Rare Case of Bilateral Renal Vein Thrombosis: A Case Report

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Abstract
Bilateral renal vein thrombosis (RVT) is a rare clinical condition. A case of a patient initially presenting with fever with a diagnosis of Plasmodium falciparum malaria was further investigated and it was found to have bilateral RVT. The patient was successfully treated with anti-coagulants and immunosuppressant.

Key words: Bilateral, Membranous glomerulonephritis, Renal vein thrombosis

INTRODUCTION
The first time, renal vein thrombosis (RVT) was described by Rayer[1] on the basis of autopsy findings in seven patients, and before 1956, the diagnosis of this disorder was made at postmortem.[2]

RVT is the formation of a clot in the vein that drains blood from the kidneys, ultimately leading to a reduction in the drainage of one or both kidneys and the possible migration of the clot to other parts of the body, leading to complications like pulmonary embolism.[3]

It is a rare condition with significant morbidity consequences.[4] It is one of the more frequent thromboembolic events and may result in kidney infarction, hypertension, chronic infection, and renal failure. Hence, early diagnosis and appropriate treatment for such a condition are crucial.

We report a case of bilateral RVT initially presenting with fever with a diagnosis of Plasmodium falciparum malaria.

CASE REPORT
A 50 years/male, smoker had a history of fever in August 2016. He was diagnosed with P. falciparum malaria and treated with antimalarial. Despite antimalarial treatment, fever was persistent. Subsequently, he was found to have right pleural effusion on X-ray chest. Considering pulmonary Koch’s, he was started on antitubercular (INH+RCIN+ETB+PZI).

On September 24, 2016, he was admitted with us with a history of abdominal pain since 4–5 days. On ultrasound sonography test (USG) abdomen, it was found to have bilateral RVT.

With a diagnosis of bilateral RVT, the patient was further investigated. Sr creatinine was 1.4 mg/dl, Sr. cholesterol was 268 mg/dl, tumor markers, i.e., serum carcinoembryonic antigen, serum prostate-specific antigen, alpha-fetoprotein, Sr. CA 19.9 were found to be normal. Factor V was negative, Sr. homocysteine was normal indicating no hypercoagulable disorder. Serum uric acid was on higher side, i.e., 9.1 mg/dl.

Erythrocyte sedimentation rate, antinuclear antibody, and anti-neutrophil cytoplasmic antibodies, Sr. C3 level, were normal ruling out vasculitis. Viral markers (HIV, hepatitis B virus surface antigen, and hepatitis C virus) were negative indicating no viral cause for bilateral RVT.

Sr. proteins showed A: G reversal. Sr. protein electrophoresis showed M band indicating multiple myeloma. Bone marrow
biopsy, immune electrophoresis, and β2 microglobulin were normal.

Computed tomography (CT) pulmonary angiogram confirmed bilateral extensive pulmonary thrombosis, CT renal angiogram confirmed the diagnosis of bilateral RVT. After CT angiography, hemodialysis was done through the right internal jugular vein dialysis catheter.

After confirmation of RVT, the patient was started anticoagulant, i.e., IV heparin and oral warfarin with monitoring of prothrombin time international normalized ratio and a partial thromboplastin time. The patient was discharged with oral anticoagulant with follow-up advice.

After 2 months, the USG abdomen was done which showed no RVT and Sr. creatinine was 1 mg/dl. An oral anticoagulant was stopped. After a week, the patient was admitted for renal biopsy, which showed signs of membranous glomerulo-nephritis.

Anti-phospholipase A2 receptor antibody (IgG) by ELISA was done, which was negative. This ruled out primary membranous glomerulonephritis.

The cause of bilateral RVT was found to be nephrotic syndrome as other causes such as vasculitis, hypercoagulable disorders, malignancy, and viral infections were ruled out on investigations as mentioned above.

DISCUSSION

RVT is a rare event but is prevalent in patients with nephrotic syndrome. Bilateral RVT is even rarer. The literature is relatively sparse in terms of the management of RVT due to its rarity and consists of a few case reports and case series.

Bilateral RVT may be found in conjunction/association with other diseases such as malignancy, trauma, infections, procoagulant states, or with use of oral contraceptives.

For diagnosis of RVT in the presence of nephrotic syndrome, both histopathology and venous angiography are needed.

CONCLUSION

We presented here a rare case of bilateral vein thrombosis in a patient who initially presented with a history of fever. On ruling out various causes of RVT, it was found to be due to underlying nephrotic syndrome. The patient was successfully treated with anticoagulants, steroids, and cyclophosphamide.

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