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Case Report

Xanthogranulomatous Pyelonephritis Presenting as Prolonged Constitutional Illness with Obstructive Uropathy and Near-Nonfunctioning Kidney: A Case Report

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ABSTRACT

Background: Xanthogranulomatous pyelonephritis is an uncommon, destructive form of chronic renal infection characterized by progressive parenchymal destruction, granulomatous inflammation, and frequent association with urinary tract obstruction, calculi, and chronic bacterial infection. Its presentation may be dominated by constitutional symptoms rather than localizing urinary complaints, which can delay diagnosis and mimic malignancy, systemic inflammatory disease, or occult infection.

Case presentation: A 48-year-old male presented with one month of generalized myalgia, fatigue, intermittent fever, and dysuria. Clinical examination revealed fever, dehydration, and tachycardia without respiratory compromise. Laboratory evaluation demonstrated marked systemic inflammation with leukocytosis, severe elevation of C-reactive protein, reactive thrombocytosis, microcytic hypochromic anemia, hyponatremia, and biochemical evidence of iron-restricted erythropoiesis. Urinalysis showed marked pyuria, proteinuria, microscopic hematuria, and strongly positive leukocyte esterase. Urine culture grew *Escherichia coli*. Ultrasonography demonstrated a left renal calculus with mild hydronephrosis and internal echoes within the pelvicalyceal system. Non-contrast computed tomography showed a bulky left kidney containing a renal pelvic calculus, multifocal low-attenuation parenchymal areas, perinephric inflammatory change, pararenal fascial thickening, and left hilar lymphadenopathy, supporting the diagnosis of xanthogranulomatous pyelonephritis. The patient underwent antimicrobial therapy and cystoscopic left double-J ureteric stenting, during which turbid efflux was noted from the left ureteric orifice. Post-procedurally, fever and urinary symptoms resolved, and inflammatory markers declined. Subsequent dimercaptosuccinic acid renal scintigraphy demonstrated severe left renal parenchymal dysfunction, with relative left renal function of approximately 4%, and left nephrectomy was advised.

Conclusion: This case highlights the diagnostic importance of considering xanthogranulomatous pyelonephritis in adults with prolonged constitutional symptoms, inflammatory anemia, marked inflammatory response, urinary infection, and renal calculi. Cross-sectional imaging and functional assessment were central to diagnosis, staging of renal damage, and planning definitive management.

Keywords: *xanthogranulomatous pyelonephritis; renal calculus; obstructive uropathy; Escherichia coli; DMSA scan; nephrectomy; chronic pyelonephritis; case report.*

INTRODUCTION

Xanthogranulomatous pyelonephritis is a rare, severe, and destructive inflammatory disorder of the kidney. It is generally regarded as a variant of chronic pyelonephritis in which renal parenchyma is progressively replaced by lipid-laden macrophages, granulomatous inflammatory tissue, fibrosis, and suppurative destruction. The disease is most often unilateral and is strongly associated with long-standing urinary obstruction, nephrolithiasis, and recurrent or persistent bacterial infection. In adults, the condition may remain clinically obscure for weeks to months because systemic symptoms may dominate over localizing urinary tract manifestations.

The diagnostic challenge arises from the breadth of its clinical mimicry. Fever, malaise, weight loss, anemia, leukocytosis, thrombocytosis, and elevated inflammatory markers may resemble renal malignancy, tuberculosis, chronic abscess, lymphoma, or systemic inflammatory disease. Radiologically, xanthogranulomatous pyelonephritis may also simulate renal neoplasm because it can produce renal enlargement, parenchymal distortion, inflammatory extension beyond the kidney, and regional lymphadenopathy. Computed tomography is therefore central to diagnosis because it defines the renal calculus burden, pelvicalyceal dilatation, parenchymal destruction, perinephric extension, fascial involvement, and adjacent organ relationships.

The present case concerns a middle-aged male with prolonged constitutional symptoms, urinary tract infection, obstructing renal pelvic calculus, extensive left renal inflammatory change, and subsequent scintigraphic demonstration of near-complete loss of left renal function. The case is clinically relevant because it illustrates the full diagnostic arc of xanthogranulomatous pyelonephritis: nonspecific presentation, marked inflammatory phenotype, imaging-based recognition, urgent decompression, biochemical response to source control, and final functional confirmation that renal salvage was unlikely.

Published literature consistently emphasizes that xanthogranulomatous pyelonephritis is both diagnostically deceptive and therapeutically consequential. Back in history, in their classical analysis, ***Malek and Elder*** described the condition as a destructive inflammatory renal disease with a strong tendency to mimic neoplastic and other chronic inflammatory disorders [1]. More recently, ***Harley et al.***, in a systematic review including more than 1000 reported cases, showed that most adult patients ultimately require nephrectomy, underscoring the importance of early recognition, accurate anatomical assessment, and functional evaluation before definitive treatment planning [3].

Case Presentation

A 48-year-old male presented to the outpatient clinic with a one-month history of generalized myalgia, fatigue, intermittent fever, and dysuria. There was no documented significant past medical history. The clinical presentation was notable for a predominantly constitutional illness, with fever and systemic symptoms occurring alongside urinary complaints.

On examination, the patient was conscious and oriented. He appeared dehydrated and febrile. Blood pressure was 110/72 mmHg, pulse rate was 100 beats/min, respiratory rate was 18 breaths/min, temperature was 38.5°C, and oxygen saturation was 99% on room air. There was no evidence of respiratory compromise at presentation. The combination of fever, tachycardia, dehydration, dysuria, and systemic symptoms suggested an active urinary source of infection with clinically significant inflammatory response.

Investigations

Hematological and biochemical findings

Initial laboratory evaluation showed leukocytosis, severe inflammatory marker elevation, reactive thrombocytosis, anemia, and mild hyponatremia. The total white blood cell count was $17.4 \times 10^3/\mu L$ and platelet count was $696 \times 10^9/L$. C-reactive protein was markedly elevated at 205 mg/L, while erythrocyte sedimentation rate was 22 mm/hr. Hemoglobin was 8.9 g/dL and hematocrit was 28.8%. Serum sodium was 132 mmol/L. Iron studies showed reduced transferrin saturation of 11.46% and total iron-binding capacity of 176 µg/dL. Peripheral smear demonstrated microcytic hypochromic anemia with leukocytosis and reactive thrombocytosis. The clinical, hematological, microbiological, radiological, procedural, and functional findings are summarized in Table 1.

The hematological pattern was consistent with severe ongoing inflammation complicated by anemia. The thrombocytosis was interpreted as reactive in the clinical setting of infection and inflammation. The degree of CRP elevation supported a significant inflammatory or infective burden rather than uncomplicated lower urinary tract infection.

Urine examination and microbiology

Urinalysis demonstrated blood 1+, protein 2+, leukocyte esterase 4+, more than 100 white blood cells/high-power field, and 4–6 red blood cells/high-power field. Urine culture grew *Escherichia coli*. Blood cultures were negative.

The urine findings established active urinary tract infection with marked pyuria and microscopic hematuria. In the presence of renal calculus, hydronephrosis, fever, and high inflammatory markers, the microbiological result supported a diagnosis of complicated upper urinary tract infection rather than isolated cystitis.

Imaging Findings

Ultrasonography

Abdominal ultrasonography demonstrated grade I fatty liver and cholelithiasis without sonographic features of acute cholecystitis. Renal imaging showed a left renal calculus with mild left hydronephrosis. Internal echoes were present within the left pelvicalyceal system, raising concern for infected obstructed urine or debris within a dilated collecting system.

Although ultrasonography identified the calculus and hydronephrosis, the extent of parenchymal and perinephric inflammatory disease required further anatomical characterization by computed tomography.

Computed tomography

Non-contrast computed tomography of the kidney, ureter, and bladder demonstrated a bulky left kidney with a 14-mm renal pelvic calculus. Multiple hypodense areas involved the upper, mid, and lower poles of the left kidney. There was significant perinephric fat stranding, thickening of the pararenal fascia, and enlarged left renal hilar lymph nodes. The overall CT appearance was consistent with severe inflammatory pyelonephritis and was suggestive of xanthogranulomatous pyelonephritis.

The supplied axial CT images show an enlarged left kidney with a central calculus and extensive surrounding inflammatory change. The kidney appears distorted by low-attenuation parenchymal regions, with perinephric stranding extending into adjacent fat planes. These findings support diffuse destructive renal inflammation rather than uncomplicated acute pyelonephritis. Representative axial CT images demonstrate left renal enlargement with extensive perinephric inflammatory change and a renal pelvic calculus associated with destructive parenchymal abnormality, as shown in Figures 1 and 2.

Renal functional imaging

Follow-up dimercaptosuccinic acid renal scintigraphy showed severe left renal parenchymal dysfunction. The reported relative function of the left kidney was approximately 4%, with preserved function predominantly in the contralateral kidney. Based on this near-nonfunctioning status, left nephrectomy was advised.

The functional scan was important because anatomical imaging alone demonstrates structural destruction, while renal scintigraphy quantifies residual cortical function. In this case, the markedly reduced split renal function supported definitive surgical planning rather than attempts at long-term renal preservation. The DMSA scintigraphy images demonstrate preserved right renal cortical uptake with markedly reduced left renal uptake, and the quantitative report confirmed severe reduction in left split renal function, as shown in Figures 3 and 4.

Initial Management and Early Clinical Course

The patient was admitted and treated with intravenous fluids and broad-spectrum intravenous antibiotics. Urology consultation was obtained because of obstructive uropathy and extensive inflammatory renal changes. He underwent cystoscopy with left double-J ureteric stent placement under spinal anesthesia. Turbid efflux was noted from the left ureteric orifice, supporting the presence of infected obstructed urine. A 6 Fr, 24-cm double-J stent was placed successfully.

Following decompression and antimicrobial therapy, the patient improved clinically. Fever subsided, and symptoms of dysuria, myalgia, and fatigue resolved. Repeat laboratory evaluation showed reduction in inflammatory burden, with CRP decreasing from 205 mg/L to 99.8 mg/L and white blood cell count decreasing from 17.4 × 10^3/µL to 12.44 × 10^3/µL. He was discharged in hemodynamically stable condition on oral antibiotics with outpatient follow-up.

The early response confirmed that antimicrobial therapy and urinary drainage achieved short-term source control. However, subsequent DMSA findings indicated that the left kidney had sustained severe functional loss, shifting management toward planned nephrectomy.

Table 1. Clinical, laboratory, microbiological, and imaging summary

Domain	Findings
Age and sex	48-year-old male
Presenting symptoms	One month of generalized myalgia, fatigue, intermittent fever, and dysuria
Vital signs	BP 110/72 mmHg; pulse 100/min; RR 18/min; temperature 38.5°C; SpO_2 99% on room air

Hematology	WBC 17.4 × 10^3/μL; platelet count 696 × 10^9/L; hemoglobin 8.9 g/dL; hematocrit 28.8%
Inflammatory markers	CRP 205 mg/L initially; reduced to 99.8 mg/L after treatment; ESR 22 mm/hr
Peripheral smear	Microcytic hypochromic anemia, leukocytosis, reactive thrombocytosis
Urinalysis	Blood 1+, protein 2+, leukocyte esterase 4+, WBC >100/HPF, RBC 4–6/HPF
Urine culture	*Escherichia coli*
Ultrasonography	Left renal calculus, mild left hydronephrosis, internal echoes in pelvicalyceal system
CT KUB	Bulky left kidney, 14-mm renal pelvic calculus, multifocal hypodense parenchymal areas, perinephric fat stranding, pararenal fascial thickening, left hilar lymphadenopathy
Procedure	Cystoscopy with left double-J ureteric stenting; turbid efflux noted
Functional imaging	DMSA scan showed severe left renal parenchymal dysfunction; relative left renal function approximately 4%
Planned definitive management	Left nephrectomy advised

Figure Legends

Figure 1. Axial non-contrast CT image showing left renal enlargement with extensive inflammatory change.
Axial CT demonstrates a bulky left kidney with distortion of the renal contour and extensive perinephric fat stranding. The inflammatory changes extend beyond the renal capsule into the perinephric region, supporting complicated chronic infective pyelonephritis rather than uncomplicated urinary tract infection.

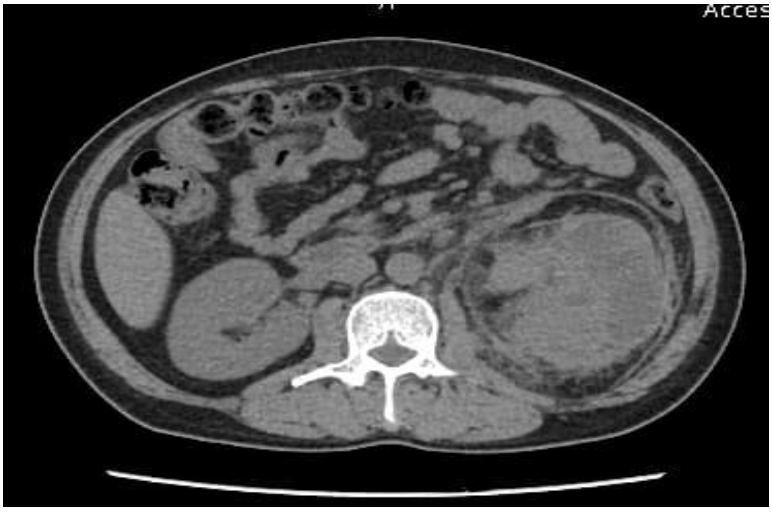


Figure 2. Axial non-contrast CT image showing left renal pelvic calculus with destructive renal inflammatory changes.

Axial CT image demonstrates a hyperdense calculus within the left renal pelvis, (yellow arrow) associated with renal enlargement, low-attenuation parenchymal areas, and marked perinephric inflammatory reaction. The imaging pattern is compatible with xanthogranulomatous pyelonephritis in the appropriate clinical and microbiological context.

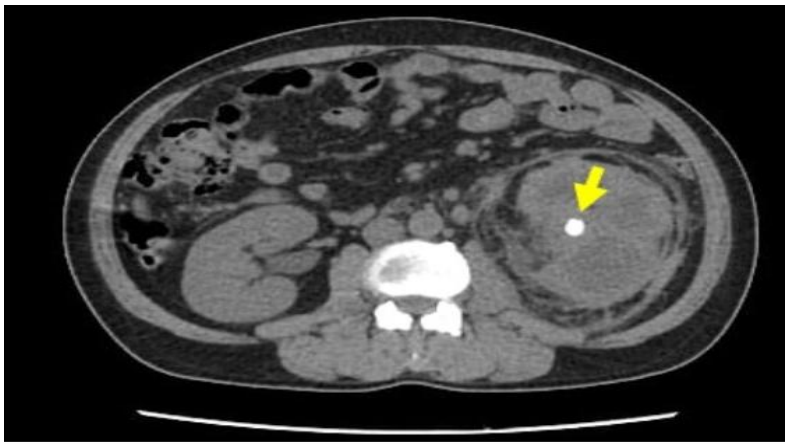
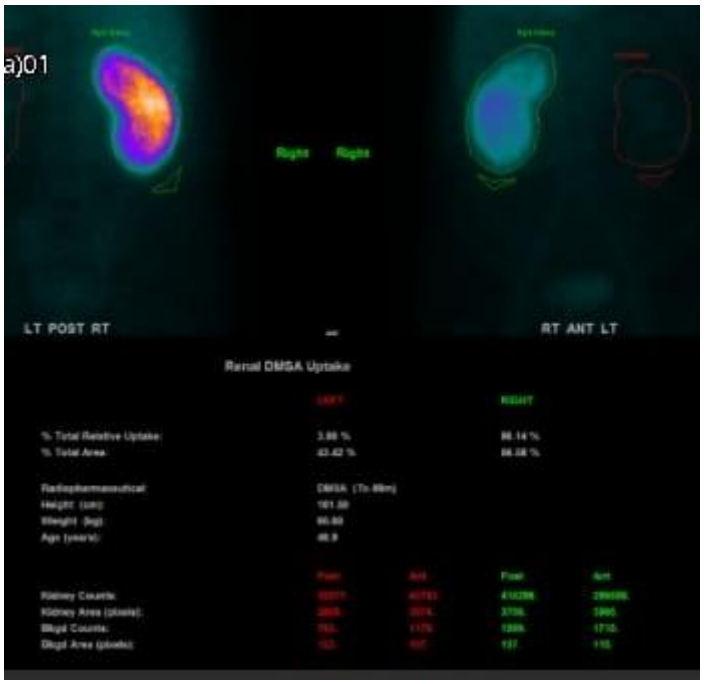


Figure 3. DMSA renal scintigraphy showing preserved right renal cortical uptake and markedly reduced left renal function.

Renal cortical scintigraphy demonstrates dominant tracer uptake in the right kidney with markedly reduced uptake in the left kidney, consistent with severe left renal parenchymal dysfunction.

Figure 4. Quantitative DMSA scan report demonstrating severely reduced split function of the left kidney.

Quantitative renal scintigraphy shows severely impaired relative function of the left kidney, reported at approximately 4%, supporting the decision for definitive surgical management.



DISCUSSION

Xanthogranulomatous pyelonephritis is an uncommon but clinically important form of chronic destructive renal infection. The present case is instructive because the initial clinical picture was dominated by prolonged constitutional symptoms, severe systemic inflammation, anemia, and only modestly localizing urinary symptoms. This pattern is well recognized in xanthogranulomatous pyelonephritis, where the clinical syndrome may mimic malignancy, occult abscess, tuberculosis, or systemic inflammatory disease before renal imaging clarifies the diagnosis. In their classical clinicopathological analysis, ***Malek and Elder*** emphasized the protean and deceptive nature of xanthogranulomatous pyelonephritis, noting that it frequently imitates neoplastic and other inflammatory renal disorders [1]. This diagnostic ambiguity remains clinically relevant, particularly when patients present late with constitutional symptoms and extensive perinephric inflammation.

The disease mechanism is generally linked to the interaction of chronic obstruction, persistent infection, suppuration, impaired urinary drainage, and parenchymal destruction. The affected kidney is progressively replaced by lipid-laden macrophages, chronic inflammatory infiltrates, abscess cavities, fibrosis, and granulomatous tissue. ***Li and Parwani*** described xanthogranulomatous pyelonephritis as a destructive inflammatory disorder associated with long-standing urinary tract obstruction and infection, with characteristic pathological accumulation of foamy histiocytes within damaged renal parenchyma [2]. In the present case, the 14-mm left renal pelvic calculus, hydronephrosis, marked pyuria,

Escherichia coli growth, and turbid ureteric efflux together support an obstructed infected system evolving into destructive renal inflammation.

The demographic profile of xanthogranulomatous pyelonephritis has classically shown female predominance, most commonly in middle age, although the disease is not restricted to women. In the systematic review by ***Harley et al.***, the typical patient was described as a woman in the fifth or sixth decade with urolithiasis, while management most often involved nephrectomy [3]. The present patient was a 48-year-old male, which makes the case less typical demographically but not biologically inconsistent. The key disease-defining elements were present: calculus-related obstruction, infected urine, renal enlargement, parenchymal destruction, severe inflammatory response, and near-complete loss of ipsilateral renal function.

The laboratory phenotype in this case was also characteristic of advanced inflammatory renal disease. The patient had leukocytosis, markedly elevated C-reactive protein, reactive thrombocytosis, microcytic hypochromic anemia, and reduced transferrin saturation. Xanthogranulomatous pyelonephritis may produce a systemic inflammatory state with anemia, malaise, fever, and weight loss, often disproportionate to the degree of urinary symptoms. In the 41-case clinical series by ***Korkes et al.***, the disease was described as an uncommon inflammation of the renal parenchyma occurring in the setting of chronic obstruction and suppuration, and computed tomography was highlighted as central to diagnosis [4]. The present hematological profile supports this concept of a chronic suppurative-inflammatory process rather than an isolated short-duration urinary tract infection.

Microbiologically, *Escherichia coli* is among the organisms most often implicated in xanthogranulomatous pyelonephritis, along with *Proteus mirabilis* and other uropathogens. In the study by Artiles-***Medina et al.***, which focused on microbiological and antibiotic resistance profiles, xanthogranulomatous pyelonephritis was treated most often with antibiotics combined with nephrectomy, and microbiological definition was considered important for targeted therapy [5]. The urine culture in the present case grew *Escherichia coli*, aligning with the common microbiological pattern. Negative blood cultures did not exclude severe renal infection, as the inflammatory burden, pyuria, imaging findings, and turbid ureteric drainage were sufficient to establish a complicated infected obstructed renal unit.

Imaging was decisive in this case. Ultrasonography first demonstrated a left renal calculus with mild hydronephrosis and internal echoes in the pelvicalyceal system, suggesting infected debris or pyonephrotic content. However, ultrasonography alone could not define the full extent of parenchymal destruction or extrarenal inflammatory spread. ***Subramanyam et al.*** reported that sonographic appearances of xanthogranulomatous pyelonephritis may include renal enlargement, a central echogenic calculus-related focus, and parenchymal anechoic or hypoechoic areas, but also cautioned that sonographic distinction from hydronephrosis with calculus may be difficult [6]. This limitation was reflected in the present case, where CT was required for definitive anatomical characterization.

Non-contrast computed tomography demonstrated a bulky left kidney, renal pelvic calculus, multifocal hypodense parenchymal areas, marked perinephric fat stranding, pararenal fascial thickening, and hilar lymphadenopathy. These findings are strongly supportive of diffuse xanthogranulomatous pyelonephritis in the appropriate clinical context. CT can identify renal enlargement, calculi, hydronephrosis, low-attenuation intrarenal collections, inflammatory parenchymal replacement, perinephric extension, and involvement of adjacent fascial planes. ***Craig et al.*** described the radiologic-pathologic basis of pyelonephritis and emphasized the role of CT in complicated renal infections, including xanthogranulomatous pyelonephritis, because it demonstrates both renal and extrarenal disease extent [7]. In the present case, the CT images were consistent with destructive inflammatory pyelonephritis rather than uncomplicated acute pyelonephritis.

The so-called “bear paw sign” is often described in diffuse xanthogranulomatous pyelonephritis, referring to a multiloculated appearance produced by dilated calyces and inflammatory low-attenuation areas surrounding a contracted or obstructed collecting system. Although the sign is not mandatory for diagnosis, its presence is useful when seen in the proper clinical setting. ***Garrido-Abad et al.*** described the bear paw sign as a characteristic CT feature in xanthogranulomatous pyelonephritis, reflecting replacement of renal parenchyma by inflammatory tissue and low-attenuation cavities [8]. The supplied CT images in the present case show renal enlargement, low-attenuation parenchymal regions, central calculus, and perinephric inflammation, which together approximate this classical imaging phenotype.

A major diagnostic issue is distinction from renal malignancy. Xanthogranulomatous pyelonephritis may simulate renal cell carcinoma because both conditions can present with constitutional symptoms, anemia, renal enlargement, local invasion-like inflammatory extension, and regional lymphadenopathy. Conversely, renal malignancy can coexist with chronic infection or be obscured by inflammatory change. This overlap requires careful radiological review and, when nephrectomy is performed, definitive histopathological assessment. ***Malek and Elder*** specifically emphasized the

tendency of xanthogranulomatous pyelonephritis to mimic renal neoplasia and other renal inflammatory diseases [1]. Therefore, in the present case, the diagnosis should be expressed as clinicoradiologically suggestive of diffuse xanthogranulomatous pyelonephritis until nephrectomy histology is available.

The functional imaging result was central to management planning. DMSA renal scintigraphy showed severe left renal parenchymal dysfunction, with relative left renal function of approximately 4%. This finding confirmed that the left kidney was nearly nonfunctioning. In diffuse xanthogranulomatous pyelonephritis, parenchymal salvage is often not possible because the kidney has undergone irreversible inflammatory destruction. The management-focused review by ***Gravestock et al.*** reported that the overwhelming majority of adult patients in included series underwent nephrectomy, reflecting the destructive and frequently non-salvageable nature of established disease [9]. In the present case, the decision to advise left nephrectomy was therefore consistent with both the anatomical burden of disease and the objective demonstration of minimal residual renal function.

Initial drainage before definitive surgery was clinically appropriate because the patient had obstructive uropathy with infected urine and systemic inflammation. The placement of a double-J ureteric stent produced decompression, confirmed by turbid efflux from the left ureteric orifice, and was followed by clinical and biochemical improvement. A staged strategy of antimicrobial therapy, urinary drainage, stabilization, and later nephrectomy is often reasonable in patients presenting with sepsis physiology, obstruction, or extensive inflammatory disease. Artiles-***Medina et al.*** highlighted the frequent use of combined antibiotic and surgical management, while ***Harley et al.*** showed that nephrectomy remains the dominant definitive treatment in contemporary series [3,5]. The present case illustrates this staged principle: urgent infection control was achieved first, while functional imaging subsequently clarified the need for definitive removal of the destroyed renal unit.

The present case also illustrates why xanthogranulomatous pyelonephritis should be considered in patients with unexplained systemic inflammation and urinary tract abnormalities. A month-long history of fever, fatigue, myalgia, anemia, pyuria, and a positive urine culture could be misclassified as recurrent uncomplicated infection if renal imaging is delayed. However, the coexistence of renal calculus, hydronephrosis, severe CRP elevation, thrombocytosis, and anemia should prompt evaluation for complicated upper tract disease. The clinical message is not merely that xanthogranulomatous pyelonephritis is rare, but that it is a diagnosis of pattern recognition: chronic systemic illness plus infected obstruction plus destructive renal imaging.

Table 2 discusses the possible differential diagnosis one has to consider at such presentations.

Table 2: Differential diagnostic considerations

Differential diagnosis	Overlapping features with the present case	Features supporting or arguing against the diagnosis in this case
Renal cell carcinoma	Constitutional symptoms, anemia, renal enlargement, regional lymphadenopathy, mass-like renal distortion	The presence of pyuria, positive *E. coli* culture, renal pelvic calculus, hydronephrosis, turbid ureteric efflux, and diffuse inflammatory CT pattern favored infective destructive pyelonephritis; histology after nephrectomy would be required to exclude coexistent malignancy definitively.
Renal abscess or pyonephrosis	Fever, leukocytosis, pyuria, obstructed infected collecting system, low-attenuation renal lesions	The chronic constitutional history, calculus-associated diffuse renal involvement, perinephric inflammatory extension, and near-nonfunctioning kidney favored diffuse xanthogranulomatous pyelonephritis rather than a localized abscess alone.
Renal tuberculosis	Constitutional symptoms, sterile pyuria, chronic renal destruction, strictures, calcification	Urine culture grew *E. coli* and the imaging pattern was associated with renal pelvic calculus and suppurative obstruction; no evidence of sterile pyuria, ureteric stricturing, pulmonary tuberculosis, or microbiological tuberculosis confirmation was provided.
Emphysematous pyelonephritis	Severe renal infection, systemic toxicity, diabetes-associated complicated pyelonephritis, possible obstruction	No gas within the renal parenchyma, collecting system, or perinephric tissues was described on CT; the imaging showed destructive inflammatory changes without emphysematous features.
Acute bacterial pyelonephritis	Fever, dysuria, pyuria, leukocytosis, positive urine culture	The renal pelvic calculus, hydronephrosis, bulky distorted kidney, multifocal hypodense parenchymal areas, perinephric extension, fascial thickening, and 4% split renal function indicated chronic destructive disease rather than uncomplicated acute infection.

Radiological-pathological correlation

The CT appearances in this case are best understood as the anatomical expression of chronic obstructed infection. The renal pelvic calculus likely contributed to impaired urinary drainage and persistent infection. The multifocal hypodense parenchymal areas correspond radiologically to destroyed renal parenchyma, inflammatory cavities, dilated infected calyces, abscess-like spaces, and xanthogranulomatous tissue. Perinephric fat stranding and pararenal fascial thickening indicate extension of inflammation beyond the kidney. Hilar lymphadenopathy is compatible with regional inflammatory response but also reinforces the need for careful exclusion of malignancy when definitive histology becomes available.

Case-specific learning points

1. Xanthogranulomatous pyelonephritis may present predominantly as prolonged constitutional illness rather than acute flank pain alone.
2. Severe inflammatory markers, reactive thrombocytosis, anemia, pyuria, and renal calculus should prompt evaluation for complicated upper tract infection.
3. CT is the pivotal imaging modality because it demonstrates calculi, renal enlargement, parenchymal destruction, perinephric extension, and fascial involvement.
4. Urgent decompression with antimicrobial therapy can stabilize the patient when infected obstruction is present.
5. DMSA renal scintigraphy is valuable when treatment planning depends on objective assessment of residual renal cortical function.
6. In the absence of nephrectomy histology, the diagnosis should be framed as clinicoradiologically suggestive of xanthogranulomatous pyelonephritis rather than histologically proven disease.

AUTHOR CONTRIBUTIONS

Author Contributions (CRediT Taxonomy)

Rajashaker Reddy Kotam: Conceptualization, patient management, data acquisition, literature review, manuscript drafting, manuscript revision, supervision, and final approval of the manuscript.

Shruthi Chamala: Data collection, clinical documentation, literature review, manuscript drafting, manuscript editing, and final approval of the manuscript.

Althaf Hussain Kadhakadi: Urological evaluation, procedural management, interpretation of urological findings, critical revision of the manuscript for important intellectual content, and final approval of the manuscript.

All authors contributed substantially to the preparation of the manuscript, reviewed the final version, and approved its submission for publication.

Declarations

Ethics Approval

Not applicable for a single-patient case report according to institutional policy. Written informed consent was obtained from the patient for publication of clinical details and anonymized images.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of Interest

The authors declare no conflicts of interest.

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Data Availability Statement

All relevant data supporting the findings of this case report are included within the article. Additional anonymized information may be available from the corresponding author upon reasonable request.

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