CLINICAL IMAGE

Nephrotic syndrome and duplication of the inferior vena cava: a series of unfortunate events

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A 41-year-old man was admitted for a diagnostic workup for nephrotic syndrome. He complained of lower extremity and face edema, as well as dull bilateral flank pain, which lasted a few months. On admission, the physical examination was unremarkable, except for the above abnormalities. Laboratory test results revealed proteinuria of up to 7 g/d, with a serum albumin concentration of 19.8 g/l. An ultrasound showed the enlargement of both kidneys. Renal biopsy revealed membranous nephropathy with a positive titer of anti-phospholipase A2 receptor antibodies. After the biopsy, the patient reported painless gross hematuria without visible blood clots, as well as a marked decrease in urinary volume.

Ultrasound examination showed kidney enlargement with prominent thickening of the pelvicalyceal walls and without any signs of hematoma. On Doppler ultrasound, a thrombus in the inferior vena cava (IVC) and both renal veins was found. Computed tomography confirmed thrombi in the renal veins and IVC, as well as revealed the duplication of the subrenal segment of the IVC (FIGURE 1). The renal pelves were wide with thickened walls, which is a typical finding in renal vein thrombosis (RVT).[1] The proximal parts of the ureters also showed wall thickening and a narrow lumen with a very thin jet of the urinary flow.

The patient received enoxaparin (1 mg/kg every 12 hours), warfarin (international normalized ratio, 2–3), alkylating agent, and steroids. Six days later, diuresis increased to 2000 ml/24 h. Thrombosis resolved at 3 months, as confirmed by control Doppler ultrasound.

RVT in the general population is rare. Its acute episode is mainly related to trauma (including kidney biopsy) and manifests as flank pain, hematuria, increased kidney size, and—if bilateral—acute kidney injury.[2] Noteworthy, the incidence of RVT reaches up to 60% in patients with nephrotic syndrome, and the risk seems to be the highest in those with membranous nephropathy.[3] Moreover, thrombosis is usually chronic and asymptomatic. The high incidence of RVT in these patients is attributed to hypercoagulable state, which is one of the hallmarks of this condition, as well as to hemoconcentration in the postglomerular circulation due to the loss of fluid across the glomerulus, which is often aggravated by forced diuresis.[4]

Duplication of the IVC is usually an accidental finding. The reported incidence of this congenital malformation is up to 3% in the general population. The anomaly was reported to be a potential risk factor for deep vein thrombosis of the lower extermities.[5] Venous stasis due to the malformation is thought to be a triggering factor.

In our patient, the coincidence of several conditions and interventions such as duplication of

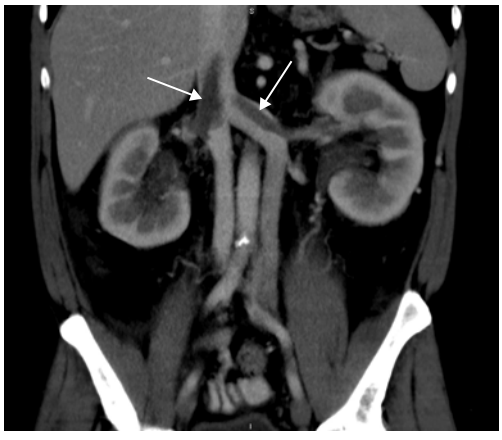

FIGURE 1 Computed tomography scan, coronal plane. The arrows show branches of the duplicated inferior vena cava with bilateral vein thrombosis. Thrombosis within the duplicated inferior vena cava extends to both renal veins.

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the IVC, membranous nephropathy with severe nephrotic syndrome, kidney trauma (kidney biopsy), and forced diuresis aimed at relieving massive edema led to a serious complication, namely, thrombosis of the IVC and bilateral renal veins. To our knowledge, this is the first report of a patient with bilateral RVT due to the coexistence of membranous nephropathy with severe nephrotic syndrome and duplication of the IVC.

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