Renal Vein Thrombosis Associated with Nephrotic Syndrome and Factor V Leiden

Dear Editor,

Renal vein thrombosis (RVT) is a well-known complication of nephrotic syndrome. [1] Here, we present a case with acute RVT diagnosed as membranous nephropathy with genetic variants that may have predisposed him to develop thrombosis.

A 20-year-old male presented with a complaint of left flank pain for a month. He denied trauma, smoking, alcohol consumption, illegal drug use, or vigorous sports. Examination relealed left costovertebral angle tenderness. Laboratory results showed increased creatinine (1.23 mg/dL) and creatine kinase (1256 U/L), proteinuria (6.89 g/day), and hypoalbuminemia (3.2 g/dL).

Abdominal computed tomography (CT) revealed increased kidney sizes, bilateral pelvicalyceal dilatation, and heterogeneous density changes consistent with fluid. CT angiography abdomen showed dilatation of the left renal vein and filling defect extending to vena cava inferior, compatible with RVT [Figure 1].

Unfractionated heparin followed by warfarin was begun and dose adjusted subsequently. A renal biopsy was performed from the right kidney, and the histological pattern was compatible with membranous nephropathy. Genetic analysis revealed heterozygosity for the pathogenic factor V Leiden (FVL) variant and homozygosity for a variant of uncertain significance of the MTHFR gene (1298A>C, p.Glu429Ala).

The risk of thrombosis in nephrotic syndrome is closely related to the severity of disease, especially low levels of albumin (<2.5 g/dL).^[2] Our patient had moderate



Figure 1: Dilatation of the left renal vein and filling defect extending to vena cava inferior compatible with renal vein thrombosis

hypoalbuminemia (albumin level was 3.2 g/dL). Therefore, we looked for and found other acquired and genetic causes of thrombophilia.

While some studies have shown that the FVL variant increase the risk of thromboembolic complications in patients with nephrotic syndrome (NS), others have concluded the opposite.^[3,4]

One of the genetic factors that have been extensively studied over the past decade is a polymorphism in the gene encoding *MTHFR*. Our patient was homozygous for a variant of uncertain significance of the *MTHFR* gene (1298A>C, p.Glu429Ala) and his homocysteine level was slightly increased. The Multiple Environmental and Genetic Assessment (MEGA) of risk factors for venous thrombosis study showed that the *MTHFR* C677T variant was not associated with the risk of venous thrombosis.^[5] In conclusion, genetic causes of thrombophilia should be investigated in addition to nephrotic syndrome in patients diagnosed with acute RVT

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms.

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Conflicts of interest

There are no conflicts of interest.

Cundullah Torun, Mirac Vural Keskinler, Banu Mesci

Department of Internal Medicine, Goztepe Training and Research Hospital, Istanbul Medeniyet University, Kadikoy/Istanbul, Turkey

Address for correspondence:

Dr. Cundullah Torun,

Goztepe Training and Research Hospital, Province of Istanbul, District of Kadıköy, Neighbourhood of Eğitim, 34722, Türkiye. E-mail: cundullaht@qmail.com

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