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Exceptional Case

Complete renal recovery from severe acute renal failure after thrombolysis of bilateral renal vein thrombosis

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Abstract
A previously healthy young man presented with acute renal failure due to extensive spontaneous deep vein thrombosis, including the inferior vena cava (IVC) and both renal veins. The patient was treated with selectively delivered thrombolytic therapy over a 7-day-period, which resulted in renal vein patency and complete recovery of renal function. A stent was placed over a segment stenosis of the IVC. No thrombophilic factors were identified. Bilateral renal vein thrombosis in young fit individuals is an unusual cause of acute renal failure. Thrombolytic therapy, even with delay, can completely restore renal function.

Keywords: acute bilateral renal venous thrombosis; inferior vena cava; renal failure; thrombolysis

Case report

A 31-year-old Caucasian man was admitted to a hospital with severe renal failure. He presented with a 1 week history of sudden onset of severe lower back and loin pain accompanied by swelling of both lower limbs and reduced urine output. He had no past medical or family history, and was an ex-smoker. Apart from his work as a taxi driver, there was no history of immobility, long airline journeys or major physical exertion. He took no regular medication. His blood pressure was 149/70 mmHg, pulse 100/min. Abdomen was distended without a palpable mass. Bladder was empty after catheterization and he remained anuric. Biochemically, he had severe renal failure (creatinine 1167 μmol/L = 13.2 mg/dL). His white cell count was $12\,800 \times 10^6$/mL, C-reactive protein 283 mg/L, erythrocyte sedimentation rate 55 mm and serum albumin 41 g/L. His clotting profile was normal, D-dimer was 15 570 ng/mL (normal below 250) and creatinine kinase was 51 IU/mL.

A central venous dialysis catheter was inserted into the right femoral vein. The operator reported difficulty in cannulating the vein but on haemodialysis high venous pressure and poor blood flow were noted. This catheter was removed and further haemodialysis was carried out via a right internal jugular venous catheter.

The next day, computed tomography (CT scan-venous phase contrast) of thorax, abdomen and pelvis revealed complete venous thrombotic occlusion extending from both common femoral veins to the supra-renal inferior vena cava (IVC), and both the renal veins were occluded with oedematous kidneys (Figure 1A). No malignancy was seen.

The interventional radiologists started thrombolytic therapy: initially, venous access was gained in the left femoral vein and digital subtraction venography demonstrated near-occlusive thrombus extending from the femoral vein to the supra-renal IVC as seen on CT. At this point, a 20 cm Cragg-McNamara thrombolysis catheter (EV3) was placed along the length of the IVC and a recombinant tissue plasminogen activator (rTPA) was administered as a bolus of 5 mg, followed by a continuous infusion of 1 mg/h for 12 h. A simultaneous infusion of unfractionated heparin at a rate of 500 IU/h was given through the femoral vascular access sheath for 48 h. Repeat venography at 12 and 48 h showed some minor improvements but still significant residual thrombus. Then, renal venography and thrombolysis were attempted via a right internal jugular vein approach after placement of a vascular access sheath. Imaging from here identified supra-renal IVC stenosis, which was angioplastied with a 12 mm diameter balloon. Venography showed heavy thrombus burden within the left renal vein and thrombus at the origin of the right renal vein. A new Cragg-McNamara (EV3) thrombolysis catheter was positioned in the left renal vein thrombus and further rTPA bolus (four pulse spray of 1 mg) followed by continuous infusion (1 mg/h) was given. Right renal vein patency was gained by suction thrombectomy of the thrombus at its origin and balloon angioplasty of the same region to disperse residual thrombus. Venography 48 h later showed complete patency of both renal veins; the supra-renal IVC

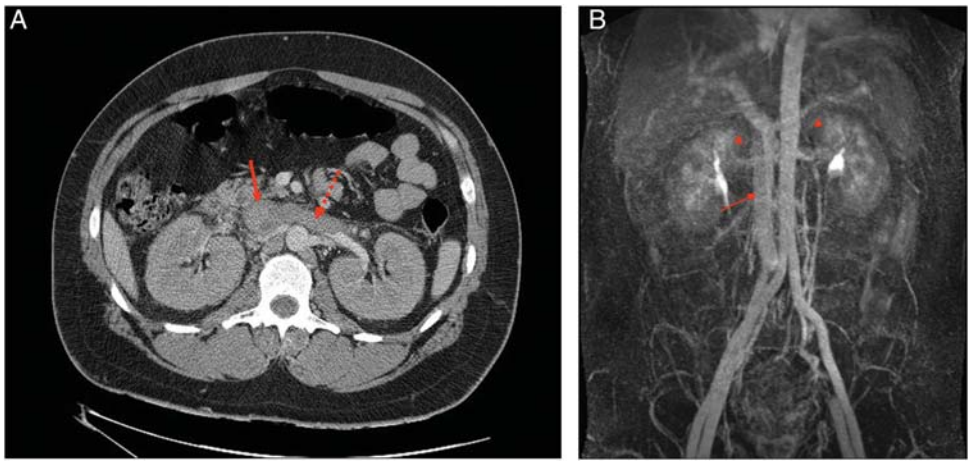

Fig. 1. Radiological imaging of renal veins. (**A**) Initial contrast enhanced CT scan demonstrating acute thrombus and venous distension within the IVC (solid arrow) and left renal vein (broken arrow). (**B**) Magnetic resonance venography 3 months later demonstrating patency of the IVC (arrow) and bilateral renal veins (arrow heads).

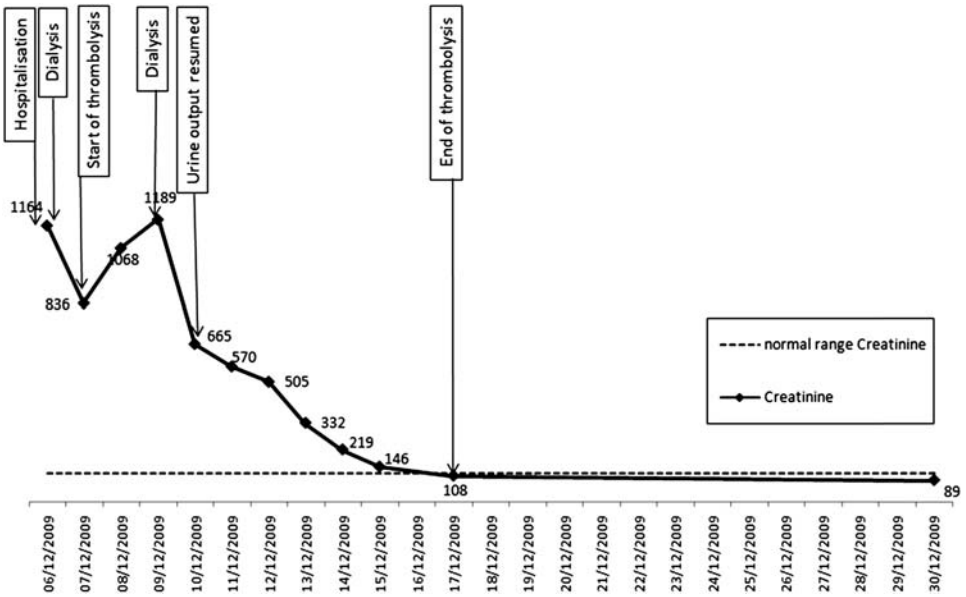

Fig. 2. Change in serum creatinine level over time.

stenosis was apparent and a 22 mm × 80 mm Sinus XL stent was deployed across this successfully. Venography then demonstrated patency of the IVC and both renal veins.

Urine output resumed on the third day of starting thrombolysis, oedema gradually settled and the creatinine level continued to improve (Figure 2). The antiphospholipid screen was negative [protein C 130 U/dL (66–122), protein S 112 U/dL (74–146), antithrombin III 134 U/dL (75–140), activated protein C ratio 2.99 [>2.1], factor V Leiden absent, lupus anticoagulant negative, anti-β2-glycoprotein I antibodies 2 U/mL (0–20), cardiolipin antibodies IgG <12 U/mL (0–12) and IgM <10 U/mL (0–10)]. The patient was anticoagulated with warfarin. Magnetic resonance venography (Figure 1B) at 3 months confirmed patency of all veins and the stented IVC segment. Eight months after initial presentation, the creatinine level was still normal at 76 μmol/L.

Discussion

Acute renal vein thrombosis is an uncommon condition that may present with loin pain and haematuria, or proteinuria. Bilateral acute, complete renal vein occlusion is very rare and results in oliguric acute renal failure [1]. Predisposing causes include nephrotic syndrome, thrombophilic states, endovascular intervention, malignancy (tumour thrombosis), major physical exertion (running a race) or trauma [2–5]. Uncommonly, no underlying cause is found as in our case [6, 7]. A short supra-renal stricture in the IVC can be contributory [2, 7, 8].

Possible treatment options beyond anticoagulation include selective or systemic thrombolysis, mechanical thrombectomy and surgery, but the optimal management for this condition is not established. Renal function recovery after complete venous occlusion may be variable [8–10]. Two decades ago, Laville *et al*. described 27

patients with renal vein thrombosis, 18 of which were bilateral; 9 patients had thrombosis involving IVC; the authors found no difference in renal function recovery between endovascular intervention versus anticoagulation alone [10]. However, these patients had underlying renal parenchymal disease including nephrotic syndrome, unlike our case. More recently, Kim *et al*. reported six patients with renal vein thrombosis (two renal allograft, three unilateral native kidneys and one bilateral native kidney). Endovascular treatment resulted in some renal function improvement in all patients but here again there was pre-existing renal disease [6]. Our patient had severe, dialysis-dependent renal failure due to bilateral renal vein occlusion, but made a complete renal recovery after 7-day treatment of intense selective venous thrombolysis, even though treatment did not start until 10 days after initial presentation. The absence of pre-existing renal disease in our patient may have aided full recovery. Although systemic thrombolysis is used by some, in our experience repeated catheter-directed treatments for such extensive thrombus burden are more effective and safe.

We conclude that aggressive endovascular intervention with selective thrombolysis can restore bilateral renal vein patency and renal function recovery, even with prolonged venous occlusion. Whilst repeated selectively delivered thrombolysis is a well-established endovascular technique, this is the first report of complete renal recovery after extensive spontaneous thrombosis. Hence, we recommend repeated selectively delivered thrombolysis for acute bilateral renal vein thrombosis.

Conflict of interest statement. None declared.

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