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# Spinal epidural abscess post-ureteroscopy: a case report

Stephanie N. Washington[1], David D. Kim[2] and Luke F. Reynolds[2*]

## Abstract

**Background** Spinal epidural abscess (SEA) is an uncommon but potentially life-threatening infection that requires prompt identification and treatment. Early SEAs may present with non-specific symptoms, including fever, chills, headache, and back pain, before progressing to neurologic dysfunction. To our knowledge, there have been no reports of immunocompetent patients with a history of recurrent UTIs presenting with SEA following ureteroscopy (URS) and lithotripsy (LL).

**Case presentation** In this article, we present two cases of spinal abscess formation after ureteroscopy in patients with histories of recurrent urinary tract infections but without previous spinal pathology. Both patients required procedural intervention and one patient sustained lasting neurological deficits.

**Conclusions** It is important that urologists be aware of this possible complication to ensure the early diagnosis of the disease to facilitate appropriate management.

**Keywords** Spinal epidural abscess, Urinary tract infection, Ureteroscopy, Laser lithotripsy

## Background

Spinal epidural abscess (SEA) is an uncommon but serious infection occurring in less than 10 in every 10,000 hospital admissions [1]. Risk factors associated with SEAs include immunosuppression, bacteremia, intravenous drug usage, and direct instrumentation within the central nervous system (CNS) [2]. SEA can often progress rapidly and lead to severe neurologic deficits if not recognized and addressed promptly [3]. SEA can be difficult to diagnose without a high level of clinical suspicion. Presenting symptoms are often nonspecific and may be misconstrued, especially in patients with early-stage SEA without neurologic dysfunction, for more common infections such as urinary tract infections (UTIs), osteomyelitis, meningitis, and inter-vertebral disc prolapse [4]. MRI is the preferred method for diagnosing SEAs. Delayed diagnosis of SEA can have an adverse impact on management effectiveness and patient outcomes, increasing the risk of paralysis or other long-term neurologic symptoms [1].

There are a handful of published cases of epidural abscesses following a urologic procedure. However, the majority of these cases presented with SEA following transrectal ultrasound (TRUS) guided biopsy with patients presenting with symptoms of fever, back pain, or neurologic symptoms one day to several months post-procedure [5–8]. There have also been a limited number of case reports on SEA following an *Escherichia coli (E. coli)* UTI in immunocompetent patients or patients with existing spinal fracture [9, 10]. To our knowledge, there have been no reports of patients with a history of

*Correspondence:
Luke F. Reynolds
lukereynolds@bsd.uchicago.edu
[1]The Pritzker School of Medicine, Biological Sciences Division, University of Chicago, Chicago, IL 60637, USA
[2]Section of Urology, Department of Surgery, University of Chicago Medical Center, Chicago, IL 60637, USA

recurrent UTIs presenting with SEA following ureteroscopy (URS) and lithotripsy (LL). In this article, we present two cases of spinal abscess formation status post ureteroscopy and laser lithotripsy. Both patients provided consent for publication of their cases. This work has been reported in line with the CARE guidelines [11].

## Case presentation

A 64-year-old male with a past medical history of obesity, hyperlipidemia, benign prostate hyperplasia, bulbar urethral stricture, recurrent UTIs, and nephrolithiasis had recently completed a course of antibiotics for a UTI that was diagnosed 6 weeks prior to URS. He was scheduled to undergo a urethroplasty and, after discussion, elected to manage his bilateral renal calculi before proceeding with treatment of his urethral stricture. Four days before his scheduled surgery date, he had a negative urine culture. He received peri-operative antibiotic prophylaxis with cefazolin and underwent an uncomplicated bilateral URS under general anesthesia using a 12/14 French ureteral access sheath and flexible ureteroscope. Laser lithotripsy was not required as small stones were found in lieu of the expected 9 mm stone reported on CT; stones were removed with the Nitinol basket, and bilateral ureteral stents were placed. The procedure lasted approximately 65 min and was well tolerated. There were no intra-operative or immediate post-operative complications. He did not have spinal anesthetic as part of his surgery.

On post-operative day (POD) 1, the patient presented with bilateral flank pain, chills, and nausea. Urinalysis (UA) was notable for 3 + blood and 3 + protein without nitrite and reflex culture, a diagnostic test automatically ordered due to an abnormal UA, resulted in < 10,000 CFU/ml of mixed bacterial flora inconsistent with infection. He was not prescribed antibiotics and was treated with oral analgesics and tamsulosin. On POD 4, he remained asymptomatic, and his stents were removed. He was not prescribed antibiotics for stent removal.

Almost a month after stent removal, he presented to the emergency department with bilateral lower extremity weakness and numbness with lower back pain. He was found to be febrile to 38 °C with a leukocytosis. Physical exam was significant for tenderness to palpation over lumbar spine, complete loss of strength in lower extremities bilaterally from the waist down, decreased sensation to touch and painful stimuli from thighs to knees, and absent patellar reflexes bilaterally. Cross-sectional imaging of the abdomen and pelvis revealed severe spinal canal stenosis at L2-L3 and L4-L5. No urologic pathology was identified within the CT scan. A cervical/thoracic/lumbar spine MRI demonstrated T5-T6 osteomyelitis and an epidural abscess with mass effect on the cord. Cord edema and severe central stenosis were both present. The infectious disease service and neurosurgery were consulted, and the patient was started on intravenous antibiotics and underwent an emergent bilateral T5-T6 laminectomies and medial facetectomies with drainage of the epidural abscess.

Cultures from his epidural space collected during surgery were positive for *Enterococcus faecalis (E. faecalis)*. His blood cultures drawn upon admission grew no bacteria. The SEA was believed to be from a genitourinary source based on the patient's recent urologic surgery and history of recurrent UTIs and with his most recent positive urine culture having grown *E. faecalis*. The patient improved clinically and was eventually transferred to the general medical floor where he began inpatient acute rehabilitation. His course was complicated by acute urinary retention requiring an indwelling urinary catheter for 9 months post-SEA drainage prior to progressing to CIC. With continuous therapy, the patient was able to ambulate with a walker 3 months following treatment. However, he continues to have neurologic deficits, requiring mobility assist devices including a power wheelchair and walker. He continues to follow up with urology to date.

The second case involves a 47-year-old female with a past medical history of obesity, hyperlipidemia, recurrent UTIs with multidrug resistant *E. coli*, and nephrolithiasis presented to the emergency department (ED) with urinary urgency and hematuria. Urinalysis was notable for large leukocyte esterase, negative nitrite, no bacteria, 11–25 RBC/hpf, and > 100 leukocytes/hpf. Reflex culture grew 60,000-100,000 CFU/mL of *E. coli*. She was treated for a presumed UTI and was compliant with her antibiotics but returned to the emergency room 3 days later for severe right flank pain, persistent urinary urgency, and hematuria. CT scan demonstrated multiple obstructing left kidney stones (Fig. 1).

Her urinalysis again demonstrated microscopic hematuria and pyuria with few bacteria with no growth on reflex culture. The next day, urology was consulted, and the patient underwent an uncomplicated left URS and LL and ureteral stent placement under general anesthesia with gentamicin for surgical prophylaxis. The surgeon used an 11/13 French ureteral access sheath and flexible ureteroscope to visualize the stone within the left renal pelvis. The stone was well fragmented using a holmium laser and larger stones were removed with a basket. The procedure lasted approximately 54 min and was well tolerated. There were no intra-operative or immediate post-operative complications. She also did not have spinal anesthetic as part of her surgery. She was discharged on the day of surgery with one week of sulfamethoxazole-trimethoprim double strength and prn hydrocodone-acetaminophen for pain.

The patient re-presented to the emergency room 2 days after discharge with right flank pain. Urinalysis was

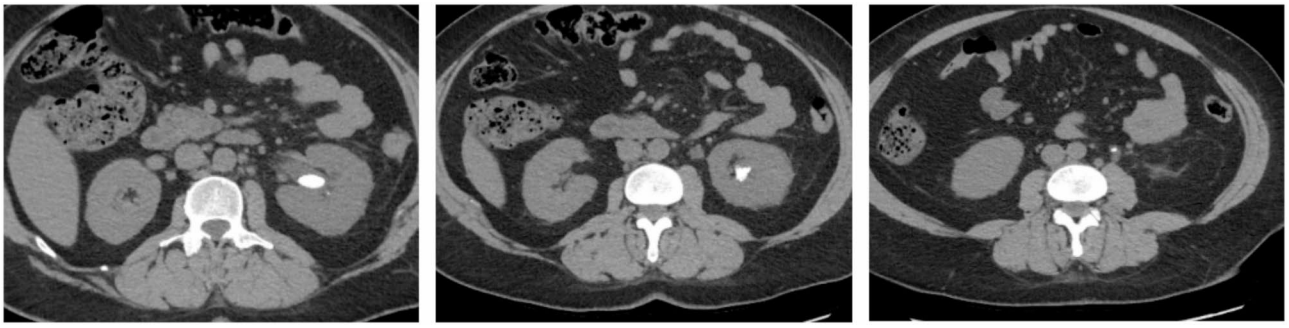

**Fig. 1** CT upper abdomen and pelvis on initial presentation. Imaging demonstrated mild to moderate hydronephrosis with four 2–3 mm stones in the mid to distal left ureter as well as additional non-obstructing renal stones

leukocyte esterase positive and nitrite negative with > 100 RBCs/hpf, 11–25 leukocytes/hpf, and few bacteria. Reflex culture grew 60,000-100,000 CFU/ml of *E. coli*; however, she left before receiving treatment. She then presented on POD 9 with nausea and severe, intermittent, right-sided flank pain. Urinalysis was again suspicious for infection and reflex culture grew 10,000–50,000 CFU/ml of *E. coli*. Imaging included an abdominal x-ray that confirmed appropriate stent positioning and a CT abdomen pelvis which demonstrated absent hydronephrosis with small non-obstructing calculi in the left collecting system. She then presented to urology clinic for ureteral stent removal 3 weeks post-surgery. Urine dip at time of removal was not concerning for infection and she was given a dose of nitrofurantoin prior to cystoscopic stent removal. Over the next few weeks, the patient visited an outside ED and was admitted to an outside hospital for 5 days for left flank and back pain. At this time, she reportedly had no evidence of infection. CT Urogram revealed multiple nonobstructing stones in the left renal pelvis and a single nonobstructing stone in the distal left ureter. Patient stay was lengthened due to an incidental vascular finding on the CT Urogram but was discharged after passing several stones and achieving adequate pain control.

One month after stent removal, she presented to the ED with worsening continuous back pain that was reported to be most prominent over the lumbar spine and coccyx. She was afebrile with unremarkable lab results. Physical exam revealed no spinal tenderness, no motor or strength deficits, strong lower extremity reflexes, and pain with movement of lumbar spine. CT scan demonstrated L3/4 discitis, not seen in CT scans prior to URS and LL, and she was started on antibiotics and admitted for further evaluation (Fig. 2). No ureteral or renal pathology was identified within the CT scan.

MRI confirmed discitis but was also suspicious for a developing right paraspinal abscess. Neurosurgery was consulted and it was determined that the patient did not require acute surgical intervention. She underwent CT-guided drainage of the abscess with interventional radiology (IR), and the fluid culture resulted in *E. coli*. Urinalysis showed no evidence of pyuria. Patient was discharged home on hospital day 8 with analgesics and IV antibiotics via a PICC line. She experienced no long term neurologic symptoms. Her PICC line was removed 7 weeks after hospitalization. She continues to follow with urology for nephrolithiasis to date.

## Discussion and conclusions

SEAs are caused by an infection of the CNS and are most commonly formed from contiguous spread or hematogenous seeding of bacteria [12]. The classic presenting symptoms of are fever, neck or back pain, and neurological deficit, however only a small percent (8–15%) presents with this triad. In addition, the specificity of these symptoms is low with a third of patients presenting with low back pain alone, two-thirds with back pain and another symptom, and less than a quarter presenting with fever [1]. This discrepancy of presentation is highlighted within this report as one patient presented with the class triad, while the other presented with only back pain without focal neurologic deficits or fever.

A systematic review of 1099 patients found the majority (63.6%) of SEAs were caused by *Staphylococcus aureus*, pathogens such as *E. Coli* and *E. faecalis* were not mentioned. This review also found the most common risk factors of SEAs to include intravenous drug use (22%), diabetes mellitus (27%), and hepatic disease (14%) [13]. An older meta-analysis of 915 patients found *E. Coli* as the causative organism in only 3% of cases [14]. The most favorable management of SEAs involves a decompressive laminectomy and debridement combined with a four to eight week course of systemic antibiotics to prevent a subsequent osteomyelitis [12]. Non-operative management may be pursued if patients are asymptomatic, able to be closely monitored for signs of progression, unable to consent to surgery, or considered to be too high risk for surgery. Of note, medical management alone has been found to have high rates of morbidity (up to 22%

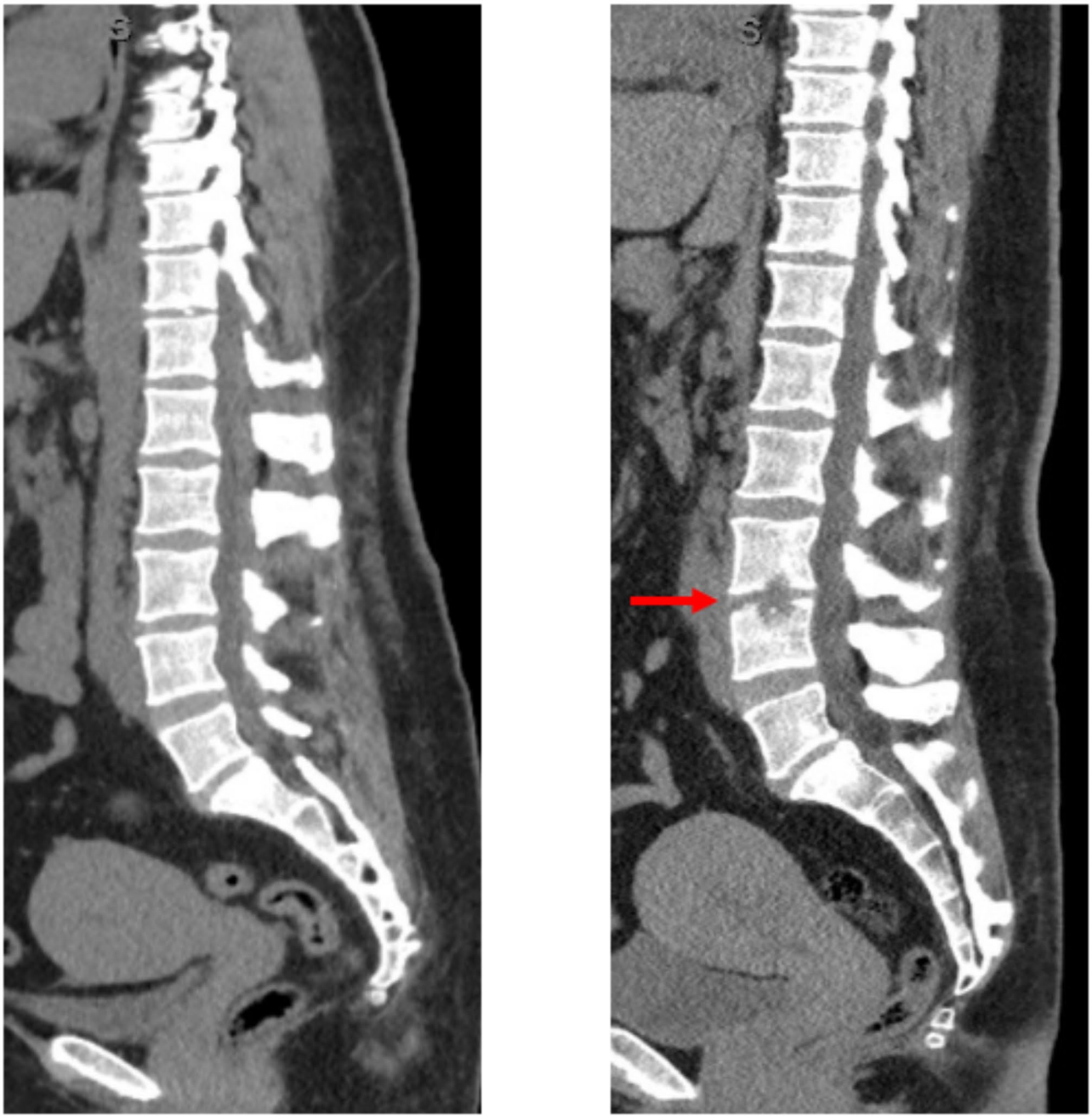

**Fig. 2** Pre- and post-operative CT scan with erosion of the L3/L4 vertebral endplate

permanent paralysis) and failure requiring surgical intervention (> 40%) [12].

Few published cases detailing SEAs after a urologic procedure exist. Of those cases, the majority of SEAs are after a TURP or TRUS and biopsy procedure [5–8]. One report described a patient with SEA following three cycles of spinal anesthesia and intravesical Bacillus Calmette-Guérin therapy for bladder cancer [15]. SEAs following a urologic procedure in a patient with a history of recurrent UTIs is exceedingly uncommon with very few cases described in the literature. The case reports describing SEAs after UTIs have included patients with pre-existing spinal pathologies, disc disease, or who are immunocompromised [9, 10]. One case study, however, detailed a spontaneous SEA in a previously healthy patient with no risk-factors [16]. While neither of the patients included in this report had common risk factors for SEA, both had a history of recurrent UTIs and presented after removal of ureteral stents following recent URS.

Similar to previously published cases, both patients presented with a SEA caused by the same organism as a previous UTI. Interestingly, neither of the patients was found to have positive blood cultures or UAs that indicated bacteriuria at the time of SEA diagnosis. The pathogenesis of a SEA following cystoscopy could be similar to bacteremia following dental procedures. It has been well documented that tissue trauma during dental instrumentation may lead to disruption of capillary bed or small vessels could serve as an entrance for virulent bacteria to spread hematogenously before seeding in the epidural space [17, 18]. It has been previously proposed that hematogenous spread of infection is similar to spread of malignant prostatic cells described in Baston's hypothesis which offered a rationale for prostate cancer's tendency to metastasize to the spine. Cells or infection may spread to the subdural space via the internal vertebral venous plexus due to lack of posterior valves and contiguous flow with the prostatic venous plexus [8, 19]. This theory could also be applied to the case in which infection is spread due to small traumas to ureteral or vesicular vasculature secondary to instrumentation during urologic procedures.

It is important to limit the risk of peri-operative infection by ensuring patients have a negative urine culture prior to surgery, administering appropriate peri-operative antibiotic prophylaxis (AP), and carefully considering the administration of peri-stent removal antibiotics in high-risk patients with history of recurrent UTIs. Best practice guidelines state that AP may be considered cystoscopy with manipulation, such as stent removal, especially when other patient risk factors are present [20].

Clinicians should remain vigilant by maintaining a high clinical suspicion for SEAs in urology patients presenting with fever and back pain to ensure the early diagnosis of the disease to facilitate timely and appropriate management.

#### Abbreviations

| | |
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| SEA | Spinal epidural abscess |
| CNS | Central nervous system |
| UTIs | Urinary tract infections |
| TRUS | Transrectal ultrasound |
| URS | Ureteroscopy |
| LL | Laser lithotripsy |
| POD | Post-operative day |
| UA | Urinalysis |
| ED | Emergency department |
| AP | Antibiotic prophylaxis |

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SNW, DDK, and LFK wrote the main manuscript text. DDK prepared Figs. 1 and 2. All authors thoroughly reviewed and revised the manuscript. All authors approve each version of the manuscript.

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#### Data availability

All data supporting the findings of this case report are presented within the manuscript. No additional data are available.

## Declarations

#### Ethics approval and consent to participate

Institutional Review Board (IRB) approval was not required for this case report, per our institution's ethics committee, as it involved a retrospective analysis of two patient's clinical data with no identifiable information. Patient consent was obtained prior to all procedures mentioned within this report in accordance with hospital policy. This study was conducted in accordance with the Declaration of Helsinki.

#### Consent for publication

Patient informed consent was obtained for publication of the information within this study.

#### Competing interests

The authors declare no competing interests.

#### Clinical trial number

Not applicable.

#### Ethics declaration

Not applicable.

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