A Case of Renal Vein Thrombosis with Posterior Nut Cracker Syndrome

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Dear Sir,

Compression of the left renal vein between the aorta and the superior mesenteric artery is known as 'nut cracker phenomenon' [1]. The compression is thought to be caused by a decrease in the angle of the superior mesenteric artery from the aorta or posterior renal ptosis with stretching of the left renal vein over the aorta [1]. A variant of this phenomenon, 'posterior nut cracker syndrome' (PNS), refers to compression of the retro-aortic renal vein between the aorta and spine [2]. It is associated with hematuria, abdominal pain and varicocele formation [1,2].

We present a case of renal vein thrombosis with PNS. A 36-year-old man was admitted to the emergency room with acute abdominal pain. Physical examination revealed left flank tenderness. Urinalysis demonstrated +++ protein and on microscopic evaluation 7-8 erythrocytes, 3-4 hyaline casts were observed. Daily urinary protein excretion was 3.5 g. Abdominal Doppler ultrasonography (USG) demonstrated compression of the left renal vein between the aorta and spine (PNS), an increase in left kidney size, minimal left renal subcapsular edema, a thrombus occluding the left renal vein extending towards the lumen of the vena cava inferior (fig. 1, 2). Abdominal CT confirmed these findings (fig. 3). Inferior ca-vography was normal. Selective venography was not performed because of the risk of dislodging the thrombus. Prothrombin time, partial thromboplastin time, α_1 -antitrypsin, anticardiolipin antibody, antithrombin III, protein and protein C levels were also normal. Renal biopsy showed features of membranous glomerulonephritis (MGN) (fig. 4). The patient was anticoagulated with heparin followed by warfarin. Abdominal Doppler USG, repeated 25 days later, showed collaterals around the left kidney and resolution of the thrombus. After 30 days of prednisolone (1 mg/kg/day), azathioprine (150 mg/day) and dipyridamole (225 mg/day) therapy, the amount of protein in 24-hour urine was 1 g.

The complaints of patients in the reported cases of nut cracker syndrome were microscopic or gross hematuria with or without abdominal or flank pain [1-5]. It is reported that hematuria from the left renal vein orifice, in the absence of any other detectable pathology, should raise the suspicion of nut cracker syndrome. The diagnosis of this anatomic variation is established by demonstrating compression of the renal vein during selective renal venography [1]. A significant

pressure gradient exists over the obstruction. Doppler USG, CT, MRI are non-invasive diagnostic alternatives to angiography[3,5].

In this case, PNS was thought to be one of the probable causes of renal vein thrombosis (RVT). Since there is no reported case of nut cracker syndrome complicated with RVT, other etiologies for RVT were also searched. Laboratory findings were consistent with nephrotic syndrome, and renal biopsy examination demonstrated MGN. It is known that MGN accounts for approximately 30% of cases of nephrotic syndrome

and 5-62% of them are associated with RVT [6, 7]. Various factors, including urinary loss of clotting inhibitors, zymogens, plasminogen, increased synthesis of fibrinogen, increased platelet aggregation, decreased renal blood flow, lead to hypercoagulability causing thromboembolic phenomena [6-11]. Recent studies demonstrated the presence of circulating immune complexes in patients with both MGN and RVT but not with MGN alone [7]. These immune complexes are believed to be the triggering factors in the coagulation process.

In our case, MGN seems to be the cause of RVT, but compression of renal vein due to the anatomic variation causing obstruction in the blood flow may have facilitated RVT in the hypercoagulability basis of MGN.

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0028-2766/95/0694-0476
\$8.00/0

Fig. 1. Doppler USG showing the proximal portion of the left renal vein, the filling defect in the left renal vein caused by the thrombus and drainage of the left renal vein to vena cava inferior between the aorta and lumbar vertebra.

Fig. 2. Abdominal USG of the patient showing a pedunculated thrombus, 1-1.5 cm in diameter, in the vena cava inferior.

Fig. 3. CT scan of the patient showing retroaortic left renal vein and a filling defect caused by the thrombus.

Fig. 4. Renal biopsy of the patient showing diffuse thickening of the capillary wall without increase in number of cells.

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