A Pain in the Back

Case

A 57-year-old man presented to the ED with a chief complaint of severe low back pain that radiated into his left buttock. The patient stated the pain started immediately after he had bent over to pick up a small refrigerator. He denied any abdominal pain, lower extremity numbness, weakness, or bowel or bladder dysfunction. The patient's medical history was significant for hypertension, for which he was taking amlodipine, and sleep apnea. The patient stated that he had a continuous positive airway pressure (CPAP) machine for his sleep apnea, but did not use it regularly. Regarding his social history, the patient denied tobacco use, but did admit to daily alcohol consumption.

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The patient's vital signs were all normal. Physical examination was remarkable only for bilateral lumbar paraspinal muscle tenderness, which was greater on the left side. There was no midline tenderness. Straight leg-raise testing was negative bilaterally, and the patient had normal strength and deep tendon reflexes in the lower extremities. The abdomen was soft and nontender.

The emergency physician (EP) diagnosed the patient with muscle strain, and discharged him home with a prescription for hydrocodone, along with instructions to follow-up with his primary care physician (PCP) within the next few days.

Three days later, the patient presented again to the same ED complaining of increased lower back pain. He denied any new injury or overuse, and continued to deny any lower extremity numbness or weakness or bowel/bladder dysfunction. Similarly, the physical examination was unchanged. The patient was given an intramuscular (IM) injection of hydromorphone with promethazine, as well as oral diazepam, and discharged home with instructions to continue to take the hydrocodone as needed for pain.

According to his wife, the patient retired to bed shortly after arriving home from the ED. Approximately 90 minutes later, she discovered the patient unresponsive in bed and called emergency medical services (EMS). He was brought back to the same hospital ED via EMS and was emergently intubated upon arrival. Unfortunately, the patient had suffered an anoxic brain injury and never regained consciousness; he died 1 week later.

The patient's wife sued the EP, claiming the anoxic brain injury was related to the drugs ordered by the EP in combination with the patient's alcohol use. She alleged that if the EP had observed the patient in the ED for signs of respiratory distress, his condition would have been treated and the anoxic brain injury would

Author's Disclosure Statement: The author reports no actual or potential conflict of interest in relation to this article.
DOI: 10.12788/emed.2017.0008

have been prevented. The plaintiff also faulted the EP for not informing the patient of the risks of drinking alcohol while taking the prescription pain medication.

The EP asserted his care of the patient was appropriate, and that there was no reason to keep the patient for observation. Regarding counseling the patient about the risks associated with concomitant alcohol consumption and pain medication, the EP stated that he had relied on the nurse who administered the medications to provide such counsel. The EP further maintained the plaintiff's death was due to the patient not using his CPAP machine as prescribed for sleep apnea, along with his alcohol consumption the evening of the event. At trial, a defense verdict was returned.

Discussion

Unfortunately, there are several unknowns in this case. Did the patient drink any alcohol after returning home from the second ED visit, prior to going to sleep? If so, how much did he consume? Did he take any of the narcotic pills prescribed from the first ED visit and, if so, how many did he take and in what time frame?

The combination of narcotics, benzodiazepines, and alcohol has long been known to be a potentially lethal combination, resulting in respiratory depression, respiratory arrest, anoxic brain injury and even death. As EPs, we are confronted with patients complaining of pain during every shift. Complicating matters, in national patient surveys concerning the care received in the ED, patients are specifically asked if their pain was adequately treated. At the same time, there is a national effort across all specialties to reduce the amount of opioids prescribed to patients. The EP should therefore attempt to select the least potent medication that will adequately control the patient's pain.

The WHO Pain Ladder

In 1986, the World Health Organization (WHO) developed a three-step analgesic ladder to guide the management of cancer pain.[1] This guide has since been expanded to include pain of noncancer etiology. Mild pain, defined on the numerical rating scale (NRS) as 1 to 3, is considered step 1.[1,2] Moderate pain (NRS of 4-6) is considered step 2, and severe pain (NRS of 7-10) is step 3. For step 1 pain, acetaminophen or a nonsteroidal anti-inflammatory drug (NSAID) is recommended. For step 2 pain, a weak narcotic (eg, codeine, hydrocodone), with or without acetaminophen or an NSAID, is recommended. For step 3 pain, a strong narcotic agent such as morphine or hydromorphone is advised.[1]

The WHO's ladder is not meant to serve as a strict protocol, but rather as a tool to guide the clinician in determining a reasonable starting point in pain management. Although the EP in this case did not ask the patient to rate his pain, from all indications it appeared to be severe (step 3) and as such, the choice of prescribing hydromorphone was a reasonable one. However, most experts agree that it is best to titrate an analgesic to the desired effect. In patients with severe pain, this means employing the intravenous (IV) route, not the IM route, which was used in this case. This is because the IM route can result in variable absorption and an unpredictable time of onset and duration of action.

Concomitant Antiemetic Therapy

It is common practice to administer an antiemetic simultaneously with a narcotic to prevent or lessen associated nausea and vomiting. The clinician must be aware, however, that all antiemetics act as central nervous system (CNS) depressants to some degree. The addition of diazepam in this case is problematic because all benzodiazepines cause sedation and anxiolysis. The combination of benzodiazepines with other CNS depressants, such as opioid analgesics, can lead to excessive sedation, resulting in partial airway obstruction, respiratory depression, and hypoxia.[3] The risk of an adverse outcome significantly increases with concomitant alcohol consumption.

The EP must carefully consider the risks and benefits any time opioids and benzodiazepines are administered in combination. In addition, the underlying health of the patient must be considered. Risk factors for opioid-induced respiratory depression includes age older than 50 years, a history of sleep apnea, preexisting pulmonary disease (eg, chronic obstructive pulmonary disease), anatomic oral or airway abnormalities, and renal or hepatic impairment.[3] Finally, patients should be informed of the dangers of mixing alcohol with opioids and benzodiazepines—whether such counsel is given by a physician, nurse, or pharmacist.

References

1. World Health Organization. *Cancer Pain Relief.* 2nd ed. Geneva, Switzerland: World Health Organization; 1996:1-69.
2. Todd KH. Pain assessment instruments for us in the emergency department. *Emerg Med Clin North Am.* 2005;23(2):285-295.
3. Jarzyna D, Jungquist CR, Pasero C, et al. American Society for Pain Management Nursing guidelines on monitoring for opioid-induced sedation and respiratory depression. *Pain Management Nursing.* 2011;12(3):118-145.

A Pain in the Butt(ocks)

Case

A 33-year-old Hispanic man, whose only spoken language was Spanish, presented to the ED for evaluation of pain in his right buttock. A coworker who accompanied the patient to the ED and served as his translator stated the patient's pain began the previous day, immediately following a work-related injury in which the patient had slipped and fallen backward, landing on his buttocks. The patient denied any head injury, loss of consciousness, or neck pain. He further stated that he was otherwise in good health and was not taking any medications. Regarding social history, the patient denied any alcohol or drug use.

The patient's vital signs were normal, as was his physical examination. The EP ordered an anteroposterior X-ray of the pelvis, which radiology services interpreted as normal. The EP diagnosed a buttock contusion secondary to fall, and discharged the patient home with instructions to take over-the-counter (OTC) ibuprofen for pain as needed.

Four days later, the patient presented to the same ED, complaining of low back pain radiating down his right leg. He denied any new injuries or falls, any lower extremity weakness, or bowel or bladder discomfort. Through a translator, the patient further noted that although he had been taking the OTC ibuprofen as prescribed, it had not alleviated his pain.

On physical examination, the patient's vital signs were: blood pressure (BP), 112/62 mm Hg; heart rate (HR), 96 beats/min; respiratory rate (RR), 20 breaths/min; and temperature (T), 101.8˚F. Oxygen saturation was 98% on room air. The lung, heart, and abdominal examinations were normal. The patient was noted to be tender to palpation over the sacral and coccygeal region. There was no documentation of a lower extremity or neurological examination.

The EP ordered a computed tomography (CT) scan of the pelvis without contrast. This was interpreted by radiology services as demonstrating moderate facet arthropathy at L4/L5 on the left side, with a facet joint cyst extending in the central canal; no fracture was identified. The radiologist suggested that these findings could be better evaluated with a magnetic resonance imaging (MRI) study of the lumbar spine, if clinically indicated. The EP decided against ordering the MRI, diagnosed the patient with a contusion of the coccyx, and discharged him home with instructions to continue taking OTC ibuprofen; he also prescribed combination acetaminophen/oxycodone for severe pain, and instructed the patient to follow-up with his PCP in 2 days.

Forty eight hours later, the patient returned to the same ED via EMS, now with the complaint of inability to walk or urinate. He continued to have a fever and was tachycardic with an HR of 110 beats/min; BP, RR, and T were normal, as was his oxygen saturation on room air. The patient was noted to have significant lower extremity weakness. A Foley catheter was placed, and 1,200 cc of urine was obtained. An emergent MRI of the lumbar spine revealed an extensive lumbar epidural abscess along the lumbar spine. The patient was started on IV antibiotics and taken to the operating room for evacuation of the epidural abscess. Unfortunately, he had a prolonged inpatient stay due to persistent bilateral lower extremity weakness and neurogenic bowel and bladder dysfunction; he eventually was discharged to a skilled nursing facility.

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The patient sued the EP and hospital for their failure to appreciate the significance of his presentation at the second ED visit. The attorneys for the plaintiff argued the defendants failed to order laboratory tests in the presence of fever, and failed to consult neurosurgery services. The plaintiff's neurosurgeon expert stated the extensive lumbar epidural abscess seen on the MRI during the third ED visit would have been appreciable on the second ED visit if an MRI had been obtained at that time. The defendants settled the case for $1 million.

Discussion

Spinal epidural abscesses (SEAs) are an uncommon but serious infection that must be recognized and treated promptly to avoid permanent neurological complications. These abscesses occur most commonly in the thoracolumbar area, where the epidural space is larger. Since the epidural space is a vertical sheath, an abscess that begins at one level commonly extends to multiple levels; SEAs frequently range three to five spinal cord segments.[1] The median age of onset for an SEA is approximately 50 years, and they are more common in men.[1] Risk factors for the development of an SEA include epidural catheter placement, paraspinal injections of glucocorticoids or analgesics, IV drug abuse, human immunodeficiency virus infection, diabetes mellitus, alcohol abuse, trauma, tattoos, acupuncture, and hemodialysis.[1,2] The most common pathogens causing an SEA are *Staphylococcus aureus*, gram-negative bacilli, and *Streptococci*.[2] The percentage of *S aureus* that are methicillin-resistant (ie, MRSA) varies by geographic location, ranging from 40% to 68%.[3,4]

Signs and Symptoms

Typically, patients with an SEA initially present with fever, malaise, and nonspecific symptoms and, as seen in this case, generally present several times to a physician before the correct diagnosis is made.[1] Unfortunately, the classic triad of fever, spinal pain, and neurological deficits is only infrequently observed. Fever is present in approximately two-thirds of patients, and spinal pain is present approximately 90% of the time.[2]

There are four stages of disease progression associated with SEAs. A typical scenario involves the initial complaint of back pain (stage I); followed by pain in the distribution of an affected nerve root (stage II); then motor weakness, sensory changes, and bladder or bowel dysfunction (stage III); and, finally, paralysis (stage IV).[1,2]

Diagnosis

Laboratory studies typically are not helpful in making the diagnosis. A complete blood count may show leukocytosis, but values can also be within the normal reference range. Acute phase reactants like erythrocyte sedimentation rate and C-reactive protein are commonly elevated with an SEA, but are neither sensitive nor specific.[1]

To make the diagnosis, the best test is a gadolinium-enhanced MRI of the spine.[2] It may be prudent to image the entire spine because multiple skip lesions are common, and a patient may not have pain or tenderness in all affected areas. If MRI is not available, a CT scan of the spine with IV contrast is an acceptable alternative.[1]

Once an SEA is identified, it is important to determine the organism(s) responsible for the infection. The best culture source is from the abscess itself (90%) followed by blood cultures (62%) and cerebrospinal fluid, which are positive only 19% of the time.[1]

Treatment

Once an SEA is diagnosed, a multidisciplinary approach involving hospitalists, interventional radiology, neurosurgery, and/or orthopedics is best. The most effective management is to treat patients with a combination of surgical decompression and drainage with systemic antibiotic therapy, typically for a minimum of 4 weeks. A minority of select patients may be treated with antibiotics alone.

References

1. Sexton, DJ, Sampson JH. Spinal epidural abscess. UpToDate Web site. http://www.uptodate.com/contents/spinal-epidural-abscess. Updated June 23, 2016. Accessed January 9, 2017.
2. Darouiche RO. Spinal epidural abscess. *N Engl J Med*. 2006;355(19): 2012-2020. doi:10.1056/NEJMra055111.
3. Chen WC, Wang JL, Wang JT, Chen YC, Chang SC. Spinal epidural abscess due to *Staphylococcus aureus*: clinical manifestations and outcomes. *J Microbiol Immunol Infect*. 2008;41(3):215-221.
4. Krishnamohan P, Berger JR. Spinal epidural abscess. *Curr Infect Dis Rep*. 2014;16(11):436. doi:10.1007/s11908-014-0436-7.