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Clinical Report

Spontaneous renal artery dissection with renal infarction

Sophie Renaud[1], Hélène Leray-Moraguès[2], Leila Chenine[2], Ludovic Canaud[3], Hélène Vernhet-Kovacsik[4] and Bernard Canaud[2]

[1]Physiology Department, George Pompidou European Hospital, APHP, Paris, France, [2]Nephrology, Dialysis and Intensive Care Unit, Lapeyronie Hospital, CHU Montpellier, Montpellier, France, [3]Department of Thoracic and Vascular Surgery, Arnaud de Villeneuve Hospital, CHU Montpellier, Montpellier, France and [4]Vascular and Radiology Department, Arnaud de Villeneuve Hospital, CHU Montpellier, Montpellier, France

Correspondence and offprint requests to: Bernard Canaud; E-mail: b-canaud@chu-montpellier.fr

Abstract

Spontaneous renal artery dissection (SRAD) is a rare entity, which often presents diagnostic difficulties because of its non-specific clinical presentation. We report six cases complicated with renal infarction, occurring in middle-aged male patients without risk factors, illustrating the difficulty and delay for diagnosing SRAD. Ultrasound and Doppler imaging were not sensitive enough to confirm the diagnosis, and contrast-enhanced abdominal computed tomography was used to correct the diagnosis and allow the clinicians to propose appropriate treatment. We conclude that considering the urgency in diagnosing and treating SRAD, contrast enhanced abdominal tomography and/or abdominal magnetic resonance imaging should be proposed as soon as a suspicion of SRAD is evoked by the clinical presentation.

Keywords: arterial dissection; extreme exertion; renal infarction; spontaneous

Introduction

Renal artery dissection occurs mainly after direct vessel injury (such as trauma or endovascular intervention) or in hypertensive patients with underlying arterial diseases (such as fibromuscular dysplasia or atherosclerosis). Spontaneous renal artery dissection (SRAD) is a rare and often unrecognized entity. The clinical presentation with acute low back or flank pain and haematuria is readily misleading and suggests renal colic. Conventional imaging, including ultrasound and Doppler explorations, is not sensitive enough to detect the dissection nor the renal infarction and patients are usually treated for urolithiasis unless a computed tomography scan (CTS) is performed. Since the first report in 1944 [1], almost 200 cases of SRADs have been published, of which ~25% were diagnosed at necropsy. SRAD is certainly more common than reported and its frequency is obviously underestimated. The availability of non-invasive and more sensitive-contrasted imaging techniques should facilitate recognition of such cases while reducing the delay in diagnosing SRAD and allowing earlier treatment.

Cases report

In this study, we report six patients who were referred to our nephrology department between 1997 and 2010 and presented with SRAD. All were men between 33 and 55 years of age. They had no cardiovascular risk factors, except one patient who had a past history of smoking. All patients presented acute unilateral flank pain with inguinal irradiation and sometimes haematuria on urinary dipstick testing and were most commonly diagnosed with renal colic, delaying the diagnosis of renal infarction by several days (Table 1). Most were hypertensive but only two patients had acute renal failure. Proteinuria was significant in two cases. Among the most frequent biochemical abnormalities, we noted elevated lactate dehydrogenase concentrations and inflammatory markers including increased white blood cells, C-reactive protein and fibrinogen levels (Table 2). Haemostasis was within the normal range, but the research for circulating anticoagulant was positive in two patients (data not shown). Renal ultrasound was performed in all cases at admission but failed to detect renal infarction. We performed contrast-enhanced abdominal CTS to diagnose the renal infarction (Figure 1) and estimate the area of extension (between 15 and 50% of the kidney surface). Renal infarction affected mainly the left side (4/6) but was bilateral in one patient. We then performed angiotomodensitometry (A-CTS) in some cases and magnetic resonance imaging angiography (A-MRI) in others, completed by percutaneous selective renal arteriography (Figure 2) to identify vascular lesions and the vessels' anatomy. Three patients had multiple renal arteries and the dissection usually affected segmental branches but concerned also the main renal artery in two cases. Signs of arterial fibrodysplasia were detected in four patients. Only one of them was treated by angioplasty with stent implantation (two stents), whilst conservative management was elected for all other patients for various reasons including the presence of a circulating false lumen, overpassed renal infarction or dissection of small branches of arteries, precluding revascularization possibilities. All patients were treated by oral anticoagulation (anti-vitamin K) or anti-platelets. Two patients were lost to follow-up; the others were followed up for a period of between

4 months and 13 years. They had normal blood pressure (one without any treatment and three with anti-hypertensive drug therapy) and their renal function remained stable.

Discussion

As illustrated by our clinical report, detection of SRAD is often delayed or missed because the condition is rare and its clinical presentation is non-specific [2]. Indeed, patients with acute renal infarction commonly have abdominal, flank or low back pain as in renal colic. The clinical presentation in our series is mainly consistent with previous reports but also illustrates new findings. First of all, there is a male predominance with a classic male to female ratio of 4:1 [3]. This predominance could be even stronger with a ratio of 10:1 as described by Edwards *et al.* [4] in the most important series to date reporting on 35 patients. SRAD occurs predominantly in the fourth decade (mean age of our series is 39 years) of patients without known cardiovascular risk factors except tobacco use. Another finding that our series showed was that the left renal artery was clearly more frequently involved. Some authors suggest that anatomical characteristics predispose the left renal artery to dissection in the case of traumatic injury or acceleration/deceleration [5–8]. Bilateral disease is encountered in 10–15% of SRAD cases [9–11] usually because of underlying arterial disease. We identified fibromuscular dysplasia in half of our cases, confirming the high prevalence reported by Lacombe *et al.* [9] in their series of 22 patients treated by surgery. The third finding that we want to underline is the fact that spontaneous dissection can occur in normal vessels under normal blood pressure, but subjected to extreme exertion, as was the case with patients five and six, who practised body building. Some authors suggest that vigourous exercise may subject the renal artery to such an unusual degree of stretching that it causes intimal tear and subsequent arterial dissection [12–15]. More discordant is the fact that our patients were not all hypertensive at diagnosis. In rats, kidney infarction induces acute hypertension associated with a transient renin elevation [16]. In humans, several authors have described the occurrence of malignant hypertension in the aftermath of a renal infarction [13, 17–19] whereas other case reports suggested that hypertension is not consistently associated, some patients remaining normotensive [12, 20–23]. It is important to note that in most of the series, patients are referred for endovascular [2] or surgical [9, 10, 24–26] revascularization, whose indication relies on the presence of uncontrolled hypertension and/or kidney failure, which easily explains the nearly 100% rate of hypertension. Moreover, there was a large proportion of fibrodysplasia (up to 90%) [4, 26] and/or atheroma [25, 27] identified in the 'surgical series'. The high prevalence of arterial pathology underlying the dissection may also help to explain the high frequency of hypertension in these series.

In cases of SRAD with renal infarction, the diagnosis must be made as early as possible to increase the chances of renal revascularization and recovery. Our experience confirms that Doppler and/or ultrasound have poor diagnostic sensitivity

Table 1. Demographic and baseline clinical and disease characteristics ($N = 6$)

Characteristics	Value
Age (years)	
Mean	42
Range	33–55
Sex	
Male	6
Female	0
Medical history	
Smoking	1
Familial hypertension	1
Disease manifestations at presentation	
Low back pain	4
Flank pain	2
Headache	1
Lipothymia	1
Ileus	1
Fever	1
Hypertension	4
Haematuria	2
Hypotheses diagnosis	
Pyelonephritis	2
Urolithiasis	6
Stomach ulcer	2
Retrocaecal appendicitis	1
Interval from symptom onset to diagnosis (days)	
Mean	9
Range	2–20

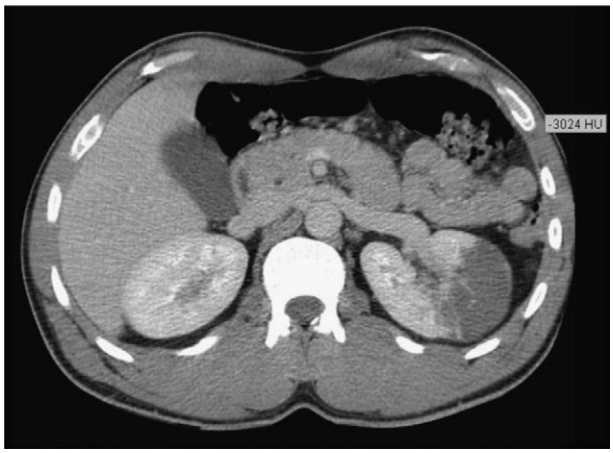

Fig. 1. Enhanced abominal CTS showing median infarction of the left kidney (patient number six).

Table 2. Biochemical parameters at diagnosis[a]

Patient number	Creatinine μmol/L	Proteinuria g/24 h	LDH UI/L	ASAT/ALAT UI/L	WBC/mm^3	CRP mg/L	Fibrinogen μmol/L (g/L)	PT/PTT %/s
1	89	0.21	NA	NA	14300	NA	0.18 (6.1)	NA/40
2	87	0.14	379	53/143	6000	6	0.12 (4.2)	90/37
3	143	0.38	NA	17/15	5100	5	0.10 (3.3)	100/28
4	127	0.08	NA	NA	6500	15	0.10 (3.6)	NA
5	74	1.15	785	33/77	8900	64.5	0.23 (8)	100/30
6	97	0.22	NA	42/44	10000	13	0.10 (3.6)	82/28

[a]CRP, C-reactive protein; NA, not available; WBC, white blood cells; ALAT, alanin aminotransferase; ASAT, aspartate aminotransferase; PT, prothrombin time; aPTT, activated partial thromboplastin time.

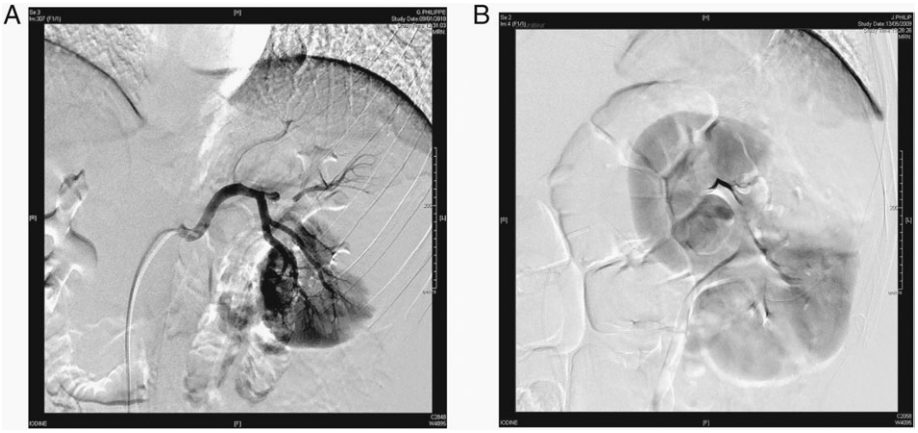

Fig. 2. Renal selective angiography; (A) patient number six; (B) patient number five.

since the dissection may affect intra-renal arterial branches and polar arteries whilst CTS, on the other hand, never failed to identify the area of renal infarction. The performance of invasive exploration by angiography is recommended at an early stage to identify the vascular lesion and propose endovascular treatment [28].

The precise mechanism by which SRAD occurs still remains poorly understood. Several hypotheses have been proposed to explain its pathophysiology. One hypothesis raised by two small series suggests that physical exertion could be the trigger for dissection by stretching the arterial wall [12, 13]. The right renal artery is less often affected than the left because its greater length allows a better distribution of shear stress. Another hypothesis, as believed by some authors, suggests that SRAD is a clinical variant of fibromuscular dysplasia or atherosclerosis. In our series, however, the demographic characteristics of our patients are not those commonly observed in arterial dysplasia or atherosclerosis as these were men without any identified cardiovascular risk factors. Additionally, in contrast to atherosclerotic lesions, the dissection occurred in the distal part of renal artery rather than the aortic ostium. Finally, in half of our cases, the dissecting process also affected intra-renal arterial branches and sometimes the polar arteries.

Once diagnosed, there are several options for the treatment of SRAD ranging from conservative treatment to endovascular and surgical intervention, depending on the stage at which the diagnosis is made and the severity of the renal lesion. There is no therapeutic consensus and revascularization is usually proposed as a second option, for patients with medically uncontrolled hypertension or progressive renal dysfunction [3, 4, 9, 24, 25]. Anti-hypertensive treatment combined with anti-aggregate platelet therapy appears to be safe and effective in most patients, with a mean follow-up reaching almost 10 years [12, 29–31]. Although the natural history of renal artery dissection has not been well documented, it seems that remodelling of the dissected artery with re-entry points may restore a nearly normal renal flow, as it occurred with patient number six and as suggested by cases of spontaneous resolution [4, 11]. Misrai *et al.* [30] studied the anatomical evolution of dissections by CT angiography with three-dimensional reconstruction and found that arterial remodelling occurs in most of the dissected branches without occlusive or aneurismal evolution. The poor outcome of surgical arterial reconstruction with a high rate of nephrectomy (40%) is attributed in part to the frequent involvement of renal artery branches. Percutaneous endovascular treatment with renal artery stenting is quite appealing and appears more likely to supplant surgical intervention since it is a less invasive and a safer treatment [2, 15, 28, 32, 33]. Nevertheless, endovascular approaches are indicated depending on how early the diagnosis is made and how quickly the revascularization occurs, which is crucial to reducing the area of infarction and preserve renal function. Due to this short time frame for intervention, we consider that direct non-invasive imaging by angio-CTS or angio-MRI is required if SRAD is suspected.

Conclusion

The clinical presentation of SRAD is misleading and the diagnosis should be advocated when renal crisis is not associated with urolithiasis or alternatively when hypertension is present. Physicians should also research any intensive exercise in the anamnestic history of the patient. Renal ultrasound and Doppler examination are not sensitive enough to make the diagnosis. CT or MRI should be proposed instead as soon as encountering any diagnostic difficulties. Such imaging techniques are invaluable for assessing renal artery morphology and area of renal infarction [17, 18]. Renal arteriography and endovascular stenting offer the best treatment opportunities. Early recognition of SRAD and endovascular treatment are essential for improving renal outcome. The six cases reported here highlight the difficulties in establishing the diagnosis of SRAD and emphasize the importance of performing appropriate renal imaging, as soon as the diagnosis is considered.

Conflict of interest statement. None declared.

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