International Journal of Research in Medical Sciences
Harshini RS et al. Int J Res Med Sci. 2026 Nov;14(11):5386-5390
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20263687

Case Report

Renal vein thrombosis with hydronephrosis in seropositive rheumatoid arthritis with hepatitis B reactivity: a diagnostic challenge suggestive of renal tuberculosis

Rathod Sai Harshini*, Yatri Patel, Pankhuri Saxena, Abhinash Jha, Minakshi Dhar

All India Institute of Medical Sciences, Rishikesh, India

Received: 15 September 2026
Revised: 28 September 2026
Accepted: 29 September 2026

***Correspondence:**
Dr. Rathod Sai Harshini,
E-mail: rathodharshini2001@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Rheumatoid arthritis (RA) is associated with an increased susceptibility to infection, while immunosuppressive treatment may further complicate the recognition and management of tuberculosis (TB). Renal TB is an indolent form of genitourinary TB that may present with non-specific constitutional and urinary manifestations and can cause obstructive uropathy. Renal vein thrombosis (RVT) is an uncommon vascular complication that may occur in the setting of systemic inflammation or infection. We report a 65-year-old man with recently diagnosed seropositive RA and hepatitis B surface antigen positivity who presented with six months of intermittent low-grade fever, constitutional symptoms and symmetrical inflammatory polyarthritis, followed by left flank pain, intermittent haematuria and bilateral lower-limb swelling. Laboratory evaluation showed anaemia, markedly elevated inflammatory markers and strongly positive rheumatoid factor and anti-CCP antibodies. HBsAg was positive, while HBV DNA was undetectable. Contrast-enhanced CT demonstrated a bulky left kidney with moderate hydronephrosis, pelvicalyceal dilatation, PUJ obstruction and left RVT with extensive collateral formation. Urine culture was sterile and renal function was preserved. Subsequent urine CBNAAT detected *Mycobacterium tuberculosis* and showed rifampicin susceptibility, establishing renal TB in the appropriate clinical and radiological context. The patient was treated with anticoagulation, RA-directed therapy, supportive measures and subsequently drug-susceptible antitubercular therapy. This case emphasizes the diagnostic challenge posed by overlapping inflammatory, infective and thrombotic manifestations and highlights the importance of considering renal TB in patients with hydronephrosis, urinary symptoms and unexplained RVT, particularly when routine bacterial cultures are sterile.

Keywords: Renal tuberculosis, Rheumatoid arthritis, Renal vein thrombosis, Hydronephrosis, Hepatitis B, Extrapulmonary tuberculosis, CBNAAT

INTRODUCTION

Genitourinary tuberculosis is an important form of extrapulmonary TB and commonly involves the kidneys. Renal TB often has an insidious course, with non-specific constitutional and urinary symptoms that can delay diagnosis. Progressive involvement may produce calyceal and ureteric abnormalities, obstruction and irreversible renal damage.[1-4] Recent literature continues to emphasize the protean clinical and radiological manifestations of genitourinary TB and the consequences of delayed recognition.[3,8]

Patients with RA may have an increased risk of TB, and the risk is further influenced by disease activity, age, previous TB and immunomodulatory treatment.[5,6] In a patient with a history of pulmonary TB, the possibility of reactivation or extrapulmonary disease therefore requires particular attention. Concurrent HBsAg positivity adds

another management consideration when immunosuppressive and antitubercular therapies are required.

RVT is uncommon and is classically associated with nephrotic syndrome, malignancy and other hypercoagulable states. Its occurrence alongside renal TB is rare, although TB-associated venous thromboembolism has been described.[7] The coexistence of seropositive RA, HBsAg positivity, hydronephrosis and RVT can therefore create substantial diagnostic overlap. We describe this case to highlight the importance of integrating clinical, radiological and microbiological findings rather than attributing all manifestations to RA or common urinary infection.

CASE REPORT

A 65-year-old man, a chronic smoker with an approximately 50 pack-year history, presented with low-grade intermittent fever of six months duration, generalised weakness, loss of appetite and progressively worsening symmetrical polyarthritis. The joint involvement affected the shoulders, elbows, knees, ankles, wrists and small joints of the hands, with early-morning stiffness lasting more than one hour and improving as the day progressed.

During the preceding month, he developed dull, aching, non-radiating left flank pain associated with intermittent haematuria and bilateral painless lower-limb swelling. He had a past history of treated pulmonary TB. There was no history of diabetes mellitus, hypertension, chronic kidney disease or malignancy.

On examination, he was pale and had left renal-angle tenderness. Musculoskeletal examination revealed active symmetrical inflammatory polyarthritis with restricted movement of the bilateral large joints and the metacarpophalangeal and the proximal interphalangeal joints.

Investigations

Investigations revealed anaemia with markedly raised inflammatory markers. The patient was HBsAg-positive with undetectable HBV DNA. Rheumatoid factor and anti-CCP antibody were strongly positive. Renal and liver function tests were within normal limits, and urine culture showed no growth.

Imaging

Contrast-enhanced CT of the thorax and abdomen demonstrated a bulky left kidney with moderate hydronephrosis, pelvicalyceal dilatation, PUJ obstruction and mild perinephric fat stranding. Left RVT with extensive collateral venous formation was also demonstrated (Figure 3).

Table 1: Investigations.

Investigations	Results
Haemoglobin	10.4 g/dl
Total leucocyte count	10 × 10³ cells/µl
Platelet count	394 × 10³/µl
ESR	110 mm/hr
C-reactive protein	54.9 mg/l
Rheumatoid factor	Strongly positive (>300 IU/ml)
Anti-CCP antibody	Positive (>300 U/ml)
HBsAg	Positive
HBV DNA	Undetectable
Blood urea / serum creatinine	15 / 0.53 mg/dl
Urine routine / microscopy	Within normal limits
Urine culture	Sterile (no growth)
Urine CBNAAT (MTB)	Detected; rifampicin susceptible
Cytology for malignancy	Negative for malignant cells



Figure 1: Dorsal view of both hands showing bilateral symmetrical swelling of the metacarpophalangeal and proximal interphalangeal joints, consistent with rheumatoid arthritis.



Figure 2: Bilateral feet showing diffuse xerosis, fine scaling, pigmentary changes, and dystrophic thickening of the toenails.

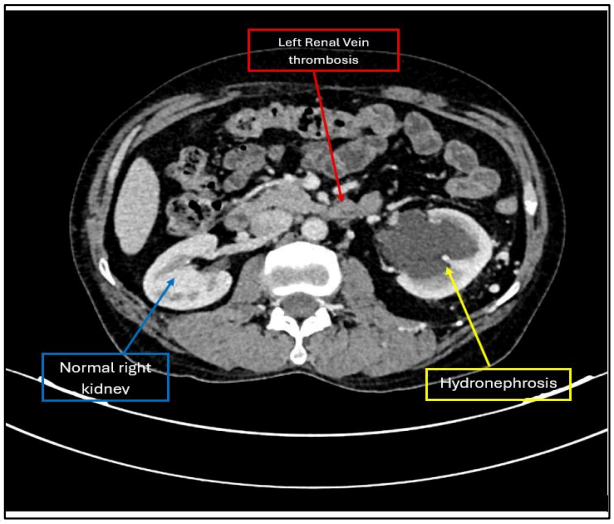


Figure 3: Contrast-enhanced CT abdomen showing a bulky left kidney with moderate hydronephrosis and pelvicalyceal dilatation, associated with left RVT and collateral venous formation.

Functional imaging

A 99mTc-DTPA renal dynamic scan was performed to assess differential renal function and drainage which revealed 20% of split function of left kidney

Microbiology

Because of persistent left flank symptoms and the radiological abnormalities, urine was sent for AFB testing and CBNAAT. Urine CBNAAT detected *Mycobacterium tuberculosis* with no rifampicin resistance.

Cytology was negative for malignant cells. In the context of the previous pulmonary TB, chronic constitutional symptoms, sterile routine bacterial culture, hydronephrosis and PUJ obstruction, the positive molecular test supported the diagnosis of renal TB.

WHO guidance supports the use of Xpert MTB/RIF on urine as an initial diagnostic test in patients with suspected genitourinary TB.[9]

Table 2: Differential diagnoses.

Differential	Features in favour	Features against / resolution
Renal TB	Previous pulmonary TB, chronic constitutional symptoms, haematuria, hydronephrosis with PUJ obstruction and sterile bacterial urine culture; subsequently supported by positive urine CBNAAT.	Initially not established; diagnosis supported when urine CBNAAT detected *M. tuberculosis.*
Acute/chronic pyelonephritis	Fever, flank pain, renal-angle tenderness and raised inflammatory markers.	Repeatedly sterile bacterial urine culture and the subsequent positive TB molecular test made pyogenic infection less likely.
Obstructive uropathy (calculus/stricture)	Hydronephrosis, pelvicalyceal dilatation and PUJ obstruction.	No calculus identified on CT; obstruction occurred in the setting of confirmed renal TB.
Urothelial/renal malignancy	Older age, chronic smoking, haematuria, bulky kidney and unilateral obstruction.	Imaging and cytology did not demonstrate malignancy.
Nephrotic syndrome-related RVT	RVT with collateral formation.	Normal renal function and absence of significant proteinuria argued against nephrotic syndrome as the explanation.
Rheumatoid renal involvement/vasculitis	Seropositive RA with high disease activity.	The imaging and microbiological findings provided a more specific infective/obstructive explanation for the renal abnormalities.

Treatment and outcome

The patient was initially managed with intravenous antibiotics, anticoagulation with apixaban, DMARDs (methotrexate and hydroxychloroquine), folic acid, analgesics and supportive measures. Following confirmation of renal TB, and with undetectable HBV DNA, he was started on a six-month course of drug-susceptible antitubercular therapy with monitoring for hepatotoxicity and HBV-related concerns. He was discharged in a haemodynamically stable condition. At follow-up, his rheumatological symptoms improved, together with clinical and biochemical improvement in his renal/urinary status.

DISCUSSION

This case illustrates the diagnostic complexity created by the simultaneous presence of inflammatory arthritis, previous TB, obstructive renal disease and venous

thrombosis. The initial presentation was dominated by prolonged fever and symmetrical inflammatory polyarthritis, with strongly positive rheumatoid factor and anti-CCP antibodies supporting the diagnosis of RA. However, the subsequent development of flank pain, haematuria and lower-limb swelling required a separate evaluation of the renal and vascular abnormalities.

Renal TB is characteristically indolent and may present with non-specific constitutional or urinary symptoms. Imaging can demonstrate a spectrum ranging from subtle calyceal abnormalities to obstruction, strictures and advanced destructive disease.[2-4,8] In this patient, the combination of a previous history of pulmonary TB, chronic constitutional symptoms, unilateral hydronephrosis, PUJ obstruction and sterile bacterial urine culture raised suspicion for genitourinary TB. Importantly, the absence of growth on routine bacterial culture did not establish TB by itself; the diagnosis was strengthened by urine CBNAAT detecting *M. tuberculosis*. WHO guidance recognizes urine Xpert MTB/RIF as an initial diagnostic option in adults with signs and symptoms suggestive of genitourinary TB.[9]

The renal vascular finding was particularly unusual. RVT is more commonly associated with nephrotic syndrome, malignancy and systemic hypercoagulable states. In this patient, there was no significant proteinuria, renal function was preserved and cytology did not reveal malignancy. The CT demonstrated extensive collateral vessels, suggesting an established thrombotic process. TB has been associated with a prothrombotic state, and a published case report described renal vein and inferior vena cava thrombosis in active TB.[7] The proposed mechanisms include systemic inflammation, endothelial injury, activation of coagulation pathways and venous stasis. However, a causal relationship between renal TB and the RVT cannot be proven from this single case, and the thrombosis may have been multifactorial.

The coexistence of RA further broadened the differential diagnosis. RA itself is associated with an increased risk of infection, and TB risk has been reported to be higher in patients with RA than in comparable populations.[5,6] Older age, previous TB and immunomodulatory treatment may further influence this risk. In the present case, it was therefore important not to attribute the fever and systemic inflammatory response solely to active RA.

HBsAg positivity with undetectable HBV DNA added another management consideration. The patient required both immunomodulatory therapy for RA and antitubercular treatment, making clinical and biochemical monitoring particularly important. The case therefore required coordination between rheumatology, infectious diseases, urology and medical teams. The key diagnostic lesson is that a positive rheumatological profile does not exclude a concurrent infection, and renal TB should remain in the differential when obstruction and urinary symptoms occur in a patient with previous TB.

The principal limitation of this report is that the diagnosis of renal TB was established by urine CBNAAT in conjunction with the clinical and radiological picture rather than by renal tissue histopathology. In addition, the precise mechanism responsible for the RVT cannot be established. Nevertheless, the temporal evolution, imaging findings and microbiological confirmation provide a clinically coherent explanation for the renal presentation.

CONCLUSION

Patients with inflammatory polyarthritis who develop flank pain, haematuria, hydronephrosis and RVT require evaluation beyond the underlying rheumatological diagnosis. In a patient with previous TB, renal TB should be considered even when routine bacterial urine cultures are sterile. Urine molecular testing can provide important microbiological evidence, while imaging helps define the extent of obstructive and vascular complications. Coexisting HBsAg positivity and the need for immunomodulatory treatment further reinforce the value of coordinated multidisciplinary care.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Cantini F, Nannini C, Niccoli L, Petrone L, Ippolito G, Goletti D. Risk of tuberculosis reactivation in patients with rheumatoid arthritis, ankylosing spondylitis, and psoriatic arthritis receiving non-anti-TNF-targeted biologics. Mediators Inflamm. 2017;2017:8909834.
2. Figueiredo AA, Lucon AM. Urogenital tuberculosis: update and review of 8961 cases from the world literature. Rev Urol. 2008;10(3):207-17.
3. Muneer A, Macrae B, Krishnamoorthy S, Zumla A. Urogenital tuberculosis-epidemiology, pathogenesis and clinical features. Nat Rev Urol. 2019;16(10):573-98.
4. Golden MP, Vikram HR. Extrapulmonary tuberculosis: an overview. Am Fam Physician. 2005;72(9):1761-8.
5. Chung TT, Ko HJ, Lau CS, Chung HY. A retrospective study on the risk of tuberculosis in patients with rheumatoid arthritis. Rheumatol Int. 2020;40(6):983-90.
6. Carmona L, Hernández-García C, Vadillo C, Pato E, Balsa A, González-Alvaro I, et al. Increased risk of tuberculosis in patients with rheumatoid arthritis. J Rheumatol. 2003;30(7):1436-9.
7. Anweri AV, Kaniyoor Nagri S, Karanth S. Inferior vena cava and renal vein thrombosis: a rare cause of acute kidney injury in tuberculosis. BMJ Case Rep. 2023;16(2):e254084.
8. Rocco R, Villar BBDLF, Ferreira LDC, Vilte RMDLCV, McBenedict B, Silva NCZ, et al. Renal

tuberculosis with genitourinary sequelae: a case report. Rev Inst Med Trop Sao Paulo. 2025;67:e22.
9. World Health Organization. WHO consolidated guidelines on tuberculosis. Module 3: diagnosis-rapid diagnostics for tuberculosis detection. 3rd ed. Geneva: World Health Organization; 2024.

Cite this article as: Harshini RS, Patel Y, Saxena P, Jha A, Dhar M. Renal vein thrombosis with hydronephrosis in seropositive rheumatoid arthritis with hepatitis B reactivity: a diagnostic challenge suggestive of renal tuberculosis. Int J Res Med Sci 2026;14:5386-90.