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Infected Spontaneous Renal Fornix Rupture Secondary to Obstructing Ureteral Calculus: Role of Urgent DJ Stenting and Culture-Guided Therapy

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Abstract

Background: Spontaneous renal fornix rupture (SRF) is an uncommon, potentially life-threatening urological emergency resulting from a precipitous rise in intraluminal pressure due to acute upper urinary tract obstruction. In infected, obstructed systems, the clinical scenario is heavily compounded by the risk of rapid urosepsis, retroperitoneal abscess formation, and permanent parenchymal damage. Urgent, minimally invasive decompression remains the cornerstone of management to protect the renal unit and interrupt the septic cascade. **Case Presentation:** A 43-year-old female presented to the emergency department with acute-onset left flank pain, high-grade fever, and constitutional symptoms. Laboratory evaluations demonstrated severe leukocytosis (WBC: 17.6 × 10^3/μL), an elevated C-reactive protein (CRP: 27.9 mg/L), and acute kidney injury with a presumed Stage 1 AKI indicated by a serum creatinine of 1.32 mg/dL against an unknown baseline. Contrast-enhanced computed tomography (CECT) urography with delayed excretory phase imaging demonstrated left-sided obstructive nephropathy secondary to an obstructing proximal ureteral calculus causing moderate hydroureteronephrosis, and active contrast extravasation from the superior calyceal fornix into the perirenal space, confirming the formation of a localized urinoma. Empiric antibiotic therapy was adjusted following urine cultures that grew *Escherichia coli* resistant to ceftriaxone but sensitive to carbapenems. Due to the high risk of systemic sepsis from an infected, obstructed urinary system, an urgent cystoscopy was performed, and a 6 French, 26 cm Double-J (DJ) stent was retrogradely

deployed under spinal anesthesia alongside the administration of intravenous Ertapenem. Following decompression, the patient showed rapid clinical improvement: acute pyrexia and severe flank pain resolved within 24 hours, while overall laboratory values and serum creatinine normalized to 0.8 mg/dL by day 3. The DJ stent was maintained in situ for 6 weeks during which the underlying calculus passed spontaneously. A post-removal retrograde pyelogram (RPG) confirmed complete restoration of pelvicalyceal architectural integrity without residual urine leakage or stricture formation. **Conclusion:** Spontaneous renal fornix rupture accompanying obstructive pyelonephritis demands swift diagnosis and immediate surgical decompression. Decompression should never be delayed by the false assumption that spontaneous rupture provides adequate systemic drainage. CECT urography with a delayed excretory phase is the diagnostic standard of care, while follow-up retrograde studies are essential to objectively confirm functional healing of the upper tract.

Keywords

Renal Fornix Rupture, Urinoma, Double-J Stent, Obstructive Pyelonephritis, CT Urography

1. Introduction

Spontaneous renal fornix rupture (SRF) is defined as a non-traumatic, non-iatrogenic disruption of the pyelocaliceal collecting system that leads to the extravasation of urine into the perinephric or retroperitoneal space. The vast majority of SRF cases (approximately 74%) are secondary to acute upper urinary tract luminal occlusion, most commonly precipitated by impacted ureteral or calyceal calculi [1] [2].

Pathophysiology and Pressure Mechanics

Physiologically, an acute mechanical obstruction causes an immediate stagnation of urine flow, triggering a significant, retrograde increase in hydrostatic pressure within the renal pelvis and calyces. According to Laplace's Law, when the expanding intraluminal pressure outpaces the structural tensile strength of the delicate calyceal fornices, a macro-perforation occurs at the reflection point of the minor calyces. This rupture acts as a temporary, endogenous decompressing "safety valve" that drops intrapelvic pressure and can paradoxically mitigate acute renal colic.

However, when the obstructed urinary column is colonized by pathogenic bacteria, this protective mechanism is lost. The extravasation of infected urine into the highly vascularized retroperitoneal spaces significantly escalates morbidity, predisposing the patient to urosepsis, perirenal abscess formation, retroperitoneal fibrosis, and irreversible ischemic nephron loss. Management strategies vary from strict conservative observation in small, sterile leaks to aggressive intervention in

complex scenarios. This case report highlights the critical role of timely endourological decompression and culture-targeted antimicrobial stewardship in managing an infected SRF secondary to obstructive urolithiasis.

2. Case Presentation

2.1. History and Clinical Examination

A 43-year-old female with no prior history of urolithiasis or renal anomalies presented to the emergency department at Desouk General Hospital with an acute, unremitting left-sided flank pain of 24 hours' duration. The pain was accompanied by high-grade fever, chills, nausea, and general malaise.

On physical examination, the patient was febrile (temperature: 38.8˚C), tachycardic (heart rate: 108 bpm), and normotensive (blood pressure: 115/75 mmHg). Extreme tenderness was elicited upon deep palpation of the left lumbar region and at the left costovertebral angle. There were no signs of generalized peritonitis, and no palpable abdominal masses or distended bladder were noted. Based on the temperature of 38.8˚C, heart rate of 108 bpm, and profound leukocytosis, the patient met the clinical criteria for Systemic Inflammatory Response Syndrome (SIRS) secondary to an acute urological infection.

2.2. Diagnostic Workup

Initial laboratory evaluations revealed a profound inflammatory response and a decline in renal function:

- White Blood Cell (WBC) Count: 17.6 × 10^3/μL (normal range: 4.0 - 11.0 × 10^3/μL) with an absolute neutrophilic shift.
- C-Reactive Protein (CRP): 27.9 mg/L (normal: <5.0 mg/L).
- Serum Creatinine: 1.32 mg/dL. While the patient's prior baseline creatinine was unknown, this elevated presentation value indicated a presumed Stage 1 Acute Kidney Injury (AKI) secondary to acute upper urinary tract obstruction.
- Urinalysis: Gross microscopic hematuria (>50 RBCs/HPF) and pyuria (>30 WBCs/HPF); positive for nitrites and leukocyte esterase.

An urgent transabdominal urological ultrasound was performed, revealing a left-sided obstructive pathology, severe distention of the extra-renal pelvis, and an irregular, hypoechoic fluid collection within the left perinephric space measuring approximately 4.5 × 3.2 cm.

To definitively characterize the collection and delineate the anatomy of the upper urinary tract, contrast-enhanced computed tomography (CECT) urography was performed. The corticomedullary and nephrographic phases confirmed moderate left hydroureteronephrosis secondary to an obstructing proximal ureteral calculus measuring 6 mm. Crucially, the delayed excretory phase (obtained 10 to 15 minutes post-injection) demonstrated a clear tear in the superior calyceal anatomy with active, high-attenuation contrast medium pooling outside the renal parenchyma into the perirenal space, confirming an acute urinoma secondary to spontaneous fornix rupture (**Figure 1**).

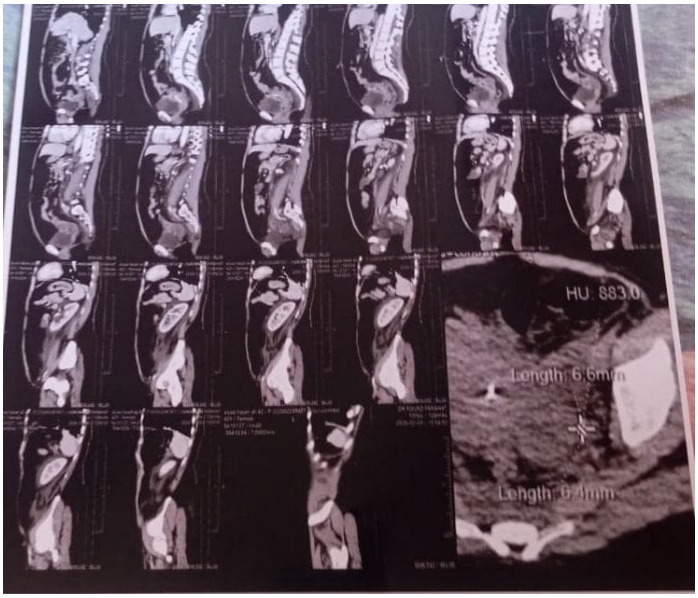

Figure 1. Axial and Coronal views of CECT Urography showing contrast extravasation from the left upper pole calyceal fornix into the retroperitoneum, establishing a localized urinoma.

Empiric broad-spectrum antibiotic therapy was initiated with third-generation cephalosporins (Ceftriaxone 2 g IV q24h). Concurrently, peripheral blood cultures were drawn, which subsequently showed no growth after 5 days. Clean-catch urine culture and sensitivity testing isolated *Escherichia coli* (>10^5 CFU/mL) displaying an extended-spectrum beta-lactamase (ESBL) phenotype, characterized by absolute resistance to Ceftriaxone but marked susceptibility to Ertapenem and Levofloxacin.

2.3. Interventional Management and Follow-Up

Given the volatile combination of an obstructed, infected upper tract, active urine extravasation, severe SIRS criteria, and an escalating AKI, immediate endourological intervention was pursued. Under spinal anesthesia, the patient was placed in the lithotomy position. Rigid cystoscopy was performed, and a retrograde ureteropyelogram was deliberately avoided at the start of the procedure to prevent forcing infected urine further into the retroperitoneum under high pressure.

Under fluoroscopic guidance, a 0.035-inch hydrophilic guidewire was successfully negotiated past the obstructing ureteral stone. A 6 French, 26 cm polyurethane Double-J (DJ) stent was retrogradely deployed across the site of rupture to bridge the defect and bypass the obstruction. Cloudy urine was drained from the bladder and sampled for secondary intraoperative culture. This secondary intraoperative specimen subsequently confirmed the presence of the identical ESBL-producing *E. coli* strain found in the initial clean-catch sample.

Postoperatively, the patient was transferred to the inpatient ward, and antimicrobial therapy was narrowed to intravenous Ertapenem (1 g once daily) based on the sensitivity profile. The clinical response was prompt:

- Within 24 hours: The patient became completely afebrile, and narcotic requirements for flank pain ceased entirely.
- Within 72 hours: Overall inflammatory markers and metabolic parameters stabilized drastically (WBC: $8.4 \times 10^3/\mu L$), and renal parameters normalized completely, with serum creatinine stabilizing at its true baseline of 0.8 mg/dL.
- A chronological summary of the patient's entire clinical timeline and lab transitions is detailed in Table 1.

Table 1. Clinical and laboratory timeline of the patient's management course.

Timeframe	Clinical Event/Intervention
Day 0 (Admission)	Presentation with left flank pain, fever (38.8˚C), severe leukocytosis, and presumed Stage 1 AKI.
Day 0 (Hours 2 - 4)	Ultrasound and CECT imaging completed; blood/urine cultures drawn; empiric IV Ceftriaxone initiated.
Day 1 (Hours 12 - 18)	Urine culture identifies ESBL *E. coli*; treatment immediately escalated to targeted IV Ertapenem.
Day 1 (Urgent)	Patient undergoes rigid cystoscopy and urgent retrograde 6Fr DJ stenting; intraoperative cultures sampled.
Day 2 (Post-op Day 1)	Rapid clinical improvement: patient becomes completely afebrile; acute narcotic requirements cease.
Day 4 (Post-op Day 3)	Complete laboratory resolution: creatinine normalizes to 0.8 mg/dL; WBC drops to $8.4 \times 10^3/\mu L$.
Day 5	Patient clinically stable for discharge; transitioned to a 10-day course of targeted oral Levofloxacin.
Week 6	Elective DJ stent removal; dynamic follow-up Retrograde Pyelogram (RPG) confirms closure and stone passage.
Month 3	Final outpatient follow-up; patient is fully asymptomatic with sterile urine and normal renal function.

The patient was discharged on day 5 post-intervention with a 10-day course of oral Levofloxacin (500 mg once daily). Although the strain exhibited an ESBL phenotype, the in-vitro sensitivity profile explicitly confirmed complete susceptibility to fluoroquinolones, making oral Levofloxacin an ideal, highly bioavailable choice for oral step-down stewardship.

The DJ stent was kept in place for 6 weeks to ensure complete structural remodeling and sealing of the disrupted calyx. At the 6-week mark, the patient returned for elective stent removal. Following cystoscopic retrieval of the DJ stent, an on-table retrograde pyelogram (RPG) was performed. The RPG demonstrated a completely intact left pelvicalyceal system with normal calyces, unobstructed downflow of contrast into the bladder, and an absolute absence of contrast extravasation or infundibular stricture formation (Figure 2). Fluoroscopic inspection and follow-up imaging at this time confirmed that the original 6 mm obstructing ure-

teral calculus had passed spontaneously, likely displaced or fractured during initial guidewire negotiation and subsequent internal stenting, leaving the patient entirely stone-free.

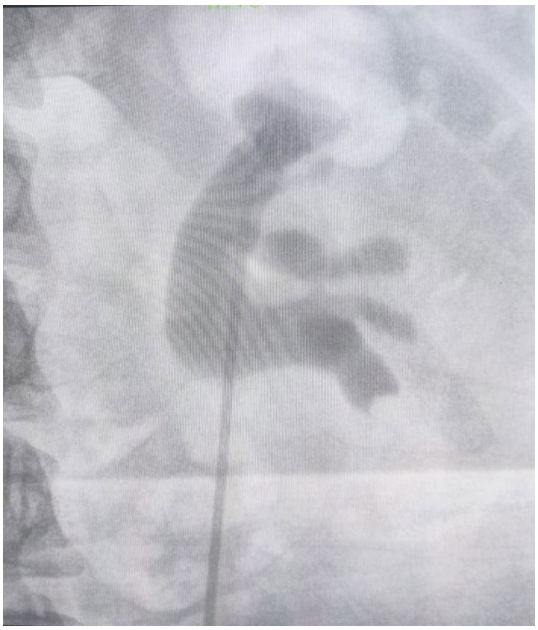

Figure 2. Retrograde Pyelogram (RPG) obtained after 6 weeks of stenting, demonstrating a normal pelvicalyceal architecture with complete resolution of the fornix leak and stone-free status.

At her 3-month outpatient follow-up, the patient remained completely asymptomatic, with sterile urine cultures and preserved renal function.

3. Discussion

Spontaneous renal fornix rupture is a critical urological condition that presents a diagnostic and therapeutic challenge to clinicians. While mechanical occlusion by urolithiasis is the predominant trigger, other potential causes include retroperitoneal malignancies, strictures, sloughed papillae, benign prostatic hyperplasia, and pregnancy-induced upper tract compression. Regardless of the underlying cause, the primary pathophysiological event is an acute rise in intrarenal pressure.

The management of SRF remains unstandardized due to its low clinical incidence. Historically, conservative management—consisting of bed rest, adequate analgesia, oral antibiotics, and medical expulsive therapy (MET)—has been advocated for small, sterile, non-obstructive caliceal tears. Studies show that up to 57.5% of uncomplicated, small stones (<5 mm) with minimal urine extravasation can be safely managed without invasive intervention, allowing the urinoma to spontaneously resorb [3] [4].

3.1. Management Paradigms of Infected SRF

However, the clinical paradigm changes completely when there is an active infec-

tion, a large urinoma, persistent pain, or worsening renal function. In this case, the patient presented with all four high-risk indicators: severe urosepsis meeting full SIRS criteria, acute kidney injury, a sizable perirenal urinoma, and an ESBL-producing *E. coli* infection. Waiting for spontaneous stone passage or relying solely on antibiotics in an obstructed, infected system carries an unacceptably high risk of septic shock and perirenal abscess formation.

Immediate urinary diversion is the standard of care for infected systems to decompress the upper collecting system. Decompression can be achieved via either retrograde Double-J stenting or percutaneous nephrostomy (PCN) tube placement. In our patient, retrograde DJ stenting was chosen because it effectively bridges the site of the caliceal tear, redirects urine flow internally, and eliminates the elevated back-pressure that drives retroperitoneal extravasation. This internal diversion allows the torn fornix to heal cleanly through secondary intention over several weeks.

3.2. Diagnostic and Follow-Up Imaging Modalities

This case also underscores the diagnostic superiority of CECT urography with delayed-phase imaging [5]. While initial ultrasound is an excellent, radiation-free tool to detect hydronephrosis and large fluid collections, it lacks the spatial resolution to accurately locate the perforation site or differentiate a sterile urinoma from an acute hematoma or abscess [5]. Delayed excretory imaging (typically 10 - 15 minutes post-contrast) is crucial, as it shows high-density contrast media actively mixing with the pooling urine in the retroperitonium, providing a clear map of the anatomical leak.

Finally, performing a retrograde pyelogram at the time of stent removal is highly recommended. It serves as an objective, dynamic verification tool to confirm that the caliceal tear has healed completely and that no late-stage ischemic complications, such as infundibular or ureteral strictures, have developed.

4. Conclusion

Spontaneous renal fornix rupture occurring alongside obstructive pyelonephritis is a high-acuity urological emergency. Endourological decompression via urgent Double-J stenting, paired with culture-guided antibiotic therapy, is highly effective for stabilizing patients and resolving infection. The spontaneous decompression caused by a fornix rupture must not delay surgical intervention when infection is present. Delayed-phase CECT remains the gold standard for initial diagnosis, while a post-recovery retrograde pyelogram is essential to confirm complete anatomical and functional healing of the upper urinary tract.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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