Case Report

Bilateral ureteral complete obstruction with huge spontaneous urinoma formation in a patient with advanced bladder cancer

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Abstract

Spontaneous rupture of the collecting system with extravasation of urine and urinoma formation is usually associated with urinary tract obstruction by a ureteral calculus. Tumor growth is an extremely rare cause of urinary extravasation. Here we report a case of bilateral obstructive uropathy with a huge spontaneous left retroperitoneal urinoma caused by advanced infiltrative transitional cell carcinoma of the urinary bladder. The point of leakage was located in the left renal pelvis. The urinary leakage ceased after percutaneous nephrostomy drainage, and the patient subsequently underwent radical cystoprostatectomy. Histopathology revealed a high-grade urothelial carcinoma of the urinary bladder with pelvic lymph node metastasis. The patient refused any adjuvant treatment and expired 6 months after the operation from disseminated metastasis from bladder cancer.

Keywords: bladder cancer; obstruction; urinoma

1. Introduction

Ureteral obstruction is a not uncommon complication of bladder cancer and is associated with advanced tumor stage and poor prognosis, while bilateral ureteral complete obstruction caused by bladder tumor is infrequent. Spontaneous urinary extravasation is not unusual and most commonly results from ureteral obstruction by calculus. Ureteral obstruction by neoplasm is a rare cause of spontaneous urinary extravasation. This case report describes a case of bilateral complete ureteral obstruction with left-sided urinary extravasation and a huge retroperitoneal urinoma caused by infiltrative transitional cell carcinoma of the urinary bladder.

2. Case report

A 77-year-old male visited our emergency department due to lack of passage of urine for 30 hours. On arrival, he had stable vital signs. Physical examination revealed a distended left abdomen without tenderness or rebounding pain. There was no distended urinary bladder and no flank knocking pain. Laboratory analysis revealed elevated serum creatinine (4.3 mg/dL) and blood urea nitrogen (46.3 mg/dL) levels. The hemoglobin level was low, at 86 g/L, and the white blood cell count was normal.

Ultrasound and abdominal noncontrast computed tomography (CT) disclosed bilateral hydronephrosis with a 20 cm cystic lesion over the left retroperitoneal space (Fig. 1) extending from the perirenal space along the psoas muscle to the pelvic cavity. The urinoma was drained percutaneously with an 8 F pigtail catheter. A persistent urinary drainage from the percutaneous retroperitoneal drain tube of about 2000–3000 mL/d was noted, and no urine was drained from a urethral Foley catheter. The serum creatinine and blood urea nitrogen levels normalized (1.0 mg/dL and 14.6 mg/dL, respectively) 2 days after drainage.

A contrast medium-enhanced CT was performed and revealed obstructed a left ureter with a point of leakage over the renal pelvis (Fig. 2). Cystoscopy was performed immediately after the CT study to attempt double-J stenting, and this
revealed a contracted urinary bladder with a diffusely ulcerative bladder mucosa that was blocking the bilateral ureteral orifices. Biopsies were taken over the ulcerative mucosa. Bilateral percutaneous nephrostomy drainage was performed after cystoscopy. Afterwards, the leakage point of the collecting system healed and the retroperitoneal drainage tube became dry.

The pathology report for the biopsy revealed high-grade transitional cell carcinoma. As there was no evidence of lymph node and distal metastasis on serial imaging studies, the patient subsequently underwent radical cystoprostatectomy with bilateral pelvic lymph node dissection and ileal conduit urinary diversion 2 weeks after the drainage by percutaneous nephrostomy.

Infiltrative tumor over the posterior wall and trigone of the urinary bladder with perivesical soft tissue involvement was noted during surgical exploration. The post-cystoprostatectomy pathology report demonstrated high-grade transitional cell carcinoma infiltrating the full thickness of the bladder wall, with perivesical soft tissue invasion and pelvic lymph node metastasis (stage pT3N1M0, according to American Joint Committee on Cancer, 6th edition). Both ureters were encased in the infiltrative bladder tumor, with no tumor growth in their lumens.

The postoperative course was smooth, and there was no recurrence of hydronephrosis or urinary extravasation on ultrasound follow-up after the operation. The patient refused any adjuvant treatment and expired 6 months after surgery from disseminated metastasis of bladder cancer.

3. Discussion

Ureteral obstruction by bladder tumor is not uncommon and is usually associated with advanced disease. In 1998, Halebian et al retrospectively evaluated 415 patients with transitional cell carcinoma of the urinary bladder who had received radical cystectomy. Their results showed a significant correlation between hydronephrosis and advanced cancer stage, as well as decreased patient survival. More than 90% of patients with bilateral obstruction had disease with extra- vesical extension.3

In 2007, Bartsch et al reviewed 788 patients who had bladder cancer treated with radical cystectomy and revealed that the incidence of unilateral hydronephrosis was 13.7% and the incidence of bilateral hydronephrosis was 3.2%. They also demonstrated that hydronephrosis without tumor involvement over the ureteral orifice was a significant marker for advanced disease.1 Leibovitch et al reviewed 122 cases of invasive transitional cell carcinoma of the urinary bladder and concluded that the 5-year survival rate in patients with ureteral obstruction was significantly lower than in those without obstruction.4

Spontaneous rupture of the collecting system is relatively rare, and is usually associated with urinary tract obstruction. Cooke and Bartucz reported 14 cases of spontaneous urinary extravasation over the upper urinary tract and demonstrated that incidence of urine extravasation was 0.1% in patients who underwent intravenous urography examination. Ureteral stone was the most common cause of obstruction.5

Koga et al reported 11 cases of spontaneous peripelvic extravasation. Urinary obstruction was caused by calculi in nine cases, by invasion of sigmoid carcinoma in one case, and by ureteral tumor (transitional cell carcinoma) in one.2 In 2006, Lien et al reviewed the literature and found that ureteral stones caused 50% of the cases of spontaneous urinary extravasation, followed in frequency by intravenous urography, pregnancy, enlarged prostate, neoplasm, and aortic aneurysm.6
There are two etiologies for spontaneous urinary extravasation from the upper urinary tract: caliceal fornix extravasation and ureteral (or peripelvic) rupture. The former is caused by a sudden increase in renal pelvis pressure and backflow of urine into the renal sinus in patients with acute obstruction. This is considered to be a protective physiological mechanism producing decompression that may protect the kidney from high-pressure injury. The latter results from direct injury by a stone during its passage.

The mechanism underlying urinary extravasation in tumor disease may be different from that caused by a stone owing to the gradual increase in intrarenal pressure. The actual mechanism of neoplasm-related urinary extravasation is unclear because only a limited number of cases have been reported.

The treatment of perirenal extravasation should be individualized. Conservative follow-up, endoscopic treatment, percutaneous urinary drainage, and surgical correction of obstruction have all been suggested depending on the underlying cause and amount of extravasated fluid.

Although ureteral obstruction induced by a bladder cancer is not uncommon, bladder cancer-related urinary tract obstruction leading to urinary leakage and urinoma formation is very rare. This case report reminds us that when peripelvic urine extravasation is noted in a patient without any sign of urinary calculi, further evaluation for malignant disease should be performed.

References