The Emergency Department Evaluation, Management, and Treatment of Back Pain

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KEYWORDS

• Back pain • Epidural abscess • Spinal stenosis • Diskitis

Back pain is one of the most common symptom-related complaints for visits to primary care physicians (PCPs) and is the most common musculoskeletal complaint that results in visits to the emergency department (ED).[1–3] Most adults (almost 90%) experience back pain at some time in their lives.[1,3,4] It contributes to high levels of lost work days, disability, and health care use.[5–7] It is the primary cause of work-related disability in persons less than 45 years of age. In 2005, back complaints prompted 139 million visits at a cost of $17.6 billion.[8] Although in the past higher use of the ED was primarily by a sicker, more disabled, and chronically ill population, recent trends suggest that EDs may be welcoming more patients who have traditionally sought care from their PCP. The 2006 Massachusetts Health Care Reform legislation sought to move state residents toward universal health care coverage and improve access to primary care medicine. Following this, ED visits soared. However, almost half (44%) of ED users noted that their visit could have been managed by their PCP had one been available.[9] This suggests that, with recent national health care initiatives moving in a similar direction, an increasing number of patients with common complaints such as back pain will visit the ED.

Although back pain has a benign course in more than 90% of patients, physicians must be vigilant and comfortable looking for warning signs of a neurologically impairing or life-threatening cause. As with many common complaints that have an overwhelmingly benign etiology, there may be a tendency toward complacency. The first goal of ED assessment of patients with back pain is to evaluate for potentially

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dangerous causes that, if not promptly recognized, could result in significant morbidity and mortality. The standard recommended approach is to obtain from all patients with lower back pain a careful and comprehensive history and to perform a thorough physical examination, and to rely on the presence of so-called ''red flags'' or alarm symptoms to guide further diagnostic tests, specialty evaluation, and treatment. This methodical approach helps to identify the small percentage of patients who have serious pathology as the underlying cause of their pain. This article focuses on the essential elements of an efficient and effective evaluation of patients with back pain in the ED, with special emphasis on epidural abscess, epidural compression syndrome, malignancy, spinal stenosis, and back pain in children.

CLINICAL HISTORY

The importance of a thorough and accurate clinical history cannot be overemphasized. As with any patient who complains of pain, symptoms should be characterized by the basic historical elements of the episode, such as the intensity, onset, character, severity, location, exacerbating and alleviating factors, and the presence of radiation. Further questions related to red flags must also be asked to identify patients at high risk. A summary of the red flag signs and symptoms can be found in **Table 1**.

Different causes of acute low back pain have different distinguishing characteristics. Typical nonspecific back pain is unilateral. It may radiate to the buttocks or posterior thigh but not past the knee. This pain is increased with movement, better with rest, and there are no complaints of numbness, weakness, or bowel or bladder dysfunction. Peripheral nerve pain may be described as a pins and needles sensation or burning, as opposed to nerve root pain, which is transient and sharp, relieved with recumbent positioning, and exacerbated by Valsalva maneuvers. Discogenic pain is typically worse with flexion, whereas pain from spondylolysis is aggravated by facet loading, which occurs in extension. Inflammatory back pain (spondyloarthritis) is insidious in onset, affects younger patients (<40 years of age), improves with exercise but not with rest, and causes increased pain at night with improvement on arising. Sciatica is sharp or burning and radiates laterally or posteriorly down the leg distal to the knee, usually to the foot or ankle. There may also be associated numbness or weakness. Epidural compression syndromes (spinal cord compression, cauda equine syndrome, and conus medullaris syndrome) are associated with numbness, weakness, bilateral leg pain, incontinence, and saddle anesthesia.

The age of the patient is an important initial consideration. Physicians must be especially cautious if the patient is less than 18 or more than 50 years of age because there

Table 1
History and physical examination red flags

Historical Red Flags	Physical Red Flags
Age <18 or >50 y	Fever
Pain lasting more than 6 wk	Writhing in pain
History of cancer	Bowel or bladder incontinence
Fever and chills	Saddle anesthesia
Night sweats, unexplained weight loss	Decreased or absent anal sphincter tone
Recent bacterial infection	Perianal or perineal sensory loss
Unremitting pain despite rest and analgesics	Severe or progressive neurologic defect
Night pain	Major motor weakness
Intravenous drug users, immunocompromised	
Major trauma	
Minor trauma in the elderly	

is a higher likelihood of serious pathology as the cause for the back pain. Tumors and infection occur with higher frequency in these age groups.[10–12] Younger patients are also at increased risk of spondylolysis and spondylolisthesis.[13,14] In the older patient, an abdominal aortic aneurysm (AAA) must always be considered as a potential cause. The presence of hematuria can make this entity resemble the classic kidney stone. Isolated low back pain is a common presentation of a contained rupture of an AAA.

The elderly may sustain fractures, including pathologic fractures, with minor trauma. This condition is likely secondary to osteoporosis. Chronic steroid users are also at increased risk for fracture, even with minor mechanisms. The immunocompromised, intravenous drug abusers (IVDAs), and those with a recent bacterial infection (eg, pneumonia or urinary tract infection) are at increased risk of spinal bacterial infections. Any patient with back pain and a history of IVDA should be assumed to have an abscess or vertebral osteomyelitis until proven otherwise.[15,16] Recent gastrointestinal or genitourinary procedures may also cause a transient bacteremia leading to an infectious cause of the patient's back pain. Patients with cancer are another high-risk group. The spine is the third most common site for cancer to metastasize.[17,18] Most patients with systemic cancer have spinal metastasis. Cancers that are known to most commonly metastasize to the spine are breast, lung, and prostate, but also include kidney, thyroid, and multiple myeloma.[19] These patients are at greater risk of epidural compression syndrome and pathologic vertebral fractures.

Most episodes of lower back pain resolve or significantly improve within 4 to 6 weeks, therefore lack of significant improvement in 6 to 8 weeks is a red flag.[20,21] It should then be established whether the patient is primarily describing back or leg symptoms. Radicular pain in a dermatomal distribution, often into the lower hamstring, knee, and foot, indicates nerve root compression or irritation (sciatica). The more common nonspecific back pain without true radiculopathy may still radiate into the buttocks or upper thighs but not past the knee, implying muscle or ligamentous strain or disc disease without associated nerve involvement.

The clinician should attempt to elicit historical features that are suggestive of infection or malignancy, such as unintentional weight loss (more than 4.5 kg in 3 months), fevers, chills, and night sweats. Back pain associated with chest, abdominal, or urinary complaints is suggestive of an extraspinal cause. Because most benign back pain is tolerable, worsened with activity, and improved with rest or lying still, symptoms such as severe night pain (especially deep bony pain) and severe unrelenting pain that is not relieved by rest, recumbency, or appropriate analgesic treatment should raise a red flag for malignancy or infection.[22–24]

Symptoms that are suggestive of epidural compression syndrome are mainly neurologic in origin, and include any loss of bowel or bladder function, urinary retention, erectile dysfunction, saddle anesthesia, or progressive distal leg numbness or weakness.[21,25] In older patients, typically those greater than 60 years of age, spinal stenosis is suggested by lower back pain with radiculopathy that is worsened with walking, prolonged standing, and back extension (standing) but is relieved with rest and forward flexion (sitting).[26–28] Pain from disc herniation is relieved by lying supine and is worsened by Valsalva maneuvers, coughing, sneezing, and positions that produce increased pressure on annular fibers, such as prolonged sitting, standing, and bending postures.[29,30]

PHYSICAL EXAMINATION

The purpose of the physical examination is to evaluate neurologic complaints discovered in the history, identify potential neurologic defects, and to uncover any red flags.

Carefully evaluate the patient's vital signs, general appearance, and back, then perform a complete neurologic examination of the legs. Lumbar spine pathology is frequently manifested in the lower extremities as an alteration in the patient's reflexes, sensation, and/or muscle strength. The neurologic examination systematically tests the clinical relationship between the reflexes and the motor and sensory components of the most commonly affected nerve roots.

First and foremost, the clinician must account for any abnormal or unstable vital signs, which may indicate an extraspinal cause of the back pain such as an AAA. Fever, present in approximately 50% of patients with osteomyelitis or spinal epidural abscess, is a red flag for infection.[12,22] The absence of fever, equally common, does not rule out a spinal infection.

Next, consider the general appearance of the patient. In most cases, patients with back pain prefer to lie still because movement worsens their pain. Those individuals in extreme pain or writhing in pain may have an underlying emergent cause. This would include both spinal causes, such as epidural abscess and osteomyelitis, and extraspinal causes such as intra-abdominal (ie, mass), retroperitoneal (ie, nephrolithiasis or hemorrhage), or vascular (ie, AAA) causes. Next, the patient's back should be fully exposed and inspected for signs of infection. The midline spinous processes should be palpated for tenderness suggesting fracture or infection (sensitivity 86%, specificity 60%).[26] Pain during lumbar flexion suggests discogenic pain, whereas pain on lumbar extension suggests facet disease. Secondary findings of paraspinal muscle spasm or edema should be noted.

A detailed distal neurologic examination must be performed that targets the 3 most common locations for disc herniation: L4, L5, and S1. More than 95% of herniated discs affect the L4 to L5 or L5 to S1 interspaces, causing impingement on the L5 and S1 nerve roots, respectively.[31,32] Patients with these radiculopathies complain of acute back pain and pain radiating down the lateral (L5) or posterior (S1) leg to the ankle or foot. Pathology at the higher lumbar spine (L1, L2, L3) causes acute back pain with anterior thigh radiation, weakness with hip flexion (iliopsoas muscle action), and anterior thigh sensory changes in the corresponding dermatome. This may be seen in older patients with symptoms of spinal stenosis. There are no individual reflexes for the L1 to L3 lumbar levels. Those with pathology at the lower sacral levels (S2–S5) have sacral or buttock pain that radiates down the posterior leg or into the perineum and can have difficulties with penile erection (S2–S4), abnormal perianal sensation (S3–S5), anal wink (S2–S4), rectal tone (S2–S5), and bladder function (S2–S4).

An understanding of the key physical components of the targeted L4 to S1 neurologic examination is essential. At each level, the corresponding muscle strength, sensation, and reflex needs to be tested and documented. True sensory loss is best tested with pinprick rather than light touch. Sensory fields can have considerable overlap so areas that are exclusively served by an individual nerve must be examined. Similarly, because many muscles have innervation from multiple roots, preserved strength may be found despite significant involvement of a single nerve root. The L4 neurologic level strength is tested with knee extension, ankle inversion, and dorsiflexion (sensitivity 49%–63%, specificity 52%–89%).[31,33,34] Sensation is tested from the medial leg down to the medial surface of the great toe (not including the first web space). The corresponding reflex is the patellar reflex (sensitivity 15%–50%, specificity 67%–93%).[34,35] The L5 neurologic level strength is tested with great toe dorsiflexion (extensor hallucis longus) (sensitivity 37%–61%, specificity 55%–71%) and heel walking.[35,36] Sensation is tested from the lateral leg, dorsum of the foot, and the first web space. There is no reliable reflex to test L5, which is the most commonly

compressed nerve root. The S1 neurologic level strength is tested with foot eversion (peroneals) and toe walking (ankle plantar flexion) (sensitivity 13%–47%, specificity 76%–100%).[33,35] Sensation is tested from the lateral foot and ankle. The corresponding reflex is the Achilles reflex (sensitivity 47%–56%, specificity 57%–90%).[31,34–36] Ankle reflexes become increasingly absent with age, lost in nearly 50% of those more than 80 years of age.[37] This loss is usually symmetric, thus unilateral absence may signify pathology.[38] The overall sensitivity and specificity of the sensory examination in the diagnosis of lumbar disc herniation is 16% to 50% and 62% to 86% respectively.[33–35]

Although impractical and unnecessary for office practice, urinary catheterization can be helpful in evaluating select patients in the ED. Measurement of a postvoid bladder residual volume tests for the presence of urinary retention with overflow incontinence. This finding is sensitive and specific for cauda equina syndrome.[6] Large postvoid residual volumes (>100 mL) indicate a denervated bladder with resultant overflow incontinence and suggest significant neurologic compromise.[26,39,40] This finding warrants immediate evaluation for epidural compression syndrome. A negative postvoid residual volume is reassuring and effectively rules out significant bilateral neurologic compromise.

Digital rectal examination is not a routine part of the physical examination in most patients with back pain but should be performed in select individuals. Appropriate patients include anyone with red flags, severe pain, and those in whom epidural compression syndrome is being considered, such as those individuals with severe pain and progressive neurologic findings. The rectal examination can aid in the assessment of sphincter tone and perianal sensation (S3–S5), anal wink (S2–S4), and in checking for masses or a possible perirectal abscess. A Babinski test should also be performed in patients with findings concerning for neurologic compromise. A positive test involves extension of the great toe with flexion and splaying of the other toes, and suggests a lesion affecting the upper motor neurons or corticospinal tract.

The straight leg raise (SLR), which tests for the presence of a herniated disc causing nerve root compression, is one of the most important tests for evaluating back pain. This examination technique is approximately 80% sensitive for a herniated disc cause of pain (sensitivity 72%–97%, specificity 11%–66%).[41] The SLR has a positive predictive value (PPV) of 67% to 89% and a negative predictive value (NPV) of 33% to 57% in patients with a high probability of having a disc herniation versus a PPV of 4% in those patients with a low probability (based on the absence of neurologic symptoms or sciatica).[41,42] A positive test causes or reproduces radicular pain below the knee of the affected leg when the leg is elevated between 30° and 70°. A positive finding also occurs if radicular symptoms are elicited when the leg is then lowered until pain is eased and the ipsilateral ankle is dorsiflexed (Braggard sign).[43] Pain below 30°, above 70°, or with reproduction of pain only in the back, hamstring, or buttock region does not constitute a positive test. Care should be taken that the patient is not actively helping in lifting the leg and that the knee remains straight throughout the examination. Pain referred to the affected leg when the opposite asymptomatic leg is tested, called a positive crossed SLR, strongly indicates nerve root irritation from a herniated disc. This finding has a high specificity (85%–100%), but low sensitivity (29%), with a PPV of 79% to 92% and NPV of 22% to 44%.[41,42]

If the patient is reluctant or unwilling to lie supine for SLR testing, the seated SLR or slump test should be attempted. The traditional supine SLR is more sensitive than the seated SLR (sensitivity of 0.67 vs 0.41).[44] To perform the slump test, the patient sits at the edge of the examination table and slumps forward while flexing the neck and trunk. This movement is followed by knee extension and ankle dorsiflexion. A positive test

reproduces radicular pain. The slump test was positive in 94% of patients with frank disc herniation, 78% of patients with bulging discs, and 75% of patients without radiographic findings.[45] The SLR and slump test demonstrate good correlation ($\kappa = 0.69$).[46] The slump test has greater sensitivity than the SLR (0.84 vs 0.52) with slightly lower specificity (0.83 vs 0.89).[47]

The clinician must also be able to distinguish between true pathologic back pain and nonorganic back pain. Waddell signs are physical examination findings that can aid in making this distinction, and can be remembered by the acronym DORST (distraction, over-reaction, regional disturbances, simulation tests, and tenderness).[48] Superficial, nonanatomic, or variable tenderness during the physical examination suggests a nonorganic cause. The clinician may also simulate back pain through provocative maneuvers such as axial loading of the head or passive rotation of the shoulders and pelvis in the same plane. Neither maneuver should elicit low back pain. There may be a discrepancy between the symptoms reported during the supine and sitting SLR. The seated version of the test, sometimes termed the distracted SLR, can be performed while distracting the patient or appearing to focus on the knee. Further, radicular pain elicited at a leg elevation of less than 30° is suspicious because the nerve root and surrounding dura do not move in the neural foramen until an elevation of more than 30° is reached. Sensory and motor findings suggestive of a nonorganic cause include stocking, glove, or nondermatomal sensory loss or weakness that can be characterized as give-away, jerky, or cogwheel. Gross overreaction is suggested by exaggerated, inconsistent, painful responses to a non-offensive stimulus.

Traditionally Waddell signs, especially if 3 or more are present, were believed to correlate with not only functional complaints (physical findings without anatomic cause) and decreased likelihood of return to work but also with malingering. The association between Waddell signs and malingering has been misinterpreted, falsely propagated, misused both clinically and medicolegally, and ignores the caveats about the interpretation and application of the signs from the original study article.[49] As originally conceived, Waddell signs were not intended for use in detecting malingering or sincerity of effort. Subsequent studies have found that Waddell signs are associated only with poorer treatment outcome, decreased functional performance, decreased likelihood of return to work, higher levels of pain intensity and duration, psychological distress, and perceived disability.[50–53] However, there has been little evidence to support the association between Waddell signs and secondary gain or malingering.[51,52] When combined with shoulder motion and neck motion producing lower back pain, Waddell signs predict a decreased probability of the individual returning to work.[54] There is poor interobserver agreement in the assessment of Waddell signs.[55] Another test worth noting is the Hoover test, which assesses the patient's voluntary effort. Patients are instructed to lift their leg while their heels are cupped by the examiner. True volitional effort results in increased downward pressure on the untested heel. ED staff may also note inconsistency in observed spontaneous activity during time of care, such as with dressing, undressing, and getting off the examination table. Although these signs can be used in the evaluation of select patients, they are merely a component of a comprehensive examination. Waddell signs should never be used independently because they lack the sensitivity to be able to distinguish nonorganic problems from true organic pathology.[51]

DIAGNOSTIC TESTING

In general, if the history and physical examination do not reveal any concerning findings or red flags for emergent pathology, no further testing is required. Diagnostic

testing is indicated only if it will help guide specific patient management strategies. Blind diagnostic testing (so-called ''shotgunning'') may lead to a cascade of further testing and false-positive results, which themselves will beget further testing and possibly unnecessary interventions. Such an approach may foster the patient's inaccurate belief that there is true pathology present, which can increase their anxiety and medical expenses. However, if red flags are uncovered in the history and physical examination, further evaluation with appropriate diagnostic testing is warranted.

Routine laboratory studies are rarely needed for the initial evaluation of nonspecific acute low back pain. Laboratory testing, consisting of a white blood cell (WBC) count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) is indicated in cases of suspected infection or malignancy. In cases of spinal infection, the sensitivity of an increased WBC count (35%–61%), ESR (76%–95%), and CRP (82%–98%) may help guide further evaluation or consideration of other entities.[56–58] An increased ESR also has a role in the diagnostic evaluation of occult malignancy (sensitivity 78%, specificity 67%).[59] Urinalysis is useful for atypical presentations of pyelonephritis. Other tests such as blood cultures, prostate-specific antigen, calcium, and alkaline phosphatase may be considered in appropriate cases.

Imaging, like laboratory testing, is not indicated in the absence of red flags or concerns for malignancy, fracture, infection, or epidural compression syndrome. Unnecessary imaging only serves to increase the cost of the visit and the length of stay, and subjects the patient to unnecessary radiation. Although the added diagnostic value of modern neuroimaging is significant, there is concern that these studies may be overused in patients with lower back pain presenting to the ED.[60,61] Patients who receive radiography are more likely to be satisfied with their care, and up to 80% of patients with low back pain would accept plain radiographs if given the choice, despite the lack of benefit associated with routine imaging.[62] This suggests a need for the ED physician to address patient preferences and expectations. Educational interventions may be effective for reducing the proportion of patients who expect routine imaging for their low back pain.[63]

Most patients in the ED with lower back pain do not require plain radiographs. Studies show that plain radiographs rarely reveal any helpful information.[20,64,65] Radiographic studies have not been shown to help diagnose uncomplicated lower back pain and rarely alter clinical decision making.[61,62] Plain radiographs (anteroposterior and lateral views) should be the first step only in cases of suspected infection, fracture, malignancy, or neurologic compromise.[66] Additional views are only indicated if spondylolysis or spondylolisthesis is suspected. They otherwise add unnecessary cost and radiation.[60] The amount of gonadal radiation from a single 2-view plain radiograph series of the lumbar spine is equivalent to being exposed to daily chest radiographs for 1 year.[67] Negative plain radiographs do not rule out disease such that, if sufficient pretest probability of a potential neurologic emergency exists, the ED physician should proceed with further imaging (computed tomography [CT] or magnetic resonance imaging [MRI]) regardless of plain radiograph results.[20] In these cases, if these studies can easily and quickly be obtained, there is no need for plain radiographs because they do not offer unique information. A bone scan can be useful when looking for spinal stress fractures, infectious processes, and spinal metastatic disease, especially in the patient without neurologic symptoms.[20] However, it is not typically ordered from the ED and has largely been replaced by MRI.[68]

In the absence of a serious or progressive neurologic deficit, or concern for epidural compression syndrome, neoplasm, or spinal infection; CT or MRI should not be part of the diagnostic evaluation in the ED. Isolated sensory loss or the absence of a reflex is not considered to be a progressive neurologic deficit. CT is superior to plain

radiographs for the detection of vertebral fractures and other bony pathology, especially fractures involving posterior spine structures, bone fragments within the spinal canal, or spinal malalignment.[69,70] CT is increasingly used as a primary screening modality for moderate to severe spine trauma. CT with myelography (or with intravenous [IV] gadolinium) may be used in those patients with concern for epidural abscess, epidural compression, or vertebral osteomyelitis who are otherwise unable to have MRI. Evaluation of vertebral body bony destruction seen on plain radiograph is best visualized by CT. However, if a neurologic deficit is present, MRI is more appropriate.

With the exception of the evaluation of acute trauma, MRI is the most informative investigative modality and identifies almost all pathologic states that benefit from surgical management. MRI is the modality of choice for evaluation of spinal infectious lesions (sensitivity 96%, specificity 92%), malignancy (sensitivity 83%–93%, specificity 90%–97%), disc herniation, and epidural compression syndrome (sensitivity 93%, specificity 97%).[71] In acute disc herniation without progressive neurologic symptoms, MRI can be delayed for 4 to 6 weeks and coordinated by the patient's PCP.[60,61,72,73] Disc disease is a component of normal aging and a nonspecific finding[74–76]; 1 in 4 of all asymptomatic persons younger than 60 years have MRI findings of a herniated disc.[77] That number increases to 1 in 3 in people older than the age of 60 years.[77] In one study, more than 50% of asymptomatic patients were identified as having a bulging disc on MRI.[78]

PHARMACOTHERAPY

One of the most important goals of treatment is to provide an acceptable level of analgesia while the underlying condition resolves or to ameliorate the suffering of those patients who await definitive therapy. When considering a particular pharmacologic therapeutic agent, recall that physicians notoriously underestimate, and subsequently undertreat, pain.[79]

First-line pharmacologic therapy should include nonopioid analgesic agents such as acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs), both of which are excellent analgesics with comparable efficacy.[65,80,81] Some clinicians consider acetaminophen superior because of its low cost, efficacy, and lower side effect profile.[80] Given the increasing recognition of acetaminophen's role in hepatotoxicity, patients should be cautioned not to take acetaminophen in any form (eg, over-the-counter preparations) in addition to opiate-acetaminophen combination narcotics.[82] NSAIDs have well-known renal and gastrointestinal side effects that should also be considered. NSAIDs may be a better option for younger patients who are less likely to have these potential complications, whereas acetaminophen may be more appropriate for older patients.[80,81] NSAIDs are slightly superior to acetaminophen for pain relief from osteoarthritis not limited to the back.[81] Ketorolac (either intramuscular or IV) may provide early improved analgesia in those unable to tolerate oral medication in the acute setting. There does not seem to be a specific NSAID that is clearly more effective than others.[80]

Opioid analgesics should be considered a third-line alternative, and best used for those experiencing severe acute back pain with inadequate control with nonnarcotic analgesics.[81] Though counterintuitive from clinical experience, the data for opioid superiority compared with other analgesics, such as NSAIDs or acetaminophen, is sparse and inconclusive. Opioids have not been shown to be more effective than either NSAIDs or acetaminophen for initial treatment of acute lower back pain, nor do they increase the likelihood of return to work.[65,81,83–85] When prescribed, opioids should be combined with an NSAID, taken on a fixed dosing schedule, and only for

a limited time.[86,87] The physician should always consider the known side effects of opioids (constipation, confusion, and sedation) especially in the elderly population. There is good evidence that muscle relaxants reduce pain and that different types are equally effective.[81,88–90] However, the high incidence of significant side effects such as dizziness and sedation limits prolonged use. Muscle relaxants seem to confer the most benefit to patients with back pain and associated muscle spasm who can tolerate the side effect profile. Benzodiazepines are second-line muscle relaxants because of concerns for their abuse potential. Both types of muscle relaxants may be beneficial in an every-bedtime capacity, thereby limiting side effects. Opioid and muscle relaxant use is not compatible with most workplace responsibilities. There is no role for starting any long-acting chronic pain medication (ie, oxycontin, methadone, fentanyl patches) in the ED. A regimen of NSAIDs, acetaminophen, and skeletal muscle relaxants may suffice for most patients.

Epidural glucocorticoid injections seem to have the best effect in patients with radiculopathy caused by a herniated disc, but do not provide benefit beyond 4 to 6 weeks.[91] Further, they do not delay surgery in those who are already surgical candidates.[92] Steroid injections do not seem to be effective for other entities such as nonspecific low back pain or spinal stenosis.[91,93] Lumbar epidural steroid injections are never indicated for acute or emergent treatment and thus have no role in the ED management of back pain. In general, this procedure is part of an outpatient pain management regimen for the patient with radiculopathy who fails to respond to several weeks of conservative therapy.

There is no clear benefit to oral glucocorticoids for patients with lower back pain, with or without sciatica.[65,81,94,95] However, some practitioners continue to use glucocorticoids for back pain with lumbosacral radiculopathy, acknowledging that the benefit is likely modest and transient.[96,97] The practitioner should always consider the negative side effect profile of steroids, including hyperglycemia, gastrointestinal bleeding, and an increased risk of infection. Nonpharmacologic analgesia can include the use of heat or cold externally applied to the lower back. There is better evidence for the use of heat than ice for treatment of lower back pain.[98,99] Anecdotal evidence suggests that cold packs may be beneficial early in symptom treatment and moist heat may help with pain and muscle spasm in the first 1 to 2 weeks. However, actual care rendered rarely reflects these evidence-based practice guidelines and recommendations.[100] Notable deviations from established treatment guidelines include infrequent inclusion of advice, education, and reassurance (20.5%) in addition to infrequent prescription of simple analgesics (17.7%). In a study of more than 3500 patients with a new episode of lower back pain, providers demonstrated an overreliance on opioids (19.6%) and early imaging (25.3%).[100] More patients were referred to imaging than received advice and education. Such usual practice is unlikely to lead to the best patient outcomes and clearly contributes to the high cost of managing lower back pain.

SUMMARY

An individual red flag does not necessarily correspond to a specific pathology; it indicates a higher probability of a serious underlying condition that may require further investigation. However, multiple red flags always require further investigation that is often initiated in the ED. Following the history and physical examination, patients with acute low back pain can be divided into 3 main categories: (1) patients with nonspecific lower back pain, (2) patients with nerve root or radicular pain, and (3) patients with serious or emergent spinal pathology, including red flag conditions

such as tumor, infection, or epidural compression syndrome. The first priority is to make sure that the back pain is of musculoskeletal origin and to rule out nonspinal pathology such as from an AAA. The next step is to exclude the presence of serious spinal pathology such as epidural compression syndrome or epidural abscess. The physician must determine whether the patient has nerve root pain. In the absence of radicular pain, the pain is classified as nonspecific low back pain.

NONSPECIFIC LOWER BACK PAIN

Most people (80%–90%), and most patients in the ED, have nonspecific lower back pain, which is pain without a clear origin and not caused by a specific disease or spinal abnormality.[65] These patients have low back pain and an otherwise negative history and physical examination.[26] This diagnosis of exclusion is made only after ruling out the more worrisome causes of back pain.[20] Typically, patients have mild to moderate pain, localized asymmetrically in the lumbar or sacral paraspinous muscles. Pain, typically characterized as an ache or spasm, may radiate into the buttocks or thigh, and is worsened with activity and relieved with rest. At times, this reported radiation can elicit an improper diagnosis of radiculopathy or disc herniation as opposed to true radicular symptoms, which radiate below the knee in a dermatomal distribution, and may be associated with sensory loss, weakness, or reflex changes. Physical examination may reveal mild to moderate paraspinal muscle tenderness and/or paravertebral muscle spasm but will not reveal any red flags or neurologic abnormalities. Because most patients with nonspecific lower back pain experience symptomatic resolution within 4 to 6 weeks, only conservative management is needed.[65] No further diagnostic testing is required beyond the history and physical examination.[29] Conservative management includes continuation of daily activities as tolerated, ice, heat, analgesia, and/or muscle relaxants, patient education, and referral for close follow-up with their PCP to ensure that the problem resolves.[81]

Persistent pain for more than 4 to 6 weeks, including a prolonged exacerbation of chronic back pain, should be evaluated with the previously mentioned laboratory tests (CBC, ESR, and/or CRP) and a 2-view plain radiograph of the lower back. Prior imaging, such as CT or MRI, should also be reviewed if possible. The ED physician should perform a thorough (re)review of red flags in the history and physical examination that may have been missed on the initial evaluation or have developed in the interim. If there are any new concerning findings, the patient should be approached like any new patient with the same findings. In the absence of new findings, chronic back pain is often regarded as one of the most challenging clinical scenarios. The underlying cause of chronic back pain is complex and multifactorial, making proper assessment and treatment nearly impossible in the ED setting. These patients usually require a multidisciplinary treatment approach for the greatest chance for success. The prescription of narcotics to these patients must be an individualized decision in accordance with the ED physician's assessment of the clinical scenario. The risk of providing narcotics to a drug-seeking patient must be carefully weighed against the risk of denying pain medicine to a patient with true pain in true need.

COCCYDYNIA

Coccydynia is another common back condition presenting to the ED. It is more common in adults, women, and the obese. It commonly occurs from acute trauma, usually caused by a fall backward into a sitting position that results in a bruised, broken, or sometimes dislocated coccyx. Other causes include repetitive minor trauma; for example, prolonged sitting on hard, ill-fitting, or narrow surfaces such as

a bicycle seat. Those with poor posture are also at increased risk because leaning backward excessively puts an increased load on the coccyx. Patients complain of pain in the tailbone that is worse with sitting, especially when leaning backward, and on standing. They may report pain with sexual intercourse or with defecation. Rectal examination is essential and the only way to fully palpate the coccyx. The ED physician will find tenderness localized to the coccyx both with external palpation in the gluteal crease and when the coccyx is grasped between the forefinger and the thumb. Differential diagnosis should included prostatitis, pelvic inflammatory disease, and rectal abscess. Imaging is only useful if considering infection, cancer, or other pathology, as confirmation of a coccygeal fracture is not always necessary because of the risks of radiation exposure and the unlikely event that imaging will change management. ED attempts at reduction are not recommended. Treatment involves protecting the tailbone from further trauma, such as by advising the patient to sit leaning forward and prescribing a donut or wedge cushion that distributes weight away from the coccyx. Heat or ice is advised, allowing the patient to choose whichever is most effective. Hot sitz-type baths may also be helpful. Analgesic medications should be selected based on symptoms, and, if opioids are chosen, a stool softener should also be provided to avoid pain with constipation and straining.

Lumbosacral Disc Disease and Radiculopathy

Lumbar radiculopathy is a clinical diagnosis of nerve root irritation and compression leading to symptoms in the distribution of the affected lumbar or sacral nerve root, such as numbness, weakness, or paresthesias. The most common causes are from disc herniation and spondylotic degeneration causing foraminal stenosis. Disc herniation is unusual before 18 years of age and is rare in the fibrotic discs of the elderly. Disc herniation occurs when the tough outer disc layer (the annulus fibrosis) tears, and the inner gelatinous material (the nucleus pulposus) prolapses, inflames, and compresses a nerve root. This herniation may be anywhere on the continuum from asymptomatic to severely painful. Disc herniation is most common at the L4 to L5 and L5 to S1 levels, causing L5 and S1 radiculopathies, respectively. Clinical symptoms are typically self-limited with a high rate of spontaneous improvement and low likelihood of progression to a neurologic emergency.

Although patients complain of pain in their lower back, unilateral lower extremity pain and radicular symptoms predominate because of the anatomic distribution of the nerve roots involved. Pain radiating below the knee is more likely to represent true radiculopathy than pain radiating only to the gluteal region or posterior thigh. Motor findings, such as focal weakness, occur less frequently than focal sensory or reflex changes. Patients typically report a combination of pain and a constellation of sometimes vague sensory symptoms such as anesthesia, dysesthesia, hyperesthesia, or paresthesia. A dermatomal pattern of sensory loss or a reduced or absent deep tendon reflex is more suggestive of a specific root lesion than the pattern of radicular pain.[38,101] Symptoms tend to become worse with Valsalva maneuvers and better with recumbent positioning. Diagnosis consists of localizing the pain and neurologic dysfunction to an isolated nerve root, as discussed earlier. In addition to disc herniation, other causes for radiculopathy should always be considered, including cord compression, spinal stenosis, tumor, and infection. Multinerve root pathology and/or the presence of bilateral symptoms are potential indicators of a spinal mass lesion or large central disc herniation that compresses multiple descending nerve roots within the spinal canal.

The natural history of disc herniation is that pain from pressure and nerve irritation improves as the local inflammation subsides. The size of the disc protrusion may

naturally decrease with time. A bulging disc is a common entity, likely a component of normal aging that may be an incidental finding on a MRI.[74,75,77,78] Although disc herniation and radiculopathy are commonly considered together, herniation is usually asymptomatic and likely only occasionally causes symptoms of sciatica.

It may be helpful for the ED practitioner to classify the typical patient with lumbar radiculopathy into one of the 3 main groups of common presentations. The first is the patient with painful radicular symptoms and a pure sensory dysfunction without other neurologic deficits. The next patient category has the same symptoms but includes a mild nonprogressive identifiable motor deficit with or without an associated reflex change. The third patient group includes severe or worsening motor deficits. Patients who have a herniated disc with symptoms in a single nerve root distribution and an otherwise normal examination do not require an urgent or emergent MRI or even specialty referral.[3,65,102] Many patients who have a herniated disc can be managed conservatively as outpatients by their PCP with follow-up and re-evaluation in 1 week. Because clinical symptoms are typically self-limited and unlikely to progress to a neurologic emergency, the ED evaluation of patients with stable symptoms is focused on pain control and activity modification and does not require emergent diagnostic testing. These patients must not have any red flags such as urinary retention or saddle anesthesia that would suggest another emergent condition (eg, epidural compression), and they must be neurologically intact (no progressive or bilateral neurologic deficits). Urgent neuroimaging and consultation initiated from the ED is necessary for patients with acute lumbar radiculopathy and red flags that suggest rapidly progressive or bilateral neurologic deficits, urinary retention or saddle anesthesia, or suspected epidural abscess or neoplasm. Patients with intractable pain should be admitted for pain control.

Patients with the common symptoms of back pain with radiculopathy should receive appropriate analgesics, be advised to avoid bed rest, and be treated like those with nonspecific lower back pain.[65,103] Patients should be reassured that most people experience symptomatic resolution within 4 to 6 weeks with conservative, nonsurgical management.[65,104] If the pain from disc herniation does persist for longer than 6 weeks, outpatient MRI is indicated.[60,65] Corticosteroid injections into the epidural space may relieve some of the inflammation associated with disc herniation and represent another outpatient option for patients in severe pain. Although some reduction of symptoms may be obtained initially, no long-term benefit or reduction in the need for later surgery has been documented.[91,92] There is also no clear benefit to the use of systemic steroids in cases of disc herniation with radiculopathy.[81] With a documented herniation, some patients with prolonged intractable pain may benefit from surgical discectomy compared with conservative management, although this remains controversial.[105–107] Other indications for surgery include worsening motor or sensory deficits.

EPIDURAL ABSCESS

Spinal epidural abscess, now diagnosed in 1 in 10,000 hospital admissions, remains a rare disease despite a doubling of the overall incidence in the past 2 decades.[12] Although traditional risk factors such as diabetes, alcoholism, and human immunodeficiency virus (HIV) remain unchanged, the aging population, increased IVDA, and the increasing use of spinal instrumentation and indwelling devices (epidural catheters, spinal stimulators, and vascular access) may help to explain this increased incidence. Another possible explanation involves the improved diagnostic sensitivity of modalities such as MRI that allow for early detection.

Epidural abscesses may originate from either remote spread (25%–50%) via the bloodstream or local contiguous spread (15%–30%) from infected adjacent skin and soft tissue such as from a psoas abscess or infected vertebral body.[12] An emerging cause is via direct inoculation into the spinal canal (eg, during spinal surgery or with implantable spinal devices). The source of infection is not identified in up to one-third of cases.[12] There are several proposed mechanisms of bacterial damage to the spinal cord, including direct mechanical compression of neural elements or blood supply, or thrombosis and thrombophlebitis of nearby veins.[12,108]

The most common isolated pathogen is *Staphylococcus aureus*, accounting for up to two-thirds of epidural abscess cases.[31] Empiric antibiotics should target the most commonly identified organisms: *S aureus* (with an increasing prevalence of methicillin-resistant *S aureus*), gram-negative bacteria (*Escherichia coli*), streptococci, coagulase-negative staphylococci (*Staphylococcus epidermidis*), and, rarely, anaerobes.[108–110]

Prompt recognition and proper management are imperative to avoid disastrous complications including sepsis, paralysis, or death. Spinal epidural abscess remains a challenging diagnosis to make, and almost half of cases are initially misdiagnosed.[12] In one study, the mean duration between symptom onset and the first ED visit was 5 days, and between symptom onset and hospital admission was 9 days. Patients averaged 2 ED visits before admission.[111] The difficulty in initially making this diagnosis likely stems from the relative infrequency of traditionally accepted signs and symptoms. The classic symptom triad of epidural abscess consists of fever, back pain, and neurologic deficits. As with most triads, few patients (<15%) have all 3 components at the time of presentation. Almost 75% have back pain and 50% are febrile initially.[12] The rate of neurologic progression is highly variable, and up to 67% of patients have a normal initial neurologic examination. Only 60% have a WBC count greater than 12,000 cells/mm^3.[15,110,112,113] These data underscore the need for a high index of clinical suspicion. Early diagnosis and treatment are imperative because the extent of preoperative neurologic deficit is an important predictor of the final neurologic outcome. Therefore, in cases in which the ED physician has a moderate to high pretest probability of disease, further workup should be pursued despite a normal WBC count, a normal initial neurologic examination, and the absence of fever.

Spinal epidural abscess commonly presents with fever and severe back pain that is usually aggravated by movement or palpation. Signs of nerve root injury or spinal cord compression may be present but are typically late findings. Left untreated, an epidural abscess will cause symptoms that progress in a typical sequence.[114] Providers see patients somewhere on this continuum. The disease starts with general malaise and a possible fever, followed by a backache that progresses to back pain and becomes severe. Patients may subsequently complain of nerve root pain and shooting pain in the distribution of the affected nerve. Many patients at this stage, characterized by nonspecific symptoms, may be misdiagnosed without a high index of clinical suspicion. These symptoms are then followed by motor weakness, sensory changes, bowel and bladder dysfunction, and, eventually, paralysis (4%–22%) or death (5%).[12,114] Patients may progress rapidly through these symptomatic stages, and some stages may be missed. Despite being initially present only half of the time, fever does help distinguish epidural abscess from other causes of back pain. If findings on the history or physical examination are worrisome for a spinal epidural abscess, patients urgently require MRI. With a low index of suspicion, including normal results of serum WBC count, ESR, CRP, and lumbosacral plain radiographs, patients can be discharged with close follow-up.

Epidural abscesses occur more commonly in the thoracolumbar area because of a larger epidural space and more infection-prone fat tissue. Because the epidural space is a vertically oriented sheath, longitudinal extension occurs so that infection at one level frequently tracks to adjacent spinal levels. Laboratory studies including blood cultures should be obtained. Lumbar puncture (LP) and direct needle aspiration have no role in the ED management of epidural abscesses. LP has a low diagnostic yield, with a Gram stain that is usually negative and cultures that are rarely positive.[12,114] MRI is the diagnostic test of choice because it aids in surgical planning and is the best test for early detection allowing for the diagnosis of small abscesses before the development of cord impingement.[12,115,116] For patients unable to have MRI, CT with gadolinium contrast is the next alternative. Plain films are rarely diagnostic early in disease.

The patient's final neurologic outcome is best predicted by their presurgical neurologic condition, highlighting the importance of timely diagnosis.[12,111,117,118] Standard treatment involves a combination of surgical and medical management.[12,15] Surgical treatment involves decompression and drainage of purulent material. Medical treatment focuses on systemic intravenous antibiotic therapy. The choice and timing of antibiotics and the decision to give steroids should be discussed with consultants from infectious disease and spine surgery if time allows. Initial antibiotics are broad spectrum, and should include vancomycin because of the increased prevalence of methicillin-resistant *S aureus*. Additional agents include metronidazole plus a third- or fourth-generation cephalosporin (eg, cefotaxime).[12] Subsequent antibiotic therapy will be based on culture and sensitivity results of blood cultures or needle aspirate. Because of the difficulty in predicting the progression of neurologic deficits, surgical consultation should be initiated from the ED with direct physician-to-physician communication.

Recent literature suggests that a subset of patients who are hemodynamically stable, neurologically intact, and at low overall risk of bad outcomes may be treated nonsurgically, although this is not the current standard of care.[119–123] These patients may include those who have serious underlying medical conditions making them an unacceptably high operative risk, those who refuse surgery, those with panspinal infection, or those with advanced neurologic deficits who are considered unlikely to improve with surgery. This last group consists of those with complete paralysis present for more than 36 to 48 hours before diagnosis and treatment. These patients may still benefit from nonemergent surgery to prevent a source of subsequent sepsis.[12,124] Some patients with small abscesses and no neurologic deficits may benefit from needle aspiration (to identify the organism) plus antibiotic therapy without surgical decompression.[119] Medical-only treatment is based on case reports and retrospective analysis that may be subject to reporting bias and does not represent the standard of care. Treatment decisions are best made by a team of consultants involving spine surgeons, internists, infectious disease specialists, and interventional radiologists.

EPIDURAL SPINAL CORD COMPRESSION

Epidural compression syndrome is a collective term encompassing spinal cord compression, cauda equina syndrome, and conus medullaris syndrome. These pathologic entities are grouped together because they share similar ED presentation, evaluation, and management. The only difference is the level of neurologic deficit at the time of presentation. The most common cause of epidural compression syndrome is a massive midline disc herniation, usually at the L4 to L5 disc level.[125] Other causes

include tumor, epidural abscess, spinal canal hematoma, or lumbar spine spondylosis.

As in epidural abscess, the primary determinant of ultimate neurologic outcome is the neurologic status at the time of diagnosis, so making an early diagnosis is critical. However, delayed diagnosis is common. Back pain with a progressive increase in intensity is often the first symptom. Unlike other causes of back pain, pain from epidural spinal cord compression is worse with recumbent positioning secondary to epidural venous plexus distention. In time, associated unilateral or bilateral radiculopathy may develop. There may be more complaints of leg pain or neurologic symptoms than of back pain in many patients. Weakness is also commonly present at the time of diagnosis. It is usually symmetric and may have progressed to the point of significant gait disturbance or paralysis. Cauda equina lesions are also associated with decreased lower extremity reflexes.

Although less common than motor findings, abnormal sensory findings may include lower extremity paresthesia and anesthesia. One of the most frequent sensory deficits is saddle region anesthesia, which denotes loss of sensation around the anus, genitals, perineum, buttocks, and posterior-superior thighs. Patients may complain about bowel, bladder, or sexual dysfunction, and may also have decreased anal sphincter tone (60%–80%) on physical examination.[26] Urinary retention with overflow incontinence (sensitivity of 90%, specificity of 95%) is a common, though late, finding.[26] The probability of cauda equina syndrome in patients without urinary retention is approximately 1 in 10,000. An attempt may be made to localize the lesion by noting the level of the neurologic deficits, such as with the loss of bowel or bladder function (S2–S5) or the loss of the ankle jerk reflex (S1–S2). Such an exercise may not be necessary because MRI scans through the entirety of the lumbosacral spine. With presumed cauda equina syndrome, a sensory level deficit or a positive Babinski reflex suggests involvement of the conus medullaris. This terminal region of the spinal cord lies in close proximity to the nerve roots. Pathology to this region can therefore yield both upper and lower motor neuron signs; a mixture of both spinal cord and nerve root dysfunction.

Similar to the pathologic appearance of spinal epidural abscess on MRI, multiple locations of pathology often coexist. Imaging should include the entire spine if there is concern of metastatic compression or infection. In other cases, regional MRI may be appropriate. Information obtained from the MRI helps the admitting team with prognosis and treatment planning. CT with myelography should be used in patients who are not candidates for MRI.

General treatment guidelines involve providing analgesia and steroids. Pain control is the most pressing need from the patient's perspective and often requires opioid analgesics. One must only imagine a patient with vertebral metastases lying on his/her back for a long MRI study to appreciate the importance of effective analgesia. Early and effective pain control also helps the physician complete the history, physical examination, and diagnostic testing in a more effective and timely manner. The administration of glucocorticoids can minimize ongoing neurologic damage from compression and edema until definitive therapy can be initiated. The optimal initial dose and duration of therapy is controversial, with a recommended dose range of dexamethasone anywhere from 10 to 100 mg intravenously.[126,127] The ED physician should administer the first dose as soon as the diagnosis is suspected, rather than waiting for confirmatory diagnostic testing that often takes hours to complete. High-dose steroids are associated with serious side effects but have proven efficacy, whereas low-dose steroids have a lower side effect profile but no randomized controlled data to support their use.[126] Traditional dosing (dexamethasone 10 mg) can be used in patients with minimal neurologic dysfunction, reserving the higher dose

(dexamethasone 100 mg) for patients with profound or rapidly progressive symptoms, such as paraparesis or paraplegia.[126,127] Some specialists may omit or delay glucocorticoids in patients with small epidural lesions without any associated neurologic abnormalities.

As previously stated, final neurologic outcomes may be predicted by the functional status of the patient on arrival. Most patients who require a catheter on arrival will continue to do so. Patients who are ambulatory on arrival will likely remain ambulatory. Patients who are too weak to walk but not paraplegic have an approximately 50% chance of walking again. Those patients who are paraplegic on arrival are unlikely to walk again.[128] These statistics highlight the need for early detection and give insight to probable prognosis. All patients with evidence of neoplastic epidural cord compression should be administered glucocorticoids, provided adequate analgesia, and be admitted to the hospital with urgent consultation and evaluation for possible operative decompression and/or radiation therapy. On average, outcome is improved if decompression takes place within 24 to 48 hours of symptom onset.[129–131] Consultation with surgery and radiation oncology initiated in the ED facilitates more timely intervention.

CANCER

Both benign and malignant tumors can cause myelopathy as a result of external compression or intramedullary growth. The most common syndrome involves metastatic spread to the epidural space, causing spinal cord compression. The thoracic spine is the most common site of bony metastasis.[132] Back pain is the initial symptom of spinal metastasis in most presentations. Patients present with pain at the site of the lesion that is often described as dull, constant, and aching. Unlike the pain associated with mechanical low back pain and disc herniation, which improves with rest, cancer-related back pain tends to be unrelieved by rest and may even worsen with recumbency. Severe nighttime pain is also characteristic. Pathologic compression fractures may present with abrupt worsening of back pain. Patients with neoplastic epidural spinal cord compression may report radicular symptoms and progressive weakness with accompanying sensory loss and bladder dysfunction. Rapid progression to paraplegia can occur secondary to vascular compression. Similarly to epidural abscess, timely diagnosis and treatment is essential because the ultimate neurologic prognosis depends on the neurologic function at the time of intervention.

Plain radiographs may or may not show destructive lesions in 1 or multiple vertebral bodies. MRI is the imaging modality of choice to assess for spinal metastasis and neoplastic epidural spinal cord compression. CT is a better imaging modality than MRI for cortical bone destruction. Although any tumor may involve the bone, the most common cancers that metastasize to the spine are breast, lung, and prostate.[133]

A systematic approach to the patient with cancer and back pain is accomplished by categorizing patients into 3 groups based on signs and symptoms. The first patient group has had a sudden or rapid change in their back pain and developed new or progressive signs or symptoms suspicious for epidural compression, such as bowel or bladder incontinence, weakness, loss of reflexes, or the development of bilateral or multiroot findings. These patients are at high risk for rapid deterioration and should be evaluated and treated as previously discussed for possible emergent epidural compression syndrome in the ED. In addition to high-dose corticosteroids, patients with a vertebral neoplasm may also benefit from radiation therapy. The second patient group has back pain with stable neurologic signs or symptoms present for days to weeks. These findings include an isolated Babinski sign or mild and stable unilateral neurologic symptoms such as weakness, sensory changes, or radiculopathy in a single

nerve root without evidence of cord compression. The presence of bilateral or multiroot involvement excludes patients from this group. Patients in group 2 should have plain radiographs performed in the ED and should also have MRI within 24 hours that can be done as an inpatient or outpatient. Considering the risk of myelopathic progression, it is safest to initiate the first dose of dexamethasone in the ED and admit these patients for pain control. With consensus and comfort between patient, ED physician, and PCP, patients can be discharged to their homes. The third patient group involves patients who have stable back pain without neurologic complaints or abnormalities suggestive of cord compression. These patients do not require treatment with dexamethasone. The ED evaluation should include plain radiographs. If there is any bony pathology, advanced imaging with MRI or CT is indicated as an outpatient within the next several days. If the plain radiographs are normal, further evaluation is not emergent. Patients must be closely followed by their PCP for improvement and lack of progressive symptoms. Follow-up appointments should occur within 1 week.

Some patients without known cancer have red flag signs and symptoms that are merely suggestive of malignancy, such as unexplained weight loss or back pain that is worse at night. As previously discussed, these patients require further risk stratification with plain radiographs and laboratory testing, including a WBC count, ESR, and CRP. With normal test results, these patients can be referred to their PCP for further workup and evaluation. With abnormal diagnostic results, such as a bone lesion on plain radiographs or an extremely increased ESR, urgent CT or MRI should be performed on an outpatient basis within the next week.

Unlike cauda equina syndrome, which usually requires only focal MRI of the lumbosacral spine, evaluation for malignancy requires screening MRI of the entire spine to evaluate for falsely localizing lesions, because clinically silent multilevel involvement is common and there is a 10% risk of distant asymptomatic metastases, which may affect subsequent treatment.[134] Additional imaging can be ordered from the ED and performed urgently from the hospital floor. Neoplastic epidural spinal cord compression is a true emergency and requires prompt diagnosis and treatment for the best possible patient outcome. Treatment in the ED would include high-dose corticosteroids with specialty consultation for radiation therapy and/or surgical decompression.

LUMBAR SPINAL STENOSIS

Lumbar spinal stenosis is usually secondary to degenerative arthritis (spondylosis). Stenosis of the vertebral canal occurs from a combination of loss of intervertebral disc height with bulging, facet joint hypertrophy and thickening of the ligamentum flavum.[27] Considering the aging population of the United States, ED physicians should expect to see this condition more frequently and increase our comfort with its diagnosis and treatment. Lumbar stenosis, in isolation, is frequently asymptomatic. Similarly to degenerative disc disease, there is poor correlation between the severity of symptoms and the degree of spinal canal stenosis seen on MRI. Neurologic symptoms are believed to be caused by both direct mechanical compression and nerve root ischemia. The classic and most common symptoms of lumbar spinal stenosis are that of neurogenic claudication: burning pain in the back that radiates to the buttocks and posterior-lateral legs.[26,27] Some patients may only note symptoms when active and not at rest. There are associated bilateral sensory changes, such as numbness or tingling and/or mild weakness affecting the legs, that are often asymmetric. Focal weakness, sensory loss, or reflex changes may occur when spinal stenosis is associated with radiculopathy. The physical examination may reveal single

or multiple lumbosacral radiculopathies with sensory loss, areflexia, and/or focal weakness in the distribution of the involved nerve roots.[28] Symptoms are usually bilateral and aggravated by walking, prolonged standing, or spinal extension, and relieved with sitting, lying, or waist flexion.[27,28] This is because erect posture narrows the cross-sectional area of both the central canal and neural foramina. Neurogenic claudication (also called pseudoclaudication) is differentiated from vascular claudication by etiology and symptomatology. Neurogenic claudication is caused by neurologic compression, not by arterial insufficiency.[26] Unlike vascular claudication, symptoms are often provoked by standing erect without walking, and may persist while at rest.[28]

Diagnosis is made with clinical findings that suggest spinal stenosis and with a neuroimaging study that shows structural narrowing. A history and physical examination can allow a presumptive diagnosis of spinal stenosis; however, a positive neuroimaging study without clinical correlation (ie, an incidental finding of canal narrowing) is insufficient for diagnosis. Just as in degenerative disc disease, radiographic spinal stenosis is an age-related population norm. More than 20% of asymptomatic persons greater than 60 years of age may have findings of spinal stenosis on imaging studies.[27] MRI is the study of choice and CT myelography is a second option in those in whom MRI is contraindicated. ED management of patients with spinal stenosis should be conservative and focus on pain control with acetaminophen, NSAIDs, and opiates. In the absence of alarming red flag findings (progressive neurologic deficit or evidence of cauda equina syndrome), these patients do not require laboratory or radiographic studies in the ED. Data supporting the role of epidural injection are sparse and inconclusive.[27] There is no role for epidural injection in the ED. Evaluation should also include outpatient referral to the patient's PCP to maximize medical management. The ED physician can consider an outpatient surgical referral for those patients with symptoms that are either severe or functionally disabling or for those in whom maximal medical therapy does not relieve symptoms sufficiently to allow for activities of daily living.

BACK PAIN IN CHILDREN

Although back pain represented only 0.4% of all visits to an inner-city pediatric department,[135] a brief mention of pediatric pathology causing back pain is warranted in this discussion. Most cases of back pain in children and adolescents receive no definitive diagnosis. However, children with back pain are more likely than their adult counterparts to have an underlying pathologic cause for their pain. Common identifiable causes include trauma (up to 25%) and nonspecific/benign musculoskeletal pain (24%–50%).[136–139] Other causes included idiopathic pain (13%), infections such as urinary tract infection (5%), viral illness (4%), and other miscellaneous causes (6%).[135]

The standard approach of the history and physical examination is to evaluate for red flags, just as in adults. Although age less than 18 years is considered a red flag, close attention should be paid to children less than 10 years old, particularly those less than 5 years old. Tumor, discitis, and malignancy occur with greater frequency in this age group.[140] Osteoid osteoma is the most common tumor in children who present with back pain. This tumor classically presents as intense nocturnal pain relieved with NSAIDs.[141] Protracted (>3–4 weeks) or worsening pain is also more concerning in children.[140] Inquire as to whether the pain interferes with activity and play. Pain preventing child play should raise concern. Inflammatory disease is suggested by morning stiffness and limited mobility that improves with a hot shower and activity and returns with rest. Inquire about recent febrile or bacterial illness. Young athletes with back pain represent a special subpopulation with a high incidence of structural injuries to

the posterior spinal elements such as spondylolysis.[14] If the child participates in sports, ask about training intensity, duration, frequency, and especially recent increases that may suggest overuse injury. Children with sickle cell disease may have avascular necrosis of vertebral bodies. Caution should be exercised when attributing back pain to scoliosis in children because it is rarely a painful condition.

Physical examination should include physician-observed ambulation and a focused lower extremity neurologic evaluation, just as in adults. Pain with flexion suggests muscle injury/spasm or injury to the anterior spinal elements. Pain with extension indicates injury to the posterior elements or sacroiliac joint. Refusal to walk may suggest occult trauma, discitis, or osteomyelitis. As in adults, the clinical evaluation should direct the laboratory and radiologic evaluation. Children with a short symptom duration who appear well, with a normal neurologic examination in the absence of any red flags, can be conservatively managed without further diagnostic testing. They can be discharged with good-quality discharge instructions and follow-up with their pediatrician. Children with concerning signs or symptoms should proceed with a WBC count and ESR. Other tests, such as urinalysis and blood cultures, should be ordered if clinically appropriate. Plain radiographs of the lower back should also be obtained, with consideration of oblique views if spondylolysis is a consideration. Routine oblique views should not be performed. Children with suspected malignancy, spine infection, or progressive neurologic findings benefit from MRI. Timing and location of the study should be coordinated with the pediatrician or hospitalist.

Spondylolysis is a unilateral or bilateral defect in the pars interarticularis portion of the vertebrae. It is a stress fracture mostly seen in the lumbar vertebrae, and most commonly L5. Spondylolisthesis can occur when bilateral spondylolytic defects allow the forward translation of 1 vertebral body on another, occurring at L5 on S1 most frequently. It is graded based on the percentage of the lower vertebral body that is now uncovered (eg, 25%), termed slippage. Repetitive microtrauma to the bone from lumbar hyperextension or repeated lumbar flexion and extension leads to pars defects and eventual spondylolysis. Progression to spondylolisthesis occurs during the growth spurt and is correlated with persistent pain and lack of healing.[142] Spondylolysis is common in adolescent athletes and presents with acute lower back pain.[14] Sports that involve lumbar hyperextension, repetitive flexion/extension, or torsion have the highest incidence (eg, gymnastics, dance, figure skating).[140,143] ED diagnosis of back strain in this population should only occur following careful consideration of more serious pathology, including spondylolysis or spondylolisthesis. The typical patient is an active adolescent who complains of insidious onset of aching lower back pain that may radiate into the gluteal region, usually with activity. Pain is relieved with rest and worsened by extension or lateral bending.[139] On physical examination, patients may be focally tender to palpation and have pain worse with lateral bending or extension than with lumbar flexion. The neurologic examination is frequently normal. Clinical suspicion may be increased with a positive single-legged hyperextension test (stork test) in which the child stands on 1 leg and bends backward, thereby exacerbating ipsilateral lower back pain.[140,144] If neurologic symptoms and/or radiculopathy are present, an alternative diagnosis should be considered, because they are rarely associated with spondylolysis. There is no role for diagnostic laboratory tests.

Diagnostic imaging should start with plain radiographs with added oblique views. Classically, oblique views show the ‘‘Scotty dog’’ sign with a crack on the dog’s neck/collar, the pars.[145] The Scotty dog’s head (superior articular facet), nose (transverse process), eye (pedicle), neck (pars interarticularis), and body (lamina) should be easily identified on the oblique radiograph. A defect in the pars may indicate an acute fracture or an old nonunion. Bony sclerosis may be seen if healing has begun. ED

management includes pain control, cessation of offending activities, particularly extension activities, and rest. Outpatient follow-up with an orthopedic specialist should be made. Inconclusive ED workup should involve discharge with close follow-up with the patient's pediatrician for consideration of single-photon emission computed tomography (SPECT) scan or MRI.[144,146] Spondylolisthesis is managed by observation. Progression usually stops when the child achieves skeletal maturity. Current recommendations are for limited contact sports in children with less than 30% to 50% slippage and surgical stabilization for children with slippage greater than 30% to 50%. Treatment becomes more aggressive if the child is symptomatic.

In addition to the infectious pathology discussed earlier, children are prone to inflammation and infection of the intervertebral discs, particularly in the lumbar region. Untreated, this may spontaneously resolve or progress to vertebral osteomyelitis or abscess formation. In comparison with children with osteomyelitis, children with discitis tend to be younger (2.8 vs 7.5 years of age), are less likely to be febrile (28% vs 78%), have a shorter duration of symptoms (22 vs 33 days) and are clinically less toxic in appearance.[10] Parents bring their young child to the ED noting irritability and reported back pain (27%), often associated with a limp or refusal to crawl or walk (63%).[147] Physical examination findings are nonspecific and may include an inability to flex the lower back (50%), loss of lumbar lordosis (40%), a tendency to lie still, and percussion tenderness over the involved spine.[140,147] Asking the child to pick up an object from the ground may be a helpful part of the physical examination. Fever is absent or low grade. Early in the disease course, systemic toxicity is rare and, if present, suggests osteomyelitis. WBC count can be normal early in the disease course, but the ESR is increased in more than 90% of patients.[10] The imaging study of choice is MRI. Children with MRI findings of discitis should be admitted, provided with pain control, and started on empiric antibiotics to cover the most common isolate, *S aureus*, in addition to other less common isolates including coagulase-negative *Staphylococcus* and *Kingella kingae*.[148] Appropriate initial antibiotics to be started in the ED include vancomycin and a third-generation cephalosporin such as ceftriaxone.

ADDITIONAL INFORMATION

In general, the recommended role of the ED physician in the management of acute lower back pain is to rule out significant pathology and obtain a correct diagnosis while avoiding excessive investigation. Subsequent goals include initiating appropriate treatment, providing analgesia, and patient education. Although patients should avoid vigorous exercise and provocative or high-impact activities after treatment, complete rest is not recommended. Bed rest has been proved to be deleterious to successful recuperation from back pain, leading to less functional recovery and slightly increased pain than in those advised to remain ambulatory.[149] Remaining active also helps with muscle spasm and atrophy. The ED physician should recommend that patients continue their daily activities and gradually increase specific exercises as tolerated. Patients should understand that back pain does not need to be totally alleviated before returning to work. Issues of return to work should be based on consideration of the work duties of the patient. Unlike a white-collar desk job, the patient with a job involving heavy manual labor may benefit from time away from work if no light-duty options are available. The ED work note should make this distinction clear.

Patients should understand that emergent evaluation by a back surgeon (neurosurgeon or orthopedic surgeon) is indicated for patients who are having back

emergencies such as severe or progressive motor weakness, epidural abscess, or signs and symptoms of epidural compression syndrome. Outpatient referral for entities such as persistent disabling symptoms, including severe pain and radiculopathy, are elective and should be done only after attempts at maximal medical treatment have failed, and made by a patient's PCP. Carefully selected and presented advice and information about back pain can have a positive effect on patients' beliefs and clinical outcomes. The ED physician should reassure the patient by acknowledging their pain and being supportive. Care should be taken to avoid negative or confusing messages. An example of this would be avoiding language (eg, ruptured disc) that may frighten the medically naive patient and that may imply a serious abnormality when none exists.[150]

It is important to provide a full explanation of the diagnosis, evaluation, treatment plan, and anticipated time course for expected recovery in terms that the patient understands. For example, patients should be educated about why they are not undergoing laboratory tests or radiographic studies of their lower back and should be reassured of the likely benign course of the pain. Most patients can be convinced by education and an explanation of radiation dosing and associated deleterious effects. This approach helps avoid misperceptions of substandard care or subsequent unnecessary return visits within 48 hours when symptoms are still present. Patients should be reassured that back pain is common, that the pain does not indicate ongoing harm or serious pathology, and that the outlook is good. As mentioned earlier, the ED physician should avoid making unnecessary presumptive diagnoses and avoid the medicalization of benign conditions by ordering unnecessary tests. This behavior, coupled with the overprescription of analgesics (particularly opiates), fosters a belief on the part of the patient of the existence of serious pathology for an otherwise benign condition. If the facility evaluating the patient is unable to obtain an appropriate imaging study (eg, MRI) when needed, consider transfer to another facility with ready radiology access and specialty consultation for the patient with suspected spinal infection or epidural compression syndrome.

Patients may ask the ED physician about complementary and alternative treatments. Some supplemental treatment modalities have been shown to be of debatable efficacy in the management of acute and chronic low back pain. These treatments include acupuncture, physiotherapy, chiropractic manipulation, massage, ultrasound, traction, and transcutaneous nerve stimulation.[65,151] These treatments may be useful adjuncts to the ED physician's armamentarium for the outpatient treatment of many back conditions and may be of particular help for patients with acute flares of chronic or subacute back pain. The greatest benefit of these modalities has been found in patients who have belief in, and higher expectations of, the efficacy of a specific treatment modality, or who have had a favorable response to a particular modality in the past. These patients are more likely to derive benefit in the future and to demonstrate greater functional improvement at 12-week follow-up.[152] These treatments are generally safe and do not involve harm, so patients can be encouraged to pursue them as an outpatient. These modalities tend to cost more than conventional medical supportive care but, like most alternative treatments, are associated with enhanced patient satisfaction. Physical therapy may be beneficial for those patients with symptoms present for 4 to 6 weeks, although not in the acute setting.[153] Spinal manipulation involves moving a joint, in this case the spinal column, beyond its usual end range of motion but not past its anatomic range of motion. There is controversial benefit, with studies showing mixed results, although it may be as effective as conventional treatment.[154,155] Acupuncture and massage do not show clear data for acute treatment, but may be helpful for chronic back pain.[156,157]

Perhaps the most important aspect of ED management of acute back pain involves the discharge instructions. All patients with back pain evaluated in the ED who are not admitted should be given clear instructions with unambiguous indications to return or go to the nearest ED with symptoms such as new or progressive leg weakness, bowel or bladder dysfunction, or saddle anesthesia. Although not practical considering the available time for patient encounters in the ED setting, time spent discussing prevention is time well spent. This discussion may take the form of preprinted written discharge instructions. The future of ED medicine likely involves a greater number of uninsured and primary care patients, so there are invaluable benefits to providing information detailing the benefits of exercise, weight loss, staying active, and avoidance of activities that involve repetitive twisting or bending or high-impact activities that increase spinal stress. Proper bending and lifting techniques should be included.

Almost all patients with nonspecific lower back pain can be discharged from the ED with follow-up with their PCP. In rare circumstances, severe ongoing pain despite treatment and/or inadequate support at home for recovery may preclude discharge. Patients with cancer and intractable bony pain may also require admission for pain control. For patients who have a red flag diagnosis of cauda equina syndrome or epidural abscess, immediate neurosurgical consultation is required for emergent surgical decompression. Patients with epidural abscess also require administration of intravenous antibiotics.

The clinical pitfall to avoid is diagnosing an emergent back pain episode as just a back strain. The ED physician should always check for the presence of red flags in all patients who have back pain. To summarize, the patients who have low back pain emergencies are: (1) those who have a past medical history of malignancy and new back pain with neurologic findings, (2) those who have back pain and symptoms of epidural compression syndrome, (3) those who have back pain with symptoms suggesting an infectious cause, (4) those who have back pain with gross muscle weakness or paralysis, and (5) those who have back pain and bilateral or multiple nerve root involvement.

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