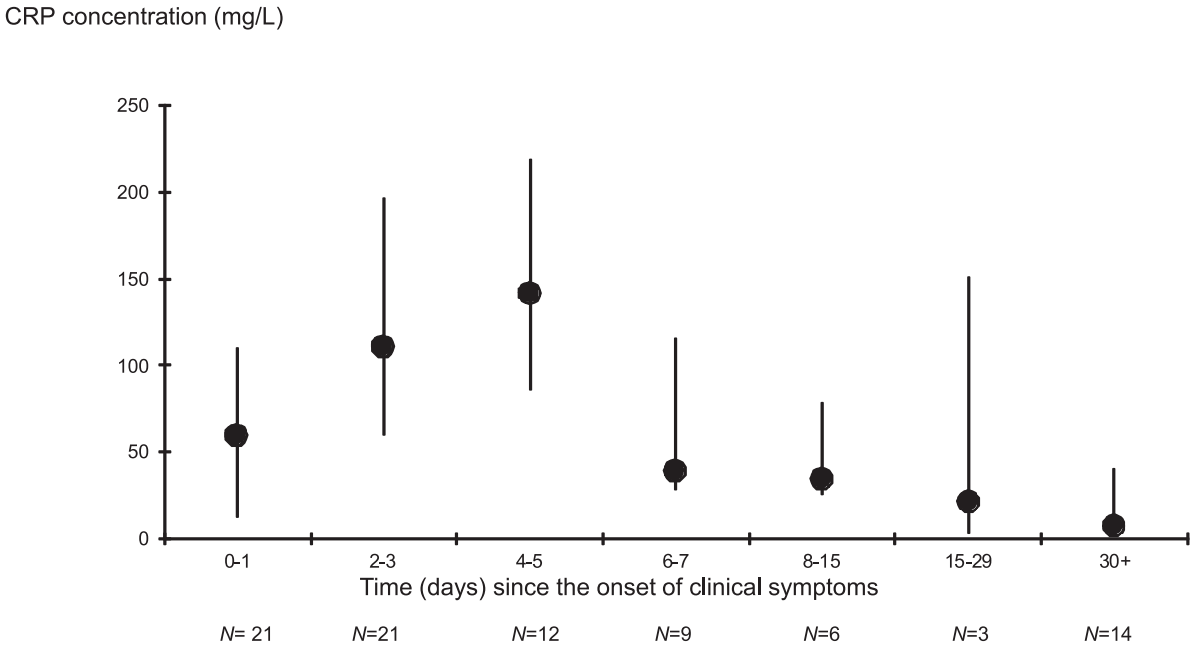

Figure 3. | C-reactive protein levels (86 values collected in 81 patients) according to the delay between the onset of clinical symptoms and blood samples collection. The error line represents values between the 25th and 75th percentiles.

The basal demographic characteristics of patients with RI have been little studied. We found that a medical history of hypertension was significantly more frequent in patients of the cardiac group, who were also significantly older than the patients of the other groups. This result may account for the higher frequency of chronic heart disease (including ischemic or arrhythmic heart diseases) in patients with RI of cardiac origin, but the incidence of other classic cardiovascular risk factors, such as diabetes mellitus or exposure to smoking, was not significantly more frequent in the cardiac group than other groups. These data contrast with the data reported in the work by Bolderman *et al.* (7), which found that patients with RI classified as idiopathic were younger, were more likely to smoke, and had a lower incidence of diabetes mellitus and arterial hypertension than other patients. The work by Domanovits *et al.* (3) suggested that a CT scan should be performed early in all patients displaying the triad of a high risk of thromboembolic events, persistent flank/abdominal/lower back pain, and high LDH levels and/or hematuria within 24 hours of the onset of pain for the detection of possible RI (3). As previously reported in an analysis of pooled data (4), we found that the spectrum of clinical signs of RI included abdominal and/or flank pain (96.8% of cases), nausea and vomiting (27.6% and 20.2% of cases, respectively), and fever (20.2% of cases). However, we also provide the first demonstration that clinical symptoms do not differ significantly between patients with different causes of RI. Mean BP at RI presentation has rarely been reported, but several case reports have suggested that some patients remain normotensive after kidney infarction (12,13). In our study, an increase in BP was frequently observed at the time of RI diagnosis (48% of cases). Median DBP was significantly higher in patients with RI caused by local renal artery injury. The work by Paris *et al.* (14) identified 55 patients with spontaneous RI among 18,287 patients referred to a hypertension unit, and 22 of these patients had malignant hypertension at initial presentation. In this previous study based exclusively on the analysis of hypertensive patients, mean DBP was higher than the mean DBP in our population (107 ± 18 versus 83 ± 15 mmHg). Underlying renal artery disease was found in 75% of the total population studied in the work by Paris *et al.* (8). These data suggest that severe hypertension occurring in a patient with long-term treated hypertension or inaugural hypertension in a normotensive patient also reporting abdominal and/or flank pain should alert the clinician to the risk of RI. Moreover, a large increase in DBP strongly suggests that the RI is directly related to primary or secondary renal artery injury. These findings are not surprising, because experimental kidney infarction is known to induce renin-dependent hypertension associated with intrarenal and/or prerenal ischemia (15).

We then investigated the possible correlation between the main cause of RI and certain laboratory findings. As previously reported, LDH concentration, a common marker of cell necrosis, was frequently high (90.5% of the total population) in patients with RI and remained above the upper limit of the normal range over a long period (15 days) after the first clinical presentation (3,4). Increase in CRP level was also frequently (77.6% of cases) observed in patients with RI. Patients with RI of cardiac origin displayed larger increases in CRP concentration than the patients of the other groups. It has been suggested that CRP, a marker of systemic inflammation, may be involved in the pathogenesis of cardiovascular diseases and could be used as a prognostic marker in patients with acute coronary syndrome (16). By contrast, the work by Bolderman *et al.* (7) reported only a moderate increase in CRP concentration that was not significantly associated with RI of

cardiac rather than idiopathic origin. These discrepancies may, in part, be accounted for by the differences in the interval between symptom onset and definitive RI diagnosis between the two studies.

Consistent with the findings of the meta-analysis carried out by Antopolsky *et al.* (4), we found that impaired renal function was frequently associated with RI (40.4% of our cases), although we cannot exclude the possibility that some patients already had renal failure before the occurrence of RI. Patients from the hypercoagulable group tended to have higher serum creatinine concentrations than patients of the other groups, although this difference was not statistically significant. This probably reflects the severity of RI in patients with hypercoagulability disorders, in whom the frequency of bilateral renal involvement was significantly higher than in other patients.

No clear treatment strategy for RI has yet been established, and the approach used depends on several factors, including the underlying cause and the time between the onset of clinical symptoms and definitive radiologic diagnosis. Thirty-three of our patients underwent renal angiography after RI diagnosis by CT scan, but curative treatment was performed in only five of these patients, highlighting the importance of rapid diagnosis. Given the rarity of this disease, it is unlikely to be possible to test the superiority of a particular treatment in prospective randomized clinical trials.

Our study has some limitations. First, our study is retrospective, and some laboratory data are lacking. Second, we cannot definitely exclude that patients from the idiopathic group exhibited a specific (but not determined) causal mechanism involved in RI occurrence. Third, the limited sample size of each subgroup did not allow us to definitively distinguish specific clinical and biologic features according to the main underlying mechanisms. The aim of this retrospective study was not to establish a diagnosis score to discriminate the cause of RI according to clinical and laboratory parameters. Prospective studies are required to determinate with accuracy whether demographic and biologic data may differentiate the underlying mechanisms involved in RI occurrence.

Disclosures

None.

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