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Cause of renal infarction: a retrospective analysis of 186 consecutive cases

Anne-Laure Faucon^{a,d}, Guillaume Bobrie^a, Anne-Sophie Jannot^{b,d,f}, Arshid Azarine^c, Pierre-François Plouin^a, Michel Azizi^{a,d,e}, and Laurence Amar^{a,d}

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Background: Renal infarction can cause abrupt and severe hypertension and less frequently renal failure. Renal infarction results from disruption of renal blood flow in the main ipsilateral renal artery or in a segmental branch. Underlying mechanism is either general, 'embolic' or 'thrombophilic', or local related to primary 'renal artery lesion'. It depends on various causes. In absence of an identified cause, renal infarction is classified as 'idiopathic'. Previous studies report a significant number of 'idiopathic' renal infarction.

Objective: The aim of this study was to analyze various renal infarction causes.

Methods: Between July 2000 and June 2015, 259 consecutive patients with renal infarction were admitted to our hospital center and retrospectively identified from weekly multidisciplinary round. Main clinical and biological characteristics were extracted from clinical data warehouse. Renal imaging was reviewed by two readers unaware of the diagnosis.

Results: Of 259 initially identified patients, 30 were excluded owing to a lack of imaging or clinical data and 43 because iatrogenic renal infarction. In the 186 studied patients, dissection was observed in 76 patients (40.8%) and occlusion in 75 (40.3%). Renal infarction mechanisms were 'renal artery lesion' ($n = 151$; 81.2%), 'embolic' ($n = 17$; 9.1%), 'thrombophilic' ($n = 11$; 5.9%) and 'idiopathic' ($n = 7$; 3.8%). Predominant renal artery lesions were atherosclerosis disease ($n = 52$; 34.4%) followed by dissecting hematoma ($n = 35$; 23.2%) and fibromuscular dysplasia ($n = 29$; 19.2%). Right and left kidneys were equally involved.

Conclusion: Renal artery lesion is the most frequent cause of renal infarction. This result underlines the need for extensive arterial exploration to identify the renal infarction mechanism and, in case of renal artery lesion, the underlying vascular disease.

Keywords: atherosclerosis disease, dissecting hematoma, fibromuscular dysplasia, renal artery dissection, renal infarction

Abbreviations: AoD, extension of an aortic dissection to the RA; ASD, atherosclerosis disease; CDW, clinical data warehouse; DAAD, dissecting or aneurysmal multifocal arterial disease; DH, dissecting hematoma; FMD, fibromuscular dysplasia

INTRODUCTION

Renal infarction results from acute disruption of the renal blood flow in the main ipsilateral renal artery or in one of its segmental branches. This can cause abrupt hypertension [1], sometimes malignant hypertension [1,2] and less frequently renal failure if the renal infarction is bilateral and extensive [3,4]. Renal infarction is an uncommon condition with an incidence of about 0.004–0.007% in emergency departments [5–7]. However, owing to nonspecific clinical presentation (flank or back pain, hematuria) [1,6–8], its incidence is probably underestimated. This is supported by data from our previous study [1] and from an autopsy series [8]. Both found a prevalence of 0.3 and 0.48%, respectively [1,8].

Renal infarction can be due to either a general or local in-situ mechanism. Classically, the main general renal infarction mechanisms are classified as 'embolic' (atrial fibrillation, valvular or aortic wall disease) or 'thrombophilic' from various hypercoagulable states. Local mechanism consists in parietal 'renal artery lesion'. Each mechanism depends on various causes. Finally, when no cause is found despite extensive workup, renal infarction is classified as 'idiopathic' [4]. In the three retrospective large series [3,7,9], renal infarction mechanism was mainly 'embolic' (25–61%), less frequently related to renal artery lesion (0–31%), and a high percentage of 'idiopathic' renal infarction (14–30%) was recorded. This is in contradistinction with our previous study where renal artery lesions reach 74.5% [1]. Furthermore, arterial occlusion or isolated dissection sometimes make the etiologic diagnosis tricky without systematic extensive explorations of other arterial sites, nor without embolic and thrombophilic investigations.

Therefore, to clarify renal infarction mechanisms and causes, 186 consecutive renal infarction inpatients

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admitted to our specialized Hypertension Unit in a tertiary hospital center, were retrospectively analyzed, over 15 years. This study constitutes an extension of our previous analysis [1]. The patients were divided into four groups according to the main mechanisms of renal infarction [4]. Specific renal artery imaging features (total occlusion, dissection, stenosis or aneurysm) were further analyzed together with different causes and related patients characteristics.

METHODS

Data source

An Electronic Health Record system is utilized in our institution to gather all patient data. It includes treatments, laboratory workup, administrative data (such as billing International Classification of Diseases 10th Revision codes and medical procedures), clinical notes, vital parameters, biological and radiological results and demographic data (birth and death date if relevant). These informations are stored in a clinical data warehouse (CDW) which is run under the Informatics for Integrating Biology and the Bed-side standard [10]. It encompasses data from more than 500 000 patients, including more than 1000 new patients per year consulting for hypertension. For these hypertensive patients, a specific questionnaire of 103 questions has been used since 1975 [11].

Blood pressure measurements

Clinical blood pressure (BP) was measured in the supine position with a validated oscillometric device (OMRON 907; Omron Co., Kyoto, Japan). It is automatized, allowing three successive BP measurements without any observer being present. The result taken into account is the average of these three measurements. Twenty-four-hour ambulatory BP monitoring was performed with the Spacelabs 90207 monitor (Spacelabs Healthcare, Issaquah, Washington, USA). BP was measured every 15 min during the day (from 0700 to 2200 h), and every 30 min overnight (from 2200 to 0700 h).

Arterial imaging

All patients had a computed tomography (CT) scan to confirm renal infarction diagnosis, to assess ipsilateral renal artery radiological features, to evaluate contralateral renal artery and other arterial sites. It intended also to determine the renal infarction mechanism and cause. Whenever necessary, patients had a carotid and Willis polygon CT scan.

Renal artery lesion

Renal artery lesion causes include atherosclerosis disease (ASD), fibromuscular dysplasia (FMD) [12], dissecting hematoma [13], extension of an aortic dissection (AoD) to the renal artery, dissecting or aneurysmal multifocal arterial disease (DAAD), iatrogenic postrenal artery catheterization/surgery complication or posttrauma complication.

As assertion of FMD diagnosis is difficult based on the sole finding of a dissected artery, it also requires the identification of suggestive FMD lesions involving other arterial sites [12]. Conversely, as in acute aortic syndromes

[14], renal artery dissecting hematoma is consecutive to a spontaneous entry tear located in the renal artery trunk or branches, or to an intimal–medial hematoma (by vasa vasorum rupture), without other arterial lesion. Hence, its diagnosis requires the exclusion of other underlying arterial disease. At the opposite, DAAD associates renal artery lesion with dissection or aneurysm on other vascular sites, any known vascular disease being excluded. This is the reason why our procedure stipulates extensive investigations of other arterial sites (contralateral renal artery, aortoiliac and digestive axis, carotid and Willis polygon). In the absence of lesions identifying arterial disease, transesophageal echocardiography, Holter-EKG and thrombophilia investigations are performed. Clinical history and all available imaging are reviewed at a weekly multidisciplinary round with hypertension specialists, nephrologists, cardiologists, vascular radiologists, vascular interventional radiologists and vascular surgeons. The conclusion of this meeting is included in the patient's electronic record.

Study protocol

Patients with renal infarction were retrospectively identified from the multidisciplinary meeting reports, queried by using four keywords: 'infarction', 'dissection', 'thrombosis' and 'nephrectomy' renal imaging was available on the Picture Archiving and Communication System. It was reviewed by two readers (A.-L.F. and G.B.), unaware of the initial radiological and multidisciplinary meeting reports. The aims of this second look were to confirm renal infarction diagnosis (typical CT scan features of renal infarction including single or multiple triangular defects in the renal parenchyma) [15], to assess ipsilateral renal artery radiological features (total occlusion, dissection, stenosis or aneurysm), to evaluate contralateral renal artery and other arterial sites. It intended also to determine the renal infarction mechanism ('embolic', 'thrombophilic', 'renal artery lesion' or 'idiopathic') and cause (ASD, FMD, dissecting hematoma, AoD, DAAD, iatrogenic postrenal artery catheterization/surgery complication or posttrauma complication). If a disagreement emerged between the reviewers and the initial renal imaging or multidisciplinary meeting report, the final diagnosis was made by a vascular radiologist (A.A.) after a third independent reading.

We excluded patients wrongly selected by keywords but without renal infarction, patients with iatrogenic renal infarction (postrenal arterial angioplasty/surgery, postadrenal surgery, postaortic angioplasty/surgery etc.) and patients without available radiology or clinical report.

Statistical analysis

The number of missing answers in the dedicated hypertension questionnaire was first determined. When more than 50% answers were lacking for a special question, this item was discarded as no relevant. Each question was analyzed in the same way: median and interquartile range were estimated for continuous variables. Frequency was determined for qualitative variables. This analysis was performed in the whole population as well as in the specific group defined by the renal infarction cause and mechanism. We

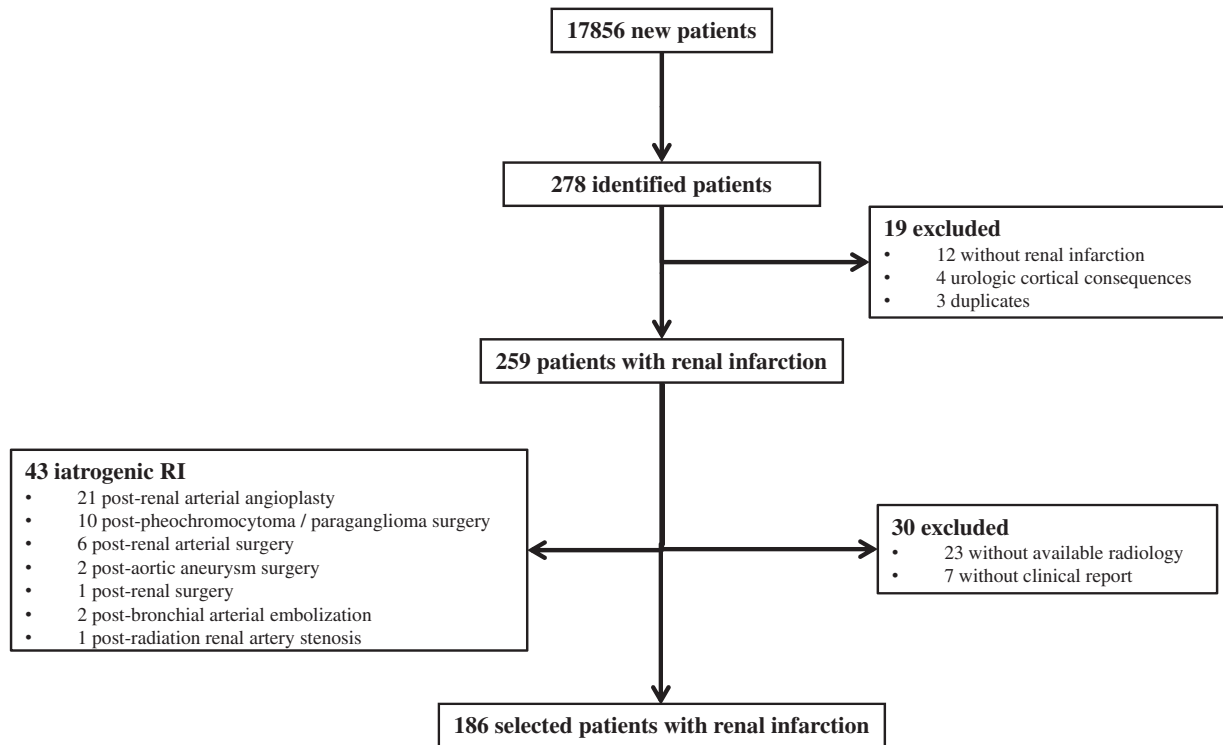


FIGURE 1 Flowchart.

first checked whether the missing data pattern was random with respect to etiology or mechanism by testing the association between missing data and etiology on one side and mechanism on the other using either a chi-square test or a Fisher's exact test when necessary for qualitative item responses. We then tested the association between item responses and mechanism on the one hand and etiology on the other using either a chi-square test or a Fisher's exact test when necessary for qualitative item responses or an analysis of variance for continuous response items. Multiple testing was taken into account using a Bonferroni correction. Overall, 49 clinical and laboratory characteristics from the hypertension questionnaire were considered as relevant for more than half of the 186 patients. Therefore, the level of significance in the subsequent analyses was considered to be 0.001.

Ethic committee

The current study was approved by our institutional review board (registration no. 00001072, 16 November 2015, delivered by the 'Comité de Protection des Personnes Ile de France 2').

RESULTS

Among the 17 856 new patients who consulted in our center between July 2000 and June 2015, 278 patients selected according to specific keywords, were retrospectively identified from the weekly multidisciplinary meeting reports as having renal infarction. According to inclusion criteria, 19 did not fit inclusion keywords and were excluded. Thus, 259 consecutively admitted patients with renal infarction were identified. Thirty of them were excluded owing to

missing imaging or clinical data, as well as a further 43 patients who had iatrogenic renal infarction (i.e. recent vascular, renal or adrenal surgery or percutaneous transluminal angioplasty or other procedures). Finally, 186 patients with renal infarction were studied (Fig. 1). Owing to discrepancies between initial diagnosis and 'naïve' reviewers, 39 patients required a third independent reading by the vascular radiologist. This third review changed the mechanism in only two cases [one thromboembolic mechanism (atrial fibrillation) was reclassified as atherosclerosis dissection and one normal renal artery was reclassified as renal artery lesion by dissecting hematoma] and the cause in only four cases (four 'FMD' were reclassified as 'dissecting hematoma'). The questionnaires of the 186 included patients were extracted from the CDW at the visit date nearest to the diagnosis (the first available imaging data supporting the evidence of renal infarction) [median interval: 3 days (1; 56)]. The renal infarction prevalence was 1.0% and the incidence 0.07%.

Median age at diagnosis was 53.3 (42.5; 64.5) years, 62% were men, 65% had known hypertension for 5 (0; 14.5) years, 43% had known dyslipidemia and 9% diabetes, and 27% were current smokers and 84% had antihypertensive drugs. The clinical BP was 141 (124; 159)/84 (73; 96) mmHg and the diurnal ambulatory BP 138 (127; 156)/85 (78; 94) mmHg with two (1; 3) antihypertensive drugs. Serum creatinine was 94 (77; 124) $\mu\text{mol/l}$ and estimated glomerular filtration rate – eGFR – (modification of diet in renal disease formula) was 70 (50; 90) ml/min per 1.73 m².

Renal infarction involved the right kidney in 60 (32.3%) patients, the left in 78 (41.9%) and both kidneys in 48 (25.8%). The ipsilateral renal artery radiological feature of the renal infarction was dissection in 76 (40.8%) cases,

TABLE 1. Renal infarction mechanisms and causes

	<i>N (%)</i>
Renal artery lesions	151 (81.2)
Atherosclerosis disease	52 (34.4)
Dissecting hematoma	35 (23.2)
Fibromuscular dysplasia	29 (19.2)
Dissecting or aneurysmal arterial disease (Renal artery lesion with dissection or aneurysm on other vascular site)	12 (7.9)
Aneurysm	8 (5.3)
Extension of an aortic dissection	5 (3.3)
Trauma complication	4 (2.6)
Miscellaneous disease	6 (3.9)
Ehlers–Danlos syndrome	1
Alagille syndrome	1
Pseudoxanthoma elasticum	1
Arteritis	2
Anatomic anomaly of contralateral renal artery	1
'Embolitic' mechanisms	17 (9.1)
Atrial fibrillation	8
Aortic atherosclerosis lesions	3
Patent foramen ovale	2
Auricular thrombosis	2
Intraauricular communication	1
Ventricular thrombosis (pheochromocytoma)	1
'Thrombophilic' mechanisms	11 (5.9)
Antiphospholipid syndrome	3
JAK2 mutation	2
Cancer	4
Multiple thrombosis	2
Idiopathic	7 (3.8)

JAK2, Janus kinase 2.

total occlusion in 75 (40.3%), stenosis in 14 (7.5%) and aneurysm in three (1.6%). In 11 (5.9%) cases, we found no ipsilateral renal artery lesion but a lesion of the contralateral renal artery or of other sites leading to identification of the underlying arterial disease.

Renal infarction mechanisms were 'renal artery lesion' in 151 (81.2%) patients, 'embolic' disease in 17 (9.1%) and 'thrombophilic' in 11 (5.9%). None of these were observed in seven (3.8%) patients. However, extreme distal renal artery lesions, or postdissection recanalization or remodeling, could not be ruled out. Clinical and laboratory characteristics were similar in the four groups (data not shown).

The causes for each renal infarction mechanism are listed in Table 1. Renal artery lesion was more frequently due to ASD ($n = 52$, 34.4%) than to dissecting hematoma ($n = 35$, 23.2%) or FMD ($n = 29$, 19.2%). 'Embolitic' renal infarction was mostly due to atrial fibrillation. As shown in Table 2, patients with renal infarction related to ASD were older, more often prone to dyslipidemia and cardiovascular disease. They also had higher SBP, lower eGFR, and they were taking more antihypertensive and lipid-lowering drugs. Whatever the cause, right and left kidneys were equally involved.

The renal infarction cause was suggested by the renal artery radiological feature. The most frequent causes for renal artery dissection ($n = 76$) were dissecting hematoma ($n = 35$, 46%), FMD ($n = 16$, 21%) and DAAD ($n = 10$, 13%). By definition, renal artery dissection is present in 100% of dissecting hematoma, but it was also the most frequent renal artery radiological feature in FMD (55% of cases). ASD was mostly responsible for renal artery occlusion (53.3%) and stenosis (71.4%). Conversely, the most frequent renal artery aspect in ASD was occlusion (76.9%).

TABLE 2. Patients' characteristics according to the three main renal infarction causes

	<i>n</i>	ASD 52	FMD 29	DH 35	<i>P</i>
Age at diagnosis (years)	116	67 (60; 74)	47 (40; 58)	46 (40; 53)	<0.001
Males, <i>n (%)</i>	116	32 (62%)	16 (55%)	25 (71%)	0.39
BMI (kg/m ²)	112	25 (22; 27)	24 (22; 27)	25 (23; 27)	0.611
Preexisting hypertension, <i>n (%)</i>	105	33 (70%)	17 (65%)	19 (59%)	0.61
Hypertension duration (years)	96	9 (2; 23)	5 (0; 13)	3 (0; 10)	0.016
Never-smokers, <i>n (%)</i>	107	20 (41%)	12 (46%)	11 (34%)	0.66
Diabetes, <i>n (%)</i>	105	6 (12%)	1 (4%)	2 (6%)	0.43
Dyslipidemia, <i>n (%)</i>	107	34 (69%)	8 (31%)	10 (31%)	<0.001
Previous cardiovascular event, <i>n (%)</i>	116	12 (23%)	2 (7%)	2 (6%)	0.03
Previous stroke, <i>n (%)</i>	116	9 (17%)	7 (24%)	4 (11%)	0.41
Clinical SBP (mmHg)	102	159 (139; 172)	134 (123; 145)	136 (116; 151)	0.001
Clinical DBP (mmHg)	102	81 (69; 94)	84 (76; 94)	85 (75; 95)	0.155
Clinical heart rate (bpm)	100	65 (59; 77)	71 (63; 82)	67 (63; 83)	0.262
Daytime ambulatory SBP (mmHg)	91	156 (138; 173)	135 (127; 147)	135 (126; 149)	<0.001
Daytime ambulatory DBP (mmHg)	91	83 (73; 96)	89 (85; 98)	85 (80; 96)	0.184
Daytime ambulatory heart rate (bpm)	86	68 (62; 75)	78 (69; 84)	78 (71; 82)	0.014
Serum creatinine (μmol/l)	113	127 (100; 155)	83 (73; 97)	85 (75; 97)	<0.001
eGFR MDRD (ml/min/1.73 m ²)	86	46 (38; 62)	83 (69; 100)	78 (66; 93)	<0.001
Microalbuminuria (mg/l)	61	16 (7; 44)	6 (3; 8)	5 (3; 18)	0.107
RI lateralization (R/L/bilateral), <i>n</i>	116	18/26/8	8/11/10	10/17/8	0.4
Antihypertensive drugs, <i>n (%)</i>	115	50 (98%)	23 (79%)	27 (77%)	0.0025
Antihypertensive drugs, <i>n</i>	115	3 (2; 4)	2 (1; 2)	2 (1; 3)	<0.001
Lipid-lowering drugs, <i>n (%)</i>	114	32 (63%)	6 (21%)	10 (29%)	<0.001
Antidiabetics drugs, <i>n (%)</i>	115	6 (12%)	1 (4%)	2 (6%)	0.38

ASD, atherosclerosis disease; bpm, beats per minute; DH, dissecting hematoma; eGFR, estimated glomerular filtration rate; FMD, fibromuscular disease; L, left; MDRD, modification of diet in renal disease formula; *n*, number; R, right; RI, renal infarction.

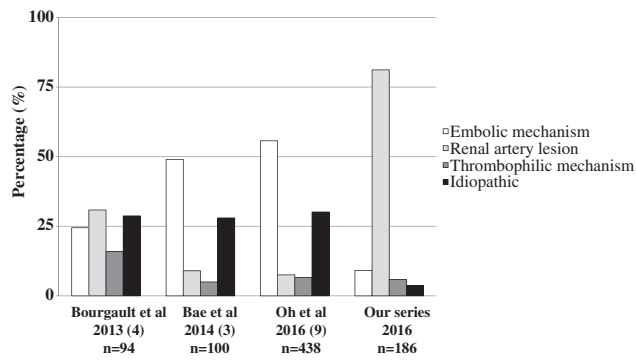


FIGURE 2 Respective frequency of renal infarction mechanisms.

DISCUSSION

We report here the largest retrospective single-center series of renal infarction. Over 15 years, 186 consecutive patients were admitted to a specialized Hypertension Unit in a tertiary hospital center. The renal infarction prevalence is 1.0% and the incidence 0.07%. The most frequent mechanism was renal artery lesion (81.2%). The most frequent radiological feature was renal artery dissection related to three major causes: dissecting hematoma, FMD and an association with arterial aneurysm/dissection in other sites. The second most frequent radiological feature was renal artery occlusion principally due to ASD. Patients clinical characteristics were as expected: patients with ASD were older and had more cardiovascular risk factors and comorbidities than patients with dissecting hematoma or FMD. ‘Embolic’ and ‘thrombophilic’ mechanisms were less frequent, respectively, 9.1 and 5.9%. We found a small percentage of idiopathic renal infarction (3.8%).

Our current results amplify those of our first study conducted in 55 renal infarction patients, between 1986 and 2004, in which 74.5% had renal artery lesion ($n = 41$): 26 dissections, 10 stenosis, five aneurysms. Renal infarction was ‘idiopathic’ in five patients (9%) [1]. It is of note that some results differ from the three other large retrospective renal infarction series [3,4,9] (Fig. 2). In the study from Bourgault *et al.* [4] (94 patients), the authors found a high

frequency of ‘renal artery lesion’ (30.8%) but also of ‘embolic’ mechanisms (24.5%). The ‘embolic’ mechanism was much more frequent in the two Korean studies (including 100 and 439 patients) reaching 49 and 55.7%, respectively. ‘Renal artery lesion’ was much less common reaching 9 and 7.5%, respectively [3,9]. Furthermore, these three studies report a much higher frequency of ‘idiopathic’ renal infarction (nearly 30%). The distribution of the various renal artery radiological features (occlusion, dissection, stenosis, aneurysms) was not available in these studies. In two studies, the most frequent renal artery lesion cause was dissection (58.6 and 63.6%, respectively) [4,9], but information about cause is lacking. Bae *et al.* [3] do not report specific renal artery lesion cause, and Oh *et al.* [9] mention no FMD. More surprisingly, none of the three studies report ASD as a renal infarction cause, whereas a literature review indicates a frequency of 5% [7]. This could be explained by the less sudden or dramatic nature of renal infarction symptoms in cases of ASD and suggests that some patients with ASD-related renal infarction might go undetected. Figure 3 shows the frequencies of renal artery lesions causes in our study compared with the three others.

The differences between these three other studies and ours can be summarized as follows. The first keypoint could be a recruitment bias. The study from Bourgault *et al.* [4] was a questionnaire-based analysis conducted in 14 Nephrology and Internal Medicine Departments involved in the management of acute renal infarction. The centers were asked to complete a questionnaire concerning patients with renal infarction ($n = 94$) who were identified from clinical diagnosis databases over 22 years (1989–2011). The study from Bae *et al.* was conducted in an Internal Medicine Department. The authors retrospectively reviewed the charts of patients diagnosed with acute renal infarction ($n = 100$) over 7 years (1995–2012) [3]. Neither this Korean study nor the French one provided further details about patient screening or selection [3,4]. The study from Oh *et al.* was conducted in nine Internal Medicine Departments by screening patient charts from January 1993 to December 2013. Renal infarction cases were identified by the code of International Classification of Disease and by a discharge diagnosis of ‘renal ischemia’, ‘renal infarction’ or ‘atherosclerosis of the renal artery’.

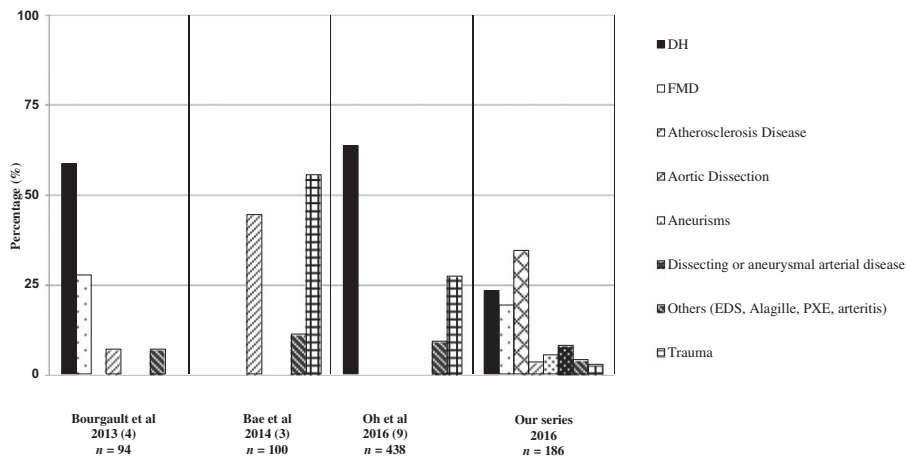


FIGURE 3 Respective frequency of renal artery lesion causes. DH, dissecting hematoma; EDS, Ehlers Danlos syndrome; FMD, fibromuscular dysplasia; PXE, pseudoxanthoma elasticum.

Finally, 1606 patients were identified and renal infarction was attested by radiological evidence which was only based on a formal report by a radiologist in 438 [9]. Our study is a single-center series, conducted in a specialized Hypertension Unit of a tertiary hospital center. The hospital includes our Hypertension Unit, a Cardiovascular and Interventional Radiology Department, a Cardio-Vascular Surgery Department and a Rare Vascular Diseases Centre, the latter especially managing patients with FMD. All patients with renal infarction were systematically presented at a weekly multidisciplinary round including hypertension specialists, nephrologists, cardiologists, vascular radiologists, interventional radiologists and vascular surgeons. All of them have a robust experience in renal artery disease management. A second difference could be the study period during which significant improvements were made in imaging, resulting in a more precise diagnosis of renal artery lesions. Another difference might be a consequence of our renal infarction diagnostic procedure in an etiologic approach. In fact, linking the radiological feature of the renal artery to a mechanism – general or local – and then to a cause might sometimes be tricky when faced with a total occlusion or an isolated dissection. As a consequence, systematic extensive investigation of other arterial sites (contralateral renal artery, aortoiliac and digestive axis, carotid and Willis polygon) is performed. If there is no evidence for a renal arterial disease, our renal infarction support procedures stipulate that each patient must undergo transesophageal echocardiography, Holter-EKG and thrombophilia investigations. Therefore, there is not only an obvious recruitment bias due to the specialization of our center, but probably also a more accurate renal artery analysis. This is attributable to the specificity of our renal infarction procedure and the involvement of a specialized multidisciplinary team. These reasons could explain the preponderance of renal artery lesion and the varieties of vascular diseases we found. Furthermore, our results could help assessing a clearer definition of renal artery dissecting hematoma and make nosology more precise (i.e. for arterial disease associating dissection and aneurysm in different sites), ensuring an improvement in renal infarction classification [13,16].

Our study displays many strengths. First, this is the largest currently available single-center study. Second, our renal infarction diagnostic procedure includes systematic extensive investigation of other arterial sites to detect other arterial lesions and sharpen the diagnostic approach. Third, clinical and all available imaging data are reviewed for all our patients with our multidisciplinary specialized staff. Fourth, all the data stored in our CDW were extracted from a specific and standardized robust designed questionnaire used since 1975.

Our study has some limitations. First of all, this is a retrospective study: therefore, some patients could have been underdiagnosed and some clinical and laboratory data were lacking. Second, this is a single-center study performed in a Hypertension Unit specialized in renal artery lesions among others: so we cannot exclude a selection bias in favor of renal artery lesions. Third, we are unable to conclusively identify specific clinical and biological features regarding the main underlying mechanisms owing to the somewhat limited sample size. Finally,

we cannot exclude that some abnormalities coexist (e.g. atherosclerosis and embolic event).

Perspectives

The differences between previous published studies and our results highlight the need for extensive arterial investigations and a multidisciplinary approach. The better the diagnosis, the better the management and the treatment. For example, if a renal artery lesion is identified, anticoagulant treatment is useless, whereas it is mandatory in ‘embolic’ and ‘thrombophilic’ mechanisms. Given the rarity of renal infarction, and even more of renal artery dissection, no randomized controlled trial has been conducted to evaluate the need and effect of anticoagulant treatment in this setting. However, we must remember that the *CADISS* trial found no difference in efficacy of antiplatelet and anticoagulant drugs to prevent stroke and death in patients with symptomatic carotid and vertebral artery dissection [17]. Likewise, identifying renal artery lesion could imply surgical management [13] or endovascular treatment [18] in certain rare cases.

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Conflicts of interest

There are no conflicts of interest.

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