Renal colic in a patient with anti-phospholipid antibodies and factor V Leiden mutation

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Introduction

Pain due to urolithiasis may present as either a dull ache in the renal angle or, more classically, as renal (ureteric) colic [1]. The latter is a severe flank pain which typically radiates from the loin to the groin, perineum, labia, scrotum or penis, often associated with vomiting. However, although renal colic is thought of almost pathognomically as representing a kidney stone, other diagnoses should be borne in mind. We observed a patient with anti-phospholipid antibodies (APLA) and factor V Leiden mutation who was admitted with ureteric colic. Initial misdiagnosis as urolithiasis resulted in almost total loss of function of her left kidney and the development of renovascular hypertension.

Case

A 50-year-old Caucasian female presented to the casualty department complaining of intense left flank pain accompanied by fever. Her symptoms had commenced abruptly ~6 h prior to her arrival at the hospital. Relevant past history was notable for an admission 3 years previously because of acute thrombosis of the left subclavian, internal jugular and brachiocephalic veins to the point of entry to the superior vena cava. The patient was known to be a heavy smoker (30 pack years). She was gravida 5, para 4, having undergone one spontaneous abortion (her fourth pregnancy). She was menstruating regularly and was not on any oral contraception. During the previous hospitalization, a coagulation screen revealed positive APLA (IgG 61 GPL units, normal <23) and a heterozygous state of factor V Leiden mutation (activated protein C (APC) resistance of 1.1, normal 2.1–4). Lupus anticoagulant, anti-thrombin III, protein C activity and total protein S were within normal limits. Anti-nuclear factor was negative.

The patient was then begun on anticoagulant therapy and eventually discharged on warfarin, to be continued permanently. She was advised to keep her INR between 2 and 3. Over the ensuing months, recanalization of the thrombosed veins occurred with resolution of the left upper limb oedema.

On her current admission, apart from a body temperature of 38°C, physical examination was unremarkable. Blood pressure was 140/80 mmHg. Serum creatinine was 1.4 mg/dl (previous basal value 0.9 mg/dl). Her INR was 1.3, prothrombin time 83%. Urinalysis showed 2–3 RBC/HPF with trace proteinuria. A plain abdominal X-ray did not reveal any calcified shadows.

Two months later, the patient developed hypertension (160/100 mmHg). Basal PRA was 1.8 increasing to 2.8 ng/ml/h after captopril administration. A repeat
Renal scintigram showed 77 and 23% uptake of the right and left kidneys, respectively. Serum creatinine had decreased to 1.1 mg/dl. Treatment with ramipril, 2.5 mg/day, promptly controlled the patient’s blood pressure.

Discussion

Acute occlusion of a renal artery in a 50-year-old female with no documented atheromatous disease is a rare occurrence. Renal fibromuscular dysplasia only very infrequently exhibits occlusive phenomena [2].

This patient presumably developed an acute thrombosis of her left main renal artery. Her presenting symptoms were renal colic and fever. She was known to harbour APLA and to be heterozygous for factor V Leiden mutation. She previously had experienced one major thrombotic episode and was on permanent warfarin therapy. In spite of this impressive history with more than a hint at a predisposition to thrombotic events, she was diagnosed initially as having urolithiasis. This resulted from misinterpretation of her mode of presentation with renal (ureteric) colic. Considered almost pathognomonic of kidney stones, renal colic must be differentiated from intestinal colic, appendiceal colic, torsion of an ovarian cyst, ruptured ectopic pregnancy and, as in our case, acute occlusion of the renal artery or vein. Interestingly, such an error in diagnosis is, unfortunately, commonplace. In a recently published nephrological textbook, the authors report on nine patients with a functional solitary kidney, in whom acute occlusion of the renal artery occurred [3].

All nine patients presented with oliguria and in seven of them, flank pain was part of the leading symptom.
In six of the nine patients, urolithiasis was the primary diagnosis.

The association of APLA with arterial or venous thrombosis is well known. However, even in this setting, renal artery occlusion is rarely documented. A literature review has yielded only 13 such cases including our own, of which five occurred in kidney allograft vessels [4].

To our knowledge, apart from our case, only once previously has factor V Leiden mutation been associated with acute renal artery thrombosis [5]. Due to the fact that our patient was both APLA positive and APC resistant, her thrombotic tendency was greatly increased. Contributing factors were smoking and the patient’s poor compliance with therapy as evidenced by an INR of 1.3 on admission.

Although there have been anecdotal reports of renal salvage even after prolonged periods of ischaemia [6], given that 72 h had elapsed since symptoms began and a total lack of perfusion on renal scintigram, no such attempt was made in our patient.

In summary, this case illustrates that the diagnosis of acute renal artery occlusion should be borne in mind in patients presenting with renal (ureteric) colic, especially so in those with a thrombotic predilection.

**Teaching point**

Not all renal colic represents kidney stone disease. Be aware of other diagnoses, in particular, renal artery thrombosis. In this condition, timely diagnosis is essential.

**References**