CASE REPORT

Pyelonephritis and obstructive uropathy: a case of acute kidney injury

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SUMMARY

We present a case of a man in his late 50s with a history of metastatic prostate carcinoma requiring bilateral ureteric stenting. He was admitted with increasing confusion and lethargy. He was diagnosed with sepsis and an acute kidney injury (AKI). Clinical suspicions of an obstructive component to his AKI were not confirmed by an ultrasound scan, which showed a unilateral hydronephrosis unchanged from a scan 1 month previously. A nephrostomy was performed, and frank pus aspirated. The patient's clinical state improved steadily thereafter. Patients who are dehydrated, or who have suffered from malignant or fibrotic processes affecting the retroperitoneum, may present with urinary obstruction without a corresponding increase in urinary tract dilation. Additionally, there must be a suspicion of pyonephrosis in a symptomatic patient with known hydronephrosis. Clinicians should be aware that clinical suspicions of urinary obstruction not demonstrated on ultrasound scanning require further investigation.

BACKGROUND

Acute kidney injury (AKI) refers to an acute decline in renal excretory function over a period of hours or days leading to the accumulation of waste products, sometimes accompanied by oliguria, hyperkalaemia, hyperphosphataemia and a metabolic acidosis.¹ In 2012, the Kidney Disease Improving Global Outcomes (KDIGO) group provided a consensus definition of AKI (table 1)² that is an amalgamation of two earlier classification systems.^{3 4} The incidence of AKI has been found to be 2147 per million population per year.⁵ AKI is a particular problem in critical care settings.⁶

The causes of AKI can be separated into pre-renal, renal and postrenal. Pre-renal AKI is caused by renal hypoperfusion due to hypotension or decreased effective circulating volume.⁷ Renal AKI can be caused by prolonged pre-renal causes, infectious agents, toxins (including endotoxin), or autoimmune processes.⁷ Postrenal AKI is due to obstruction of the urinary tract, usually associated with hydronephrosis.^{7 8}

In patients found to have an AKI, an ultrasound scan of the urinary tract should be offered within 24 h if obstruction is suspected or the aetiology unclear, or within 6 h if pyonephrosis is suspected.⁹ Many cases of AKI may be preventable by fluid administration and cessation of antihypertensive or nephrotoxic medications—thus fluid balance, urine output recording, and biochemical and drug assessment are warranted.¹⁰ Postrenal AKI can be treated via nephrostomy or stenting. General measures

include volume support, the treatment or prevention of hyperkalaemia and metabolic acidosis, and adjustments in medication regimens to account for the renal impairment.²

CASE PRESENTATION

A farmer in his late 50s with a background of terminal metastatic prostate carcinoma presented to accident and emergency with lethargy, reduced exercise tolerance, productive cough, reduced appetite, vomiting, twitching and confusion. Three weeks previously, he had undergone bilateral nephrostomies with antegrade ureteric stent insertion for urinary obstruction, at which time an ultrasound scan showed significant asymmetry in renal size (kidney length 12.2 cm on the left, 9.5 cm on the right, with a corresponding decrease in cortical mass). He had also had a more recent admission with frank haematuria. His baseline estimated glomerular filtration rate (eGFR) was 30–35 mL/min. Physical examination found pallor, dehydration and bibasal crepitations. His blood pressure was 147/84 mm Hg, heart rate 95 bpm, respiratory rate 15 per minute, temperature 37°C and his oxygen saturations were 97.1% on air. He was passing urine. He had a tender left loin on palpation of his abdomen.

INVESTIGATIONS

Arterial blood gas (ABG) analysis showed a metabolic acidosis (pH 7.33, pCO₂ 3.66 kPa, pO₂ 12.4 kPa, HCO₃⁻ 16.1 mmol/L, base excess -10.6 mmol/L). A full blood count revealed the patient's haemoglobin to be 83 g/L, and he had a white cell count of 6.6×10⁹/L and a platelet count of 72×10⁹/L. His urea and electrolytes showed Na⁺ 125 mmol/L, K⁺ 7.7 mmol/L, urea 52.8 mmol/L and creatinine 773 µmol/L (his baseline was 150–200 µmol/L). His C reactive protein was 362.1 mg/L and albumin, 22 g/L. An ECG showed 2 mm ST elevation in V₁–V₃ and tented T waves in V₂–V₄. A chest X-ray showed bibasal changes and prominent pulmonary veins. A portable ultrasound scan showed bilateral ureteric stents and left-sided moderate hydronephrosis, unchanged from what had been shown in a previous scan 1 month earlier. No hydronephrosis of the right kidney was visualised. Further imaging with CT scan was deemed unnecessary at the time of acute imaging, although this may have provided better evidence of pyonephrosis with perinephric stranding or gas in the collecting system, had it been performed.

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Table 1 KDIGO classification system for AKI²

Criteria		
Stage	Serum creatinine	Urine output
1	1.5–1.9×baseline, or increase of ≥ 0.3 mg/dL (26.5 μ mol/L)	<0.5 mL/kg/h for 6–12 h
2	2–2.9×baseline	<0.5 mL/kg/h for ≥ 12 h
3	≥ 3 ×baseline, or increase to ≥ 4 mg/dL (354 μ mol/L), or initiation of renal replacement therapy, or in patients <18 years decrease in estimated GFR to <35 mL/min/1.73 m ²	<0.3 mL/kg/h for ≥ 24 h, or anuria for ≥ 12 h

AKI, acute kidney injury; GFR, glomerular filtration rate; KDIGO, Kidney Disease Improving Global Outcomes.

DIFFERENTIAL DIAGNOSIS

This patient had chronic kidney disease stage 3b evidenced by his baseline eGFR of approximately 30–35 mL/min. He was correctly diagnosed as having an AKI stage 3 in the KDIGO classification.²

The AKI was initially attributed to sepsis or hypotension-induced acute tubular necrosis (ATN), a pre-renal cause, secondary to pneumonia or a urinary tract infection (UTI). An obstructive component to the AKI was not considered due to the unchanged ultrasound appearances.

The patient's confusion was assumed to be due to a combination of septic encephalopathy and uraemia.

The initially dismissed alternative diagnosis was an obstruction to the left ureter, with accompanying left-sided hydronephrosis and suppurative pyelonephritis, also known as a pyonephrosis.¹¹ This is often only demonstrable on intervention.

TREATMENT

Initial medical management consisted of intravenous antibiotics, fluids, calcium gluconate and dextrose with insulin. When a second ABG demonstrated the hyperkalaemia persisting despite medical management, haemodialysis was performed. This improved the biochemical picture (Na^+ 129 mmol/L, K^+ 4.8 mmol/L, urea 26.4 mmol/L, creatinine 432 μ mol/L) but not the clinical state. The patient was increasingly confused and displaying myoclonic jerks. His blood pressure had dropped to 120/70 mm Hg, and his heart rate was 130–150 bpm and now irregularly irregular. His temperature was 36.2°C. A decision was made to perform a nephrostomy without further imaging, after discussion with an interventional radiologist (figure 1). Frank pus was aspirated, confirming a left-sided pyonephrosis.

OUTCOME AND FOLLOW-UP

A few hours following the nephrostomy, the patient's confusion had improved, and his other observations improved in the days following as the sepsis resolved. The exception was his blood pressure, which had dropped to 85/67 mm Hg, probably due to postobstructive polyuria and bacteraemia following the nephrostomy insertion. Clearly, source control with drainage of the collection was key to improving this patient's clinical outcome. At one point, he was passing 100–200 mL/h via his nephrostomy along with a further 20–30 mL/h via a catheter. The ultrasound-visualised asymmetry in the renal mass 3 weeks earlier suggested that the majority of the patient's renal reserve was left-sided. Thus a left-sided ureteric obstruction is likely to have caused a predominantly postrenal AKI, although the right kidney would still have been affected by ATN as a result of the

Figure 1 Image from radiological nephrostomy procedure. Gross hydronephrosis is seen.

pyonephrosis and accompanying urosepsis. He was discharged home 10 days later after responding to his supportive care. On his final day in hospital, his urea and electrolyte results were: Na^+ 136 mmol/L, K^+ 5.2 mmol/L, urea 16.4 mmol/L and creatinine 233 μ mol/L (representing his new baseline).

The patient was admitted 11 days later with increasing confusion. An MRI of his spine showed extensive pelvic metastases. He rapidly deteriorated, becoming increasingly confused, although a CT scan of his head showed no brain metastases. The patient died 2 weeks later.

DISCUSSION

Urology patients have a high risk of developing AKI.⁸ The pathophysiology of sepsis-induced pre-renal AKI is multifactorial, including ischaemia secondary to hypoperfusion and intrarenal vasoconstriction, as well as direct lipopolysaccharide-induced damage.⁷ Postrenal causes inflict damage by increasing the pressure within the renal tubules, leading to cellular damage.¹² Sepsis is a clinical syndrome defined as the presence of both infection and a systemic inflammatory response.¹³ Commonly, sepsis is said to be present where there is suspected or confirmed infection along with the presence of two or more markers of severe inflammatory response syndrome.¹³ Severe sepsis is defined as tissue hypoperfusion or organ dysfunction as a result of infection,¹³ and it has a mortality rate of 20–40%.¹⁴ Septic encephalopathy, as had occurred in this case, is the most frequent complication of sepsis.¹⁵

Sepsis due to a UTI is known as urosepsis.¹⁴ Urosepsis occurs most commonly as a result of a UTI with complicating factors such as obstruction to the flow of urine.¹⁶ Infectious organisms may reach the male bladder through instrumentation, such as with urinary catheters. Patients with bladder outflow obstruction (such as that occurring with prostatic neoplasms) are less likely

to be able to empty the bladder completely, allowing any infectious organisms left within to multiply.¹⁷ Ascension of these organisms to the upper urinary tract can occur directly through the ureter or through lymphatic channels,¹¹ potentially leading to pyelonephritis or pyonephrosis. Alternatively, organisms can reach the upper urinary tract haematogenously, but this is less common.¹¹ Pyonephrosis is a potentially deadly condition.¹¹ Although it typically presents acutely, it can cause a more insidious presentation and thus should be considered as a potential diagnosis in any patient with known hydronephrosis and fever.¹⁸

The detection of a postrenal component to the AKI required a very high index of suspicion on behalf of the clinicians involved, due to the lack of change in the ultrasound scan-visualised hydronephrosis. Medical students are taught that good history-taking and examination techniques are still the most important skills in modern medicine, despite the vast array of modern imaging techniques available. In this case, it may have been tempting to believe that the scan had ruled out obstruction, something that was strongly suggested by the patient's history. However, there are case reports of obstruction occurring in the absence of dilation.^{19–22} One study found that, out of 80 patients with acute obstructive anuria, four lacked any evidence of urinary tract dilation on ultrasound scan.²³

Lyons *et al*¹⁹ discussed three cases of non-dilated obstructive uropathy. Two of these were males in their eighth decade, with urological malignancies, presenting with symptoms and signs suggestive of obstruction without radiological evidence. Renal functional improvement occurred in both cases following nephrostomy. Canavese *et al*²⁰ presented a case of non-dilated obstructive uropathy in a 59-year-old man with metastatic pancreatic cancer. Ultrasonography and CT scanning were unable to demonstrate urinary tract dilation. Five days later, a repeat scan was undertaken and mild dilation was seen—thus ureteric stenting was performed. A further two cases of non-dilated obstructive uropathy occurring on a background of malignancy are presented by Onuigbo *et al*,²¹ the first being a 56-year-old man who was eventually diagnosed with a urothelial carcinoma and the second, a 59-year-old woman with a history metastatic uterine cervical adenocarcinoma. Leong *et al*²² presented a case in a 31-year-old man with a history of ulcerative colitis, who presented in a similar way to the case presented here, with flank pain, dehydration and sepsis. The lack of dilation in these cases may be due to hypovolaemia, oedema, retroperitoneal metastases, retroperitoneal fibrosis or a retroperitoneal tumour.^{19–22} Urogenital malignancies may infiltrate retroperitoneal tissue leading to a lack of dilation, which may be compounded by dehydration.²¹

Where there is clinical suspicion of obstruction without radiological evidence, further diagnostic testing should be considered.^{19–21} This may take the form of CT scanning (to demonstrate tumours),¹⁹ or antegrade pyelography.^{19–21} Multiple factors predisposing to AKI may be present in any one patient.⁵ The rapid-onset, extreme loss of renal function occurring here, in combination with the clinical examination findings, were consistent with a postrenal component to the AKI.²⁴ Obstruction in the presence of suppurative pyelonephritis, or pyonephrosis, is a particularly serious condition that has the ability to lead to severe sepsis, and thus death.¹⁴ The index for suspicion should thus be very high in a patient with known hydronephrosis. This case illustrates that being confident in one's history-taking and examination findings can be rewarding, even in the face of equivocal radiological evidence.

Learning points

- ▶ When presented with a case of acute kidney injury (AKI), consider the possibility that multiple aetiologies may be responsible in any single patient.
- ▶ Have a high index of suspicion for postrenal causes of AKI. These are associated with extreme disturbances in renal function while also being the most immediately amenable to treatment.
- ▶ The presence of urogenital malignancy, retroperitoneal fibrosis or oliguria may lead to false-negative results when assessing for ureteric obstruction utilising an ultrasound scan.
- ▶ In a symptomatic patient with known hydronephrosis, there should be a high index of suspicion of pyonephrosis.

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Reminder of important clinical lesson

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