Renal colic in a dialysis patient: a case of renal stone disease

Olympia Koulouri • Sarah Jones • Richard Beable • Jonathan Barratt

University Hospitals of Leicester NHS Trust, Leicester, UK

Correspondence to: Jonathan Barratt. Email: jonathan.barratt@uhl-tr.nhs.uk

DECLARATIONS

Competing interests
None declared

Funding
None

Ethical approval
Written informed consent to publication was obtained from the patient or next of kin

Guarantor
JB

Contributorship
OK and SJ wrote the report; RB collated and annotated the radiological images; JB edited the final report

Acknowledgements
None

Reviewer
John Bradley

This report highlights the importance of always considering renal colic as a cause of abdominal pain in patients on dialysis.

Case report

A 45-year old man on home haemodialysis presented with a 6-h history of severe right-sided abdominal colic, non-radiating and associated with rigors and nausea but no vomiting. The pain had not been relieved by simple measures including a change of posture or over the counter analgesics. Twenty-four years previously he had undergone a panproctocolectomy and ileostomy for Crohn's disease. His ileostomy had been functioning normally with no recent change in content or output. He had been anuric for over 12 months prior to this presentation and described no new lower urinary tract symptoms.

He had developed end-stage renal disease secondary to chronic pyelonephritis at the age of 36 years and had been receiving home haemodialysis treatment for the preceding two years after a failed renal transplant. He was awaiting a parathyroidectomy for tertiary hyperparathyroidism. He had suffered an episode of right ureteric obstruction secondary to a calculus 11 years previously, when dialysis independent, which was treated successfully with a right nephrostomy. An antegrade nephrostogram at the time of nephrostomy insertion demonstrated a lucent filling defect in the upper third of the right ureter. A repeat nephrostogram one week later demonstrated clearance of the previously identified filling defect and the nephrostomy was removed. He had also presented two years previously while on dialysis with painless visible haematuria which resolved spontaneously. A computed tomography (CT) scan of the kidneys, ureter and bladder at that time demonstrated a 6-mm right-sided distal ureteric stone (Figure 1). It was decided at that point to adopt a watchful waiting strategy and he remained completely asymptomatic for the following two years.

On examination he was afebrile, blood pressure 165/98 mmHg, heart rate 102 bpm. Abdomen was soft and non-distended but tender on palpation over the right flank with no signs of peritonism. Bowel sounds were normal, and a functioning ileostomy was noted in the right iliac fossa. Examination of the cardiovascular and respiratory systems was unremarkable.

Full blood count revealed a normocytic normochromic anaemia (Hb of 11.5 gd/L), WBC 8.9 $\times$ 10^9/L and Plts 305 $\times$ 10^9/L. Serum biochemistry was consistent with tertiary hyperparathyroidism – parathyroid hormone 166 pmol/L, adjusted Ca 2.38 mmol/L, PO_4 1.28 mmol/L, ALP 360 U/L. Liver function tests and amylase were normal. C-reactive protein was elevated at 129 mg/L. An abdominal ultrasound scan demonstrated atrophic kidneys with no evidence of hydronephrosis or other intra-abdominal pathology (Figure 2). A CT scan of the kidneys, ureter and bladder was performed because of persistent abdominal pain and this again identified a 6-mm calculus in the distal section of the right ureter (Figure 3).

Following urological review the patient underwent a right ureteroscopy and laser lithotripsy with removal of the stone fragments. Immediately postprocedure the patient's pain resolved completely and he was discharged home 24 h later.

Figure 1
Non-contrast CT scan taken during the earlier presentation with painless visible haematuria demonstrating parenchymal calcification in a small right kidney (upper arrow) and a calculus in the distal third of the right ureter (lower arrow)

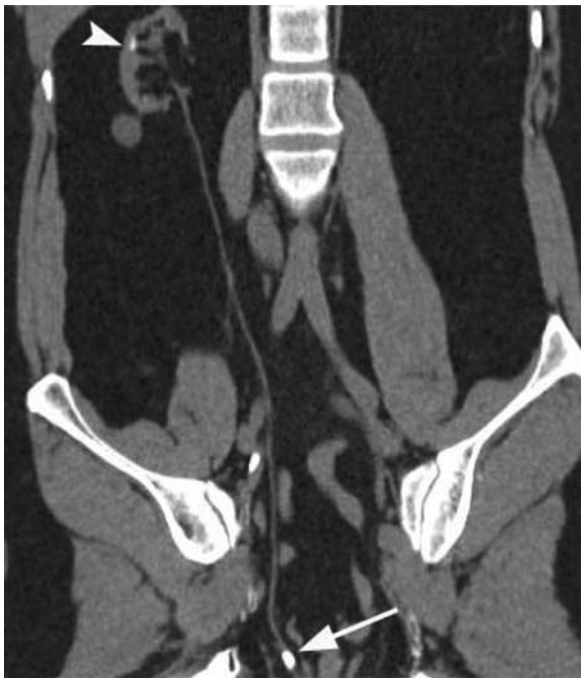
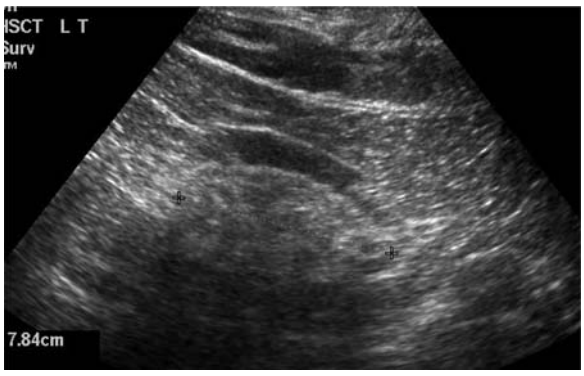


Figure 2
Ultrasound scan of the right kidney taken during the emergency presentation with right-sided renal colic demonstrating a small right kidney (7.84 cm). As the patient was anuric hydronephrosis did not develop despite complete ureteric obstruction

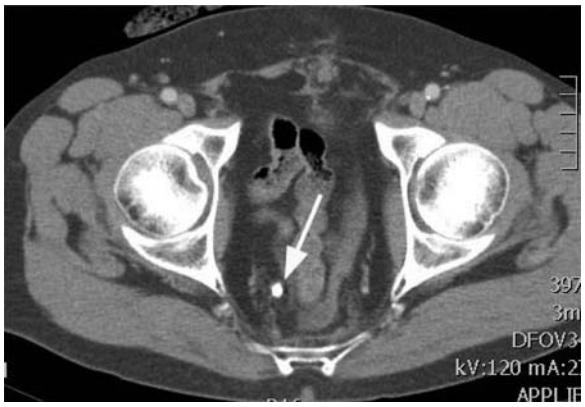


Discussion

Renal colic is often overlooked as a cause of abdominal pain in patients with end-stage renal disease receiving dialysis, particularly if they are anuric. Many physicians assume that patients on dialysis do not develop symptomatic stone disease due to their low or absent urine output. However, the incidence of symptomatic renal calculi in patients undergoing dialysis has been reported to be as high as 13%, with a mean incidence of 6.6% in a review of five studies involving a total of 531 dialysis patients.[1] This is similar to the incidence of kidney stones in the non-dialysis population.[2] As in the general population dialysis patients may present with classic ureteric colic, seen in this case, or more commonly vague abdominal, flank or lumbar pain.[3] A pre-dialysis history of kidney stones, as in this case, is not always present as many patients present with their first kidney stone after they have commenced dialysis. Furthermore, it has been reported that over 80% of dialysis patients presenting with renal stones will develop recurrent stones on dialysis.

The precise mechanism for stone formation in oliguric and anuric dialysis patients is not known. In contrast to the non-dialysis population where calcium-containing stones are by far the most common type of stone, dialysis patients predominantly form variably mineralized protein stones comprising a mixture of β2-microglobulin, lysozyme, serum amyloid P protein, albumin and Tamm-Horsfall protein.[4,5] The variable calcium oxalate content of these stones means they are more difficult to visualize on CT scanning and may in some cases be entirely radiolucent. In these latter cases the patient may present with recurrent episodes of abdominal pain and, if still producing appreciable amounts of urine, unexplained intermittent hydronephrosis. In this case it is not possible to be sure which factors contributed to stone formation, however the history of Crohn's disease and previous intestinal surgery have both been strongly linked to the development of renal tract stones. Crohn's disease can result in variable degrees of fat malabsorption which in turn leads to increased delivery of fatty acids to the colon and excessive colonic absorption of oxalate, a condition known as enteric hyperoxaluria. Ileostomies with high fluid output lead to

Figure 3
Non-contrast CT scan taken during the emergency presentation with right-sided renal colic demonstrating a 6-mm calculus in the distal segment of the right ureter



hypokalaemia and low urine volumes, hypocitraturia and low urine pH, all of which promote stone formation.[6]

Treatment of renal stone disease in dialysis patients is similar to that in the general population. Most patients will spontaneously pass their stones, however extracorporeal shock wave lithotripsy can be used depending on the patients urine output. If urine output is insufficient, as in this case, laser lithotripsy is necessary with manual removal of the stone fragments.

This case highlights the importance of considering renal stone disease in any dialysis patient presenting with abdominal pain to the medical or surgical admissions unit. Even if a dialysis patient reports minimal or no urine output they are still capable of forming symptomatic renal tract stones and importantly these may on occasion be radiolucent.

References

1 Viterbo R, Mydlo JH. Incidence and management of dialysis patients with renal calculi. *Urol Int* 2002;**69**:306–8
2 Parmar MS. Kidney stones. *BMJ* 2004;**328**:1420–4
3 Caralps A, Lloveras J, Andreu J, *et al.* Urinary calculi in chronic dialysis patients. *Lancet* 1979;**2**:1024–5
4 Oren A, Husdan H, Cheng PT, *et al.* Calcium oxalate kidney stones in patients on continuous ambulatory peritoneal dialysis. *Kidney Int* 1984;**25**:534–8
5 Daudon M, Lacour B, Jungers P, *et al.* Urolithiasis in patients with end stage renal failure. *J Urol* 1992;**147**:977–80
6 Bambach CP, Robertson WG, Peacock M, Hill GL. Effect of intestinal surgery on the risk of urinary stone formation. *Gut* 1981;**22**:257–63