Spinal cord compression

The right clinical information, right where it's needed
Table of Contents

Summary 3
Basics 4
  Definition 4
  Epidemiology 4
  Aetiology 5
  Pathophysiology 6
  Classification 6
Prevention 8
  Primary prevention 8
Diagnosis 9
  Case history 9
  Step-by-step diagnostic approach 9
  Risk factors 14
  History & examination factors 15
  Diagnostic tests 18
  Differential diagnosis 20
  Diagnostic criteria 23
Treatment 25
  Step-by-step treatment approach 25
  Treatment details overview 28
  Treatment options 30
  Emerging 41
Follow up 42
  Recommendations 42
  Complications 42
  Prognosis 44
Guidelines 45
  Diagnostic guidelines 45
  Treatment guidelines 45
Evidence scores 46
References 47
Images 56
Disclaimer 59
Can occur as a result of spine trauma, vertebral compression fracture, intervertebral disc herniation, primary or metastatic spinal tumour, or infection.

The resulting spinal cord injury may be acute, sub-acute, or chronic and occurs due to direct cord damage, by compression and/or infiltration, or by compromise of the vascular supply to the cord.

Diagnosis is made by x-ray or MRI of the spine, but spinal cord injury may occur with no findings on imaging.

Acute spinal cord compression (SCC) is a medical emergency that requires swift diagnosis and treatment to prevent irreversible spinal cord injury and long-term disability.

Treatment of acute SCC may include corticosteroids, surgery, or radiotherapy.
**Definition**

Spinal cord compression (SCC) results from processes that compress or displace arterial, venous, and cerebrospinal fluid spaces, as well as the cord itself. This can occur as a result of extrinsic causes and lesions, or intrinsic aetiologies of the cord substance. Examples include trauma or tumour affecting the cord substance, and lesions that compromise cord function emanating from surrounding elements or vascular sources. The presenting symptoms are a result of spinal cord injury (SCI) or root dysfunction and include paresis, sensory changes or loss of sensation, sphincter dysfunction (urinary or anal), and erectile problems. Diagnosis is made by x-ray, CT or MRI of the spine, but SCI due to SCC can occur with no findings on imaging, a situation termed SCI without roentgen abnormality (SCIWORA).

**Epidemiology**

Spinal cord compression (SCC) is a devastating condition affecting people across all age groups worldwide. Despite this, little international data exist regarding incidence and prevalence; therefore, most of the information included stems from US-based research. Where possible, international statistics have been mentioned.

Trauma is the main cause of acute SCC. In the US there are approximately 11,000 traumatic spinal cord injuries annually. The global prevalence of spinal cord injury (SCI) has been reported to vary from 236 to 1298 per million inhabitants. In the US, the case rate remains an estimate at 40/1,000,000. The worldwide incidence of SCI varies from 8 to 246 cases per million inhabitants per year.[2] Over half (53%) of those sustaining SCI are aged 16 to 30 years.[3] Most studies suggest an increasing incidence over the last 10 years.

Overall, the leading 5 causes of acute SCI in people ≤65 years of age are as follows, with the highest listed first:[3]

- Males: automobile accidents, falls, gunshot wounds, diving accidents, motorcycle accidents
- Females: automobile accidents, falls, gunshot wounds, medical/surgical complications, diving accidents.

More than 80% of all reported SCI and 90% of all sports-related SCI affect males. Sports- and recreation-related SCI primarily affect people under 29 years of age.[3] [4] [5]

In people aged 65 years of age or older, falls are the leading cause of spinal cord injuries. Fractures in this age group are most commonly commonly associated with osteoporosis.[6] It has been estimated that there are approximately 550,000 osteoporosis-related vertebral fractures in the US each year. These can occur spontaneously or with relatively minor trauma. According to the National Osteoporosis Foundation, low bone mass affects over 44 million Americans, and frank osteoporosis affects 15% of postmenopausal white women and 35% of all women >65 years of age. It is estimated that 50% of white women will sustain an osteoporotic fracture at some time during their lifetime. Osteoporotic fractures affect women 6 times more frequently than men. Men have a lower, but still significant, osteoporotic fracture risk which peaks 10 years later than in women.[6] [7]

Intervertebral disk disease commonly causes sciatica, which affects approximately 2% of all people in the US, usually between 30 and 50 years of age. Eighty percent of all people will experience disk-related back pain in their lives; 90% will improve without surgery.[4] [5]
Each year in the US, approximately 20,000 people with cancer develop spinal cord compression. This group represents 5% to 10% of the general cancer population.[8] [9] [10] Eighty-five percent of all spinal cord tumours are metastatic, with the remainder comprising primary central nervous system tumours (e.g., meningeal, glial, ependymal). The international incidence rate parallels that of the US.[9] [10]

Spinal epidural abscess is found in 2.8 cases per 10,000 hospital admissions. The incidence is thought to be increasing in relation to intravenous drug abuse.[11] The most commonly reported organism in epidural abscess is *Staphylococcus aureus*, although many other bacteria have been implicated, including *Streptococcus* and *Pseudomonas* species, *Escherichia coli*, and *Mycobacterium tuberculosis*. MRSA is increasingly reported, particularly in patients with spinal surgery or implanted devices.

**Aetiology**

Spinal cord compression (SCC) can occur as a result of spine trauma, vertebral fracture, intervertebral disc herniation, primary or metastatic spinal tumour, or infection.

Trauma is a leading cause of acute SCC and is commonly due to:

- Car accidents
- Falls
- Gunshot wounds
- Sports injuries
- Diving
- Motorcycle accidents
- Knife wounds
- Iatrogenic causes such as surgery and injection procedures.

Vertebral compression fractures are usually due to low-energy trauma in weakened bone and are seen in older people and in patients with:

- Osteoporosis
- Corticosteroid therapy
- Osteomalacia
- Osteomyelitis
- Tumour infiltration causing pathological fractures
- Spinal subluxation.

**Intervertebral disc disease**

- Intervertebral disc herniation describes the rupture of the nucleus pulposus in the intervertebral space, through the fibres of the annulus fibrosis. The herniation may cause 1 or more fragments of the nucleus pulposus to compress or irritate the adjacent nerve roots.
- The symptoms of paraesthesia, pain, and weakness are indicative of lumbar radiculopathy, or sciatica.
- MRI shows evidence of disc protrusion or extrusion, nerve root impingement, or thecal sac compression.

Tumours causing SCC include:

- Primary sarcoma
- Central nervous system tumours (ependymoma, meningioma, glioma)
Spinal cord compression

Basics

- Multiple myeloma
- Metastatic small cell lung cancer
- Metastatic non-small cell lung cancer
- Metastatic breast cancer
- Metastatic prostate cancer
- Metastatic renal cancer.

Infection may cause cord compression by external pressure or by direct involvement of the cord, and may be due to:

- Discitis
- Tuberculosis (Pott’s disease of the spine)
- Epidural abscess.

Pathophysiology

The spinal cord extends from the foramen magnum down to the level of the first and second lumbar vertebrae (at birth, down to the second and third lumbar vertebrae). The conus medullaris is the cone-shaped termination of the caudal cord. The pia mater continues caudally as the filum terminale through the dural sac, and attaches to the coccyx. The cord is protected by a bony skeleton, the vertebral column, which is partially mobile and allows for movement of the spine. The cord is wrapped by the dura mater, as is the brain, and the vessels supplying and draining the cord are organised within that space. The vessels enter at various levels from the main vascular system. The cord floats, as in an aquarium, in the cerebrospinal fluid space. This acts as a buffer to movement and early degrees of compression. The cord substance contains a grey area centrally and is surrounded by white-matter communication tracts, both ascending and descending.

Acute spinal cord compression (SCC) can result from trauma, disc herniation, bony fracture, spinal subluxation, or penetrating injuries (e.g., gunshot wounds, knife wounds, iatrogenic causes). Chronic SCC results from degenerative bony reaction, slow tumour growth, or infection in the spaces around the thecal sac. Both acute and chronic presentations will reach a common endpoint, when the cord or nerve roots can no longer function correctly.

Injury to the spinal cord or nerve roots arises from stretching or from pressure. This results in injury to the white matter (myelinated tracts) and the grey matter (cell bodies) in the cord with loss of all or some of the sensory modalities (pinprick, joint position sense, vibration, hot/cold, pressure) and motor function.

The spinal cord and nerve roots depend on a constant blood supply for appropriate energy stores and substrate, to perform axonal signalling. Conditions that interfere, either directly or indirectly, with the blood supply will cause malfunction of the transmission pathway. Nerve tracts most vulnerable to mechanical pressure include the corticospinal and spinocerebellar tracts, and the posterior spinal columns.

Classification

American Spinal Injury Association (ASIA) sensory and motor impairment scale[1]

ASIA sub-types or syndromes
Spinal cord compression

• Complete cord transection syndrome results in a group of symptoms known as spinal shock, which when seen at the high cervical level include quadriplegia, respiratory insufficiency, loss of bladder and bowel function, anaesthesia below the affected level, and neurogenic shock (hypotension and hypothermia).

• A central cord syndrome, seen in syringomyelia, central canal ependymoma, and haemorrhage following trauma, is associated with greater loss of upper limb function compared to the lower limbs.

• Brown-Sequard's syndrome results from a hemisection lesion of the spinal cord.

• Anterior cord syndrome occurs when the injury affects the anterior spinal tracts, including the vestibulospinal tract.

• Posterior cord syndrome is rare and occurs when the injury affects the posterior spinal tracts.

• Cauda equina syndrome occurs with damage to the spinal roots of the lumbar cord of the cauda equina and is associated with pain, radicular sensory changes, leg weakness, and loss of bowel and bladder function.

Clinical classification of spinal cord injury (SCI)

Traumatic and non-traumatic causes of spinal cord compression may result in:

• Acute SCI
• Sub-acute SCI
• Chronic SCI.

All forms of SCI may result in:

• Complete neurological impairment: sensory and motor functions absent in the lowest sacral segments
• Incomplete neurological impairment: sensory or motor function preserved below the level of injury, including the lowest sacral segments.
Primary prevention

High-risk occupations for trauma and disc herniation (construction workers, agricultural workers, seamen, bricklayers, road menders, vehicle drivers, military personnel, law enforcement officers, and firefighters) will require adequate safety measures and preventative measures to be instituted by employers, including education, safe work practices, and restraint systems. These same measures apply to organised recreational activities (vehicle racing, mountaineering, diving, horse riding, and gymnastics).
Case history

Case history #1

A 25-year-old man presents to the emergency department after an automobile accident. He was ejected from the vehicle. He complains of numbness in both lower extremities and cannot move his legs. There is no pinprick sensation below the umbilicus except for an anal wink, and there is no rectal tone. The bulbocavernosus reflex is weakly present. Power in the lower extremities is graded at 1/5.

Case history #2

A 40-year-old woman presents with back pain and difficulty with her gait. She has a long history of smoking and has had some haemoptysis recently. Her examination reveals diminished pinprick sensation from the nipple line caudally, power in the lower extremities of 4/5, absent joint position sense in the lower extremities, and diminished vibratory sense. Anal sphincter tone is intact.

Other presentations

The clinical findings of pain, sensory changes, and motor loss (including sphincter dysfunction) can be seen in most forms of spinal cord compression. However, the onset of severe back or neck pain, in the historical setting of intravenous drug use, or the historical setting of chronic, low-grade back pain, should raise a suspicion of epidural compression of the cord elements by infection. Rarely, an acute presentation will exhibit unilateral spastic paralysis on the same side of the body, as well as ipsilateral loss of vibration and proprioception (position sense), with pain and temperature sensation being lost from the contralateral side beginning 1 or 2 segments below the lesion. This is known as Brown-Sequard's syndrome and can result from a lateral cord compression (from tumour or disc herniation). An additional group of patients present after a fall occurring while the neck is extended. The result is intact motor and sensory function in the legs, and absent motor and/or sensory function in the arms. This is known as central cord syndrome.

Step-by-step diagnostic approach

The diagnosis of spinal cord compression (SCC) is based on the clinical history and radiographic findings. Other serious causes of loss of sensory, motor, or autonomic function should be excluded when making the diagnosis.

History

Acute spinal cord injury (SCI) should be suspected in any patient following trauma, particularly trauma to the head or neck and when the patient is unresponsive due to hypotension and has respiratory compromise. Acute SCI is a medical emergency. Patients should be managed at a trauma centre with spinal cord experience and in-house neurosurgical facilities. While history and examination are started, the patient should be immobilised with a cervical collar and backboard/head strap.

A history of the precise circumstances of the accident, timing, initial clinical examination data, treatment received, and presence of combined initial injuries should be taken from witnesses, relatives, and attending police, paramedic, or medical staff. In patients with multiple injuries following trauma and who
may be confused or have loss of short-term memory, SCI must be considered until all investigations have been completed.

Patients who are able to give a history and who present with sub-acute or chronic cord compression or with severe localised back pain are questioned about risk factors such as recent or chronic trauma, occupation, recreational activities, underlying degenerative vertebral disease, congenital spinal abnormalities, osteoporosis, intravenous drug use, medicines, recent surgery, and immunosuppression.

Incomplete spinal cord syndromes may be associated with the following acute, sub-acute, or chronic symptoms:

Sensory symptoms

- Altered sensation below a certain level (e.g., pin, touch, vibration, temperature)
- Hemisensory loss.

Motor symptoms

- Hemiplegia or hemiparesis (sparing the face)
- Paraplegia or paraparesis
- Tetraplegia or tetraparesis.

Autonomic symptoms

- Constipation
- Urinary retention
- Dizziness (due to hypotension)
- Cold, shivering, and drowsiness (due to hypothermia)
- Erectile dysfunction
- Abdominal pain and distension (due to ileus)
- Syncope (due to bradycardia).

Pain is a common presenting symptom for cord compression. Pain can be local, mechanical, or radicular, but these patterns often overlap. Patients will describe gnawing or aching pain in the mechanical segment, and sharp radicular pain. Axial mechanical pain is increased with movement, especially flexion, extension, or rotation of the spinal segments. Patients who present with pain, either axial or radicular, and mild motor symptoms should be imaged if the pain has been present for ≥3 months. If significant weakness is present, consultation with a neurosurgery specialist is appropriate.

Cervical spinal stenosis can present with arm wasting or weakness and leg spasticity. Patients with lumbar spinal stenosis may complain of leg claudication with leg weakness or numbness. Central cord compression syndrome presents with arm weakness with preserved leg function.

Patients should be asked about the clinical history of malignancy if involvement of the spine is suspected. The most common tumours primarily involving the spine include central nervous system tumours such as ependymoma, glioma and meningioma, multiple myeloma, and osteosarcoma. The most common tumours metastasising to the spine include small cell lung cancer, non-small cell lung cancer, breast cancer, prostate cancer, and renal cancer.

Clinical presentation of epidural abscess may be quite variable. The clinical triad of fever, back pain, and neurological deficit is not present in most patients. Early presentations may be subtle, and atypical
presentations are not unusual. For epidural abscess, a 4-phase sequential evolution has been described, with the following symptoms:

- Localised spinal pain
- Radicular pain and paraesthesias
- Muscular weakness, sensory loss, and sphincter dysfunction
- Paralysis.

**Examination**

Following a history of acute spinal trauma and while examination is undertaken, the patient should be immobilised with a cervical collar and backboard/head strap.

The neurological examination is started using the American Spinal Injury Association (ASIA) sensory and motor impairment scoring system, which will be repeated during the clinical course to get an idea of evolution towards improvement or aggravation.[14] [15] The examination is fast and the patient is kept immobilised.

Examination may be combined with investigations for associated traumatic injuries (thoracic trauma, head trauma, and lesions of intra-abdominal organs). On the basis of these initial investigations, acute imaging and surgical management may be planned.

Motor changes can be tested using the Medical Research Council manual muscle motor test scoring system of 0 to 5.[16] Any acute or sub-acute motor weakness without obvious alternative explanation is an indication for CT or MRI (preferred) imaging and neurological consultation. Motor loss is a presenting symptom in 60% of patients with disc-/bone-related cord compression.[17]

To detect a sensory level, separate tests for pinprick and vibration sense are done by applying the stimulus and moving it rostrally until a change is noted.[14]

Reflexes are lost following SCI. The following reflex tests may be made:

- Superficial abdominal reflexes are tested by running a stimulus in any abdominal quadrant towards the umbilicus and noting the umbilical movement towards the stimulus.
- An anal wink is contraction of the anal sphincter on stimulation of the perineal area.
- The bulbocavernosus reflex is elicited by tapping the dorsum of the penis resulting in contraction of the pelvic floor muscles.
- The cremasteric reflex is scrotal elevation due to contraction of the cremasteric muscle following stimulus to the upper inner thigh.
- Babinski’s sign is an abnormal plantar reflex consisting of extension (upwards motion) of the great toe on stimulation of the lateral side of the sole, starting from the heel to the base of the toes.

Spinal shock is initially associated with areflexia and hypo-reflexia, but is replaced by increased tone, hyper-reflexia and a positive Babinski’s sign after a few weeks. Bilateral Babinski’s signs, and decreased anal sphincter tone and bulbocavernosus reflex are late signs in tumour, but may be present on first examination in the trauma patient. They are uncommon in disc herniation. Their detection warrants urgent imaging (MRI, myelogram, CT with contrast) and neurosurgical referral.[16]

Hyper-reflexia and loss of pinprick sensation, temperature, position, and vibratory sensation may occur early, especially when associated with malignancy. Sensory changes that are symmetrical raise the possibility of neuropathy, even if associated with mild weakness. Pain that progresses down the
Asymptomatic (or less symptomatic) limb caused by straight-leg raising may suggest cord compression or disc herniation.

From the initial patient examination, a number of classic syndromes of incomplete SCI exist and knowledge of these may direct further investigations and treatment:

- **Complete cord transection syndrome** results in a group of symptoms known as spinal shock, which, when seen at the high cervical level, include quadriplegia, respiratory insufficiency, loss of bladder and bowel function, anaesthesia below the affected level, and neurogenic shock (hypotension and hypothermia). Lower cervical transection spares the respiratory muscles. Horner's syndrome may be seen with higher transections due to loss of descending sympathetic pathways from the hypothalamus and consists of miosis, anhidrosis, and ipsilateral ptosis. High thoracic lesions result in paraparesis with autonomic function loss. Lower thoracic, lumbar, and sacral cord transection result in loss of bowel and bladder function.

- **Central cord syndrome**, seen in syringomyelia, central canal ependymoma, and haemorrhage following trauma, is associated with greater loss of upper limb function compared to the lower limbs. Acute, traumatic central cord syndrome affects the corticospinal tract axons that control the muscles of the hands. There may be a history of trauma with neck hyper-extension and initial quadriplegia that is replaced by leg recovery after a few minutes. Proximal arm weakness, sensory loss, and bladder dysfunction are followed by varying degrees of recovery.

- **Brown-Sequard’s syndrome** results from a hemisection lesion of the spinal cord and is more commonly seen after trauma. Ipsilateral segmental anaesthesia is seen at the level of the lesion with ipsilateral paralysis, loss of vibration and position sense below the level of the lesion, and hyper-reflexia. Contralateral loss of pain and temperature sensation is seen 2 or 3 segments below the lesion.

- **Anterior cord syndrome** occurs when the injury affects the anterior spinal tracts, including the vestibulospinal tract. It is most commonly associated with anterior spinal artery occlusion. There is loss of pain, loss of temperature sensation, and paralysis below the level of the lesion, with sparing of touch, position, and vibration sensation.

- **Posterior cord syndrome** is rare and occurs when the injury affects the posterior spinal tracts, most commonly due to posterior spinal artery occlusion. Loss of pain, position, and vibration sensation is seen below the level of the lesion.

- **Cauda equina syndrome** is commonly due to disc compression and stenosis of the spinal canal. The syndrome consists of saddle (perineal) anaesthesia, bladder retention, and leg weakness. The reported incidence of cauda equina syndrome resulting from herniated lumbar disc varies from 1% to 15% and commonly affects males aged 40 to 60 years. Bladder dysfunction may present as incontinence, but often presents earlier as difficulty starting or stopping a stream of urine. Urinary incontinence is due to overflow.

**Imaging**

Patients with acute SCI should be immobilised with a cervical collar and backboard/head strap while imaging investigations are being undertaken. MRI and CT imaging are preferred, with anteroposterior, lateral, and special views being required to show alignment of bone structures. In the patient with potential traumatic myelopathy, assessing mechanical stability of the spine is the first priority. Plain radiographs are useful for this purpose, but CT may be more useful. At some centres, routine multi-detector CT with sagittal and coronal reconstructions is supplanting the role of plain radiographs, especially in the setting of multiple trauma. MRI is the study of choice when there is incomplete paralysis or under other...
Spinal cord compression

Diagnosis

Circumstances where direct visualisation of neural or ligamentous structures is clinically necessary.[18] [19] [Fig-1]

[Fig-2]

SCI due to SCC can sometimes occur with no findings on imaging, a situation termed SCI without roentgen abnormality (SCIWORA).[20] This is much more common in children than adults and does not necessarily apply to MRI, as many adult patients with SCIWORA will have findings on MRI.

Patients with chronic pain only may be imaged with plain x-ray films, and if non-diagnostic, with MRI imaging of the involved region of the spine. MRI has largely replaced CT scanning in the non-invasive evaluation of patients with painful myelopathy because of its superior soft-tissue resolution and multiplanar capability. When MRI is not available, or to answer specific questions before surgical intervention, myelography and CT myelography may be useful.[18] CT imaging can show bone changes and some soft tissue changes including cysts, abscesses, haemorrhage, cord oedema, and calcification. CT myelography is preferred for detection of spinal canal abnormalities. MRI is the best imaging method for evaluation of neural tissue.[18] [21] Urgent MRI is recommended for all patients who have new-onset urinary symptoms with associated back pain or sciatica.[20]

In patients with suspected tumours, both unenhanced and enhanced imaging can be used. If available, 3D reconstruction aids decision making.[22]

Patients who present with a tumour history, especially of a type commonly recognised as being associated with spinal metastases (e.g., breast, prostate, renal or lung tumours, sarcoma, and multiple myeloma), should undergo CT- or MRI-enhanced imaging. Twenty-eight percent of patients with spinal metastases will have multiple areas of epidural/bony involvement.[23] A thorough metastatic work-up is paramount in patients with spinal metastasis. In patients with rapidly progressing symptoms of malignancy, chest radiography and physical examination may be all that is warranted. Plain radiography of the entire spine should then be performed, followed by CT or MRI with and without contrast enhancement. CT-guided biopsy of suspected tumours can confirm the diagnosis. [Fig-3]

Epidural abscess is best detected by MRI with or without gadolinium enhancement. The region for MRI examination is dictated by the location of the pain. CT guidance may also allow for surgical aspiration and diagnosis of infection or drainage of epidural abscess. Whole-body MRI and MRI imaging of the bone marrow is a new modality for the detection of metastatic carcinoma and multiple myeloma of the bone with high diagnostic accuracy.[24] Studies have utilised positive emission tomography scanning to aid in the identification of lesions of concern through the detection of hypermetabolism. It has also been suggested that these images aid in the prediction of surgical outcomes.[25]

Urodynamic studies are useful to evaluate the degree and cause of sphincter dysfunction, as well as to monitor recovery of bladder function following decompression surgery.[26]

Laboratory tests

Pre-operative laboratory tests for patients with acute traumatic SCI include clotting studies, full blood count, and electrolyte examination. There are no specific laboratory tests for diagnosis of acute SCI, but cerebrospinal fluid analysis may be helpful in excluding other non-traumatic causes of SCI, such as transverse myelitis, HIV myelopathy, and infection.
[VIDEO: Diagnostic lumbar puncture in adults: animated demonstration]

Patients with pain due to chronic SCC and with history of immunocompromise, IV drug use, or recent invasive procedures should undergo laboratory testing for erythrocyte sedimentation rate and C-reactive protein. If these are elevated, MRI with and without gadolinium is ordered to exclude infection. The most commonly reported organism in epidural abscess is *Staphylococcus aureus*, although many other bacteria have been implicated. Blood cultures or local aspiration can provide bacteriological confirmation in patients with or without neurological signs due to infection.[33] [34] [35]

Definitive diagnosis of primary or metastatic spinal tumour is done by CT-guided biopsy and histopathology.

**Risk factors**

**Strong**

**age between 16 and 30 years and male sex**

- The incidence of spinal cord injury (SCI) is highest among people age 16 to 30 years (53%). Car accidents are the leading cause of SCI in the US for people age under 65 years. Males represent >80% of all reported cases of SCI and 90% of all sports-related injuries. Sports- and recreation-related spinal cord injuries primarily affect people under 29 years of age.[3]
- Disc desiccation, disc herniation, and progressive bony stenosis are associated with ageing. Falls are the leading cause of spinal cord injuries for people aged ≥65 years.[3]

**trauma**

- Falls in older people and motor vehicle accidents in the younger age group are often part of the history of these patients.  
  [Fig-1]
-  
  [Fig-2]

**tumour**

- A history of breast, prostate, renal, or lung malignancy, sarcoma, and multiple myeloma is associated with chronic spinal cord compression.[12]  
  [Fig-3]

**osteoarthritis**

- Osteoporosis in older women is associated with vertebral compression fracture.[6] [13]

**high-risk occupation**

- High-risk occupations for trauma and disc herniation include construction workers, agricultural workers, seamen, bricklayers, road menders, vehicle drivers, military personnel, law enforcement officers, and firefighters.

**high-risk recreational activities**

- High-risk recreational activities for trauma include vehicle racing, mountaineering, diving, horse riding, and gymnastics.
**Weak intravenous drug use**

- An increased incidence of spinal epidural abscess is associated with intravenous drug abuse.[11]

**Immunosuppression**

- May be associated with increased risk of discitis or epidural abscess and of opportunistic infections involving the spine. Diabetes should also be considered as a risk factor for epidural abscess.

### History & examination factors

#### Key diagnostic factors

**Presence of risk factors (common)**

- Key factors include a history of trauma including motor vehicle accident, and high-risk occupation or sports activity.

**Age group 16 to 30 years (trauma) (common)**

- Trauma is the most likely cause and most commonly involves C4/5 and C5/6.

**Age group 30 to 50 years (disc disease) (common)**

- With radicular pain, the most common cause is disc disease (C5/6 or L4/5, L5/S1).[36]

**Age group 40 to 75 years (malignancy) (common)**

- Malignancy involving the spine is a most common cause in this age group.

**Acute onset and duration of symptoms (common)**

- Acute symptoms are commonly associated with traumatic cord compression or disc herniation.

**Chronic onset and duration of symptoms (common)**

- Chronic symptoms are associated with osteoporosis, osteomyelitis, or malignancy.

**Back pain (common)**

- Back pain without sensory, motor, or autonomic symptoms may have causes other than cord compression or may be associated with degenerative spine disease (spondylosis).
- Metastatic malignancy, osteoporotic compression fractures, and osteomyelitis of the spine commonly cause back pain and investigations should be done to rule them out.

**Numbness or paraesthesias (common)**

- Acute, severe symptoms occur in acute cord compression, commonly due to trauma.
- Mild symptoms may be associated with early forms of chronic cord compression.
- Other, non-spinal causes should be ruled out if these are the only symptoms, for example, diabetic neuropathy or other peripheral neuropathies.

**Weakness or paralysis (common)**

- Acute, severe symptoms occur in acute cord compression, commonly due to trauma.
- Mild symptoms may be associated with early forms of chronic cord compression.
Spinal cord compression

Diagnosis

• Other, non-spinal causes should be ruled out if these are the only symptoms, for example, motor neuron disease and inflammatory myopathies.

bladder or bowel dysfunction (common)

• Seen with longstanding lumbar cord compression, particularly spinal stenosis, and as part of cauda equina syndrome (with bowel and/or bladder incontinence, lower extremity weakness, and saddle paraesthesias).

hyper-reflexia (common)

• An early sign of spinal cord compression and is more commonly associated with malignancy.

sensory loss (common)

• Loss of pinprick, temperature, position, and vibratory sensation occurs early in cord compression, especially when due to malignancy.
• Sensory changes that are symmetrical raise the possibility of neuropathy, even if associated with mild weakness.
• The American Spinal Injury Association (ASIA) sensory and motor impairment scale is used for classifying and grading acute traumatic spinal cord injury and acute non-traumatic spinal cord injury according to motor and sensory signs (A to E).[1]

muscle weakness or wasting (common)

• Assessed on a scale of 0 to 5 using Medical Research Council manual muscle testing.[16]
• The American Spinal Injury Association (ASIA) sensory and motor impairment scale is used for classifying and grading acute traumatic spinal cord injury and acute non-traumatic spinal cord injury according to motor and sensory signs (A to E).[1]

loss of tone below level of suspected injury (spinal shock) (common)

• In spinal shock, there is loss of reflexes, motor paralysis, and hypo-reflexia or areflexia. This is different from neurogenic shock.[40]

hypotension and bradycardia (neurogenic shock) (common)

• Neurogenic shock usually follows cervical or high thoracic injury. Patients show hypotension, bradycardia, warm, dry extremities, peripheral vasodilation, venous pooling, poikilothermia, priapism, and decreased cardiac output.

complete cord transection syndrome (common)

• Results in a group of symptoms known as spinal shock, which, when seen at the high cervical level, include quadriplegia, respiratory insufficiency, loss of bladder and bowel function, anaesthesia below the affected level, and neurogenic shock (hypotension and hypothermia).
• Lower cervical transection spares the respiratory muscles. Horner’s syndrome may be seen with higher transections due to loss of descending sympathetic pathways from the hypothalamus and consists of miosis, anhidrosis, and ipsilateral ptosis.
• High thoracic lesions result in paraparesis with autonomic function loss. Lower thoracic, lumbar, and sacral cord transection result in loss of bowel and bladder function.

cauda equina syndrome (common)

• Due to disc compression and stenosis of the spinal canal. The syndrome consists of saddle (perineal) anaesthesia, bladder retention, and leg weakness.
Spinal cord compression

Diagnosis

- The reported incidence of cauda equina syndrome resulting from herniated lumbar disc varies from 1% to 15% and commonly affects males aged 40 to 60 years.
- Bladder dysfunction may present as incontinence but often presents earlier as difficulty starting or stopping a stream of urine. Urinary incontinence is on the basis of overflow.

Central cord syndrome (common)

- An acute form of cord compression injury, often associated with hyper-extension injury in an individual with cervical spondylosis. It occurs more frequently among older people.
- Central cord syndrome is associated with greater loss of upper limb function compared to the lower limbs, including the vestibulospinal tract.

Other diagnostic factors

History of malignancy (uncommon)

- History of breast, prostate, renal or lung malignancy, sarcoma, and multiple myeloma.
- Metastases involve the thoracic (70%), lumbar (20%), and cervical (10%) spine.
- Forty percent of patients with non-spinal bone metastasis will have epidural spine compression.[37]
- Cadaveric surveys indicate 40% to 90% of cancer patients have metastatic disease to the spine; 38% are in multiple sites in the epidural space.[38] [39]
- Any patient with suspected or known cancer should undergo CT or MRI when presenting with back pain.

Immunosuppression (uncommon)

- Immunosuppressed patients and those with HIV may develop vertebral osteomyelitis, discitis, or epidural abscess following a skin infection or urinary tract infection. They may also have a history of weight loss and fever. Diabetes should also be considered as a risk factor for epidural abscess.

Intravenous drug use (uncommon)

- The incidence of spinal infection, particularly epidural abscess, is thought to be increasing in relation to intravenous drug abuse.[11]

Loss of rectal sphincter reflex (uncommon)

- Patulous rectal tone and history of recent incontinence are signs of severe cord involvement.

Loss of appendicular reflexes (uncommon)

- Asymmetric loss of biceps, triceps, knee, and ankle reflexes.

Local deformity of spine on palpation (uncommon)

- Acute traumatic injury or infectious kyphosis (angulation of spine) is associated with impending cord compression.

Brown-Sequard's syndrome (uncommon)

- Results from a hemisection lesion of the spinal cord (due to tumour or disc herniation).
- In the acute presentation, signs include unilateral spastic paralysis on the same side of the body, as well as ipsilateral loss of vibration and proprioception (position sense), with pain and temperature sensation being lost from the contralateral side beginning 1 or 2 segments below the lesion.

Anterior cord syndrome (uncommon)
Spinal cord compression

**Diagnosis**

- Occurs when the blood supply to the anterior portion of the spinal cord is interrupted.
- It is characterised by loss of motor function below the level of injury, loss of sensations carried by the anterior columns of the spinal cord (pain and temperature), and preservation of sensations carried by the posterior columns (fine touch and proprioception).

**Posterior cord syndrome (uncommon)**

- Caused by a lesion of the posterior portion of the spinal cord. It can be caused by an interruption to the posterior spinal artery. Unlike anterior cord syndrome, it is a very rare condition.
- It is possible for it to present as Brown-Sequard's syndrome with unilateral spastic paralysis on the same side of the body as well as ipsilateral loss of vibration and proprioception (position sense), with pain and temperature sensation being lost from the contralateral side beginning 1 or 2 segments below the lesion.

**Diagnostic tests**

**1st test to order**

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MRI spine</strong></td>
<td>disc displacement, epidural enhancement, mass effect, T2 cord signal</td>
</tr>
<tr>
<td>- This is the imaging study of choice for suspected cord compression of any aetiology. Conventional T2 and T2-weighted fast spin-echo images are used in the diagnosis of degenerative disc disease. [41] [19]</td>
<td></td>
</tr>
<tr>
<td>- T2-weighted cord signal increase in disc compression and trauma allows for some prognostication about eventual recovery of function. [42] [21]</td>
<td></td>
</tr>
<tr>
<td>- MRI imaging for tumours presenting as spinal cord compression will yield multiple sites in 28%. [43]</td>
<td></td>
</tr>
<tr>
<td><strong>Gadolinium-enhanced MRI spine</strong></td>
<td>Infection: epidural space and bone involvement; metastatic disease: visualisation of tumour</td>
</tr>
<tr>
<td>- The imaging modality of choice in infection including osteomyelitis and epidural abscess.</td>
<td></td>
</tr>
<tr>
<td><strong>Plain spine x-ray</strong></td>
<td>decreased disc space height (disc compression), loss of bony detail (tumour, infection), misalignment of vertebral elements (trauma), loss of end-plate definition (infection)</td>
</tr>
<tr>
<td>- Plain films are of limited value if the diagnostic possibilities are disc prolapse or spinal stenosis. In the setting of trauma, plain films combined with CT are the tests of choice. [44] [45]</td>
<td></td>
</tr>
<tr>
<td>- False-positive findings are more common in plain films than in CT scans.</td>
<td></td>
</tr>
<tr>
<td>- In tumour diagnosis, plain films require 50% bone loss before noticeable change is seen on the radiograph. [46] Current studies indicate that MRI is the preferred imaging method for metastatic spinal disease. [47]</td>
<td></td>
</tr>
<tr>
<td><strong>CT spine</strong></td>
<td>cord compression from tumour expansion into the canal or bony fragments from pathological fracture</td>
</tr>
<tr>
<td>- Best used in combination with myelography or when MRI is unavailable. Can be used for surgical planning. Distinguishes between neural compression from bone and ligaments or disc protrusion.</td>
<td></td>
</tr>
</tbody>
</table>
## Diagnosis

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CT myelography</strong></td>
<td>classical hour-glass constriction shape of the dye column</td>
</tr>
<tr>
<td>• Performed less often now that MRI is widely available. It is still used for patients with implanted pacemakers or who may suffer from claustrophobia, or when there is any other reason that a good-quality MRI cannot be obtained.</td>
<td></td>
</tr>
<tr>
<td>• Myelography allows imaging of the degree of cord compression in flexion and extension.</td>
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</tr>
</tbody>
</table>

### Other tests to consider

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>full blood count with differential</strong></td>
<td>raised white blood cell count with neutrophilia seen in infection</td>
</tr>
<tr>
<td>• Laboratory studies are not generally helpful in establishing the diagnosis for tumour-, trauma-, or disc-related compression of the cord, but may assist in the diagnosis of infection.</td>
<td></td>
</tr>
<tr>
<td><strong>erythrocyte sedimentation rate and C-reactive protein</strong></td>
<td>elevated in infection or inflammation</td>
</tr>
<tr>
<td>• Non-specific markers of infection and inflammation. Persistent elevation may herald osteomyelitis or epidural abscess.</td>
<td></td>
</tr>
<tr>
<td><strong>blood or cerebrospinal fluid cultures</strong></td>
<td>positive in epidural abscess, discitis, or osteomyelitis</td>
</tr>
<tr>
<td>• The most commonly reported organism in epidural abscess is <em>Staphylococcus aureus</em>, although many other bacteria have been implicated, including <em>Streptococcus</em> and <em>Pseudomonas</em> species, <em>Escherichia coli</em>, and <em>Mycobacterium tuberculosis</em>. MRSA is increasingly reported, particularly in patients with spinal surgery or implanted devices.</td>
<td></td>
</tr>
<tr>
<td><strong>tumour biopsy and histopathology</strong></td>
<td>tissue diagnosis of malignancy, if present</td>
</tr>
<tr>
<td>• CT-guided biopsy and histopathology confirm the diagnosis of primary or metastatic spinal cord tumour.</td>
<td></td>
</tr>
<tr>
<td><strong>urodynamic studies</strong></td>
<td>reduced bladder contractility sphincter dysfunction</td>
</tr>
<tr>
<td>• Urodynamic studies are useful to evaluate the degree and cause of sphincter dysfunction, as well as to monitor recovery of bladder function following decompression surgery.[26]</td>
<td></td>
</tr>
<tr>
<td><strong>positive emission tomography (PET) scan of the spine</strong></td>
<td>areas of hypermetabolism detected</td>
</tr>
<tr>
<td>• Studies have utilised positive emission tomography scanning to aid in the identification of lesions of concern through the detection of hypermetabolism. It has also been suggested that these images aid in the prediction of surgical outcomes.[25]</td>
<td></td>
</tr>
</tbody>
</table>
## Differential diagnosis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Differentiating signs / symptoms</th>
<th>Differentiating tests</th>
</tr>
</thead>
</table>
| Transverse myelitis           | • Approximately one third of patients report a febrile illness preceding the symptoms. Most have leg weakness of varying degrees of severity. The arms are involved in a minority of cases.   | • Cerebrospinal fluid analysis shows pleocytosis with a modest number of lymphocytes and increase in total protein.  
  • MRI shows focal demyelination with possible enhancement at the appropriate level. Lyme titres are occasionally found to be high.[48] |
| Guillain-Barre syndrome (GBS) | • Two-thirds have a history of gastroenteritis or influenza-like illness weeks before onset of neurological symptoms. It is frequently severe and presents with features which are similar to those of spinal cord compression, as an ascending paralysis initially with weakness in the legs that spreads to the upper limbs and the face, along with complete loss of deep tendon reflexes. Autonomic signs may be present in some variations. May develop progressive respiratory muscle weakness requiring ventilation. | • Typical cerebrospinal fluid findings include albumin-cytological dissociation, that is, an elevated protein level (1-10 g/L or 100-1000 mg/dL) without an accompanying increased cell count. A sustained increased WBC count may indicate an alternative diagnosis such as infection.  
  • Electrodiagnostics (electromyogram and nerve conduction studies) may show prolonged distal latencies, conduction slowing, conduction block, and temporal dispersion of compound action potential in demyelinating cases. In primary axonal damage, the findings include reduced amplitude of the action potentials without conduction slowing.[49]  
  • Nearly 40% of patients are seropositive for *Campylobacter jejuni*. [50] |
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>HIV-related myelopathy</td>
<td>• History of HIV infection or high-risk behaviours (IV drug use, HIV-infected blood transfusion, unprotected sex).&lt;br&gt;• Signs and symptoms referable to the spinal cord lesions, including paraparesis, often accompanied by spasticity or ataxia (or both) coupled with dementia.</td>
<td>• Enzyme-linked immunosorbent assay (ELISA) testing should be ordered when HIV testing is indicated. False-negatives may occur during window period immediately after infection and before antibodies to HIV have developed. A positive result should be confirmed with a Western blot or second ELISA. The window period can be reduced to 2 to 4 weeks by using fourth-generation tests and those that include IgM antibodies to HIV.&lt;br&gt;• Cerebrospinal fluid, microbiological, and spinal imaging studies may be inconclusive or non-specific. [51] [52]</td>
</tr>
<tr>
<td>Amyotrophic lateral sclerosis (ALS)</td>
<td>• Presents as a combination of upper motor neuron (UMN) and lower motor neuron (LMN) symptoms and signs.&lt;br&gt;• Approximately 60% of ALS patients experience muscle weakness and stiffness as the initial symptoms. The neurological examination usually shows evidence of muscle weakness (localised or widespread, depending on the extent of the disease). The examination also reveals muscle atrophy. The muscles may be so stiff that, when the neurologist moves them, they continue to move abnormally afterwards. When the neurologist tests the knee jerk reaction, the movement is abnormally quick (hyper-reflexia).</td>
<td>• Electromyogram (EMG) testing is a significant part of the diagnosis. The current criteria define a positive EMG when signs of active denervation include fibrillation potentials and positive sharp waves, with fasciculation potentials. [53]</td>
</tr>
<tr>
<td>Condition</td>
<td>Differentiating signs / symptoms</td>
<td>Differentiating tests</td>
</tr>
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</table>
| **Multiple sclerosis**  | • Can mimic clinical symptoms of spinal cord compression. However, in almost all cases of multiple sclerosis there are also brain lesions.  
  • Has a variable presentation with multiple episodes separated by space (i.e., neurological symptoms result from lesions in different central nervous system sites) and time. Common symptoms include progressive limb weakness, gait difficulty, ataxia, loss of balance, and paroxysmal vertigo.  
  • In the setting of acute paraparesis, visual symptoms (visual loss) may be present. This is neuromyelitis optica (NMO). NMO is thought to be a distinct entity from MS by many neurologists. It has a relapsing course (80% to 90%), and predominantly affects females.[54] | • Brain MRI typically shows areas of demyelination.  
  • Cerebrospinal fluid examination shows raised IgG and oligoclonal banding. If NMO suspected (optic neuritis, myelitis, longitudinal cord lesion on MRI), NMO-IgG seropositivity.[54]                                                                                                                                                  |
| **Diabetic neuropathy** | • History of diabetes mellitus. Pain and loss of sensation in the feet in a glove-and-stocking distribution. Bladder dysfunction may be present due to autonomic neuropathy.                                                                                                                                                                                                                                           | • Nerve conduction studies show reduction in sensory nerve conduction velocity and a decrease in amplitude.                                                                                                                                                                                     |
| **Polymyositis**        | • Symmetrical weakness of shoulder and pelvic girdles.                                                                                                                                                                                                                                                                                                                                                                     | • Elevated muscle enzyme levels (e.g., CK), often with a positive ANA titre.  
  • Characteristic changes in electromyogram include increased needle insertion activity, spontaneous fibrillations, low-amplitude short-duration polyphasic motor potentials, and complex repetitive discharges.  
  • Diagnosis is confirmed with muscle biopsy, indicating immune cell infiltration and destruction of muscle fibres.                                                                                                                                                                           |
### Condition | Differentiating signs / symptoms | Differentiating tests
--- | --- | ---
Hereditary muscular dystrophy | • Severe proximal and distal muscle weakness without sensory changes from an early age. | • MRI and electromyogram/nerve conduction studies will show only myopathic changes and no spinal cord compression.
Peripheral neuropathy | • Peripheral neuropathy and neuropathies due to diabetes or thyroid disease may be difficult to differentiate from neurological symptoms of compression neuropathies. | • Nerve conduction studies and electromyogram are helpful in confirming neuropathy and characterising the neuropathy, that is, demyelinating, axonal, polyneuropathy, mononeuropathy multiplex, radiculopathy, or plexopathy.

### Diagnostic criteria

**The Medical Research Council (MRC) manual muscle test (MMT) scale**[16][55]

Manual muscle test scale

- Grade 5: patient can hold the position against maximum resistance and through complete range of motion (ROM).
- Grade 4: patient can hold the position against strong-to-moderate resistance and has full ROM.
- Grade 3: patient can tolerate no resistance but can perform the movement through the full ROM.
- Grade 2: patient has all or partial ROM in the gravity eliminated position.
- Grade 1: the muscle/muscles can be palpated while the patient is performing the action in the gravity eliminated position.
- Grade 0: no contractile activity can be felt in the gravity eliminated position.

**American Spinal Injury Association (ASIA) sensory and motor impairment scale**[1]

Patients with acute, traumatic spinal cord injury are stratified based on the ASIA impairment scale which identifies the spinal level of the lesion (cervical, thoracic or lumbar) and the degree of impairment (A to E).

- A - Complete: no sensory or motor function is preserved in sacral segments S4 to S5.
- B - Incomplete: sensory, but not motor, function is preserved below the neurological level and extends through sacral segments S4 to S5.
- C - Incomplete: motor function is preserved below the neurological level, and most key muscles below the neurological level have muscle grade <3.
- D - Incomplete: motor function is preserved below the neurological level, and most key muscles below the neurological level have muscle grade ≥3.
- E - Normal: sensory and motor functions are normal.
Spinal cord compression

Diagnosis

Frankel sensory and motor impairment scale[56]

An older and simpler classification scheme that may be used for acute traumatic spinal cord injury and acute non-traumatic spinal cord injury:

- A: no neurological function
- B: sensory function only
- C: some sensory and motor function preserved
- D: useful motor function
- E: normal.
Step-by-step treatment approach

The main goal of treatment is to prevent clinical deterioration from progressive degenerative changes of spinal cord injury (SCI), to relieve pain and symptoms, and to restore functional ability. The following patient groups can be considered in the treatment approach: patients with acute, traumatic SCI; patients with intervertebral disc compression (cauda equina syndrome); patients with malignant spinal cord compression (SCC); and patients with epidural abscess (infection).

**Acute, traumatic SCI**

Patients with acute, traumatic SCI are stratified based on the American Spinal Injury Association (ASIA) impairment scale, which identifies the spinal level of the lesion (cervical, thoracic, or lumbar) and the degree of impairment (A to E). Acute, traumatic SCI is a medical emergency and management should be undertaken at a trauma centre with experience in SCI and in-house neurosurgical expertise.

All patients should be immobilised with a cervical collar and backboard/head strap while clinical and imaging studies are being performed. For patients with confirmed spinal injury, neurosurgical evaluation with neural decompression and spinal stabilisation is recommended within 24 hours. The role of rapid surgery for compression is based on the postulate that early decompressive surgery significantly improves outcome and reduces complication rates, but this remains controversial.

There is an increasing trend towards early surgery, although one study found no significant neurological benefit between early (<72 hours) and late (>5 days). Although the most common treatment offered in industrialised countries, there is a lack of data to support the use of post-traumatic internal fixation. Good-quality controlled trials are needed to answer this question. External orthoses are an alternative to surgery; thoracic injuries respond better to external immobilisation than do lumbar injuries. The Spine Studies Trauma Group assessed the literature and canvassed expert opinion to develop an algorithm for the surgical approach to subaxial cervical spine injuries, based on the sub-axial cervical spine injury classification (SLIC) system algorithm. However, this algorithm is limited in its generalisability. One study suggested that early surgery in central cord syndrome yielded better motor scores at 1 year.

Clinically apparent deep venous thrombosis occurs in approximately 15% and pulmonary embolism in approximately 5% of acute SCI patients. Stasis from paralysed muscles and hyper-coagulability remain the 2 major factors contributing to the development of thrombosis in this patient population. Compression stockings, intermittent pneumatic compression devices, and low-molecular-weight heparin are the mainstays of both prevention and treatment. All patients should be given prophylaxis to prevent venous thromboembolism and possible pulmonary embolism. Treatment should begin no later than 72 hours following SCI. First-line treatment is low-molecular-weight heparin, with second-line treatment being un-fractionated heparin. In patients with contraindications to anticoagulation, an inferior vena cava filter can be used.

For both non-operative and operative candidates, the use of high-dose methylprednisolone (given within 8 hours) is in widespread use, as supported by the National Acute Spinal Cord Injury Study III (NASCIS III) study (US). However, more research is required on the use of corticosteroids, which may not be suitable for all patients and which may not be recommended by all clinicians. If methylprednisolone is used, initiation of treatment should be within 8 hours of injury. A 30-mg/kg intravenous bolus is given over 15 minutes followed by a 5.4-mg/kg/hour infusion for 24 hours if therapy is initiated within 3 hours of injury. If the infusion is initiated 3 to 8 hours after injury, the infusion is for 48 hours. Some clinicians do not recommend the use of corticosteroids due to their...
complications of gastrointestinal haemorrhage, corticosteroid myopathy, and increased risk of infection. It is contraindicated in gunshot wounds to the spine because it has no demonstrated benefit.[63] [64] [65]

Treatment of autonomic dysfunction and prevention of hypotension may be required, particularly in patients with cervical trauma. Hypotension may be associated with spinal shock, neurogenic shock, hypovolaemia, sepsis, bradycardia, or cardiogenic shock. Hypotension should be treated, as it contributes to neurological damage. Treatment consists of central line placement, volume resuscitation, and vasopressors.

Prevention of stress ulceration with proton-pump inhibitors or H2 antagonists is indicated for 4 weeks following SCI.

Nutritional support should begin within 72 hours of SCI, with isotonic feeds and evaluation of dysphagia. Mechanically assisted ventilation or manually assisted cough may be required. Bladder catheterisation may be continuous or intermittent. Laxatives and bowel evacuation may be required. Passive and active range of motion (ROM) is advised with prevention of pressure ulcers of the occiput, sacrum, and heels through manual or automatic turning every 2 hours.

[VIDEO: Tracheal intubation animated demonstration ]
[VIDEO: Bag-valve-mask ventilation animated demonstration ]

**Intervertebral disc compression (cauda equina syndrome)**

Emergency decompression of the spinal canal is the appropriate treatment option within 48 hours after the onset of symptoms. Timing of surgery is generally divided into 2 groups of patients: caudal equina syndrome with bladder symptoms, and without bladder symptoms. In the latter group, surgery within 24 hours results in fewer bladder symptoms post-procedure. If the presentation is with bladder symptoms, timing of surgery does not seem to make a difference.[69] [70] Surgery of choice is wide decompressive laminectomy. Intra-operative monitoring of somatosensory and motor-evoked potentials allows for evaluation of radiculopathy and neuropathy.[71] Urodynamic studies are useful to evaluate the degree and cause of sphincter dysfunction, as well as to monitor recovery of bladder function following decompression surgery.[26] Pharmacological treatments for disc herniation have not been shown to be of benefit.[72] 1[C]Evidence

Patients with bilateral sciatica have been reported to have a less favourable prognosis than patients with unilateral pain. Patients with complete perineal anaesthesia are more likely to have permanent paralysis of the bladder. The extent of perineal or saddle sensory deficit has been reported to be the most important predictor of recovery. Females and patients with bowel dysfunction have been reported to have worse outcomes postoperatively.[73] [74]

All patients should be given prophylaxis to prevent venous thromboembolism and possible pulmonary embolism. Treatment of autonomic dysfunction and prevention of hypotension may be required, particularly in patients with cervical trauma. Prevention of stress ulceration with proton-pump inhibitors or H2 antagonists is indicated for 4 weeks following SCI. Nutritional support should begin within 72 hours with isotonic feeds and evaluation of dysphagia. Mechanically assisted ventilation or manually assisted cough may be required. Bladder catheterisation may be continuous or intermittent. Laxatives and bowel evacuation may be required. Passive and active ROM is advised with prevention of pressure ulcers of the occiput, sacrum, and heels through manual or automatic turning every 2 hours.
Malignant spinal cord compression

Treatment of spinal metastasis is palliative. Historically, treatment has consisted of corticosteroids, decompressive laminectomy/vertebrectomy, and radiation.[75] 

Corticosteroids provide relief from pain, reduce tumour-associated oedema, and may be oncolytic for some tumours.[76] 

There is no agreement on the type of corticosteroid or on the dose intensity or duration.[78] Guidelines published in 2008 from the National Institute for Health and Care Excellence (NICE) recommend use of dexamethasone.[79]

Controversy exists between advocates of surgery alone and radiotherapy alone.[80] Studies found no difference for radiotherapy alone when compared to decompressive surgery.[81] Current surgical procedures that deal with the entirety of the disease (vertebral body, lamina, lateral elements) raise the prospect for improved functional outcomes, and perhaps improved survival.[82] Reviews have investigated neurological outcomes after laminectomy with and without radiotherapy.[84] Studies have reported that surgery followed by radiation is more effective than radiation alone in treating patients with SCC caused by metastatic cancer.[85] This group emphasised decompression, attempted complete local tumour removal, and spinal stabilisation. They reported a post-surgery ambulation rate of 82%. ASIA scores and Frankel grades were also improved.[85] 

The age of a patient may help guide the choice of therapy: outcome measures of ability to walk and survival are both affected by age. As age increases to the seventh decade, the outcomes of surgery and radiation alone are nearly equal.[88]

Indications for radiotherapy alone include:

- Radiosensitive tumours (small cell lung carcinoma and myeloma)
- No instability
- Rapidly progressive neurological decline with limited life expectancy
- The presence of significant medical comorbidities.

Radiotherapy plus surgery is recommended for:[89] [90] [91]

- Patients who need a tissue diagnosis
- Patients who present with spinal instability
- Patients with radio-resistant tumours.

The morbidity of either surgical approach is about 25%. Complications include infection, spinal instability, root/cord injury, and wound breakdown.[92] [93]

Epidural abscess

Treatment usually involves surgery in combination with intravenous antibiotics. The single most important predictor of the final neurological outcome is the patient's neurological status immediately before surgery. Identification and prompt treatment of the offending organism is paramount. Choice of antibiotic depends upon the results of microbiological culture and sensitivity. Antibiotic treatment is advised for at least 12 weeks.

The most common pathogen in epidural abscess is *Staphylococcus aureus*, although many other bacteria have been implicated, including *Streptococcus* and *Pseudomonas* species, *Escherichia coli*, and *Mycobacterium tuberculosis*. MRSA is increasingly reported, particularly in patients with spinal surgery or implanted devices.
Surgery can be performed with open techniques, allowing for cord decompression as well as sampling of the tissues. For patients with less disabling symptoms, CT-guided needle aspiration of lesions can be performed. Complications of surgery include progressive kyphosis and late neurological compromise.[94]

### Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

<table>
<thead>
<tr>
<th>Acute</th>
<th>( summary )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>acute traumatic spinal cord injury</strong></td>
<td></td>
</tr>
<tr>
<td>1st</td>
<td>immobilisation + decompressive/stabilisation surgery</td>
</tr>
<tr>
<td>adjunct</td>
<td>intravenous corticosteroids</td>
</tr>
<tr>
<td>plus</td>
<td>prevention of venous thromboembolism</td>
</tr>
<tr>
<td>plus</td>
<td>maintenance of volume and blood pressure</td>
</tr>
<tr>
<td>plus</td>
<td>prevention of gastric stress ulcers</td>
</tr>
<tr>
<td>plus</td>
<td>supportive therapies</td>
</tr>
<tr>
<td><strong>non-traumatic intervertebral disc compression (cauda equina syndrome)</strong></td>
<td></td>
</tr>
<tr>
<td>1st</td>
<td>decompressive laminectomy</td>
</tr>
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<td><strong>malignant spinal cord compression</strong></td>
<td></td>
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<td>corticosteroids ± surgery ± radiation therapy</td>
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<tr>
<td><strong>epidural abscess</strong></td>
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<tr>
<td>1st</td>
<td>antibiotics ± surgery</td>
</tr>
</tbody>
</table>
### Spinal cord compression

#### Treatment

<table>
<thead>
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<tbody>
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</tbody>
</table>
## Treatment options

### Acute

#### acute traumatic spinal cord injury

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<tr>
<th>1st</th>
<th>immobilisation + decompressive/stabilisation surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>»</td>
<td>Immobilisation in a cervical collar or backboard/head strap should be done while examination and imaging are being performed. If spinal cord injury is confirmed, neurosurgical investigation with spinal cord decompression and spinal stabilisation is performed. The role of rapid surgery for compression is based on the postulate that early decompressive surgery significantly improves outcomes and reduces complication rates in patients with spinal cord injury.</td>
</tr>
<tr>
<td>»</td>
<td>There is an increasing trend towards early surgery, although one study found no significant neurological benefit between early (&lt;72 hours) and late (&gt;5 days).[58] However, there is a lack of data to support post-traumatic internal fixation. Good-quality controlled trials are needed to answer this question. The Spine Studies Trauma Group assessed the literature and canvassed expert opinion to develop an algorithm for the surgical approach to subaxial cervical spine injuries, based on the SLIC classification system algorithm. However, this algorithm is limited in its generalisability.[59] One study suggested that early surgery in central cord syndrome yielded better motor scores at 1 year.[60]</td>
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<table>
<thead>
<tr>
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<th>intravenous corticosteroids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary options</td>
<td></td>
</tr>
<tr>
<td>» methylprednisolone: 30 mg/kg intravenously as a bolus given over 15 minutes, followed by 5.4 mg/kg/hour intravenous infusion for 24 hours (if &lt;3 hours since injury) or for 48 hours (if 3-8 hours since injury)</td>
<td></td>
</tr>
<tr>
<td>» For both non-operative and operative candidates, the use of high-dose methylprednisolone (given within 8 hours) is in widespread use, as supported by the National Acute Spinal Cord Injury Study III (NASCIS III) study (US).[63]</td>
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<td></td>
</tr>
</tbody>
</table>
Spinal cord compression

Treatment

Acute

because it has no demonstrated benefit.[63] [64] [65] More research is required on the use of corticosteroids, which may not be suitable for all patients and which may not be recommended by all clinicians.[64] [65]

plus prevention of venous thromboembolism

Primary options

» enoxaparin: 40 mg subcutaneously once daily
  -and-
  » compression stockings or pneumatic intermittent compression

Secondary options

» heparin: 5000 units subcutaneously every 8-12 hours
  -and-
  » compression stockings or pneumatic intermittent compression

Tertiary options

» IVC filter

» All patients should be given prophylaxis to prevent venous thromboembolism and possible pulmonary embolism. Treatment should begin no later than 72 hours following spinal cord injury. First-line treatment is low-molecular-weight heparin, with second-line treatment being unfractionated heparin. Treatment duration is at the discretion of the clinician and based on patient degree of immobility.

» In patients with contraindications to anticoagulation, an inferior vena cava (IVC) filter can be used.

» Compression stockings and pneumatic intermittent compression devices may also be beneficial.

plus maintenance of volume and blood pressure

Primary options

» volume resuscitation
  -and/or-
  » dopamine: 1-50 micrograms/kg/minute intravenously, titrate gradually according to response

» Treatment of autonomic dysfunction and prevention of hypotension may be required, particularly in patients with cervical trauma.
### Acute Treatment

Hypotension may be associated with spinal shock, neurogenic shock, hypovolaemia, sepsis, bradycardia, or cardiogenic shock. Hypotension should be treated, as it contributes to neurological damage.

- **Treatment consists of central line placement, volume resuscitation, and vasopressors.** The aim is to maintain a blood pressure of >100 mmHg systolic and an adequate urine output (0.5 mL/kg/hour).

- **Dose should be started low and titrated according to response.**

### Prevention of Gastric Stress Ulcers

**Primary Options**

- **Omeprazole:** 40 mg orally once daily

- **Cimetidine:** 300 mg orally/intravenously every 6 hours

- **Famotidine:** 40 mg orally once daily; 20 mg intravenously every 12 hours

**Prevention of stress ulceration with proton-pump inhibitors or H2-antagonists is indicated for 4 weeks following spinal cord injury.**

### Supportive Therapies

- **Nutritional support** should begin within 72 hours with isotonic feeds and evaluation of dysphagia.

- **Mechanically assisted ventilation** or manually assisted cough may be required.

- **Bladder catheterisation** may be continuous or intermittent.

- **Laxatives and bowel evacuation** may be required.

- **Passive and active range of motion** is advised with prevention of pressure ulcers of the occiput,
Acute

<table>
<thead>
<tr>
<th>non-traumatic intervertebral disc compression (cauda equina syndrome)</th>
<th>1st decompressive laminectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Emergency decompression of the spinal canal is the appropriate treatment option within 48 hours after the onset of symptoms. Pharmacological treatments have not been shown to be of benefit.</td>
<td></td>
</tr>
<tr>
<td>Evidence Urodynamic studies are useful to evaluate the degree and cause of sphincter dysfunction, as well as to monitor recovery of bladder function following decompression surgery.</td>
<td></td>
</tr>
<tr>
<td>plus prevention of venous thromboembolism</td>
<td></td>
</tr>
<tr>
<td>Primary options</td>
<td></td>
</tr>
<tr>
<td>• enoxaparin: 40 mg subcutaneously once daily -and-</td>
<td></td>
</tr>
<tr>
<td>• compression stockings or pneumatic intermittent compression</td>
<td></td>
</tr>
<tr>
<td>Secondary options</td>
<td></td>
</tr>
<tr>
<td>• heparin: 5000 units subcutaneously every 8-12 hours -and-</td>
<td></td>
</tr>
<tr>
<td>• compression stockings or pneumatic intermittent compression</td>
<td></td>
</tr>
<tr>
<td>Tertiary options</td>
<td></td>
</tr>
<tr>
<td>• IVC filter</td>
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</tr>
<tr>
<td>• Compression stockings and pneumatic intermittent compression devices may also be beneficial.</td>
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## Treatment

### Acute

<table>
<thead>
<tr>
<th>plus</th>
<th>maintenance of volume and blood pressure</th>
</tr>
</thead>
</table>

**Primary options**

- volume resuscitation
- dopamine: 1-50 micrograms/kg/minute intravenously, titrate gradually according to response

» Treatment of autonomic dysfunction and prevention of hypotension may be required, particularly in patients with cervical trauma. Hypotension may be associated with spinal shock, neurogenic shock, hypovolaemia, sepsis, bradycardia, or cardiogenic shock. Hypotension should be treated, as it contributes to neurological damage.

- Treatment consists of central line placement, volume resuscitation, and vasopressors. The aim is to maintain a blood pressure of >100 mmHg systolic and an adequate urine output (0.5 mL/kg/hour).

- Dose should be started low and titrated according to response.

<table>
<thead>
<tr>
<th>plus</th>
<th>prevention of gastric stress ulcers</th>
</tr>
</thead>
</table>

**Primary options**

- omeprazole: 40 mg orally once daily
- cimetidine: 300 mg orally/intravenously every 6 hours
- famotidine: 40 mg orally once daily; 20 mg intravenously every 12 hours

» Prevention of stress ulceration with proton-pump inhibitors or H2-antagonists is indicated for 4 weeks following spinal cord injury.

<table>
<thead>
<tr>
<th>plus</th>
<th>supportive therapies</th>
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</thead>
</table>

- Nutritional support should begin within 72 hours with isotonic feeds and evaluation of dysphagia.

- Mechanically assisted ventilation or manually assisted cough may be required.
**Spinal cord compression**

**Acute**

<table>
<thead>
<tr>
<th>[VIDEO: Tracheal intubation animated demonstration ]</th>
<th>[VIDEO: Bag-valve-mask ventilation animated demonstration ]</th>
</tr>
</thead>
</table>

» Bladder catheterisation may be continuous or intermittent.

» Laxatives and bowel evacuation may be required.

» Passive and active range of motion is advised with prevention of pressure ulcers of the occiput, sacrum, and heels through manual or automatic turning every 2 hours.

**malignant spinal cord compression**

1st  

corticosteroids ± surgery ± radiation therapy  

**Primary options**

» methylprednisolone: consult specialist for guidance on dose  

OR  

» dexamethasone: consult specialist for guidance on dose  

» Treatment of spinal metastasis is largely palliative. Historically, treatment has consisted of corticosteroids, surgery (decompressive laminectomy/vertebrectomy), and radiation.[75] Evidence Corticosteroids provide relief from pain, reduce tumour-associated oedema, and may be oncolytic for some tumours.[76] There is no agreement on the type of corticosteroid or the dose intensity or duration.[77] [78]  

3B Evidence  

» Guidelines from the National Institute for Health and Care Excellence (NICE) recommend dexamethasone.[79]  

» Controversy exists between advocates of surgery alone and radiotherapy alone.[80] Some studies have found no difference for radiotherapy alone when compared to decompressive surgery.[81] Studies have reported that surgery followed by radiation is more effective than radiation alone in treating patients with spinal cord compression caused by metastatic cancer.[85] [86]  

4B Evidence The age of a patient may help guide the choice of therapy: outcome measures of ability to walk and survival are both affected by age. As age increases to
### Treatment

| Acute | 
|-------|----------------------------------|
| plus  | prevention of venous thromboembolism |

#### Primary options

- enoxaparin: 40 mg subcutaneously once daily
  - and-
  - compression stockings or pneumatic intermittent compression

#### Secondary options

- heparin: 5000 units subcutaneously every 8-12 hours
  - and-
  - compression stockings or pneumatic intermittent compression

#### Tertiary options

- IVC filter

- All patients should be given prophylaxis to prevent venous thromboembolism and possible pulmonary embolism. Treatment should begin no later than 72 hours following spinal cord injury. First-line treatment is low-molecular-weight heparin, with second-line treatment unfractionated heparin. Treatment duration is at the discretion of the clinician and based on patient degree of immobility.

- In patients with contraindications to anticoagulation, an inferior vena cava (IVC) filter can be used.

- Compression stockings and pneumatic intermittent compression devices may also be beneficial.

#### plus maintenance of volume and blood pressure

#### Primary options

- volume resuscitation
  - and/or-
  - dopamine: 1-50 micrograms/kg/minute intravenously, titrate gradually according to response

- Treatment of autonomic dysfunction and prevention of hypotension may be required, particularly in patients with cervical trauma. Hypotension may be associated with spinal shock, neurogenic shock, hypovolaemia, sepsis, bradycardia, or cardiogenic shock.
## Acute

Hypotension should be treated, as it contributes to neurological damage.

- Treatment consists of central line placement, volume resuscitation, and vasopressors. The aim is to maintain a blood pressure of >100 mmHg systolic and an adequate urine output (0.5 mL/kg/hour).
- Dose should be started low and titrated according to response.

### plus prevention of gastric stress ulcers

#### Primary options

- **omeprazole**: 40 mg orally once daily


- **cimetidine**: 300 mg orally/intravenously every 6 hours


- **famotidine**: 40 mg orally once daily; 20 mg intravenously every 12 hours

- Prevention of stress ulceration with proton-pump inhibitors or H2-antagonists may be indicated for 4 weeks following spinal cord injury or surgery.

### plus supportive therapies

- Nutritional support should include isotonic feeds and evaluation of dysphagia.

- Mechanically assisted ventilation or manually assisted cough may be required.

- Bladder catheterisation may be continuous or intermittent.

- Laxatives and bowel evacuation may be required.

- Passive and active range of motion is advised with prevention of pressure ulcers of the occiput, sacrum, and heels through manual or automatic turning every 2 hours.
<table>
<thead>
<tr>
<th>Acute epidural abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1st antibiotics ± surgery</strong></td>
</tr>
<tr>
<td><strong>Primary options</strong></td>
</tr>
<tr>
<td>« vancomycin: 15-20 mg/kg intravenously every 8-12 hours <strong>and</strong></td>
</tr>
<tr>
<td>« metronidazole: 500 mg intravenously every 6 hours <strong>and</strong></td>
</tr>
<tr>
<td>« cefotaxime: 2 g intravenously every 6 hours <strong>and</strong></td>
</tr>
</tbody>
</table>

The most common pathogen in epidural abscess is *Staphylococcus aureus*, although many other bacteria have been implicated, including *Streptococcus* and *Pseudomonas* species, *Escherichia coli*, and *Mycobacterium tuberculosis*. MRSA is increasingly reported, particularly in patients with spinal surgery or implanted devices.

**Primary options**

- Treatment usually involves surgery in combination with intravenous antibiotics. The single most important predictor of the final neurological outcome is the patient's neurological status immediately before surgery. Patients who present with back pain, low-grade fever, and no neurological signs may be candidates for antibiotics and close observation alone. Any progression of signs is an indication for re-evaluation of surgery.
- Identification and prompt treatment of the offending organism is paramount. Choice of antibiotic depends upon the results of microbiological culture and sensitivity. Antibiotic treatment is advised for at least 12 weeks. Empirical antibiotic therapy should be administered until culture and sensitivities are known. A typical empirical regimen would involve a combination of vancomycin, metronidazole, and cefotaxime.
- Surgery can be performed with open techniques, allowing for cord decompression as well as sampling of the tissues. For patients with less disabling symptoms, CT-guided needle aspiration of intra-discal/intra-bony lesions can be performed. Complications of surgery include progressive kyphosis and late neurological compromise.[94]

**plus prevention of venous thromboembolism**

**Primary options**
### Acute

- **enoxaparin**: 40 mg subcutaneously once daily
- **compression stockings or pneumatic intermittent compression**

### Secondary options

- **heparin**: 5000 units subcutaneously every 8-12 hours
- **compression stockings or pneumatic intermittent compression**

### Tertiary options

- **IVC filter**

All patients should be given prophylaxis to prevent venous thromboembolism and possible pulmonary embolism. Treatment should begin no later than 72 hours following spinal cord injury. First-line treatment is low-molecular-weight heparin, with second-line treatment unfractionated heparin. Treatment duration is at the discretion of the clinician and based on patient degree of immobility.

In patients with contraindications to anticoagulation, an inferior vena cava (IVC) filter can be used.

Compression stockings and pneumatic intermittent compression devices may also be beneficial.

**plus**  
**maintenance of volume and blood pressure**

### Primary options

- **volume resuscitation**
  - **dopamine**: 1-50 micrograms/kg/minute intravenously, titrate gradually according to response

Treatment of autonomic dysfunction and prevention of hypotension may be required, particularly in patients with cervical involvement with infection.

Treatment consists of central line placement, volume resuscitation, and vasopressors. The aim is to maintain a blood pressure of >100 mmHg systolic and an adequate urine output (0.5 mL/kg/hour).
### Spinal Cord Compression: Treatment

<table>
<thead>
<tr>
<th>Acute</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dose</strong></td>
<td>should be started low and titrated according to response.</td>
</tr>
</tbody>
</table>

**plus** prevention of gastric stress ulcers

**Primary options**

- **Omeprazole**: 40 mg orally once daily

**OR**

- **Cimetidine**: 300 mg orally/intravenously every 6 hours

**OR**

- **Famotidine**: 40 mg orally once daily; 20 mg intravenously every 12 hours

**Prevention of stress ulceration with proton-pump inhibitors or H2-antagonists may be indicated for 4 weeks following spinal cord injury or surgery.**

**plus** supportive therapies

- Nutritional support should include isotonic feeds and evaluation of dysphagia.

- Mechanically assisted ventilation or manually assisted cough may be required.

**[VIDEO: Tracheal intubation animated demonstration]**

**[VIDEO: Bag-valve-mask ventilation animated demonstration]**

- Bladder catheterisation may be continuous or intermittent.

- Laxatives and bowel evacuation may be required.

- Passive and active range of motion are advised with prevention of pressure ulcers of the occiput, sacrum, and heels through manual or automatic turning every 2 hours.
Emerging

Denosumab

A monoclonal antibody with a notable mechanism of action. It inhibits the receptor activator of nuclear factor-kB ligand (RANKL). RANKL is responsible for osteoclast differentiation, activation, and survival and so denosumab acts in part as an anti-resorptive agent; it was first approved for use in osteoporosis. The effect of altering this signalling pathway plays a role in its alteration of the immune system. Decreased osteoclast activity helps reduce the risk of bone-related events in patients with cancer that has spread to the bone. This may explain its effects on the bony metastatic process. Safety and efficacy were addressed in preliminary studies, showing bone mineral density was increased in drug-active patients. The recent accelerated reviews in both the US and European Union found evidence for the bone-saving effects in metastatic disease.[95] [96]

Zoledronic acid

This is a bisphosphonate that has been marketed primarily for osteoporosis, with the advantage of once-yearly dosing. When used in patients taking concomitant hormonal therapy for breast carcinoma, it was noted to demonstrate an additive effect on decreasing breast cancer relapses at bone or other sites. A 2017 Cochrane review concluded that in women with metastatic breast cancer and bone metastases, bisphosphonates reduce the risk of developing skeletal-related events, delay the median time to a skeletal-related event, and appear to reduce bone pain compared with placebo or no bisphosphonate. In women with advanced breast cancer without clinically evident bone metastases, the authors found no evidence of effect of bisphosphonates on bone metastases or overall survival compared with placebo or no bisphosphonates.[97]

Lidocaine infusion

Lidocaine is a local anaesthetic and a class 1b anti-arrythmic agent. There is growing interest in the role of lidocaine, administered as an infusion, for the management of systemic pain. Lidocaine has anti-inflammatory and anti-hyperalgesic properties. These may be related to its effects on N-methyl-D-aspartate (NMDA) receptors and G-coupled proteins. It is theorised that lidocaine decreases central sensitisation. There is research on the use of lidocaine in cancer-related pain. Lidocaine may have a role in the treatment of postoperative pain after spinal cord compression surgery.[98] [99] [100] [101] [102] [103]
**Recommendations**

**Monitoring**

Following acute trauma, the patient is monitored during the rehabilitation cycle for late conditions, such as spasticity in the affected limb(s), loss of functional level(s) due to syrinx, or bony misalignment.

Following treatment for malignancy, the patient is monitored for adverse effects of therapy.

For patients who have had epidural abscess, the patient's erythrocyte sedimentation rate (ESR) is monitored during the 12-week antibiotic course at least every 2 weeks. A rising ESR suggests relapse, intercurrent infection, or sequestrum of isolated bone/granulation tissue.

For patients who have had disc herniation, a life-long programme of muscle activity to maintain healthy core lumbar and pelvic girdle muscles is important to avoid continued degeneration and recurrence.

**Patient instructions**

All patients who have experienced spinal cord compression will need to be instructed in daily rehabilitation techniques (stretching, muscle use), bladder care (if applicable), bowel hygiene (if applicable), and skin surveillance for breakdown.

Bladder care involves regular emptying of the bladder, which may require intermittent clean catheterisation.

Bowel hygiene involves regular evacuation of the distal bowel, either manually or with enemas, or oral cathartics. In addition, the patient will need instruction on optimal fluid status for both means of elimination.

Skin surveillance and avoidance of breakdown in numb or insensate areas involves padding at pressure points, frequent position changes, and use of emollients.

Good communication between healthcare professionals and people with metastatic spinal cord compression (MSCC) is essential. Treatment, care, and support, as well as the information given about it, should be culturally appropriate. It should also be accessible to people with additional needs (such as physical, sensory, or learning disabilities) and to people who do not speak or read English. People with MSCC should have access to an interpreter or advocate if needed. Patients who experience MSCC should be instructed as to signs and symptoms that may herald a recurrence, as well as to steps that can be taken to ensure timely access to appropriate care in order to avoid further injury or loss.

**Complications**

<table>
<thead>
<tr>
<th>Complications</th>
<th>Timeframe</th>
<th>Likelihood</th>
</tr>
</thead>
<tbody>
<tr>
<td>pressure ulcers</td>
<td>short term</td>
<td>high</td>
</tr>
<tr>
<td>Loss of sensation and poor mobility combine to put pressure points at risk.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>discectomy-related complications</td>
<td>short term</td>
<td>medium</td>
</tr>
<tr>
<td>Complications</td>
<td>Timeframe</td>
<td>Likelihood</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------------</td>
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</tr>
<tr>
<td>These complications occur in 15% to 30% of cases and include haemorrhage, soft-tissue infection, nerve root injury, dural tear, recurrent or residual disc herniation, epidural scar formation, discitis, arachnoiditis, pseudomeningocoele, facet joint fracture (iatrogenic or stress related), spinal stenosis, and epidural haematoma. The risks of spine surgery are related to patient factors (weight, smoking, lifestyle) and surgical technique.[114]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>postoperative autonomic dysfunction</td>
<td>short term</td>
<td>medium</td>
</tr>
<tr>
<td>Autonomic function is transmitted in the anterior interomedial tract. The sympathetic nervous system fibres exit from the spinal cord between C7 and L1. The parasympathetic system nerves exit between S2 and S4. Therefore, progressively higher spinal cord lesions or injury cause increasing degrees of autonomic dysfunction. Symptoms suggesting autonomic dysfunction include orthostatic hypotension, heat intolerance, and loss of bladder and bowel control. Erectile dysfunction is an early symptom.[6]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>cardiovascular dysfunction</td>
<td>short term</td>
<td>medium</td>
</tr>
<tr>
<td>Well-recognised complications following acute spinal cord compression include persistent sinus bradycardia (including re-polarisation changes), atrioventricular blocks, supraventricular tachycardia, ventricular tachycardia, and primary cardiac arrest. These complications are related in part to loss of supra-spinal sympathetic control. This would typically be found in injuries to the upper thoracic (T-6) and cervical cord.[118] [119] [120]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>heterotopic ossification</td>
<td>long term</td>
<td>medium</td>
</tr>
<tr>
<td>The incidence of heterotopic ossification in patients with spinal cord injury (SCI) is between 16% and 53%. The incidence of clinically significant cases is between 18% and 27%. The pathophysiology involves an inflammatory process with increased blood flow in soft tissue. Bisphosphonates and/or surgery are reserved for cases with moderate pain or joint dysfunction.[112] [113]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>deep venous thrombosis</td>
<td>variable</td>
<td>high</td>
</tr>
<tr>
<td>In prospective studies, the incidence of deep vein thrombosis (DVT) following acute spinal cord injury (SCI) has been reported at between 18% and 100%, depending on the diagnostic technique used, time after injury, and concurrent risk factors.[61] Overall incidence without prophylaxis is estimated to be 40% based on meta-analysis of DVT in patients with acute SCI. Clinically apparent DVT occurs in approximately 15% and pulmonary embolism in approximately 5% of acute SCI patients. Hypercoagulability and stasis from paralysed muscles remain the 2 major factors contributing to the development of thrombosis in this patient population. Compression stockings, intermittent pressure devices, and low-molecular-weight heparin are the mainstays of both prevention and treatment.[61] [62]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>urinary tract infections</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>Patients with poor bladder emptying are at risk from developing retrograde influx of organisms and infection.[111]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pulmonary embolism</td>
<td>variable</td>
<td>low</td>
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</table>
Pulmonary embolism (PE) is estimated to occur in 5% of patients affected by spinal cord injury. However, due to the wide clinical variations in presentation and the clinical un-reliability of signs and symptoms in PE, that number is suspect. Signs that can be present include uni- or bilateral leg swelling (>3 cm in change), low-grade fever without obvious source, and pleural rub. Chest x-ray findings are quite non-specific, and the recommended diagnostic test is a contrasted helical CT of the chest. Treatment can include un-fractionated heparin, low-molecular-weight heparin, or fondaparinux. In patients with right heart strain or other haemodynamic compromise, the use of thrombolytic therapy is recommended.

MRSA infection
- Timeframe: variable
- Likelihood: low

MRSA is increasingly reported, particularly in patients with spinal surgery or implanted devices.

Prognosis

**Traumatic spinal cord compression**

The likelihood of recurrence after a traumatic spinal cord injury is estimated to be 2% to 5%, either due to spinal instability or because patients may continue with high-risk activities. Approximately 30% of paraparetic patients and 5% of paraplegic patients can be expected to retain or regain the ability to walk; 45% of patients require a urinary catheter before treatment, and only 21% of these patients subsequently become catheter-free.

**Malignant spinal cord compression**

Recurrence rates of malignant spinal cord compression range from 7% to 9%. Patients with multiple sites of metastasis at presentation are at highest risk.

**Intervertebral disc disease (cauda equina syndrome)**

Recurrence rates are reported as being from 5% to 15% but no single factor has been associated with same-level recurrence.

**Infection**

Recurrence, even after treatment, is not unusual.
## Diagnostic guidelines

### Europe

**Metastatic spinal cord compression in adults: risk assessment, diagnosis and management**

*Published by: National Institute for Health and Care Excellence  Last published: 2008*

## Treatment guidelines

### Europe

**Metastatic spinal cord compression in adults: risk assessment, diagnosis and management**

*Published by: National Institute for Health and Care Excellence  Last published: 2008*

### International

**An integrated multidisciplinary algorithm for the management of spinal metastases**

*Published by: International Spine Oncology Consortium  Last published: 2018*

### North America

**Palliative radiation therapy for bone metastases: update of an ASTRO evidence-based guideline**

*Published by: American Society for Radiation Oncology  Last published: 2016*

**Guidelines for the management of acute cervical spine and spinal cord injuries**

*Published by: Joint Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons  Last published: 2013*

**A 2011 updated systematic review and clinical practice guideline for the management of malignant extradural spinal cord compression**

*Published by: International Journal of Radiation Oncology  Last published: 2012*

**The role of bisphosphonates in the management of skeletal complications for patients with multiple myeloma**

*Published by: Cancer Care Ontario  Last published: 2012*
Evidence scores

1. Treatment of disc herniation: there is poor-quality evidence that epidural corticosteroids are effective in treating herniated discs.[72]
   **Evidence level C**: Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

2. Treatment of spinal breast cancer metastases: there is poor-quality evidence that radiotherapy improves outcomes, but timing of treatment is important as less than 10% of people walk again if severe deterioration of motor function occurs before radiotherapy is commenced.[75]
   **Evidence level C**: Poor quality observational (cohort) studies or methodologically flawed randomized controlled trials (RCTs) of <200 participants.

3. Treatment of spinal metastases: there is medium-quality evidence that adding high-dose corticosteroids to radiotherapy improves the chance of walking 6 months after treatment when compared with radiotherapy alone.[77]
   **Evidence level B**: Randomized controlled trials (RCTs) of <200 participants, methodologically flawed RCTs of >200 participants, methodologically flawed systematic reviews (SRs) or good quality observational (cohort) studies.

4. Treatment of spinal metastases: there is medium-quality evidence that surgery followed by radiation is more effective than radiation alone in treating patients with spinal cord compression caused by metastatic cancer.[85] [86]
   **Evidence level B**: Randomized controlled trials (RCTs) of <200 participants, methodologically flawed RCTs of >200 participants, methodologically flawed systematic reviews (SRs) or good quality observational (cohort) studies.
Key articles


References


<table>
<thead>
<tr>
<th>Reference</th>
<th>Details</th>
</tr>
</thead>
</table>


119. Collins HL, Rodenbaugh DW, DiCarlo SE. Spinal cord injury alters cardiac electrophysiology and increases the susceptibility to ventricular arrhythmias. Prog Brain Res. 2006;152:275-88. Abstract


Figure 1: Burst fracture of L1 vertebra due to axial load, causing compression of conus and cauda equina of the spinal cord; there is also a fracture in the body of L2.

From the collection of Kenneth F. Casey, MD, FACS; used with permission.
**Figure 2: Axial view of L1 burst fracture showing bone fragment occupying the spinal canal**

*From the collection of Kenneth F. Casey, MD, FACS; used with permission*
Figure 3: Compression from spinal column or epidural tumours, as shown, are often heralded by pain and spine deformity

From the collection of Kenneth F. Casey, MD, FACS; used with permission
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Contributors:

// Authors:

Kenneth F. Casey, MD, FACS
Clinical Associate Professor Surgery (Neurosurgery)
Michigan State University, Clinical Associate Professor (Physical Medicine and Rehabilitation), Wayne State University School of Medicine, Detroit, MI
DISCLOSURES: KFC declares that he has no competing interests.

// Peer Reviewers:

Marc Chamberlain, MD
Professor of Neurology
Moffitt Cancer Center and Research Institute, University of Washington, Seattle, WA
DISCLOSURES: MC is an author of several references cited in this topic.

Alexios G. Carayannopoulos, DO, MPH
Interventional Spine Physiatrist
Pain Medicine Specialist, Medical Director, Spine Center, Lahey Clinic, Burlington, MA
DISCLOSURES: AGC declares that he has no competing interests.

William A. Petri, Jr, MD, PhD, FACP
Chief and Professor of Medicine
Division of Infectious Diseases and International Health, University of Virginia Health System, Charlottesville, VA
DISCLOSURES: WAP declares that he has no competing interests.

Shuxun Hou, MD, PhD
Professor and Chief Physician
Orthopaedic Department, Clinic of the General Hospital of CPLA, Beijing, China
DISCLOSURES: SH declares that he has no competing interests.