Inferior Vena Cava Thrombosis Associated with a Distended Ileal Neobladder

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Abstract

Venous stasis is generally accepted to be a predisposing factor for venous thrombosis. However, benign causes of inferior vena cava (IVC) obstruction with associated thrombus formation have not been well described. We herein present a case of IVC compression caused by a distended ileal neobladder measuring 2,000 mL in capacity that led to IVC thrombosis. Following transurethral drainage for six weeks and anticoagulation therapy with warfarin for six months, the thrombus completely disappeared. The patient was considered to have a hypercoagulable state resulting from an acute urinary tract infection, a condition that may be associated with an increased risk of thrombosis.

Key words: deep vein thrombosis, inferior vena cava, ileal neobladder, urinary tract infection

(DOI: 10.2169/internalmedicine.54.2606)

Introduction

Orthotopic neobladder reconstruction has become a standard procedure in patients undergoing radical cystectomy for invasive bladder cancer. This technique has eliminated the need for cutaneous stoma, urostomy appliances and/or intermittent catheterization. The use of a detubularized ileum for total bladder reconstruction provides a highly compliant, low-pressure bladder. However, a distended ileal neobladder may result in clinical manifestations of severe complications with almost no sensation of bladder fullness (1). We herein present a case of external compression of the inferior vena cava (IVC) caused by a massively distended ileal neobladder that led to IVC thrombosis.

Case Report

A 59-year-old man presented with a one-week history of abdominal distension and decreased urine output with a high-grade fever. The patient had undergone retroperitoneoscopic nephroureterectomy 22 months earlier for urothelial carcinoma of the right ureter followed by radical cystoprostatectomy and construction of an orthotopic ileal neobladder 17 months earlier for urothelial carcinoma of the bladder. The reservoir was a detubularized, ileal, U-shaped neobladder constructed using 45 cm of the terminal ileum. The patient’s postoperative course had been uneventful. Twelve months after neobladder construction, he had a bladder capacity of 400-500 mL, a post-void residual urine volume of <50 mL and no need for clean intermittent catheterization.

A physical examination revealed abdominal bulging and bilateral lower extremity swelling. The laboratory findings were as follows: white blood cell count, 10,700/mm³; platelets, 200,000/mm³; serum creatinine, 2.15 mg/dL; and C-reactive protein, 30.74 mg/dL. The prothrombin and activated partial thromboplastin times were within the reference ranges. Unenhanced computed tomography (CT) revealed that the massively distended ileal neobladder was compressing the IVC bifurcation against the fifth lumbar vertebra, where the right common iliac artery intersected in front of the bifurcation, with left-sided hydronephrosis and hydroureter (Fig. 1). Urethrocystoscopy confirmed the presence of neovesicourethral anastomotic stricture, which was treated with endoscopic urethrotomy. A 20-Fr Foley catheter was
introduced, and 2,000 mL of *Staphylococcus agalactiae*-infected urine was collected, resulting in a reduction in the lower extremity swelling. Intravenous ampicillin sodium/sulbactam sodium was subsequently administered to treat the bacterial infection. After one week of therapy, the patient’s renal function returned to normal, and contrast-enhanced CT identified a 5×5×12-mm thrombus in the IVC bifurcation, without pulmonary embolism (Fig. 2). According to the retrospective evaluation of contrast-enhanced CT scans obtained 12 months after neobladder construction, there was no evidence of venous thrombi in the iliac veins or IVC. After excluding the possibility of other risk factors for deep vein thrombosis (DVT), we presumed that compression by the distended neobladder had led to thrombus formation in the IVC.

Following the administration of transurethral drainage for six weeks and anticoagulation therapy with warfarin for six months, the thrombus completely disappeared, as observed on CT scans. After undergoing urethral catheter removal, the patient voided spontaneously, with a post-void residual urine volume of <50 mL.

**Discussion**

Venous stasis is generally accepted to be a predisposing factor for venous thrombosis, as Virchow postulated in 1856 (2). Occlusion of the IVC is a well-recognized complication of malignancy, either indirectly due to an increased tendency for thrombus formation or directly as a result of tumor thrombus extension, as in cases of renal cell carcinoma. In contrast, benign causes of IVC obstruction with associated thrombus formation have not been well described. There are few reports of uncommon benign causes, such as hepatic cysts, an enlarged renal pelvis due to pelviureteric junction obstruction (3) and polycystic kidneys (4) with thrombotic complications. To the best of our knowledge, this is the first report of IVC thrombosis as a complication of a distended ileal neobladder.

In the present case, IVC thrombosis developed distal (toward the heart) to the site of compression. Ideally, venous thrombosis should occur proximal (toward the limb) to venous compression. Thrombus formation is a dynamic proc-
ess in which some platelets adhere to, while others separate from, the developing thrombus, where the shear, flow, turbulence and number of platelets in the circulation greatly influence the architecture of the clot (5). Our hypothesis regarding the present case is that thrombus formation initially occurred at a region of venous stasis in the compressed IVC and then continued to develop downstream in the native geometry of the IVC via decompression of the neobladder due to various factors, including turbulence around the thrombus. In addition, the lack of use of antithrombotic therapy during this period may also have contributed to the development of the thrombus.

Venous obstruction secondary to compression of a distended urinary bladder was first described in 1960 (6). Recently, Evans et al. reviewed 15 previously reported cases of iliac venous obstruction due to bladder distention with no thrombotic complications (7). All but one patient presented with painless bilateral lower extremity edema, and bladder decompression using catheter drainage resulted in the prompt resolution of symptoms. A single case of IVC obstruction secondary to bladder distension has previously been reported (7). However, external compression of the iliac veins or IVC is not often associated with DVT (8). Venous stasis alone, although an important factor, is usually insufficient to produce thrombosis, and other thrombotic risk factors are often required for thrombus formation (9).

Acute infection and associated systemic inflammation may induce a hypercoagulable state. These pathologic conditions increase the thrombomodulin level, platelet reactivity and fibrinogen content, thus inhibiting fibrinolysis (10). Schmidt et al. reported that acute infections, including those of the respiratory tract, urinary tract, skin and intra-abdominal region, are associated with a ≥2-fold increased risk of DVT; this association was strongest during the initial two weeks after the onset of infection, gradually declining thereafter (11). However, symptomatic urinary tract infection (UTI) is an uncommon complication of neobladder reconstruction in the late postoperative period. In a study of 79 patients with orthotopic neobladder, new events of symptomatic UTI occurred in 36% of cases during the first three postoperative months, compared to only 10% between three and six months and 8% between six and 12 months (12). The common pathogens of symptomatic UTI were *Pseudomonas aeruginosa* (24%), *Escherichia coli* (24%) and *Klebsiella pneumoniae* (22%). In the present case, the patient was considered to have a hypercoagulable state induced by acute UTI, a condition that may be associated with an increased risk of thrombosis.

In conclusion, external compression of the IVC caused by a distended ileal neobladder may result in IVC thrombosis. However, venous stasis alone is usually insufficient to produce thrombosis, and other thrombotic risk factors, such as acute UTI, may be required for thrombus formation in the IVC.

The authors state that they have no Conflict of Interest (COI).

**References**


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