CASE REPORT

The bladder ran dry: bilateral ureteral obstruction

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SUMMARY

A relatively young healthy man (barring obesity and distant gouty arthritis) was admitted with severe acute kidney injury (serum creatinine, 15.9 mg/dL) following acute gastroenteritis and occasional use of diclofenac. Abdominal ultrasound revealed mild left hydronephrosis due to staghorn stone and normal right kidney. Soon after, complete anuria necessitating haemodialysis developed without pain or evidence of infection. CT imaging revealed stones obstructing the right ureter. Following urological surgery, postobstructive diuresis developed and the serum creatinine came down to near normal. The stones were identified as uric acid stones.

Anuria has a relatively narrow differential and painless (partially non-dilated) bilateral ureteral obstruction is a distinctly unusual cause. A review of the literature to cover all reported causes of bilateral ureteral obstruction is presented. Only a minority of cases were not associated with an underlying malignant disease or its treatment. The multifactorial aetiology of the patient's acute kidney injury (volume depletion, diclofenac and obstructive uropathy) is presented and discussed.

BACKGROUND

Anuria has a relatively narrow differential and painless bilateral ureteral obstruction due to a benign aetiology, uric acid urolithiasis obstructing urinary flow bilaterally (but at different levels) as its cause, is distinctly unusual.

CASE PRESENTATION

A 42-year-old man presented with acute onset (1 day) of lethargy and dyspnoea. His medical history was notable for morbid obesity and intermittent gouty arthritis treated with colchicine. He had one episode of renal colic 12 years prior which was not investigated.

Over the preceding week, he had vomiting, diffuse abdominal pain and watery diarrhoea and used occasional diclofenac 100 mg tablets. On admission, he had neither pain nor gastrointestinal symptoms. Vital signs were normal and examination was unremarkable excepting leg oedema. Laboratory tests revealed serum creatinine 15.9 mg/dL, blood urea nitrogen 86 mg/dL, sodium 144 mmol/L, potassium 6.4 mmol/L, phosphate 11.8 mmol/L, calcium 6.4 mmol/L, magnesium 1.6 mmol/L and uric acid 13.2 mg/dL, and pH 7.12 (BE -16.5), Hb 10.2 g/dL with normal glucose, liver enzymes and albumin (4.3 g/dL). Urinalysis showed microscopic haematuria, sodium 12 mmol/L, negative casts and cultures. Chest X-ray and plain abdominal radiography were normal. On abdominal ultrasound, the right kidney was normal and the left showed a stone at the renal pelvis with some hydronephrosis. The urinary bladder was partially filled.

He was hydrated and given Kayexalate, bicarbonate and calcium. On the next day anuria developed, serum creatinine rose to 17.3 mg/dL and urgent haemodialysis was required. Low-dose CT without contrast revealed bilateral

Figure 1 (A) Patient's abdominal CT without contrast, coronal-oblique view, showing left hydronephrosis due to a staghorn stone in the kidney's pelvis (arrow). The left ureter is normal. On the right side, hydroureter can be seen (broken arrow) and the kidney is normal. (B) Patient's abdominal CT without contrast, coronal-oblique view, showing longitudinally placed stones in the lower third of the right ureter (so-called 'steinstrasse'—'stone street', although usually in the context of lithotripsy) causing right hydroureter (broken arrows). The stone in the kidney pelvis on the left can also be seen (arrow). At the time of the imaging, the patient's urinary output was nil.

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ureteral obstruction by stones at different levels (figure 1A,B). Ureteroscopy with stone removal (right) and leaving of double J stent (left) was performed. Another haemodialysis was necessary before improvement was achieved, marked by postobstructive diuresis (up to 7 L/day) and gradual decrease of serum creatinine to 2.7 mg/dL on discharge. Analysis of the stones confirmed uric acid nephrolithiasis. Allopurinol was added to the treatment and was well tolerated, given in addition to a low-purine diet and raising urine pH. Approximately 1 month after his discharge, percutaneous nephrolithotomy (PNL) was successfully performed to remove the left staghorn calculus. If left untreated, a staghorn stone will eventually destroy the kidney. He remains asymptomatic on a follow-up (at 6 months), and the last serum creatinine value is 1.6 mg/dL.

DISCUSSION

Acute kidney injury (AKI) can be non-oliguric, oliguric or anuric—a classification that may be useful in the need to quickly establish aetiology and determine prognosis.^{1,2} Anuria

(0–50 mL urine/day) often requires renal replacement therapy, as in our patient, who presented with the most severe stage of AKI according to RIFLE criteria. Indeed, the AKI was multifactorial as he had severe upper (recurrent vomiting) and lower (watery diarrhoea) gastrointestinal fluid losses which suffice to mediate volume depletion and marked prerenal failure. In addition, in this setting, the use of non-steroidal anti-inflammatory drugs (NSAIDs) significantly increases the risk of NSAID nephrotoxicity.³ Thus, we did not suspect a predominant role of obstructive uropathy here until complete anuria in a patient with a history of gout and ‘unilateral’ nephrolithiasis led to the CT-based diagnosis of acute bilateral ureteral obstruction, possibly precipitated by the volume depletion and oliguria.⁴ The absence of renal colic and the false-negative plain films (since uric acid stones are radiolucent) and ultrasound contributed to the diagnostic delay which ended when anuria supervened. In most reported series, postrenal aetiologies are usually responsible for only ~1:10 of AKI. Myriad aetiologies of bilateral ureteral obstruction have been identified in a literature search (box 1). Most patients had an underlying malignant disease.⁵ Nephrolithiasis was seldom the cause, and when it occurs, the majority of patients have a moderate to severe renal colic and normal urinary output.⁶ In conclusion, our patient’s AKI was first thought to be prerenal and NSAID induced, and his unilateral hydronephrosis was not considered emergent by the consultant urologists. However, complete anuria suggested otherwise and CT demonstrated unequivocal bilateral ureteric obstruction by stones at the level of the ureteropelvic junction (left) and at the lower ureter (right). These were treated successfully, and the patient’s severe AKI quickly regressed to near-normal glomerular filtration rate (GFR). Thus, neither the absence of pain⁶ nor the absence of dilatation^{7,8} can rule out obstructive uropathy which has a wide differential. The absence of convincing hydronephrosis could be due either to a low GFR, as in this case, or to an infiltrating malignancy.⁸ Early imaging by CT is highly useful in timely diagnosis and in guiding treatment⁹ and needs to be done early to enable renal salvage by rapid decompression.

Box 1 Causes of bilateral ureteral obstruction

I. Malignancy related (~80%) (3)

Intrinsic or extrinsic obstruction (by periureteral metastases or metastatic lymph nodes) due to non-urological malignancy (primarily cervix, stomach, colorectal and ovary) or renal/bladder/prostate cancer. Rarely reported in lymphoma, myeloid leukaemia (chloroma), primary systemic amyloidosis and myelofibrosis. See also, ‘latrogenic’.

II. Benign (~20%)*

1. Ureteral stones (uric acid stones, calcium stones)
2. Retroperitoneal fibrosis and/or inflammatory abdominal aortic aneurysm (rarely, idiopathic ureteral fibrosis)
3. Acute bleeding—retroperitoneal haematoma (massive) or pelvic haematoma (eg, after fracture); renal pelvis/ureter haematoma;
4. Papillary necrosis (eg, associated with diabetes mellitus)
5. Infection—primarily ureteropelvic fungus balls (candida, aspergillus and saccharomyces); tuberculosis; bladder condylomata acuminata
6. Iatrogenic—surgical injuries such as reported after correction of vesicoureteral reflux, aortofemoral bypass graft or management of malignancy; radiation-induced strictures; adverse drug effects (warfarin, carboplatin, indinavir and guaifenesin)
7. Ureteral vasculitis—polyarteritis nodosa, Churg-Strauss syndrome
8. Women’s health—pregnancy; abdominal pregnancy; endometriosis; accidental ureteral ligation during hysterectomy; uterine prolapse (see also, I. Malignancy related)
9. Varia—postappendicitis; appendiceal mucocele; complete scrotal urinary bladder herniation; detrusor hypertrophy in prostatic hyperplasia; bladder wall oedema (eg, after vaginal hysterectomy); chronic granulomatous disease. Transient reflex anuria in unilateral ureteral obstruction had also been reported.

Based on PubMed search of ‘bilateral ureteral obstruction’ since inception (English, human, adult) yielding 143 articles, and their citations. References can be obtained on request.

* Several entities are quite rare.

Patient’s perspective

I thought I was doomed for dialysis forever. It is amazing that both kidneys could be obstructed at once and that so many stones did not cause any pain. I only hope this would not recur.

Learning points

- ▶ Complete anuria has a narrow differential, and its presence can serve as a useful diagnostic clue.
- ▶ Acute bilateral ureteral obstruction is an unusual cause of anuria and can sometimes present without pain or without convincing hydronephrosis or both.
- ▶ Gout with uric acid stones are radiolucent and may be missed but will be accurately depicted by low-dose unenhanced CT.
- ▶ Reported cases of bilateral ureteral obstruction have been reviewed and reveal a wide and intriguing differential diagnosis.

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REFERENCES

- 1 Choi HM, Kim SC, Kim MG, *et al.* Etiology and outcomes of anuria in acute kidney injury: a single center study. *Kidney Res Clin Pract* 2015;34:13–19.
- 2 Klahr S, Miller SB. Acute oliguria. *N Engl J Med* 1998;338:671–5.
- 3 Whelton A. Nephrotoxicity of nonsteroidal anti-inflammatory drugs: physiologic foundations and clinical implications. *Am J Med* 1999;106:13S–24.
- 4 Wiederkehr MR, Moe OW. Uric acid nephrothiasis: a systemic metabolic disorder. *Clin Rev Bone Miner Metab* 2011;9:207–17.
- 5 Organ M, Norman RW. Acute reversible kidney injury secondary to bilateral ureteric obstruction. *Can Urol Assoc J* 2011;5:392–6.
- 6 Salter CA, Lang C, Altamar HO. Anuria secondary to bilateral obstructing ureteral stones in the absence of renal colic. *J Endourol Case Rep* 2016;2:93–5.
- 7 Maillet PJ, Pelle-Francoz D, Laville M, *et al.* Nondilated obstructive acute renal failure: diagnostic procedures and therapeutic management. *Radiology* 1986;160:659–62.
- 8 Kocurek JN, Orihuela E, Saltzstein DR. Nondilated obstructive uropathy and renal failure as a result of carcinoma of the intrapelvic area. *Surg Gynecol Obstet* 1991;173:470–2.
- 9 Türk C, Petfik A, Sarica K, *et al.* EAU guidelines on diagnosis and conservative management of urolithiasis. *Eur Urol* 2016;69:468–74.

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