Atraumatic Back Pain

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KEYWORDS

• Back pain • Spinal cord and cauda equina compression • Spinal epidural abscess
• Spontaneous epidural hematoma • Malignant epidural spinal cord compression

KEY POINTS

- Approach the complaint of back pain broadly—a common reason for missing a neurologic emergency in patients presenting with back pain is failure to consider the diagnosis.
- A detailed history and neurologic examination are crucial in differentiating emergent from nonemergent causes of back pain.
- Decide imaging levels carefully: atraumatic myelopathies and skip lesions from infection or metastases may occur outside of the cauda equina, with similar neurologic presentation.

INTRODUCTION

Atraumatic back pain, or lumbago, is a prevalent condition affecting a substantial portion of the adult population. The Global Burden of Disease study has shed light on the extensive impact of low back pain, consistently ranking it among the top 4 most burdensome nonfatal conditions worldwide.[1] Epidemiologic data indicate that approximately 80% of individuals will experience low back pain at some point in their lives, establishing it as a leading complaint in both primary care and emergency departments (EDs). According to the National Hospital Ambulatory Medical Care Survey, back pain makes up a considerable proportion of ED visits in the United States. In 2021, approximately 4.4 million ED visits were attributed to back pain in the United States, comprising 2% to 4% of total visits.[2,3] The prevalence of serious spinal pathology in patients presenting with low back pain is higher (2.5%–5.1%) in those presenting to the ED than in those presenting to primary care (<1%).[4] ED clinicians, therefore, must navigate the diagnostic challenge of a common chief complaint that can typically be managed in the outpatient setting with a small subset at risk for serious neurologic injury, paralysis, and death. We have, therefore, structured this review within a clinical

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Abbreviations	
AAA	abdominal aortic aneurysm
CSF	cerebrospinal fluid
CT	computed tomography
DDD	degenerative disc disease
DVIs	disco-vertebral infections
ED	emergency department
ESCC	epidural spinal cord compression
GBS	Guillain–Barre syndrome
IDU	injection drug use
IR	interventional radiology
IV	intravenously
LMN	lower motor neuron
MESCC	malignant epidural spinal cord compression
MI	myocardial infarction
MOUD	medications for opioid use disorder
MRSA	*methicillin-resistant Staphylococcus aureus*
NSAIDs	nonsteroidal anti-inflammatory drugs
OA	osteoarthritis
PE	pulmonary embolism
PLEX	plasma exchange
RP	retroperitoneal
SCC	spinal cord compression
SEA	spinal epidural abscess
SEH	spinal epidural hematoma
TB	tuberculosis
TM	transverse myelitis
UMN	upper motor neuron
VCF	vertebral compression fracture

framework that separates emergent, "worst first" causes of atraumatic back pain requiring immediate intervention, and nonemergent causes that are less time-sensitive, although some still ultimately require emergent management or admission.

History and Physical Examination

History and physical examination are critically important in differentiating patients who present with atraumatic back pain and are safe for discharge from patients who require emergent intervention.

- History
  - The differential for patients presenting with atraumatic back pain is broad, and diagnosis is dependent on a detailed history. Historical details guide the physical examination, risk stratification, and imaging decisions. A classification and description of the pain is the starting point. It is important to determine the onset (sudden vs gradual), location, radiation, quality, intensity, and aggravating/alleviating factors for the pain. Next, one should determine a temporal timeline of the episodes—with focus on the frequency, severity, treatment, and any imaging history. Certain elements of a patient's past medical history are also critical. History of cancer (especially with bone metastases), injection drug use (IDU), anticoagulation, osteoporosis, immunosuppression (human immunodeficiency virus [HIV], organ transplant, and chronic steroid use), autoimmune disease (rheumatoid arthritis, ankylosing spondylitis, and multiple sclerosis), and recent infection, surgery or invasive spinal procedures can all provide important clues. The review of systems is equally important as findings

can lead to proper diagnosis. While not all-encompassing, systemic symptoms such as fever, chills, unexplained weight loss, night sweats are concerning. Documenting the presence or absence of neurologic symptoms is paramount. Patients reporting weakness (focal or progressive), numbness, radiculopathy, gait disturbance, saddle anesthesia, and bowel or bladder dysfunction (urinary retention and urinary or fecal incontinence) should be moved into the emergent category.

- Physical examination
  - A thorough physical examination, guided by the history presented, is the next step in the evaluation process for all patients presenting with atraumatic back pain. Physical examination should touch on the general appearance including signs of systemic illness (fever and pallor). Second, a spinal examination should be performed, including palpation for tenderness, step-offs, deformity, and assessment of range of motion (note limitations and pain with movement). A very detailed neurologic examination is critical. This should assess motor strength, sensation, reflexes, and gait. The neurologic examination should specifically test for, and document, any signs of spinal cord compression (SCC) including cauda equina, though these are unreliably present on examination and historical risk factors should still lead to further workup (see examination findings later).[5] Finally, an abdominal examination palpating for abdominal tenderness or pulsatile mass and vascular examination assessing peripheral pulses will provide a complete examination.
  - Findings highly concerning for SCC include perianal/saddle anesthesia, bowel/bladder dysfunction, bilateral extremity weakness, and upper motor neuron (UMN) or lower motor neuron (LMN) findings. UMN findings include hyperreflexia, clonus, spasticity and Babinski sign, occurring with SCC above L1. Made up of the spinal nerve roots below L1, cauda equina compression can result in an LMN syndrome with resultant hyporeflexia/areflexia and ultimately flaccid paralysis. Saddle anesthesia and fecal incontinence are also uniquely associated with cauda equina compression. It is important to note the classic feature of urinary incontinence or retention in "cauda equina syndrome" can also occur with SCC of the thoracic spine. Many patients struggle to characterize early urinary sphincter dysfunction, and ensuring that the postvoid residual is less than 150 cc provides an objective measure of intact function.
- Concerning findings or "red flags"
  - Reproducible data behind "red flags" are elusive, and the term "red flag" is often a grab bag for both historical and examination findings, which should be clarified in documentation.[6–8] The term "red flags" should therefore be utilized with some caution in clinical decision-making, though these bear considering in patients complaining of low back pain—anticoagulation use, new urinary retention, and disturbance of saddle sensation.[4,7–9] Etiology-specific risk factors will be discussed later.
- Imaging
  - Imaging is important in the evaluation of patients with atraumatic back pain from both emergent and nonemergent causes (**Fig. 1**). Preferred modalities will be discussed in the individual sections. While advanced imaging is often necessary in the setting of risk factors and/or neurologic deficits on examination, it is important to note that routine imaging without clinical correlation is not recommended and can lead to unnecessary and potentially harmful interventions.[10,11]

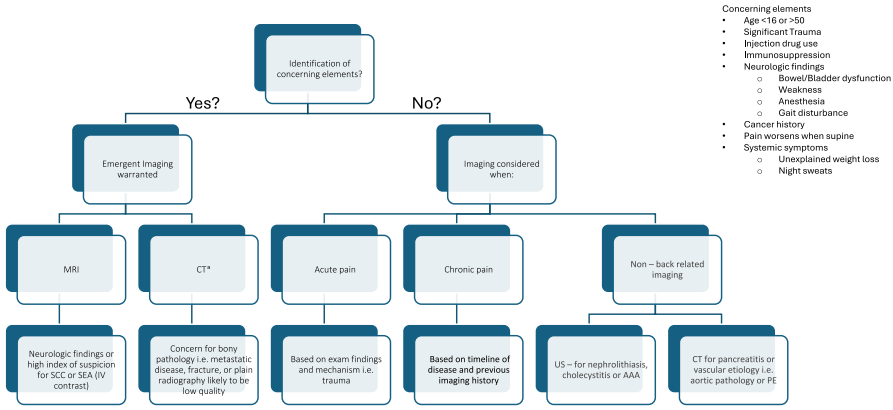

Fig. 1. Evaluation of patients with atraumatic back pain from both emergent and nonemergent causes. [a]Plain radiography can be considered first line based on patient condition and presentation, however, CT has higher sensitivity in patients with concerning findings.

Overview of Emergent Causes

Emergent, spinal causes of atraumatic back pain require immediate intervention due to either a compressive or noncompressive myelopathy, which can lead to permanent neurologic damage or death (**Table 1**). Nonspinal causes of back pain that require immediate intervention should also be on the differential, including, but not limited to aortic dissection, symptomatic abdominal aortic aneurysm (AAA), spontaneous retroperitoneal (RP) bleed, pulmonary embolism (PE), and myocardial infarction (MI). Completing a detailed history, neurologic examination, and vascular examination are critical diagnostic steps and care should be taken to document these findings as they inform clinical decision-making.

Compressive Myelopathies

Compressive myelopathies have numerous atraumatic causes including malignancy, abscess, hematoma, degenerative spondylosis, and disc herniation. Clinical presentation varies by etiology and level of compression as discussed earlier, though pain, weakness, and gait disturbance are common features.[12,13] Cervical cord compression is the most common location, followed by lumbar or cauda equina and then the thoracic cord.[13,14] This cervical predominance contradicts the traditional focus on cauda equina compression in the ED literature and physician training, which may lead to premature closure and diagnostic delay. Additionally, while level-specific history and examination findings may help localize spinal pathology, ED management is largely based on etiology not spinal level. We have, therefore, approached spinal cord and cauda equina compression (SCC) collectively in this review.

Degenerative Spondylosis and Disc Herniation

Degenerative spondylosis is age-associated degeneration of the vertebral bodies, intervertebral/uncovertebral (cervical) joints, and facet joints, which can result in spinal stenosis and subsequent myelopathy. It is most common in adults aged older than 55 years. Massive disc herniation, defined as 33% to 75% narrowing of the spinal canal resulting in SCC can be seen as early as the third decade but is most common after the age of 40 years. Disc herniation and degenerative spondylosis are the most common causes of SCC in the United States, though global literature illustrates varying etiologic patterns.[13–18] Emergent presentations are similar across entities and no

Table 1
Diagnosis and management of emergent atraumatic back pain

	Diagnosis	Risk Factors	Imaging	ED Management[a]	Definitive Management
Compressive[b] myelopathy	SEA/DVIs	IDU, indwelling line, internal hardware, immunocompromise, diabetes, spinal surgery, and contiguous infection	Blood culture X2 CRP/ESR Total spine MRI w/contrast (consider CT to assess for psoas abscess)	Empiric antibiotics:[c] Ceftriaxone 2 g IV Vancomycin 25–35 mg/kg IV Pain control Early MOUD[d]	Surgical decompression and antibiotics (select cases IR drainage with antibiotics)
	SEH	Anticoagulation and neuraxial procedure	MRI w/contrast	Reverse anticoagulation Pain control	Surgical decompression
	MESCC	Cancer or examination concerning for cancer	Total spine MRI w/contrast	Dexamethasone 10 mg IV Pain control	Radiotherapy and surgical decompression
	Degenerative spondylosis/disc herniation	Age >55 y or age >40 y, respectively	MRI w/o contrast (unless concern for above etiologies)	Pain control	Surgical decompression
Noncompressive myelopathy[e]	TM/GBS	Preceding infection and autoimmune disorder	MRI w/contrast and lumbar puncture	Corticosteroids (TM)/ intravenous immunoglobulin (IVIG) (GBS)	Corticosteroids, IVIG, PLEX, and cause-specific treatment
Nonspinal etiology[e]	Aortic pathology, RP bleed, PE, and MI	—	—	—	—

[a] Pain control should be initiated for all causes.
[b] Spine surgery consultation should occur for all emergent causes of compressive myelopathies.
[c] Consider pseudomonal coverage if risk factors.
[d] MOUD, Medications for opioid use disorder.
[e] Broad differential diagnosis–select review included here.

historical or physical examination features are specifically suggestive of this etiology of SCC.[8] MRI is the imaging modality of choice and should include gadolinium if there is suspicion for alternative etiology such as malignancy or infection.

Definitive treatment of SCC secondary to spondylosis and disc herniation is surgical decompression. A spine surgeon (orthopedic or neurosurgery) should be emergently consulted, or the patient transferred for surgical evaluation if spine surgery is unavailable at the presenting center. Multimodal pain control and, as needed, anxiolysis should be initiated for patient comfort and MRI tolerance. Strategies for short-term, multimodal pain control include nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, lidocaine patches, opioids, physical repositioning, and cryotherapy. The evidence behind corticosteroid use remains limited. In a 2022 Cochrane review for systemic corticosteroids in low back pain, short-term pain and function were only slightly improved in radicular back pain and no clear benefit was found in nonradicular presentations. Medical management, however, does not improve outcomes in the acute SCC secondary to massive disc herniation or degenerative spondylosis. Even with surgical decompression, many patients experience persistent motor or sensory deficits at discharge.[13]

Malignant Epidural Spinal Cord Compression

Malignant epidural spinal cord compression (MESCC) is a painful complication of cancer with high associated morbidity and a median survival rate of 3 to 6 months following diagnosis.[19] The average annual incidence of MESCC is 3% to 5% in patients dying of cancer, though incidence varies widely by primary cancer site.[19–22] Lung cancer, prostate cancer, multiple myeloma, and breast cancer are the 4 most common underlying malignancies.[19] Notably for ED clinicians, 20% of patients have no preceding cancer diagnosis and epidural spinal cord compression (ESCC) is the presenting diagnostic event.[23] The majority of MESCC cases is due to metastatic tumor in the vertebral bone through hematogenous or contiguous spread.[20] Tumor in the epidural space compresses the epidural venous plexus, resulting in vasogenic edema, and ultimately, if untreated, spinal infarct. MESCC most commonly occurs in the thoracic spine (60%), followed by the lumbosacral (25%) and then cervical spine (15%), and multiple sites of compression are possible.[20] Risk factors associated with MESCC are history of cancer or an examination concerning for cancer. Patients typically develop pain (95%) as the initial symptom of MESCC and associated radiculopathy is common. Mechanical back pain (with movement) is concerning for spinal instability. Weakness followed by sensory deficit are often also noted at initial presentation, which may be described as heaviness or clumsiness. Sensory deficits and bowel or bladder dysfunction are later findings and a poor prognostic indicator. MRI with and without contrast is highly accurate for diagnosing MESCC and should be urgently pursued. CT is a second-line option if MRI is contraindicated or unavailable. Up to one-third of patients have more than one site of compression, so the full spine should be imaged.

First-line treatment in MESCC are corticosteroids, serving as a bridge to definitive treatment and a component of multimodal pain control. Data are limited, and recommendations are largely based on expert opinion. In the ED, patients with neurologic deficits or pain should be loaded with 10 mg dexamethasone intravenously (IV). It has been proposed that very high-dose dexamethasone (96 mg IV) be used in cases of paraplegia associated with MESCC. However, a 2015 Cochrane review showed no benefit to this approach and associated toxicity with high-dose steroids.[24] Some recommendations suggest avoiding steroid initiation in patients with small lesions, normal neurologic examination, and no pain, which is an unusual constellation in ED

presentations. Opioid pain management may be necessary in the setting of malignant pain, which can be severe. In this setting, it is crucial to consider the morphine milligram equivalents of baseline pain regimens to ensure breakthrough pain is adequately treated. Definitive management consists of radiotherapy and/or surgical decompression, weighing multiple factors including grade of compression, radiosensitivity of the primary tumor, mechanical instability, and life expectancy given in the setting of invasive surgery.[20,24] Ultimately, this is a multidisciplinary decision made with collaboration between the patient, their primary oncologist, radiation oncology, and spine surgery. These services should be consulted emergently and transfer initiated if they are not available at your institution. A palliative care consult should also be considered. Prognosis is largely based on neurologic function at diagnosis and rapidity of onset. Patients with slow symptom onset who can walk prior to treatment have better functional outcomes.[20,23] As previously discussed, mortality is high in this patient population given underlying malignancy and advanced metastatic burden.

Spinal Epidural Hematoma

Atraumatic spinal epidural hematoma (SEH) can be spontaneous or procedure-related. Spontaneous events are exceedingly rare, with an incidence of 0.01 per 100,000.[25] Coagulopathy, vascular malformations, and tumors are risk factors, and incidence is likely increasing with the rising use of anticoagulation. Procedure-related SEH is more common (1:200,000), though still unusual.[26] Thus, neuraxial analgesia and lumbar punctures are significant risk factors. SEH and the diagnosis should be considered in presentations with this history.[27] In all contexts, patients typically present with severe pain that occurs over days followed by loss of motor, sensory, and bowel or bladder function. MRI with contrast is the gold standard for diagnosis (**Fig. 2**B). SEH associated with severe or progressive neurologic deficits requires definitive management with decompressive evacuation and laminectomy.[28] Either a spine surgeon should be emergently consulted or the patient transferred if definitive management is not available at the presenting center. Some patients with SEH on imaging but reassuring neurologic examinations may be monitored conservatively.[28,29] Reversal of anticoagulation should be strongly considered. Outcomes are directly tied to the severity of symptoms at the time of diagnosis.[28,30]

Spinal Epidural Abscess

Spinal epidural abscess (SEA) is an uncommon and serious cause of back pain with associated morbidity and mortality. It is defined as a collection of pus between the dura and vertebral body and can occur at any level of the spine.[31] Route of infection is most often bacteremia (IDU, infected hardware), contiguous spread (osteomyelitis, spondylodiscitis, or psoas abscess), and recent spinal procedure.[32] Prevalence is highest at ages 50 to 70 years.[33] Between 2005 and 2015, the incidence of SEA in the United States has increased by 2 fold to 3 fold (5.1 cases per 10,000 admissions), influenced by multiple factors, including the growing age of the population with associated comorbidities such as diabetes and the evolving opioid epidemic with associated IDU.[32,34] One in 5 patients with SEA aged 15 to 64 years injects drugs (IDU-SEA).[34]

SEA is the most frequently missed serious neurologic condition in patients presenting to the ED with low back pain (44%).[4] Missed or delayed diagnoses of SEA are associated with poor outcomes, particularly given the risk of sudden decompensation due to cord infarction from vascular thrombosis.[31,35] Presentations of SEA are classically described as a triad of fever, back pain, and neurologic deficit. While this triad is highly concerning for SEA, it is rarely present, and diagnosis can be elusive.[11] Fever is,

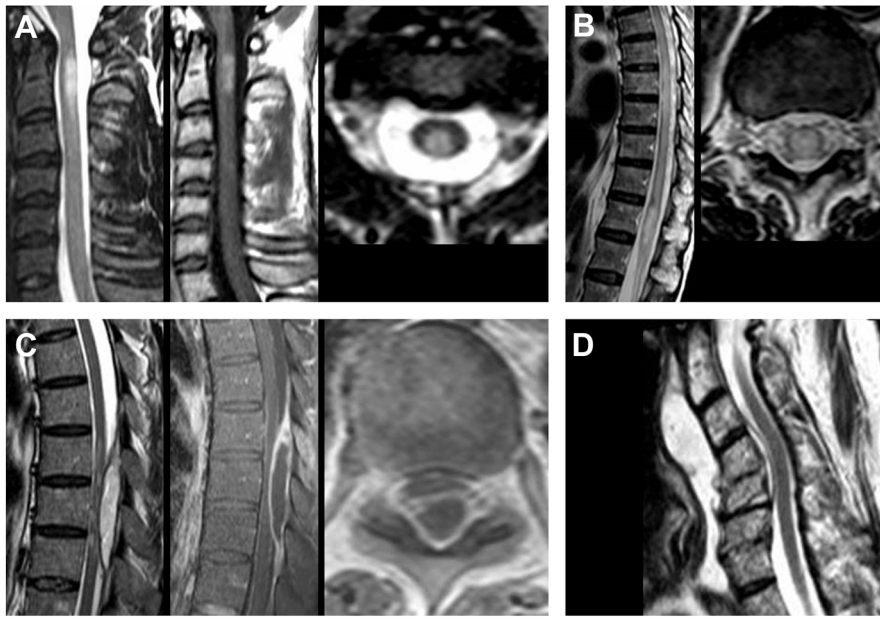

Fig. 2. MRI of acute myelopathies. (*A*) Myelitis isolated to the posterior aspect of the upper cervical cord; (*B*) Dural arterio-venous fistula with extensive longitudinal lesions of the thoracic cord and conus medullaris; (*C*) Epidural abscess resulting in posterior compression of the thoracic cord; (*D*) Spondylodiscitis resulting in posterior dislocation of the middle thoracic cord.[18] (*From* Stasolla A, Prosperini L, Haggiag S, et al. Non-traumatic acute myelopathies: Clinical and imaging features in a real world emergency setting. Neuroradiol J. 2022;35(6):727–35. https://doi.org/10.1177/19714009221096823.)

in fact, uncommon (32%); thus, a high index of suspicion is required in patients with back pain and risk factors.[33] Historical risk factors with a high positive likelihood ratio for SEA include those that predispose for bacteremia, such as IDU, concomitant infection site, and indwelling vascular catheter or hardware.[8,36] Diabetes, immunocompromise, and recent invasive spinal procedure or fracture have also been associated with SEA.[8,32,36] Lack of these risk factors suggests against SEA as the underlying etiology; however, historical red flags are frequently missed during history and chart review leading to delayed diagnosis and subsequent patient harm.[31] A thorough history and examination is, therefore, critical in the diagnosis of SEA, which is further emphasized by the limited utility of laboratory studies. ertythrocyte sedimentation rate (ESR) greater than 20 and c-reactive protein (CRP) greater than 1.0 are sensitive (ESR 100% and CRP 87%) in the setting of a risk factor but nonspecific, and leukocytosis is not routinely present.[8,32,36]

MRI with contrast is the gold standard when evaluating for SEA (**Fig. 2**C). The full spine should be imaged given the prevalence of skip lesions. *Staphylococcus aureus* is the most common causative bacteria with *methicillin-resistant Staphylococcus aureus* (MRSA) comprising 25% of those cases.[32] However gram-negative bacilli, including *Pseudomonas aeruginosa*, comprise up to 20% of cases and *Mycobacterium tuberculosis* is also a consideration, indicating the need for culture data.[32,37] Antibiotics should be initiated immediately after obtaining 2 sets of blood cultures. A reasonable empiric regimen pending culture data is ceftriaxone 2 g IV and vancomycin 25 to 35 mg/kg IV. Cefepime or meropenem should be considered if *Pseudomonas* is a

concern (eg, intensive care unit patient and indwelling spinal hardware). Most patients will require surgical decompression, and a spine surgeon (neurosurgery or orthopedics) should be emergently consulted for definitive management or the patient transferred if these are not available at the presenting center. Occasionally, medical management combined with interventional radiology (IR)-guided aspiration may be sufficient; however, data are limited. Multimodal pain control is important in the recovery process, particularly in the setting of IDU. Patients with IDU-SEA are more likely to leave against medical advice, often in the setting of withdrawal, which leads to delays in treatment and increased morbidity.[34] Integrated, early treatment of patients' substance use disorder is, therefore, a critical component of SEA management.[38]

Disco-Vertebral Infections

Disco-vertebral infections (DVIs), such as spondylodiscitis and vertebral osteomyelitis, present similarly to SEA and share associated risk factors. Compressive myelopathy can be present, though many patients present in pain without neurologic deficit. Contiguous spread may lead to concomitant DVIs and SEA infections that present simultaneously.[34,39] *S aureus* is again the most common causative bacteria and MRSA a concern.[40,41] As with SEA, spondylitis and vertebral osteomyelitis may also be secondary to other bacteria such as *Pseudomonas*, particularly after spinal surgery, and spinal tuberculosis (TB; Potts Disease).[40] Potts disease should be considered in a patient with back pain and known or suspected TB. Urgent MRI with contrast is the imaging modality of choice in DVIs (**Fig. 2**D).[18] If MRI is not available, or there is a contraindication to MRI, an urgent computed tomography (CT) myelogram should be considered. CT with IV contrast is not sensitive for DVIs or SEA and should never be used in isolation. However, CT may inform clinical management when MRI or CT myelogram is not immediately available and can concurrently evaluate for an associated psoas abscess. In hemodynamically stable patients without acute neurologic findings on examination, antibiotics can be deferred pending DVI culture data.[41] In all other contexts (hemodynamic instability, sepsis, or progressive neurologic symptoms), antibiotics should be started empirically following the same regimen as discussed earlier for SEA.[41]

Noncompressive Myelopathies

Vast in etiology, many noncompressive myelopathies have no associated back pain. This review, therefore, focuses primarily on transverse myelitis (TM).

Transverse Myelitis

TM is an inflammatory myelopathy of the spinal cord, broadly divided into 2 categories: (1) idiopathic TM, which is typically postinfectious and (2) secondary TM most commonly in the setting of autoimmune and paraneoplastic processes. TM is rare with a reported annual incidence ranging from 1 to 8.6 cases per million,[42,43] although a cohort study from the Kaiser Permanente Northern California database reported a much higher annual incidence of 31 cases per million in a diverse patient population.[44] Nearly all cases occur before the age of 40 years and 20% are aged under 18 years. TM results in sensory, motor, and, less commonly, autonomic dysfunction below the affected spinal cord level. The most common presenting symptoms are sensory change (38.7%), followed by weakness (25.1%) and back pain (22%).[45] Changes in continence and balance may also be noted but are less common.[45] Onset tends to be acute and symptom nadir ranges from 1 to 21 days.[46] A clear sensory level without cephalad progression is highly indicative of myelopathy and an urgent MRI with contrast should be obtained (**Fig. 2**A). UMN findings in TM can help to differentiate it from LMN findings in Guillain–Barre syndrome (GBS), which is the most common misdiagnosis though

less frequently associated with back pain.[45] Lumbar puncture results may also help differentiate TM (cerebrospinal fluid [CSF] pleocytosis) from GBS (elevated protein in the absence of CSF pleocytosis), although these are not universally present. Standard of care treatment of idiopathic TM is a short course of high-dose glucocorticoids and possible plasma exchange (PLEX) if motor findings are noted on examination. Cause-specific TM treatment varies by etiology; however, steroids may still be indicated in the acute phase.[46] Most patients have at least partial recovery, though functional outcomes are variable. A delayed diagnosis resulting in discharge to home after initial presentation results in a greater burden of symptoms, emphasizing the importance of appropriate diagnostic pathways in the ED.[45] If there is a high index of concern for TM or it is identified on MRI, patients should be admitted and neurology consulted.

Emergent Nonspinal Causes

Though not the focus on this review, it is important to note that aortic pathology, spontaneous RP bleeds, PE, and MI can present as acute onset thoracic or lumbar back pain. All are associated with high morbidity and mortality if untreated and should be considered on the differential for emergent etiology of back pain in patients presenting to the ED. Ruptured AAA is a classic example of this phenomena—back pain is a presenting symptom in 42% of cases and rates of misdiagnosis in this are high (30%–40%), leaving ED clinicians vulnerable to missing this fatal condition.[47,48]

Overview of Nonemergent Causes

Atraumatic back pain can stem from various nonemergent causes that can be broadly subdivided into mechanical and nonmechanical causes.

Mechanical Causes

Mechanical causes of back pain are those that refer to conditions resulting from structural issues within the spine and musculoskeletal system. Examples include compression fractures, degenerative disc disease (DDD), osteoarthritis (OA), and muscle imbalances. Nonmechanical etiologies encompass conditions that are not primarily due to musculoskeletal structures but can still produce back pain. Some of these include psychogenic factors, infections like shingles, and organ-related pain (such as pancreatitis and nephrolithiasis).

Compression Fractures

Compression fractures are very common. In the United States alone, approximately 750,000 people are diagnosed with compression fractures annually.[49] Patients usually have osteoporosis diagnosis associated with their vertebral compression fracture (VCF). VCF affects approximately 25% of postmenopausal women, and the incidence increases with age as 40% of women aged 18 years and older will experience a VCF.[50] Patients with VCF usually present with severe onset of back pain, and this may be associated with trauma but does not necessarily have to be in the history to have compression fractures in the differential diagnosis. Some crucial factors in the patient's history may include midline back pain, in the setting of little or no trauma, or increased pain with changes in position, most notably standing. These elements of the history are especially relevant when taking a history in patients who are of the proper demographic (elderly and have osteoporosis). Patients may also suffer from neurologic complications, that is, paresthesia, weakness, or even SCC if the fracture fragment is in an area that affects the spinal cord. Physical examination in a patient who is complaining of back pain should always include a detailed neurologic examination. Using the algorithm (see **Fig. 1**), imaging will need to be obtained in patients who have midline back

pain. Imaging should ideally start with plain radiography of the painful area; it is prudent to image the adjacent areas of the spine if the physical examination is not successful in localizing the pain. Due to the nature of plain radiography in elderly patients, many clinicians will prefer to use CT scan as the initial test of choice. The use of CT scan will often be necessary, as it will give increased detail including compromise of the spinal canal. This is essential in patients with osteoporosis as interpreting plain radiography in patients with decreased bone density can be exceedingly difficult. Emergent MRI evaluation is necessary if the patient has neurologic findings associated with the compression fracture. Once diagnosed, patients with VCF should have multimodal pain management including NSAIDs and acetaminophen as tolerated. Due to comorbidities, patients who are elderly and suffer from compression fractures also have contraindications to many analgesics. Opiates and adjuvant medications like topical analgesics and gabapentin may also be used to alleviate pain. Emergent neurosurgical or orthopedic spine consultations are necessary if the patient has any neurologic findings. Depending on the severity of the pain and the patient's functional status, the patient may require admission to the hospital.

Degenerative Arthropathies

OA of the spine, spinal stenosis, and DDD are a spectrum of degenerative diseases that can present with back pain. Advancing age and obesity are significant factors in the development of these entities. Patients with autoimmune and rheumatologic diseases, like ankylosing spondylitis or rheumatoid arthritis, can also have advanced arthropathies. Patients with spinal arthritis and DDD often present similarly, so they will be covered together in this section. OA of the spine usually causes pain due to its effects on the facet joints, while the most advanced presentation of OA is spinal stenosis. DDD, as can be inferred from the name, is due to the degeneration of the intervertebral discs and their loss of compliance leading to stiffness and pain. Both OA and DDD entities are classified by gradual onset of back pain. Symptoms are usually worse with prolonged inactivity and in the morning. Pain can be relieved by movement and light exercise. In spinal arthritis, one can have radicular symptoms if there is foraminal encroachment that causes nerve compression. DDD usually does not have radicular symptoms unless there is a disc herniation causing nerve compression. Once again, history and physical examination are crucial in determining the ED management of these patients, as imaging and further testing are predicated on them. In these disease processes, since the pain is usually gradual onset, imaging in the ED is not called for unless the patient has "red flags" that would indicate the need for advanced imaging. If the patient lacks high prevalence risk factors, has had prolonged pain and no previous imaging, plain radiography should suffice. CT scans and MRIs are unlikely to be helpful in these situations; however, they will be useful for long-term management, and specialists will often recommend them if consulted. Treatment in the ED should include multimodal pain management. NSAIDs Should be considered the first line of treatment of patients with these issues if they are tolerated. Medications such as acetaminophen and muscle relaxants can be added as needed. There is, however, no good data to support the use of muscle relaxants—and a risk–benefit analysis for each patient must be considered. In the ED, opiates should not be used as they have shown no benefit in the management of these patients. One mainstay of treatment of degenerative arthropathies is physical therapy. Patients with these conditions often benefit from rehabilitation methods. If the patient can comply with physical therapy, there are tremendous benefits, in both the acute recovery and long-term recurrence of these arthropathies. One of the goals of ED management should be to set expectations for the patient that there is no rapid treatment option that will reverse years of degenerative changes.

Muscular Imbalances

Quite often in the evaluation of the patient presenting with lumbago, a skeletal etiology leads to muscular imbalances. As the patient develops pain, either acute or chronic, they can develop a muscular imbalance as a protective mechanism to compensate for the pain. In the acute setting, one may elicit muscular spasms or tenderness on examination. When one suspects muscular imbalance as a cause of back pain, they should make sure to expand the history to account for lower extremity issues such as knee or foot and ankle pain. The paraspinal muscles are responsible for spinal stability, if the patient is compensating for a lower extremity injury or pain, they may be using paraspinal muscles differently than they are accustomed to, causing back pain. Once again, the mainstay of treatment should be focused on correcting the underlying etiology that is causing the muscular imbalance, including evaluation of arthritis of knee or possibly podiatry for foot and ankle issues. For the patient to tolerate these corrections, however, analgesics will often be necessary. NSAIDs and muscle relaxants should be first-line treatments. As with other etiologies, opioid medications have not been shown to improve outcomes in patients with muscular back pain.

Nonmechanical Causes

In the evaluation of a patient with back pain, the differential diagnosis should always include nonmechanical causes of the pain. While the list can be quite extensive, and emergent causes have already been discussed earlier, there are a few conditions that warrant discussion.

Herpes Zoster (Shingles)

The physical manifestation of the reactivation of varicella-zoster infection can be extremely painful. While usually associated with a vesicular rash in a dermatomal pattern, pain can precede and linger after the rash, commonly referred to as postherpetic neuralgia. Considered to be one of the more painful disease entities, postherpetic neuralgia can be exceedingly difficult to diagnose, especially when the pathognomonic rash is not present. Characteristically seen in older and immunocompromised individuals, pain from zoster is difficult to manage. Treatment with oral antiviral medications (valacyclovir 1000 mg 3 times a day) in the acute setting, within 72 hours of rash appearance, is the standard of care. Oral steroids can be added to help with inflammation and pain. This is often prescribed at a prednisone taper starting at 40 mg for days 1 to 6 and ending at 10 mg for days 10 to 14. If the patient does have postherpetic neuralgia, treatment with tricyclic antidepressants for example, amitriptyline, gabapentin, and topical lidocaine patches are all acceptable choices. In the case of zoster and postherpetic neuralgia, the best treatment is prevention with vaccination, as this has been proven to decrease incidence.

Renal Diseases

When evaluating back pain, one cannot overlook the possibility of internal organs as the etiology of the pain. Flank, lower thoracic, and lumbar back pain can be due to renal disease processes. As above with aortic aneurysm, renal pathology can be the underlying cause of the pain. The history and presentation of the pain can give some direction to help stratify the differential diagnosis. Renal colic due to nephrolithiasis tends to be acute in onset, quite severe and can be described as migratory with or without hematuria or dysuria. Pyelonephritis can be accompanied by infectious symptoms and description of dysuria.

After a thorough history and physical examination, patients presenting with acute onset of back and flank pain that does not appear to be musculoskeletal will often require a diagnostic evaluation with laboratory test and radiology examination. Point of care ultrasound examination can be useful in evaluating for hydronephrosis. If the pain is from nephrolithiasis or infection, urinalysis may show hematuria or bacteriuria. Although the absence of these does not fully rule out renal disease processes. CT imaging will often be necessary to make a final diagnosis and help with final management. Parenteral pain control and antibiotics may be warranted based on the patient's diagnosis, clinical status, and ability to tolerate oral medications.

Pancreatitis

Much like the kidneys, the pancreas is found in the retro-peritoneal space, albeit partially. Thus, pancreatic pathology can present as thoracic or upper lumbar back pain. While pancreatic pathology is outside of the scope of a neurologic article, clinicians should keep this on the differential diagnosis if other causes of back pain are ruled out.

Somatization

Chronic back pain can be a demoralizing affliction, as it can limit quality of life and interfere with activities of daily living. As such, there are several scoring systems that attempt to quantify the measure of disability from back pain including the Oswestry Disability Index and the Back Pain Functional scale.[51] As stated previously, two-thirds of adults experience back pain at some point, and many of these will not have any diagnostic or radiographic cause identified. In these cases, somatization and psychological stress can contribute to the patient's pain. The emergency medicine clinician should, however, not default to these as causative, unless all other etiologies—especially emergent causes—have been evaluated.

SUMMARY

This is a review article that provides a structured approach to evaluating and managing patients presenting with atraumatic back pain or lumbago, emphasizing the importance of identifying serious emergencies. While common and usually benign, back pain does have some very serious disease entities that are associated with significant morbidity and mortality. The history and physical examination are crucial for risk stratification, with a primary focus on red flags such as neurologic deficits, cancer history, IDU, and systemic symptoms. Imaging is often necessary and should be guided by the clinical scenario, this often requires advanced imaging including CT, or MRI instead of plain radiography. Emergent causes of back pain often require the assistance of a specialist based on availability, for example, neurosurgery or spinal orthopedics.

CLINICS CARE POINTS

- Consider emergent causes of acute back pain in your differential to avoid diagnostic bias.
- Augment clinical decision making with a detailed history and exam in patients who present with acute back pain.
- Consider Imaging the entire spine in patients who present with urinary incontinence or retention.
- The term "red flags" is non-specific and should be utilized with caution in documentation.

DISCLOSURE

None.

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