

Postoperative Encrusted Cystitis After Robot-Assisted Simple Prostatectomy: A Case Report

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Abstract

Encrusted cystitis is an uncommon disorder characterized by adherent mineral deposits on inflamed bladder mucosa. We report a 73-year-old man with persistent dysuria, hematuria, urinary frequency, weak stream, and fluctuating pyuria after robot-assisted simple prostatectomy. Day 1 denotes the first diagnostic cystoscopy. Cystoscopy showed extensive encrustation involving the prostatic surgical bed, bladder neck, and nearly all bladder walls; computed tomography demonstrated mural calcification and bladder-wall thickening. Urine pH reached 8.80 on day 15. Transurethral resection and debridement on day 16 removed more than 15 cm² of visibly involved mucosa. Histopathology showed inflammation and calcium deposition, and analysis of the removed encrustations identified calcium phosphate (apatite). A bladder aspirate grew *Enterococcus faecalis* at 100,000 colony-forming units/mL. *Corynebacterium urealyticum* was not isolated after 72-hour incubation on standard media or detected in the removed deposits; targeted molecular testing was unavailable. The cause of the encrustation therefore remained unconfirmed, with both an infectious mechanism and postoperative dystrophic calcification considered. Treatment included debridement, temporary urinary drainage, susceptibility-supported levofloxacin, a 14-day empirical course of intravenous vancomycin, and eight weeks of oral L-methionine. No infusion reactions or serious systemic adverse effects were recorded. Follow-up urine cultures were negative. Cystoscopy on day 117 showed no visible encrustations, although mucosal inflammation remained. By day 131, symptoms had improved substantially, with the International Prostate Symptom Score decreasing from 22 to 6, while pyuria persisted. This case illustrates the need to investigate a structural cause of persistent urinary symptoms after prostate surgery and to distinguish the diagnosis of encrusted cystitis from confirmation of its etiology. Improvement followed combined treatment; the separate contributions of empirical vancomycin and urinary acidification could not be determined.

Categories: Urology, Internal Medicine, Infectious Disease

Keywords: encrusted cystitis, enterococcus faecalis, postoperative complication, robotic prostatectomy, vancomycin therapy

Introduction

Encrusted cystitis is a rare inflammatory disorder in which mineral deposits form firmly adherent plaques on the bladder mucosa. Patients may present with dysuria, urinary frequency, hematuria, and persistent or recurrent pyuria, findings that can be mistaken for a routine urinary tract infection. In the classical infectious form, urease-producing organisms, particularly *Corynebacterium urealyticum*, hydrolyze urea to ammonia, raising urine pH and promoting precipitation of struvite and carbonated apatite. Prior instrumentation, catheterization, and urothelial injury are recognized predisposing factors [1]. Cases have been described after transurethral and open prostate surgery [2,3].

Calcification after surgery is not always caused by infection. Dystrophic calcification may develop within injured or necrotic tissue and may resemble or coexist with encrusted cystitis [4]. Mineral composition and microbiological findings should therefore be interpreted alongside the clinical, endoscopic, and histopathological features. Recognizing the structural lesion is important because antimicrobial treatment alone may not address adherent deposits or postoperative tissue injury.

We report extensive postoperative encrusted cystitis after robot-assisted simple prostatectomy. The deposits extended beyond the surgical bed and bladder neck across nearly all bladder walls. Although one urine sample was markedly alkaline, *C. urealyticum* was not recovered after 72-hour incubation on standard media or detected in the removed encrustations. The case illustrates the distinction between recognizing encrusted cystitis and establishing its cause, as well as the need to assess symptoms, cultures, and mucosal recovery separately during follow-up.

Case Presentation

Clinical history

A 73-year-old man was evaluated for persistent urinary frequency, dysuria, hematuria, weak urinary stream, and a sensation of incomplete bladder emptying after prostate surgery. His medical history included

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coronary artery disease, hypertension, obesity, and longstanding lower urinary tract symptoms. Before surgery, he had received tamsulosin-containing therapy. Day 1 denotes the first diagnostic cystoscopy for the persistent postoperative symptoms; subsequent time points are reported relative to that assessment.

Approximately six months before day 1, prostate volume was 181 cm³ and postvoid residual urine was 93 mL. Baseline urine microscopy showed 15,000 leukocytes/mL at pH 6.40, with no erythrocytes reported. Serum creatinine was 107 µmol/L. Urinary cell counts were measured using the Nechiporenko method (local upper reference limits: 4,000 leukocytes/mL and 2,000 erythrocytes/mL). Published reference intervals of 4.6–8.0 for urinary pH [5] and 62–115 µmol/L for serum creatinine in adult men [6] are provided for context; laboratory-specific intervals for these two tests were unavailable. Urinary pH is dimensionless.

A preoperative urine culture showed no bacterial growth, so the baseline leukocyturia did not constitute microbiologically confirmed infection. He received a single 2 g dose of cefazolin for prophylaxis within 60 minutes before surgery and underwent robot-assisted simple prostatectomy using a transvesical approach with adenoma enucleation 147 days before day 1. Prostate histopathology showed benign prostatic hyperplasia. A urethral catheter was inserted during surgery and remained in place for seven days. Urinary symptoms and hematuria persisted. A subsequent bacterial urine culture obtained eight days after prostatectomy (139 days before day 1) also showed no growth despite 125,000 leukocytes/mL. During the later diagnostic assessment, he denied fever, chills, and renal colic. The abdomen was soft, and there was no costovertebral-angle tenderness.

Diagnostic assessment

Cystoscopy on day 1 demonstrated adherent encrustation involving the prostatic surgical bed and bladder neck, with widespread extension across nearly all bladder walls and associated edematous, friable mucosa. Pelvic computed tomography (CT) on day 5 showed linear and coarse calcific deposits along the inner bladder wall, measuring up to 5.7 × 5.4 cm with attenuation of 314–576 Hounsfield units. Bladder wall thickness was 7–14 mm, with perivesical inflammatory change; the bladder was incompletely distended (Figure 1).

Both cystoscopy and CT supported a diagnosis of postoperative encrusted cystitis. The main competing explanation was dystrophic postoperative calcification of injured tissue. Bladder stones were less likely because the deposits adhered to the bladder wall. An infection-associated process remained clinically plausible because markedly alkaline urine (pH 8.80) had been documented one day before debridement in the setting of recent urological instrumentation.

Urinary leukocyte counts were not consistently elevated before debridement. Samples collected on days 5 and 10 contained 1,250 leukocytes/mL, within the local reference range, despite erythrocyte counts of 3,000,000/mL and 500,000/mL, respectively. On day 15, urinary pH was 8.80 with 2,500 leukocytes/mL. This was a midstream voided specimen collected in a sterile container and delivered to the laboratory within two hours. The value represents a single documented episode of marked alkalinity rather than evidence of sustained alkaline urine.

Routine blood chemistry, including renal function tests, was within reference limits. Ionized calcium measured on day 15 was 1.29 mmol/L, within the published reference interval of 1.12–1.32 mmol/L [7]. In view of the unremarkable initial biochemical findings, the treating team did not pursue serum phosphate or parathyroid hormone testing. A comprehensive urinary metabolic profile was not available. Ultrasonography on day 1 showed no upper urinary tract abnormalities. The CT findings are shown in Figure 1.

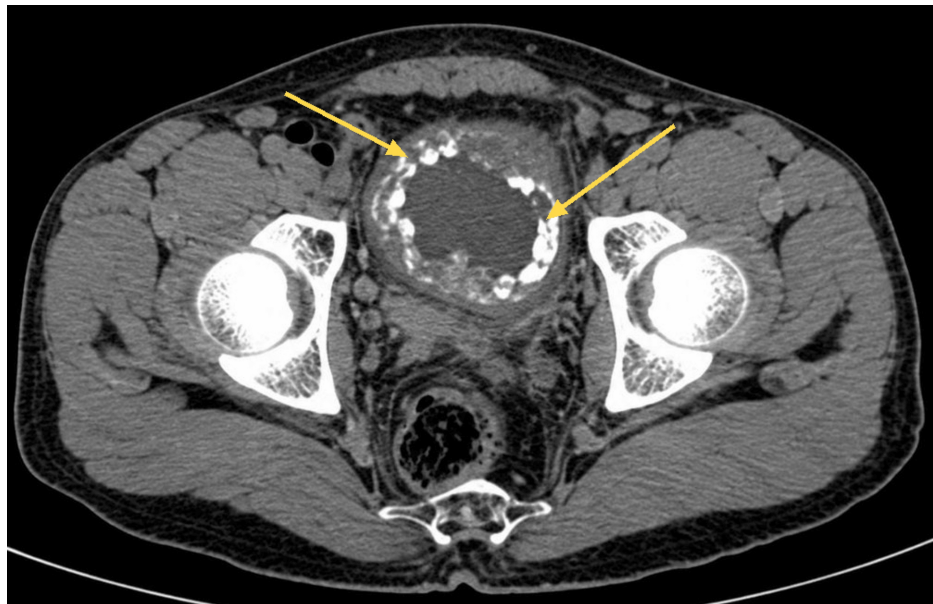


FIGURE 1: Axial pelvic computed tomography showing bladder-wall encrustation

Axial pelvic computed tomography obtained on day 5 demonstrates coarse calcific deposits along the inner bladder wall (yellow arrows), with associated bladder-wall thickening. The bladder is incompletely distended. Day 1 denotes the first diagnostic cystoscopy.

Endoscopic intervention, histopathology, and microbiology

Debridement took place on day 16 (15 days after the first diagnostic cystoscopy) because the patient was unable to undergo the procedure immediately and requested a delay. Operative inspection confirmed extensive encrustation involving the prostatic surgical bed and bladder neck and distributed across nearly all bladder walls. Transurethral resection and debridement removed encrusted and inflamed tissue, including deposits from both lateral walls and the anterior bladder wall. The total visibly involved mucosal area was estimated intraoperatively to exceed 15 cm². Biopsy material was obtained. A 22-Fr three-way urethral catheter was placed and was removed on day 22.

Histopathological examination of the bladder biopsy demonstrated fibromuscular tissue with inflammatory-cell infiltration and calcium deposits. Postoperative analysis of the removed encrustations identified calcium phosphate (apatite). Microbiological examination of the encrustations did not detect *C. urealyticum*. The biopsy, mineral analysis, imaging, and cystoscopy supported encrusted cystitis, but its microbial cause remained uncertain.

A bladder aspirate obtained during the day 16 procedure grew *Enterococcus faecalis* at 100,000 colony-forming units (CFU)/mL. The isolate was reported susceptible to ampicillin, ciprofloxacin, and levofloxacin; minimum inhibitory concentrations and a vancomycin susceptibility result were not provided. The specimen was incubated for 72 hours on standard culture media. *C. urealyticum* and other urease-producing organisms were not isolated. Targeted polymerase chain reaction (PCR) or other molecular testing for *C. urealyticum* was unavailable because of limited resources. The *E. faecalis* culture documented bacteriuria during the clinical episode but did not establish the cause of the encrustation.

Additional evaluation at a specialized tuberculosis center yielded negative urine PCR for *Mycobacterium tuberculosis* and a negative Mantoux tuberculin skin test. A fungal urine culture, collected on the same day as a routine bacterial urine culture, showed no fungal growth. These investigations did not identify an alternative infectious cause.

Therapeutic interventions

Levofloxacin (500 mg orally every 24 hours for approximately 10 days) was initiated empirically for a suspected urinary tract infection on day 16, the day of endoscopic debridement. The bladder aspirate culture, reported on day 22, subsequently confirmed that the *E. faecalis* isolate was susceptible to levofloxacin, and treatment was continued to complete the course. Persistent urinary symptoms did not, by themselves, indicate resistance of the cultured isolate.

On day 30, vancomycin was initiated empirically at 15 mg/kg per dose intravenously every 12 hours for 14 days. The treating physicians selected this course because urinary symptoms persisted after debridement and the extensive encrustations, together with the earlier alkaline urine sample, raised concern for possible *C. urealyticum*-associated disease. This was an individualized empirical decision, without microbiological confirmation of that organism. Follow-up assessments and infusion monitoring recorded no infusion-related reactions or serious systemic adverse effects. The prescribed dose remained unchanged throughout the course, and renal function remained clinically stable.

L-methionine was started on day 30 at 500 mg orally three times daily (1.5 g/day) and continued for eight weeks. Urinary pH was already 6.65 at initiation. The intention was to maintain an acidic urinary environment during mucosal recovery and potentially reduce further phosphate precipitation. The duration was selected by the treating physicians. Management therefore combined debridement, temporary urinary drainage, antimicrobial therapy, and urinary acidification.

Follow-up and outcomes

Cystoscopy on day 22 showed diffuse postoperative mucosal inflammation and fibrinous/sloughing changes at the treated sites. The recorded filling volume was 140 mL, with first urge at 80 mL. Postoperative progress notes documented normal temperature and stable hemodynamics. Separate voided urine cultures collected on days 25 and 79 showed no growth.

Despite negative follow-up cultures, pyuria increased to 3,000,000 leukocytes/mL on day 30, then decreased to 750,000/mL on day 36 and 250,000/mL on day 82. Serum creatinine was 98 $\mu\text{mol/L}$ on day 95 (published adult male reference interval: 62–115 $\mu\text{mol/L}$).

Follow-up cystoscopy on day 117, approximately 14 weeks after debridement, showed postoperative healing changes with residual mucosal edema, friability, and hypervascularity but no visible encrustation. The recorded filling volume was 145 mL, with first urge at 80 mL. These and the day 22 volumes were procedural observations rather than formal measurements of bladder capacity or compliance. Ultrasonography on day 117 again showed no upper urinary tract abnormalities. On day 131, the urinary leukocyte count had decreased to 30,000/mL and the erythrocyte count to 500/mL, within the local reference range for erythrocytes.

Symptoms had improved substantially by the last clinical follow-up on day 131. The International Prostate Symptom Score (IPSS), based on the validated seven-item urinary symptom index [8], decreased from 22 on day 1 to 6 on day 131, corresponding to a change from severe to mild lower urinary tract symptoms [9]. On day 1, maximum urinary flow rate (Q_{max}) was 8 mL/s with a voided volume of 105 mL and a postvoid residual volume of 40 mL. On day 131, Q_{max} was 14 mL/s with a voided volume of 190 mL and a postvoid residual volume of 35 mL. These results document improvement with mild residual symptoms, rather than a completely asymptomatic state. Representative cystoscopic appearances before debridement and at follow-up are shown in Figure 2A and Figure 2B, respectively.

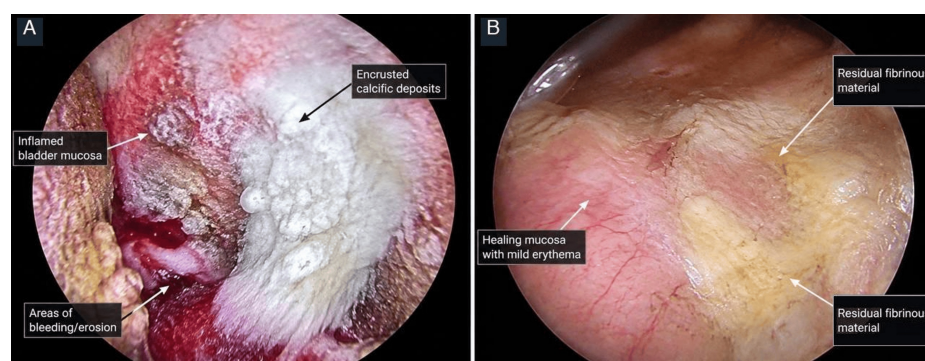


FIGURE 2: Cystoscopic appearance before debridement and during follow-up after treatment

(A) Pre-debridement cystoscopy showing extensive whitish encrustation over inflamed bladder mucosa with areas of bleeding/erosion. (B) Follow-up cystoscopy on day 117 (approximately 14 weeks after debridement) showing healing changes with residual mucosal edema, friability, and hypervascularity, with residual fibrinous material and no visible encrustation. Day 1 denotes the first diagnostic cystoscopy.

The clinical timeline is summarized in Table 1.

Time point	Clinical event
191 days before day 1	Lower urinary tract symptoms; prostate volume 181 cm ³ ; postvoid residual 93 mL; leukocyturia.
Before prostatectomy	Preoperative bacterial urine culture showed no growth.
147 days before day 1	Robot-assisted simple prostatectomy after a single 2 g dose of cefazolin given within 60 minutes before surgery; benign prostatic hyperplasia on histology. A catheter was inserted during surgery and retained for seven days.
139 days before day 1	Eight days after prostatectomy: 125,000 urinary leukocytes/mL; bacterial urine culture showed no growth.
Days 1 and 5	Cystoscopy showed encrustation of the surgical bed, bladder neck, and nearly all bladder walls; computed tomography confirmed mural calcification and inflammatory changes. Day 1 ultrasonography showed no upper urinary tract abnormalities.
Day 15	Urinary pH 8.80 in a midstream sample delivered to the laboratory within two hours; ionized calcium 1.29 mmol/L.
Day 16	Transurethral resection and debridement of >15 cm ² of visibly involved mucosa after a delay requested by the patient; biopsy and bladder aspirate obtained. Empirical levofloxacin initiated.
Day 22	Catheter removal; cystoscopy showed postoperative inflammation and healing changes. Aspirate culture reported levofloxacin-susceptible <i>Enterococcus faecalis</i> ; levofloxacin continued.
Day 25	Voided urine culture showed no growth during the reported levofloxacin course and before vancomycin initiation.
Day 30	Empirical vancomycin initiated for 14 days and L-methionine for eight weeks. No infusion reactions, serious systemic adverse effects, or vancomycin dose changes were recorded.
Day 79	Voided urine culture showed no growth.
Day 117	Cystoscopy showed residual inflammation without visible encrustation. Ultrasonography showed no upper urinary tract abnormalities.
Day 131	Symptoms substantially improved; IPSS 6 compared with 22 on day 1. Urinary erythrocytes 500/mL and leukocytes 30,000/mL.

TABLE 1: Clinical timeline

Day 1 denotes the first diagnostic cystoscopy. Urinary pH is dimensionless (published reference interval: 4.6-8.0[5]). The published ionized calcium reference interval is 1.12-1.32 mmol/L [7]. Local upper reference limits for urine microscopy were 4,000 leukocytes/mL and 2,000 erythrocytes/mL. Published intervals are provided for context and are not laboratory-specific.

IPSS: International Prostate Symptom Score.

Serial urinary pH and leukocyte and erythrocyte counts are presented in Table 2.

Time point	Urinary pH	Leukocytes (cells/mL)	Erythrocytes (cells/mL)
191 days before day 1	6.40	15,000	Absent
139 days before day 1	7.08	125,000	1,000,000
Day 5	7.10	1,250	3,000,000
Day 10	7.10	1,250	300,000
Day 15	8.80	2,500	Too numerous
Day 18	6.00	650,000	500,000
Day 22	6.56	112,500	2,000,000
Day 25	6.69	112,500	Too numerous
Day 30	6.65	3,000,000	2,000,000
Day 36	5.94	750,000	1,650,000
Day 82	5.93	250,000	23,750
Day 120	6.00	81,250	15,000
Day 131	5.82	30,000	500

TABLE 2: Serial urinary pH and leukocyte and erythrocyte counts
Day 1 denotes the first diagnostic cystoscopy. Urinary pH is dimensionless (published reference interval: 4.6–8.0[5]); the laboratory-specific pH interval was unavailable. Urinary cell counts were measured using the Nechiporenko method; local upper reference limits were 4,000 leukocytes/mL and 2,000 erythrocytes/mL. "Too numerous" means too numerous to count.

Figure 3A shows the peri-treatment urinary pH trajectory, while Figure 3B summarizes the timing of endoscopic intervention, antimicrobial therapy, urinary acidification, and follow-up cultures. Urine had already become acidic before L-methionine was initiated on day 30.

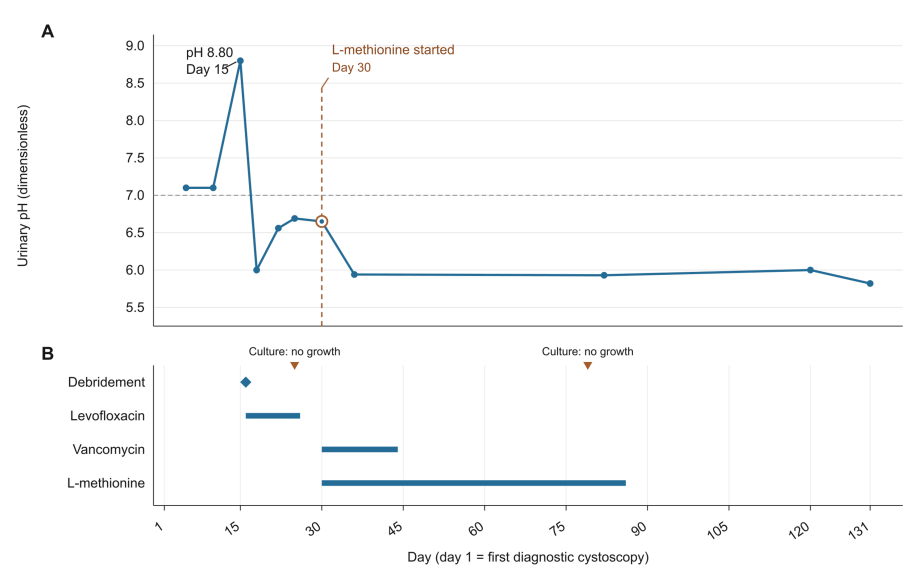


FIGURE 3: Peri-treatment urinary pH trajectory and timing of major therapeutic interventions
(A) Serial urinary pH (dimensionless; published reference interval: 4.6–8.0[5]). The pH of 8.80 was recorded on day 15, one day before debridement. The horizontal dashed line denotes neutral pH 7; the vertical dashed line marks initiation of L-methionine on day 30. (B) Timing of debridement, antimicrobial treatment, urinary acidification, and negative follow-up cultures on days 25 and 79. Treatment bars show reported approximate course durations. Both panels use the same time axis; day 1 denotes the first diagnostic cystoscopy.

Urine and encrustation microbiology results are summarized in Table 3.

Time point	Specimen	Result
Before prostatectomy	Urine	Bacterial culture: no growth.
139 days before day 1	Urine collected eight days after prostatectomy	Bacterial culture: no growth.
Day 16	Bladder aspirate	<i>Enterococcus faecalis</i> , 100,000 CFU/mL; susceptible to ampicillin, ciprofloxacin, and levofloxacin. <i>Corynebacterium urealyticum</i> and other urease-producing organisms were not isolated after 72-hour incubation on standard media.
Day 16	Removed encrustations	<i>C. urealyticum</i> was not detected on microbiological examination.
Day 25	Voided urine	No growth; collected during levofloxacin treatment and before vancomycin initiation.
Day 79	Voided urine	No growth.

TABLE 3: Urine and encrustation microbiology

Day 1 denotes the first diagnostic cystoscopy. Negative culture results are qualitative; no numerical reference interval applies. Targeted molecular testing for *C. urealyticum* was unavailable. A fungal urine culture, collected on the same day as a routine bacterial urine culture during the diagnostic workup, also showed no fungal growth.

CFU: colony-forming units.

Discussion

Diagnosis and possible mechanisms

The diagnosis rested on adherent bladder-wall deposits, CT findings, and inflammation with calcium deposition on biopsy. Their distribution across nearly all bladder walls distinguished the lesion from a free bladder stone or calcification confined to the prostatic cavity. Urinary leukocyte counts were intermittently within the local reference range before debridement, showing that the structural lesion could not be assessed from pyuria alone.

The mineral analysis identified calcium phosphate (apatite); struvite was not reported. Apatite can occur in urease-associated encrustation, but calcium phosphate deposition can also accompany postoperative tissue injury and dystrophic calcification [1,4]. A single pH of 8.80 raised suspicion of a urease-producing organism without proving sustained urease activity. Ionized calcium of 1.29 mmol/L and otherwise normal routine blood chemistry, including renal function tests, did not suggest hypercalcemia or renal impairment. Normal results on these tests do not exclude local dystrophic calcification or all urinary metabolic risk factors. A retained suture or hemostatic material could also act as a local nidus, although such a nidus was not established in this case.

Microbiological interpretation

C. urealyticum is slow-growing and may be missed by conventional urine culture. Recovery depends on incubation time, suitable media, and recognition of small colonies [10,11]. In a study of 302 urine specimens, a selective medium recovered 14 isolates compared with four on blood agar [12]. Another study found higher recovery with selective than conventional media, particularly among asymptomatic patients [13]. These studies demonstrate method-dependent detection; they do not provide a universal false-negative rate for patients with encrusted cystitis. Molecular detection has also identified *C. urealyticum* in a patient whose initial conventional cultures were negative [14].

In the present case, 72-hour incubation on standard media and examination of the removed encrustations did not identify *C. urealyticum*. Those results reduce microbiological support for a corynebacterial cause, although they do not exclude it with certainty. Targeted molecular testing was unavailable because of limited resources, and selective culture and urease testing were not documented. No other urease-producing organism was isolated, but this does not establish exclusion of fastidious organisms such as *Ureaplasma* species without directed testing. Negative fungal culture and the negative tuberculosis investigations provided no evidence for those alternative causes in the tests performed. The etiology therefore remains unconfirmed.

E. faecalis was recovered from the bladder aspirate, but it is not the usual urease-producing pathogen associated with encrusted cystitis. Its isolation documented bacteriuria during a symptomatic episode, rather than proving that it caused the mineral deposits. Likewise, clinical improvement after treatment cannot retrospectively establish an undetected corynebacterial infection.

Antimicrobial treatment and duration

Levofloxacin was started empirically for suspected urinary tract infection and continued after susceptibility testing supported activity against the isolated *E. faecalis*. Culture-directed reassessment is consistent with complicated urinary tract infection guidance [15]. However, susceptibility of this isolate cannot be extrapolated to an organism that was not recovered. Glycopeptides commonly retain activity against *C. urealyticum*; one series of 40 isolates found all susceptible to vancomycin, whereas 38 were resistant to levofloxacin [16]. This supports the choice of coverage when corynebacterial disease is suspected, but not an automatic indication for vancomycin after a negative culture.

The treating physicians selected a 14-day empirical vancomycin course after debridement because symptoms persisted and the extensive deposits and earlier marked alkalinity remained concerning. Treatment durations in the literature vary with disease extent. Pagnoux et al. described cystoscopic removal of most bladder deposits combined with 14 days of teicoplanin in microbiologically confirmed corynebacterial cystitis [17]. By contrast, the series of Sakhi et al., composed predominantly of encrusted pyelitis, used a median antibiotic duration of nine weeks [11]. Neither report validates a fixed 14-day vancomycin regimen for culture-negative postoperative cystitis; the course here was an individualized decision.

The day 25 urine culture was already negative during levofloxacin treatment and before vancomycin began on day 30. Subsequent culture negativity therefore cannot be attributed to vancomycin. Debridement, drainage, antimicrobial treatment, and tissue healing occurred together, preventing assessment of the separate benefit of empirical glycopeptide therapy. No infusion reactions or serious systemic adverse effects were recorded, and the available creatinine values were 107 µmol/L at baseline and 98 µmol/L on day 95.

Urinary acidification

L-methionine was intended to maintain acidic urine during recovery and potentially limit phosphate precipitation. European Association of Urology (EAU) guidance lists methionine as an option for urinary acidification in infection-stone management, with a weak recommendation; this is not a validated treatment schedule for postoperative encrusted cystitis [7]. Sabiote et al. reported prolonged L-methionine use for persistent encrusted uropathy after urine had already become acidic, providing a clinical precedent for maintenance acidification [18]. Their case involved upper tract disease and a different treatment course.

Here, urine pH had fallen to 6.00 on day 18 and remained below 7 before L-methionine was started on day 30. The initial pH change cannot therefore be attributed to methionine. The eight-week duration reflected the treating physicians' judgment during mucosal recovery, rather than an established regimen. Its additional benefit after debridement remains uncertain.

Recovery and follow-up

No visible encrustations remained at cystoscopy on day 117, and symptoms improved from severe to mild by day 131. Qmax increased from 8 to 14 mL/s, but the voided volume also increased from 105 to 190 mL. Uroflowmetry is volume dependent, and measurements at a voided volume below 150 mL require cautious interpretation [9]. The findings indicate improvement without establishing normal bladder function or excluding residual outlet obstruction or detrusor underactivity. The small volumes recorded during cystoscopy should not be treated as formal cystometric capacity or compliance measurements.

Pyuria persisted despite negative follow-up cultures and improvement in symptoms and endoscopic findings. Residual mucosal inflammation provides a plausible explanation, although a single case cannot establish its mechanism. Guidance cautions against using pyuria alone to diagnose catheter-associated infection [19]; this principle requires clinical context in a patient who is no longer catheterized. Follow-up every three to six months is recommended to reassess urinary symptoms and IPSS. Recurrent or worsening symptoms or signs of infection should prompt earlier evaluation and additional investigations.

Context in the literature

A focused narrative search of PubMed-indexed reports using the terms “encrusted cystitis,” “encrusted uropathy,” “prostatectomy,” and “dystrophic calcification” identified related cases after prostate surgery [2-4]. This was not a systematic review, and no claim of a first reported occurrence is made. The contribution of the present case is the diffuse postoperative distribution of deposits, the unresolved infectious versus dystrophic mechanism, and the documented differences between symptom improvement, culture results, and mucosal recovery.

Limitations

Molecular testing for *C. urealyticum* was unavailable, and the microbial etiology remained unconfirmed despite examination of urine and removed encrustations. Combined treatment with debridement, antibiotics, drainage, and acidification prevented assessment of the individual contributions of vancomycin and L-methionine to clinical improvement.

Conclusions

Persistent urinary symptoms after prostate surgery warrant assessment for a structural cause when the clinical course remains unexplained. In this patient, diffuse adherent bladder-wall deposits, inflammation on biopsy, and calcium phosphate (apatite) supported postoperative encrusted cystitis, while its etiology remained unconfirmed. Negative cultures did not exclude every possible infectious contributor, but neither the isolated *Enterococcus faecalis* nor a single alkaline urine sample established the cause. Postoperative dystrophic calcification remained a plausible alternative.

Visible encrustations were absent at cystoscopy on day 117, and symptoms improved substantially by day 131, with the International Prostate Symptom Score decreasing from 22 to 6. Improvement followed debridement, temporary drainage, antibiotics, and urinary acidification. The separate benefits of empirical vancomycin and L-methionine could not be determined. Follow-up should assess symptoms, microbiological findings, and urinary function together, with longer observation needed to evaluate sustained recovery and recurrence.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Boburkhon Muzaffarkhonov, Nodir Dadakhanov

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Supervision: Nodir Dadakhanov

Disclosures

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