Published in final edited form as:

Infect Dis Clin Pract (Baltim Md). 2018 July ; 26(4): 237–239. doi:10.1097/IPC.0000000000000565.

Spinal epidural abscess caused by *Gardnerella vaginalis* and *Prevotella amnii*

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Abstract

We report a case of spinal epidural abscess (SEA) in a 58-year-old woman who had recently been diagnosed with gonococcal infection, but did not receive guideline-recommended therapy. She presented with back pain and signs and symptoms of pelvic inflammatory disease (PID). MRI of the spine demonstrated epidural abscess extending from L4–L5 to T10. She underwent T10–L1 and L3–L4 laminectomies for evacuation of the abscess and *Gardnerella vaginalis* and *Prevotella amnii* were isolated from the abscess fluid cultures. Our case demonstrates SEA as a rare, but morbid complication of PID and highlights the pathogenic potential of the anaerobic flora associated with PID.

Keywords

spinal epidural abscess; pelvic inflammatory disease; Gardnerella; Prevotella; gonorrhea

Case

A 58-year-old female with a history of hypertension, type-II diabetes mellitus, and depression presented to the emergency department (ED) with one week of back pain and lower extremity weakness. The patient had been notified by a sexual partner to seek testing for sexually transmitted infections (STIs) and, sixteen days prior to presentation in the ED, she had sought care from her primary care physician for this reason. At the primary care visit, the patient also complained of vaginal discharge and right second toe pain and was prescribed seven days of prednisone for possible gout flare. Urine was obtained for nucleic acid amplification testing (NAAT) for *Neisseria gonorrhea* and *Chlamydia trachomatis* at this visit and subsequently returned positive for *N. gonorrhea*. The patient was prescribed doxycycline 100 mg tabs to be taken twice daily for seven days. Five days into the course of

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Potential conflicts of interest:

The authors have no conflicts of interest to disclose.

doxycycline she began having severe mid-back pain, bilateral lower extremity weakness, and difficulty ambulating. The patient also noted constant, diffuse abdominal pain. After one week without improvement of her symptoms she presented to the ED. At this time, she denied fevers, chills, nausea, vomiting, diarrhea, urinary symptoms, or vaginal discharge. She denied recent hospitalization, surgery, or medical procedures. She denied a history of intravenous drug use. Home medications included metformin with hemoglobin A1c recently 5.6%.

Physical examination revealed fever of 101.4 °F, blood pressure of 122/97 mmHg, heart rate of 86 beats/min, respiratory rate of 18 breaths/min, and pulse oximetry saturation of 95%. The patient appeared non-toxic. Auscultation of the chest was unremarkable with normal heart sounds, no murmurs, and clear lungs. Palpation of the spine revealed tenderness over the lumbar region. Neurologic examination of the lower extremities revealed bilateral distal weakness (strength 4/5), 1+ reflexes, and intact sensation. Rectal tone was normal. Palpation of the abdomen revealed diffuse tenderness without peritoneal signs and no organomegaly. Pelvic examination was also abnormal with cervical motion tenderness, but no cervical discharge or friability was noted. Skin and joint examinations were unremarkable.

Clinical data were significant for a white blood cell count of 22.4×10^9 cells/liter (78.4% neutrophils, 18.8% lymphocytes), hemoglobin of 12.5 g/dL, platelets of 332×10^9 cells/liter, and erythrocyte sedimentation rate of 101 mm/hr. Total bilirubin was 0.9 mg/dL, AST 87 IU/L, ALT 66 IU/L, and alkaline phosphatase 224 IU/L. Urinalysis was normal. Two sets of blood cultures were negative. HIV testing was negative. NAAT for *N. gonorrhea* and *C. trachomatis* was performed on an endocervical swab from the patient and was negative. Nucleic acid probe assay for Candida species, *Trichomonas vaginalis* and *Gardnerella vaginalis* was also performed and detected *G. vaginalis*. Ultrasound of the abdomen and pelvis showed mild gallbladder wall thickening and a mildly dilated common bile duct (CBD), but no evidence of tubo-ovarian abscess. The CBD dilation was later evaluated with MRCP and was favored to be due to a distal stricture of unknown etiology. MRI of the cervical, thoracic and lumbar spine demonstrated epidural abscess extending from L4–L5 to T10 with significant thecal sac compression, phlegmonous changes extending superiorly as far as the clivus, and abscesses in the paraspinal musculature. There was no evidence of spinal osteomyelitis (Figure 1).

The patient was initiated on vancomycin, ceftriaxone, and doxycycline in the ED and transferred to another institution for urgent neurosurgical evaluation and spinal decompression. Metronidazole was added post-operatively to cover for a potential anaerobic component as well. During transfer, she developed progressive lower extremity weakness and acute urinary retention. She underwent T10–L1 and L3–L4 laminectomies and purulence was observed in the epidural and paraspinal spaces.

Cultures were obtained from both the epidural and the paraspinal fluid collections. Gram stain of both fluids showed moderate polymorphonuclear leukocytes and moderate to many gram negative and gram variable rods. The aerobic culture of the paraspinal abscess demonstrated a few pinpoint colonies on blood agar on the second day of incubation which

were identified as *G. vaginalis* by Matrix Assisted Laser Desorption/Ionization Time of Flight (MALDI-TOF) mass spectrometry (VITEK MS, bioMerieux, Inc.). The anaerobic culture of the epidural abscess grew a moderate amount of the same organism. Additionally, the anaerobic cultures of both fluids grew a strictly anaerobic gram negative rod on Brucella blood agar, which could not be definitively identified by MALDI-TOF. This isolate was subsequently identified as *Prevotella amnii* by sequencing of 16S rDNA.

The patient remained in stable condition post-operatively. The antimicrobial regimen was ultimately changed to ampicillin-sulbactam 3g IV every 6 hours, which was continued for six weeks. She was then transitioned to oral therapy with amoxicillin-clavulanate 875 mg twice daily for an additional four weeks. She had full return to her normal, baseline neurological function.

Discussion

To our knowledge, our case is the first published example of spinal epidural abscess (SEA) caused by pelvic inflammatory disease (PID). The patient initially presented to primary care with a known STI-contact and was diagnosed with an uncomplicated gonococcal infection. However, she was prescribed doxycycline mono-therapy, a treatment that is not recommended based on current CDC guidelines due to the high rates of resistance and lack of proven efficacy.¹ Then, upon presentation to the ED, she displayed signs and symptoms of PID and was diagnosed with an anaerobic SEA due to *G. vaginalis* and *P. amnii*. PID is a known complication of gonococcal cervicitis but can also be caused by mixed bacterial flora in the setting of bacterial vaginosis (BV). In either case, cervical inflammation triggers an ascending polymicrobial infection due to a mix of aerobic and anaerobic commensals.² Thus, one possibility is that our patient was sub-optimally treated for gonorrhea and this resulted in progression to PID. Alternatively, PID may have developed regardless of the treatment chosen or have been unrelated to gonococcus and potentially triggered by an episode of BV.

There are several features which support our suspicion that the SEA originated from infection in the genital tract and additional factors that may have contributed to the pathogenesis of SEA. First, *G. vaginalis* and *P. amnii* were recovered from the SEA and both are associated specifically with the female genital tract. *G. vaginalis* may colonize the lower tract, cause BV, and is associated with PID. *P. amnii* has specifically been associated with BV³ and, in retrospect, has likely been recovered from cases of PID which had been attributed to the highly-related species *P. bivia*.^{4,5} Second, the patient had *G. vaginalis* detected via a DNA probe assay from an endocervical swab obtained in the ED, indicating outgrowth of this organism. Third, the patient met clinical criteria for the diagnosis of PID. Given the abnormal liver imaging and elevated aminotransferases, we also considered a complication of PID known as perihepatitis, or Fitz-Hugh-Curtis syndrome. However, the isolated imaging findings of a thickened gallbladder wall and mildly dilated CBD with distal stricture are atypical in the absence of the more commonly described imaging findings demonstrating inflammation of the anterior liver capsule with adhesions.⁶ Finally, the hematogenous spread of urogenital infections to the spine has been described in other cases as occurring through a network of veins known as Batson's plexus.⁷ Batson's plexus

connects venous drainage of the urogenital tract to vertebral veins and may have been the mode of spread of this patient's infection to epidural and paraspinal spaces. Finally, both diabetes and steroid use have been risk factors associated with SEA and may have contributed to the pathology in this case.⁸

G. vaginalis has been implicated in infections other than BV and PID. It has been associated with chorioamnionitis and postpartum endometritis.⁹ Furthermore, there have been six previously reported osteoarticular infections associated with *G. vaginalis*. These cases include a parietal bone osteomyelitis in a neonate,¹⁰ a hip arthritis in a renal transplant recipient,¹¹ two hip prosthesis infections^{12,13} and two cases of spinal infections. The first spinal infection reported was of a 50-year-old woman who presented with back pain. MRI showed inflammation of the L5-S1 disk space and an epidural abscess. Culture from biopsy of the L5-S1 disc space grew *G. vaginalis* alone.¹⁴ The second case was a 38-year-old woman who initially presented with headache and evaluation with MRI revealed vertebral osteomyelitis, discitis and a small epidural fluid collection at L2–3. Culture from biopsy of L2–L3 grew only *G. vaginalis*.¹⁵ The etiology of these osteoarticular infections was thought to be from either transient *G. vaginalis* bacteremia or through the Batson's plexus, but there was no direct evidence of genital infection in any of these cases.

Our case is notable, as it clearly illustrates anaerobic SEA as a sequela of PID. Most cases of SEA are due to *Staphylococcus aureus*, coagulase-negative staphylococci, streptococcus species, and other aerobic bacteria, whereas anaerobic SEA is relatively rare, representing less than 2% of cases.⁸ As such, empiric therapy for SEA does not typically include anaerobic coverage unless a gastrointestinal source of infection is suspected. Therefore, our case leads us to recommend anaerobic coverage as a component of antimicrobial therapy for SEA whenever PID is the suspected etiology.

Acknowledgments

We are grateful to Dr. Jessie Torgersen for help and suggestions on the manuscript.

Sources of support:

Dr. Culyba is partially supported by NIH Infectious Diseases Training Grant 1-T32-AI-118684.

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Figure 1. MRI of the vertebral column demonstrating diffuse disease

A, B) Lumbar spine study. Sagittal (A) and axial (B) views of post-contrast T1-weighted images showing dorsal epidural (A, arrows) and paraspinal (B, arrow) disease. **C, D)** Thoracic spine study. Sagittal views of pre- (C) and post-contrast (D) T1-weighted images demonstrating dorsal compression (D, arrow) from abscess. The most significant cord compression is at T10-L1 and L3-L4. **E)** Cervical spine study. Sagittal view of post-contrast T1-weighted image demonstrating extension of phlegmon to level of clivus (right-pointing arrow) and dorsal abscess at cervico-thoracic junction (left-pointing arrow).