ACUTE RENAL INFARCTION: A SINGLE CENTRE EXPERIENCE.

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INTRODUCTION

Acute renal infarction, due to an abrupt interruption of blood flow in the renal artery, is frequently not recognized. Non specific clinical presentation, mimicking other conditions (i.e., nephrolithiasis, pyelonephritis), may explain the possible delay in diagnosis. Major causal factors include atrial fibrillation, valvular or ischaemic heart disease, renal artery thrombosis/dissection, coagulopathies. Etiology remains in many cases unknown.

METHODS

We retrospectively analysed the medical records of 16 patients (12 M and 4 F) with renal infarction diagnosed by computed tomography (CT) or magnetic resonance imaging (MRI) admitted to our Nephrology Department between 1999 and 2012 and subsequently followed for 10 years as outpatients. We describe the clinical characteristics and outcome of 16 patients with acute renal infarction.

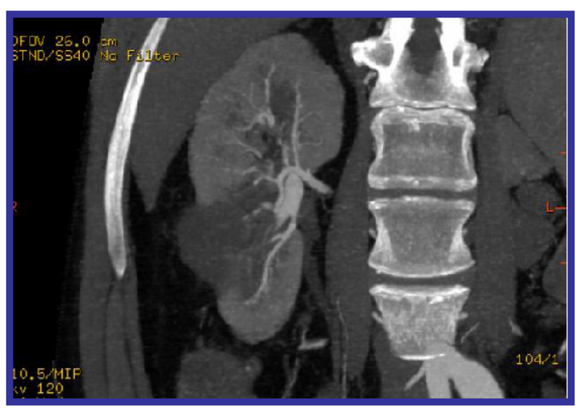
RESULTS

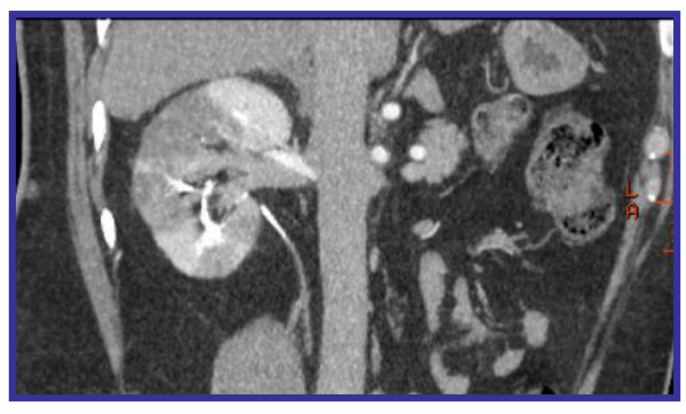
Mean age was 58.62 years; eleven patients presented with lumbar pain, 3 with diffuse abdominal pain. In 2 patients renal infarction was painless. Associated symptoms included macroscopic haematuria (3 patients), arterial hypertension (9), hyperthermia (4), oliguria (2) and dysuria (1).
Possible associated risk factors included obesity/overweight (6 patients), current smoking (3), cocaine abuse (1), estroprogestinic therapy (2), atrial fibrillation (4), and atrial mixoma (1 patient).
Six out of 11 patients were found to have antophospholipid antibodies at the diagnosis (anti-β2GPI antibodies in 3, anticardiolipin antibodies in 2, LAC in 1), but only one had these antibodies still detectable 12 weeks later. Systolic arterial pressure was 147.69±18.99 mmHg, diastolic 83±10 mmHg. Laboratory investigations at onset are shown in table 1.
Acute kidney injury occurred in two patients at presentation. CT scan/MRI imaging showed alterations of renal arteries in 9 cases (thrombosis in 5 patients, renal artery dissection in 1, fibromuscular dysplasia in 2 and both thrombosis and dissection in 1). CT scan images included single or multiple triangular defects in the renal parenchyma (Fig. 1 and 2)
Thirteen patients were treated with LMWH,2 with aspirin alone; one patient did not receive anticoagulant/antiplatelet treatment because of severe arterial hypertension.
At the end of follow-up (23.6±36 months in 9 patients), serum creatinine was 1.1 ± 0.2 mg/dL; one patient remained on chronic hemodialysis. In 4/6 patients who underwent renal scintigraphy after a median of 28 months (range 6-120), the contribution of the affected kidney to total renal function was reduced.

Table 1

Clinical and laboratory parameters	
PAS	147,69 18,99 mmHg
PAD mmHg	83 10 mmHg
Leukocytes	10.465 4036 x 10^3/ul
LDH	1.793 618 UI/l
CRP vn< 0,5 mg/dl	14 12,2 mg/dl
Serum Creatinine	1,4 0,6 mg/dl
eGFR	65 30 ml/min/1,73 m2

Fig. 1 and 2





Single or multiple triangular defects in the renal parenchyma

CONCLUSIONS

Fifty % of our patients had an idiopathic renal infarction. Renal artery abnormalities (fibromuscular dysplasia, renal artery dissection) were present in 25% of patients, cardioembolic etiology in 25%. Clinical presentation was confirmed to be non specific. Further studies should focus on etiology and evolution of kidney function in patients with acute renal infarction.

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149--SP
H) Clinical Nephrology, CKD. H1) Lab methods, progression & risk factors for CKD 1-5, nutrition in CKD, renal diseases (except GNs and cystic diseases).
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