A 61-year-old male with a past medical history of hypertension initially presented in the emergency department with fever, chest pain, and tachycardia. He was found to have a left lower lobe infiltrate on admission chest x-ray. He also had left lower extremity edema. A CT angiogram of the thorax was negative for pulmonary embolism and a lower extremity Doppler study was negative for deep vein thrombosis. The patient was treated for sepsis secondary to community-acquired pneumonia. His blood cultures grew Streptococcus pneumoniae (S. pneumonia) susceptible to ceftriaxone. Symptoms improved with ceftriaxone and azithromycin. His repeat blood cultures were negative and he was discharged home on a course of cefpodoxime.

The patient returned one month later with fever to 103.5°F, altered mental status, nuchal rigidity and back pain. He was found to have leukocytosis (WBC = 12,600/μL, 86% neutrophils). Physical exam was remarkable for tenderness of the lumbar spine. Lungs were clear to auscultation; heart sounds were of normal intensity without murmurs or gallops and he did not have any focal neurological deficits. CT of the head was unremarkable. A lumbar puncture revealed a white blood cell count of 2680 /μL (60% neutrophils, 20% lymphocytes). The patient was started empirically on ceftriaxone, vancomycin, and ampicillin for suspected meningitis. MRI of the cervical, thoracic and lumbar spine demonstrated spondylodiscitis at the C5-C6 and T5-T6 levels and fasciitis at the L4-L5 level with a posterior epidural phlegmon and an adjacent 2mm abscess. Neurosurgery did not recommend drainage of the minor abscess but IV antibiotics were continued.

An MRI of the head with and without gadolinium demonstrated abnormal signal within the right frontal and parietal sulci with FLAIR hyperintensity of the cortex, suspicious for both meningitis and cerebritis (Figure 1). Diffusion-weighted images demonstrated areas of diffusion hyperintensity within the dependent portions of the bilateral atria suspicious for proteinaceous material.

Laboratory workup including HIV Ab, IgA/M/G and total complement were within normal limits. Blood cultures were positive for S. pneumonia. CSF culture returned negative. He also had an elevated IgE to 1000 IU/mL and his maximum erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were 40 mm/hr and 6.68 mg/dL respectively.

The patient returned to baseline mental status. His headache, neck pain, and back pain resolved. Repeat blood cultures were obtained and remained negative for five days. Infectious Disease Service was consulted and recommended eight weeks of intravenous ceftriaxone. The patient received a peripherally inserted central catheter and was discharged on ceftriaxone with close follow up by his primary care doctor, infectious disease, neurology, and spine surgery.

DISCUSSION OF DIAGNOSIS

Spinal and paraspinal infections from S. pneumoniae are rare.1-5 The largest case series described eight cases over a
span of 13 years from Nottingham, UK. Even less described is vertebral osteomyelitis in setting of meningitis. Our patient had persistent bacteremia, vertebral osteomyelitis, meningitis and a lumbar epidural abscess on MRI (Figure 1).

The literature suggests that upper and lower respiratory tract infections caused by pneumococci, sickle cell anemia, and bony trauma may be predisposing factors in pneumococcal vertebral osteomyelitis. Common comorbidities include sickle cell anemia, diabetes and heavy alcohol intake. Our patient did not have any of the above comorbidities but developed a community acquired pneumonia with *S. pneumoniae* bacteremia one month prior to presentation.

Our patient presented with fever, back pain and had an elevated ESR. In the setting of bacteremia these findings are suggestive of the clinical diagnosis of vertebral osteomyelitis. Imaging usually supports the diagnosis. Loss of disc height, cortical bone erosion, and epidural abscess formation are common imaging findings and were present in our case.

In cases in which the diagnosis of vertebral osteomyelitis has been established with clinical/imaging findings, isolation of the causative agent and specific treatment are of utmost importance in limiting the morbidity and mortality of the disease. Our patient was treated with high-dose ceftriaxone and made a full recovery. His mental status changes resolved and he remained without neurological deficits upon discharge.

It is important to note that in the case of the patient’s initial presentation of pneumonia with pneumococcal bacteremia, there are no Infectious Diseases Society of America (IDSA) guidelines regarding treatment. There have been no controlled trials on the optimal duration of antibiotics for the treatment of invasive pneumococcal infection in the setting of lower respiratory tract infection. It is important that more work is done to optimize the duration of therapy.

**CONCLUSION**

This case shows that vertebral osteomyelitis can complicate the course of pneumococcal bacteremia without a history of invasive procedures, back injury, or other co-morbidities mentioned above. The possibility of pneumococcal vertebral osteomyelitis should be considered in a patient with pneumococcal meningitis since this will alter the duration of antibiotic treatment. Imaging modalities such as MRI and CT scan help identify the extent of the infection. We recommend consultation with infectious disease and neurosurgery, as these patients will require extended antibiotic treatment with close physician follow-up.

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**Figure 1.** A: Post-contrast, sagittal fat saturated, T1-weighted MRI demonstrates a posterior epidural phlegmon at the level of L4-5 with 2mm abscess (arrow). B: Post-contrast, axial fat saturated, T1-weighted MRI demonstrates not only phlegmon with abscess (arrow), but involvement of the facets, lamina and spinous process with adjacent soft tissue enhancement. C: Pre-contrast, axial FLAIR MRI demonstrating abnormal signal within the subarachnoid space with possible enhancement of the gyri themselves suggesting cerebritis. D: Pre-contrast and E: post-contrast axial, T1-weighted spin echo MRIs demonstrating no sulcal enhancement post-contrast 4 days after the initiation of antibiotics.