

Case Report

Extensive ileocaval and renal vein thrombosis mimicking acute pyelonephritis

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ABSTRACT

Renal vein thrombosis (RVT) is a rare clinical diagnosis which is the presence of thrombus in the major renal veins or its tributaries. Major causes include nephrotic syndrome, trauma, malignancy, hypercoagulable states. Presentation could be unilateral or bilateral flank pain and/or haematuria. Symptoms could be confused with nephrolithiasis. A normal ultrasonography (USG) in the initial stage could delay the diagnosis and hence propensity for dreaded complications such as loss of renal function and pulmonary embolism. Anticoagulation is the mainstay of therapy for RVT. Review of the literature reveals that thrombolytic therapy can also be used in patients with the most severe disease. Here we present a case of a young male, who initially suspected to have acute pyelonephritis, turned out to have extensive Iliocaval and unilateral RVT secondary to factor V Leiden mutation who underwent catheter directed thrombolysis and mechanical thrombectomy.

Keywords: Catheter-directed thrombolysis, Renal vein thrombosis, Flank pain

INTRODUCTION

Iliocaval thrombosis with renal vein thrombosis (RVT) is a rare clinical diagnosis which is the presence of thrombus in the iliac vessels, the inferior vena cava, and major renal veins or its tributaries. It can present acutely or go unnoticed and can result in acute kidney injury or chronic kidney disease. It complicates approximately 5-50% of cases of nephrotic syndrome and leads to significant morbidity, however clinically apparent renal vein thrombosis is rare.¹ Most patients remain asymptomatic for it, and spontaneous recovery occurs more frequently than is suggested clinically, thus amounting to high incidence of chronic RVT.² It is also associated with malignancy, primary or secondary hypercoagulable disorders such as antiphospholipid antibody syndrome and resistance to activated protein C caused by factor V Leiden, trauma, extrinsic compression, infections, or as a post-renal transplant complication. The kidney swells and

becomes engorged due to severe passive congestion which leads to degeneration of nephrons and can cause symptoms of flank pain, haematuria, and decreased urine output.³ In general, anticoagulation is the mainstay of therapy for RVT. Review of the literature reveals that thrombolytic therapy can also be used in patients with the most severe disease as well local thrombolysis with or without catheter directed mechanical thrombectomy (CDT) has also been proposed for selected cases with RVT.

CASE REPORT

A 23-year-old young male, with no prior medical history, presented with gradual onset right sided flank pain. It was of moderate intensity with progressive intermittent character of about a week's duration and associated with mild fever. There was one episode of self-limiting mild haematuria as well. Other than a recent history of a long road trip of about 6 hours, there was no contributory history. There was no history of dysuria, diarrhoeal illness,

oedema, weight loss, any trauma or prior renal stone disease. Physical examination showed tachycardia (pulse rate 110/min), blood pressure of 120/70 mmHg, and right-side flank tenderness. Laboratory parameters showed haemoglobin 11.3 g/dl, total white cell count $13.5 \times 10^9/l$, platelet count 265000/cumm, serum creatinine 1.03 mg/dl (no baseline creatinine was available), serum albumin was 3.5 g/dl (3.4–4.8g/dl). Urinalysis revealed 1+ proteinuria on dipstick, 1 to 2 RBC and 3 to 4 WBC on microscopy. Urinary albumin creatinine ratio was 49.64 mg/gm. He was negative for coronavirus disease 2019 (COVID-19). Ultrasound abdomen revealed a bulky right kidney. With the presumptive diagnosis of acute pyelonephritis, he was initiated on I.V. antibiotics. Despite I.V. antibiotics he continued to have right flank pain and was referred to us. For the significantly bulky right kidney on ultrasound, the patient appeared relatively nontoxic. Being a young male, with no previous history of urogenital issues and no obvious anatomical abnormalities conspicuous on ultrasound, a contrast enhanced computed tomography (CECT) abdomen was advised. The CECT revealed thrombosis of inferior vena cava along with involvement of right renal vein, right iliac vein, and bilateral common femoral vein and superficial veins (Figure 1). Urine culture was sterile. His blood sample was collected for the procoagulant panel, and then was initiated on with unfractionated heparin. His work-up was negative for antinuclear antibodies, anticardiolipin antibody, anti-beta-2 glycoprotein antibody, lupus anticoagulant, protein C and protein S levels were normal while antithrombin III levels were 46% and he was positive for factor V Leiden mutation. He remained symptomatic so was posted for catheter directed thrombolysis via alteplase at 0.8 mg/hour dosage for 25 hours along with unfractionated heparin at 500 units/hour. Serial haematocrit, and partial thromboplastin times were checked every 6 hours and remained below 50 seconds throughout the period and later underwent mechanical thrombectomy. He was continued on unfractionated heparin at dosage of 1500 units/hour and 24 hours later was switched over to enoxaparin for 3 days followed by rivaroxaban at dosage of 15 mg twice daily for 3 weeks with later dosage at 20 mg at bedtime. He has been symptom free. Followed up after 1 month, his DTPA scan showed a total GFR of 91.4 ml/min, with right kidney 15.7 ml/min and left kidney 75.7 ml/min.

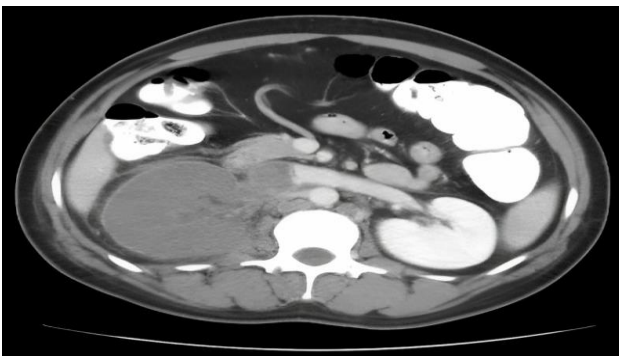


Figure 1: venous phase of CECT showing filling defect suggestive of thrombosis in right renal vein.

DISCUSSION

RVT is a rare but serious condition which could be unilateral or bilateral and should always be considered in the differentials of flank pain and haematuria. Our patient had unilateral flank pain, who initially was worked up with suspicion of pyelonephritis and later diagnosed with RVT on CT angiography. As his renal function were normal, contrast study was first choice, otherwise contrast-enhanced, magnetic resonance venography (3D-CE-MRV) could have been another imaging of choice as is as sensitive as CT and has the major benefit of avoidance of radiation and intravenous (IV) contrast. Both CT and MRV can help detect RVT as well as help in delineating another aetiology like nephrolithiasis and presence of renal cell cancer. Renal USG shows an enlarged kidney and hyper-echogenic kidney in approximately 90% of the patients in the early phase of acute RVT.⁴

Colour Doppler or contrast enhanced ultrasonography could be more sensitive. In adults, causes of RVT include nephrotic syndrome, malignancy, trauma, extrinsic compression, infections, underlying hypercoagulable state including activated protein C and protein S deficiency, antithrombin III deficiency, factor V Leiden and antiphospholipid antibody syndrome. In our case, antithrombin III levels were 46% and he was positive for factor V Leiden mutation.

As samples were drawn before starting therapy, we were able to get the appropriate results. Anticoagulation should be initiated early to prevent thrombus propagation and serious thromboembolism, as was done in our case. However, thrombectomy/thrombolysis should be considered in cases of bilateral RVT, thrombus extension into inferior vena cava or treatment failure while on anticoagulation.⁵⁻⁷

As our patient had persistent pain and had thrombosis of inferior vena cava along with involvement of right renal vein, right iliac vein, and bilateral common femoral vein and superficial veins; catheter directed thrombolysis and mechanical thrombectomy was performed, to which he had good response. He was discharged on direct-acting oral anticoagulant. Anticoagulation should be prescribed for as long as the underlying cause is not treated. If the thrombosis is idiopathic or due to an irreversible cause, lifelong anticoagulation may be indicated.

CONCLUSION

A high index of suspicion is essential for the diagnosis and early treatment of RVT, as it can mimic common renal conditions like acute pyelonephritis. Delay in therapy can result in renal infarction, extension of thrombosis to nearby venous structures, or ureteral obstruction due to compression.

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