A rare case of renal vein thrombosis secondary to *Klebsiella pneumoniae* pyelonephritis

Kelly L. Lurz, Dany N. Hanna, Brian H. McGreen, Francis J. Schanne
Department of Urology, Hahnemann University Hospital, Philadelphia, Pennsylvania, USA

**Abstract**

Renal vein thrombosis (RVT) is most often an implication of nephrotic syndrome. Pyelonephritis has been associated at a much lower rate, with the incidence of *Klebsiella pneumoniae* causation being extremely rare. In our case, a 35-year-old female patient presented with right-sided *K. pneumoniae*-positive acute pyelonephritis complicated by perinephric abscess and renal vein thrombosis. She was successfully treated with anticoagulation and extended antibiotic therapy. The possibility of RVT in patients with *K. pneumoniae*-induced pyelonephritis warrants consideration.

**Keywords:** Hypercoagulability, *Klebsiella pneumoniae*, nephrotic syndrome, pyelonephritis, renal vein thrombosis

**INTRODUCTION**

The current data describe renal vein thrombosis (RVT) to be most often associated with nephrotic syndrome, specifically minimal change disease, and systemic lupus erythematosus (SLE).[1] To the best of our knowledge, acute pyelonephritis has been reported as an etiology in a small number of case reports, with only three identifying *Klebsiella pneumoniae* as the causative pathogen.[2‑4] We report the rare finding of RVT in a patient with *K. pneumoniae* pyelonephritis.

**CASE REPORT**

A 35-year-old female patient presented to our hospital as a transfer for further workup and treatment of a right perinephric abscess with associated pyelonephritis, hydronephrosis, and a 7 mm lower pole calculus. At the previous institution, a ureteral stent and nephrostomy tube were placed, and urine and renal aspirate cultures grew pan-sensitive *K. pneumoniae*. On admission to our urology service, she was stable, afebrile, and mildly tachycardic with a leukocyte count of 16 × 10⁹/L and creatinine of 1.02 mg/dL. A MAG3 nuclear scan showed 29.8% renal function on the right. In addition to the above findings, a computerized tomography (CT) abdomen and pelvis with contrast demonstrated right RVT extending to the inferior vena cava (IVC) confluence [Figure 1]. Nephrology and infectious disease were consulted, and she was started on warfarin with a heparin bridge and ceftriaxone. The patient's history was significant for a documented *K. pneumoniae* urinary tract infection (UTI) 1 year prior, and 2 episodes of right nephrolithiasis that did not require surgical intervention with the most recent episode being 2 years ago. She took no home

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How to cite this article: Lurz KL, Hanna DN, McGreen BH, Schanne FJ. A rare case of renal vein thrombosis secondary to *Klebsiella pneumoniae* pyelonephritis. Urol Ann 2018;10:103-5.
medications and denied personal and family histories of blood dyscrasias. A complete coagulopathy workup, including markers for SLE, antiphospholipid syndrome, and Factor V Leiden mutation were negative. Her hospital course was uneventful, and after 2 weeks, the nephrostomy tube was removed with a resolution of the abscess. She was discharged on ciprofloxacin and warfarin. The patient subsequently presented for an unremarkable laser lithotripsy of right lower pole calculus 6 months later.

**DISCUSSION**

The affiliation between nephrotic syndrome and RVT is driven by disease-induced hypercoagulability. The pathogenesis of hypercoagulability is unknown but is thought to be due to the loss of antithrombin and plasminogen in the urine.[1] Studies have reported a wide range of incidence rates of RVT in patients with nephrotic syndrome, spanning from 0.5% to 60%. The diagnosis of nephrotic syndrome is made when there is >3.5 g protein in a 24-h urine collection.[1] Our patient’s 24-h urine collection contained only 1.4 g of protein, making the diagnosis of nephrotic syndrome unlikely.

Renal cell carcinoma (RCC) is another contributor to RVT. Hypercoagulability is due to tumor production of inflammatory cytokines and procoagulant substances. The mean age of RCC diagnosis is 64 years and is two-fold more common in men. The tumor would appear as a region of enhancement on CT.[8] Our patient’s demographics, negative family history, and hypo-enhancement of the right kidney on CT makes the probability of RCC highly improbable.

*Klebsiella pneumoniae* is a Gram-negative rod from the Enterobacteriaceae family, often found within oral and intestinal flora. As carrier rates are markedly higher in hospitalized patients (77% hospitalized vs. 23% nonhospitalized have positive stool cultures), infections with *K. pneumoniae* are usually hospital-acquired. Major risk factors for *K. pneumoniae* infections include indwelling foreign bodies and prior antibiotic use.[6] The organism has developed several virulence factors that help it evade the immune system. The mucoid phenotype often exists with aerobactin production, a high-affinity siderophore, which chelates iron for bacterial growth. K1 and K2 capsular serotypes lack a mannose receptor that macrophages utilize for phagocytosis.[7] A new carbepenemase-producing strain has also emerged. *K. pneumoniae* is the cause of acute pyelonephritis in 4.8% of cases.[8] The organism has been associated with renal and perinephric abscesses, emphysematous UTIs, and post-transplant UTIs.[9]

Our patient’s *K. pneumoniae*-positive pyelonephritis along with the exclusion of other causes makes the diagnosis of *K. pneumoniae*-induced renal vein thrombosis highly likely. The pathogenesis of hypercoagulability begins with immune cell recognition of pathogen-associated molecular patterns, like lipopolysaccharide. The inflammatory cascade commences increasing the release tissue factor, a pro-coagulant of the extrinsic pathway.[10]

The gold standard for imaging RVT is selective renal venography; however, this is falling out of favor for less invasive options like Doppler ultrasonography, spiral CT with contrast, and magnetic resonance imaging.[1] Treatment should include infection control with culture-directed antibiotics, dissolution of thrombus, and anticoagulation to prevent further thrombus formation.[2]

A limited number of case reports have been published describing the association between RVT and *K. pneumoniae* infection. Bassilios reported on a 45-year-old female who presented for fever and dysuria, with history significant for diet-controlled diabetes. Urine and blood cultures grew *K. pneumoniae*, and imaging revealed a right RVT extending into the right atrium. Workup was negative for blood dyscrasias, nephrotic syndrome, and renal malignancy.[2] Similarly, Harris described a 62-year-old female with sepsis from acute pyelonephritis, and RVT confirmed on CT. Urine cultures grew *K. pneumoniae*. Just like our patient, both females described above were successfully treated with an extended course of antibiotics and anticoagulation. In 1986, Eijsten reported on a 54-year-old male who presented with fever, weight loss, and a painful right
upper quadrant mass. Intravenous pyelogram, renal angiography, and abdominal ultrasound exposed a heterogeneous mass in the right kidney with vessel irregularity and a central calculus. Suspicious for malignancy, a right nephroadrenalectomy was performed with subsequent renal vein and IVC wall removal due to occluding thrombotic masses found intraoperatively. Blood cultures grew *K. pneumoniae*. Pathology revealed multiple renal and perinephric abscesses with extensive thrombosis into the renal vein and IVC wall. Eijsten's case represents the variety and severity of presentation of *K. pneumoniae*-induced renal vein thrombosis.

**CONCLUSION**

*K. pneumoniae* UTIs can have multiple implications. RVT is a rare, but serious complication. Proper imaging paired with urine or blood cultures will help guide toward this diagnosis. Treatment consists of managing the infection and anticoagulation.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

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