

CASE REPORT

Gastrointestinal obstruction secondary to chronic obstructive nephrolithiasis: A case report

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Abstract

Background: Nephrolithiasis is a common urologic condition, with increasing prevalence in the United States. This can often present with flank pain and hematuria; however, sequelae such as hydroureteronephrosis, forniceal rupture, and pyelonephritis can lead to more severe complications.

Case Presentation: This case report describes a 43-year-old female who presented to the hospital with left upper quadrant pain, fever, nausea, and vomiting. Imaging revealed an obstructive 0.7 cm kidney stone at the right ureterovesical junction with severe hydroureteronephrosis resulting in mass effect on the duodenum and upstream gastric dilation. Percutaneous nephrostomy placement resolved both the hydroureteronephrosis and the secondary gastrointestinal obstruction.

Conclusion: This case study highlights the rare complication of bowel obstruction caused by obstructive nephrolithiasis, underscoring the diverse clinical presentations of nephrolithiasis and the importance of timely, conservative management to avoid clinical decompensation.

KEYWORDS

adult urology, calculus disease, gastrointestinal obstruction, megaureter, urology

1 | INTRODUCTION

In the United States, the prevalence of nephrolithiasis amongst adults has risen over the last three decades—from 1 of every 20 in 1988–1994, to 1 of every 11 in 2007–2010, to 1 of every 10 in 2017–2020^[1,2]. Depending on location and size, kidney stones may persist asymptotically, pass spontaneously, present with renal colic, or require urologic intervention^[3]. When symptomatic, patients may present with flank pain, hematuria, nausea, vomiting, and dysuria^[4]. Acute sequelae of nephrolithiasis include but are not limited to hydroureteronephrosis, pyelonephritis, or abscess^[5,6]. Severe or chronic obstructive nephrolithiasis may result in irreversible loss of renal function, hydroureteronephrosis leading to forniceal rupture and possible urinoma formation^[4].

Although uncommon, severe hydroureter may cause mass effect on adjacent organs. Currently, there is limited literature documenting secondary obstructive megaureter leading to bowel obstruction, especially in the adult population. One case report of a congenital giant megaureter leading to neonatal bowel obstruction has been documented^[7]. Another case report described a 30-year-old male who was found to have a severely hydronephrotic kidney that mimicked the symptoms of bowel obstruction^[8]. While not related to bowel obstruction, another case report documented a 16-year-old female with right-sided ureteral obstruction due to contralateral congenital giant megaureter in a duplicated collecting system^[9].

Megaureter can be classified into two types: primary or secondary. While primary megaureter is typically diagnosed in neonatal and pediatric patients and is

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defined as intrinsic issues with the ureter itself, for example, congenital ureterovesical junction (UVJ) obstruction, vesicoureteral reflux, or aperistaltic ureter^[10]. Secondary megaureter can present in both pediatric or adult populations and develops as sequelae from other conditions such as nephrolithiasis, mass effect from adjacent organs, neurogenic bladder, posterior urethral valves, or urethral atresia^[10].

This case report documents the rare presentation of an obstructive kidney stone resulting in gastrointestinal obstruction, not previously described in the current literature.

2 | CASE PRESENTATION

A 43-year-old female patient presented to the emergency department (ED) at a county hospital with a 1-week history of subjective fever, left upper quadrant abdominal pain, and 1 day of vomiting. The patient's medical history was significant for a debilitating cerebral

vascular accident and consequent sustained neurologic deficits, neurogenic bowel and bladder, and gastrostomy tube (G-tube) dependence.

In the ED, patient was afebrile, normotensive, and mildly tachypneic. Physical exam was unremarkable besides mild erythema surrounding the G-tube, although the exam was limited by the patient's nonverbal status. Initial labs demonstrated mild leukocytosis white blood cell count of $13.1 \times 10^9/L$ and normal renal function with glomerular filtration rate of $120 \text{ mL/min/1.73 m}^2$, pyuria and bacteriuria on urinalysis. Computed tomography (CT) of the abdomen and pelvis with contrast revealed severe right hydroureteronephrosis to the level of the right UVJ secondary to an obstructive 0.7 cm obstructive kidney stone (Figure 1). There was a severe hydroureter, measuring 1.8 cm axially. Additionally, a perinephric complex multiseptated hypodense cystic structure measuring $8.6 \text{ cm} \times 7.0 \text{ cm}$ was observed abutting the right medial and inferior kidney (Figures 2 and 3), causing mass effect on the duodenum and upstream gastric dilation. It was initially unclear where this cystic structure originated as there were no prior imaging studies available.

While in the ED, a nasogastric (NG) tube was placed for gastric decompression and broad-spectrum intravenous antibiotics were initiated upon presentation. Urology was consulted and recommended for

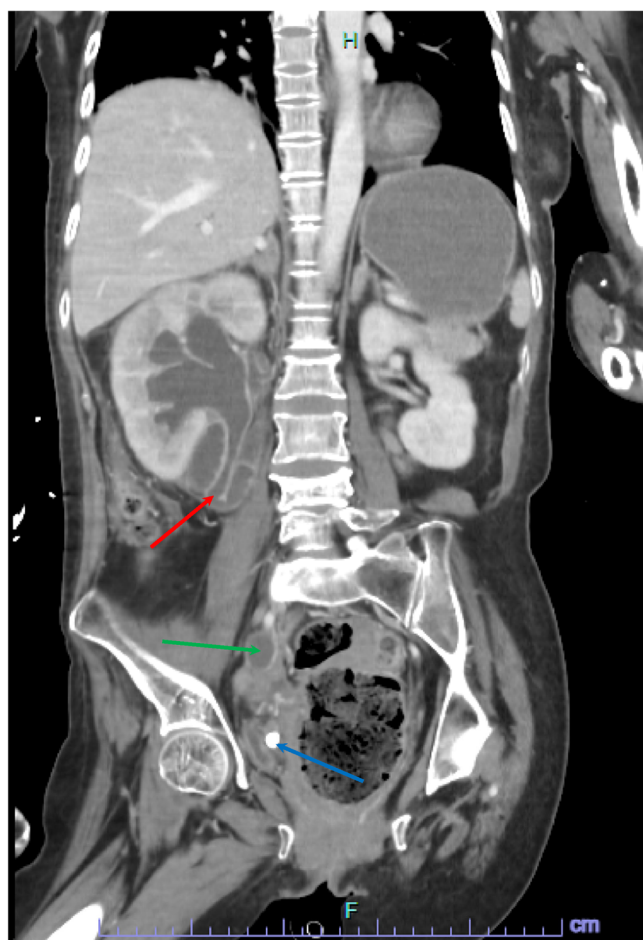


FIGURE 1 Computed tomography abdomen/pelvis with contrast (coronal view) on Day 1 of presentation. Dilated tortuous proximal ureter (red arrow) and distal ureter (green arrow). Distal obstructing 0.7 cm right ureterovesical junction stone (blue arrow).

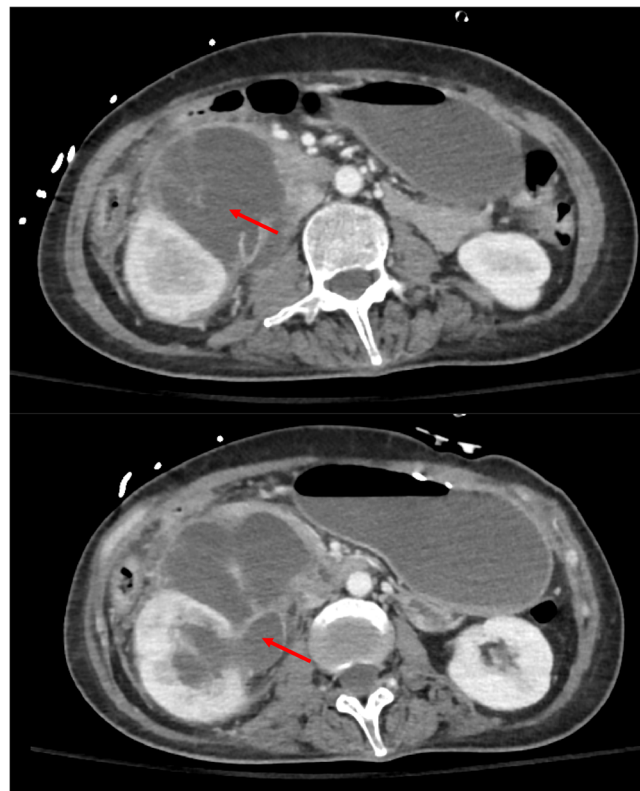


FIGURE 2 Computed tomography abdomen/pelvis with contrast (axial view) on Day 1 of presentation. Dilated tortuous right proximal ureter (red arrow) appearing as a perinephric complex cystic structure.



FIGURE 3 Computed tomography abdomen/pelvis with contrast (coronal view) on Day 1 of presentation. Dilated, tortuous proximal ureter appearing as perinephric cystic fluid collection (1) causing mass effect and subsequent dilation of the duodenum (2) and upstream dilation of the stomach (3).

interventional radiology (IR) to place percutaneous nephrostomy tube (PCN). After placement of PCN on hospital Day 2, repeat CT on Day 4 demonstrated resolution of the hydroureteronephrosis, reduction in size of the perinephric cystic mass, and improved duodenal dilation (Figures 4 and 5). NG tube was removed, and G-tube feeds were resumed on Day 5. On Day 7, patient was discharged home with PCN and planned follow-up in four weeks for further surgical planning of stone removal.

Patient was brought for ureteroscopy and laser lithotripsy with Urology 4 weeks later. Intra-operatively, the UVJ stone was no longer visualized and was assumed to have passed spontaneously prior to intervention, which was later confirmed with post-op imaging. Retrograde pyelogram revealed severe J-hooking of the proximal ureter that was unable to be traversed with a guidewire. No ureteral stent was placed and the PCN was placed to drainage. Patient was later seen in clinic 3 weeks later and had the PCN capped. Patient passed a PCN capping trial with

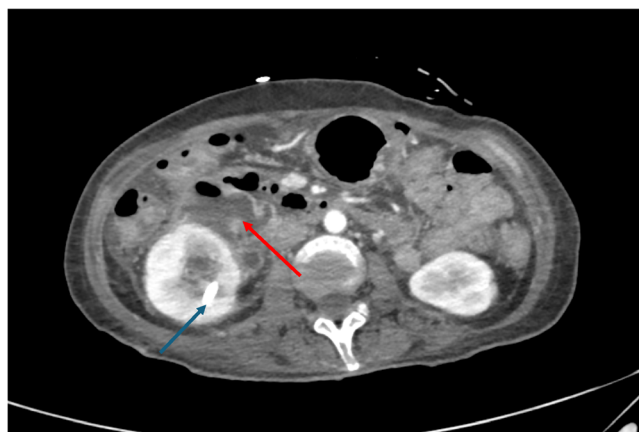


FIGURE 4 Computed tomography abdomen/pelvis with contrast (axial view) on Day 4 of presentation. Reduced hydroureteronephrosis (red arrow) after placement of percutaneous nephrostomy (blue arrow).



FIGURE 5 Computed tomography abdomen/pelvis with contrast (axial view) on Day 4 of presentation. Reduced hydroureteronephrosis (red arrow) after percutaneous nephrostomy placement.

subsequent removal of PCN 2 weeks later. Table outlining the timeline of the patient treatment course from the day of presentation up to the last patient follow-up can be found below (Table 1).

TABLE 1 Timeline of the patient's clinical course, diagnostic evaluations, and therapeutic interventions from initial presentation through final follow-up.

Timeline	Event/clinic findings	Intervention
Hospital day 1	Arrival to ED. Initial imaging showed 0.7 cm obstructing right UVJ stone, dilated urinary collecting system, gastric and duodenal obstruction. Urinalysis significant for leukocytosis, pyuria, bacteriuria. Labs showing normal kidney function.	Urology consulted recommended IR placement of PCN. NG tube placed for decompression. Broad-spectrum antibiotics initiated.
Hospital day 2-3	Urology recommending PCN placement and improvement of acute illness before definitive stone management.	Continued bowel decompression and broad-spectrum antibiotics. Placement of right-sided PCN by IR.
Hospital day 4-5	Repeat imaging demonstrated improvement of hydroureteronephrosis dilation, resolution of gastrointestinal dilation. Nephrostomy urine cultures positive for <i>Proteus</i> .	Broad-spectrum antibiotics discontinued, trimethoprim-sulfamethoxazole started.
Hospital day 8	Patient with resolution of symptoms.	Discharged with urology appointment in 4 weeks.
4 weeks after hospital discharge	Patient continuing to do well, without fevers, flank pain. PCN in place with good urine output.	Plan for ureteroscopy and laser lithotripsy in 3 weeks.
3 weeks after initial clinic appointment	Presented for surgery. Previously seen right UVJ stone had spontaneously passed. Retrograde pyelogram showing severe J-hooking of right proximal ureter that was unable to be traversed with guidewire. No ureteral stent placed and PCN capped.	Follow-up in clinic for PCN removal in 2 weeks.
2-week follow-up appointment	Patient urinating normally, no fevers, chills, nausea, vomiting. Passed PCN capping trial. Normal renal function.	PCN removed.

Abbreviations: ED, emergency department; IR, interventional radiology; NG, nasogastric; PCN, percutaneous nephrostomy; UVJ, ureterovesical junction.

3 | DISCUSSION

This case report outlines the variability and complexity in the presentation and clinical management of obstructive nephrolithiasis. The patient's initial CT showed impressive gastric and duodenal dilation due to compression from the adjacent 8.6 cm complex perinephric cystic structure (Figure 3), which is supported by the patient's presenting symptoms of nausea and vomiting. It was initially unclear whether this structure was the severely dilated collecting system, urinoma, abscess, or neoplasm on CT. Given the rapid decompression of this cystic structure upon placement of PCN (Figures 4 and 5) and the severe J-hooking of the proximal ureter seen on retrograde pyelogram, it was determined that the perinephric cystic structure was a severely dilated and torturous proximal ureter rather than a urinoma, abscess, or neoplasm. There was also no extravasation of contrast during the retrograde pyelogram, making forniceal rupture and urinoma less likely (Figure 6). This presentation of gastrointestinal obstruction and symptom resolution with PCN placement is most similarly seen in one other case report, albeit, not in the setting of nephrolithiasis^[8]. This patient had megaureter secondary to obstructive ureteral stone resulting in mass effect on adjacent organs (Figure 1).

The decision for PCN instead of retrograde stent placement was multifactorial. Because the patient had high aspiration risk with general anesthesia, PCN was opted for as it only requires moderate sedation with high success and low complication rates^[11].

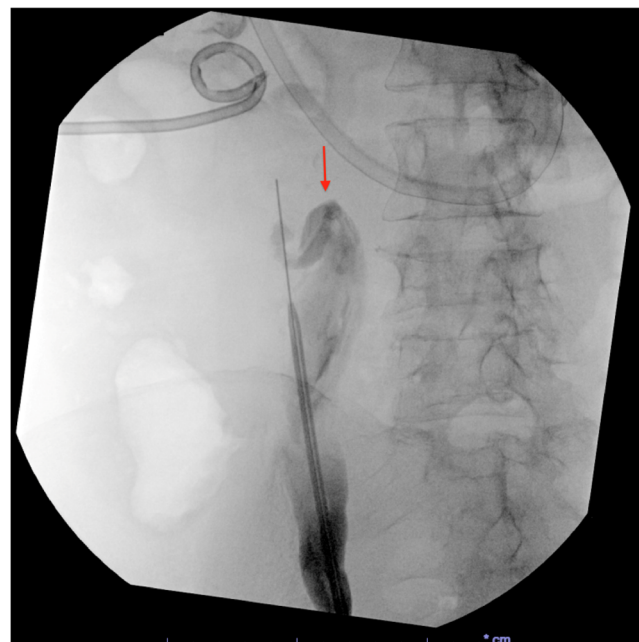


FIGURE 6 Retrograde pyelogram demonstrating right-sided hydroureter and severe J-hooking at the proximal ureter (red arrow).

While stent placement for an obstructing distal ureteral stone is within the American Urological Association guidelines for nephrolithiasis^[12], the severity of hydroureteronephrosis in this patient was not likely to improve with stenting. In addition, the stone appeared to be possibly impacted, as

evidenced by the ureteral wall thickening measuring 2.1 mm on CT. Although ureteral wall thickness of 3.35 mm or greater is associated with failure of initial retrograde stent placement, which is thicker than the ureteral wall described in our patient^[13], the patient's aspiration risk pushed the decision for PCN placement. Surprisingly, the 0.7 cm UVJ stone had spontaneously passed by the time patient was brought for ureteroscopy. Although ureteral stones greater than 0.5 mm in size have a lower likelihood of spontaneous passage^[14], ureteral thickness less than 2.3 mm has been described as the optimal cutoff to predict spontaneous passage^[15], which is supported by our patient's case.

Nephrolithiasis is a common urologic pathology that can present symptomatically or be asymptomatic and discovered incidentally. Symptomatic presentation can be variable; however, classic symptoms include hematuria, flank pain, dysuria, urgency, nausea, or vomiting^[4]. Additionally, nephrolithiasis may lead to a variety of complications including hydronephrosis, forniceal rupture resulting in urinoma, pyelonephritis, or abscess^[4-6]. Such complications may lead to patient clinical decompensation and result in more urgent intervention for nephrolithiasis.

There is limited literature describing bowel obstruction caused by secondary obstructive megaureter^[7-9]. No case reports of bowel obstruction in the setting of nephrolithiasis can be found from literature review, underscoring the importance of documenting this presentation. This case study illustrates the variability of presentation among patients with nephrolithiasis and subsequent complications in a complex patient with multiple comorbidities. In situations where a patient is nonverbal, conventional symptoms of flank pain and dysuria cannot be reliably elicited to evaluate for obstructive nephrolithiasis. However, when a patient presents with nausea and vomiting, the differential diagnosis of gastrointestinal obstruction must always be investigated, and the possibility of secondary gastrointestinal obstruction due to megaureter should not be overlooked.

Thoughtful consideration of the differential diagnosis is necessary to drive medical decision making towards the proper interventions to prevent clinical decompensation. As seen in this case, prompt resolution of the dilated collecting system and gastrointestinal obstruction was achieved with PCN placement alone, without requiring more invasive interventions in the acute setting. This case study is the first to document secondary obstructive megaureter from nephrolithiasis resulting in gastrointestinal obstruction.

AUTHOR CONTRIBUTIONS

Jacob Galan and Shannon Leung participated in data acquisition, data analysis, and drafting of the manuscript. Shannon Leung also participated in the study concept and design. Steven E. Canfield participated in study supervision, validation, and critical revision of the manuscript.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ETHICS STATEMENT

This case report was conducted in accordance with institutional ethical standards and the Declaration of Helsinki. Written informed consent for publication of clinical details and images was obtained from the patient.

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