Renal infarction management: towards an etiological approach?

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In 1993, a 66-year-old woman was admitted to our Emergency Department for hypertensive crisis. She complained of nausea, vomiting and abdominal pain. A biological work-up disclosed moderate renal impairment (plasma creatinine: 1.7 mg/dl, eGFR: 32 ml/min per 1.73 m²) with neutrophilic leukocytosis (20.180/µl, 93% neutrophils) and Lactate Dehydrogenase elevation (985 U/l). Renal infarction of the left kidney was demonstrated by computed tomography-angiography (CTA). Arteriography highlighted a severe stenosis (90%) of the left renal artery with partial thrombosis of a poststenotic dilatation. Local thrombolysis with urokinase and angioplasty were performed, with a residual stenosis of 30% [1]. This case raises questions about the cause (fibromuscular dysplasia versus atherosclerosis), optimal diagnostic approach and management of renal infarction. Twenty-five years later, these questions remain largely unaddressed.

In the case of renal infarction, brutal reduction of the flow in a main renal artery and/or its branches usually leads to nonspecific clinical manifestations, with a diagnosis frequently delayed from hours to days [2]. Renal infarction may indeed mimic a large range of abdominal disorders (pyelonephritis, nephrolithiasis, appendicitis, mesenteric ischemia ...), or other renal diseases (hypertensive crisis, acute renal failure, ...), and its frequency is probably underestimated [3]. Furthermore, the pathophysiology and optimal management of renal infarction remain an unexplored field, as only retrospective studies and case-reports are available [2–5]. In 2013, a practical classification of four different groups of renal infarction was proposed by Bourgault et al. [2]: renal infarction (i) of cardiac (embolic) origin, (ii) associated with renal artery injury, (iii) associated with hypercoagulability disorders and (iv) apparently idiopathic forms. Since then, all retrospective studies used this classification. In some publications [3,4], the most frequent cause was the embolic form (~50%), whereas other reported an equal distribution (25% each) of embolic, vascular and idiopathic forms [2].

In the current issue of the Journal of Hypertension, Faucon et al. [5] carefully reviewed 186 cases of renal infarction admitted in their tertiary center from July 2000 to June 2015. They reported the causes of renal infarction according to the four categories described previously [2]. In this large series, they highlight the added value of a multidisciplinary approach to limit the number of idiopathic forms (n = 7) and correctly classify each patient [5]. It seems indeed crucial to assess the diagnosis accurately to avoid unnecessary anticoagulation, thrombolysis, surgery or stenting. In ‘vascular forms’, Faucon et al. [5] also stress the importance of screening other vascular territories (contralateral renal artery, carotid arteries, ...), to differentiate atherosclerotic renal artery stenosis, dissecting forms of fibromuscular dysplasia or isolated dissecting hematoma. In particular, fibromuscular dysplasia and dissecting hematoma are probably overlooked in a substantial number of cases. Systemic exploration of other vascular beds may contribute to decrease the proportion of idiopathic forms (3.8% in this series versus 30% in the three other recent series) [2–5] and help the clinician to distinguish between dissecting forms. Indeed, fibromuscular dysplasia affects two or more arterial territories in over 50% of cases [6,7]. Accordingly, the identification of a typical string-of-beads in another artery may uncover the cause of renal artery dissection.

Interestingly, in contrast with previous studies, in the series of Faucon et al. [5], the ‘vascular’ form of renal infarction (~80%) was predominant, much more frequent than the ‘embolic’ form (~10%). This may be partly due to a much lower prevalence of atrial fibrillation: 4.3% in this series versus 21–48% in previous reports [2–5]. As acknowledged by the authors, this low prevalence probably reflects a recruitment bias linked to the expertise of the center (i.e. hypertension and arterial diseases) [5]. In less selected cohorts, the proportion of embolic forms may be higher and will likely increase further during the next decade, in the wake of the announced atrial fibrillation ‘epidemic’ in our ageing Western world [8].

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Finally, iatrogenic forms of renal infarction should not be overlooked. Although the latter were excluded from the current publication of Faucon et al. [5], in a preliminary analysis of their data presented at the 2016 European Meeting on Hypertension in Paris [9], the proportion of renal infarction due to endovascular or surgical procedures was 43 of 229 (19%). With the wider use of endovascular surgery of the aorta and its branches, renal infarction and renal artery thrombosis may become an increasing matter of concern [10].

Beyond academic discussions, in clinical practice, the challenge is to make the diagnosis and identify the cause of renal infarction to individualize management and orient treatment and follow-up (Fig. 1). For the diagnosis, in the absence of contraindication, the imaging modality of choice is definitely abdominal CTA. Common findings include wedge-shaped, peripheral nonenhancing areas and, in some cases, the typical ‘cortical rim sign’, which corresponds to preserved perfusion of a thin rim of the cortex by perforating branches of the renal capsular artery (Fig. 2a). The alternative diagnostic method is magnetic resonance-angiography (MRA) (Fig. 2b). Renal arteriography remains the gold-standard [1–5] and can be associated with endovascular treatment.

Once the diagnosis is established, the mechanism of renal infarction (embolic, vascular, thrombophilic, iatrogenic) needs to be identified. The clinical context (family history, atrial fibrillation, recent aortic procedure, ...) should be taken into account. Systemic thromboembolism originating from the heart or the aorta should be researched using transthoracic/transesophageal echocardiography (particularly in case of atrial fibrillation) and thoracoabdominal CTA or MRA. Aortic assessment by CTA may also identify rare vascular diseases (aortitis, ...) or dissection. Primary or acquired hypercoagulability should be assessed by expert clinicians in the field of hemostasis to avoid futile biological assays [11]. Finally, in the absence of clear cause during the acute phase, a second assessment of renal arteries seems appropriate 3–6 months after renal infarction, particularly in case of renal infarction associated with renal artery dissection. Indeed, subtle lesions of fibromuscular dysplasia may be difficult to ascertain during the acute phase of renal infarction due to local inflammation. If not already done, vascular work-up should be extended to extrarenal territories – particularly cervicocephalic arteries – to detect lesions suggestive of fibromuscular dysplasia, vascular Ehlers–Danlos syndrome or rare inherited vasculopathies. As highlighted by Faucon et al. [5], the expertise of a multidisciplinary team (clinicians, radiologists) is invaluable to optimize the analysis of the underlying mechanisms (i.e. local ‘vascular’ or general ‘embolic’) of renal infarction and facilitate reclassification of idiopathic causes of renal infarction.

Therapeutic options include conservative management (pain, hypertension, ...), anticoagulation and/or revascularization depending on the cause of renal infarction, the severity of the disease and the experience of surgeons, interventional radiologists or cardiologists of the center [1–5]. Anticoagulation with heparin will be started in the majority of patients, particularly in the embolic and hypercoagulable categories. Intra-arterial thrombolysis is a therapy of choice when the thrombus extends to intrarenal arteries. Combination of local thrombolysis and thrombectomy is useful in case of bilateral renal infarction, of solitary kidney or when the thrombus is localized in the proximal part of the renal artery. Renal artery stenting and antiplatelet therapy are sometimes advocated in case of associated atherosclerotic lesions. Nephrectomy has become exceptional today [1–3,12].

In conclusion, given the infrequent occurrence of renal infarction, the optimal strategy of diagnosis and management of the disease remains elusive. With their large
retrospective case-series, Faucon et al. [5] shed new light on etiological work-up and causes of renal infarction. Their approach would deserve to be applied prospectively in other centers with different patient recruitment, preferably in the context of national and international registries, as is currently done for fibromuscular dysplasia [13,14].

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Conflicts of interest
There are no conflicts of interest.

REFERENCES