Case Report

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Infected calcium oxalate stone leading to pyogenic spondylodiscitis and bilateral lower limb weakness: a case report

Cheng-Yang Wu, MD[a], Chi-Shin Tseng, MD[b,*], Yuan-Ju Lee, MD[b,*]

Introduction and importance: It is rare for calcium oxalate renal stone, presented mainly in sterile urine, to result in urinary tract infection. The stone-related infection could develop spondylodiscitis, causing neurological deficits. To date, there are no reports about calcium oxalate partial staghorn stone and spondylodiscitis.

Case presentation: A 62-year-old male suffered from haematuria, fever, and flank pain. He came to the urology outpatient department, where acute pyelonephritis was diagnosed, and a left partial staghorn stone was seen on computed tomography. Oral antibiotics were prescribed with improvement. Two weeks after antibiotics treatment, he developed bilateral lower limb weakness and numbness under the nipple level. He was brought to the emergency department, where the spine MRI revealed T2–T3 spondylodiscitis with epidural abscess and spinal cord compression. He underwent T2–T3 spine operation with improvement in muscle power and hypesthesia. The culture of the surgical lesion yielded Citrobacter koseri, the same as the urine culture obtained at his first visit. Left-side percutaneous nephrolithotomy was performed 1 month after with successful stone removal and resolution of pyuria. Stone analyses reported calcium oxalate. Follow-up MRI showed marked improvement with resolution of spondylodiscitis.

Clinical discussion: Urinary tract infection resulting from partial staghorn stone, with additional hematogenous spread causing spondylodiscitis, is scarcely discussed. The authors illustrated a case with calcium oxalate stone, belonging to sterile Jensen's classification type 1. However, a urinary tract infection could be seen in urine stasis or obstruction.

Conclusion: With accurate diagnosis and essential interventions, the patient had immediate neurological improvement and reached disease-free status.

Keywords: case report, citrobacter koseri, spondylodiscitis, staghorn stone, urinary tract infection

Introduction

Urolithiasis is the most common urological pathology, with an estimated incidence rate of around 10–15%[1]. The connection between renal stones and infections has been identified with urinary alkalinity, phosphate calculi, and increased urinary ammonia[2]. Infection stones would mainly form staghorn, which could be complete or partial[3]. Usually, they are composed of struvite (magnesium ammonium phosphate), linked to some urease-producing bacteria, including Proteus, Providencia, Staphylococcus, and Klebsiella[4].

HIGHLIGHTS

- Infected calcium oxalate partial staghorn stone leads to pyogenic spondylodiscitis is a rare occurrence.
- This patient was initially diagnosed with acute pyelonephritis but later developed bilateral lower limb weakness and numbness due to spinal cord involvement.
- Spondylodiscitis was not anticipated, highlighting the difficulty in diagnosing the underlying cause.
- Surgical intervention resulted in improved muscle power and hypesthesia, with culture confirming pathogen corresponding with the previous urine culture.
- This case highlights the connection between urinary tract infection, calcium oxalate stones, and the development of pyogenic spondylodiscitis.

Calcium oxalate is the predominant stone composition, with a 60% occurrence rate, while struvite accounts for 7%[5]. Notably, calcium oxalate stones are rarely associated with urinary tract infections.

Spondylodiscitis is a rare disease characterized by an infection or inflammation of the intervertebral disc[6,7]. The aetiology could be a transient bacteremia causing seeding to the spine or direct extension from an infected lesion, even as a complication of urinary tract infection[8]. Hematogenous spread of bacteria is the most common cause of pyogenic spondylodiscitis and is reported chiefly caused by *Staphylococcus aureus* (>50%)[9].

[a]Department of Medical Education, National Taiwan University Hospital and [b]Department of Urology, National Taiwan University Hospital, National Taiwan University, Taipei, Taiwan

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*Corresponding author. Address: Department of Urology, National Taiwan University Hospital, 7 Chung-Shan South Road, Taipei, Taiwan. Tel.: +886 223 123 456, Ext 51 2135; fax: +886 223 219 145. E-mail: leeyuanju@hotmail.com (Y-J. Lee), and clifford1987tcs@gmail.com (C-S. Tseng).

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The association between renal stones and spondylodiscitis is rarely discussed. Rehman *et al.*[10] have reported that a 74-year-old woman with ureteric stone causing acute flank pain received ureteric stent initially, which led to *E. coli*-associated bacteremia and subsequent spondylodiscitis. The case Rehman and colleagues presented did not experience any neurological deficit. We demonstrate a calcium oxalate partial staghorn case, which later developed thoracic spondylodiscitis and bilateral lower limb muscle asthenia without prior stone interventions and systemic bacteremia. The case has been reported in line with the SCARE criteria[11].

Case presentation

The patient was a 62-year-old male without systemic medical diseases and denied social history. He experienced gross haematuria, accompanying intermittent fever up to 38.5°C, and acute left flank pain. He was treated at our urology clinic where urine analysis revealed pyuria [white blood cell (WBC): ≥ 100/high power field (HPF)], and ultrasonography showed a left renal stone. Acute pyelonephritis was impressed and oral cefuroxime was prescribed. Urine culture was obtained and yielded *Citrobacter koseri*. An abdominal computed tomography scan showed a left partial staghorn renal stone (Fig. 1). Retrograde intrarenal surgery was suggested, yet the patient hesitated.

Two weeks later, he developed acute bilateral lower limbs weakness with numbness under the nipple level. He then came to our emergency department. The physical exam showed decreased bilateral lower limbs muscle power (4/5). Lab data yielded leukocytosis (WBC: 9.22 K/μl Seg:78.1%) and pyuria (WBC: ≥ 100/HPF). Spine MRI revealed T2–T3 discitis with pyogenic infection resulting in retropulsion and compression of the spinal cord (Fig. 2).

Figure 1. Computed tomography scan of the abdomen and pelvis in coronal view showed left partial staghorn renal stones measuring 1.8 cm, with Hounsfield unit (HU) around 1100.

Thoracic myelopathy due to T2–T3 spondylodiscitis with epidural abscess and spinal cord compression was diagnosed. The patient received T2–T3 laminoplasty/pediculectomy/facetectomy and T2–3 discectomy to remove the epidural abscess and spinal cord decompression, as well as bilateral T1, T2, and T4 posterior fixation with pedicle screw. His muscle power and hypesthesia had improved after the operation. The culture of the surgical lesion yielded *Citrobacter koseri*, the same as the prior urine culture result.

Postoperative laboratory data showed resolved leukocytosis and pyuria, and follow-up MRI showed marked improvement with spinal stenosis and epidural abscess (Fig. 3). Cefixime for 6–8 weeks was prescribed.

One month after the spine surgery, urine analysis showed mild pyuria (WBC: 10–19/HPF), and Ceftriaxone was administered 3 days before nephrolithotomy. Left-side percutaneous nephrolithotomy was performed smoothly with no complication. Ceftriaxone was kept until discharge. The stone analysis reported the composition of calcium oxalate. Outpatient department follow-ups showed bilateral no hydronephrosis and negative pyuria in urine analysis.

Discussion and conclusions

We presented a case of unusual infective renal calculi, ensuing hematogenous pyogenic spondylodiscitis with epidural abscess at the T2–T3 level causing bilateral lower limb muscle weakness. This case demonstrates the resolution of symptoms after surgical management.

Calcium oxalate stones have rarely formed into staghorn stones, less likely to develop spondylodiscitis. Even so, the stone compositional analysis is crucial in defining the aetiology and determining treatment[12]. Identification of the stone compositions could be classified according to Jensen's system[13]. Type I stones are defined as those found in sterile and acidic urine, commonly with calcium-based calculi. Type II stones are infection calculi and alkaline urine, whereas type III stones result from metabolic abnormalities, such as uric acid-based calculi. According to this classification, calcium oxalate, classified as Jensen type 1, is present in sterile urine and less associated with infection. However, conditions causing urine stasis or urinary tract obstruction could result in urinary tract infection[14].

The infection mechanism hypothesizes that the preceding formed-calcium oxalate stone encounters episodes of urinary tract infection, which turn the sterile calculi into an infection staghorn stone, complicating with systemic bacteremia and subsequent general illness. Few cases describe urolithiasis-related urinary tract infection (UTI)-causing spondylodiscitis. Rehman *et al.*[10] reported that a 74-year-old female underwent retrograde intrarenal surgery for ureteric stone, which later developed Escherichia coli bacteriemia and subsequent spondylodiscitis at T10/T11. Secondary spondylodiscitis of L3, L4, and L5 as a complication of urosepsis related to extracorporeal shock wave lithotripsy was observed by Gallina *et al.*[15]. Those studies shared the element of pre-existing intervention to stones, causing bacteriemia and urosepsis, or

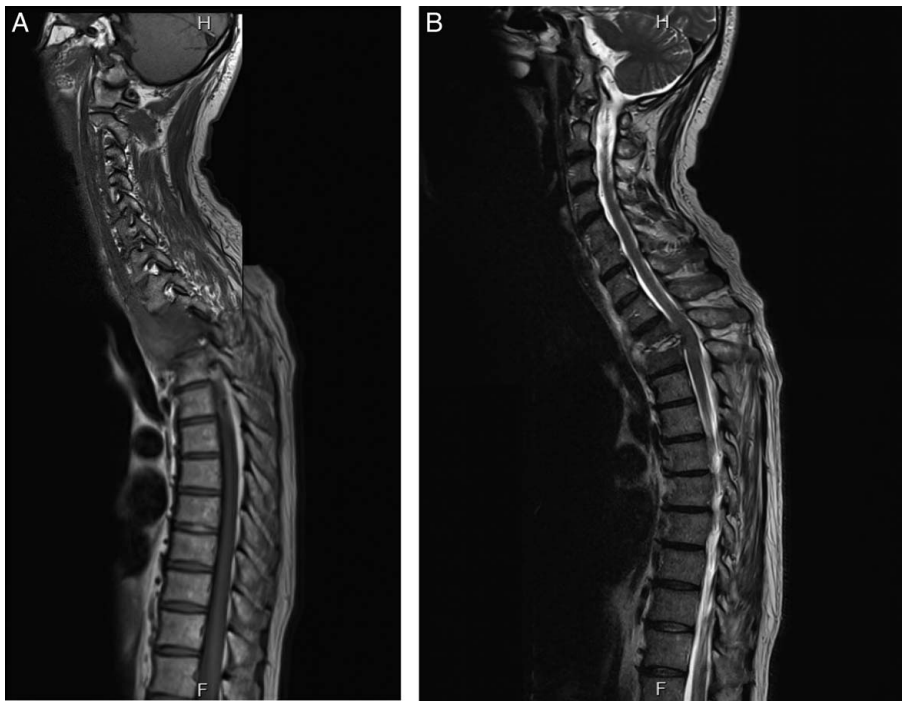

Figure 2. Contrast-enhanced MRI of the spine showed irregular endplate change and disc destruction with heterogeneous paraspinal enhancing soft tissue and abscess formation at T2–3 level [(A) T1WI sagittal view; (B) T2WI sagittal view].

local abscess formation invading kidney-level vertebrae, ultimately resulting in spondylodiscitis.

Contrarily, there was no precedent stone intervention and no observed systematic bacteremia in our case. However, distant infection to T2–T3 pyogenic spondylodiscitis was presented, implying the hematogenous spread of bacteria[10]. In conclusion, the infective renal calculi have the potential to progress into bacteremia from urinary tract infection, leading to severe diseases.

In our case, the surgical wound and urine culture yielded a Gram-negative bacillus of the Enterobacteriaceae family named Citrobacter. Among its species, the most commonly isolated from human and infection-associated are *C. koseri* and *C. freundii*[16]. Citrobacter infection is rarely identified and almost exclusively presented in infants, and immune-compromised hosts, resulting primarily in meningitis[17]. Nonetheless, in adult patients, *C. freundii* was frequently isolated from the gastrointestinal tract,

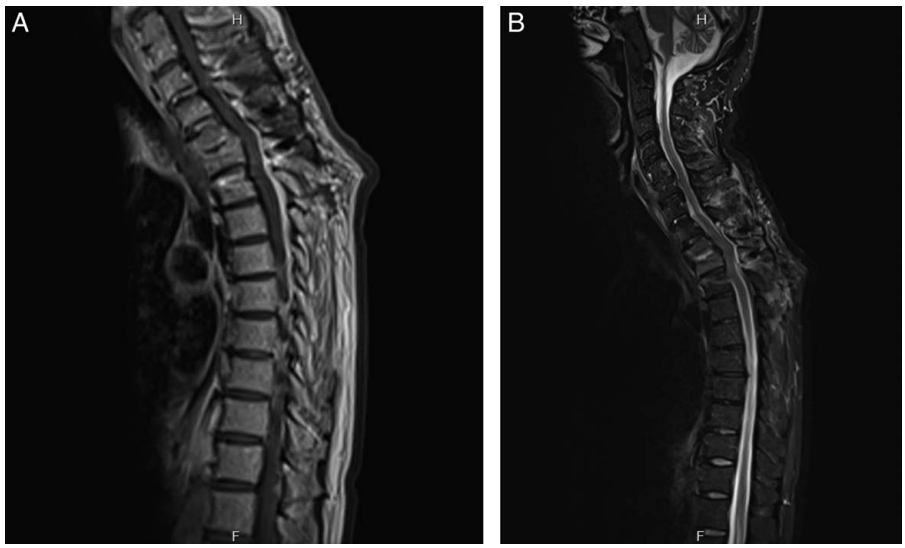

Figure 3. Contrast-enhanced MRI of the spine showed residual enhancement at T2 and T3 vertebrae and fluid accumulation in the intervertebral disc [(A) T1WI sagittal view; (B) STIR sagittal view]. Moreover, no more spinal stenosis and epidural components were seen.

and *C. koseri* most commonly leads to urinary tract infections, most of which are pyelonephritis[18].

Few cases describe *Citrobacter koseri*-related UTI and spondylodiscitis. Hayati *et al.*[18] reported a 75-year-old female with lower urinary tract symptoms and *Citrobacter koseri* bacteremia complicated by spondylodiscitis. Stewart and colleagues demonstrated a case of staghorn calculus and *C. koseri* UTI progressing to a peri-nephric abscess and empyema. They both pointed out the potential for systemic illness in patients with *C. koseri* UTI, especially in the setting of nephrolithiasis[17].

Calcium oxalate partial staghorn with *Citrobacrter koseri* urinary tract infection is uncommon, let alone the complication of pyogenic spondylodiscitis. This case highlights the association between *Citrobacrter koseri* infection and hematogenous-related spondylodiscitis; moreover, signifying the importance of possible severe complications and neurological sequelae in staghorn stone patients.

Ethical approval

None declared.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution

C.-Ya.W. analyzed the patient data, wrote the main manuscript text, and prepared figures. C.-S.T. and Y.-J.L. provided expertise and edited the manuscript. All authors read and approved the final manuscript.

Conflicts of interest disclosure

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Data availability

Data sharing does not apply to this article as no datasets were generated or analyzed during the current study.

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