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Spinal Emergencies in Primary Care Practice

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ABSTRACT

Atraumatic spinal emergencies often present a diagnostic and management dilemma for health care practitioners. Spinal epidural abscess, cauda equina syndrome, and spinal epidural hematoma are conditions that can insidiously present to outpatient medical offices, urgent care centers, and emergency departments. Unless a high level of clinical suspicion is maintained, these clinical entities may be initially misdiagnosed and mismanaged. Permanent neurologic sequela and even death can result if delays in appropriate treatment occur. A focused, critical review of 34 peer-reviewed articles was performed to identify current data about accurate diagnosis of spinal emergencies. This review highlights the key features of these 3 pathological entities with an emphasis on appropriate diagnostic strategy to intervene efficiently and minimize morbidity.

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KEYWORDS: Cauda equina syndrome; Spinal emergencies; Spinal epidural abscess; Spinal epidural hematoma

INTRODUCTION

Spinal emergencies are conditions that can cause permanent neurologic injury if not diagnosed and treated in a timely manner. Exclusive of trauma and neoplasm, the most common spinal emergencies encountered in the outpatient and emergency department settings are spinal epidural abscess, cauda equina syndrome, and spontaneous spinal epidural hematoma. These conditions may initially manifest nonspecific symptoms and often present a diagnostic challenge to first-line providers.

Atraumatic spinal emergencies are rarely evaluated first by a spine specialist. Rather, these 3 conditions most often present in the outpatient setting to primary care providers or to urgent care/emergency department practitioners. Recognizing the potential for a spinal emergency and ordering of magnetic resonance imaging (MRI) provides the most effective means of detecting the presence and cause of neural element compression.

This focused literature review of 34 peer-reviewed studies was performed to provide additional clarity about the diagnosis and management of spinal epidural abscess, cauda equina syndrome, and spontaneous spinal epidural hematoma. Pertinent studies about the diagnosis and management of these spinal emergencies were included for review.

This study aims to improve the outcomes of atraumatic spinal emergencies by providing an improved understanding of patient risk factors and presenting symptoms, along with the need for efficient diagnosis and urgent management of these 3 conditions.

CLINICAL PRESENTATION OF NEUROLOGICAL COMPRESSION

Neural compression in the central spinal canal at the spinal cord level (Occiput to L1) can lead to upper motor neuron dysfunction. Compression of the nerve roots in the foramina at any level or within the central canal caudal to L1 typically leads to lower motor neuron dysfunction.

Neural element compression at the cervical level can result in axial pain, neck stiffness, and a variety of neurologic syndromes inclusive of monoradiculopathy, myelopathy, and myeloradiculopathy. Cervical monoradiculopathy is caused by isolated dysfunction of a single nerve root.

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This clinical syndrome produces a distribution of neurologic findings characterized by sensory alteration in a single dermatome or weakness of muscles innervated by a specific nerve root.

Cervical myelopathy is due to compression of the spinal cord within the central canal. This syndrome is typified by bilateral sensory findings (paresthesias, numbness, and altered proprioception) and motor changes (weakness, increased muscle tone) in the upper, and possibly, all 4 extremities. Other potential findings include altered hand dexterity, gait and balance difficulty, and rarely, urinary difficulties. Upper motor neuron signs on physical examination (eg, spasticity, hyperreflexia, Hoffman's sign, inverted radial reflex, clonus, or an extensor plantar response) correlate with cervical cord dysfunction but are not always present in the setting of acute cord compression. Myelopathy can occur in conjunction with dysfunction of one or more nerve roots to produce cervical myeloradiculopathy.

CLINICAL SIGNIFICANCE

- Time to decompression is an important factor correlated to outcomes, emphasizing early diagnosis.
- Irreversible neurologic sequelae can occur if there is initial misdiagnosis or delay in diagnosis.
- Managing bleeding dyscrasias can be an important first step in the treatment of epidural hematoma.
- Bowel/bladder symptoms and perianal numbness can differentiate sciatica from cauda equina syndrome.

In the thoracic region, compression of a single nerve root can produce torso radicular symptoms. While uncommon, thoracic radiculopathy may produce truncal paresthesias/dysesthesias in a dermatomal distribution or chest pain that can often mislead practitioners. Spinal cord compression at the thoracic level can produce thoracic myelopathy.

The spinal cord most often terminates between the first and second lumbar vertebrae. Compression of single or multiple nerve roots at the lumbar level can produce low back pain, radiculopathy in the lower limbs (often referred to as sciatica) or neurogenic claudication. Lumbar radiculopathy is typically produced by nerve compression in the foramina or lateral aspect of the spinal canal by disc herniation or spondylotic changes. Particularly in the setting of disc herniation, radicular symptoms can be provoked on examination by the straight leg raise maneuver. Longstanding central canal stenosis beyond the termination of the spinal cord (ie, caudal to the L1-2 level) is most commonly produced by spondylosis, and typically manifests as neurogenic claudication. When the caudal nerve roots are subjected to acute compression in the central spinal canal (eg, a large central disc herniation), cauda equina syndrome may develop.

SPINAL EPIDURAL ABSCESSES

Spinal epidural abscess was first described by Italian anatomist, Giovanni Battista Morgagni in 1761, as a pyogenic infection of the space between the vertebral body/ligamentous complex and dura mater of the spine.[1] Once perceived to be an uncommon disease process with an incidence of 1:20,000 hospital admissions 20 years ago,[2] spinal epidural abscess is becoming more prevalent. In the 21st century, spinal epidural abscess still often poses a diagnostic dilemma for treating physicians. Initial misdiagnosis rates were historically as high as 75%-89% and can result in permanent neurologic dysfunction, paralysis, and even death.[3,4]

Patients with spinal epidural abscess most commonly present with axial pain, which is present in over two-thirds of patients.[5,6] The "classic triad" of spinal epidural abscess consisting of fever, axial pain, and neurologic deficit is seen in only 8% of patients, and therefore should no longer be considered the classic presentation of an epidural abscess.[5] Patients may present with radiculopathy or myelopathy, depending on the location of the abscess along the spinal axis and within the spinal canal; bacteremia or sepsis may also be evident.

To improve the sensitivity of diagnosing epidural abscesses, it is important to consider patient comorbidities and other predisposing factors. The most common presenting comorbidities for spinal epidural abscess include: previous focus of infection (22%-54%), diabetes (22%-51%), intravenous (IV) drug abuse (29%-39%), and renal disease (7%-37%).[5,6] However, many patients who develop epidural abscesses have no risk factors, prompting physicians to maintain a high level of suspicion.

Further assistance in diagnosis can be provided by the elevation of nonspecific inflammatory markers. Complete blood counts will show a leukocytosis in up to 66% of patients,[2,4] while erythrocyte sedimentation rate and C-reactive protein (CRP) levels are elevated in nearly all cases.[2,4,7-10] Positive blood cultures can be found in 63.3%-70%[4,5] of cases of spinal epidural abscess, and if positive, correspond to the offending organism nearly 100% of the time (most often, *Staphylococcus aureus*).[2,4]

MRI with IV gadolinium or computed tomography (CT) with myelography are the 2 imaging modalities available to evaluate epidural abscesses and should be obtained of the entire spinal axis (cervical, thoracic, and lumbosacral) due to the possibility of infectious foci at multiple spinal locations. Both methods of advanced imaging produce similar diagnostic sensitivity of 90% and above.[2,7] MRI is the diagnostic modality of choice as it offers the benefit of being less invasive and provides improved soft tissue detail. Some patients require CT myelogram due to a contraindication to MRI (eg, pacemaker or vascular clips). Plain radiographs can show evidence of spinal column infection such as osteomyelitis or sagittal alignment changes. However,

x-ray study findings can take weeks to manifest and do not replace MRI or CT myelography in the diagnostic process.

Management of spinal epidural abscesses is based upon multiple factors, including the patient's medical history, the infectious organism, the location and size of the abscess, and most importantly, the patient's neurological examination. Urgent referral to spine and infectious disease specialists is always warranted. Decompressive and debridement surgery followed by long-term antibiotics is the treatment of choice and has been shown to significantly improve outcomes in patients with neurologic compromise.[5]

Patel et al[6] performed a retrospective review identifying risk factors for failure of nonoperative management of epidural abscess after 41% of their medically managed patients failed treatment, ultimately requiring surgery. The authors found that important risk factors for failure of medical management included diabetes (odds ratio [OR] 3.8; $P = .057$), CRP > 115 (OR 4.7; $P = .045$), white blood cell count > 12.5 (OR 3.3; $P = .045$), positive blood cultures (OR 3.5; $P = .035$); the presence of multiple risk factors produced an additive effect on failure rate.[6]

For patients suffering from spinal epidural abscesses, the greatest predictor of overall outcome, regardless of management, is the initial neurologic presentation at the time of definitive diagnosis.[2,5,6,11] The rapidity of identifying the diagnosis, involving appropriate consultants, and initiation of treatment is paramount to optimizing patient outcomes.[12]

Case 1

A 51-year-old man presented with 2 weeks of atraumatic back pain, bilateral lower extremity weakness and paresthesias, gait instability, and urinary retention. He denied fevers/chills, history of invasive spinal procedure, diabetes, or IV drug use

Physical examination: afebrile; palpation tenderness in the cervicothoracic spine; bilateral lower extremity weakness (grade 3/5); altered pain sensation in both lower extremities; normal rectal tone and sensation; normoreflexia.

Labs: white blood cell count 8.7; erythrocyte sedimentation rate 42; CRP 35.38; blood cultures: negative.

MRI: the entire spine with gadolinium contrast (Figure 1); large, contrast-enhancing C5-T6 ventral and dorsal collections with multifocal cord compression and signal change.

Management: Within 10 hours of presentation, this patient underwent emergent posterior decompression of his cervicothoracic spine, with intraoperative cultures positive for *Propionibacterium acnes*. Intravenous antibiotic therapy was administered for 8 weeks.

Initial response to treatment: During the index hospital admission, lower extremity sensation improved, as did the motor examination, with the left lower extremity strength graded 5/5 and the right 3+/5. This patient failed a voiding trial and was discharged with a Foley catheter.

CAUDA EQUINA SYNDROME

The spinal cord terminates in adults between the lumbar 1 and lumbar 2 vertebrae; caudal to this level, individual nerve roots extend distally and give the appearance of a horse's tail or, in Latin, "*cauda equina*."[13] Severe compression of the neural elements caudal to the distal extent of the spinal cord can produce a constellation of motor, sensory, and visceral symptoms known as cauda equina syndrome.

Compression of the caudal nerve roots in the central spinal canal can occur secondary to multiple etiologies including: intervertebral disc herniation, traumatic fracture of the lumbosacral vertebrae, epidural hematoma, neoplasm, epidural abscess, and progressive chronic lumbar stenosis. Acute or sub-acute intervertebral disc herniation is by far the most common cause of cauda equina syndrome. Note,

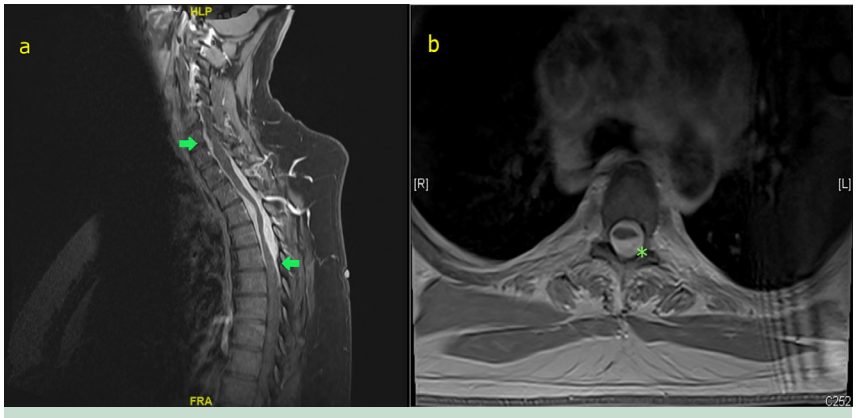

Figure 1 Contrast-enhanced T1-MRI showing a large cervicothoracic spinal epidural abscess. (**A**) Mid-sagittal section; (**B**) axial section at upper thoracic level. Arrows indicate the craniocaudal extent of the abscess spanning from C6 to T5. Asterisk showing abscess collected in the posterior epidural space.

however, that cauda equina syndrome occurs in only approximately 2% of acute disc herniations.[13]

Difficulty in accurately diagnosing cauda equina syndrome is, in large part, due to the similarity in presentation to disc herniation with radiculopathy. In fact, patients may even develop pain-related or narcotic-related urinary retention in some cases of acute disc herniation in the absence of neurologic bladder dysfunction.[14]

The 2 most common presenting symptoms of cauda equina syndrome include severe lower back pain (83%-100% of cases) and unilateral or bilateral sciatica (90%-100% of cases).[13,15,16] Up to 71% of patients with cauda equina syndrome have a previous history of back pain/sciatica.[13] Additional "red-flag" symptoms of cauda equina syndrome include motor or sensory deficits in one or both legs, groin or perirectal sensation loss (saddle anesthesia), and bowel/bladder/sexual dysfunction. Identification of saddle anesthesia, reduced rectal tone, or the presence of urinary retention (with overflow incontinence) is key in distinguishing isolated radiculopathy from cauda equina syndrome. Measuring postvoid residual of >100-200 mL by bladder scanning or placement of a Foley catheter can help diagnose cauda equina syndrome, and also will serve to decompress the bladder.[13,17,18] Clinical suspicion of cauda equina syndrome should prompt emergent MRI and spine surgical consultation.

Clinical diagnosis is preferably confirmed with an emergent MRI (CT myelography can be performed if MRI is contraindicated). Intravenous gadolinium contrast should be utilized when there is suspicion for an infectious or oncologic etiology.

Neurologic status at presentation and at the time of decompression are the factors most closely tied to outcomes, emphasizing the need for expeditious diagnosis and early surgical consultation. If appropriately managed in a timely fashion, the long-term prognosis after decompression of cauda equina syndrome can be favorable, although permanent bowel, bladder, and sexual dysfunction is common, especially in cauda equina syndrome complete with urinary retention.[14]

Case 2

A 33-year-old woman presented with 5 days of worsening, atraumatic, acute-on-chronic back pain and right lower extremity pain with bilateral lower extremity numbness and weakness. She had no bowel/bladder incontinence or urinary retention.

Physical examination: Lumbar spine tenderness to palpation. **Neurologic:** bilateral weakness in ankle/toe dorsiflexion and plantarflexion (2/5 grade); absent sensation bilaterally in the L4-S1 distribution; normal sensation in S2-5 dermatomes; rectal tone normal.

T2-weighted MRI showed a canal-obliterating disc extrusion at the L4-L5 level (Figures 2 and 3).

Within 12 hours of presentation, this patient was urgently taken to the operating room for a laminectomy and discectomy. A 1.5-cm large, extruded disc was removed from the intervertebral space (Figure 4). Postoperatively, there was return of 4/5 strength to bilateral anterior tibialis and gastrocnemius and soleus muscles (L4 and S1), but extensor hallucis longus (L5) function was 0/5 bilaterally. She was able to urinate spontaneously without retention and was discharged home.

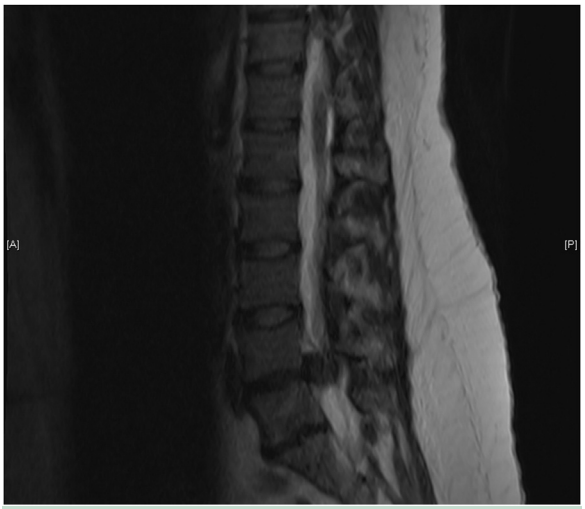

Figure 2 T2-weighted MRI (mid-sagittal section) showing a large L4-L5 disc extrusion causing severe compression of the cauda equina in the central spinal canal.

SPINAL EPIDURAL HEMATOMA

Spinal epidural hematoma is another neural element of compressive disease with the potential for a catastrophic outcome. Spinal epidural hematomas can be the result of traumatic events including spinal fractures and ligamentous disruptions. Spinal epidural hematoma may also be iatrogenically induced by spinal surgical procedures, lumbar puncture, or invasive epidural/facet joint analgesic administration, or may be spontaneous with no known cause.

Previous studies are inconsistent in their description of spontaneous spinal epidural hematoma, with some authors including patients with coagulopathies and those on anticoagulant therapy, while others include only patients without a history of invasive procedures or coagulopathic risk factors (idiopathic).[19–26] Spontaneous spinal epidural hematoma can also, rarely, be the result of rupture of arteriovenous malformations.[25–27]

The incidence of spinal epidural hematoma ranges from 1:150,000-1:220,000 after epidural or spinal block or in 0.1:100,000 individuals without an ascertainable cause (spontaneous spinal epidural hematoma).[25,28,29] The majority of spinal epidural hematoma and spontaneous spinal epidural hematoma have been found to occur in the cervicothoracic region, with 53%-80% located in the posterior epidural space.[21,26,27]

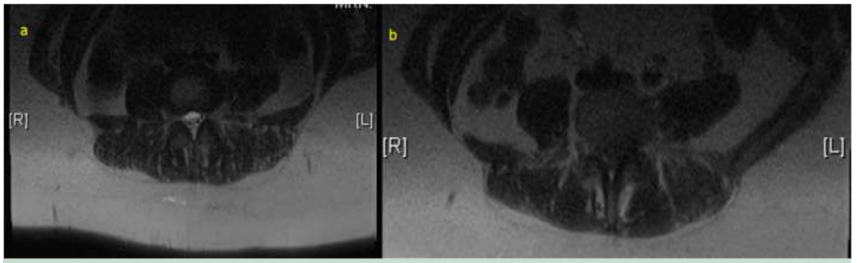

Figure 3 T2-weighted MRI (axial sections). (**A**) At one level above disc extrusion there is no canal compromise. (**B**) At level of disc extrusion there is near complete canal occlusion.

The most common initial presenting symptom is axial spine pain. Depending on the location of the compressive hematoma and time from onset to presentation, patients may also complain of radiculopathy or dermatomal referred pain, varying distributions of paresis or paralysis, myelopathy, and bowel/bladder dysfunction.[25,28] The progression of neurologic dysfunction, however, often follows a variable rate and severity.[27]

Spinal epidural hematoma can be difficult to distinguish from other compressive spinal pathologies. Therefore, suspicion should be high anytime a patient has severe spinal pain and a history of invasive spinal procedure, perispinal injections or epidural catheter placement, coagulopathy, or ongoing use of blood-thinning medications. Wysowski et al[30] reported on 41 patients who developed spinal epidural hematoma while being given prophylaxis for deep venous thrombosis with enoxaparin, a low-molecular-weight heparin medication, over a 5-year period. Thirty-nine of the 41 patients had recent history of preoperative epidural analgesia or spinal fracture.[30] When suggestive risk factors and symptoms are present, emergent advanced imaging and spine surgical consultation are necessary. The risk of failure to identify or appropriately manage spinal epidural hematoma can lead to paralysis.

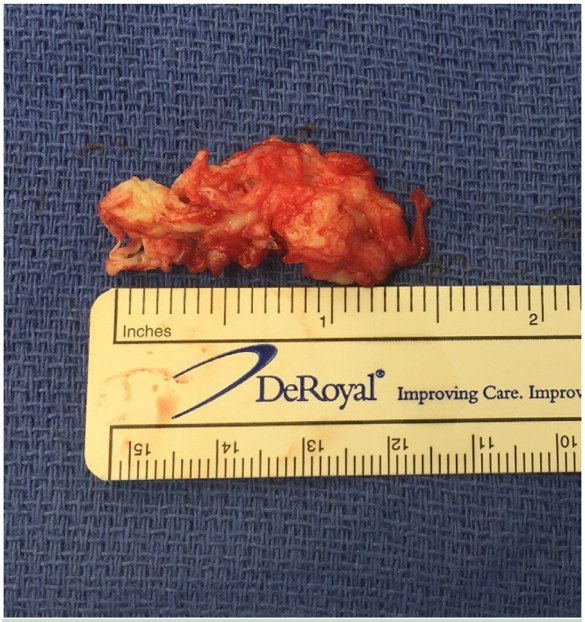

Figure 4 Large extruded portion of intervertebral disc.

Much like epidural abscesses and cauda equina syndrome, MRI is the gold standard for evaluation of spinal epidural hematoma.

Initial medical management should include removal of any offending anticoagulant agents, whether in the form of prescription medications or homeopathic remedies such as fish oil or garlic. Correction or reversal of any bleeding dyscrasias with appropriate factors, blood product, or reversal agents is essential in minimizing the patient's continued bleeding risk.

Surgical decompression, most often in the form of a laminectomy, is almost always recommended for patients with spinal epidural hematoma.[23,24,27,28,31-33] Mukerji and Todd's[34] meta-analysis demonstrated that surgical decompression within 12 hours provided the best chance for complete recovery even in patients with complete or near-complete paralysis. Medical management and close neurologic monitoring can, however, provide acceptable outcomes in the rare instances where an early trajectory of rapid neurologic recovery has already been observed and immediate spine surgical care is available 24 hours per day in case the patient deteriorates neurologically.

Case 3

A 71-year-old man presented to the emergency department with progressive bilateral lower extremity weakness and truncal paresthesias. Twelve hours earlier, the patient underwent a cervical epidural steroid injection and had not discontinued taking his daily aspirin. The patient went home after the injection and later developed thoracic numbness/paresthesias, which spread to his lower extremities.

Physical examination: Tenderness to palpation in his cervicothoracic spine. **Neurologic:** 0/5 strength and decreased sensation in his bilateral lower extremities. He also had decreased sensation in his trunk. Upper limb strength was 2/5 in his left hand; intrinsic muscle weakness was evident at 2/5; otherwise, sensorimotor function was

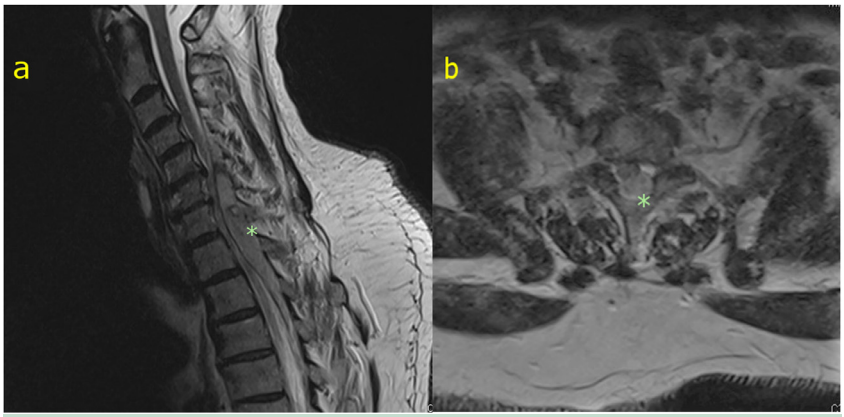

Figure 5 T2-weighted MRI of the cervical spine. (**A**) Mid-sagittal section. Note the large, dorsal spinal epidural hematoma extending from C6-T4. (**B**) Axial section. There is severe central canal narrowing as a result of spinal epidural hematoma. The hematoma (*) is situated in the posterior epidural space with dorsal compression and ventral displacement of the spinal cord.

normal in the upper extremities. The patient had a positive Babinski sign, decreased perianal sensation, and absent rectal tone. He had no pathologic clonus but was hyperreflexic in his lower extremity reflexes.

T2-weighted MRI demonstrated a large, complex dorsal epidural fluid collection extending from C6-T4, producing mass effect on the spinal cord. Disc bulges are also present, most significantly at C5-C6 and C6-C7, contributing to canal stenosis (Figure 5).

This patient was taken to the operating room within 6 hours of presentation for a posterior laminectomy and evacuation of the hematoma. He was discharged 8 days later with 5/5 strength and normal sensation in all extremities. Intermittent self-catheterization for urinary dysfunction was necessary on a long-term basis.

CONCLUSION

Spinal emergencies have historically presented as a diagnostic challenge for medical practitioners. Delays in diagnosis and management of spinal emergencies can produce life-altering consequences for patients and practitioners alike. Extensive literature has been published to identify the correlation between timely decompression of the neural elements and patient outcomes.

Being equipped with an understanding of the risk factors and presentation of patients with spinal epidural abscess, cauda equina syndrome, and spinal epidural hematoma empowers practitioners to confidently and appropriately manage these conditions in a timely and outcome-optimizing manner.

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