# Imaging Guidelines and Recommendations for Diagnosis, Surveillance, and Management of Pediatric CNS and Spinal Tumors

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## Abstract

Central nervous system (CNS) tumors are the second most common cause of cancer in children when incidence rates of cancer are estimated according to the Indian population dynamics based on 2011 consensus. As per the estimates, CNS tumors account for 20.1% of cancer burden in children aged between 0 and 14 years and 16.8% when 0 to 19 years age group is considered. The most common pediatric brain tumors are astrocytoma and medulloblastoma followed by other embryonal tumors, craniopharyngioma, and ependymal tumors. The incidence of CNS tumors in children from India is similar to the western high-income countries, other than slightly higher incidence of craniopharyngioma in Indian children.

**Keywords**
- MRI
- pediatric brain tumors
- protocol
- spinal tumor

## Introduction

Central nervous system (CNS) tumors are the second most common cause of cancer in children when incidence rates of cancer are estimated according to the Indian population dynamics based on 2011 consensus. As per the estimates, CNS tumors account for 20.1% of cancer burden in children aged between 0 and 14 years and 16.8% when 0 to 19 years age group is considered.[1] The most common pediatric brain tumors are astrocytoma and medulloblastoma followed by other embryonal tumors, craniopharyngioma (CP), and ependymal tumors.[2] The incidence of CNS tumors in children from India is similar to the western high-income countries

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(HIC), other than slightly higher incidence of CP in Indian children.[2,3]

## Symptoms/Presentation

Central nervous system (CNS) tumors are the most common solid tumors in children. The worldwide incidence of pediatric CNS tumors varies from 1.12 to 5.14 per 1,00,000 individuals.[4] The peak age for pediatric CNS tumors is less than 5 years after which the incidence of CNS tumors is less till up to 15 years of age after which the incidence rates are almost like adults.[5] In the Indian population, the age-adjusted rates for the incidence of childhood CNS tumors range from 6.6 to 19.8 per million for boys and 3.0 to 16.0 per million for girls.[6]

The common presenting symptoms of pediatric CNS tumors are headache, vomiting, seizures, or cranial nerve deficits, which are quite nonspecific. This can lead to delayed or missed diagnosis.

## Prognosis/Survival

The data to evaluate prognosis or study survival statistics in children with CNS tumors in India is scarce. The overall incidence of pediatric CNS tumors in India is low compared to the HIC, probably due to missed diagnosis or failure to recognize symptoms attributable to CNS tumors. As per a study from South India with data on institutional follow-up for pediatric CNS tumors, the mortality was found to be approximately 15.3% with significant treatment drop-out.[7] The 5-year survival data from a systematic review of worldwide literature from 1980 to 2009 concludes variability in survival statistics over the decades depending on tumor grade. There was significant improvement in survival for embryonal tumors (37% in 1980 to 60% in 2009), whereas the survival for astrocytoma changed very little (78% in 1982 to 89% in 2009) over the years.[8]

## Role of Imaging

Magnetic resonance imaging (MRI) is the modality of choice for initial baseline assessment of a brain tumor and for follow-up imaging to assess for treatment response. Computed tomographic (CT) scan can be useful for certain cases when the intracranial lesion is heavily calcified (e.g., CP) and for tumors near the skull base or involving the skull vault to assess for bony changes. However, CT scan is usually always performed in conjunction with an MRI for complete assessment.

Across India, there are multiple cancer treatment hospitals, either managed by the government or run privately. The various institutions have a variety of MRI/CT scanners from different vendors with the MRI field magnet strength ranging from 0.5T to 3T. Also, many scans are performed at standalone private diagnostic centers and images are provided as hard prints on films. Some of the other challenges in pediatric brain/spine imaging are lack of anesthetic support or lack of expertise in reporting those scans. This greatly limits the prospect of multicenter study trials due to the inhomogeneity in scan planning and acquisition. It is a pressing priority to have a standardized imaging protocol across various institutions to be able to include more patients in pediatric CNS tumor trials and improve treatment outcomes.

The Response Assessment in Pediatric Neuro-Oncology (RAPNO) committee have recently published recommendations for image acquisition and have clearly defined response criteria for the pediatric brain tumors.[9–12] Based on these recommendations, the European Society of Paediatric Oncology Brain Tumour imaging Working Group has provided their own recommendations for pediatric brain tumor imaging that consists of minimal essential/mandatory sequences and additional sequences including advanced imaging methods that can be performed as per local imaging policy.[13] Applying these recommendations to the Indian scenario poses unique challenges as discussed above and thus we have formed imaging guidelines that will most suite to the Indian subcontinent in terms of utilizing the limited imaging capacity and improvise on the reporting standards with a clear guideline for timing the scans. We have also included templates for reporting formats to achieve minimal variability in reporting standards and facilitate wider acceptance of cases for future pediatric CNS tumor trials.

### Pediatric CNS Tumor Grade on Imaging

The conventional T2-weighted (T2W) and fluid-attenuated inversion recovery (FLAIR) sequences give a good overview of the extent as well as the characteristics of the lesion with information on perilesional edema and mass effect on surrounding structures. The highly cellular lesions cause T2 shortening and generally appear iso to hypointense on T2W images. FLAIR signal changes combined with diffusion-weighted imaging/apparent diffusion coefficient (DWI/ADC) maps give information on perilesional edema versus tumor infiltration. Some low-grade gliomas (LGGs) have a bubbly appearance on imaging with a FLAIR hyperintense rim (highly suggestive of a dysembryoplastic neuroepithelial tumour [DNET]). Calcifications can be seen in low-grade tumors like pilocytic astrocytoma (PA), intermediate grade lesions like supratentorial ependymomas (EP) and in high-grade tumors like atypical teratoid rhabdoid tumor (ATRT). However, when the lesion is largely calcified with no associated enhancing component or abnormal diffusion changes, it is usually a low-grade lesion. T1W imaging is helpful specially to look for hemorrhage or calcification in the lesion as gradient or susceptibility imaging sequences are not generally part of tumor imaging protocol. The other important utility of precontrast T1W imaging is on the postoperative scan to distinguish postoperative hemorrhage from abnormal postcontrast enhancement along the resection margins that can indicate residual disease.

Postcontrast enhancement at baseline imaging in a pediatric CNS tumor does not play as big a role as it does for adult CNS tumors, as it does not reflect tumor grade in pediatric cases. However, postcontrast enhancement is particularly important in some metastasizing low-grade tumors (e.g., 5–10% of PA can metastasize) that only show enhancement

and this can be the only feature on imaging to pick up smaller lesions or leptomeningeal spread.[14] Enhancement plays an important role for tumor follow-up (f/u) for the lesions that showed enhancement at baseline imaging, to map the tumor extent or recurrence on f/u imaging (e.g., PA) and also for nonenhancing lesions at baseline that show abnormal postcontrast enhancement on f/u suggesting progression or change in lesion grade (e.g., World Health Organization [WHO] grade I diffuse astrocytoma *MYB or MYBL1 altered*).[14] Postcontrast imaging can also help to assess tumor response as LGG show significantly reduced enhancement after treatment with MEK inhibitors or BRAF inhibitors; however, this should be weighed with tumor size measurement on T2W/FLAIR imaging.[9]

DWI with ADC maps is one of the most important sequences for pediatric CNS tumor grading. Tumors can be graded by obtaining ADC value of the lesion by drawing a region of interest on the ADC map where the lower ADC values correspond to higher grade and cellularity. Some higher grade lesions do not show postcontrast enhancement and in such cases a lower ADC value can help assess for high tumor cellularity as well as for nonenhancing metastasis, a classic example being leptomeningeal and spinal metastasis in grade 4 medulloblastoma showing a contrast enhancement-DWI mismatch.[15] Diffusion has a crucial role in determining progression for the nonenhancing tumors on antiangiogenic therapy and to distinguish recurrence from pseudo-progression in high-grade tumors.[16,17]

## Pediatric Brain Tumor Types

The pediatric brain tumors have several classification systems that are based on histology, molecular features, and/or site of origin of the lesion. Approximately two-third of CNS tumors in adults are supratentorial, whereas, in children approximately two-third of CNS tumors are infratentorial. Some genetic syndromes like neurofibromatosis (NF) type 1 and 2, tuberous sclerosis and Li-Fraumeni syndrome show propensity to develop brain tumors.

The most recent update by the new WHO 2021 classification of CNS tumors (WHO CNS 5) have introduced and merged many categories form the previous classification. The WHO CNS5 have classified brain tumors mainly based on the combination of histological and molecular features. The common pediatric infratentorial tumors are medulloblastoma, cerebellar astrocytoma, EP, brain stem diffuse midline glioma (including diffuse intrinsic pontine glioma [DIPG]), and ATRT. The common pediatric supratentorial tumors are LGG, high-grade gliomas (HGG), embryonal tumors, pituitary tumors, and pineal tumors.

### Low-Grade Gliomas and Other Low-Grade Tumors (WHO grade I/11)

LGG are tumors of glial origin and are WHO grade I and II tumors. LGG are the most common pediatric CNS tumors accounting for nearly 40 to 50% of all CNS tumors.[18] In the WHO CNS5 classification, pediatric LGGs are classified as circumscribed, diffuse, and glioneuronal tumors. The LGG commonly arise in the hypothalamic-chiasmatic region (40%), cerebellum (25%), and cerebral hemispheres (17%), with a small proportion occurring in the brain stem (9%).[18,19] Optic pathway lesions are generally more diffuse and quite extensive in children often involving the hypothalamic–chiasmatic axis when sporadic. When associated with a cancer predisposition syndrome, the LGG are classically seen involving the optic pathway and brain stem (10–15% of NF type 1 patients).[20] PA is the most common LGG (~16%).[21] The other less common low-grade lesions are diffuse astrocytoma, pilomyxoid astrocytoma, pleomorphic xanthoastrocytoma, and DNET. The recent 2021 WHO classification of CNS tumors have added a new category for diffuse LGGs with four entities under this category: diffuse astrocytoma, MYB- or MYBL1-altered; angiocentric glioma; polymorphous low-grade neuroepithelial tumor of the young; and diffuse LGG, MAPK pathway-altered.[22]

### High-Grade Gliomas and Embryonal Tumors

HGG encompass a variety of WHO grade III and IV glial tumors and the second most common pediatric CNS tumors. The common HGG gliomas are anaplastic astrocytoma (WHO III), glioblastoma multiforme (WHO IV), and diffuse midline gliomas. The new diffuse pediatric HGG category in the new 2021 WHO CNS tumor classification has four entities under it: diffuse midline glioma (H3 K27-altered), diffuse hemispheric glioma (H3 G34-mutant), diffuse pediatric-type HGG (H3-wild-type and IDH-wild-type), and infant-type hemispheric glioma.[22]

Embryonal tumors are one of the most common high-grade CNS lesions in children and medulloblastoma is the most common CNS embryonal tumor accounting for 10-15% of pediatric CNS tumors.[12] The new WHO CNS5 classification has molecularly divided medulloblastoma (MBL) into four categories (WNT-activated, sonic hedgehog [SHH]-activated TP53 wild-type, SHH-activated TP53-mutant, and non-WNT/non-SHH group that mainly comprises group 3 and 4 lesions) and the previously described histological subtypes are now combined in one category, MBL histologically defined.[22] The other common embryonal tumors are ATRTs and embryonal tumors with multilayered rosettes.

### Ependymal and other Pediatric CNS Tumors (WHO I–III)

EP are classified according histopathological/molecular features as well as anatomic site, and are divided into molecular groups across the supratentorial (ZFTA fusion-positive and YAP1 fusion-positive), posterior fossa (two groups PFA and PFB), and spinal compartments (MYCN amplified and myxopapillary EP).[22] The supratentorial EP are associated with calcification and cysts. Leptomeningeal spread can be seen in EP and is not limited to any particular tumor location. The intracranial EP have a significant risk for recurrence and the 5-year overall survival is approximately 50 to 70%.[23]

The most common pituitary tumor in children is CP accounting for 6 to 9% of pediatric CNS tumors.[24] CP arises from Rathke's pouch remnant and can be sellar or suprasellar and follows the rule of 90% (approximately 90% have calcification, 90% are cystic, and 90% show enhancement). Pituitary microadenoma and macroadenoma are the less common

tumors in this category. The adamantinomatous variety of CP is more common in children and papillary variant is more common in adults.

The common pineal tumors are germ cell tumors that can sometimes present as a bifocal tumor and can involve the neurohypophyseal region.[25] Tumor markers show good correlation with different types of germ cell tumors. Calcification is common in pineal tumors. The usual clinical presentation is due to obstructive hydrocephalus due to mass effect on the cerebral aqueduct.

## Management of Pediatric CNS Tumors

Surgery, chemotherapy, and radiotherapy (RT) are the main treatment options for pediatric CNS tumors and the choice of treatment depends on the stage of the disease, lesion grade, and anatomical location of the lesion.

Surgery is often the initial treatment choice especially when the tumor is causing significant mass effect or hydrocephalus or raised intracranial pressure. Surgery planning is mostly done on MRI with assistance from other advanced imaging techniques like diffusion-tensor imaging (DTI), functional MRI, and intraoperative monitoring with ultrasonography or intraoperative MRI. Surgery for pediatric CNS tumors is undertaken with curative intent and is extremely valuable in most of the nondiffuse CNS tumors (e.g., medulloblastoma or PA). However, the immediate postoperative MRI (<72 hours) is extremely useful to classify the surgery into subtotal, gross total, supramaximal, or complete resection. The extent of resection is particularly important in higher grade lesions for prognostication.

An extraventricular drain or a ventriculoperitoneal shunt is often required for hydrocephalus and for tumors around the cerebral aqueduct or in the posterior fossa, a third ventriculostomy may be required. For certain tumors like CP, achieving complete resection is often difficult due to its extent and involvement of adjacent structures making surgical debulking a suitable option. However, the CP cysts often fill up and in such cases an Omaya reservoir is inserted to achieve cyst drainage and can also be used for chemotherapy administration.[26]

RT is one of the main treatment modalities for unresectable pediatric CNS tumors and residual disease or for preventing recurrence. Various forms of RT can be used including the conventional photon bean therapy or brachytherapy or the upcoming proton beam therapy. Proton beam therapy is more focused with less complications due to less irradiation of the adjacent uninvolved structures.[27]

Role of chemotherapy in pediatric brain tumors is limited due to poor permeability of chemotherapeutic drugs across the brood–brain barrier; however, it is useful adjunct for certain tumors. Intrathecal chemotherapy is useful for intracranial hemato-lymphoid tumors.

## Timing for Imaging

A presurgical baseline MRI brain or spine imaging should be performed for all patients. For patients undergoing surgery, a postoperative MRI scan to assess for residual disease should be performed around 48 hours (within 24–72 hours) as per RAPNO recommendations.[9,10] This postoperative scan will then be the baseline scan for further imaging follow-up. If only biopsy is performed, then a postbiopsy scan is not required. Follow-up imaging is recommended every 3 months for surveillance in the first year and the interval can be then be slowly increased.[20,28] In some cases of relapse, follow-up imaging can be performed every 2 months.[10] Approximately 5 to 10% of pediatric CNS low-grade tumors and 10 to 30% HGG[29] present with metastasis on presentation or develop secondarily.[30] Thus, spine MRI for brain tumors should be performed at baseline; similarly, baseline brain imaging should be performed for primary spinal cord tumors (SCT). If metastasis is detected, then repeat surveillance MRI of the spine or brain is recommended by the RAPNO committee at the same intervals as the primary tumor site.[9]

NF-1-related low-grade CNS lesions should have similar imaging protocols with additional sequences specific for site, like for orbital imaging (►**Supplementary Table S1**).[9] RAPNO has defined the radiological response assessment criteria for pediatric LGG and HGG (►**Supplementary Tables S2** and **S3**).

The largest measurable lesion or lesions or the most symptomatic/actively growing lesion should be the target lesion. Measurements are usually done in two or three dimensions and the most reproducible way is to measure the longest dimension in perpendicular planes.[9]

## Introduction to Spinal Cord Tumors

SCT are classified based on location as intramedullary (IM), intradural–extramedullary (IDEM), or extradural (ED).[31] Spinal cord neoplasms are comparatively rare. They account for 5 to 10% of all central nervous system tumors, of which 70 to 80% are IDEM in location.[32] The intramedullary neoplasms can be glial and nonglial histopathologically. The glial neoplasms include EP, myxopapillary EP, subependymoma, astrocytoma, and ganglioglioma. The nonglial neoplasms include hemangioblastoma, paraganglioma, dermoid, epidermoids, lipomas, hemangiomas, metastasis, and lymphoma.[33,34] EP are the most commonly seen in adults, while astrocytomas are more common in the pediatric population. The IDEM and ED neoplasms primarily include meningioma and nerve sheath tumors.[32] Other lesions include cysts, metastases, paraganglioma, dermoid, and epidermoids. ED tumors also include the spinal column, of which bone metastasis is the most common.

## Etiopathogenesis and Epidemiology

The etiopathogenesis of most primary spinal tumors is unknown. However, exposure to cancer-causing agents may be attributed to some. A genetic role is suspected as these tumors are linked to known inherited syndromes. NF 2 and von Hippel-Lindau disease are the common ones associated with neurofibromas/meningiomas and hemangioblastomas, respectively.

There is scarce data on the incidence of primary SCT. Schellinger et al quoted an overall incidence of spinal cord tumors as 0.74 per 100,000 person-years, with an incidence of 0.77/100,000 in females and 0.70/100,000 in males.[35] Two-third of all spinal tumors are IDEM, and 10% are IM SCT.[36] According to literature, the primary spinal tumors are more commonly seen in females in the Western population, whereas a slight male preponderance is seen in Asian studies.[37,38] Male to female ratio of nearly 1.4:1 has been reported in Indian studies.[39,40] The mean age of presentation of IDEM tumors was 35.8 years, IM was 25.7 years, and ED tumors was 30.7 years.[41] In a study by Chamberlain and Tredway, the mean age of patients with intramedullary spinal cord tumor (IMSCT) was 41 years, which is higher than Indian data.[36]

## Clinical Presentation

Back pain, paresthesias, weakness in limbs, and sensory loss are the most frequent presenting symptoms. Scoliosis or other spinal deformities may occur due to weakness of paraspinal muscles when anterior horn cells of the spinal cord are involved. Autonomic dysfunction, including loss of bowel or bladder control and erectile dysfunction may also occur.[40,42,43]

## Clinical/ Diagnostic Workup Excluding Imaging

A thorough clinical history and examination with a detailed neurological assessment mark the beginning of the assessment. IM tumors have a longer duration of clinical history as compared to IDEM tumors. IDEM tumors commonly have radicular pain, whereas IM tumors present with dull aching pain. Paralysis may occur in different body parts, depending on which level of the spinal cord or corticospinal tracts are involved (distal to proximal muscle weakness occurs in IDEM and proximal to distal weakness in IM tumors). Loss of sensation in the legs, arms, or chest may occur when spinothalamic tracts are involved. Dissociative anesthesia and suspended sensory loss are the classical presentations of IM tumors. Loss of bowel or bladder function occurs earlier in intramedullary tumors than in IDEM tumors. Anterior horn cell involvement, which occurs more commonly in IM tumors, leads to paraspinal muscle wasting causing secondary scoliosis.[40,42,43]

Cerebrospinal fluid (CSF) analysis may be considered to assess tumor cells. Laboratory studies are usually not helpful in establishing the diagnosis.

To identify the location and appearance of the tumor, imaging is required in the form of an MRI or CT scan.

## Imaging Guidelines

MRI without and with intravenous gadolinium contrast is the modality of choice. It helps differentiate various spinal cord neoplasms; however, the appearances are not always pathognomic.[33] Radiographs and CT scans do not have a role in assessing the primary tumor. They may reveal associated bony changes like scoliosis, widened interpeduncular distance, and bone erosion.[33]

MRI protocol should include T2W sagittal images through the whole spine with tailored down T2 and T1 axials through the area of abnormality. Postcontrast images are at least obtained in sagittal and axial planes with an additional whole spine postcontrast screening to look for additional lesions or drop metastasis. In selected cases, DWI or DTI may be performed.[44] The recommended brain tumor protocol and spinal tumor protocol and metastatic workup imaging evaluation have been described in ►**Tables 1** and **2**, respectively.

MRI helps distinguish IM versus IDEM versus ED tumors in most cases. It helps identify the characteristic features, tailoring down the diagnostic considerations to one most likely diagnosis to be posited. Among IM neoplasm, EP is most common in adults, followed by astrocytoma. Central location, well-circumscribed lesion, presence of hemorrhage (cap sign), presence of cysts, and focal intense homogeneous enhancement are the features favoring EP. Myxopapillary EP is the most common neoplasm of the conus medullaris/filum terminale. Astrocytomas, as opposed, are poorly defined and eccentric with patchy irregular enhancement.[33,44] Meningiomas and schwannomas are the common IDEM tumors associated with NF. Meningiomas are more common in the thoracic spine followed by the cervical spine and reveal intense enhancement with a dural tail. Schwannomas are common in the dorsal spinal nerve root, reveal foraminal extension with intense enhancement, which may be heterogeneous in larger lesions.[32] Both of these can also present as ED masses.

## Principles of Management

A multidisciplinary treatment decision making is often required, comprising neurosurgeons, spinal surgeons, medical oncologists, radiation oncologists, and other medical specialists. Preoperative imaging diagnosis is essential as it guides the surgeon in appropriate planning for the excision of the tumor. Intraoperative neuromonitoring (motor evoked potentials & somatosensory evoked potentials) plays an important role in SCT surgery, especially in intramedullary tumors where tracts are in extremely close proximity to the tumor. A meticulous dissection of the tumor from the cord interface is carried out. Any intraoperative drop in motor evoked potentials or somatosensory evoked potentials alarms the surgeon and allows maximum safe resection of tumors. The main aim of surgery in SCT, especially in IMSCT, is maximum safe resection with preservation of motor and sensory function. Histopathology report guides the further line of management. Adjuvant treatment in the form of radiation therapy and chemotherapy is suggested in malignant SCT and cases of drop metastases to the spinal cord.[40,42,43,45]

## Follow-Up Imaging

MRI is the modality of choice for postoperative imaging. In the immediate postoperative period, MRI may be performed

**Table 1** Pediatric brain tumor MRI protocol recommendations (adapted from the recent RAPNO and SIOPE recommendations)[2–6]

| MRI sequence | Slice thickness/parameters | Imaging plane | Comments |
|---|---|---|---|
| **Basic protocol (essential)** | | | |
| a. T1W TSE/FSE or<br>b. 3D T1W | a. Slice thickness $\leq$ 4 mm and slice gap $\leq$1 mm<br>or<br>b. Slice thickness <1mm and no slice gap | a. Axial<br>or<br>b. Sagittal | The 3D MPR sequence should be isotropic. Avoid fat saturation technique |
| T2W TSE/FSE | Slice thickness $\leq$ 4 mm and slice gap $\leq$1 mm | Axial | None |
| T2 FLAIR TSE/FSE | Slice thickness $\leq$ 4 mm and slice gap $\leq$1 mm | Axial | None |
| DWI/ADC | Slice thickness $\leq$ 4 mm and slice gap $\leq$1 mm | Axial | A minimum of two *b*-values ($b = 0$ and $b = 1000$), preferably three b-values ($b = 0$, $b = 500$, $b = 1000$) |
| **Postcontrast**<br>a. T1W TSE/FSE or<br>b. 3D T1W | a. Slice thickness $\leq$ 4 mm and slice gap $\leq$1 mm<br>or<br>b. Slice thickness <1mm and no slice gap | a. Axial, sagittal and coronal<br>or<br>b. Sagittal | The 3D MPR sequence should be isotropic. Avoid fat saturation technique |
| **Additional sequences** | | | |
| MRI sequence | Slice thickness / parameters | Imaging plane | Comments |
| Postcontrast T2 FLAIR TSE/FSE | Slice thickness $\leq$ 4 mm and slice gap $\leq$1 mm | Axial | |
| Advanced MRI sequences | Perfusion, MR spectroscopy and DTI | | |

Abbreviations: 3D, three-dimensional; ADC, apparent diffusion coefficient; DTI, diffusion-tensor imaging; FLAIR, fluid-attenuated inversion recovery; FSE, fast spin echo; MPR, multiplanar reconstruction; MRI, magnetic resonance imaging; RAPNO, Response Assessment in Pediatric Neuro-Oncology; SIOPE, European Society of Paediatric Oncology; T1W, T1-weighted; TSE, turbo spin echo.

**Table 2** Pediatric spine MRI protocol guidelines for brain tumors and for primary spinal tumors (adapted from the recent RAPNO and SIOPE recommendations)[2–6]

| MRI sequence | Slice thickness/parameters | Imaging plane | Comments |
|---|---|---|---|
| **Spine imaging for brain tumors (metastatic workup)** | | | |
| T1W postcontrast SE/TSE | Slice thickness $\leq$ 3 mm and slice gap $<$ 0.5 mm | Sagittal | Whole spine (will need two blocks for older kids) |
| T1W postcontrast SE/TSE | Slice thickness 4–5 mm and no slice gap | Axial | Will require two blocks (upper and lower spine) |
| **Spine imaging for primary spinal tumors** | | | |
| T2W SE/TSE | Slice thickness $\leq$ 3 mm and slice gap $<$ 0.5 mm | Sagittal | Whole spine (will need two blocks for older kids) |
| T1W SE/TSE | Slice thickness $\leq$ 3 mm and slice gap $<$ 0.5 mm | Sagittal | Whole spine (will need two blocks for older kids) |
| **Postcontrast** | | | |
| T1W SE/TSE | Slice thickness $\leq$ 3 mm and slice gap $<$ 0.5 mm | Sagittal | Whole spine (will need two blocks for older kids) |
| T1W SE/TSE<br>or<br>T1W VIBE/THRIVE/LAVA | Slice thickness 4-5mm and no slice gap<br>or<br>Slice thickness $\leq$ 3 mm and no slice gap | Axial | Will require two blocks (upper and lower spine) |
| **Additional sequences** | | | |
| T2W SE/TSE | Slice thickness 4–5mm and no slice gap | Axial | At the level of abnormality |
| Heavily T2W | 3D FIESTA/CISS/SPACE/VISTA | Sagittal | At the level of abnormality |

Abbreviations: 3D, three-dimensional; MRI, magnetic resonance imaging; RAPNO, Response Assessment in Pediatric Neuro-Oncology; SIOPE, European Society of Paediatric Oncology; T1W, T1-weighted; TSE, turbo spin echo.

to assess the extent of resection. This also serves as a baseline imaging for further follow-up (pre-RT and chemotherapy baseline). It is usually performed within 24 hours following surgery before neovascularity and scarring develops. It also helps assess complications like hematoma, ischemia, infection, CSF leak, and malpositioning of hardware in symptomatic patients. Hardware positioning and integrity are better evaluated with CT.[46]

In asymptomatic patients, a routine follow-up MRI imaging is performed at 4 to 6 months postsurgery to assess for tumor recurrence or disease progression. In symptomatic patients, it again helps assess tumor recurrence, hardware failure, and treatment-related complications like compression fracture, radiation myelitis, and radiation myositis. Radiation myositis on MRI reveals edema in the radiation field with straight sharp margins extending across the muscle and subcutaneous fat. Radiation myelopathy is a rare and late complication, usually seen within 4 years after radiation therapy and dependent upon the radiation dose. MRI reveals cord edema in the early course with atrophy in delayed phase.[17]

Conflict of Interest
None declared.

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