

Spinal Tumours

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Abstract: Spinal tumours arise from a wide spectrum of different tissues. Primary spinal tumours originate from the spinal cord or the vertebral segment. Metastatic tumours of the spine are secondary tumours of the spine that metastasise to or reach these spinal regions from a distant area. Intramedullary neoplasms arise from glial and support cells in the spinal cord; extramedullary neoplasms originate from peripheral nerve roots. Between 4% and 16% of all adult central nervous system (CNS) tumours are primary neoplasms of the spinal cord. Dumbbell tumours are tumours that may be located in various compartments. Intradural–intramedullary neoplasms and dumbbell neoplasms make up 18% and 22%, respectively, of all primary spinal cord neoplasms; intradural–extramedullary tumours account for 54% of all such neoplasms. Meningioma and schwannoma are the most common intradural–extramedullary spinal tumours. Glioma, ependymoma, cavernoma, and haemangioblastoma are frequent intramedullary tumours. Clinical features include pain, weakness (paresis/paralysis), and sphincter dysfunction. Contrast MRI and CT scan of the spine are the right imaging modalities in most cases. Microsurgical treatment is the definitive treatment. This chapter briefly discusses the management of common and rare spinal tumours, including meningioma, schwannoma, astrocytoma, ependymoma, dumbbell tumours, and metastatic tumours.

Abbreviations

ABC	aneurysmal bone cyst	CNS	central nervous system
CT	computed tomography	CSF	cerebrospinal fluid
DWI	dissemination-weighted imaging	EG	eosinophilic granuloma
GCT	giant-cell tumour	IDEM	intradural–extramedullary
IM	intradural–intramedullary	IMSCT	intramedullary spinal cord tumour
MPNST	malignant peripheral nerve sheath tumour	NF	neurofibromatosis type 1
NST	nerve sheath tumour	NF2	neurofibromatosis type 2
NOMS	neurological, oncological, mechanical, systemic	PCNSL	primary CNS lymphoma
SEC	spinal extraosseous chordoma	STIR	short TI inversion recovery

1. Epidemiology of Spinal Tumours

Spinal and spinal cord tumours arise from a wide spectrum of different tissues, like bone, nerves, soft tissues, and blood vessels. These tumours should be assessed with very different preoperative treatment strategies according to the benign or malignant behaviour of the tumour. Primary spinal tumours are a group of tumours which originate from the spinal cord or the vertebral segment. In opposite, metastatic tumours in these areas are secondary tumours of the spine that metastasise to or reach these spinal regions from an area that is significantly far away via a haematogenous route or from nearby organs. The primary tumour group incorporates a wide variety of tumours which can occur all through the spine. These tumours of the spine usually originate from neural, osseous, cartilaginous, and perineural structures. However, these tumours contrast from one another in terms of location and origin. An inappropriate utilization of the terminology related to spinal tumour origin and subtypes can be seen in the literature. To correct it, and for the sake of simplicity, an explanation of this nomenclature is greatly needed. Logical inconsistency is often seen in the use of the expressions “spinal tumour” and “spinal cord tumour.” These two terms are sometimes utilized interchangeably. Even when it does not originate from the spinal cord, any pathology influencing the spinal cord, regardless of whether it is a primary tumour of a vertebral segment or a metastatic (extradural) tumour, can be named a spinal cord tumour, regardless of its root. The expression “spinal tumour” is a general term and incorporates both extradural and intradural lesions. Spinal tumours are comprehensively ordered by their cause and comprise both “spinal cord tumours” and “vertebral segment tumours.” Vertebral segment tumours can be primary or metastatic and originate from osseous and cartilaginous components. Intradural–intramedullary tumours are the term used to describe spinal cord neoplasms that essentially develop from the spinal cord’s cell components. However, complete intradural–extramedullary pathologies, such as meningiomas, can be considered primary spinal cord

tumours even though they do not arise from the spinal cord. Extradural, intramedullary, or both types of intradural tumours are possible. While intramedullary neoplasms arise from glial and support cells of the spinal cord, extradural neoplasms originate from peripheral nerve roots, which are not a fundamental component of the spinal cord. The nomenclature “spinal cord tumour” will be utilized to refer to all intradural diseases in this section. Between 4 and 16% of all adult central nervous system (CNS) tumours are primary neoplasms of the spinal cord. The overall age-dependent incidence rate ranges from 0.74 to 2.5 per 100,000 individuals (Elia-Pasquet et al. 2004; Kurland 1958; Liigant et al. 2000; Materljan et al. 2000). Despite sharing a comparable histopathology with their cranial counterparts, primary spinal cord neoplasms are less typical. Dumbbell tumours are tumours that may be located in various locations. Intradural–intramedullary neoplasms and dumbbell neoplasms make up 18% and 22%, respectively, of all primary spinal cord neoplasms; intradural–extramedullary tumours account for 54% of all such neoplasms (Conti et al. 2004) (Table 1).

No specific neurological sign or symptom may be used to diagnose these tumours. The compression of neural tissues is a characteristic of spinal cord tumours. These tumours’ clinical manifestations can cover a wide variety of adverse effects, from minor sensory complaints to serious motor impairments.

Table 1. Classification of spinal tumours.

Extradural	Intradural
Primary (malignant tumours)	Primary (intramedullary tumours)
Chordoma	Astrocytoma
Chondrosarcoma	Ependymoma
Fibrosarcoma	Dermoid tumour
Ewing’s sarcoma	Epidermoid tumour
Lymphoma	Teratoma
Osteosarcoma	Lipoma
	Haemangioblastoma
	Ganglioglioma
Benign tumours	Extradural tumours
Osteoid osteoma	Meningioma
Osteblastoma	Neurofibroma
Osteochondroma	Schwannoma
Chondroblastoma	
Fibroma	
Giant-cell tumour	
Haemangioma	
Aneurysmal bone cyst	
Secondary	Secondary
Metastatic tumours	Metastatic tumours

Source: Authors’ compilation based on data from Conti et al. (2004).

2. Classification of Spinal Tumours

2.1. Intradural–Extradural Tumours

Intradural–extramedullary (IDEM) tumours mostly consist of nerve sheath tumours (neurofibromas and schwannomas), meningiomas, and myxopapillary ependymomas at the filum terminale. Spinal meningiomas are the most continuous intradural tumours and typically occur in the dorsal region. The psammomatous subtype, which mimics the intracranial subtypes in which numerous psammoma bodies can be seen, is the most well-known histologic subtype (Gottfried et al. 2003; Schaller 2005). Meningiomas tend to affect older people aged 50–70 and are more frequent in women, with a female-to-male ratio of 3 to 1 (Preston-Martin 1990). Both genders are affected by nerve sheath tumours in a similar way, and their occurrence peaks in the fourth and fifth decades of life. The far more prevalent subtype in this group, schwannomas, typically occurs sporadically but can also be detected in neurofibromatosis (Seppälä et al. 1995). The dorsal nerve rootlet gradually becomes normal as spinal nerve sheath tumours (NSTs) develop from either the ventral or dorsal nerve rootlets. These neoplasms can be intradural only, particularly in the cervical regions, or extradural; they can also have both extra- and intradural segments and manifest as dumbbell-like patterns. Schwannomas are thought to form in the area where oligodendrocytes transition into Schwann cells, where they provide myelin. As they swell up, these all-around-capsulated neoplasms can put pressure on nearby functioning fascicles (Kim et al. 1989). Depending on the kind, schwannomas can have a reduced cell structure with palisading Verocay bodies (Antoni A) or have

fewer cells (Antoni B) (Requena and Sangüeza 1995). Neurofibromas are most frequently encountered in people with neurofibromatosis type 2, but they can also occur randomly. In these tumours, which differ significantly from schwannomas in that they can involve different nerve fascicles and grow along the entire nerve, it can occasionally be challenging to completely excise the neoplasm without the deliberate cutting of the nerve root. Damage from schwannomas can be distinguished thanks to the close proximity of axons in net pathology. The most common age for the incidence of filum terminale ependymomas, which are all-around-capsulated tumours, is 36 years old (Sonneland et al. 1985). Histologic smears show myxopapillary-appearing vascularized myxoid cores surrounded by well-differentiated cuboidal or columnar cells that are radially oriented.

IDEM tumours (Figure 1) are often benign, slow-growing neoplasms, and there may be a long lag interval between the onset of clinical features and discovery. The main side effect, axial back discomfort, may not be discovered for quite some time. Another negative effect, particularly in NST patients, is radicular pain. Cauda equina syndrome or myelopathy may result from spinal cord compression. Affected patients may become aware of adverse effects at a younger age if a functional or neurological deficit progresses quickly over time.

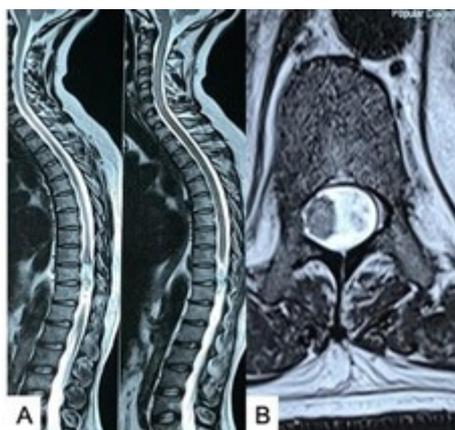


Figure 1. (A,B) MRI dorsal spine (T2W sagittal and axial images) showing intradural–extramedullary tumour (schwannoma). Source: Figure by authors.

Surgery should be performed on all individuals with dynamic neural or practical impedance as well as those whose sequential MR tests show rapid tumour progression. Surgery is not required (on symptomatic grounds only) in asymptomatic patients; the main exception would be in the case of myxopapillary ependymoma, where asymptomatic cases would be asked to undergo cautious excision to prevent CSF leaks (Mridha et al. 2007; Fassett et al. 2005). Due to the difficulties and limitations of the transthoracic approach published by Bohlman, which necessitates the resection of significant portions of the lung and may result in severe vascular injury, thoracic IDEM tumours are typically treated using traditional methods (Bohlman and Zdeblick 1988). Various techniques for removing ventral thoracic lesions have been published; these include the conventional extracavitary procedure described by Larson, which is appropriate for both ventrally located neoplasms and neoplasms with large extraforaminal parts, and the costotransversectomy strategy, which is also appropriate for lateral and ventrolateral injuries, but not for anteriorly located neoplasms due to limited view of the opposite side of the lung (McCormick 1996).

2.1.1. Nerve Sheath Tumours (Schwannoma and Neurofibroma)

About 25% of neoplasms that appear in the intradural–extramedullary region are nerve sheath tumours (Figures 1–4) (Levy et al. 1986).

Nerve sheath tumours occasionally extend to the spinal cord or extramedullary compartment, although the most of the intradural–extramedullary spinal tumours are constrained to the intradural–extramedullary region. Schwannomas make up about 65% of intradural nerve sheath tumours, and neurofibromas make up the majority of the remainder. Malignant NSTs are rare, making up only 5% of these tumours. Both schwannomas and neurofibromas are rare diseases that most frequently present as solitary neoplasms and have no underlying hereditary disease. One of three genetic diseases, namely neurofibromatosis type 1 (NF1), neurofibromatosis type 2 (NF2), or schwannomatosis, will be responsible for a small percentage of isolated lesions and nearly all

occurrences of metastases. Neurofibromas can develop at any age, although schwannomas seldom affect children and peak in incidence in the fourth to sixth decades of life.

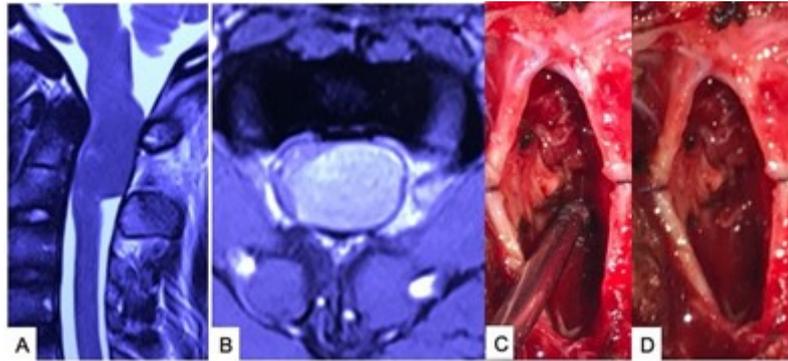


Figure 2. (A,B) MRI of craniocervical junction (CVJ) (sagittal and axial T2W images) showing intradural C2 schwannoma. (C,D) Intraoperative images after removal of tumour. Source: Figure by authors.

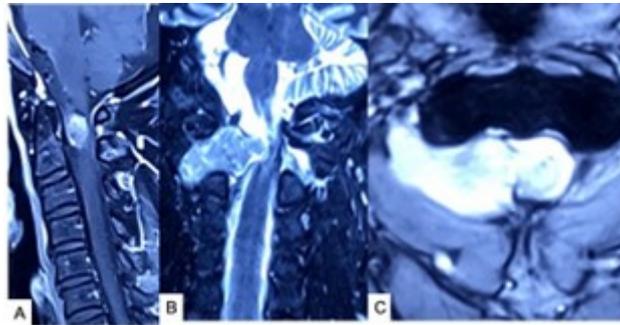


Figure 3. MRI of craniocervical junction (A) contrast sagittal view; (B,C) T2W coronal view) showing C2 schwannoma with both extra- and intradural (extra-arachnoidal) extension on right side. Source: Figure by authors.

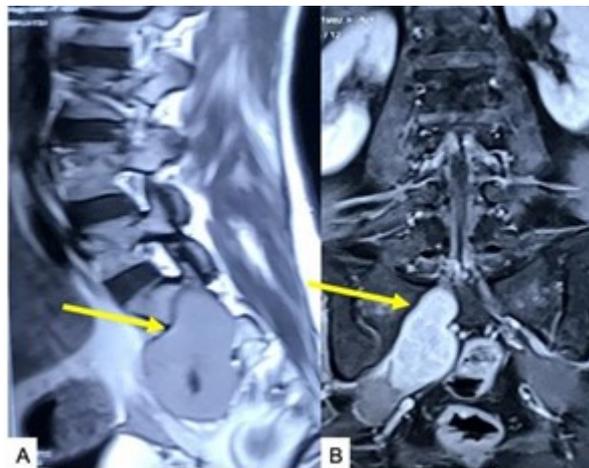


Figure 4. MRI of lumbosacral spine (A) T1W sagittal image and (B) contrast coronal image showing right S2 schwannoma (arrow marked) extending into pelvis (posterior to rectum). Source: Figure by authors.

According to histology, conventional schwannomas have two distinct morphologies and are made of neoplastic Schwann cells (Figure 1). Schwann cells that are constantly spindled to a minimum are organized in fascicles that run in diverse directions in the Antoni A pattern. Spinal schwannomas have a strong propensity for tactile nerve roots and much less frequently affect the autonomic or motor nerves. The majority of schwannomas are relatively slow-growing neoplasms that do not recur and sporadically undergo detrimental alteration. Recurrences occur in 32–40% of cases of plexiform schwannomas and spinal cell schwannomas, which are fairly common.

The majority of irregular neurofibromas manifest as cutaneous lesions, and occasionally, the spinal roots are also involved. However, spinal inclusion is common in NF1 patients, and many neoplasms can be connected to scoliosis and the risk of detrimental alteration (Khalid et al. 2018). Atypical and plexiform neurofibromas are two different types of neurofibromas. High cellularity, scattered mitotic figures, cytological atypia, monomorphic cytology, as well as/or fascicular development characterizes atypical neurofibromas, which can be challenging to distinguish from low-grade peripheral nerve sheath tumours.

2.1.2. Malignant Peripheral Nerve Sheath Tumours (MPNSTs)

Even though there have been a few rare cases of spinal MPNSTs, these tumours usually develop in the retroperitoneum, appendages, head, and neck and make up 3–10% of all soft-tissue sarcomas. If the tumour is resectable, careful excision is the preferred course of treatment because MPNSTs have a high risk of metastasizing (Ducatman et al. 1986); nevertheless, there is currently no effective primary therapy available. The visualization of metastatic or unresectable MPNSTs is incredibly poor, especially in the spinal region, where the mortality rates can reach 80%; larger lesions are therefore inevitably associated with more morbidity (Lang et al. 2012; Endo et al. 2011). The extremely rare condition known as spinal extraosseous chordoma (SEC) typically affects the cervical and epidural regions. SECs are more accurate, less malignant, and recur and metastasize at a slower rate than those originating from the bone. An unusual, dangerous tumour that develops from bone or soft tissues is called mesenchymal chondrosarcoma. Tumours may contain calcification, which could influence or reflect the progression of the disease.

2.1.3. Spinal Meningiomas

Spinal meningioma (Figure 5) develops in the membranes that surround the spinal cord (Duong et al. 2012). Compared to men, women experience these much more frequently (Hoa and Slattery 2012). Meningiomas are intradural–extramedullary in 90–95% of cases (Conti et al. 2004), and the shape of an intradural tumour, also known as a dumbbell tumour, is particularly characteristic. Spinal meningiomas comprises nearly 1.2–12.7% of all meningiomas as well as 25% of all spinal cord tumors. The thoracic region is where these tumours are most frequently encountered, followed by the cervical and lumbar regions. The peak incidence of meningiomas occurs in the sixth and seventh decades of life and they are often only diagnosed after the age of 50.



Figure 5. Contrast MRI of spine (sagittal view) showing (A) upper dorsal meningioma, (B) cervical plaque meningioma, and (C,D) lower dorsal meningiomas (arrow marked). Source: Figure by authors.

Spinal meningiomas have a low risk of tumor recurrence and a generally good oncological and surgical prognosis. Slowly expanding and occasionally engulfing the nearby arachnoid, but infrequently the pia, spinal meningiomas grow laterally into the subarachnoid region. Surgery is the usual course of treatment, with the goals being full excision of the tumor and the restoration and improvement of neurologic function. Although radiotherapy is usually used as an adjuvant therapy for spinal meningioma, it may be explored in cases of tumor recurrence, for difficult surgical cases, and for individuals with higher-grade lesions. Novel molecular and genetic profiling contributes to our understanding of spinal meningioma and could lead to the discovery of new therapeutic avenues (Hohenberger et al. 2023).

2.2. Intradural–Intramedullary Tumours

Intradural–intramedullary tumours (Figures 6 and 7) can be of the following varieties.

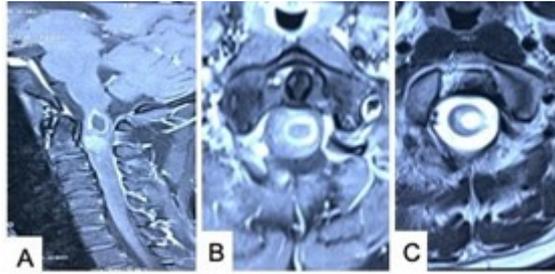


Figure 6. Contrast-enhanced MRI of cervical spine (A) contrast sagittal, (B) contrast axial, and (C) T2W axial images) revealing cystic and solid lesions to the upper cervical spine (astrocytoma grade 2). Source: Figure by authors.

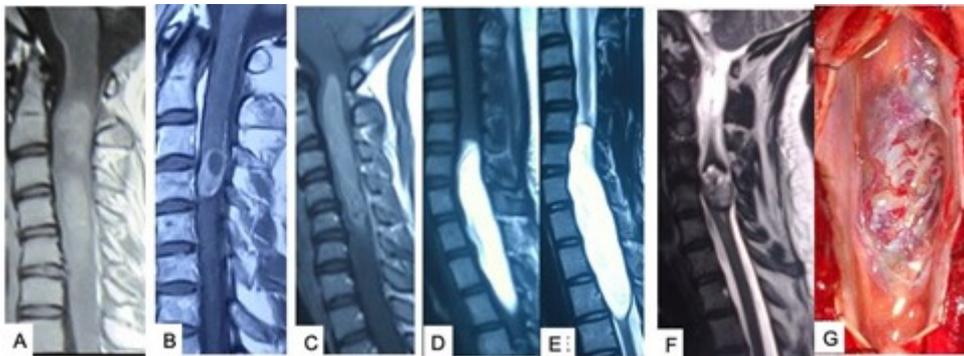


Figure 7. MRI of spine (sagittal sections) showing intramedullary spinal tumours: (A) long-segment cervical ependymoma; (B) cervical haemangioblastoma; (C) cervical astrocytoma; (D,E) cervicodorsal lipoma; and (F) cervical cavernoma. (G) Intraoperative picture of cervical cavernoma after durotomy in patient in (F). Source: Figure by authors.

2.2.1. Astrocytoma

This tumour (Figures 6 and 7C) has a low rate of one-year survival. Incidence is most common between the third and fifth decades of life. The female-to-male = 1:1.5. The proportion of low-grade to high-grade tumours is 3:1 in all age groups. It can occur at all possible levels.

A total of 38% of cases are cystic; the cystic fluid, as a rule, has a high protein content (Greenberg 2010).

2.2.2. Ependymoma

The most widely occurring tumours of the lower spinal cord, conus, and filum are ependymomas (Figures 7A and 8), and they are slow to develop. There is a slight male predominance, and they are commoner in the third to the sixth decades of life. The majority occur in the filum, and the cervical region comes in second. Histologically speaking, myxopapillary ependymoma (Figure 8) is the most frequent. Other histological types include papillary, cell, epithelial, and mixed. A total of 46% of affected people have cystic degeneration, which may extend into the spinal canal. In the filum, it is typically barely vascular (Greenberg 2010).

2.2.3. Haemangioblastoma

Haemangioblastoma (Figure 7B) is generally non-invasive, sharply demarcated, and may include peripheral cysts. Some 33% of cases of spinal haemangioblastoma are linked to von Hippel–Lindau disease. It requires a microsurgical approach like AVM, and may be associated with intraoperative hypotension (Greenberg 2010).



Figure 8. (A,B) MRI of lumbosacral spine showing myxopapillary ependymoma at D12 to L3. Source: Figure by authors.

2.2.4. Dermoid and Epidermoid

Epidermoids are uncommon before the late teens/young adulthood. There is a slight female predominance. Upper thoracic and cervical localizations are uncommon; incidence in the conus is frequent. Generally, these are IDEM neoplasms, yet in the conus/cauda equina they may have an IM (intramedullary) part.

2.2.5. Lipoma

Lipoma (Figure 7D,E) may develop related to spinal dysraphism. Below, we consider lipomas that happen without spinal dysraphism. Onset peaks in the second, third, and fifth decades of life. There is no sex prevalence. Typically, they are IDEM (there is a sub-type that is exclusively IM, in the spinal cord); the cervicothoracic level is the most well-known area of occurrence. Faecal incontinence is common with low tumours. Nearby subcutaneous dimples or masses are visible. Malis suggests early subtotal resection at around 1 year from onset in symptomless patients (Malis 1978). Superficial extrasacral resection is insufficient, as it results in thick intraspinal scarring which may prompt acute serious neurological damage with poor salvageability.

3. Extradural Tumours

3.1. Chordoma

The primary sacral spine tumour that is most commonly diagnosed today is chordoma. These tumours rarely develop in persons under the age of 40 and are twice as frequent in men than in women. The sacral spine, the notochord's embryologic endpoint, is where it typically develops from remaining notochord cells. Chordomas develop gradually but are malignant. Patients commonly present with bladder and bowel incontinence, sexual dysfunction, dull pain, or neurological repercussions. Huge sacral chordomas in patients might press on the internal organs, resulting in bowel blockages, urine incontinence, and abdominal pain. CT scans of the sacrum might be insufficient for visualizing these tumours. Magnetic resonance imaging is the favoured imaging methodology, permitting the clinician to assess the size of the tumour, explicit tumour segments, and the degree of soft-tissue involvement. On separate T1W imaging and T2W imaging scans, chordomas are frequently isointense and hyperintense. With gadolinium, they improve heterogeneously. The sacral cortex can be reconstructed in computed tomography (CT), and neuroforaminal widening can be shown. Additionally, some chordomas may have calcifications. A thorough biopsy is frequently recommended for tissue analysis and to manage additional treatment. Endoscopy and biopsy of disputed lesions should be completed if it is proven that the lesion originated primarily from the rectum. Transrectal biopsy should be avoided to prevent the spread of tumour cells to unaffected regions. The remaining tumours should be analyzed via CT-guided biopsy, and the biopsy tract is confined within the confines of the concomitant excision. Several histologic subtypes exist. Conventional chordomas contain a bottomless myxoid framework and the cell cytoplasm has a "bubble-like" physaliferous design. Other types of chordomas incorporate chondroids and de-differentiated chordomas. For the most part, chordomas affect soft tissues, and are typically avascular (Arnautovic and Gokaslan 2019).

3.2. Epidural Tumours

This section on extradural spinal cord tumours must make a distinction between two categories: bone and soft-tissue tumours. It is possible to further classify the latter into primary and secondary neoplasms. These neoplasms differ in a number of crucial ways: although soft-tissue tumours are typically benign and do not directly affect the biomechanical qualities of the spinal column, bony tumours interfere with spinal cohesion and are predominately caused by malignant agents. In the past two years, there have been notable advancements in the surgical management of bone tumours, including a better perception of the spinal cord's biomechanics and improved repair and reconstruction techniques (Klekamp and Samii 2007).

3.2.1. Sign and Symptoms

Pain, motor weakness, gait ataxia, sensory deficits, dysaesthesias, sphincter issues, localized swelling, etc., are the earliest symptoms of epidural spinal tumours (including soft-tissue and bone tumours). Most patients with epidural tumours have a characteristic clinical presentation, which includes systemic pain, spastic para- or tetraparesis, and radicular symptoms. Local discomfort is the primary symptom in most individuals with epidural neoplasms (65% with soft-tissue and 79% with bone tumours). At first, only a small number of patients are seen to have motor or gait issues (Klekamp and Samii 2007).

Soft-tissue tumours that originate from the epidural region push epidural veins and fat aside before compressing the dura. They frequently advance into the paraspinous spaces in the direction of the intervertebral foramina as they grow along and around the dural sac. The foramina may then become enlarged as a result of bone erosion. The vertebral bodies may become twisted or dysplastic if this phenomenon starts in early childhood.

Depending on whether they are benign or malignant, bone tumours tend to damage or alter the bony anatomy but otherwise have similar clinical characteristics. Local pain, which is the primary initial symptom in 79% of patients and is brought on by periosteum and bone infiltration, is present. Before the tumour affects the spinal cord, radicular symptoms may appear after it compresses the intervertebral foramina or bursts into the soft tissue. In a paper on spinal metastases, for instance, it was demonstrated that 80% of patients progressed from local discomfort to neurological features within 2 months (Helweg-Larsen and Sørensen 1994).

However, due to the risk that malignant tumours, in particular, pose to the integrity of the spine, quick clinical diagnoses following vertebral collapse and spinal instability are not unheard of.

Patients with bone tumours (Figure 9) typically experience gait issues and localized pain. Analogously to patients with extra- and intramedullary tumours, people with epidural tumours have noticeably higher levels of discomfort (McCormick et al. 1990; Solero et al. 1989). People with epidural neoplasms had the highest percentage of progressive spinal cord damage, in contrast (i.e., they are incapable of walking or experience bowel and bladder incontinence). This is mostly caused by bony tumours that suddenly compress the cord following pathological fractures and by a high rate of malignant tumours. In other words, because indications of spinal dysfunction may obscure those of tumour progression as in intra- or extramedullary tumours, the clinical course is not always straightforward.

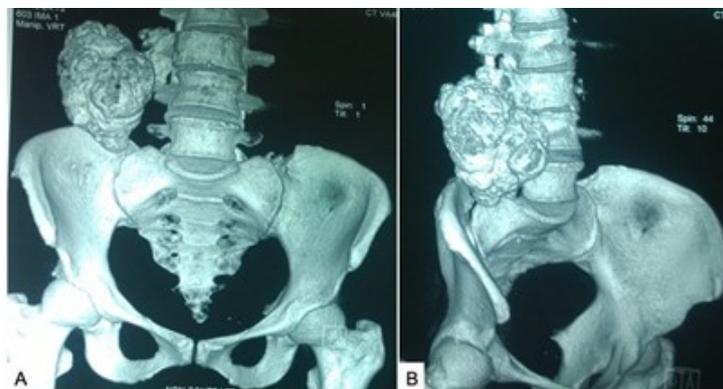


Figure 9. (A,B) Three-dimensional reconstruction CT scan of lumbosacral spine and pelvis showing L3 right transverse process-originating osteochondroma. Source: Figure by authors.

3.2.2. Neuroradiology

While MRI is currently almost exclusively used to make a neuroradiological diagnosis of extra- and intramedullary tumours (Figures 1–8 and 10–12), this is not the case with extradural tumours, notably bone tumours. CT and straightforward X-rays are absolutely necessary for spine bone tumours and are also advised for extradural soft-tissue tumours, despite the fact that contemporary MRI affords excellent visualization of soft-tissue and bone structures.



Figure 10. MRI of spine (sagittal view) showing D2 and D7 vertebral body collapse due to metastatic tumours (with compression of thecal sac). Source: Figure by authors.



Figure 11. MRI of dorsal spine (sagittal views) showing multiple vertebral involvement of tumours (multiple myeloma) with collapse of multiple vertebral bodies. Source: Figure by authors.



Figure 12. (A,B) MRI of cervical spine (sagittal views) showing cervical neuro-enteric cyst. Source: Figure by authors.

An extradural tumour's neuroradiological evaluation must reveal the following information:

1. The tumour's location and size, to start.
2. Distinction between bone tumours and soft-tissue tumours.
3. Distinction between a metastatic and a malignant tumour.
4. The reaction of the surrounding tissues.
5. Major vessel involvement.
6. Spinal stability.

Oblique X-ray imaging can show the intervertebral foramina. Although schwannomas account for the majority of cases, widening of the foramina can also be seen in a range of other histologies, including osteblastomas, chondrosarcomas, and meningoceles (Zibis et al. 2000). Some epidural tumours can grow to huge sizes within the paraspinal spaces. Epidural schwannomas with bright contrast enhancement can be solid or cystic. They are generally laterally placed, compress the dura, and develop in the extraspinal space along the sheath of the nerve via the neuroforamen. The neuroforamen is widened. The extraspinal part is in most cases larger than the intraspinal part. Sometimes, minor intradural tumour extensions cannot be ruled out on MRI; in these cases, the dura needs to be extended along the nerve root for intradural examination. Epidural cavernomas show a varied signal pattern on MRI based on the quantity and chemical condition of haemoglobin derivatives linked to smaller haemorrhages. They may relate to significant haemorrhagic cysts. Synovial cysts have fatty liquid in them. The signal pattern on MRI is inhomogeneous and can look like a cavernoma. Computed tomography shows the linkage to the intervertebral joint and facilitates the differential diagnosis.

Epidural arachnoid cysts are very simple to spot. The degree of spinal compression exerted by those cysts is complicated to ascertain. Some behave as slit-like dura dissections, while others only slightly or severely pinch the dura and spinal cord. The diagnostic difficulty is to show a dura shortage and, consequently, the communication location between the cyst and the subarachnoid space. The surgical plan is determined by this location. When there has been a history of trauma, dura deficiencies along a nerve root sleeve are more commonly linked to epidural arachnoid cysts. To show this communication, myelography and post-myelographic CT may be necessary. The spinal cord may occasionally herniate into this dura defect. On T1W images, lymphomas and other infiltrating neoplasms like soft-tissue sarcomas are iso- or hypointense, but on T2W images, they exhibit high signal intensity. They develop in the epidural region around the dural sac and homogeneously accumulate contrast without breaking down the bone (Boukobza et al. 1996).

Epidural diseases that may be mistaken for neoplasms include spinal lipomatosis, disc prolapses, abscesses, and chronic epidural haematomas. MRI signal intensities for chronic extradural haematomas vary according to the age of the haemorrhage. In patients with haemorrhagic diatheses, they may spread throughout numerous spinal segments or be localized. No trauma history is required. Sequential exams may be used to prove spontaneous resorption. Spinal epidural abscesses can be brought on by haematologic conditions or local spread from a nearby vertebral abscess or spondylodiscitis. Spinal lipomatosis, which is correlated with total body fat, can be seen in patients who are very overweight. It might also be connected to steroid therapy. Severe compression of fat tissue by the dural sac is the distinguishing feature.

3.3. Metastasis

The most prevalent spinal tumours are spinal metastases (Figures 10 and 11), which account for around 90% of the masses seen on spinal imaging. Although not restricted to bone metastases, spinal metastases are more frequently detected as bone metastases and about 20% of patients also have invasion of the spinal canal and cord compression. The dorsal region of the spinal column is where metastasis is most frequently discovered, followed by the lumbar region. The cervical region is where metastasis is least likely to be discovered (Mundy 2002).

The sparing of intervertebral disc space is a distinguishing characteristic of these lesions when analysing spinal metastases on MRI images. This disc area is almost always involved during an infection. The pathways by which metastatic spine disorders spread include direct tumour growth, venous haematogenous dissemination as opposed to arterial spread, and, eventually, lymphatic spread. The most frequent route for tumour embolization and spinal invasion among these is haematogenous dissemination through Batson's plexus system. The following tumours, listed in descending order, are the most prevalent primary malignancies that largely spread to the spine: thyroid (2.5%), renal (5%), gastrointestinal (4.5%), lung (19%), prostate (7.5%), and breast (21%). All tumours have the potential to spread to the spine; however, the cancers indicated above metastasize into the spinal cord at an early stage of the disease (Ziu et al. 2022).

3.3.1. Epidemiology

When treating cancer patients, doctors frequently run into the situation of spinal metastases. The morbidity associated with spinal metastasis includes spinal cord compression brought on by the invasion of epidural space, pathological bone fractures needing large dosages of narcotic drugs for therapy, and hypercalcemia. Rarely will spinal metastasis with no apparent involvement of the bone seed within the spinal cord itself. In such cases, the diagnosis of metastatic lesions is complicated because there is no known history of the primary tumour, so the proper diagnosis is only determined after testing identifies the type of tumour.

3.3.2. Clinical Symptoms and Physical Examination

Pain is the primary symptom experienced by people with spinal metastases. Any oncologic patient who experiences back or neck pain should be given a high level of clinical suspicion. Pain is the most common starting symptom that needs to be evaluated by the doctor in therapy, despite the fact that it is not the most dreadful or deadly symptom of spinal metastases. In addition to being an early symptom, metastasized neck and back pain frequently requires additional diagnostic imaging in patients who cannot undergo imaging of the full spine. Patients sometimes awaken from sleep due to the severe and excruciating pain. Additionally, if nerve injury has occurred, the pain is intense and firing in a particular dermatomal range, which may signal a greater tumour enlargement. In the event that the tumour has spread into the spinal canal, sensory and motor impairment could be permanent and even more concerning. The degree to which the deficit is weak and extends is a key concept for treating spinal metastases. The larger the deficit at presentation, the worse the odds of recovery. Furthermore, the amount of time between the onset of the deficiency and the doctor discovering the cause affects the possibility and probability of regeneration.

3.3.3. Diagnosis

The simplest and most frequently accessible diagnostic for evaluating an oncologic patient with neck or back discomfort is an X-ray of the spine. Simple anterior, posterior, and lateral pictures are frequently insensitive or complicated and need at least 50% bone disintegration before an issue may be noticed. MRI of the spine is the gold standard for assessing these abnormalities (Figures 10 and 11). Expansion, invasion rates, spinal canal obstruction, and metastatic aetiology are all revealed by MRI. However, it is not always available, for example if a patient has an internal or external pacemaker. Myelography should be utilized with or without CT imaging for patients who are not suitable for MRI. Myelography has the advantage of sending CSF for pathological investigation; however, its utility is severely limited when the canal is entirely blocked by a lesion. In these cases, multiple contrast injections into the spinal cord could be required to overcome the obstruction stage.

3.3.4. Treatment

Spinal Instability Neoplastic Score (SINS) (Fisher et al. 2010). Scoring is as follows:

- Location:
 - Junctional (occiput–C2, C7–T1, T11–L1, L5–S1)—3 points;
 - Mobile spine (C3–6, L2–4)—2 points;
 - Semirigid (t3–T10)—1 point; rigid (s2–5)—0 points.
- Pain:
 - Yes—3 points;
 - Occasional but not mechanical pain—2 points;
 - Pain-free lesion—0 points.
- Bone lesion:
 - Lytic—2 points;
 - Mixed (lytic/blastic)—1 point;
 - Blastic—0 points.
- Spinal alignment:
 - Subluxation/translation present—4 points;
 - De novo deformity (kyphosis/scoliosis)—2 points;
 - Normal alignment—0 points.

- Vertebral body collapse:
 - >50% collapse—3 points;
 - <50% collapse—2 points;
 - No collapse with >50% body involved—1 point;
 - None of the above—0 points.
- Posterolateral involvement of the spinal elements:
 - Bilateral—3 points;
 - Unilateral—1 point;
 - None of the above—0 points.

A score of 0–6 indicates stability, a score of 7 to 12 defines intermediate (possibly impending) instability, and a score of 13–18 indicates instability. A score of more than 7 warrants a surgical consultation.

Patients can be managed without surgery if metastasis affects many bony structures without compromising the cord or causing a bone fracture. However, consulting a spine surgeon is crucial if you want guidance on spinal instability caused by several spine metastases. However, radiation therapy and chemotherapy may be the main treatment options for the majority of patients with multiple spinal metastases and normal neurological examinations. An image-guided bone lesion biopsy may be beneficial if tissue is required for pathological diagnosis and there are no primary metastases or metastases that are easily accessible using any other approach. Rarely is an open diagnosis necessary after numerous unsuccessful efforts with a needle biopsy (Aielli et al. 2019; Kam et al. 2019; Yang et al. 2018).

When a spinal cord tumour is present, the approach to treatment must change considerably, and immediate surgical consultation is necessary because these patients may proceed to bed-bound status within days. Research has demonstrated that paralysis brought on by metastatic spine disease considerably reduces the life expectancy of malignant patients. Contrarily, surgical intervention will greatly lower the mortality and morbidity linked to acute paralysis in patients with acute paralysis brought on by metastatic disease compression of the spinal cord (Patchell et al. 2005; van den Bent 2005).

3.3.5. Role of Steroids

Depending on the speed and severity of neurological deterioration, treatment may require the administration of steroids. In clinical trials, dexamethasone has been found to reduce pain and ameliorate symptoms. The precise dose that would help the patient the most is unknown, though. No discernible therapeutic advantage was seen with the larger dose in research comparing a 100 mg dexamethasone bolus dose with a 10 mg initial injection of dexamethasone bolus. A 10 mg IV bolus dosage followed by a 4 mg maintenance dose given every 6 h, tapered over the course of 2 weeks, as permitted by the clinical scenario, could serve as a successful starting dose. Immediate scans to assess growth and possible surgical involvement should be made available.

Surgical intervention is acceptable when there is little reason to suspect the presence of an extremely radiosensitive tumour, when total paralysis has been present for longer than 24 h, or when the patient's projected survival is shorter than 3 or 4 months. Following surgery, further care should include chemotherapy and targeted radiation using a multidisciplinary approach. Radiation therapy typically involves 30–40 Gy in ten treatments. After radiation and chemotherapy, wound closure becomes a worry; thus, the patient needs to be continuously watched to spot and treat the wound as soon as possible (Le et al. 2018; Osborn et al. 2018).

The NOMS (neurological, oncological, mechanical, systemic) paradigm is the foundation of the current therapeutic strategy for spinal metastases. This framework's neurological component evaluates the patient's neurological status and the grade of epidural spinal cord compression as determined by MRI; the oncological and mechanical components describe the radiosensitivity of the primary tumour and the mechanical stability of the spine as determined by the neoplastic spinal instability score, respectively. These parameters determine the broad management categories for these patients, which may include decompression and surgical stabilization, spinal stereotactic radiation therapy, conventional external beam radiation therapy alone or with stabilization, or a combination of these following separation surgery (Table 2).

Table 2. Augmentation and radiosurgery in spinal metastases.

Surgery	Vertebral Augmentation	Radiosurgery
Manifested spinal cord compression by a tumour that is not highly radiosensitive, mechanical instability, uncertain diagnosis.	Vertebral body fracture with pain without significant epidural spinal cord compression.	Failure of radiotherapy to control disease.

Patients without any of the above are generally candidates for radiation therapy. Source: Authors' compilation based on data from Patchell et al. (2005); van den Bent (2005); Bilsky et al. (1999).

3.3.6. Indications for Surgery

Primary surgery (Bilsky et al. 1999):

- Radioresistant tumour (sarcoma, renal cell carcinoma);
- Spinal instability;
- Pathological fracture with bone in the spinal canal;
- Occult primary tumour;
- Circumferential epidural tumour; moderate to highly radioresistant tumour (colon, lung).
- Secondary surgery after chemo/radiotherapy.
- Progressive neurologic symptoms.

4. Sacral Spinal Tumours

Up to 7% of all spinal tumours are sacral spine tumours, which are extremely unusual (Feldenzer et al. 1989). They can be categorized thoroughly into primary or metastatic groups. In addition, primary sacral neoplasms may be divided into three categories based on their origin: congenital, osseous, or neurogenic. Chordoma is the most well-known primary sacral neoplasm. Metastasis-related sacral tumour is the most well-known type. It is worth identifying the differences between primary and metastatic sacral tumours since they may have a big impact on future treatment paradigms and interdisciplinary debates. For example, a primary sacral neoplasm should be given cautious consideration for biopsy, with detailed technique considerations and follow-up treatment plans. On the other hand, biopsy is probably unsuitable for patients who have complete spinal injury and a metastatic tumour with sacral linkage. In these patients, carefully removing tumour tissue or plans for palliative care that include chemotherapy or radiation might be more appropriate. Sacral tumours present noteworthy management difficulties and require careful treatment because of a wide range of accompanying side effects, tumour types, and complex accessory bone structures. The bone structure of the sacral spine is one of a kind as it is close to neighbouring neurovascular structures, bony components, joints, and retroperitoneal organs, which requires an interdisciplinary approach before starting treatment.

4.1. Lymphoma

The CNS may be afflicted by lymphoma, a deadly lymphocytic tumour, either as the primary or secondary manifestation of another systemic illness. "Primary CNS lymphoma" (PCNSL) is lymphoma of the spinal cord or brain that excludes all other regions (apart from the visual structures). Over the past 20 years, CNS lymphoma has increased in frequency across all age groups.

4.2. PCNSL

PCNSL has also been referred to in the literature as microgliomatosis, immunoblastic sarcoma, malignant reticulosis, and perivascular sarcoma. When present, it most frequently manifests as single or multiple central brain damage in middle-aged adults. The patient's immunologic capacity affects how the neurologic involvement presents. Nearly 4% of intracranial tumours are primary CNS lymphomas. For up to two decades, critical CNS lymphoma rates have been steadily rising (Grommes and DeAngelis 2017).

An isodense or tolerably hyperdense lesion that improves firmly and uniformly with differentiation is seen by a CT scan. Oedema that is mild to moderate is usual. Particularly in immunocompromised patients, tumours may exhibit an upgraded ring structure. A typical region is the periventricular zone, where most tumours border the ependyma. On T1W MR images, primary CNS lymphomas are often isointense or mildly hypointense, and on T2W MR images, they are isointense or mildly hyperintense. Benign tumours have been seen; however, they are uncommon. Treatment modalities for PCNSL have advanced significantly in the last

decade. Chemotherapy and radiotherapy, separately or in combination, have altogether expanded the prognosis of numerous immunocompetent patients with PCNSL.

5. Rare Spinal Cord Tumours

5.1. Epidemiology

Although the most recent classification of intramedullary spinal cord tumours (IMSCTs) states they only comprise 2–4% of all CNS tumours, spinal cord neoplasms actually make up about 15% of all primary central nervous system (CNS) tumours (Kopelson et al. 1980). Astrocytoma and ependymoma, two of the more prevalent intramedullary lesions, are described in detail elsewhere in this book. Astrocytomas and ependymomas are the commonest IMSCTs and usually occur in children, although ependymomas have also been seen in adults. Haemangioblastomas and spinal cord metastases, which are far commoner in the adult population, are less common IMSCTs.

5.2. Diagnostics

Assessment should initially start with a careful physical test with an emphasis on distinguishing upper motor neuron signs to identify the neurologic level of injury. When the physical test has been finished, the highest-quality imaging modality is magnetic resonance imaging (i.e., MRI). Gadolinium is commonly used as the contrast material. Histologic conclusions can sometimes be reached by surveying the MRI qualities of the lesion, as every subtype has a particular imaging appearance. T2W imaging is the modality of choice to detect spinal canal widening as lesions are traditionally hyperintense (Arima et al. 2014). Thus, primary intramedullary spinal cord lymphoma appears hyperintense on T2W MRI. These neoplasms are frequently multicentric and ineffectively described without syrinx evidence (Nakamizo et al. 2002). Homogenous enhancement and solid qualities are visible on gadolinium-enhanced images and an isointense signal on T1 (Flanagan et al. 2011). On T1 and T2W imaging, intramedullary lipomas are hyperintense and follow fat signal on all sequences (Shen et al. 2001). Cord lipomas are usually singular; however, some patients have multiple lipomas (Patwardhan et al. 2000). Tumours are generally situated along the ventral segment of the spinal cord in the intradural–extramedullary area, yet they can invade the intramedullary space in rare cases (Menezes and Traynelis 2006).

6. Dumbbell Tumours of the Spine

Heuer first used the phrase “dumbbell tumour” in 1929 to refer to spinal tumours that develop an hourglass shape as they grow inside an anatomical barrier, like a nerve root foramen, the dura mater, or other bone components (Heuer 1929; Eden 1941; Love and Dodge 1952). Based on the site of the tumour, spinal tumours with significant intraspinal or/and paravertebral involvement are categorized into four groups: intramedullary, intradural–extramedullary, epidural, and dumbbell (McCormick 1996). Dumbbell tumours can be divided into different groups as indicated by the constricting structure and details of tumour location (Asazuma et al. 2004).

Today, the phrase “dumbbell tumour” refers to a specific type of tumour that involves at least two distinct places and is associated with them, like the intradural or epidural space or regions external to the spinal canal, rather than the hourglass shape (Ozawa et al. 2007).

The location and size of spinal dumbbell tumours influence their presentation. The majority of individuals with spinal dumbbell neoplasms present with similar clinical features, regardless of the underlying pathology. Non-radicular pain is a typical side effect, followed by sensory deficits, gait difficulties, radiculopathy, motor impairments, ataxia, and bowel and bladder dysfunction (Safaei et al. 2015; Sowash et al. 2017). Non-radicular pain can continue as the disease progresses, while radiculopathy will in general settle following surgery (Sowash et al. 2017). Dumbbell tumours are more likely to be malignant among paediatric patients than among adult patients.

7. Spinal Arachnoid Cysts

Arachnoid cysts may compress the spinal cord and nerve roots, or simply obstruct the flow of the CSF, depending on their size and position, which could be the cause of a syrinx (Clifton et al. 1987; Inoue et al. 2001; Mallucci et al. 1997; Wang et al. 2003). They might be brought about by injury or another condition that causes arachnoid scarring, for example, meningitis, subarachnoid drain, or surgery, to name but a few (Andrews et al. 1988; Bassiouni et al. 2004; Buczek and Jagodziński 1994; Fobe et al. 1998; Kang et al. 2000; Osenbach et al. 1992). Patients with ankylosing spondylitis have been shown to experience multiple lumbar arachnoid cysts (Rosenkranz

1971; Shaw et al. 1990). Congenital cysts and cysts related to various of mutations have also been described (Baysefer et al. 2001; Jamjoom et al. 1991; Wakai and Chiu 1984). Some of them may be a benign mutation (Fortuna and Mercuri 1983; Perret et al. 1962). Arachnoid cysts can communicate with or be separated from the subarachnoid area. The weight of the cyst and, thus, the clinical side effects, may change during the course of the patient's treatment depending on how well they correspond with the subarachnoid space. Rarely, spinal cord herniation and dura abnormalities are linked to intradural arachnoid cysts. Arachnoid cysts may be detected posteriorly in the midline or may only affect one side of posterior back subarachnoid space if they are limited to either side of the posterior median arachnoid septum (Perret et al. 1962). Anterior cysts are usually a consequence of a surgical procedure, such as a lumbar puncture or an injection, or a sequela of trauma or meningitis.

8. Cysts and Tumour-like Lesions

Throughout the CNS, there are a few locations where cysts with cuboidal to columnar mucin-delivering epithelium are seen. These refer to Rathke's cleft cysts in the sella, colloid lesions in the third ventricle, and neurenteric (neuroepithelial, neuroglial, enterogenous, or bronchogenic) lesions (Figure 12) when they develop in the anterior spinal canal or intracranially. These cysts are thought to be benign rather than malignant. Radiographic presentation is comparable among these benign lesions, including Rathke's cleft cysts, colloid lesions, enterogenous lesions, neuroglial growths, and epidermoid and dermoid cysts (Berger and Wilson 1985).

9. Diagnostics and Differential Diagnostics of Spinal Cord Tumours

Spinal cord tumours are uncommon tumours with vague clinical indications; they are usually diagnosed late. Radicular symptoms, for example, back pain, progressive neurologic deficiencies, or skeletal deformities, are ordinarily seen in children. Approximately 20–30% of primary intradural spinal tumours are spinal cord tumours. The intradural–extramedullary compartment is where the remaining approximately 70–80% of primary intradural neoplasms are located (Duong et al. 2012). Spinal cord tumour detection and evaluation are performed via magnetic resonance imaging. T1W and T2W sagittal and axial views should be used in the imaging protocol. In the sagittal, axial, and coronal planes, those configurations should include contrast-enhanced T1W series. Similar to when abnormal bone is found, short-TI inversion recovery (STIR) must be used to detect spinal cord tumours. As of late, some CT procedures, for example, dissemination-weighted imaging (DWI) and diffusion tensor imaging (DTI), have been described in the assessment spinal lesions (Landi et al. 2016).

10. Benign Tumours of the Spinal Column

Benign tumours of the axial skeleton are usually found in children and young people. When they happen in adults, they are commonly found in people somewhere in the range of 20 and 30 years old, in a posterior area. Osteochondroma, osteoid osteoma, and osteblastoma are the more common types of benign lesions that are less likely to recur if a thorough resection can be performed. The removal of the primary tumour itself is typically therapeutic, unlike in malignant tumours, which call for the removal of a large portion of healthy tissue surrounding the lesion. Other "benign" tumours, such as eosinophilic granulomas, giant-cell tumours, aneurysmal bone cysts, and haemangiomas can be linked to underlying infection, occur in many locations, or be locally aggressive, respectively. Osteochondroma (Figure 9) is a ligament-topped, hard projection that may develop from a cartilaginous remnant of the physis or from a nearby physis. Of all benign bone tumours, osteochondromas are the most well-known. Osteochondromatosis, one of the commonest skeletal dysplasias, can manifest as a consequence of hereditary osteochondromas. Clinical features range from dull spinal pain (smaller tumours) to decreased mobility or deformity (bigger tumours). Although they have similar pathogenic origins, osteblastoma and osteoid osteoma differ in size and frequency of spinal contribution. These lesions are thought to represent chronic inflammatory responses rather than actual neoplasms. Neurological disorders are rare, but the most well-known cause of painful scoliosis is osteoid osteoma. It can be seen radiographically as a radiolucent area with a focal nidus and an appropriate degree of surrounding sclerosis. Treatment is through extraction. Even though small deformities will be resolved with resection alone, severe scoliosis may necessitate combination treatment. Aneurysmal bone cysts (ABCs) are benign, non-neoplastic, proliferative lesion. ABCs affect the axial skeleton in 12–25% of all documented cases, although they account for just about 1–2% of all primary bone tumours. Although the pathophysiology is unclear, theories include a concealed tumour or traumatic arteriovenous malformation,

which would lead to the formation of a cyst. ABCs have fluid-filled chambers that are partitioned by fibrous septa. ABCs most frequently occur in the thoracolumbar region (Moore and Newell 2006).

Benign bony tumours are usually found in the posterior region, with the posterior segments accounting for 60% of spinal aneurysmal bone cysts. ABCs typically manifest in younger patients, in their second decade of life. A multiloculated, expansile, deeply vascular osteolytic lesion with an eggshell-like cortical edge can be seen on radiographic imaging with CT and MRI. In up to 40% of cases, different degrees of vertebral inclusion can occur. Preoperative embolization, complete resection, or embolization alone for regions of the spine that are challenging to access are all forms of treatment. If not enough edges are resected, postoperative radiotherapy may be necessary (Park et al. 2016).

11. Spinal Osteosarcoma

Osteosarcoma, which makes up 0.5% of all malignant tumours, is the most prevalent type of bone sarcoma. Relatively uncommon, spinal osteosarcoma makes for 3–5% of all spine cancers. The sacral region is the primary affected area, with the lumbar and thoracic segments following suit. Clinical treatment of spinal osteosarcoma has always been difficult due to the disease's diverse anatomic locations and significant neurological abnormalities (Wang et al. 2023).

Pain is the most common symptom of osteosarcoma and affects nearly all patients; over 70% of patients also have neurologic impairment. Research indicates that in 80% of cases of osteolysis, CT shows matrix mineralization; in terms of illustrating cortical damage, CT is more accurate than both plain radiography and MR imaging. MRI findings are nonspecific (Katonis et al. 2013).

The current standard of care for osteosarcoma consists of postoperative adjuvant chemotherapy, surgical removal of all clinically relevant metastases, and surgical resection of the main tumour following neoadjuvant chemotherapy. Although there has been significant progress in treating limb osteosarcoma, treating spinal osteosarcoma still presents significant difficulties because of the disease's high recurrence, susceptibility to metastases, and mortality rate (Wang et al. 2023).

The ideal resection depends on the location and extent of the tumour in the spinal column, even though wide en block resection is the procedure with the best outcomes. When the tumour does not impact at least one pedicle and there is no indication that the disease has spread, wide en block excision should be taken into consideration. En block excision is nearly impossible when tumours that affect both pedicles extend into the lamina, the vertebral artery foramen, or the tip of the odontoid. When this occurs, intralesional surgical resection ought to be taken into account. The prognosis is much poorer than that of limb osteosarcoma (Katonis et al. 2013).

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References

- Aielli, Federica, Marco Ponzetti, and Nadia Rucci. 2019. Bone Metastasis Pain, from the Bench to the Bedside. *International Journal of Molecular Sciences* 20: 280. [CrossRef] [PubMed]
- Andrews, Brian T., Philip R. Weinstein, Mark L. Rosenblum, and Nicholas M. Barbaro. 1988. Intradural arachnoid cysts of the spinal canal associated with intramedullary cysts. *Journal of Neurosurgery* 68: 544–49. [CrossRef] [PubMed]
- Arima, H., T. Hasegawa, D. Togawa, Y. Yamato, S. Kobayashi, T. Yasuda, and Y. Matsuyama. 2014. Feasibility of a novel diagnostic chart of intramedullary spinal cord tumours in magnetic resonance imaging. *Spinal Cord* 52: 769–73. [CrossRef] [PubMed]
- Arnautovic, Kenan I., and Ziya L. Gokaslan. 2019. *Spinal Cord Tumors*. Cham: Springer.
- Asazuma, Takashi, Yoshiaki Toyama, Hirofumi Maruiwa, Yoshikazu Fujimura, and Kiyoshi Hirabayashi. 2004. Surgical strategy for cervical dumbbell tumours based on a three-dimensional classification. *Spine* 29: E10–E14. [CrossRef]

- Bassiouni, Hischam, Anja Hunold, Siamak Asgari, Uwe Hübschen, Hermann-Josef König, and Dietmar Stolke. 2004. Spinal intradural juxtamedullary cysts in the adult: Surgical management and outcome. *Neurosurgery* 55: 1352–60. [CrossRef]
- Baysefer, Alper, Yusuf Izci, and Ersin Erdoğan. 2001. Lateral intrathoracic meningocele associated with a spinal intradural arachnoid cyst. *Pediatric Neurosurgery* 35: 107–10. [CrossRef]
- Berger, Mitchel S., and Charles B. Wilson. 1985. Epidermoid cysts of the posterior fossa. *Journal of neurosurgery* 62: 214–19. [CrossRef]
- Bilsky, Mark H., Eric Lis, Jeffrey Raizer, Henry Lee, and Patrick Boland. 1999. The diagnosis and treatment of metastatic spinal tumor. *The Oncologist* 4: 459–69. [CrossRef]
- Bohlman, H. H., and T. A. Zdeblick. 1988. Anterior excision of herniated thoracic discs. *The Journal of Bone and Joint Surgery. American* 20: 1038–47. [CrossRef]
- Boukobza, M., C. Mazel, and E. Touboul. 1996. Primary vertebral and spinal epidural non-Hodgkin's lymphoma with spinal cord compression. *Neuroradiology* 38: 333–37. [CrossRef]
- Buczek, M., and Z. Jagodziński. 1994. Diagnosis and treatment of central nervous system arachnoid cyst. *Neurologia i Neurochirurgia Polska* 28: 211–20. [PubMed]
- Clifton, A. G., L. Ginsberg, W. J. S. Webb, and A. R. Valentine. 1987. Idiopathic spinal arachnoid cyst, and syringomyelia. *The British Journal of Radiology* 60: 1023–25. [CrossRef] [PubMed]
- Conti, Piero, Gastone Pansini, Homere Mouchaty, Christian Capuano, and Renato Conti. 2004. Spinal neurinomas: Retrospective analysis and long-term outcome of 179 consecutively operated cases and review of the literature. *Surgical Neurology* 61: 34–43. [CrossRef] [PubMed]
- Ducatman, Barbara S., Bernd W. Scheithauer, David G. Piepgras, Herbert M. Reiman, and Duane M. Ilstrup. 1986. Malignant peripheral nerve sheath tumours. A clinicopathologic study of 120 cases. *Cancer* 57: 2006–21. [CrossRef]
- Duong, Linh M., Bridget J. McCarthy, Roger E. McLendon, Therese A. Dolecek, Carol Kruchko, Lynda L. Douglas, and Umed A. Ajani. 2012. Descriptive epidemiology of malignant and nonmalignant primary spinal cord, spinal meninges, and cauda equina tumours, United States, 2004–2007. *Cancer* 118: 4220–27. [CrossRef]
- Eden, Kenneth. 1941. The dumb-bell tumours of the spine. *British Journal of Surgery* 28: 549–70. [CrossRef]
- Elia-Pasquet, Sandrine, Dorothee Provost, Anne Jaffré, Hugues Loiseau, Anne Vital, Guy Kantor, Jean-Philippe Maire, Michel Dautheribes, Vincent Darrouzet, Jean-François Dartigues, and et al. 2004. Incidence of central nervous system tumours in Gironde, France. *Neuroepidemiology* 23: 110–17. [CrossRef]
- Endo, Makoto, Chikashi Kobayashi, Nokitaka Setsu, Yusuke Takahashi, Kenichi Kohashi, Hidetaka Yamamoto, Sadafumi Tamiya, Shuichi Matsuda, Yukihide Iwamoto, Masazumi Tsuneyoshi, and et al. 2011. Prognostic significance of p14ARF, p15INK4b, and p16INK4a inactivation in malignant peripheral nerve sheath tumours. *Clinical Cancer Research* 17: 3771–82. [CrossRef]
- Fassett, Daniel R., James Pingree, and John RW Kestle. 2005. The high incidence of tumour dissemination in myxopapillary ependymoma in pediatric patients. Report of five cases and review of the literature. *Journal of Neurosurgery* 102: 59–64. [CrossRef]
- Feldenzer, John A., James L. McGauley, and John E. McGillicuddy. 1989. Sacral and presacral tumours: Problems in diagnosis and management. *Neurosurgery* 25: 884–91. [CrossRef]
- Fisher, Charles G., Christian P. DiPaola, Timothy C. Ryken, Mark H. Bilsky, Christopher I. Shaffrey, Sigurd H. Berven, James S. Harrop, Michael G. Fehlings, Stefano Boriani, Dean Chou, and et al. 2010. A novel classification system for spinal instability in neoplastic disease: An evidence-based approach and expert consensus from the Spine Oncology Study Group. *Spine* 35: E1221–E1229. [CrossRef] [PubMed]
- Flanagan, E. P., B. P. O'neill, A. B. Porter, G. Lanzino, T. M. Haberman, and B. M. Keegan. 2011. Primary intramedullary spinal cord lymphoma. *Neurology* 77: 784–91. [CrossRef] [PubMed]
- Fobe, J. L., K. Nishikuni, and M. A. Gianni. 1998. Evolving magnetic resonance spinal cord trauma in child: From hemorrhage to intradural arachnoid cyst. *Spinal Cord* 36: 864–66. [CrossRef] [PubMed]
- Fortuna, A., and S. Mercuri. 1983. Intradural spinal cysts. *Acta Neurochirurgica* 68: 289–314. [CrossRef] [PubMed]
- Gottfried, Oren N., Wayne Gluf, Alfredo Quinones-Hinojosa, Peter Kan, and Meic H. Schmidt. 2003. Spinal meningiomas: Surgical management and outcome. *Neurosurgical Focus* 14: e2. [CrossRef]
- Greenberg, Mark S. 2010. *Handbook of Neurosurgery*. New York: Thieme.
- Grommes, Christian, and Lisa M. DeAngelis. 2017. Primary CNS Lymphoma. *Journal of Clinical Oncology* 35: 2410–18. [CrossRef]

- Helweg-Larsen, Susanne, and Per Soelberg Sørensen. 1994. Symptoms and signs in metastatic spinal cord compression: A study of progression from first symptom until diagnosis in 153 patients. *European Journal of Cancer* 30A: 396–98. [CrossRef]
- Heuer, George J. 1929. The so-called hour-glass tumours of the spine. *Archives of Surgery* 18: 935–81. [CrossRef]
- Hoa, Michael, and William H. Slattery, III. 2012. Neurofibromatosis 2. *Otolaryngologic Clinics of North America* 45: 315–32. [CrossRef]
- Hohenberger, Christoph, Peter Hau, Karl-Michael Schebesch, Oliver Kölbl, Markus J. Riemenschneider, Fabian Pohl, Martin Proeschold, and Nils Ole Schmidt. 2023. Spinal meningiomas. *Neuro-Oncology Advances* 5: i112–i121. [CrossRef]
- Inoue, Y., Y. Nemoto, K. Ohata, H. Daikokuya, A. Hakuba, T. Tashiro, M. Shakudo, K. Nagai, K. Nakayama, and R. Yamada. 2001. Syringomyelia associated with adhesive spinal arachnoiditis: MRI. *Neuroradiology* 43: 325–30. [CrossRef]
- Jamjoom, Abdulhakim B., Bruce G. Mathew, and Hugh B. Coakham. 1991. A variant of the syndrome of spinal arachnoid cysts with multiple congenital defects. *British journal of neurosurgery* 5: 77–82. [CrossRef] [PubMed]
- Kam, Tsz Yeung, Oscar Siu Hong Chan, Albert Wai Man Hung, and Rebecca Mei Wan Yeung. 2019. Utilization of stereotactic ablative radiotherapy in oligometastatic & oligoprogressive skeletal metastases: Results and pattern of failure. *Asia-Pacific Journal of Clinical Oncology* 15: 14–19. [CrossRef] [PubMed]
- Kang, H. S., C. K. Chung, and H. J. Kim. 2000. Spontaneous spinal subdural hematoma with spontaneous resolution. *Spinal Cord* 38: 192–96. [CrossRef] [PubMed]
- Katonis, P., G. Datsis, A. Karantanas, A. Kampouroglou, S. Lianoudakis, S. Licoudis, E. Papoutsopoulou, and K. Alpantaki. 2013. Spinal Osteosarcoma. *Clinical Medicine Insights: Oncology* 7: S10099. [CrossRef] [PubMed]
- Khalid, Syed I., Owoicho Adogwa, Ryan Kelly, Ankit Metha, Carlos Bagley, Joseph Cheng, and John O'Toole. 2018. Adult spinal Ependymomas: An epidemiologic study. *World Neurosurgery* 111: e53–61. [CrossRef]
- Kim, Phyo, Michael J. Ebersold, Burton M. Onofrio, and Lynn M. Quast. 1989. Surgery of spinal nerve schwannoma. Risk of neurological deficit after resection of involved root. *Journal of Neurosurgery* 71: 810–14. [CrossRef]
- Klekamp, Jörg, and Madjid Samii. 2007. Epidural Tumors. In *Surgery of Spinal Tumors*. Berlin and Heidelberg: Springer. [CrossRef]
- Kopelson, Gene, Rita M. Linggood, George M. Kleinman, Joanne Doucette, and C. C. Wang. 1980. Management of intramedullary spinal cord tumours. *Radiology* 135: 473–79. [CrossRef]
- Kurland, Leonard T. 1958. The frequency of intracranial and intraspinal neoplasms in the resident population of Rochester, Minnesota. *Journal of Neurosurgery* 15: 627–41. [CrossRef]
- Landi, Alessandro, Valeria Palmarini, Alessandro D'Elia, Nicola Marotta, Maurizio Salvati, Antonio Santoro, and Roberto Delfini. 2016. Magnetic resonance diffusion tensor imaging and fiber-tracking diffusion tensor tractography in the management of spinal astrocytomas. *World Journal of Clinical Cases* 4: 1–4. [CrossRef]
- Lang, Ning, Xiao-Guang Liu, and Hui-Shu Yuan. 2012. Malignant peripheral nerve sheath tumour in spine: Imaging manifestations. *Clinical Imaging* 36: 209–15. [CrossRef]
- Le, Robert, Jeremy D. Tran, Mel Lizaso, Ramin Beheshti, and Austin Moats. 2018. Surgical Intervention vs. Radiation Therapy: The Shifting Paradigm in Treating Metastatic Spinal Disease. *Cureus* 10: e3406. [CrossRef]
- Levy, Walter J., John Latchaw, Joseph F. Hahn, Buphinda Sawhny, Janet Bay, and Donald F. Dohn. 1986. Spinal neurofibromas: A report of 66 cases and a comparison with meningiomas. *Neurosurgery* 18: 331–34. [CrossRef] [PubMed]
- Liigant, Aive, Toomas Asser, Andres Kulla, and Ain-Elmar Kaasik. 2000. Epidemiology of primary central nervous system tumours in Estonia. *Neuroepidemiology* 19: 300–11. [CrossRef] [PubMed]
- Love, J. Grafton, and H. W. Dodge. 1952. Dumbbell (hourglass) neurofibromas affecting the spinal cord. *Surgery, Gynecology & Obstetrics* 94: 161–72.
- Malis, Leonard I. 1978. Intramedullary Spinal Cord Tumours. *Clinical Neurosurgery* 25: 512–39. [CrossRef] [PubMed]
- Mallucci, C. L., R. J. Stacey, J. B. Miles, and B. Williams. 1997. Idiopathic syringomyelia and the importance of occult arachnoid webs, pouches, and cysts. *British Journal of Neurosurgery* 11: 306–9. [CrossRef]
- Materljan, Eris, Branka Materljan, J. Sepcic, Lidija Tuskan-Mohar, Gordana Zamolo, and Irene Erman-Baldini. 2000. Epidemiology of central nervous system tumours in Labin area, Croatia, 1974–2001. *Croatian Medical Journal* 45: 206–12.
- McCormick, Paul C. 1996. Surgical management of dumbbell and paraspinal tumours of the thoracic and lumbar spine. *Neurosurgery* 38: 67–74. [CrossRef]

- McCormick, Paul C., Kalmon D. Post, and Bennett M. Stein. 1990. Intradural extramedullary tumours in adults. *Neurosurgery Clinics of North America* 1: 591–608. [CrossRef]
- Menezes, Arnold H., and Vincent C. Traynelis. 2006. Spinal neurenteric cysts in the magnetic resonance imaging era. *Neurosurgery* 58: 97–105. [CrossRef]
- Moore, Anne J., and David W. Newell. 2006. *Tumour Neurosurgery*. London: Springer.
- Mridha, A. R., M. C. Sharma, C. Sarkar, V. Suri, A. Rishi, A. Garg, and A. Suri. 2007. Myxopapillary ependymoma of lumbosacral region with metastasis to both cerebellopontine angles: Report of a rare case. *Child's Nervous System* 24: 1209–13. [CrossRef] [PubMed]
- Mundy, Gregory R. 2002. Metastasis to bone: Causes, consequences and therapeutic opportunities. *Nature Reviews Cancer* 2: 584–93. [CrossRef] [PubMed]
- Nakamizo, Tomoki, Haruhisa Inoue, Fukashi Uda, Masaya Oda, Makoto Kawai, Kengo Uemura, Makio Takahashi, Kazuto Nishinaka, Hideyuki Sawada, Masakuni Kameyama, and et al. 2002. Magnetic resonance imaging of primary spinal intramedullary lymphoma. *Journal of Neuroimaging* 12: 183–86. [CrossRef] [PubMed]
- Osborn, Virginia W., Anna Lee, and Yoshiya Yamada. 2018. Stereotactic Body Radiation Therapy for Spinal Malignancies. *Technology in Cancer Research & Treatment* 17. [CrossRef]
- Osenbach, Richard K., John C. Godersky, Vincent C. Traynelis, and Robert D. Schelper. 1992. Intradural extramedullary cysts of the spinal canal: Clinical presentation, radiographic diagnosis, and surgical management. *Neurosurgery* 30: 35–42. [CrossRef]
- Ozawa, Hiroshi, Shoichi Kokubun, Toshimi Aizawa, Takeshi Hoshikawa, and Chikashi Kawahara. 2007. Spinal dumbbell tumours: An analysis of a series of 118 cases. *Journal of Neurosurgery: Spine* 7: 587–93. [CrossRef]
- Park, Howard Y., Sara K. Yang, William L. Sheppard, Vishal Hegde, Stephen D. Zoller, Scott D. Nelson, Noah Federman, and Nicholas M. Bernthal. 2016. Current management of aneurysmal bone cysts. *Current Reviews in Musculoskeletal Medicine* 9: 435–44. [CrossRef]
- Patchell, Roy A., Phillip A. Tibbs, William F. Regine, Richard Payne, Stephen Saris, Richard J. Kryscio, Mohammed Mohiuddin, and Byron Young. 2005. Direct decompressive surgical resection in the treatment of spinal cord compression caused by metastatic cancer: A randomised trial. *Lancet* 366: 643–48. [CrossRef]
- Patwardhan, Vasudha, Tufail Patanakar, Diane Armao, and Suresh K. Mukherji. 2000. MR imaging findings of intramedullary lipomas. *American Journal of Roentgenology* 174: 1792–93. [CrossRef]
- Perret, George, David Green, and John Keller. 1962. Diagnosis and treatment of intradural arachnoid cysts of the thoracic spine. *Radiology* 79: 425–29. [CrossRef]
- Preston-Martin, Susan. 1990. Descriptive epidemiology of primary tumours of the spinal cord and spinal meninges in Los Angeles County, 1972–1985. *Neuroepidemiology* 9: 106–11. [CrossRef] [PubMed]
- Requena, Luis, and Omar P. Sangüeza. 1995. Benign neoplasms with neural differentiation: A review. *The American Journal of Dermatopathology* 17: 75–96. [CrossRef] [PubMed]
- Rosenkranz, Wolf. 1971. Ankylosing spondylitis: Cauda equina syndrome with multiple spinal arachnoid cysts. Case report. *Journal of Neurosurgery* 34: 241–43. [CrossRef] [PubMed]
- Safaei, Michael, Andrew T. Parsa, Nicholas M. Barbaro, Dean Chou, Praveen V. Mummaneni, Philip R. Weinstein, Tarik Tihan, and Christopher P. Ames. 2015. Association of tumour location, extent of resection, and neurofibromatosis status with clinical outcomes for 221 spinal nerve sheath tumours. *Neurosurgical Focus* 39: E5. [CrossRef] [PubMed]
- Schaller, Bernhard. 2005. Spinal meningioma: Relationship between histological subtypes and surgical outcome? *Journal of Neuro-Oncology* 75: 157–61. [CrossRef]
- Seppälä, Matti T., Matti J Haltia, Risto J. Sankila, Juha E. Jääskeläinen, and Olli Heiskanen. 1995. Long-term outcome after removal of spinal schwannoma: A clinicopathological study of 187 cases. *Journal of Neurosurgery* 83: 621–26. [CrossRef]
- Shaw, Pamela J., David A. Allcutt, David Bates, and Peter J Crawford. 1990. Cauda equina syndrome associated with multiple lumbar arachnoid cysts in ankylosing spondylitis: Improvement following surgical therapy. *Journal of Neurology, Neurosurgery & Psychiatry* 53: 1076–79. [CrossRef]
- Shen, Shu-Huei, Jiing-Feng Lin, Feng-Chi Chang, Jing-Ying Lee, Chao-Bao Luo, Shing-Su Chen, and Cheng-Yen Chang. 2001. Magnetic resonance imaging appearance of intradural spinal lipoma. *Chinese Medical Journal (Taipei)* 64: 364–68.
- Solero, Carlo L., Maurizio Fornari, Sergio Giombini, Giovanni Lasio, Giuseppe Oliveri, Carlo Cimino, and Franco Pluchino. 1989. Spinal meningiomas: Review of 174 operated cases. *Neurosurgery* 25: 153–60. [CrossRef]

- Sonneland, Paula R. L., Bernd W. Scheithauer, and Burton M. Onofrio. 1985. Myxopapillary ependymoma. A clinicopathologic and immunocytochemical study of 77 cases. *Cancer* 56: 883–93. [CrossRef]
- Sowash, Madeleine, Ori Barzilai, Sweena Kahn, Lily McLaughlin, Patrick Boland, Mark H. Bilsky, and Ilya Laufer. 2017. Clinical outcomes following resection of giant spinal schwannomas: A case series of 32 patients. *Journal of Neurosurgery: Spine* 26: 494–500. [CrossRef] [PubMed]
- van den Bent, Martin J. 2005. Surgical resection improves outcome in metastatic epidural spinal cord compression. *Lancet* 366: 609–10. [CrossRef] [PubMed]
- Wakai, Susumu, and Chin-Wen Chiu. 1984. Rare combination of spinal lesions and spina bifida occulta: Case report. *Developmental Medicine & Child Neurology* 26: 117–21. [CrossRef]
- Wang, Jing, Xiang-zhi Ni, Ming-lei Yang, Xing Huang, Shu-ming Hou, Cheng Peng, Jia-shi Cao, and Tie-Long Liu. 2023. Prognostic factors and treatment outcomes of spinal osteosarcoma: Surveillance, epidemiology, and end results database analysis. *Frontiers in Oncology* 13: 1083776. [CrossRef]
- Wang, Michael Y., Allan DO Levi, and Barth A. Green. 2003. Intradural spinal arachnoid cysts in adults. *Surgical Neurology* 60: 49–55. [CrossRef]
- Yang, Xiong-gang, Deng-xing Lun, Yong-cheng Hu, Yong-heng Liu, Feng Wang, Jiang-tao Feng, Kun-chi Hua, Li Yang, Hao Zhang, Ming-you Xu, and et al. 2018. Prognostic effect of factors involved in revised Tokuhashi score system for patients with spinal metastases: A systematic review and Meta-analysis. *BMC Cancer* 18: 1248. [CrossRef]
- Zibis, A. H., A. Markonis, and A. H. Karantanas. 2000. Unusual causes of spinal foraminal widening. *European Radiology* 10: 144–48. [CrossRef]
- Ziu, Endrit, Vibhu Krishnan Viswanathan, and Fassil B. Mesfin. 2022. Spinal Metastasis. In *StatPearls [Internet]*; Treasure Island: StatPearls Publishing. Available online: <https://www.ncbi.nlm.nih.gov/books/NBK441950/?cv=1&msclkid=b63e0727cbd611e> (accessed on 23 May 2023).

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