

Bilateral Alkaline-Encrusted Pyelitis Mimicking Staghorn Calculi: A Case Report

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Abstract: Alkaline-encrusted pyelitis is a rare urinary tract disorder characterized by calcific encrustation of the urothelial lining of the renal collecting system. It is commonly associated with urease-producing organisms, particularly *Corynebacterium urealyticum*, and may be difficult to diagnose because routine urine cultures can be negative. We report a case of a 51-year-old female presenting with hematuria for 3 months, altered sensorium and decreased oral intake, with severe renal dysfunction and hyperkalemia. Initial CT was reported as bilateral staghorn calculi. On subsequent review, the calcifications were noted to represent encrustation involving the bilateral pelvicalyceal systems and upper ureters. The patient was managed medically along with supportive therapy. Renal function subsequently improved. This case highlights the importance of recognizing urothelial calcific encrustation on CT and differentiating it from conventional staghorn calculi, particularly in patients presenting with hematuria and renal dysfunction.

Keywords: Alkaline-encrusted pyelitis; Urothelial encrustation; Staghorn calculi; *Corynebacterium urealyticum*; Hematuria

1. Introduction

Alkaline-encrusted pyelitis is a rare urinary tract disorder characterized by calcific encrustation of the urothelial lining of the renal pelvis, calyces, and ureters. It is most commonly associated with urease-producing organisms, particularly *Corynebacterium urealyticum*. Urease-mediated hydrolysis of urea increases urinary alkalinity and promotes precipitation of magnesium ammonium phosphate and calcium phosphate, resulting in calcification of the urothelial surface.

The condition is associated with several predisposing factors, including previous urological procedures, urinary catheterization, prolonged hospitalization, prior antibiotic exposure, immunosuppression, and pre-existing urothelial disease. Clinical manifestations are often nonspecific and may include hematuria, pyuria, flank pain, urinary obstruction, urinary debris, and deterioration of renal function. Fever may be absent.

Diagnosis can be challenging because *C. urealyticum* is slow-growing and may not be detected by routine urine culture. Prolonged incubation and appropriate culture media may be required. Consequently, a negative routine urine culture does not exclude an encrusted urinary tract infection when the clinical and radiological findings are suggestive.

Non-contrast computed tomography (CT) is the key diagnostic examination. It demonstrates the distribution, morphology, and relationship of calcification to the urothelial surface. Unlike conventional renal calculi, which are typically located within the collecting-system lumen, urothelial encrustation appears as mural, plaque-like, curvilinear, or irregular calcification conforming to the walls of the renal pelvis, calyces, and ureters. Extensive encrustation may mimic conventional renal calculi, including staghorn calculi.

Recognition of these imaging features is therefore essential for accurate diagnosis and appropriate management.

We report a case of bilateral upper urinary tract calcific encrustation initially interpreted as bilateral staghorn calculi in a patient with severe renal dysfunction, hematuria, and a negative routine aerobic urine culture. The case emphasizes the importance of recognizing the distribution and morphology of calcification on CT and distinguishing urothelial encrustation from intraluminal nephrolithiasis.

2. Case Presentation

The patient presented with a 3-month history of hematuria, altered sensorium, and decreased oral intake. There was no history of fever. The patient had a history of previous hip replacement surgery, and a Foley catheter was in situ.

Initial laboratory investigations demonstrated severe renal dysfunction and electrolyte abnormalities. Serum urea was 212 mg/dL, blood urea nitrogen was 99.4 mg/dL, serum creatinine was 7.4 mg/dL, sodium was 131 mmol/L, potassium was 7.0 mmol/L, and chloride was 129 mmol/L.

Urinalysis showed 4+ albumin and numerous red blood cells. Routine aerobic urine culture showed no growth. Urine pH, pyuria, struvite crystalluria, prolonged culture for *C. urealyticum*, and chemical analysis of the calcific material were not available.

Initial CT and USG imaging was reported as demonstrating bilateral staghorn calculi involving the pelvicalyceal systems. On retrospective review, the calcification was not confined to the collecting-system lumen. Instead, it followed the contours of the bilateral renal pelvises, calyces, and upper ureters, producing irregular mural and plaque-like calcific

encrustation. The distribution and morphology favored urothelial encrustation over conventional free or staghorn calculi.

A thin rim of perinephric fluid was noted along the anterior cortex, measuring up to 8.5 mm in maximum thickness over a length of approximately 4.8 cm. This collection communicated with another collection in the posterior pararenal space. Mild surrounding fat stranding was present, with thickening of the anterior and posterior pararenal fascia.

The previously described left renal and perinephric collections were also noted. These findings were interpreted in conjunction with the surrounding inflammatory changes and the extensive calcific abnormality of the collecting system.

Additional hyperdense encrustation was observed surrounding the Foley catheter bulb.

The patient was managed medically with intravenous meropenem 500 mg every 8 hours and linezolid 600 mg every 12 hours, together with supportive therapy and other supplements. No dialysis was performed. No cystoscopy, ureteroscopy, urinary diversion, or other endoscopic intervention was undertaken. The encrustation was not removed and was not available for chemical or microbiological analysis.

Follow-up laboratory investigations demonstrated substantial improvement in renal function.

3. Case Discussion

Alkaline-encrusted pyelitis is a rare urinary tract infection characterized by calcific encrustation of the urothelial wall of the renal pelvis, calyces and ureters. It is most commonly associated with urease-producing organisms, particularly *Corynebacterium urealyticum*. The clinical presentation is nonspecific and may include hematuria, pyuria, flank pain, urinary obstruction and deterioration of renal function. Fever is not a constant finding.

Our case demonstrates an extensive bilateral upper urinary tract encrustation initially reported as bilateral staghorn calculi. On review of the CT images, the calcification was seen to follow the contours of the bilateral pelvicalyceal systems and upper ureters, favouring mural urothelial encrustation rather than conventional intraluminal calculi. Extensive encrustation of the collecting system may closely resemble a staghorn calculus and can therefore present a diagnostic challenge.

The distribution and morphology of calcification are important imaging features in differentiating encrusted pyelitis from conventional nephrolithiasis. Urothelial encrustation may appear as thin, linear or irregular calcification closely related to the wall of the renal pelvis, calyces or ureters. In extensive disease, the calcification may become bulky and lobulated, producing a staghorn-like appearance. CT is particularly useful in demonstrating the relationship of the calcification to the urothelial surface.

In our patient, associated inflammatory changes were also present. A thin rim of perinephric fluid was seen along the anterior cortex, communicating with a collection in the posterior pararenal space. Mild surrounding fat stranding with thickening of the anterior and posterior pararenal fascia was noted. These findings suggest associated inflammatory changes in the setting of extensive urinary tract disease.

Another notable finding was the presence of hyperdense encrustation around the Foley catheter bulb. This finding raises the possibility of a more extensive encrusting process involving the urinary tract.

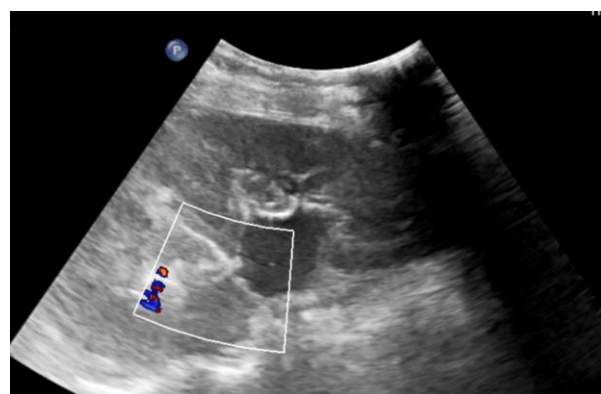


Figure 1: USG image of the right kidney, showing linear mural calcification along the dilated pelvicalyceal system, with twinkling artifacts suggesting calcification



Figure 2: USG image of the left kidney, showing mural calcifications of the pelvicalyceal system. However, no significant hydronephrosis is noted in these images.



Figure 2: Axial section of the abdomen (CT) – shows linear hyperdense calcific encrustation of the pelvicalyceal systems on both sides.

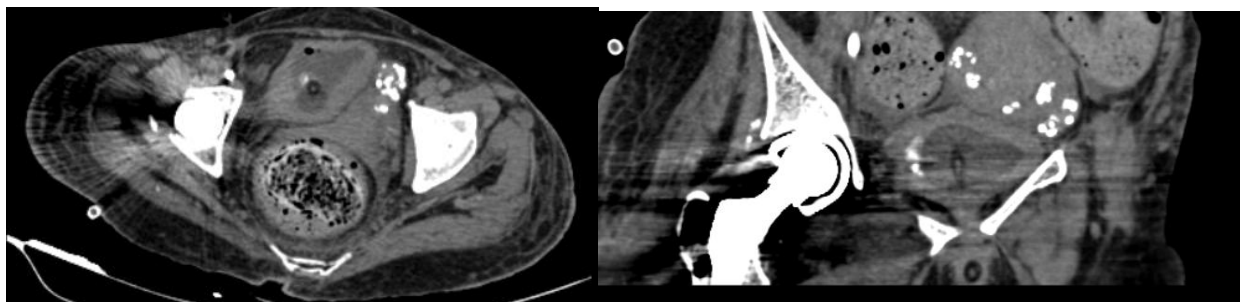


Figure 3: Axial and coronal sections of the abdomen on CT imaging, through the urinary bladder shows few calcific foci in the urinary bladder, and along the Foley's bulb



Figure 4: Sagittal section of the abdomen (CT) – shows few well defined collections in the perinephric space near the lower pole of the kidney.

The patient presented with prolonged hematuria and severe renal dysfunction, with an initial serum creatinine of 7.4 mg/dL and potassium of 7.0 mmol/L. Routine aerobic urine culture showed no growth. Standard urine culture may fail to identify *C. urealyticum*, as the organism is slow growing and may require prolonged incubation on appropriate media. In the present case, however, prolonged culture was not available and no causative organism was isolated.

Diagnosis of alkaline-encrusted pyelitis is usually supported by the combination of characteristic imaging findings, alkaline urine, pyuria and identification of a urea-splitting organism. In our case, urine pH and microbiological analysis of the encrustation were not available. Therefore, the diagnosis was based predominantly on the characteristic radiological appearance and clinical presentation.

Treatment of alkaline-encrusted pyelitis generally consists of appropriate antibiotic therapy along with urinary acidification and, when required, removal of the encrustations or urinary drainage procedures. Our patient was treated medically with intravenous meropenem 500 mg every 8 hours and linezolid 600 mg every 12 hours along with supportive therapy. No dialysis or endoscopic intervention was performed.

Follow-up investigations showed significant improvement in renal function. Serum creatinine decreased from 5.1 mg/dL on 29 April 2026 to 1.6 mg/dL on 10 May 2026, while urea decreased from 121.3 to 53 mg/dL and BUN from 56.7 to 25 mg/dL. This improvement occurred without dialysis or removal of the encrustations.

Our case highlights the importance of recognizing urothelial calcification on CT and differentiating it from conventional

renal calculi. In patients presenting with hematuria and renal dysfunction, particularly when calcification involves the wall of the renal pelvis, calyces and ureters, alkaline-encrusted pyelitis should be considered as a differential diagnosis. Careful review of the distribution and morphology of calcification may help avoid misdiagnosis as staghorn calculi and facilitate appropriate microbiological evaluation and management

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