

Received: 2026.05.16

Accepted: 2026.08.11

Available online: 2026.09.07

Published: 2026.XX.XX

Bilateral Xanthogranulomatous Pyelonephritis in a Patient With Spina Bifida and Neurogenic Bladder: A Case Report

EF Dagmawi Dereje Wale 

F Heera Tesfaye Erega

F Iman Omer

F Alvin Billy

F Advith Suresh

F Tigabu Bishaw

F Tadlo Belete

F Tsebelu Shirshaw 

F Vishal A. Poddar

EF Shaheen Alvi

Department of Internal Medicine, Howard University Hospital,
Washington, DC, USA

Authors' Contribution:

Study Design A

Data Collection B

Statistical Analysis C

Data Interpretation D

Manuscript Preparation E

Literature Search F

Funds Collection G

Corresponding Author: Dagmawi D. Wale, Department of Internal Medicine, Howard University Hospital, 2041 Georgia Avenue, NW, 5th Floor, Suite 5100, Washington, DC 20060, USA, Phone: (202) 865-6100, e-mail: dwale@huhosp.org

Financial support: None declared

Conflict of interest: None declared

Patient: Male, 30-year-old

Final Diagnosis: Xanthogranulomatous pyelonephritis

Symptoms: Poor oral intake • shortness of breath • weight loss

Clinical Procedure: —

Specialty: Critical Care Medicine • Infectious Diseases • General and Internal Medicine • Nephrology • Urology

Objective: Rare disease

Background: Xanthogranulomatous pyelonephritis (XGP) is a rare chronic inflammatory renal infection associated with urinary obstruction, recurrent infections, and progressive renal parenchymal destruction. Bilateral involvement is exceedingly uncommon and associated with substantial morbidity and mortality, particularly in patients with neurogenic bladder and impaired longitudinal urologic follow-up.

Case Report: A 30-year-old man with spina bifida complicated by neurogenic bladder requiring intermittent catheterization presented with respiratory distress, profound anemia, and progressive functional decline. Imaging demonstrated severe bilateral hydronephrosis, diffuse renal parenchymal destruction and calcifications, and right-sided stag-horn calculi, consistent with advanced bilateral XGP. The patient rapidly developed septic shock and acute-on-chronic hypercapnic respiratory failure requiring mechanical ventilation and vasopressor support. Percutaneous nephrostomy drainage yielded purulent material, and cultures grew *Morganella morganii*, *Candida parapsilosis*, and *Candida tropicalis*. His hospital course was complicated by dialysis-dependent renal failure, recurrent respiratory failure, and prolonged ventilatory dependence. Definitive surgical intervention was considered but deferred because of severe anatomical deformities, clinical instability, and prohibitive operative risk. After prolonged hospitalization, he was discharged to a long-term acute care facility but later died from complications related to tracheostomy.

Conclusions: This case illustrates the severe clinical consequences of advanced bilateral XGP in a high-risk patient with neurogenic bladder. The coexistence of recurrent infection, urinary obstruction, and disrupted longitudinal follow-up is consistent with recognized risk factors for upper urinary tract deterioration, although their individual contribution to this patient's disease progression cannot be established. The case underscores the management challenges that arise when bilateral renal destruction and systemic instability limit definitive source-control options.

Keywords: Dialysis • Hydronephrosis • Pyelonephritis • Pyelonephritis, Xanthogranulomatous • Renal Insufficiency, Chronic • Sepsis • Spinal Cord Diseases • Spinal Dysraphism • Staghorn Calculi

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/954146>

 1447

 —

 4

 14

Publisher's note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher



Introduction

Xanthogranulomatous pyelonephritis (XGP) is a rare chronic inflammatory renal condition characterized by progressive parenchymal destruction secondary to long-standing urinary obstruction and recurrent infections [1,2]. Histologically, the disease is marked by lipid-laden macrophages replacing normal renal tissue [3]. Computed tomography (CT) remains the diagnostic modality of choice and frequently demonstrates hydronephrosis, renal enlargement, parenchymal destruction, calculi, and the classic “bear paw sign” [4,5].

Although XGP typically presents unilaterally, bilateral disease is rare and associated with severe systemic complications, including sepsis, renal failure, and increased mortality [1,6]. In this manuscript, the term “advanced XGP” is used descriptively for severe destructive disease with major loss of renal parenchyma and associated obstructive changes; contemporary literature describes extensive XGP as higher-stage disease that may extend beyond the kidney into perirenal or retroperitoneal tissues [7]. Patients with spina bifida are particularly vulnerable because of neurogenic bladder, chronic urinary stasis, recurrent infections, and upper urinary tract deterioration associated with impaired bladder dynamics and inconsistent longitudinal follow-up [8-11].

Given the rarity and severity of bilateral XGP, the primary contribution of this report is to describe an extensive bilateral presentation in a patient with spina bifida and neurogenic bladder, characterized by severe bilateral hydronephrosis, diffuse renal parenchymal destruction, staghorn calculi, septic shock, and major limitations to definitive surgical management. Recurrent infections, chronic obstruction, and disrupted longitudinal care were present in this patient and are discussed as clinically plausible, literature-consistent contributors rather than as causal findings derived from this single case.

Case Report

A 30-year-old man with a history of congenital spina bifida complicated by neurogenic bladder requiring intermittent catheterization was transferred from an outside hospital for percutaneous nephrostomy tube placement. His clinical course was notable for recurrent urinary tract infections since transitioning from pediatric to adult care at age 21, with poor longitudinal follow-up thereafter. He had experienced 3 prior hospital admissions within the preceding 6 months, related to urologic complications. He presented with several weeks of progressive weight loss, functional decline, and poor oral intake, culminating in acute respiratory distress. His medical history was also significant for nephrolithiasis, and he lacked consistent outpatient medical care.



Figure 1. Computed tomography scout view demonstrating severe musculoskeletal deformities consistent with underlying spina bifida, including marked thoracolumbar scoliosis and fixed lower extremity contractures.

On presentation, the patient appeared cachectic with severe musculoskeletal deformities, including thoracolumbar scoliosis and fixed contractures of the lower extremities (**Figure 1**). Vital signs were notable for tachycardia and mild hypoxemia. Laboratory evaluation demonstrated profound anemia (hemoglobin 4.7 g/dL), leukocytosis, acute kidney injury, and significant pyuria. Arterial blood gas analysis revealed severe acute-on-chronic hypercapnic respiratory failure with a pH of 7.16 and PCO_2 in the 90s, consistent with profound acidemia, necessitating immediate transfer to the intensive care unit and endotracheal intubation.

CT of the abdomen and pelvis demonstrated severe bilateral hydronephrosis with diffuse renal parenchymal destruction and calcifications consistent with advanced bilateral XGP (**Figure 2**), along with right-sided staghorn calculi (**Figure 3**). Within 24 hours of admission, the patient developed septic shock requiring vasopressor support. A right-sided percutaneous nephrostomy tube was placed, which drained purulent material and supported the urinary tract as a major source of infection.



Figure 2. Contrast-enhanced computed tomography of the abdomen and pelvis showing bilateral hydronephrosis with diffuse renal parenchymal destruction and calyceal calcifications, findings consistent with advanced xanthogranulomatous pyelonephritis.

The hospital course was marked by multiple complications, including dialysis-dependent acute kidney injury, persistent ventilator dependence, ventilator-associated pneumonia, urinary tract infection with *Morganella morganii*, and candidemia due to *Candida parapsilosis* and *Candida tropicalis*. Although the patient was intermittently extubated, his course was further complicated by recurrent episodes of hypercapnic respiratory failure requiring repeated re-intubation.

The urology team was actively involved throughout the hospitalization, and definitive surgical management with bilateral nephrectomy was considered. However, the procedure was deemed technically unfeasible due to complex anatomical constraints, severe musculoskeletal deformities, and positioning limitations. Multiple tertiary care centers were consulted for potential transfer and surgical intervention, but all attempts were unsuccessful due to the patient's high operative risk and clinical instability.

After a prolonged hospitalization, the patient achieved hemodynamic stabilization with control of sepsis and was successfully weaned off vasopressor support. He was subsequently discharged to a long-term acute care facility for continued



Figure 3. Axial computed tomography image demonstrating large staghorn calculi occupying the right renal collecting system, contributing to chronic obstruction and inflammatory renal destruction.

ventilatory support, rehabilitation, and ongoing management with the nephrostomy tube in place, with plans for delayed surgical evaluation. Unfortunately, within 1 month of discharge, the patient was readmitted to an outside hospital and ultimately died due to complications related to tracheostomy.

This report illustrates an extreme bilateral XGP presentation in a high-risk patient with neurogenic bladder, in whom advanced renal destruction and severe systemic illness coincided with limited surgical feasibility and constrained options for definitive source control.

Discussion

This case primarily contributes to the literature by illustrating the clinical complexity and management constraints of advanced bilateral XGP in a high-risk patient with neurogenic bladder. Although XGP accounts for less than 1% of chronic pyelonephritis cases, bilateral disease remains uncommon and is associated with substantial morbidity [1,2,6]. In this patient, chronic urinary stasis, recurrent infections, obstructive nephrolithiasis, and interrupted longitudinal care coexisted with severe bilateral renal destruction. These factors are biologically and clinically plausible contributors based on the published literature, but their individual effects, temporal sequence, and

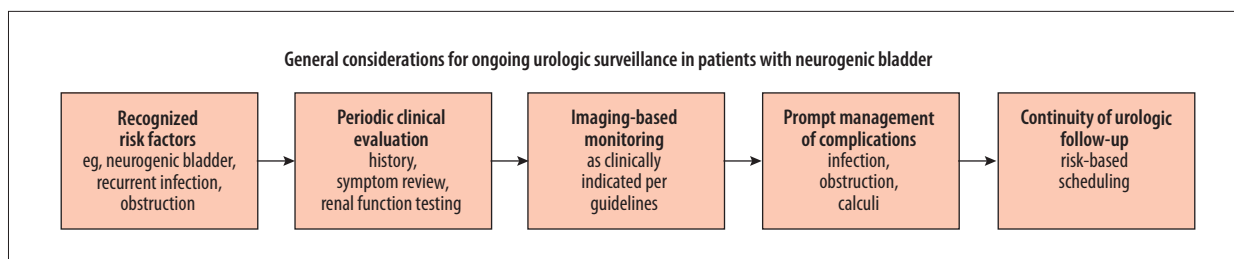


Figure 4. General framework of urologic surveillance considerations for patients with neurogenic bladder.

causal relationship to disease severity cannot be determined from a single-case observation [9-11].

The clinical severity in this patient—including polymicrobial infection, septic shock, dialysis-dependent renal failure, and recurrent respiratory failure—illustrates the extreme end of the XGP disease spectrum. Prior reports and systematic reviews describe substantial morbidity in advanced XGP, particularly when infection, obstruction, and loss of functional renal parenchyma are present [6,12]. *Morganella morganii* and *Candida* species in this case also reflect the complexity of infection in a heavily instrumented and critically ill patient [13,14]. While the diseased urinary tract was a major infectious source, the degree to which persistent renal infection independently drove each subsequent systemic complication cannot be established from this case alone.

The case also illustrates the recognized vulnerability of patients with spina bifida and neurogenic bladder to upper urinary tract deterioration. Long-term studies and contemporary guidance associate neurogenic lower urinary tract dysfunction with hydronephrosis, recurrent infection, renal scarring, and the need for risk-based surveillance [9-11]. In this patient, disrupted longitudinal follow-up preceded presentation with advanced bilateral disease; however, this temporal association does not demonstrate that the disease would have been detected earlier, prevented, or altered by a specific surveillance strategy. Rather, the observation is consistent with the broader literature supporting continued urologic follow-up in high-risk neurogenic bladder populations.

From a management perspective, the clearest lesson of this case is the difficulty of achieving definitive source control once bilateral renal destruction, severe systemic illness, and complex anatomy coexist. Nephrectomy remains the definitive treatment for many patients with diffuse XGP, but contemporary surgical literature emphasizes that these operations can be technically demanding and associated with substantial perioperative morbidity [6,12]. In this patient, bilateral nephrectomy was considered but was not feasible because of severe musculoskeletal deformities, positioning limitations, clinical instability, and prohibitive operative risk. Percutaneous nephrostomy provided decompression and drainage but could not substitute for definitive surgical source control. Thus, the case demonstrates how therapeutic options may become markedly constrained in

advanced bilateral disease rather than establishing that an earlier intervention would necessarily have changed the outcome.

A broader implication is that continuity of urologic care remains important for patients with neurogenic bladder who are at risk for upper urinary tract deterioration [9-11]. However, this case cannot determine whether a particular surveillance schedule or earlier intervention would have prevented XGP or improved this patient's outcome. Future studies should better define risk-based surveillance and management strategies in high-risk neurogenic bladder populations (Figure 4); these questions remain secondary to the principal case-specific lesson of severe bilateral XGP with limited surgical feasibility.

This report has the limitations inherent to a single-case observation, including limited generalizability and inability to establish causality, preventability, or the effect of alternative surveillance or treatment strategies. Its value lies in illustrating the convergence of advanced bilateral XGP, severe systemic deterioration, and restricted surgical options in a patient with spina bifida and neurogenic bladder.

Conclusions

This case illustrates the severe clinical burden of advanced bilateral XGP in a patient with spina bifida and neurogenic bladder. The principal contribution is an example of profound bilateral renal destruction complicated by septic shock, dialysis-dependent renal failure, and limited feasibility of definitive surgical source control. Recurrent infection, obstruction, and disrupted longitudinal follow-up were present and are consistent with recognized risk factors in the literature, but causality, preventability, and the potential benefit of earlier intervention cannot be inferred from this single case. The case reinforces the clinical importance of ongoing risk-based urologic surveillance while emphasizing the formidable management constraints of advanced bilateral XGP.

Department and Institution Where Work Was Done

Department of Internal Medicine, Howard University Hospital, Washington, DC, USA.

Informed Consent

Consent was obtained.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Jang TL, McKoy T, Hakim J, Polenakovik HM. Xanthogranulomatous pyelonephritis – A diagnostic and therapeutic dilemma. *Am J Med Sci.* 2023;365(3):294-301
2. Guennouni A, Laridi A, Maslouhi K, et al. Xanthogranulomatous pyelonephritis: A case report and literature review. *Radiol Case Rep.* 2025;20(10):5004-9
3. Prasad P, Sudha S, Niranjana G, et al. Xanthogranulomatous pyelonephritis in the tropics. *Discoveries (Craiova).* 2025;13(3):e212
4. Mynarski NJ, Shen L, Negrete LM. I saw the “bear paw” sign – Massive renal xanthogranulomatous pyelonephritis. *Clin Imaging.* 2023;93:70-74
5. Pais JS, Rocha MB, Muglia VF, et al. Xanthogranulomatous pyelonephritis: Case series – Clinical, radiologic, therapeutic, and histological aspects. *Urol Ann.* 2022;14(4):383-88
6. Harley F, Wei G, O’Callaghan M, et al. Xanthogranulomatous pyelonephritis: A systematic review of treatment and mortality in more than 1000 cases. *BJU Int.* 2023;131(4):395-407
7. Ritter S, Bieri U, Caruso J, et al. A rare case of successful conservative treatment of an extensive xanthogranulomatous pyelonephritis: Case report and review of the literature. *Clin Case Rep.* 2025;13(8):e70699
8. Fairchild RJ, Aksenov LI, Hobbs KT, et al. Medical management of neurogenic bladder in patients with spina bifida: A scoping review. *J Pediatr Urol.* 2023;19(1):55-63
9. Brownrigg N, Lorenzo AJ, Rickard M, Dos Santos J. The urological evaluation and management of neurogenic bladder in children and adolescents-what every pediatric nephrologist needs to know. *Pediatr Nephrol.* 2024;39(2):409-21
10. Fazelinia H, Ding H, Taylor D, et al. Stratification of neurogenic bladder risk in spina bifida using the urinary peptidome. *Am J Physiol Renal Physiol.* 2024;326(2):F241-F48
11. Ginsberg D, Gousse A, Keays-White D, et al. Adult neurogenic lower urinary tract dysfunction: AUA/SUFU guideline. *J Urol.* 2021;206(5):1097-105
12. Faridi MS, Sridhar K, Bhatia D. Simple nephrectomy for Xanthogranulomatous pyelonephritis – Is it really simple? *Trop Doct.* 2025;55(3):244-46
13. Alsaadi A, Alghamdi AA, Akkielah L, et al. Epidemiology and clinical characteristics of *Morganella morganii* infections: A multicenter retrospective study. *J Infect Public Health.* 2024;17(3):430-34
14. Asogan M, Kim HY, Kidd S, et al. *Candida parapsilosis*: A systematic review to inform the World Health Organization fungal priority pathogens list. *Med Mycol.* 2024;62(6):myad131