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Malignant Spinal Cord Compression-Adapting Conventional Rehabilitation Approaches

Lisa Marie Ruppert, MD

Assistant Attending Rehabilitation Medicine Service-Memorial Sloan-Kettering Cancer Center;
New York, NY

Assistant Professor of Rehabilitation Medicine-Weill Cornell Medical College; New York, NY

Synopsis

Spinal tumors are classically grouped into three categories, extradural tumors, intradural extramedullary tumors and intradural intramedullary tumors. Spinal tumors may cause spinal cord compression and vascular compromise resulting in pain or neurological compromise. They may also alter the architecture of the spinal column, resulting in spinal instability. Oncologic management of spinal tumors varies according to the stability of the spine, neurological status and presence of pain. Treatment options include surgical intervention, radiation therapy, chemotherapy and hormonal manipulation. When combined with this management, rehabilitation can serve to relieve symptoms, improve quality of life, enhance functional independence, and prevent further complications in patients with malignant spinal cord compression

Keywords

Malignant Spinal Cord Compression; Neoplastic Spinal Cord Injury; Rehabilitation

Introduction

When combined with medical, radiation and surgical oncology care, rehabilitation can serve to relieve symptoms, improve quality of life, enhance functional independence, and prevent further complications in patients with malignant spinal cord compression.¹

Epidemiology and Pathophysiology

Spinal tumors are classically grouped into three categories, extradural tumors, intradural extramedullary tumors and intradural intramedullary tumors (Table 1).

Author Contact Information: Memorial Sloan Kettering Cancer Center, 515 Madison Avenue, 5th Floor, New York, NY10022, rupperl1@mskcc.org.

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Extradural Tumors

Extradural (epidural) tumors refer to lesions outside of the dura mater, in the vertebral bodies and neural arches. These tumors can be primary or secondary to metastatic disease and are more commonly malignant in nature.² Primary extradural tumors arise from osteoblasts, chondrocytes, fibroblasts and hematopoietic cells. The majority of extradural metastases occur through hematogenous spread.³ Direct extension of primary tumors may also lead to metastases in the spinal column. For example, prostate, bladder and colorectal cancers may become locally aggressive and invade the lumbar or sacral epidural space.⁴

Primary and metastatic lesions may be osteolytic, osteoblastic or mixed. Osteolytic lesions are more common in adults and with breast, lung and thyroid cancers. Osteoblastic lesions typically occur with prostate and bladder cancers and carcinoid tumors. Mixed lytic/blastic lesions can be seen with lung, breast, cervical and ovarian cancers.² Osteolytic lesions result in bone destruction greater than bone formation, whereas osteoblastic lesions result in bone deposition without breakdown of old bone first.²

Both osteolytic and osteoblastic lesions alter normal bone architecture and can result in deformity or collapse of the affected vertebral body. This deformity can lead to spinal instability by increasing strain on the support elements of the spine including muscles, tendons, ligaments and joint capsules.⁴ It can also result in retropulsion of fractured bone fragments into the epidural space causing spinal cord compression.⁵

Extradural lesions (Figure 1) may grow into the epidural space resulting in spinal cord compression. Epidural spinal cord compression (ESCC) results in mechanical injury to axons and myelin. ESCC also results vascular compromise of the spinal arteries and epidural venous plexus, leading to spinal cord ischemia and/or infarction.

Depending on the underlying malignancy, 2–5% of patients will develop clinical signs and symptoms of ESCC during the course of their disease.³ ESCC is most commonly diagnosed in thoracic lesions; though cadaveric studies have shown that the most common site of tumor burden in the spine is the lumbar region.⁵

Intradural Extramedullary Tumors

Primary intradural extramedullary tumors are located within the dura mater but outside of the spinal cord parenchyma. These tumors arise from peripheral nerves, nerve sheaths and sympathetic ganglion. They are most commonly benign. Extramedullary metastases (Figure 2), often referred to as leptomeningeal disease (LMD), are a relatively common complication of cancer, occurring in 3–8% of all patients. The most common site of involvement is the dorsal aspect of the spinal cord, particularly at the level of the cauda equina.²

Metastatic disease is thought to reach the leptomeninges through hematogenous spread, CSF seeding, and direct extension. CSF seeding can occur spontaneously or as a byproduct of surgical resection. Direct extension can occur along the epineurium or perineurium of spinal nerves or along veins exiting the vertebral body bone marrow.^{2,6}

Similar to extradural lesions, intradural extramedullary lesions can result in spinal cord compression and vascular compromise. Vascular compromise in this setting can result not only in ischemia, but in spinal subarachnoid hemorrhage. The risk of hemorrhage is greatest in patients receiving anticoagulation therapy.²

Intradural Intramedullary Tumors

Primary intradural intramedullary tumors are located within the spinal cord parenchyma (Figure 3) and arise from glial cells, neuronal cells, and other connective tissue cells. These tumors account for 4–5% of all primary central nervous system tumors, and are mostly benign.^{2,5} Intramedullary tumors are classified as low, intermediate or high grade based on cytology. They can be found in any region of the spinal cord, although cervical and cervicothoracic segments are slightly more favored.²

Intramedullary spinal cord metastases (ISCM) are diagnosed in less than 1% of patients with cancer.³ Metastatic lesions reach the intramedullary space either by hematogenous spread or via direct extension along leptomeninges and nerve roots, or through the Virchow-Robin spaces.³

ISCM usually occurs in the setting of extensive metastatic disease and is rarely the first manifestation of systemic malignancy.³ Metastatic lesions can be seen throughout the spinal cord usually as a solitary lesion. The cervical spinal cord, a vascular rich area, is the most common site of involvement.²

It is generally thought that intramedullary lesions cause neurological injury through direct compression of the surrounding spinal cord and vascular compromise².

Clinical Manifestations

Extradural Tumors

Pain is the most common initial symptom in patients with ESCC (89–90%) and may precede the development of other neurological symptoms by weeks to months. Three classically defined types of pain in the setting of extradural involvement are local, mechanical and radicular pain. An individual with epidural involvement may be affected by one or more of these pain types.⁴

Localized pain is thought to be the result of periosteal stretching and inflammation caused by tumor growth. It is characterized as a deep gnawing or aching pain. It is often nocturnal and improves with activity and antiinflammatory medications.⁴

Mechanical pain varies with position or activity, is indicative of impending or established spinal instability, and characteristically occurs with transitional movements or axial loading of the spine. This pain may also be elicited by lying prone or supine, particularly in the thoracic spine. Unlike localized pain, mechanical pain is often refractory to antiinflammatory agents. Mechanical pain responds well to stabilization of the spine with bracing or surgical fixation.^{3,4}

Radicular pain occurs in the setting of nerve root compression and is often described as sharp, shooting or stabbing in nature. In the thoracic region, radicular pain is typically bilateral and described as a tight band around the chest or abdomen. In the cervical and lumbar regions, it is usually unilateral, radiating to the upper or lower extremity respectively.^{3,4}

Motor weakness results from dysfunction of pathways that include the anterior horn cells and mediate movement⁷. It is the second most common symptom of ESCC, and is present in 35–85% of patients with metastases at the time of presentation. This weakness may be upper motor neuron, lower motor neuron or a combination of both depending on the area of the cord involved.⁴

Sensory impairments are usually present at the time of diagnosis (60%) in individuals with ESCC, but are rarely the initial symptom. The pattern of impairment is dependent upon the spinal pathway involved. Involvement of the lateral spinothalamic tract reduces pain and temperature perception on the contralateral side of the body, one or two dermatomes below the level of the lesion, but rarely causes paresthesias. Bilateral lesions may effect erection, ejaculation, and orgasm.⁷

Dorsal column involvement results in loss of proprioception, vibration and touch from the ipsilateral body and information about visceral distension, and may result in paresthesias. Individuals may experience limb or gait ataxia as the result of their proprioceptive loss. Lhermitte's phenomenon, an electric shock sensation that extends into the back and sometimes the limbs with changes in head or neck position, is frequently noted with dorsal column involvement in cervical and upper thoracic lesions.^{3,7}

Autonomic symptoms include bowel, bladder and sexual dysfunction, loss of sweating below the lesion and orthostatic hypotension. They are unusual as an initial symptom, but are often present at the time of diagnosis. Autonomic symptoms usually correlate with the degree of motor involvement.^{3,4}

Gait and truncal ataxia mimicking cerebellar involvement can be seen. This ataxia likely results from compression of the spinocerebellar tracts and can be differentiated from cerebellar lesions by the absence of dysarthria and nystagmus.³

Unusual signs of ESCC include the eruption of herpetic zoster at the level of cord compression, Horner's syndrome with C7-T1 involvement and neuropathic facial pain with high cervical ESCC involving the descending fibers of the trigeminal-thalamic tract.³

Intradural Extramedullary Tumors

Primary extradural tumors and Leptomenigeal disease present in a similar fashion to ESCC but with a higher incidence of neurological impairments. Approximately 70–90% of individuals will have pain as an initial symptom. This pain may be axial and/or radicular, and worsened by recumbency. More than 60% of individuals undergoing surgical resection for LMD have some degree of weakness. Motor deficits may manifest in the absence of pain.²

Almost all patients will have some degree of sensory involvement. Bowel, bladder and sexual dysfunction have been noted in 30–80% of individuals with LMD, usually as an early finding. LMD may present with radiculopathy, neuropathy or in a Brown-Séquard, conus medullaris or cauda equina pattern.⁷

Intramedullary Tumors

Intramedullary tumors may present with clinical manifestations surprisingly similar to epidural tumors. Pain, the most common initial symptom (30–85%), may be described as radicular, posterior midline, dull, aching and/or as paravertebral tightness and stiffness.²

Neurological deficits are common and usually involve cord segments below the level of the tumor. Overall, more than 92% of patients have some degree of motor deficit on examination. Sensory deficits are seen in 62–87% and bowel and/or bladder dysfunction in about 70% of patients.²

The constellation of motor and sensory findings indicative of Brown-Séquard syndrome is seen in 6–22% of patients. About 4% will have evidence of a Horner's syndrome. Other patterns that can be seen in individuals with intramedullary tumors include central cord syndrome, conus medullaris syndrome, and cauda equina syndrome.^{2,7}

Spinal Instability

Spinal instability is defined as a loss of spinal integrity as a result of a neoplastic process that is associated with movement related pain, symptomatic and progressive deformity and/or neurological compromise under a normal physiologic load. Factors considered when evaluating the structural stability of the spinal column include location of the lesion (Table 3), spinal alignment of the involved segment, extent of vertebral body involvement, involvement of posterior elements, bone lesion quality, overall bone mineral density, presence of multilevel contiguous and non contiguous lesions, previous surgical intervention, cancer treatments such as radiation therapy and hormonal manipulation, degenerative changes, and presence of mechanical pain.⁸

Various scoring scales are available to assess spinal stability; the Spine Instability Neoplastic Score is the most commonly used (Table 4).⁹

Diagnosis

Patients with suspected spine or spinal column involvement require a thorough diagnostic work up, including a history and physical examination. History taking should include inquiries about smoking history, environmental or occupational exposures to carcinogens, travel history, recent screening examinations and familial cancers.⁴

Physical examination should include an assessment of strength, sensation, reflexes and sphincter function. The International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) can be used as a guide for completing this examination but not to determine prognosis.⁹

Imaging

Magnetic resonance imaging (MRI) is considered the gold standard for assessing spinal involvement. MRI's resolution allows for accurate anatomical assessment of the soft tissue structures in the spine, including the intervertebral discs, spinal cord, spinal nerve roots, meninges, spinal musculature and ligaments. Plain films are a useful screening test to identify lytic or sclerotic lesions, pathological fractures, spinal deformities and large masses. Computerized tomography scans (CT) provide highly detailed imaging of the osseous anatomy of the spine and degree of tumor involvement. The addition of myelography allows for assessment of spaces occupied by neural elements and identification of compressed structures. In addition to CT of the spine, patients with suspected metastatic disease should have imaging of the chest, abdomen and pelvis to establish the extent of disease or identify the primary tumor.⁴

Serologies and other tests

Blood cell counts, chemistries and cancer specific laboratory testing such as prostate specific antigen (PSA), breast cancer genes 1 and 2 (BRCA1 and 2), carcinoembryonic antigen (CEA) and serum and urine protein electrophoresis should be examined based on clinical suspicions.⁴ Biopsy of an epidural lesion should be considered in patients without a prior history of cancer, unknown primary, or history of only limited stage or "cured" malignancy. Lumbar puncture for CSF analysis can be performed upon completion of neuraxial imaging in patients with intradural involvement.³

Oncologic Management

Management of spinal tumors varies according to tumor type, treatment history, spinal stability, neurological status and pain intensity. Treatment options include surgical intervention, radiation therapy, chemotherapy and hormonal manipulation.⁵ Indications for surgical intervention include paraplegia lasting more than 12–24 hours in patients with prior radiation to the spine, spinal instability, and bony compression of the spinal cord. The primary goals for surgical management are to preserve neurological function, reduce pain, and ensure mechanical stability.^{5,10}

Surgical intervention has risks. Potential complications include instrument failure, respiratory complications, deep venous thrombosis, CSF leak, wound infection and/or dehiscence and worsening of neurological symptoms from vasogenic edema.⁵

Radiation therapy plays an important role in pain relief, prevention of pathological fractures, and stabilization of neurological function. Radiosensitive tumors include myeloma, lymphoma and solid tumors such as prostate and breast. Relatively radioresistant tumors include sarcoma and renal cell carcinoma.¹¹

Unfortunately radiation may also cause adverse effects including gastrointestinal toxicity, mucositis, bone marrow suppression and radiation induced myelopathy. Radiation myelopathies, although uncommon, can be seen with radiation treatment of primary spine/spinal cord tumors, prophylactic radiation to prevent metastases, and when the spinal cord is included in the field of radiation such as with colorectal cancers.^{3,11}

Radiation myelopathies are generally divided into four subtypes which include acute complete paraplegia/tetraplegia, lower motor neuron disease, acute transient radiation myelopathy and chronic progressive radiation myelopathy. Acute complete radiation myelopathy is rare and presumed to be related to radiation induced vascular damage resulting in spinal cord infarction. Lower motor neuron disease is extremely rare and presumed to result from anterior horn cell damage.^{3,12}

Acute transient radiation myelopathy (ATRM) is the most common form. ATRM typically occurs 1–29 months after completion of radiation therapy and is hypothesized to result from demyelination of the dorsal columns. ATRM is generally associated with cervical spine irradiation, but can occasionally be seen in other cord segments. Clinical manifestations include Lhermitte's sign without neurological changes on examination. Treatment is reassurance as symptoms resolve over weeks-months.^{3,12}

Chronic progressive radiation myelopathy (CPRM) occurs 9–15 months after radiation therapy. It is the most feared form and has been reported in 1–5% of patients who survive 1 year post treatment. CPRM is classically characterized by a latent period during which the patient is asymptomatic. Clinical onset is usually painless and insidious. Manifestations include ascending weakness, diminished sensation and clumsiness. A Brown Séquard pattern of deficits has also been described. Literature suggests a steady progression of neurological deficits over the course of weeks to months.^{3,10,12}

The Pallis criteria for the diagnosis of CPRM states that the spinal cord must have been included in the field of radiation therapy, the main neurological deficit must be within the segment of the cord exposed to radiation, and that metastases or other primary cord lesions must be ruled out.¹²

There is no effective treatment. Corticosteroids are often tried with varying results. Anticoagulation and hyperbaric oxygen have occasionally been noted to improve or stabilize symptoms. Bevacizumab has shown benefit anecdotally.³

Chemotherapy is considered in the setting of highly chemo-sensitive tumors such as lymphomas, neuroblastomas and germ cell tumors. It can be used as an adjuvant therapy for metastatic disease from breast and prostate cancers and melanoma. Spinal metastases in the setting of breast and prostate cancer are also often sensitive to hormonal manipulation. However, for most patients, chemotherapy plays a limited role largely because of the slow and unpredictable response of the tumor and the urgent need to decompress the spinal cord.^{5,11}

Chemotherapy induced myelopathy is an exceedingly rare complication most associated with chemotherapeutic agents administered directly into the CSF. The exact pathogenesis is unknown, but ascending paresthesias, weakness and sphincter dysfunction have been reported. Lhermitte's phenomenon has been noted after IV administration of Cisplatin and reflects injury to the dorsal root ganglion. This symptom is usually transient, although patients may be left with sensory ataxia after multiple cycles. There is no definitive treatment.³

Corticosteroids remain part of the initial treatment for spinal tumors. They reduce tumor and spinal cord vasogenic edema resulting in improvement or at least stabilization of neurological deficits while definitive treatment is initiated. Corticosteroids also provide analgesia for pain and have direct cytotoxic effects on lymphoma and melanoma.³

Significant variability exists with regard to initial dose and tapering schedule. Noted side effects from steroids include hyperglycemia, increased risk of infection, gastrointestinal irritation, mood disturbances, fluid retention, impaired wound healing and steroid myopathy.^{3,5}

Bisphosphonates, which inhibit osteoclast activity and suppress bone reabsorption associated with spinal metastases, have been proven effective in reducing the risk of pathological fractures, relieving pain and reducing malignancy associated hypercalcemia in metastatic breast cancer, multiple myeloma, and other cancers that produce osteolytic metastases.¹¹

Rehabilitation

Benefits of rehabilitation in traumatic spinal cord injury are well established. Studies in neoplastic spinal cord injuries have shown similarly positive results and indicate a complementary role to oncologic management for this patient population.¹

The demographic profiles (Table 2), mechanism of injury and medical comorbidities of neoplastic spinal cord injuries may differ from traumatic injuries, but similar principles of neuro-rehabilitation can be applied. These principles aim to relieve symptoms, prevent further complications, enhance functional independence and improve quality of life.¹

Symptomatic Treatments

Pain is reported to be one of the most common symptoms. Determining the etiology of pain is pivotal to management. Several options are available for treatment including postural bracing (for non-surgical or residual mechanical pain), medications and modalities.⁵

Postural bracing can be achieved through custom and commercial orthotics and can accommodate all regions of the spine. Care should be taken when evaluating spinal alignment at involved segments with and without brace donned to ensure appropriate correction is achieved, involved segment is included within the brace, and brace is tolerated. The least restrictive brace appropriate for the patient should be chosen to achieve stability while preventing further muscle weakness.

Medication management for pain may include steroids, non-steroidal anti-inflammatories, anti-convulsants, tricyclic antidepressants and opioids⁵. Potential side effects, in the context of an individual's medical and functional status, are a key consideration for use. Physical modalities including heat, cold, ultrasound and electrical stimulation may be incorporated into pain management. Caution must be used with application over areas with sensory loss. Also use caution with modalities that promote increased blood flow as there is a potential risk of disease spread.⁵

Spasticity, Bowel/ Bladder and Sexual Dysfunction

Spasticity is a common complication of upper motor neuron lesions. It may prove to be beneficial for mobility and performance of ADLs, but may also cause pain and interfere with hygiene and function. Patients should be educated on possible benefits of spasticity and functional use of tone promoted during physical and occupational therapies. Management options for negative symptoms of spasticity include continuous passive stretching exercises, stretching splints and oral agents such as baclofen, tizanidine and benzodiazepines. Intrathecal baclofen and localized treatments such as phenol and botulinum toxin can also be considered.

Bladder dysfunction can result in difficulty with urinary drainage and abnormalities in intravesicular pressure, increasing risk for infections, renal disease, skin breakdown, and social embarrassment. Bladder symptoms can range from frequency and urgency to complete urinary retention. A thorough neurological examination (including sphincter function and reflexes), voiding diary, measurement of post void residual volumes and urodynamic studies can be used to assess an individual's bladder pattern and aid in establishment of a bladder program.

Intermittent catheterization or indwelling catheters can be used for both upper motor neuron (detrusor over activity with or without sphincter dyssynergia) and lower motor neuron bladder patterns (detrusor hypocontractility or a-contractility). Of note, caution must be taken in individuals with neutropenia or severe thrombocytopenia as they are at risk for infection and bleeding. Anticholinergic agents can be considered in patients with upper motor neuron patterns where as cholinergics and manual techniques such as Credé and double void considered in patients with lower motor neuron patterns. Depending on severity of injury, pelvic floor physical therapy may be considered for sensory retraining, pelvic muscle/sphincter coordination, and biofeedback.

Bowel dysfunction can result in social inconvenience, embarrassment, and skin compromise. As with the bladder, a thorough neurological assessment can help establish an individual's bowel pattern. Stool diaries, stool studies and abdominal imaging may also be helpful. Once a pattern is established, a bowel program can be initiated to allow for control over time and place of bowel movements with desired frequency and without incontinence. Medications such as stool softeners, oral stimulants and contact irritants (suppositories) can be initiated for upper motor neuron (hyperreflexic) bowel patterns. Digital stimulation can also be used in place of suppository. Lower motor neuron (hyporeflexic) bowel patterns can be managed with oral bulk forming agents and manual removal of stool. Pelvic floor therapy may be considered for incomplete injuries. Caution is necessary with digital stimulation, suppository use and manual removal in patients with neutropenia or severe thrombocytopenia.

Sexual dysfunction may be the result of the spinal cord injury, the primary cancer, attendant mood disorders, or side effects of treatment. Management should be based on the underlying cause of dysfunction. Options for sexual dysfunction related to the spinal cord injury include education, oral medications, intracavernous injection therapy and assistive devices (vacuum device and prosthesis) pending level of injury.⁵

Preventing Further Complications

Loss of sensation and mobility, bowel and bladder dysfunction, the catabolic state of cancer and malnutrition can place a patient at risk for skin breakdown. Pressure ulcers are preventable and maintenance of skin integrity is vital. Education should include techniques for pressure relief, skin hygiene and importance of nutrition.⁵ Formal wound care should be initiated if breakdown occurs.

Weakness, extradural tumor involvement, hormonal manipulation and radiation exposure place a patient at risk for spinal instability even after surgical intervention. To minimize the load on the spinal column, spinal precautions should be placed on activities. These may vary based on region of the spine involved, but generally include no excessive flexion or extension, twisting movements or lifting more than 10lbs. The duration of these spinal precautions is dependent on an individual's disease status and response to oncologic treatment. Use of bracing and physical therapy for core strengthening and postural training may also help to prevent instability.

Enhancing Functional Independence

Determination of neurological level of injury, severity of injury, oncologic prognosis and patient/caregiver expectations is essential to establishing realistic rehabilitation goals and determining the appropriate setting for rehabilitation efforts.

Physical and occupational therapy play a large role in these efforts. Based on neurological and functional status, focus is placed on strengthening exercises, range of motion, sensory reintegration, transfer training, balance, wheelchair mobility, gait training, activities of daily living and assessment for appropriate assistive devices. If indicated, upper and lower extremity bracing can be used to provide functional positioning, joint stability, compensation for weakness and proprioceptive feedback.

During the rehabilitation course, it is important to monitor for medical comorbidities related to cancer and its treatment. These comorbidities include fatigue, cytopenias, electrolyte disturbances, vitamin deficiencies, depression, infection and deep venous thrombosis.⁵ They may require supportive care and modification of the rehabilitation care plan.

Improving Quality of Life

Studies have shown that rehabilitation efforts in individuals with malignant spinal cord compression improve function, mood, pain levels, quality of life and survival. Incomplete injuries with the most neurological deficits are found to benefit most.^{1,5,13}

Conclusion

As survival rates for individuals with spinal tumors improve, it becomes even more important for clinicians to be aware of the potential long term neurological impact of these tumors and their treatments. It is also important for clinicians to understand how to apply rehabilitation principles and practices to this patient population.

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Key Points

- Spinal tumors are classically grouped into three categories: extradural tumors, intradural extramedullary and intradural intramedullary tumors
- Localized spine pain is the most common symptom in patients with epidural spinal cord compression at time of diagnosis
- Motor weakness is the second most common symptom in patients with epidural spinal cord compression at time of diagnosis
- Management of spinal tumors varies according to the stability of the spine, neurological status and pain. Treatment options include surgical intervention, radiation therapy and systemic treatments such as chemotherapy and hormonal therapy.
- Principles of neuro-rehabilitation applied to patients with traumatic spinal cord injury are equally appropriate for patients with spinal tumors.

Figure 1.
Extradural Tumor with Epidural Spinal Cord Compression

Figure 2.
Intradural-Extramedullary Lesion

Figure 3.
Intradural-Intramedullary Tumor

Table 1**Tumors of the Spine**

Extradural Spinal Tumors	
<i>Primary Malignant Tumors</i>	<i>Sources of Epidural Metastases</i>
	<u>Adult</u>
Lymphoma	Prostate Cancer
Osteosarcoma	Breast Cancer
Ewing sarcoma	Lung Cancer
Chondrosarcoma	Thyroid Cancer
Chordoma	Non-Hodgkin's Lymphoma
Sacrococcygeal Teratoma	Hodgkin's disease
Malignant Fibrous Histiocytoma	Multiple Myeloma
Solitary Plasmacytoma	Renal Cell Carcinoma
Fibrosarcoma	Colorectal Cancers
	Sarcoma
	Germ Cell Tumor
<i>Primary Benign Tumors</i>	Unknown Primary
Vertebral Hemangioma	<u>Pediatrics</u>
Giant Cell Tumor	Sarcoma- <i>Primarily Ewing's Sarcoma</i>
Osteochondroma	Neuroblastoma
Osteoid Osteoma	Germ Cell Tumors
Osteoblastoma	Hodgkin's disease
Intradural Extramedullary Tumors	
<i>Primary Malignant Tumors</i>	<i>Sources of Leptomeningeal Disease</i>
Malignant nerve sheath tumors	Glioblastoma
Hemangiopericytoma	Central Nervous System Lymphoma
	Leukemia
	Lymphoma
<i>Primary Benign Tumors</i>	Breast Cancer
Meningioma	Lung Cancer
Schwannoma	Melanoma
Neurofibroma	
Paraganglioma	
Ganglioneuroma	
Intradural Intramedullary Tumors	
<i>Primary Tumors</i>	<i>Sources of Intramedullary Metastases</i>
Ependymoma	Lung Cancer
Myxopapillary Ependymoma, Subependymoma	Breast Cancer
Astrocytoma	Melanoma
Hemangioblastoma	Lymphoma
Cavernous Angioma	Renal Cell Carcinoma

Intradural Intramedullary Tumors

Neurocytic Tumors

Oligodendroglioma

Embryonal Neoplasm

Lipoma

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Table 2

Traumatic versus Neoplastic Spinal Cord Injury-Patient Demographics

	Traumatic SCI	Neoplastic SCI
Gender	M>F	M=F
Age	16–30	50–70
NLI	Tetraplegia = Paraplegia	Paraplegia > Tetraplegia
Severity	Complete = Incomplete	Incomplete > Complete

Table 3

Regions of the Spine

Region	Segments	Articulations	Risk for Instability
Junctional Spine	Occiput-C2 C7-T2 T11-L1 L5-S1		*Highest risk for instability, subject to translational forces and unique blood supply characteristics
Mobile Spine	C3–C6 L2–L4		*High risk for instability
Semi-rigid Spine	T3–T10	Articulation with rib cage	*Ribs provide biomechanical protection against instability
Rigid Spine	S2–S5	Articulation with pelvis	*Pelvis provides biomechanical protection against instability

Table 4

Elements of the SINS Criteria

Element of SINS	Score
<i>Location</i>	
Junctional Spine (occipit-C2, C7-T2, T11-L1, L5-S1)	3
Mobile Spine (C3–C6, L2–L4)	2
Semi-Rigid Spine (T3–T10)	1
Rigid Spine (S2–S5)	0
<i>Pain relief with recumbency and/or pain with movement/loading of the spine</i>	
Yes	3
No (occasional pain but not mechanical)	1
Pain free lesion	0
<i>Bone Lesion</i>	
Lytic	2
Mixed (lytic/blastic)	1
Blastic	0
<i>Radiographic spinal alignment</i>	
Subluxation/translation present	4
De novo deformity (kyphosis/scoliosis)	2
Normal alignment	0
<i>Vertebral body collapse</i>	
>50% collapse	3
<50% collapse	2
No collapse with >50% vertebral body involved	1
None of the above	0
<i>Posterolateral involvement of the spinal elements (facet, pedicle or costovertebral joint fracture or replacement with tumor)</i>	
Bilateral	3
Unilateral	1
None of the above	0

SINS SCORE

Score	
0–6	Stable spine
7–12	“Indeterminate” (possible impending) instability
13–18	Instability

* The SINS score is generated by tallying each score from the 6 individual elements

* SINS scores from 7–18 warrant surgical consultation