Renal artery embolism successfully revascularized by surgery after 5 days' anuria. Is it never too late?

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Introduction

Renal artery embolism (RAE) is an infrequent but significant cause of renal loss in patients suffering either from valvular cardiopathy or from aortic atheromatosis [1]. Although according to different authors [2] the duration of renal ischaemia is important, prolonged delay in revascularization does not defer recovery of renal function [3], since preserved vascularization could be present if incomplete obstruction or collateral circulation is patent. When arterial flow is re-established, and if massive infarction has not occurred, renal output can return to normal once tubular lesions have healed. We report a patient who had only one kidney, since the contralateral had been removed a year before due to renal-cell carcinoma. She suffered from a renal artery embolism and was successfully revascularized after 5 days of anuria.

Case report

A 76-year-old female was admitted to our unit with anuria and haematuria together with an acute right lumbar pain of 120 h evolution. A year before she had suffered a left nephrectomy due to a renal-cell carcinoma. No local relapse was seen in an abdominal scan carried out 6 months later.

She was also suffering from a intrathoracic hyperfunctional goitre, which was being treated with antithyroidal drugs. At the same time she was being given digoxin for chronic atrial fibrillation. On admission her blood pressure was 180–90. A systolic murmur was audible and bilateral oedema in the legs was clearly visible. An electrocardiogram showed atrial fibrillation at a rate of 110/min. Laboratory analysis revealed a serum urea of 66.75 mmol/l, serum creatinine of 786.7 μmol/l, K⁺ of 5.4 mmol/l, and lactic dehydrogenase of 2000 units/l. A renal echography showed a normal-size right kidney without urinary tract obstruction. A renal angiography confirmed complete occlusion of the main right renal artery (Figure 1) and selective catheterization through the embolus disclosed a patent inferior lobar artery as well as segmental and intrarenal arteries at the inferior lower pole of the kidney (Figure 2).

Her renal function steadily improved (serum creatinine 282.8 μmol/l, GFR 10.4 cc/min), allowing us to discontinue dialysis treatment. The patient has been on erythropoietin treatment since initiation of dialysis,
and she now presents a haemoglobin level of 10 g/l on a subcutaneous twice-weekly dose of erythropoietin.

After 3 months without dialysis the patient is now enjoying good health without any further clinical symptoms of disease. Her renal function reveals a GFR of 12 cc/min.

Discussion

The first bibliographic reference regarding kidney embolic disease dates back to 1856 [4]. Since clinical diagnosis of renal artery embolism is quite difficult, the real incidence of this disease is probably underestimated, as is shown in post-mortem studies [5].

It is known that the diagnosis of unilateral embolism can often be too late and can even go undetected. Laboratory analysis showing renal insufficiency is only seen in those patients suffering from renal bilateral embolism or in patients with renal unilateral embolism in whom the contralateral kidney is non-functioning or has been removed.

The rise in lactic dehydrogenase (I and II fractions) has been considered a good biological marker of screening in patients suffering from embolism [6], despite the fact that it can occur in many other conditions.

Transfemoral arteriography, performed early, seems to offer the best diagnostic sensitivity [7], although renal echo-Doppler studies and renographic studies have been found useful.

The choice of treatment is still controversial [8]. Several authors [9,10] are in favour of surgical embolectomy when renal artery embolism is bilateral, or unilateral with a solitary functional kidney, the embolus taking place at the main renal artery.

Although different authors [2] suggest that the duration of renal ischaemia is an important factor in the recuperation of renal function, successful late revascularization has been described [3]. Preserved vascularization either by incomplete obstruction or by collateral circulation enables the ischaemic kidney to restore its renal output when arterial renal flow is re-established and tubular lesions have healed.

Intra-arterial fibrinolytics have been used as a therapy of choice when the embolism takes place at intrarenal arteries [11,12].

Acute renal failure due to renal artery embolism should be considered in the differential diagnosis of elderly patients with atrial fibrillation or valvular cardiopathy who are suffering from abnormal lumbar pain and anuria resembling nephritic cholic pain.

It is well known that an appreciable number of elderly patients have only a single kidney, which contributes to glomerular filtration. In our particular case the patient had only one kidney, the contralateral having been removed a year before. Even though our patient had already been complaining of lumbar pain 5 days before we first saw her, surgery was still entirely successful, showing that surgery should not be denied when arteriographic studies shows possible revascularization.

When renal artery embolism occurs, quick diagnosis and treatment are compulsory in order to restore renal function and avoid end-stage renal disease and chronic dialysis, which in these patients is badly tolerated and entails a high social cost and morbidity.

Before surgery, haemodialysis was performed. Surgical revascularization was indicated and embolectomy at the main right renal artery was successfully carried out, thereby restoring normal blood backflow. An intra-arterial serum saline solution with 100,000 units of urokinase was administered as a bolus during surgical procedure.

Forty-eight hours after surgery, urine output was restored, but the patient required dialysis during the following days.

A cardiac echography showed a non-obstructive hypertrophic myocardiopathy with mild mitral insufficiency and no evidence of thrombi. A thyroid scintigraphy showed a multinodular goitre, with normal hormone levels (T4: 99.0 nmol/l. T3: 1.8 nmol/l).
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References

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