



Case Report

A Case of Septic Renal Vein Thrombophlebitis Secondary to Pyelonephritis Responded to Medical Treatment

Mansour Alghamdi^{1*}, Abdulrahman Attar², Ahmad Alotaibi³

¹Internal Medicine Consultant, Alhada Armed Forces Hospital, 3394 Al Hada, Taif 26792, Saudi Arabia

²Consultant Infectious Disease, Alhada Armed Forces Hospital, 3394 Al Hada, Taif 26792, Saudi Arabia

³Alhada Armed Forces Hospital, 3394 Al Hada, Taif 26792, Saudi Arabia

***Corresponding author:** Mansour Alghamdi, Internal Medicine Consultant, Alhada Armed Forces Hospital, 3394 Al Hada, Taif 26792, Saudi Arabia

Citation: Alghamdi M, Attar A, Alotaibi A (2023) A Case of Septic Renal Vein Thrombophlebitis Secondary to Pyelonephritis Responded to Medical Treatment. Infect Dis Diag Treat 7: 237. DOI: 10.29011/2577-1515.100237

Received Date: 11 October 2023; **Accepted Date:** 17 October 2023; **Published Date:** 20 October 2023

Abstract

Septic thrombophlebitis is a condition characterized by venous thrombosis, inflammation, and bacteremia or fungemia [1]. Several distinct clinical conditions have been identified, depending on the vessel involved. Septic phlebitis of the deep venous system is a rare, but life-threatening emergency that may fail to respond to even the most aggressive therapy. Renal vein septic thrombophlebitis is a very rare disease and carries a high mortality rate. Here we report a case admitted as community acquired pneumonia and found to have picture of septic emboli in the lungs and imaging showed acute pyelonephritis and renal vein thrombosis in which she responded to treatment and recovered fully with antibiotics and anticoagulation.

Keywords: Renal vein thrombosis; Septic thrombophlebitis; Septic pulmonary embolism; Pyelonephritis

Introduction

Renal vein thrombosis (RVT) is a medical condition that, while not always immediately evident, can have significant consequences for patients. This condition, characterized by the formation of a thrombus in the renal veins, can manifest in various ways, from acute symptoms to silent progression until a severe complication arises. The underlying causes and incidence rates of RVT vary, with some conditions like nephrotic syndrome being more commonly associated. Clinical manifestations can be diverse, and the diagnostic tools available range in their accuracy, cost, and invasiveness. Furthermore, the management and treatment of RVT, especially septic RVT, remains a topic of debate and research.

Case Report

57 years old female known case of type 2 diabetes mellitus and status post cholecystectomy 18 years ago. Presented with cough and right upper quadrant abdominal and right flank pain with significant weight loss-of 8 kg in the last month. Patient denied any urinary symptoms but reported fever in the last few days in which she was prescribed oral antibiotics (not known). On examination patient was ill with no fever or hypotension, she was tachypneic 22 breaths /minute and her heart rate was 110 and regular. Chest examination revealed bilateral fine crepitations more in the lower lobes. Abdominal examination showed mild right upper quadrant and right renal angle tenderness. No lymphadenopathy or lower limb edema noticed. Her investigations revealed respiratory alkalosis with mild leukocytosis (WBC 12.3 mainly neutrophils) c-reactive protein was 294 mg/dl, Sodium 133 mEq/L, Potassium 3 mEq/L, creatinine 64 umol/L, albumin 30 g/L.

Blood cultures after 3 days showed *Klebsiella pneumoniae* fully sensitive except to ampicillin. And urine culture showed the same organism.

CT KUB on admission showed dilated retro aortic left renal vein (figure 1). The next day CT chest was done and showed bilateral sub-pleural triangular opacities with consolidation and attempted cavitory process suggestive of septic emboli and no filling defect was seen in the pulmonary arteries (figure 2). After that CT abdomen with contrast requested and showed large hypodense non-enhancing area posteriorly located at the left kidney and similar lesion is noticed in the right kidney lower pole associated with filling defect at the right renal vein (partial thrombus) and left renal vein was heterogeneously opacified with dilated caliber denoting sluggish flow (figure 3). No suspicious masses or significant lymph nodes noted.

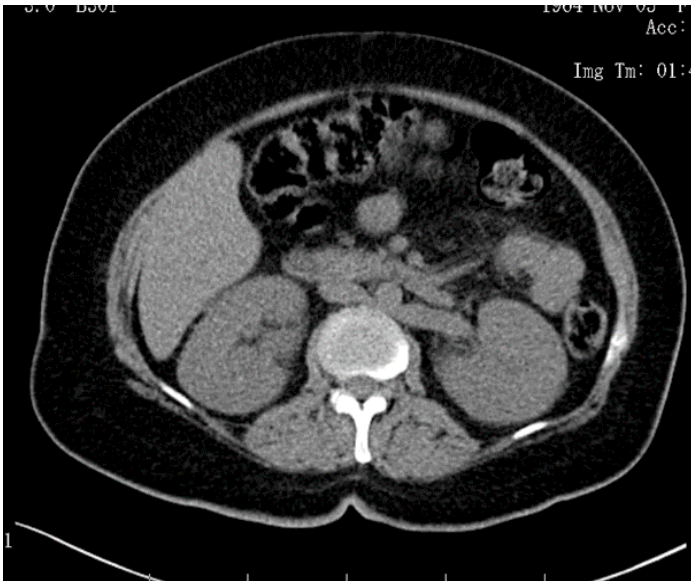


Figure 1: Dilated left renal vein with retro aortic course.

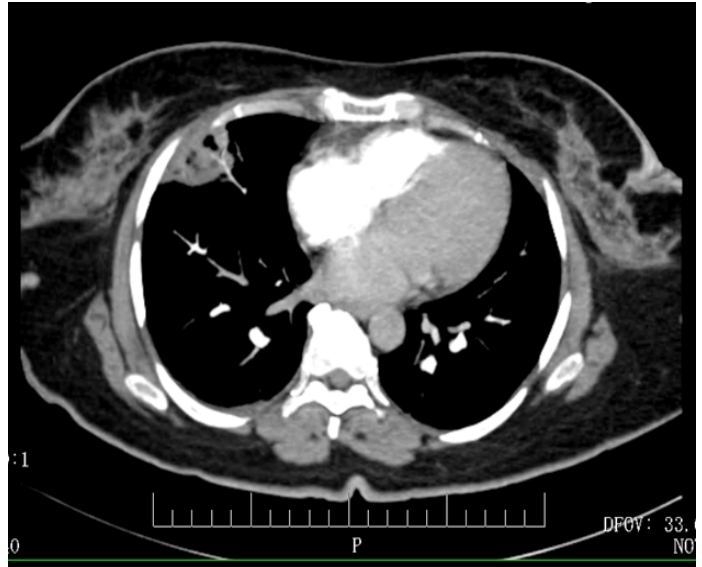


Figure 2: Triangular shaped opacity with ongoing cavitation process in the right lung.



Figure 3: Right renal vein filling defect with heterogenous opacity in the left renal vein and non-enhancing opacity of left kidney.

The patient started empirically on ceftriaxone and clarithromycin. After the CT finding patient started on apixaban therapeutic DVT dose and clarithromycin was stopped. She continued ceftriaxone 2gm iv daily as an OPAT for total of 6 weeks and apixaban for total of 3 months.

A follow up CT chest after around 6 weeks showed marked regression of the lung lesions. CT abdomen after 3 months of treatment showed normal parenchyma of both kidneys and patent renal veins with no filling defects (figure 4). The patient was reassured and discharged from the clinic to follow with her family physician.

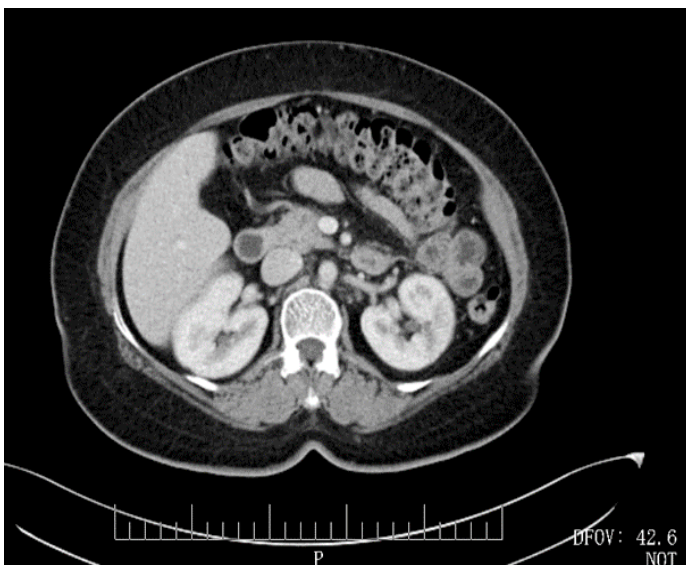


Figure 4: Follow CT after 3 months showed resolution of the right renal vein-filling defect the left renal vein caliber regressed to normal size.

Discussion

The term renal vein thrombosis (RVT) is used to describe presence of thrombus in the major renal veins or their tributaries. This condition may either present with acute symptoms or go unnoticed because of lack of symptoms until a complication like pulmonary embolism or worsening renal function, draws attention to it [1]. The exact incidence of RVT due to other causes is not known but the incidence in nephrotic syndrome and membranous nephropathy, the commonest causes of RVT, ranges from 5–62% [2].

The clinical features suggestive of RVT include nausea, vomiting, fever, flank pain, gross hematuria and palpably enlarged kidneys. Other manifestations include renal failure, thrombocytopenia, and anemia. As loin pain and hematuria predominates the problem may be mistaken for renal colic or

pyelonephritis [3]. A combination of renal vein obstruction and absence of collateral tracks for venous return is thought to be responsible for these symptoms. It is more common on the left side, presumably due to the longer length of the left renal vein in comparison to the right [4]. RVT secondary to acute pyelonephritis is rare and only few cases are described in the literature [5-10]. Despite the very low number of patients, it seems that RVT secondary to acute pyelonephritis occurred more frequently on the right side (60%), in patients with a mean age of 59.1 years and a mean white blood cell count of 18 450/mL. *E. coli* and *Klebsiella pneumoniae* accounted for 80% of cases [4]. This inflammation triggers the activation of platelets, which may accompany damage to the endothelium, resulting in fibrin deposition and thrombus formation. This process is often referred to as thrombo-inflammation. Strikingly, despite its clinical importance and despite thrombi being induced to many different pathogens, it is still unclear whether the mechanisms underlying this process are conserved and how we can best understand this process [11].

Computed tomography (CT) with intravenous (IV) contrast is regarded as the imaging modality of choice for diagnosing RVT, having nearly 100% sensitivity and specificity [1]. Renal ultrasonography (US) is safe and noninvasive and remains an accurate and highly sensitive method of diagnosing RVT. Defining characteristics of RVT in US include renal enlargement without hydronephrosis, renal vein dilation, or visualized thrombus. Color Doppler may also show low or no flow or an obstructive signal. Renal US also allows the clinician to visualize the thrombus and determine its acuity based on its echogenicity. Ultrasonograms are low-cost and easy to acquire; however, quality may vary, depending on the performing technician and on patient-specific factors. Magnetic resonance imaging (MRI) is capable of imaging and diagnosing RVT with high definition and of differentiating soft-tissue structures with high accuracy, however, it is expensive and time-consuming and can be used only for patients who has mild renal impairment and are at high risk for CT contrast nephropathy.

The mainstay of management of STP is antibiotic therapy. The empiric regimens recommended for each form are based on the sensitivity of the bacteria that most commonly cause the underlying infection, and management should be subsequently guided by blood cultures [12].

The use of anticoagulation in STP is a matter of ongoing controversy. Because STP is rare, few clinical trials have been conducted. Warfarin is well studied in cases RVT without infection, especially in patients with nephrotic syndrome, and has therefore remained the standard treatment. On the other hand, DOACs have not been well studied in RVT because the phase III studies of these medications have excluded patients with venous thrombosis in atypical locations [13].

In a retrospective study conducted at Cleveland Clinic on eight patients. Six started on rivaroxaban, and two started on apixaban. Thirty-six weeks later, follow-up imaging showed partial or complete resolution in 87% of the patients [14].

There are few cases of septic RVT that were treated conservatively, and our case is the only published case that we are aware of where NOACs were used with antibiotics for successfully treating such patients.

Conclusion

The presented case report underscores the rare manifestation of renal vein thrombosis (RVT) secondary to acute pyelonephritis, predominantly caused by *Klebsiella pneumoniae*. The clinical presentation of RVT can often be misleading, mimicking renal colic or pyelonephritis, emphasizing the importance of a high index of suspicion and timely diagnostic interventions. Computed tomography (CT) with intravenous contrast remains the gold standard for diagnosis, offering near-perfect sensitivity and specificity. While the primary treatment for septic thrombophlebitis (STP) remains antibiotic therapy, the role of anticoagulation, especially with direct oral anticoagulants (DOACs), is still a topic of debate due to limited clinical trials. However, our case demonstrates the successful use of apixaban in conjunction with antibiotics, suggesting a potential therapeutic approach for similar clinical scenarios. This case not only adds to the limited literature on septic RVT but also highlights the potential benefits of combining NOACs with antibiotics for effective management. Continuous research and more extensive clinical studies are essential to establish standardized treatment protocols for such rare clinical presentations.

References

1. Asghar M, Ahmed K, Shah SS, Siddique MK, Dasgupta P, et al., (2007) Renal vein thrombosis. *Eur J Vasc Endovasc Surg* 34:217-223.
2. Choudhary A, Majee P, Gupta R, Basu S, Das RK (2016) Adult Idiopathic Renal Vein Thrombosis Mimicking Acute Pyelonephritis. *J Clin Diagn Res* 10: PD18-PD19.
3. COSGRIFF SW, DIEFENBACH AF, VOGT W Jr. (1950) Hypercoagulability of the blood associated with ACTH and cortisone therapy. *Am J Med* 9:752-756.
4. Yildiz H, Van Nieuwenhove S, Doyen M, Yombi JC (2016) Acute pyelonephritis and renal vein thrombosis: A case report and review of the literature. *J Infect Chemother* 22:759-761.
5. Bassilios N, Tassart M, Restoux A, Bigot JM, Rondeau E, et al., (2004) Inferior vena cava thrombosis due to acute pyelonephritis. *Nephrol Dial Transpl* 19: 981-983.
6. Kumar S, Singh SK, Mavuduru RS, Acharya NC, Agarwal MM, et al., (2009) Acute pyelonephritis with renal vein and inferior vena cava thrombosis in a case of hyperhomocysteinemia. *Int Urol Nephrol* 41:185-188.
7. Eijsten A, Leisinger HJ, Jucker A (1986) Unilateral pyonephrosis with septic thrombosis of the renal vein and vena cava. *Urol Int* 41:77-79.
8. Mamzer-Bruneel MF, Anglicheau D, Correas JM, Skhiri H, Jacobs F, et al., (1997) Renal venous thrombosis: a forgotten complication of acute pyelonephritis. *Presse Med* 26:1334-1336.
9. Novelli L, Raynaud A, Pellerin O, Carreres T, Sapoval M (2007) Percutaneous manual aspiration embolectomy of renal vein thrombosis due to acute pyelonephritis. *Cardiovasc Intervent Radiol* 30:1075-1078.
10. Harris Leslie A, Van Every Marvin J, Fundell Larry J (2012) Acute bilateral renal vein thrombosis secondary to sepsis from pyelonephritis. *Int Braz J Urol* 38:132-134.
11. Beristain-Covarrubias N, Perez-Toledo M, Thomas MR, Henderson IR, Watson SP, et al., (2019) Understanding Infection-Induced Thrombosis: Lessons Learned From Animal Models. *Front Immunol* 10:2569.
12. Valerio L, Riva N (2020) Head, Neck, and Abdominopelvic Septic Thrombophlebitis: Current Evidence and Challenges in Diagnosis and Treatment. *Hamostaseologie* 40:301-310.
13. van Es N, Coppens M, Schulman S, Middeldorp S, Büller HR (2014) Direct oral anticoagulants compared with vitamin K antagonists for acute venous thromboembolism: evidence from phase 3 trials. *Blood* 124:1968-1975.
14. Matta A, Elenizi K, AlHarthi R, Moussallem N, Elhajjaji N, et al., (2019) A case of isolated unilateral right renal vein thrombosis associated with bilateral pulmonary embolism treated with rivaroxaban a direct-acting oral anticoagulant. *Am J Case Rep* 20:1152-1154.