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Diagnostic challenges of postrenal acute kidney injury: a case report

Abstract

One of the causes of acute kidney injury (AKI) is urinary tract obstruction. Here, we report a case of a patient with a rare complication after transurethral resection of bladder tumor: iatrogenic supravesical obstruction of both ureters with secondary postrenal AKI, rupture of both ureters at the pyeloureteral junction, urine leakage into the retroperitoneal space, and peritoneal irritation. The correct diagnosis was established after abdomen and pelvis computed tomography (with intravenous iodinated contrast agent). Bilateral nephrostomy drainage resulted in resolution of symptoms.

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Key words: postrenal acute kidney injury, ureteral obstruction, urine leakage

INTRODUCTION

Acute kidney injury (AKI) is a clinical syndrome secondary to impaired kidney perfusion (prerenal AKI), damage to the kidneys (renal or intrinsic AKI) or obstruction of the urinary tract (postrenal AKI) [1]. According to *Kidney Disease: Improving Global Outcomes* (KDIGO) 2012 guidelines, AKI can be diagnosed when at least one of the following criteria is met:

— increase in serum creatinine (sCr) by at least 0.3 mg/dL (26.5 umol/L) over 48 hours;
— increase in sCr by at least 50% within the prior 7 days;
— reduced urine volume below 0.5 mL/kg of body weight for 6 hours.

Early diagnosis of AKI and identification of the underlying pathology allows for appropriate therapy. Most of the literature focuses on differentiating between prerenal and renal AKI [3]. In our presented case, the clinical suspicion of postrenal AKI enabled rapid diagnosis and successful treatment.

CASE REPORT

A 55-year-old male patient with the history of benign prostate hyperplasia as well as the right-sided orchidectomy and radiotherapy for seminoma of the right testicle has been admitted to the first-level (district) hospital on day 3 following transurethral resection of bladder tumor (TURBT) because of AKI of unknown origin. Before the surgery, the patient's filtration rate was normal and sCr was 0.7 mg/dL. After TURBT, the urinary catheter was left in the bladder and the patient was discharged home. On the following day, the patient presented to the hospital again complaining about anuria, not passing stools or gas, and lumbar pain. In the urology department, the diagnosis of AKI was made (rise in sCr to 5.6 mg/dL) accompanied by raised C-reactive protein (CRP) level — 45 mg/L. Due to a clinical suspicion of urinary bladder or lower urinary tract injury, abdominal computed tomography (CT) without IV contrast but with intravesical contrast enhancement (CT urography) was ordered. No bladder rupture or lower urinary tract pathology was identified. On abdominal ultrasound, marked pelvicalyceal system of the left kidney was reported. Chest HRCT showed signs of inflammation not characteristic of a viral infection. During the patient's hospital stay, an empirical antibiotic was introduced (amoxicillin with clavulanic acid). Forced diuresis with furosemide was unsuccessful. In the face of ineffective treatment and persistent anuria, worsen-

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ing symptoms, increasing sCr and CRP, the patient was transferred to our department.

On admission, the patient's general condition was assessed as moderate. He complained about abdominal pain, bloating and not passing gas. The urinary bladder was catheterized, and there was a trace of bloody urine in the bag. On physical examination, there was mild peripheral edema, bloated abdomen, tender lower abdomen, positive rebound tenderness (Blumberg's sign), reduced bowel sounds. The laboratory tests showed raised sCr (8.8 mg/dL), BUN (43 mg/dL), CRP (149 mg/L), leukocytosis (19.3 G/L), creatine kinase (708 U/L) and lactic dehydrogenase (341 U/L). The abdominal ultrasound showed no hydronephrosis or intraperitoneal free fluid. Abdominal and pelvic CT with and without IV contrast was obtained. The reporting radiologist, while looking at each phase of the scan, initially excluded postrenal kidney failure over the telephone consultation due to normal appearance of the pelvicalyceal systems. In our opinion, postrenal cause of AKI had to be sought considering the recent urological intervention, sudden onset of anuria and abdominal symptoms consistent with urine leakage. On the same day, cytoscopy was performed with an attempt to probe the ureters, which turned out unsuccessful due to local edema and post-surgical deformation; the diagnosis of perivesicular ureteral stricture was made and bladder injury was excluded. The antibiotic was converted to ceftazidime. Because of anuria and raised renal parameters, a double-lumen catheter was implanted in the right internal jugular vein and the patient underwent hemodialysis on the next day; also, right-sided nephrostomy was created in the operating theatre. We also received a written report from CT urography from the previous day, which stated the diagnosis of bilateral damage to the pelvicoureteral junctions (in the excretory phase, there was a slight extravasation of the contrast outside the urinary tract; free fluid was visible in the perirenal and retroperitoneal space, and a small volume of fluid was present within the peritoneum). The ureters showed contrast enhancement along their entire length; the left ureter was slightly dilated (up to 13 mm) in the pelvis. The kidneys were of normal size; however, marked pelvicalyceal systems were reported. The bladder was shrunk over the catheter, showing thickened irregular wall and a single gas bubble.

As a result of the intervention, we observed an increase in diuresis and lowering sCr level (10.22 → 6.62 mg/dL). The patient did not require renal replacement therapy anymore. Two days later, left-sided nephrostomy was created because of persistent symptoms relating to urine leakage (abdominal pain, bloating, marked peritonitis). The abdominal symptoms resolved and the diuresis increased to 4150 mL; sCr normalized over the next couple of days (1.06 mg/dL). The patient was in good general condition, with two functioning nephrostomies, and he was discharged home. He was booked for further outpatient treatment.

After about three weeks since his hospital stay, a free flow of urine through the urethra was observed following an attempted closing of both nephrostomies. The consulting urologist requested a descending pyelography. After administering contrast through nephrostomies to the renal pelvis on both sides, a critical stricture of the pelvicoureteral junction on the right side was noted. Additionally, due to the ureteral stricture in its supravesical part on the left side, the urine outflow from the left kidney was slightly compromised and the ureter was dilated. Both renal calyces were dilated and distended, probably secondary to inflammation. Despite the identified pathologies, the patient opted for nephrostomy closing. The intervention was performed and the patient was advised to present to hospital if any worrying symptoms develop, and further follow-up at the outpatient clinic was planned.

DISCUSSION

In this study, we reported a case of a male patient with an atypical postoperative complication, namely a iatrogenic obstruction of both ureters in the perivesical part following TURBT with secondary postrenal AKI, bilateral injury to the pelvicoureteral junctions and urine leakage to the retroperitoneal space causing peritonitis. In order to make diagnosis, both cystoscopy (which visualized the ureteral obstruction) and CT urography (to show urine leakage outside the urinary tract) were necessary. We achieved total resolution of AKI after bilateral nephrostomies were created. The physiological urine outflow was observed after a couple of weeks, most probably because the ureterovesical junction edema resolved and the subpelvic ureteral injuries healed. Ureteral stricture and impaired urine flow shown on imaging studies after the nephrostomies were closed imply the need for careful observation,

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Lack of hydronephrosis on abdominal ultrasound did not exclude the postrenal cause of AKI. Non-dilated obstructive uropathy accounts for 5% of postrenal AKI

Ureteral obstruction generated hydrostatic pressure, which, apart from impaired glomerular filtration and renal blood flow, led to an uncommon complication — subpelvic ureteral injury

and the patient may need reconstructive surgery in the future.

Caddeo et al. studied the incidence of AKI after urological surgeries in Royal Derby Hospital, Great Britain. In 21 patients with AKI following TURBT, the prerenal mechanism (postoperative bleeding or sepsis) was identified in 9 patients (43%), while the postrenal mechanism (ureteral obstruction) was identified in 6 patients (29%). In the remaining 6 patients, the exact mechanism of AKI was not identified [4]. In the case of our patient, the prerenal mechanism was deemed unlikely because of no signs of dehydration or hypotension. Because of anuria, instead of oliguria, the postrenal AKI appeared very likely. Ureteral obstruction generated hydrostatic pressure, which, apart from impaired glomerular filtration and renal blood flow, led to an uncommon complication — subpelvic ureteral injury.

Due to a lack of characteristic symptoms, ureteral injury is one of the most challenging diagnoses. Hematuria is present in 50–75% of patients, and the common complaints include: prolonged ileus, fever, abdominal pain, palpable abdominal mass (urinoma) [5]. The study of choice in suspected ureteral injury is CT urography, which should show leakage of the contrasted urine in the late phase (5–20 min after administration) [5, 6]. The ureteral injury can be secondary to both intraureteral (urolithiasis, posterior urethral valve) and extraureteral causes of impaired urine flow (pelvic masses, pregnancy, retroperitoneal fibrosis or Ormond's disease) [6]. In our patient, all of the mentioned symptoms except for urinoma were present, and the correct diagnosis in the primary urology ward was delayed because the contrast was initially administered only to the bladder. However, such a management can be justified, because it would make the diagnosis of post-TURBT bladder injury, which seemed more likely at the time.

It should be emphasized that the lack of hydronephrosis on abdominal ultrasound did not exclude the postrenal cause of AKI. Non-dilated obstructive uropathy accounts for 5% of postrenal AKI, mainly in dehydrated patients, in early obstruction or when the ureter is unable to dilate (malignant infiltration, retropetitoneal fibrosis) [7]. In our patient, an additional cause might have been tissue fragility resulting from previous radiotherapy for testicular seminoma. In the case of early ultrasound diagnosis, doppler ultrasound and evaluation of resistance index (RI) can be beneficial. Increased RI > 0.7 can precede hydronephrosis even by 18 hours; however, isolated RI value holds little clinical significance [8]. Also, the difference in RI between both kidneys can be helpful; if it exceeds 0.08–0.10, it can be suggestive of urinary tract obstruction on the side of the increased RI. RI is diagnostic within 6–48 h since the onset of urinary obstruction [9]. The use of Doppler ultrasound in the early phase, as in our case, could have helped initially clarify the 'marked pelvicalyceal system' and lead to a decision about urgent CT urography and finally to the correct diagnosis.

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