

Emergency Neurological Life Support: Spinal Cord Compression (SCC)

Kristine H. O'Phelan · E. Bradshaw Bunney ·
Scott D. Weingart · Wade S. Smith

© Neurocritical Care Society 2012

Abstract Acute spinal cord compression (SCC) is the most serious of the diseases of the cord and should be accorded special attention in neurocritical care. Patients with SCC have a combination of motor and sensory dysfunction that has a distribution referable to one, or a few contiguous, spinal levels. Bowel and bladder dysfunction and neck or back pain are usually part of the clinical presentation but are not uniformly present. Because interventions are time-sensitive, the recognition and treatment of SCC was chosen as an ENLS protocol.

Keywords Myelopathy · Epidural infection · Epidural abscess · Paraplegia · Quadriplegia · Protocol

The ENLS suggested algorithm for the initial management of SCC is shown in Fig. 1. Suggested items to complete within the first hour of evaluating a patient with SCC are shown in Table 1.

K. H. O'Phelan (✉)
Department of Neurology, University of Miami, Miller School
of Medicine, Miami, FL, USA
e-mail: kophelan@med.miami.edu

E. B. Bunney
Department of Emergency Medicine, University of Illinois
at Chicago, Chicago, IL, USA

S. D. Weingart
ENLS Course Co-Chair, Division of ED Critical Care,
Mount Sinai School of Medicine, New York, NY, USA

W. S. Smith
ENLS Course Co-Chair, Department of Neurology,
University of California, San Francisco, CA, USA
e-mail: SmithW@neurology.ucsf.edu

Quadriplegia

Quadriplegia represents the most serious presentation of SCC. In an alert and responsive patient, quadriplegia indicates a cervical lesion. Depending on the level of the cervical lesion, there may also be respiratory failure. Pulmonary function tests such as negative inspiratory force or vital capacity may be helpful in assessing this status (see the *Acute Weakness* protocol for indications on intubation).

The patient's own assessment of his respiratory status by way of dyspnea is frequently accurate but becomes unreliable if the patient requires pain medication or sedation. When in doubt, the airway should be secured by intubation and assisted with ventilation, particularly if the clinical syndrome is rapidly progressing.

The degree of recovery is often fixed at 48–72 h after injury [1]. Therefore, if the history and physical exam narrow the possible etiology to infectious or metastatic processes, it is prudent to begin antibiotics or steroids if there will be a delay in establishing the cause of cord compression by imaging.

If cervical instability is suspected on the basis of spondylolisthesis, traumatic ligamentous injury or multiple destructive bone lesions in one spinal segment, particularly in the posterior elements, the patient should be placed in a cervical collar until imaging can determine cervical stability.

Emergent Transfer

Advanced imaging as well as specialists in neurology, neurosurgery, radiation oncology, and infectious diseases may be needed to insure the best possible outcome for a patient with SCC [2]. Emergency departments and other

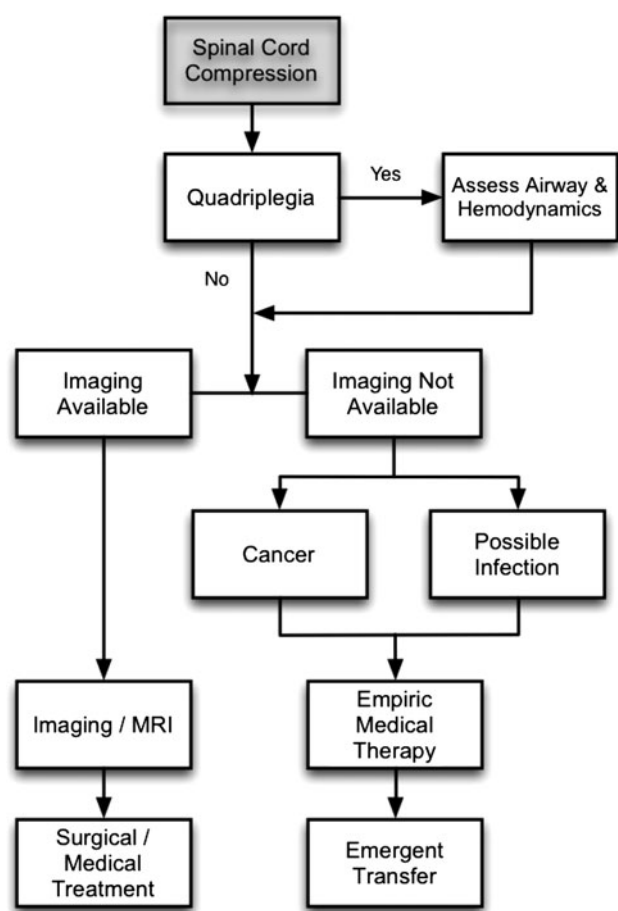


Fig. 1 ENLS spinal cord compression protocol

Table 1 Spinal cord compression checklist for the first hour

Quadriplegia? Insure proper ventilation
Attain emergent spine imaging (MRI unless contraindicated)
Alert spine surgeon if indicated
Labs: CBC, platelets, PT, PTT, creatinine
Suspected metastatic disease, contact radiation oncology; administer steroids if spinal metastasis with SCC is confirmed
Suspect epidural infection: ESR, start antibiotics

acute care facilities should have arrangements with tertiary care facilities that can provide these resources rapidly.

Distances between hospitals may be far enough to warrant transport via aircraft. In these cases, additional considerations such as landing areas for helicopters or ground transport to and from fixed wing aircrafts should be resolved well in advance and articulated in a pre-determined transportation plan. Finally, a contingency plan

should be established for use of ground transportation when weather conditions will not allow flying.

Whenever a delay in transfer or lengthy transport is anticipated, empirical therapy should be considered even if the diagnosis is not confirmed. Where an infectious cause is suspected, antibiotics should be started before transfer. If a metastatic lesion is suspected, steroids should be discussed with the accepting physician at the tertiary care facility and started if deemed appropriate.

Imaging Not Available

Some acute care facilities do not have magnetic resonance imaging (MRI) available continuously, or at all. In these cases, computed tomography (CT) may be helpful if a bony lesion is suspected. However, if a hematoma or neoplastic or infectious cause is suspected, CT may not provide adequate diagnostic information.

Therefore, an acute care facility that does not have around-the-clock MRI availability should have an arrangement with a facility that does. Owing to the rapid progression of some acute SCC syndromes, these transfer agreements should be pre-established so as to avoid prolonged attempts to find a facility.

Cancer

Patients with a known history of cancer and a high suspicion for metastatic disease to the spine with cord compression should be given high-dose corticosteroids during transfer to a center with MRI services (dosing is discussed below under the Acute Disk Herniation section).

Infection

Patients with evidence of infection such as fever, leukocytosis, intravenous (IV) drug use, or a known infectious source should be started on empiric antibiotics after blood cultures are drawn. Anti-microbial coverage should include *Staphylococcus*, *Streptococcus*, and methicillin resistant *Staphylococcus aureus* (MRSA). If there is a history of a recent neurosurgical procedure, coverage for gram negative organisms should be added. These empiric therapies may be coordinated with the accepting facility's physicians.

Imaging Available

Imaging of the spine is essential in identifying which compressive lesions may benefit from surgical treatment.

Magnetic resonance imaging (MRI) is the preferred technique because it demonstrates the surrounding supporting structures of the spinal column and the intrinsic aspects of the cord adjacent to the spinal lesion [3]. MRI can demonstrate abnormalities of the soft tissues around the spine and in the discrete regions (extradural, intradural extramedullary, and intramedullary) of the spinal canal itself. In addition, MRI allows the evaluation of long segments of the spine and spinal cord in a single examination.

The inclusion of a contrast enhanced study may be required to identify infectious, inflammatory, and neoplastic lesions. Myelography and CT may be used to identify traumatic and compressive lesions in patients who are not eligible for MRI.

The initial imaging studies should be oriented to begin at least two spinal segments above the level of the clinical deficit and continue down to the conus. Many patients will present with back pain without signs of a clear motor or sensory level or sphincter dysfunction [4–6]. For this reason, in patients with an active or recent cancer or a high suspicion of metastatic disease, the entire spine should be imaged.

Neoplastic Disease

In patients with metastatic SCC, motor function is the most important factor in determining outcome [4]. These lesions most frequently present in the thoracic spine (60 %) and less frequently in the lumbosacral (30 %) or cervical (10 %), reflecting the number of segments in each region and their blood supplies [5].

SCC can be the presenting symptom of cancer in 20 % of patients; in other words, an evident diagnosis of malignancy is not always present. Tumors of the lung, breast, and prostate, as well as lymphoma and renal cell cancer, are the most common neoplasms causing metastatic SCC. Metastatic disease occurs most often by hematogenous spread to the marrow of the vertebral bodies. There is contiguous spread from paraspinal tumors in less than 15 % of compressive metastatic lesions; this occurs particularly with Pancoast tumors and lymphomas. Compression of venous structures may also contribute to vasogenic edema and spinal cord infarcts [7].

Primary spinal cord tumors such as multiple myeloma, chordoma, chondrosarcoma, Ewing's sarcoma, and osteogenic sarcoma have also been associated with SCC though they represent only 10 % of cases of extradural SCC. Meningiomas and nerve sheath tumors are the most common intradural extramedullary lesions causing compressive symptoms, and ependymoma and astrocytomas are the most common intramedullary tumors.

Intramedullary spinal cord metastases can occur from primary lung neoplasm and breast cancer and rarely from lymphoma. Patients with intramedullary tumors can present with variants of a hemicord syndrome, which typically includes ipsilateral hemiplegia, loss of vibratory sense, and fine touch with contralateral loss of pain and temperature sensation [8].

Multiple cranial or spinal nerve dysfunction and signs of meningeal irritation suggest leptomeningeal metastases. This occurs in up to 8 % of cancer patients, and prognosis is poor [9]. Symptoms may include motor or sensory deficits in several non-contiguous sites in the central nervous system. Bowel and bladder involvement is less common. These patients often have abnormal spinal fluid studies that show an elevated protein level and pleocytosis with hypoglycorrhachia. Cerebral spinal fluid should also be sent for cytology in such cases.

A combination of radiotherapy and surgical treatment is recommended for metastatic SCC [10]. In addition, patients with leptomeningeal metastasis can be treated with intrathecal chemotherapy. Chemotherapy for tumors of the spinal column may also be reasonable for those with tumors known to be chemosensitive such as lymphoma, breast cancer, and neuroblastoma [11, 12].

Surgery may be particularly beneficial for patients with a single region of spinal compression or instability. Timely referral for surgical evaluation facilitates rapid alleviation of compressive neural damage and prevents further decline [13]. Though patients who are non-ambulatory at presentation have a shorter life expectancy, even those with significant disease at presentation may benefit from palliative surgical stabilization or radiation to mitigate symptoms [14, 15].

There are few useful studies, but some suggest that there is an advantage to surgical decompression within 24 h in patients who have lost the ability to walk or have rapidly declining ambulation. After that period, there may not be the same urgency, and the surgical efficacy has not been established. The overall prognosis for patients with metastatic SCC is poor: the one-year survival rate is 30 % [16]. However, older age is a prognostic factor and may favor radiation over surgery.

Patients without a previous history of cancer should have a complete blood count, sedimentation rate or CRP, electrolytes, liver function tests, serum light chains, immunoglobulins and electrophoresis, urinary Bence-Jones proteins, and a chest radiograph. CT scans of the chest, abdomen, and pelvis should be obtained, but should not delay treatment. Tumor markers are not specific enough to diagnose cancer but, if they are significantly elevated, may be used to suggest possible primary sites of malignancy [16].

Patients found at presentation to have compressive spinal lesions due to metastases and motor symptoms

should generally be treated with corticosteroids. While this therapy did not extend survival in several studies, it has consistently been associated with improvement in ambulation compared with placebo [17]. Whether there is an advantage to the massive doses in the range of 100 mg of dexamethasone used by some units is unknown. There is some evidence that high dose steroid regimens before radiation may have some clinical advantages, but this approach is also associated with a higher risk of complications including psychosis and gastric ulcers with hemorrhage [18]. Protein pump inhibitors are administered in parallel with corticosteroids.

Management of patients includes urinary catheterization for patients with urinary retention, hydration and attention to nutritional status, and prevention of pressure ulcers and thromboembolism. Prophylaxis is given for venous thrombosis with heparin or fractionated heparin. Analgesia to control pain and bowel regimens to prevent constipation are also fundamental to improving quality of life for these patients [19].

Acute Spinal Hemorrhage

Spinal hematomas can present with rapidly developing paraparesis or tetraparesis with local or radicular pain. The most common causes of non-traumatic spinal cord hematoma are vascular malformations, coagulopathy, inflammatory myelitis, spinal tumors, and syringomyelia [20, 22]. In rare cases, spinal hematoma can be a late complication after radiotherapy, presumably due to bleeding from telangiectasia caused by the radiotherapy [21].

MRI with and without gadolinium are used to evaluate the spinal hematoma, as it will demonstrate both the hematoma and any associated underlying pathology. Because this is relatively rare, the literature consists mainly of case reports and case series. There are little evidence-based data available to guide therapy, and most approaches are empiric.

There is considerable variation among surgeons regarding the timing of surgical intervention. Some surgeons choose to evacuate the clot early to relieve compression, while others believe it is best to allow the neurologic symptoms to plateau before surgical intervention to prevent further damage to viable tissue. Regardless of timing, the underlying cause must be addressed, particularly correction of coagulopathy.

Acute Disk Herniation

Back pain that is localized or radicular is often associated with disk herniation. This symptom is occasionally

accompanied by loss of sensory or motor function, which is usually referable to the radiculopathy.

Acute non-traumatic myelopathy from a herniated disk is rare, but has been reported [22]. The pain is typically worse when standing or sitting and better in the recumbent position. This is distinct from the pain associated with epidural tumors, which is typically worse when the patient lies down.

Acute myelopathy from disk herniations in the cervical and thoracic regions is caused by direct compression of the spinal cord as well as by compression of the blood supply to the cord. MRI will demonstrate the herniated disk as well as signal changes in the injured cord on T2 signal sequences.

Patients with acute neurologic deficits from disk herniation should have evaluation for surgical treatment. There is currently no consensus regarding recommendations for the timing of decompression or type of surgery recommended, but most experts operate quickly after signs of cord compression have become evident.

Steroids are often given to treat pain and inflammation in patients with disk herniations and radicular symptoms. In cases of severe acute deterioration in motor function with an upper motor neuron distribution, methylprednisolone may be given if started within the first 8 h. The prevailing dosage recommendation is 30 mg/kg IV bolus followed by 5.4 mg/kg/h by 23 h. However, this is a weak, level 2C recommendation based on evidence from the national acute spinal cord injury Study 2, a randomized controlled trial that has been criticized as flawed [23].

Infectious Lesion

Suppurative infections of the spinal epidural space can cause injury to the spinal cord through direct compression and also by the involvement of the vascular supply to the spinal cord. Diagnosis is often delayed because the initial symptom may only be back pain. The classic triad of fever, back pain, and neurologic deficit is not seen in most patients [24]. Symptoms may include localized back pain, radiculopathy, weakness, sensory changes, and sphincter dysfunction.

The mechanism of spread of bacteria is probably hematogenous; therefore, a history of IV drug use or an indwelling catheter is important. Direct spread of infection from vertebral osteomyelitis can occur. Most epidural abscesses occur in the posterior epidural space, which contains fat, small arteries, and the venous plexus. The anterior epidural space is more difficult to invade. *Staphylococcus aureus* is the most commonly reported pathogen though many other bacteria such as gram negative

organisms and *Mycobacterium tuberculosis* can cause a subacute spinal epidural abscess [25].

It may be difficult to distinguish infections from leptomeningeal metastases, particularly in immunosuppressed patients or those with lymphoma who may be susceptible to both. Patients with infections tend to develop cranial nerve and spinal abnormalities later in the course of illness as compared with patients with leptomeningeal neoplasm. Plain radiographs of the spine that show abnormalities involving two vertebral bodies across a disk space indicate an infectious process as it is rare for metastatic tumor to cross the disk space.

The work-up for suspected epidural abscess includes complete blood count (CBC), erythrocyte sedimentation rate (ESR), blood cultures, and preoperative lab studies. However, abnormalities of the CBC and ESR are non-specific: two-thirds of patients have a leukocytosis, and some may have a highly elevated ESR [24]. Spinal fluid studies may show elevated protein and leukocyte levels, but may also be within normal ranges. However, an ESR of less than 20 has excellent sensitivity for excluding a diagnosis of spinal epidural abscess [24].

Treatment of epidural abscess includes both medical and surgical therapies. Surgical decompression and drainage of the abscess is often indicated, and a surgical evaluation should be requested. Antibiotic coverage should include anti-staphylococcal coverage, including coverage for methicillin-resistant *Staphylococcus aureus*. If there is a history of recent neurosurgical procedure, antibiotic coverage should be broadened to include gram negative organisms (third generation cephalosporin and aminoglycoside) until culture results can definitively guide therapy. The recommendation to delay antibiotics until cultures are obtained in patients who have a mechanically stable spine and a stable neurologic examination is controversial [26].

CT guided aspiration of the epidural space is occasionally used to obtain cultures in patients without neurologic deficits. Cultures should be sent for aerobic and anaerobic bacteria, fungi, and tuberculosis. However, cultures may remain negative in 20–40 % of cases.

Steroids may be used during the course of treatment if the infection involves the spinal cord itself (intramedullary) to reduce associated spinal cord edema. Dexamethasone is most frequently used in doses ranging from 4–10 mg every 6 h.

Non-Compressive Acute Spinal Lesions

Patients with acute and severe symptoms of spinal cord dysfunction may have spinal cord infarct; inflammatory, infectious, and parainfectious myelitis; or demyelinating diseases. The leading causes of this syndrome are infarctive

Table 2 Spinal cord compression communication regarding assessment and referral

Airway status
Abnormal vital signs
Onset and duration of symptoms
Bowel or bladder involvement
Clinical spinal level of pathology
Results of any imaging
Coexistence of cancer, systemic illness
Which therapy has been started
Inquire which further therapy to start now

and are all uncommon; they are cartilaginous disk embolus, atherosclerotic disease of the aorta, and the ischemic effects of a dural arteriovenous fistula. These forms of spinal cord ischemia are difficult to diagnose and require further imaging with spinal angiography to identify arteriovenous malformation, and serum and cerebral spinal fluid (CSF) studies should usually be sent for diagnosis of infection and vasculitis. IgG index in CSF and serum as well as oligoclonal bands may be suggestive of an intrathecal infectious or inflammatory process (See the *Acute Non-Traumatic Weakness* protocol).

Normal Imaging

MRI that is unrevealing in a patient with acute disturbance of motor and or sensory function should prompt evaluation of the peripheral nerves and neuromuscular junction. Electrophysiologic studies may establish a diagnosis of acute demyelinating or axonal polyradiculopathy, myasthenia gravis, or Lambert Eaton syndrome or motorneuron disease (see the *Acute Non-Traumatic Weakness* protocol).

Communication

When communicating to an accepting or referring physician about this patient, consider including the key elements listed in Table 2.

References

1. Wuermsier LA, Ho CH, Chiodo AE, Priebe MM, Kirshblum SC, Scelza WM. Spinal cord injury medicine. 2. Acute care management of traumatic and nontraumatic injury. Arch Phys Med Rehabil. 2007;88:S55–61.
2. Schmidt RD, Markovchick V. Nontraumatic spinal cord compression. J Emerg Med. 1992;10:189–99.
3. Bammer R, Fazekas F. Diffusion imaging of the human spinal cord and the vertebral column. Top Magn Reson Imaging. 2003;14:461–76.

4. Husband DJ. Malignant spinal cord compression: prospective study of delays in referral and treatment. *BMJ*. 1998;317:18–21.
5. Klimo P Jr, Thompson CJ, Kestle JR, Schmidt MH. A meta-analysis of surgery versus conventional radiotherapy for the treatment of metastatic spinal epidural disease. *Neuro Oncol*. 2005;7:64–76.
6. Gilbert RW, Kim JH, Posner JB. Epidural spinal cord compression from metastatic tumor: diagnosis and treatment. *Ann Neurol*. 1978;3:40–51.
7. Bach F, Larsen BH, Rohde K, et al. Metastatic spinal cord compression. Occurrence, symptoms, clinical presentations and prognosis in 398 patients with spinal cord compression. *Acta Neurochir (Wien)*. 1990;107:37–43.
8. Schiff D, O'Neill BP. Intramedullary spinal cord metastases: clinical features and treatment outcome. *Neurology*. 1996;47:906–12.
9. Mut M, Schiff D, Shaffrey ME. Metastasis to nervous system: spinal epidural and intramedullary metastases. *J Neurooncol*. 2005;75:43–56.
10. Sundaresan N, Sachdev VP, Holland JF, et al. Surgical treatment of spinal cord compression from epidural metastasis. *J Clin Oncol*. 1995;13:2330–5.
11. Burch PA, Grossman SA. Treatment of epidural cord compressions from Hodgkin's disease with chemotherapy. A report of two cases and a review of the literature. *Am J Med*. 1988;84:555–8.
12. Boogerd W, van der Sande JJ, Kroger R, Bruning PF, Somers R. Effective systemic therapy for spinal epidural metastases from breast carcinoma. *Eur J Cancer Clin Oncol*. 1989;25:149–53.
13. Chen YJ, Chang GC, Chen HT, et al. Surgical results of metastatic spinal cord compression secondary to non-small cell lung cancer. *Spine (Phila Pa 1976)*. 2007;32:E413–8.
14. Patchell RA, Tibbs PA, Regine WF, et al. Direct decompressive surgical resection in the treatment of spinal cord compression caused by metastatic cancer: a randomised trial. *Lancet*. 2005;366:643–8.
15. Mannion RJ, Wilby M, Godward S, Lyraztopoulos G, Laing RJ. The surgical management of metastatic spinal disease: prospective assessment and long-term follow-up. *Br J Neurosurg*. 2007;21:593–8.
16. Walji N, Chan AK, Peake DR. Common acute oncological emergencies: diagnosis, investigation and management. *Postgrad Med J*. 2008;84:418–27.
17. Delattre JY, Arbit E, Rosenblum MK, et al. High dose versus low dose dexamethasone in experimental epidural spinal cord compression. *Neurosurgery*. 1988;22:1005–7.
18. Sorensen S, Helweg-Larsen S, Mouridsen H, Hansen HH. Effect of high-dose dexamethasone in carcinomatous metastatic spinal cord compression treated with radiotherapy: a randomised trial. *Eur J Cancer*. 1994;30A:22–7.
19. Loblaw DA, Perry J, Chambers A, Laperriere NJ. Systematic review of the diagnosis and management of malignant extradural spinal cord compression: the cancer care ontario practice guidelines initiative's neuro-oncology disease site group. *J Clin Oncol*. 2005;23:2028–37.
20. Rodesch G, Hurth M, Alvarez H, Tadie M, Lasjaunias P. Spinal cord intradural arteriovenous fistulae: anatomic, clinical, and therapeutic considerations in a series of 32 consecutive patients seen between 1981 and 2000 with emphasis on endovascular therapy. *Neurosurgery*. 2005;57:973–83; discussion 83.
21. Allen JC, Miller DC, Budzilovich GN, Epstein FJ. Brain and spinal cord hemorrhage in long-term survivors of malignant pediatric brain tumors: a possible late effect of therapy. *Neurology*. 1991;41:148–50.
22. Suzuki T, Abe E, Murai H, Kobayashi T. Nontraumatic acute complete paraplegia resulting from cervical disc herniation: a case report. *Spine*. 2003;28:E125–8.
23. Bracken MB, Holford TR. Effects of timing of methylprednisolone or naloxone administration on recovery of segmental and long-tract neurological function in NASCIS 2. *J Neurosurg*. 1993;79:500–7.
24. Mehta SH, Shih R. Cervical epidural abscess associated with massively elevated erythrocyte sedimentation rate. *J Emerg Med*. 2004;26:107–9.
25. Joshi SM, Hatfield RH, Martin J, Taylor W. Spinal epidural abscess: a diagnostic challenge. *Br J Neurosurg*. 2003;17:160–3.
26. Pradilla G, Ardila GP, Hsu W, Rigamonti D. Epidural abscesses of the CNS. *Lancet Neurol*. 2009;8:292–300.