

MORNING REPORT: PART II

An Unusual Case of Back Pain in Primary Care Clinic

Eric Tanenbaum, MD (presenter), and Meghan Kiefer, MD, MPH (discussant, in italic)

r. R is a 65-year-old gentleman with no significant past medical history who presents to this primary care clinic with a chief complaint of back pain. He describes razor-like bilateral upper back pain, 10/10 in intensity, starting adjacent to his spine and radiating to the upper chest above his nipples in a band-like distribution. The pain started six weeks ago while he was on a cross-country bike ride. The pain is made worse by inhalation but is unaffected by exercise or movement. The patient cannot recall any trauma to his back or chest. Two weeks prior to the onset of pain, Mr. R had experienced subjective fevers, lethargy, diffuse muscle weakness, night sweats, headache, dysuria, and difficulty urinating; all of these symptoms gradually resolved over a two-week period. The back pain began as his constitutional and urinary symptoms resolved.

On review of systems, the patient reports a red rash on his wrists and ankles approximately one year ago but no skin changes on his chest or back. He endorses contact with ticks and states that one to two years ago he removed a brown tick from his leg but could not recall further details. His travel history over the past year includes riding his bike through the American southwest, northeast, and northwest, as well as southeastern Canada. He endorses minimal consumption of alcohol, denies tobacco use, and denies ever using illicit or intravenous drugs.

The patient had sought care for back pain several times prior to this visit. Five weeks ago, within days of the initial onset of his pain, he had presented to a different primary care clinic. Plain chest radiography was performed, revealing a possible

left-sided pleural effusion. He was prescribed nonsteroidal anti-inflammatory drugs.

The pain continued, and he presented to an emergency department four weeks ago. At that time, he was afebrile, and his vital signs were within the normal range. A workup for acute coronary syndrome was negative. CBC revealed a mild normocytic anemia (Hb 12.9) with mild monocytosis. Basic chemistries and liver function tests were within normal limits. A PA-L chest radiograph did not reveal any significant abnormalities, and a CT angiogram of the chest revealed a 7 mm pulmonary nodule in the lower left lobe and was negative for pulmonary embolism. No bony abnormality was noted.

Two weeks ago, he presented to yet another primary care office with persistent back pain. He was prescribed gabapentin 100 mg twice daily. At this time the patient began taking ibuprofen 600 mg four times daily and oxycodone (obtained from a friend) 5 mg three to four times daily. His pain was still poorly controlled.

At the current visit, vital signs are: BP 105/74, pulse 90, T 37.6C. Physical exam reveals a healthy-appearing man in no apparent distress who appears younger than his stated age. Heart and lung exams are normal. Neurological exam reveals: CN II-XII intact, strength 5/5 throughout, reflexes 2+ throughout. He has no sensory deficits and no spinal or paraspinal tenderness; range of motion in his spine is normal. Digital rectal exam shows normal rectal tone and normal prostate. No rashes are seen. Lab testing reveals microcytic anemia (Hb 12.0) with mild monocytosis, HIV negative, UA normal, hepatitis C negative, Quantiferon Gold negative, ESR 62. The etiology of his

pain remains unclear. Due to the neuropathic quality of his pain, gabapentin is increased to 300 BID.

Mr. R is called one week after this initial visit to our clinic. He reports worsening pain. Nortriptyline 25 mg daily and naproxen 500 mg twice daily are added to his gabapentin, and a thoracic MRI with contrast is ordered.

The MRI reveals discitis/osteomyelitis of T4-T5 with erosive changes and associated ventral epidural abscesses. Cord compression is noted without signal abnormality. The patient is contacted and instructed to go to the emergency department. He is hospitalized and admitted to the spine service.

Spinal biopsy is performed and culture reveals methicillin-sensitive Staphylococcus aureus (MSSA). T3-6 laminectomy and T4-T5 corpectomy are performed with surgical stabilization and hardware placement. Broad spectrum antibiotics are begun but later narrowed to nafcillin. His hospital course is complicated by meningitis and later interstitial nephritis presumed to be caused by nafcillin. He is switched to levofloxacin and discharged with levofloxacin 750 mg PO daily and rifampin 300 mg PO twice daily to complete a six-week course. He is neurologically intact and ambulating independently upon discharge.

Spinal epidural abscess (SEA) is a rare cause of back pain. In one study the rate of spontaneous SEA was found to be 0.88 cases/100,000 person years.¹ Peak incidence is believed to be between ages 50 and 70. Major risk factors for SEA in the absence of spinal surgery or instrumentation include concurrent infection, diabetes mellitus, immune system compromise, and intra-

continued on page 2



MORNING REPORT: PART II

continued from page 1

venous drug use.^{2,3} Other risk factors include alcohol abuse, recent spinal fracture, indwelling catheter, cancer, and chronic renal failure. It is unusual for a patient to present with an SEA in the absence of risk factors; in one emergency department based study, 98% of patients had at least one listed risk factor.³

S. aureus is the most common causative agent and is present in up to 70% of cases.⁴ The most common bacterial source identified in cases of SEA is skin and soft tissue infection, although urinary tract infections, respiratory tract infections, and prior sepsis from an unknown source have also been implicated. Spinal instrumentation and invasive procedures are estimated to be responsible for 15% of cases

SEA is often missed on initial presentation because the classic triad of spine pain, fever, and neurologic deficits is rare (i.e. 8% of patients on initial visit), so diagnostic delay, as occurred in this case, is common.³ Back pain is the most common complaint with SEA, with 95% of patients describing this at their initial visit, compared to 41% reporting a neurologic deficit and 33% reporting fevers.³

MRI with gadolinium contrast is the diagnostic study of choice for SEA, with a sensitivity approaching 100%. MRI revealed or suggested the diagnosis in all 59 patients in one study.⁵ Although rarely diagnostic, X-ray of the spine can often suggest an abnormality (i.e. vertebral collapse, osteomyelitis) and the need for

further imaging. The exact sensitivity of plain radiography for SEA is unknown, but in a systematic review of pyogenic osteomyelitis, plain radiography revealed a bony abnormality in 89% of cases.⁶

This patient was an interesting case as despite his serious diagnosis he presented to a primary care clinic with the common complaint of back pain with normal vital signs and prior negative imaging. His red flags were new back pain at an older age and the non-mechanical nature of the pain. The sharp nature of the pain and apparent dermatomal pattern raised concern for a neurologic source. The primary diagnoses considered at the time of the clinic visit included infectious causes (i.e. bilateral herpes zoster, West Nile Virus, Lyme disease, other viral nerve root infections); structural causes (i.e. malignancy, including metastatic prostate cancer; other mass causing bilateral nerve root compression; spinal fracture); and musculoskeletal causes. It is notable that this patient had no known risk factors for SEA. While MRI clearly revealed the etiology of this patient's pain, multiple PA-L chest radiographs did not reveal signs of SEA nor did thoracic CT angiography. Potential bacterial sources for this patient's SEA include unnoticed skin and soft tissue infection (i.e. possible saddle sores during his bike ride) and urinary tract infection, as suggested by his urinary symptoms but less likely given that S. aureus was found to be the offending organism.

Back pain is a common complaint in primary care, and new back pain in an elderly patient—especially non-mechanical back pain—must be taken seriously. We should keep in mind that SEA is often missed on initial presentation and can present as isolated back pain.

References

- Ptaszyski AE, Hooten WM, Huntoon MA. The incidence of spontaneous epidural abscess in Olmsted County from 1990 through 2000: a rare cause of spinal pain. Pain Med 2007; 8(4):338-43.
- Sendi P, Breginzer T, Zimmerli W. Spinal epidural abscess in clinical practice. QJM 2008; 101(1):1-12.
- Davis DP, Wold RM, Patel RJ, et al. The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. J Emerg Med 2004; 26(3):285-91.
- Tompkins M, Panuncialman I, Lucas P, et al. Spinal epidural abscess. J Emerg Med 2010; 39:384-90.
- Rigamonti D, Liem L, Sampath P, et al. Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. Surg Neurol 1999; 52(2):189-96.
- Mylona E, Samarkos M, Kakalou E, Fanourgiakis P, Skoutelis A. Pyogenic vertebral osteomyelitis: a systematic review of clinical characteristics. Semin Arthritis Rheum 2009; 39(1):10-7.

SGIM