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Iliopsoas involvement complicating longstanding nephrolithiasis with chronic infection: a three-patient case series reconsidering “malignant lithiasis”

Donghui Li
Affiliated Haikou Hospital of Xiangya Medical College, Central South University
Meijiang Meng
Affiliated Haikou Hospital of Xiangya Medical College, Central South University
Xianlai Yin
Affiliated Haikou Hospital of Xiangya Medical College, Central South University
Zhiming Bai
Affiliated Haikou Hospital of Xiangya Medical College, Central South University
Haixia Xu
Affiliated Haikou Hospital of Xiangya Medical College, Central South University
Cai Lv
lvcai815@163.com

Affiliated Haikou Hospital of Xiangya Medical College, Central South University

Case Report

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Abstract

Background

Severe complications of longstanding nephrolithiasis with chronic infection remain clinically relevant despite continuing advances in endourological techniques. This phenotype may extend beyond the collecting system, resulting in extensive retroperitoneal infection with extrarenal extension, irreversible renal functional loss, and, in rare instances, occult malignancy. Reconsidering the historical descriptor “malignant lithiasis” may help frame this aggressive clinical trajectory, particularly in patients with delayed presentation or limited access to timely care.

Case presentation:

We report three cases of longstanding nephrolithiasis with chronic infection with iliopsoas involvement. Case 1 was a 50-year-old woman with perinephric and iliopsoas abscesses, with inferior tracking along the iliopsoas compartment into the obturator region. Case 2 was a 53-year-old man with perinephric and iliopsoas abscesses and inflammatory involvement extending to the colon. Case 3 was a 59-year-old man with a large renal abscess who underwent nephrectomy to achieve definitive source control for uncontrolled infection, revealing an incidental diagnosis of renal pelvic squamous cell carcinoma. Across cases, infection was recurrent or refractory, temporizing measures (e.g., percutaneous drainage and/or nephrostomy) did not achieve sustained definitive source control, and two patients developed non-functioning kidneys requiring nephrectomy after infection control.

Conclusions

These cases illustrate an aggressive phenotype of longstanding nephrolithiasis with chronic infection, consistent with “malignant lithiasis” as a marker of clinical severity rather than malignant histology. Recognizing red flags—recurrent or refractory infection, imaging evidence of extrarenal extension (e.g., iliopsoas and perinephric involvement), and progressive functional decline—may support timely escalation to definitive source control. Routine histopathology of nephrectomy specimens is critical to minimize missed malignancy in stone-related non-functioning kidneys.

Background

Nephrolithiasis is a common chronic condition with a high recurrence rate and a substantial global health burden. Approximately 11% of the global population is affected by urolithiasis, with marked geographic variation in prevalence [1]. Although minimally invasive techniques have improved stone clearance, recurrence remains common and long-term prevention and follow-up are often required [2]. In regions with large populations and uneven access to healthcare, nephrolithiasis-related complications—

including severe infection and secondary malignancy—continue to be encountered in routine clinical practice [3].

In the era of open surgery, complex staghorn calculi associated with persistent infection and a destructive clinical course were described as “malignant lithiasis” (also termed “malignant nephrolithiasis” in historical reports) to convey clinical severity and poor outcomes rather than malignant histology [4]. Despite continuing advances in endourology, similar high-risk phenotypes persist, particularly in patients with longstanding stone disease complicated by chronic infection, delayed presentation, or inadequate follow-up, in whom recurrent or refractory infection, progressive stone growth, and renal functional decline may still occur. In this context, reconsidering this historical descriptor may help reinforce clinical vigilance and support risk stratification and timely escalation to definitive source control.

However, contemporary reports that systematically characterize this aggressive phenotype—namely, longstanding nephrolithiasis with chronic infection complicated by extensive extrarenal extension with retroperitoneal abscess formation and/or occult malignancy—remain limited.

Case presentation

Case 1

A 50-year-old woman was admitted on 27 July 2020 with recurrent right flank pain for 2 years, worsened over the preceding 3 months, with right lower-limb distension and pain and no recent fever. She had a 10-year history of right renal calculi without structured follow-up and denied tuberculosis exposure. At an outside hospital, contrast-enhanced CT excluded an overt renal malignant lesion but raised suspicion of genitourinary tuberculosis (GU-TB); empiric anti-tuberculous therapy for 2 months produced no improvement.

On admission, temperature was 36.2 °C. Examination showed right costovertebral angle tenderness and a firm, tender mass extending from the right lower abdomen to the right inguinal region and medial thigh. Laboratory tests showed WBC 13.20 × 10^9/L, hemoglobin 96 g/L, and serum creatinine 72 μmol/L. Urine culture and cytology were negative; interferon-γ release assay (IGRA) and serum anti-Mycobacterium tuberculosis IgG were negative.

Non-contrast CT (NCCT) demonstrated multiple right renal calculi with extensive perinephric inflammatory change and abscess formation, tracking inferiorly through the inguinal canal to the proximal thigh near the obturator region (Fig. 1). No adjacent organ involvement was reported, and GU-TB could not be excluded initially.

Aspiration from the inguinal abscess was negative for Mycobacterium tuberculosis by quantitative polymerase chain reaction (PCR) and smear microscopy, whereas culture grew *Proteus mirabilis*, excluding GU-TB. Incision and drainage (I&D) of the inguinal abscess was performed, followed by right

percutaneous nephrostomy (PCN). One month later, the nephrostomy drained 5–15 mL/day of turbid yellow pus and the inguinal drain produced 50–80 mL/day of purulent fluid. Repeat CT showed only slight reduction of the iliac collection, with no significant change in the perinephric, psoas major, or iliopsoas lesions. Given inadequate response to drainage and antibiotics, percutaneous nephrolithotomy (PCNL) was performed to reduce stone burden and improve drainage for source control.

Intraoperatively, a large staghorn calculus occupied the renal pelvis and calyces with copious pus and necrotic tissue; the renal pelvic outlet could not be identified, and both antegrade and retrograde ureteral stent placement failed, suggesting complete obstruction. On postoperative day 1, she developed fever and hypotension consistent with urosepsis and septic shock, which improved with intensive supportive care. Infrared spectroscopic analysis confirmed a struvite (magnesium ammonium phosphate) stone. She was discharged with the nephrostomy tube in situ.

She was readmitted on 19 April 2021. Contrast-enhanced CT on 20 April 2021 showed marked right renal atrophy with residual calculi and substantial regression of prior extrarenal extension (Fig. 2). Renal dynamic scintigraphy showed left GFR 88.65 mL/min and right GFR 6.36 mL/min, consistent with a non-functioning right kidney. After infection control, open right nephrectomy with retroperitoneal abscess debridement was performed on 25 April 2021. Histopathology showed chronic inflammation and necrosis; periodic acid–Schiff (PAS) and acid-fast stains were negative. She was discharged 2 weeks postoperatively and remained clinically well at approximately 5-year follow-up, with normal serum creatinine.

Case 2

A 53-year-old man was admitted on 19 February 2024 with persistent left flank pain for 1 month and no recent fever. He had untreated left renal calculi diagnosed approximately 30 years earlier and denied tuberculosis exposure. Temperature was 36.3 °C, and examination showed left costovertebral angle tenderness. Laboratory tests showed WBC 7.66 × 10^9/L, hemoglobin 119 g/L, platelets 439 × 10^9/L, and serum creatinine 96 μmol/L. Urinalysis and urine culture were negative; repeated urine cytology was negative. IGRA and serum anti-Mycobacterium tuberculosis IgG were negative.

NCCT demonstrated multiple left renal calculi with marked renal atrophy and a perinephric abscess involving the ipsilateral psoas major muscle, abutting the splenic flexure of the colon (Fig. 3). Concomitant non-obstructing right renal calculi were present. Contrast-enhanced CT showed no definite renal malignant lesion, and GU-TB remained in the differential diagnosis. Ultrasound-guided perinephric drainage yielded 5–20 mL/day of purulent fluid. Quantitative PCR and smear microscopy for Mycobacterium tuberculosis were negative, whereas culture grew *Bacteroides fragilis*, excluding GU-TB. Targeted antimicrobial therapy was initiated with symptomatic improvement; drainage output gradually ceased and the catheter dislodged spontaneously without complication.

On 17 June 2024, he was readmitted with anorexia and progressive anemia (hemoglobin 86 g/L), without fever or urinary symptoms. Renal dynamic scintigraphy showed right GFR 63.24 mL/min and left GFR 6.32 mL/min, consistent with a non-functioning left kidney. Repeat CT on 19 June 2024 showed no meaningful reduction in the perinephric or psoas collections. After multidisciplinary discussion, open left nephrectomy with retroperitoneal abscess debridement was performed on 25 June 2024; dense adhesions at the splenic flexure necessitated partial colectomy. Histopathology was consistent with a calculous renal abscess. He recovered uneventfully and was discharged 10 days postoperatively. One month later, he underwent holmium laser lithotripsy for right renal calculi. At approximately 18 months postoperatively, he remained clinically well without recurrent pain; serum creatinine was normal and there was no recurrence of right renal stones.

Case 3

A 59-year-old man was admitted on 11 October 2024 with intermittent right flank pain for 6 months, worsened over the preceding month, with purulent urine and abdominal distension, without recent fever. He had a >20-year history of nephrolithiasis with multiple prior stone-related procedures and residual right renal calculi; he denied tuberculosis. Temperature was 36.3 °C. Laboratory tests showed WBC 9.27 × 10^9/L, hemoglobin 109 g/L, platelets 419 × 10^9/L, and serum creatinine 169 μmol/L. Urinalysis showed RBC 86/μL and WBC 19,477/μL. Urine culture grew *Pseudomonas aeruginosa*, and repeated urine cytology was negative. Renal dynamic scintigraphy showed impaired bilateral renal function, predominantly affecting the right kidney (right GFR 10.92 mL/min; left GFR 29.42 mL/min).

Non-contrast and contrast-enhanced CT urography demonstrated multiple right renal calculi with severe hydronephrosis and pyonephrosis, with suspected involvement of the right psoas major muscle (Fig. 4A–D). Percutaneous nephrostomy (PCN) was performed; because the renal pelvis was small with markedly dilated calyces, two nephrostomy tubes were placed (upper calyx and mid-to-lower calyces) to optimize drainage. Despite drainage and targeted antimicrobial therapy, symptoms worsened, and repeat imaging showed persistent infection with suspected pyonephrosis and progressive retroperitoneal spread (Fig. 4E–G). Open right nephrectomy with psoas abscess resection/debridement was therefore performed to achieve definitive source control for uncontrolled infection.

Unexpectedly, histopathological examination of the nephrectomy specimen demonstrated a moderately to poorly differentiated squamous cell carcinoma (SCC) of the renal pelvis/upper urinary tract (Fig. 5). According to the final pathology report, the tumor invaded the renal sinus fat and perirenal adipose tissue with vascular tumor emboli, and metastatic carcinoma was present in 4 of 7 regional lymph nodes (pathological stage pT3N2Mx). Additional findings in the final pathology report not shown in Fig. 5 included focal sarcomatoid change, negativity for PAX8 and GATA3, and a high Ki-67 proliferation index (approximately 50%). He was discharged 2 weeks postoperatively but experienced rapid systemic progression and died 1 month later due to widespread metastases and multiorgan dysfunction.

Table 1. Clinical characteristics, interventions, and outcomes of three patients

Key elements	Case 1	Case 2	Case 3
Age / Sex	50 / Female	53 / Male	59 / Male
History	10-year renal calculi; no structured follow-up	~30-year left renal calculi; untreated	>20-year; multiple prior procedures
Extrarenal extension (CT)	Abscess tracking to inguinal canal and proximal thigh	Perinephric abscess involving ipsilateral psoas major; abutting splenic flexure	Suspected pyonephrosis and progressive retroperitoneal spread
Microbiology	Abscess: *P. mirabilis*	Abscess: *B. fragilis*	Urine: *P. aeruginosa*
Key interventions	I&D; PCN; PCNL → Open nephrectomy + debridement	Drainage → Open nephrectomy + debridement (partial colectomy)	PCN → Open nephrectomy + psoas abscess debridement
Split function (GFR, mL/min)	Right: 6.36; Left: 88.65	Right: 63.24; Left: 6.32	Right: 10.92; Left: 29.42
Pathology	Chronic inflammation/necrosis	Calculous renal abscess	Squamous cell carcinoma (SCC)
Outcome	Recovered; ~5-year follow-up	Recovered; ~18 months follow-up	Died 1 month postoperatively

Abbreviations: CT, computed tomography; GFR, glomerular filtration rate; I&D, incision and drainage; PCN, percutaneous nephrostomy; PCNL, percutaneous nephrolithotomy; SCC, squamous cell carcinoma.

Discussion

Across our three patients, longstanding nephrolithiasis with chronic infection followed a destructive trajectory: persistent stones complicated by infection; recurrent or smoldering infection; radiographically evident extrarenal extension; and progressive loss of renal function ultimately necessitating nephrectomy. In one case, systematic histopathological evaluation of the nephrectomy specimen revealed occult squamous cell carcinoma. These cases foreground three practical red flags that should trigger timely escalation of evaluation and management: (1) recurrent or refractory infection; (2) imaging evidence of extrarenal extension; and (3) progressive decline in split renal function. In this context, we reconsider “malignant lithiasis” as a historical descriptor of a high-risk clinical course rather than a diagnostic label implying malignant histology.

Urolithiasis is increasingly recognized as a chronic condition with a notable global health burden that requires long-term prevention and follow-up [1–2]. Despite advances in minimally invasive techniques, recurrence remains common, and severe infectious complications and renal destruction still occur in a subset of patients. In this report, we use “nephrolithiasis with chronic infection” to describe longstanding stones complicated by persistent or recurrent infection, regardless of stone composition; infection-related (struvite) stones are a common, but not exclusive, substrate for this phenotype. Clinically, this

phenotype can be operationally suspected when a prolonged stone history coexists with the red flags above—recurrent or refractory infection, imaging evidence of extrarenal inflammatory extension (e.g., perinephric or iliopsoas involvement), and/or progressive loss of ipsilateral renal function—prompting timely progression from temporizing measures to definitive source control.

Historically, in the era of open surgery, complex staghorn calculi associated with chronic infection were described as “malignant lithiasis” to convey clinical severity and poor prognosis rather than malignant histology [4]. Martínez-Piñeiro et al. emphasized the close association between staghorn calculi and infection, including a high likelihood of recurrence and progressive stone growth in the presence of persistent bacteriuria—features that can drive repeated infectious episodes and progressive renal damage when management is delayed or incomplete [4]. Subsequent work and contemporary reviews continue to underscore this infection–recurrence–progression paradigm and the risk of renal functional decline when definitive management is delayed or incomplete, supporting the ongoing clinical relevance of this historical descriptor even in the modern era [5]. In a related context, Pagano et al. used “malignant lithiasis” to describe cases with severely compromised renal function and heterogeneous postoperative renal functional trajectories, reinforcing that this label captured a function-threatening course rather than malignant histology [6]. Cystine stones, characterized by frequent recurrence and rapid progression, have also been discussed under this descriptor as a marker of a high-risk course rather than malignant histology [7]. More broadly, infection-related stones constitute a high-risk subtype, often characterized by rapid growth, persistent bacterial colonization, frequent recurrence, and an increased risk of functional decline or nephrectomy if definitive management is delayed [8–10]. Inadequate treatment may progress to recurrent pyelonephritis, urosepsis, and even death [11,12]. Infection/struvite stones have also been associated with increased postoperative sepsis after endourological stone surgery; these findings align with evidence linking positive stone culture to higher postoperative sepsis rates, supporting the concept that stone-associated bacterial reservoirs can amplify infectious risk [13,14]. Together, these data reinforce the value of recognizing high-risk phenotypes consistent with the historical concept of “malignant lithiasis” as a descriptor of destructive stone disease with potential irreversible renal functional loss.

Although nephrolithiasis is often perceived as benign, the mortality risk—while overall low—is real in severe or complicated disease. A systematic review spanning the past two decades summarized reported deaths related to kidney stone disease, identified sepsis as the leading cause, and noted that high stone burden and comorbidities were common among fatal cases [15]. These findings reinforce meticulous preoperative optimization and timely definitive management when stone disease is complicated by infection and systemic illness, consistent with guideline-based principles for surgical management of urinary stones [16].

Severe renal infection can be complicated by extensive perinephric/pararenal involvement on CT, and escalation to definitive management may be required [17,18]. In longstanding nephrolithiasis, extrarenal extension can mark one of the most severe trajectories of infection [17,18]. Perinephric infection may extend contiguously beyond the perirenal space into the iliopsoas compartment, resulting in iliopsoas

abscesses associated with notable morbidity; mortality has been reported in severe or advanced cases [19,20,23]. Cross-sectional imaging—particularly CT—defines disease extent and guides drainage and surgical planning [18,21]. Because iliopsoas abscesses are frequently secondary to adjacent or distant foci, careful evaluation of potential sources, including genitourinary infection, remains important [23]. Management emphasizes timely source control with appropriate antimicrobial therapy, often combined with image-guided drainage when feasible [18,22,24]. After stabilization, minimally invasive drainage and other source control strategies may be used to control infection and, in selected patients, facilitate subsequent definitive stone management [25]. When the affected kidney is irreversibly damaged, definitive source control may require surgical intervention, and nephrectomy should be considered in selected cases [10,18].

A further concern is the association between longstanding nephrolithiasis and upper urinary tract malignancy, particularly renal pelvic squamous cell carcinoma (SCC) [26–28]. Chronic irritation and infection are recognized drivers of squamous metaplasia and malignant transformation [28], yet preoperative diagnosis remains challenging because stones and inflammation can render clinical and radiological findings nonspecific [27,30]. Upper urinary tract malignancies have been incidentally detected during or after percutaneous nephrolithotomy (PCNL), underscoring that cancer may coexist with stone disease and be missed on preoperative assessment [29,30]. Epidemiological data also suggest an increased risk of upper urinary tract urothelial carcinoma (UTUC)—and, in some cohorts, renal cell carcinoma—in patients with nephrolithiasis [3,26,31]. Tumors with squamous differentiation tend to be high-risk [27], while xanthogranulomatous pyelonephritis or genitourinary tuberculosis can mimic malignancy and further complicate the differential diagnosis [32,36]. Accordingly, European Association of Urology (EAU) guidance supports diagnostic ureteroscopy (preferably without biopsy) when imaging and/or voided urine cytology are insufficient for diagnosis and/or risk stratification [33]. When suspicious upper urinary tract mucosal abnormalities are encountered, targeted biopsy has been advocated to facilitate timely diagnosis in selected high-risk patients [34]. Occult malignancy has also been reported in nephrectomy specimens from stone-related non-functioning kidneys, supporting systematic histopathological assessment in this setting [35].

These challenges may be amplified in socioeconomically disadvantaged settings, where barriers to timely care and fragmented follow-up are more prevalent. Evidence suggests that neighborhood socioeconomic disadvantage is associated with worse urinary risk factors for recurrent urolithiasis and reduced access to medical management, while adherence to guideline-based prevention and follow-up remains suboptimal in diverse low-resource populations [37,38].

Taken together, the clinical trajectory observed in our cases and the supporting literature support a pragmatic, escalation-oriented approach anchored on the three red flags above and operationalized as practical management considerations (summarized in the Practical points section below).

Practical points.

1. **Risk recognition:** Treat longstanding nephrolithiasis with chronic or refractory infection, extrarenal extension (e.g., perinephric abscess), or progressive renal functional decline as a high-risk clinical entity [10,39]. Socioeconomic disadvantage may further increase the likelihood of irreversible complications [37,38]. Early recognition should prompt timely intervention to optimize outcomes [10,16].
2. **Initial source control and exclusion of mimics:** After genitourinary tuberculosis has been reasonably excluded [36], prioritize early urinary drainage (retrograde ureteral stenting or percutaneous nephrostomy) and stabilization; defer definitive stone manipulation until sepsis is controlled and infection has been appropriately treated [10].
3. **Definitive management and functional triage:** Aim for complete stone clearance where feasible [9]. If clearance is incomplete, counsel patients and ensure structured follow-up, as residual fragments—especially in infection/struvite stone disease—may perpetuate bacteriuria, recurrent infection, and stone regrowth [9,11,40]. Quantify split renal function early (e.g., nuclear renography) and reassess after infection control to guide kidney-preserving versus extirpative surgery [10,40]. Consider timely nephrectomy for stone-related non-functioning kidneys to achieve definitive source control and to exclude occult malignancy, which can be challenging to detect preoperatively [35,40–42].
4. **Operative planning:** In advanced infection with extrarenal extension, anticipate dense adhesions, distorted hilar anatomy, and potential adjacent-organ involvement, which may increase procedural complexity and complications [32,42]. Meticulous imaging review, proactive planning, and multidisciplinary collaboration may improve safety [16,40].
5. **Malignancy vigilance:** Maintain a high index of suspicion for occult malignancy and perform systematic histopathological evaluation of nephrectomy specimens, particularly in stone-related non-functioning kidneys [26,31,35].

Limitations and future directions.

This small case series is subject to selection and referral biases and cannot quantify risk or establish causality between chronic infection, extrarenal extension, renal functional loss, and management decisions. Clinical heterogeneity (patient factors, microbiology, prior interventions, and timing of definitive surgery) may have influenced outcomes. “Nephrolithiasis with chronic infection” is used as a pragmatic clinical construct rather than a validated classification. Multicenter cohorts are needed to refine imaging- and function-based criteria, identify predictors of irreversible damage and extrarenal spread, and evaluate whether earlier escalation to definitive source control improves outcomes.

Conclusions

Longstanding nephrolithiasis with chronic infection may follow an aggressive, destructive course, leading to extensive retroperitoneal infection, renal functional loss, and, occasionally, occult malignancy [35,40]. Despite improved stone clearance with endoscopic and other minimally invasive approaches, recurrence remains substantial and delayed care continues to contribute to irreversible complications

[1,2,10,40]. Reconsidering “malignant lithiasis” as a historical descriptor of a destructive clinical trajectory—rather than a diagnostic label—may help clinicians recognize high-risk presentations earlier, including recurrent or refractory infection, imaging evidence of extrarenal extension, and progressive decline in split renal function, and prompt clinical vigilance, structured follow-up, and timely escalation to definitive source control [4,6,16,40]. Accordingly, in refractory or complex stone disease, timely progression to definitive treatment (including nephrectomy for non-functioning kidneys) and routine histopathology of nephrectomy specimens should be considered to support individualized decision-making and minimize missed malignancy [16,35].

Abbreviations

CT: Computed tomography; EAU: European Association of Urology; GFR: Glomerular filtration rate; GU-TB: Genitourinary tuberculosis; I&D: Incision and drainage; PCN: Percutaneous nephrostomy; PCNL: Percutaneous nephrolithotomy; SCC: Squamous cell carcinoma; UTUC: Upper urinary tract urothelial carcinoma.

Declarations

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Authors’ contributions

DL drafted the manuscript and reviewed the literature. MM and XY acquired the clinical data and contributed to drafting the manuscript. ZB interpreted the imaging findings and reviewed the literature. HX interpreted the pathological findings. CL revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Availability of data and materials

No datasets were generated or analysed during the current study.

Ethics approval and consent to participate

This case series was approved by the Ethics Committee of Haikou Municipal People’s Hospital.

Consent for publication

Written informed consent was obtained from the patients in Cases 1 and 2 for publication of this case series and any accompanying images. For Case 3, written informed consent for publication (including any accompanying images) was obtained from the patient's legal next of kin because the patient is deceased.

Competing interests

The authors declare that they have no competing interests.

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Figures

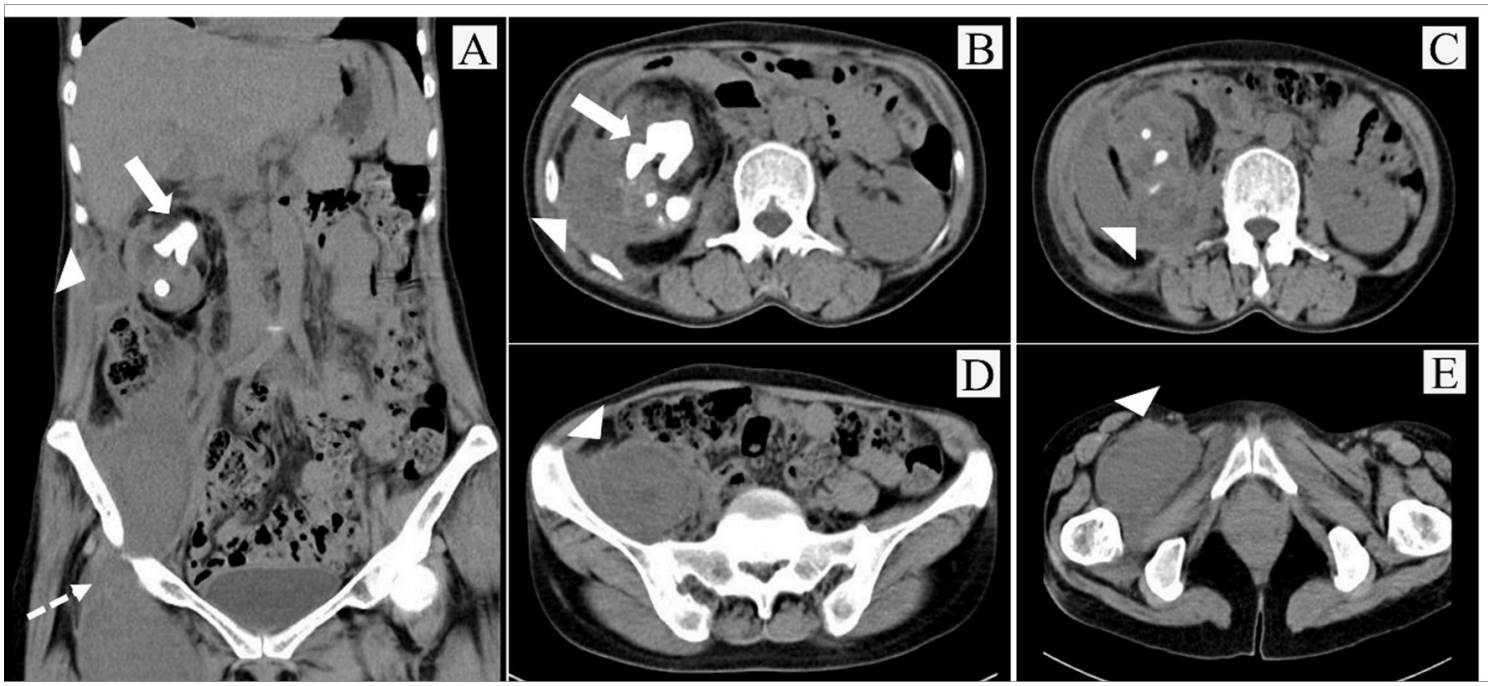


Figure 1

Non-contrast CT (NCCT) demonstrating extensive inferior tracking of perinephric inflammatory change/collection beyond the perinephric space in Case 1 (July 2020).
(A) Coronal NCCT shows a right staghorn calculus within the collecting system (solid arrow) with marked perinephric inflammatory change and a perinephric collection (arrowhead). The inflammatory change/collection tracks inferiorly along the retroperitoneum toward the pelvic outlet and proximal thigh (dashed arrow, indicating inferior extension).
(B) Axial NCCT at the renal level confirms the staghorn calculus (solid arrow) and the multiloculated perinephric collection (arrowhead).
(C) Axial NCCT slightly caudal to the renal hilum shows extension of inflammatory change/collection beyond the perinephric space along the iliopsoas compartment (arrowhead).
(D) Axial NCCT at the pelvic level demonstrates continued inferior tracking of inflammatory change/collection along the iliopsoas compartment (arrowhead).
(E) Axial NCCT at the level of the pelvic outlet/proximal thigh shows further inferior extension into the anteromedial proximal thigh, near the obturator region (arrowhead).

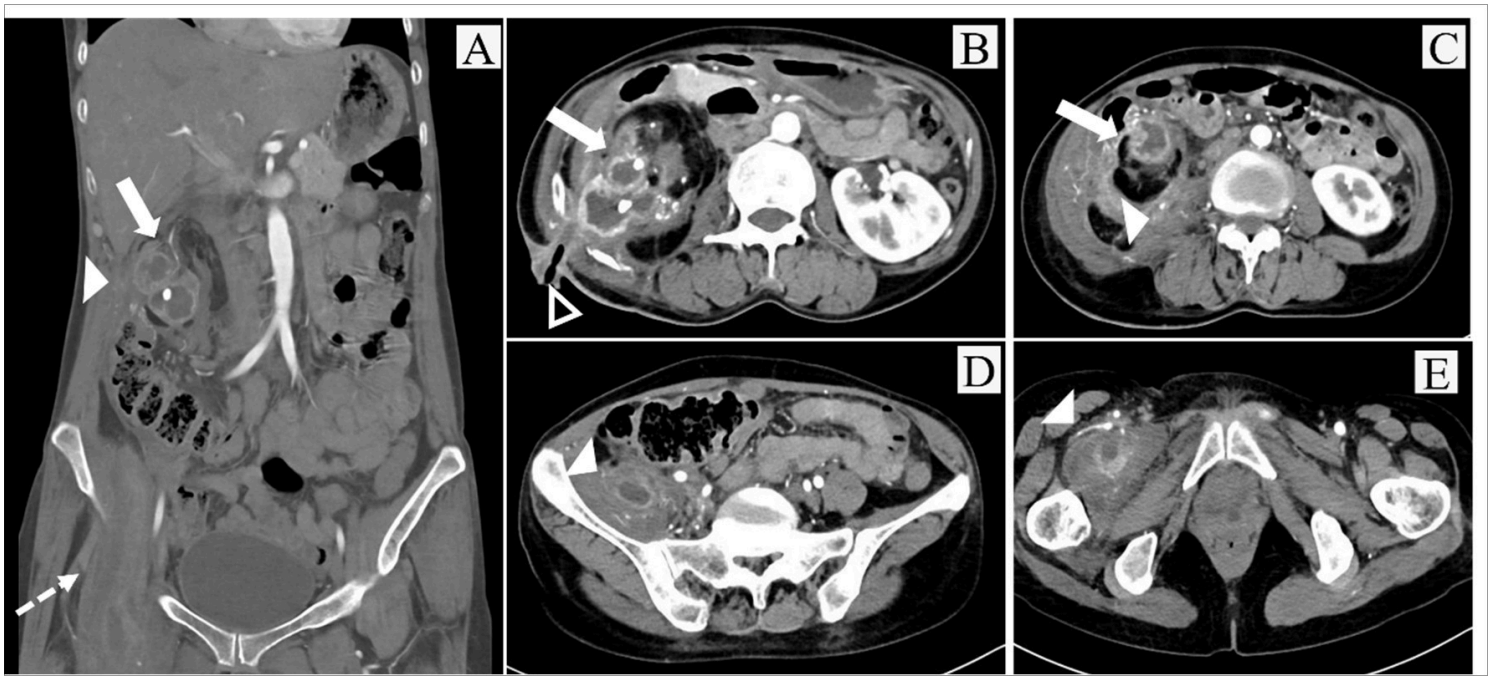


Figure 2

Follow-up contrast-enhanced CT demonstrating marked interval regression of extrarenal extension, with right renal atrophy and residual calculi in Case 1 (April 2021).

(A) Coronal contrast-enhanced CT shows marked right renal atrophy with residual calculi (solid arrow) and marked interval improvement of perinephric/retroperitoneal inflammatory change/collection (arrowhead), with regression of the previously inferiorly tracking collections toward the pelvic outlet and proximal thigh (dashed arrow, indicating prior inferior extent).

(B) Axial contrast-enhanced CT at the renal level shows marked right renal atrophy with residual calculi (solid arrow) and reduced perinephric inflammatory change/collection (arrowhead); an indwelling nephrostomy tube is present (open arrowhead).

(C) Axial contrast-enhanced CT slightly caudal to the renal hilum shows improved perinephric inflammation with regression of inflammatory extension along the iliopsoas compartment (arrowhead).

(D) Axial contrast-enhanced CT at the pelvic level shows further regression of the prior inflammatory change/collection tracking along the iliopsoas compartment (arrowhead).

(E) Axial contrast-enhanced CT at the pelvic outlet/proximal thigh shows marked regression of the prior anteromedial proximal thigh involvement near the obturator region (arrowhead).

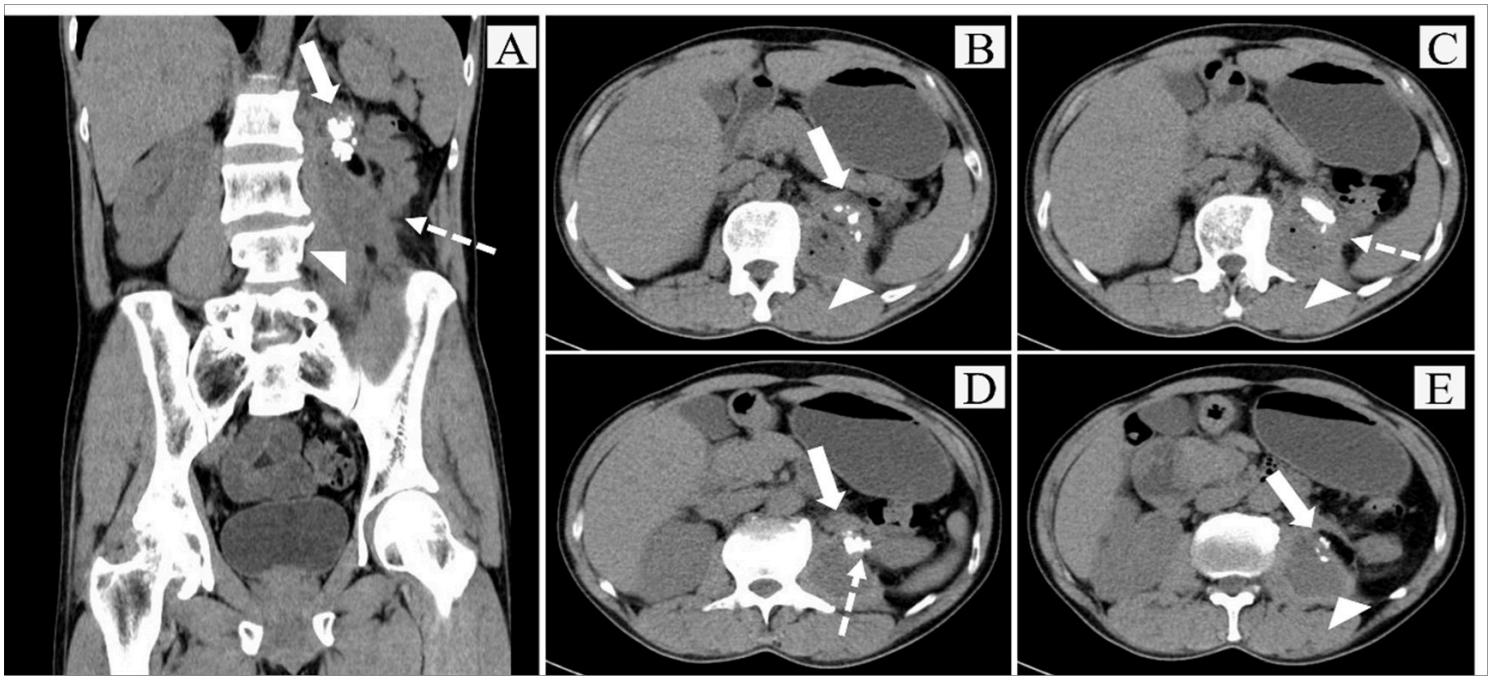


Figure 3

Non-contrast CT findings in Case 2 (February 2024).
(A) Coronal non-contrast CT demonstrates marked left renal atrophy with multiple intrarenal calculi (solid arrow). Extensive perinephric inflammatory change/collection is present with involvement of the left psoas major, suggestive of abscess formation (arrowhead). The fat plane between the perinephric inflammatory change/collection and the splenic flexure of the colon is indistinct (dashed arrow).
(B) Axial non-contrast CT at the renal level shows severe left renal atrophy with intrarenal calculi (solid arrow) and extension of the perinephric collection into the adjacent psoas major compartment (arrowhead).
(C) Axial non-contrast CT demonstrates an ill-defined interface between the perinephric inflammatory change/collection and the splenic flexure of the colon (dashed arrow), compatible with contiguous inflammatory involvement; adjacent extrarenal inflammatory change/collection is also present (arrowhead).
(D) Axial non-contrast CT at the level of the lower pole demonstrates hyperdense foci compatible with calculi within the adjacent perinephric/psoas collection (solid arrow); the normal fat plane between the left kidney/perinephric inflammatory change/collection and the adjacent colon is near-completely effaced (dashed arrow), suggesting dense inflammatory adhesion.
(E) Axial non-contrast CT demonstrates hyperdense foci compatible with calculi within the perinephric/psoas major collection (solid arrow), in keeping with inflammatory disruption and the presence of calculi within the surrounding collection (arrowhead).

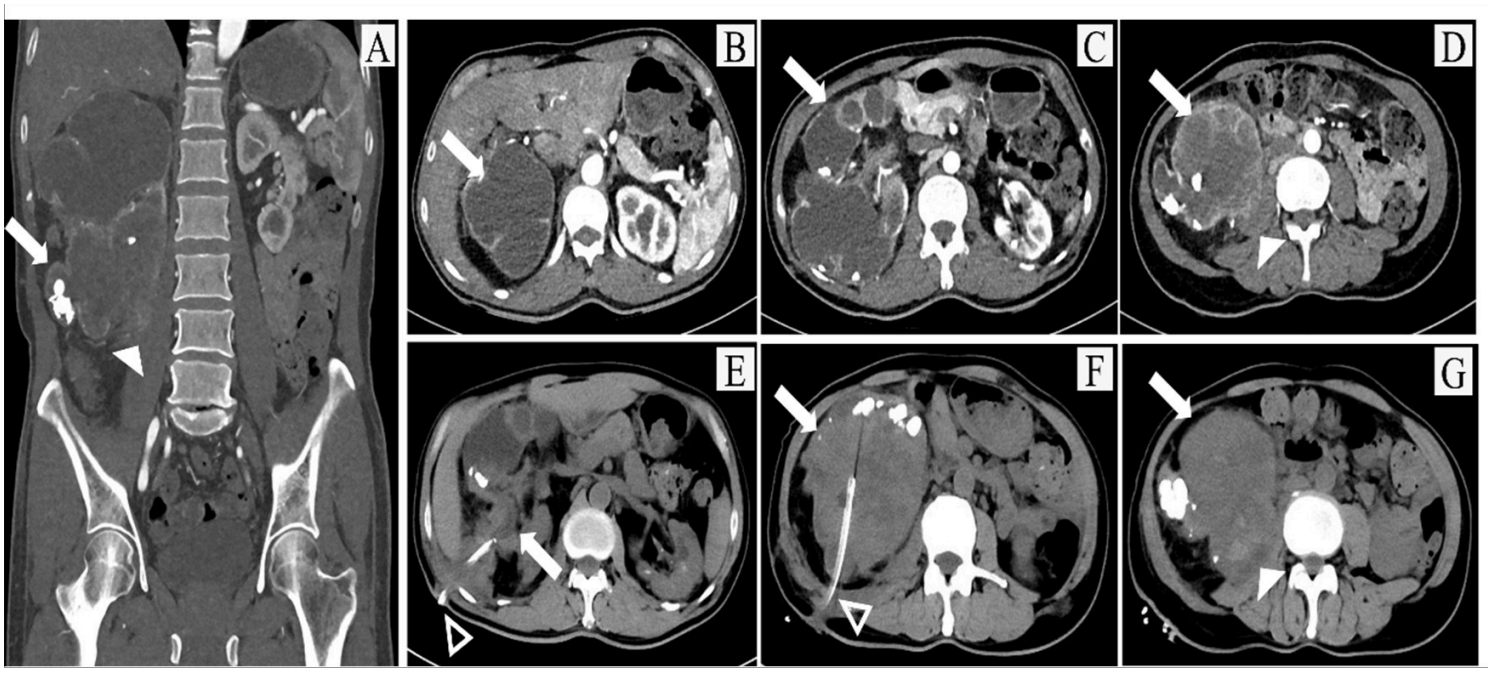


Figure 4

Imaging findings in Case 3 before and after percutaneous drainage.
(A) Coronal contrast-enhanced CT demonstrates a markedly enlarged right kidney with multiple calculi and severe hydronephrosis with pyonephrosis (solid arrow), with suspected contiguous involvement of the right psoas major muscle suggestive of abscess formation (arrowhead). No definite enhancing renal soft-tissue mass is identified.
(B–C) Axial contrast-enhanced CT images show multiple right renal calculi and a markedly dilated, multiloculated collecting system compatible with pyonephrosis (solid arrows).
(D) Axial contrast-enhanced CT demonstrates extension toward/into the right psoas major muscle (arrowhead).
(E) Axial non-contrast CT shows interval regression of pyonephrosis in the upper calyx (solid arrow) after placement of a nephrostomy tube (open arrowhead).
(F) Axial non-contrast CT shows increased pyonephrosis involving the mid-to-lower calyces (solid arrow) despite an additional nephrostomy tube in situ (open arrowhead), with hyperdense components compatible with hemorrhagic change.
(G) Axial non-contrast CT demonstrates further increase of pyonephrosis involving the mid-to-lower calyces (solid arrow) with progression of inflammatory involvement of the right psoas major muscle (arrowhead).

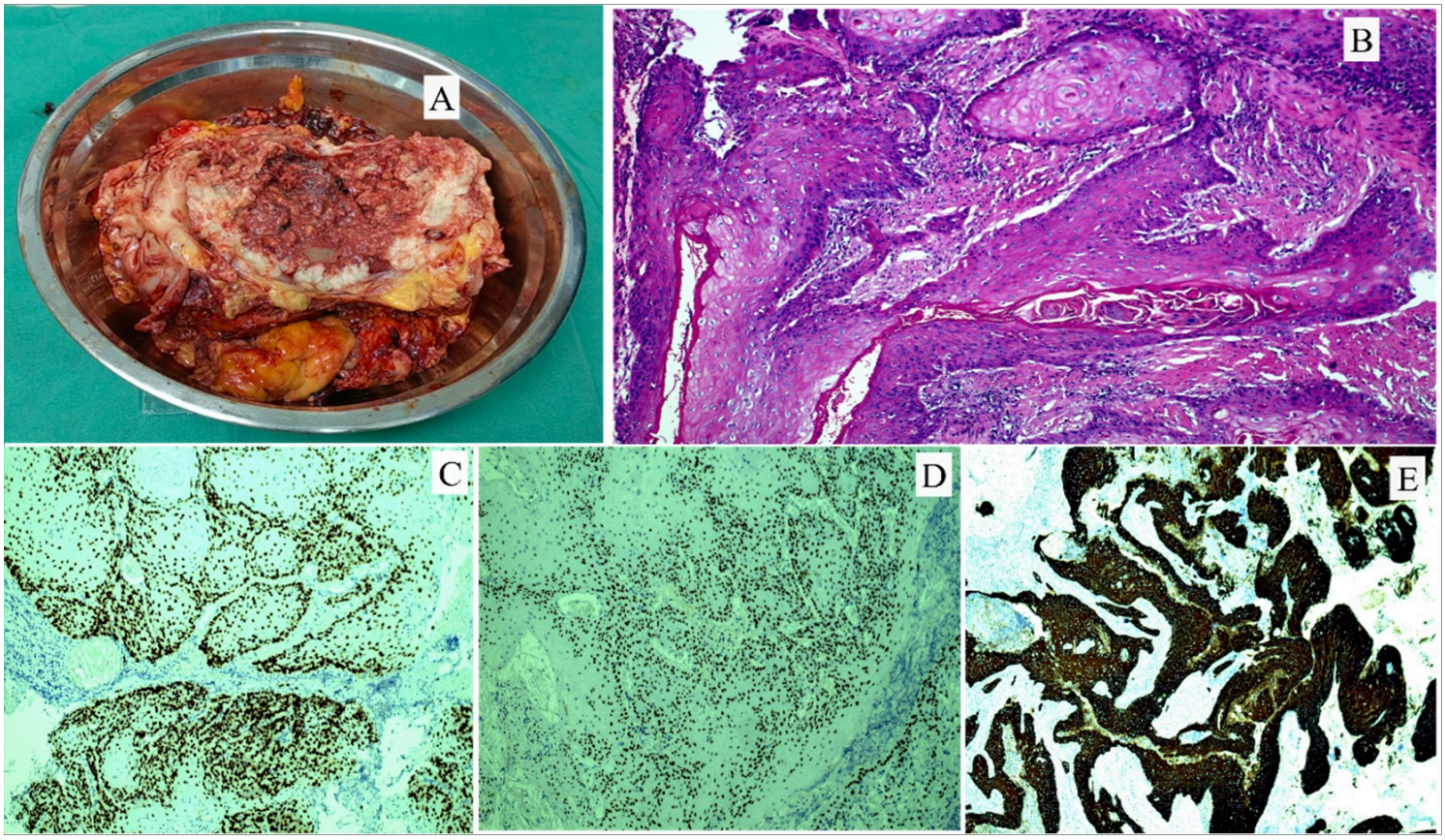


Figure 5

Gross and histopathological findings of the resected right kidney in Case 3.
(A) Gross examination of the nephrectomy specimen reveals a large, cauliflower-like mass (approximately 8 × 6 cm) centered in the renal pelvis region. The cut surface is hemorrhagic and friable, with associated cystic dilatation and renal calculi.
(B) Microscopic examination (hematoxylin and eosin staining, ×100) demonstrates irregular nests and sheets of atypical malignant cells infiltrating the stroma. The tumor exhibits moderate-to-poor differentiation, consistent with squamous cell carcinoma of the renal pelvis/upper urinary tract.
(C–E) Immunohistochemical staining supports squamous differentiation. The tumor cells show diffuse strong positivity for p63 (nuclear staining, ×100; Panel C), p40 (nuclear staining, ×100; Panel D), and CK5/6 (cytoplasmic/membranous staining, ×40; Panel E).