Chronic Post-thrombotic Obstruction of the Inferior Vena Cava: Both Renal Veins being the Cause of Painless Gross Hematuria from Pelviureteral Mucosal Varices in Normal Functioning Kidney

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A 31-year-old man presented with painless gross hematuria. His serum-creatinine level was within the normal range. Abdominopelvic CT showed an infrahepatic calcified fibrous cord, which was suggestive of inferior vena cava (IVC) remnant. Extensive venous collateralization around both kidneys and venous drainage of the extremities via the inferior epigastric and internal thoracic veins were shown. We report a case of chronic post-thrombotic obstruction of the IVC, involving both renal veins, which was believed to be the cause of painless gross hematuria from mucosal varices of the pelviureteral system in normal functioning kidney.

Key words: Hematuria, IVC, renal vein, thrombosis

INTRODUCTION

There are many cases of unexplained or essential renal hematuria, for which the definite cause of bleeding cannot be determined by routine urological and clinical examinations.

Thrombosis of the inferior vena cava (IVC) involving both renal veins is a rare condition that can result from various underlying disease entities, including deep vein thrombosis, major trauma, hypercoagulability disorder, obstruction due to tumor invasion, and idiopathic cause.

Chronic obstructive of the IVC involving both renal veins may not be recognized because of the lack of signs and symptoms. If painless gross hematuria was developed, the congestion of the tortuous engorged veins in the pelviureteral mucosa as a kind of collateralization should be considered as one of the possible causes.

CASE REPORT

A 31-year-old man presented with painless gross hematuria. Physical examination showed dilatation of abdominal superficial vessels and mild, painful swelling of the left lower leg. He had a history of operation for femoral bone fracture and bed rest for 6 months 4 years previously. Serum creatinine level was within the normal range.

Excretory urography demonstrated an irregular filling defect on the renal pelvis of the left kidney, which was thought to be blood clots. Abdominopelvic CT and CT-angiogram showed infrahepatic calcified fibrous cord, which was suggestive of IVC remnant, and IVC obliteration continued to the common femoral veins. Both renal veins were also completely obliteration. Extensive venous collateralizations were shown around both kidneys (Fig. 1). CT-venogram showed that venous drainages of the extremities via the inferior epigastric and internal thoracic veins entered to the subclavian vein, azygous vein, or suprahepatic vena cava (Fig. 2).
Cystoscopy showed no bladder lesions, but blood-tinged urine was noted from the left ureteral orifice. Diagnostic ureteroscopic examination showed no other ureteral lesions to account for the hematuria except for the tortuous engorgement of the veins of the ureteral mucosa.

DISCUSSION

Obstruction of the IVC is commonly developed by migration of clot cephalad from the iliac veins, although thrombosis of the vena cava may occur spontaneously.

There are two unique findings for this entity. Firstly, the disappearance of the old thrombosed segment of this vessel due to organization and cicatization, as it can be referred to the “lonely aorta” sign, and the formation of a fibrous cord, which may become calcified, from the thrombosed IVC. Secondly, the absence of the posterior low-density area of the liver on the unenhanced cuts, representing the hepatic segment of the IVC.3

Other common causes of obstruction of IVC include congenital anomalies of the IVC, direct invasion by tumor, external compression by an intra-abdominal mass, and primary venous neoplasm.4

Acute obstruction of the IVC above the renal veins often causes renal vein thrombosis and nephrotic syndrome. But if the obstructive process is gradual, this condition may cause debilitating lower extremity pain and swelling, back pain, weakness, and venous stasis ulceration.1 Therefore, in this case, it was presumed that the unexplained gross hematuria was associated with the congestion of the tortuous engorged veins in the pelviureteral mucosa as a kind of collateralization. The development of collateral venous circulation can also preserve renal function.1 Common collateral flow patterns include the ascending lumbar veins, the anterior abdominal veins, the internal paravertebral venous plexus and the hemiazygous orazygous vein.4

Anticoagulation therapy to prevent de novo thromboembolism generally is not required if an extensive collateral venous supply is established.5

The etiology of our patient’s caval obstruction may come into question. But, because of the presence of an infrahepatic calcified fibrous cord in abdominopelvic CT, a history of operation for femoral bone fracture with bed rest for 6 months, and marked varices on abdominal skin, we thought that obstruction of IVC involving both renal veins was probably acquired by thrombosis, and that the congestion of the tortuous engorged veins in the pelviureteral mucosa as a kind of collateralization should be considered to be the cause of painless gross hematuria.
REFERENCES