MRI of the Posterior Cranial Fossa: Spectrum of Abnormalities in Pediatric Patients.

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Learning objectives

The posterior cranial fossa is part of the intracranial cavity, located between the foramen magnum and tentorium cerebelli. Anteriorly, it extends to the apex of the petrous temporal bone and posteriorly, it is enclosed by the occipital bone. It contains the brainstem and cerebellum.

The posterior fossa is affected by a multitude of conditions in children; which includes normal variants, developmental, inflammatory-infectious, metabolic, vascular and neoplastic diseases. An understanding of these pathologies, common as well as uncommon one, will greatly facilitate to improve patient care.

MRI has high sensitivity for detection and determining the extent of disease process. Because of its better soft tissue resolution and multiplanar capability, MRI has most potential amongst all imaging modalities to allow complete diagnosis. MRI is the imaging modality of choice for the investigation of disorders of posterior cranial fossa. Accurate interpretation of MR examinations requires an understanding of the anatomy, histology and spectrum of pathology affecting the posterior cranial fossa.

The purpose of our essay is to,

- Illustrate the spectrum of posterior cranial fossa lesions in pediatric patients.
- Describe the imaging and clinical features that will help to accurately characterize these lesions and improve diagnostic yield.
Background

The posterior fossa is affected by a multitude of conditions in children. MRI can provide important information about the anatomic location, size, and shape of the lesions as well as their mass effect on adjacent structures. The pattern of signal intensity, morphology, and involvement of other areas on MR imaging along with appropriate clinical details can help accurately characterize the disease process. Understanding the spectrum of appearances of the various posterior fossa lesions improves the diagnostic yield and facilitates patient care.

In this presentation, we will illustrate the MRI findings of various posterior fossa lesions according to the pathogenesis. We will describe the imaging features along with relevant clinical findings which will help discriminate different disease processes.

1. Normal variants:

   - **Dandy Walker variant**: A Dandy-Walker variant is a less severe posterior fossa anomaly than the classic Dandy-Walker malformation in which prominent retrocerebellar space of cerebrospinal fluid communicates freely with the fourth ventricle Fig. 1 on page 8. The vermis is usually hypoplastic and the posterior fossa is not as enlarged as in the classic Dandy-Walker malformation.

   - **Mega Cisterna Magna**: Mega cisterna magna Fig. 2 on page 8 is a developmental variation of the posterior fossa characterized by expansion of retrocerebellar cistern (cisterna magna) with normal cerebellar morphology.

2. Developmental:

   - **Arnold Chiari malformations**: The Chiari malformations are a group of defects associated with congenital caudal displacement of the cerebellum and brainstem. Chiari I malformation is most common in which peg-like cerebellar tonsils are displaced into the upper cervical canal. Chiari II malformation shows displacement of the medulla, fourth ventricle and cerebellum through the foramen magnum Fig. 3 on page 9, Fig. 4 on page 10 and is usually with associated with a lumbosacral spinal myelomeningocele. In Chiari III features similar to Chiari II are seen with an occipital and / or high cervical encephalocele. Chiari IV malformation shows severe cerebellar hypoplasia without displacement of the cerebellum through the foramen magnum.

   - **Dandy Walker malformations**: The "classic" Dandy-Walker malformation shows three features:(a)vermian hypoplasia with cephalad rotation of the vermian remnant, Fig. 8 on page 14 (b)cystic dilatation of the posterior fossa communicating with the fourth ventricle, Fig. 5 on page 11.
7 on page 13 and (c) enlargement of the posterior fossa causing an abnormally high tentorium and torcular, the latter lying above the level of the lambdoid (ie, torcular-lambdoid inversion) Fig. 6 on page 12.

- **Arachnoid cyst**: Arachnoid cyst is a congenital lesion of the arachnoid membrane that expands with CSF secretion. They are filled with clear CSF Fig. 10 on page 15, Fig. 9 on page 16 and do not communicate with the ventricular system.

- **Joubert syndrome**: Joubert anomaly also known as molar tooth midbrain-hindbrain malformation has neuromaging hallmarks of vermian hypoplasia and molar tooth sign. Molar tooth sign Fig. 11 on page 17 results from a midbrain-hindbrain malformation characterized by thickened and elongated superior cerebellar peduncle and an abnormally deep interpeduncular fossa.

### 3. Inflammatory and Infectious:

- **Tuberculosis**: Intracranial tuberculomas may occur either in isolation or combined with tuberculous meningitis. These tuberculomas are isointense to grey-matter on T1 weighted images and isointense on T2 images which may have central region of hypointensity representing abundant monocyte infiltration Fig. 12 on page 18, Fig. 14 on page 20.

- **Neurocysticercosis**: Neurocysticercosis is a neurologic parasitic disease caused by the encysted larva of the tapeworm *Taenia solium* and is the most important parasitic disease of the human central nervous system. Parenchymal neurocysticercosis is the second most common form of neurocysticercosis and is frequently found at the subarachnoid space near the gray matter-white matter junction or in the basal ganglia, cerebellum Fig. 15 on page 21, brainstem.

- **Acute Disseminated Encephalomyelitis**: Acute disseminated encephalomyelitis is a monophasic severe, acute, demyelinating disease of the CNS; usually triggered by an inflammatory response to viral infections and vaccinations. The typical MR imaging findings are widespread, bilateral, asymmetric areas of increased signal intensity on T2-weighted imaging within the white matter Fig. 18 on page 24, deep gray nuclei, and spinal cord. Infratentorial lesions are common, including the brainstem Fig. 17 on page 23 and cerebellar white matter Fig. 16 on page 22. Contrast enhancement and diffusion changes are variable and highly dependent on the stage of the disease. If DW imaging is completed within the first 7 days of clinical onset, a pattern of restricted diffusion Fig. 19 on page 25 may be seen.

### 4. Metabolic and Genetic:
• **Metachromatic Leukodystrophy:** Metachromatic leukodystrophy is a lysosomal storage diseases causing dysmyelination. It arises due to deficiency of enzyme Arylsulfatase A. Bilateral symmetrical confluent areas of signal change are seen in periventricular white matter Fig. 20 on page 26 with sparing of subcortical U fibers. Corpus callosum is invariably affected. Cerebellar white matter is also involved Fig. 21 on page 27.

• **Krabbe disease:** Krabbe Disease / Globoid Cell Leukodystrophy is an autosomal recessive disease caused due to deficiency of beta Galactocerebrosidase. Increased signal on T2W images is seen involving periventricular white matter, centrum semiovale, and deep gray matter Fig. 22 on page 28. Subcortical U fibres may be spared until late in the course of the disease. When there is corticospinal tract involvement, changes are seen in posterior limbs of the internal capsule and brainstem Fig. 23 on page 29.

• **Mitochondrial disorders:** Mitochondrial diseases are a heterogeneous group of disorders caused by defects in intracellular energy production. Kearns-Sayre syndrome is one of the mitochondrial disorder in which symptoms include ataxia, ophthalmoplegia, retinitis pigmentosa, increased cerebrospinal fluid (CSF) protein, hearing deficit, endocrinologic dysfunction and conductive heart block. On imaging both gray and white matter are affected, most often the brain stem tegmentum Fig. 25 on page 31, and the white matter of the cerebrum, cerebellum and basal ganglia Fig. 24 on page 30.

• **Maple Syrup Urine Disease:** Maple syrup urine disease (MSUD) is a very rare inborn error of amino acid metabolism, which classically affects the brain tissue resulting in impairment or death if untreated. MRI may show diffuse swelling of the brain due to extensive oedema of the white matter. The MSUD edema is seen, which involves the deep cerebellar white matter Fig. 28 on page 34, the dorsal part of the brainstem Fig. 27 on page 33, the cerebral peduncles, and the dorsal limb of the internal capsule Fig. 26 on page 32.

• **Wilson's disease:** Wilson's disease is a rare autosomal recessive disorder of copper metabolism, affecting multiple organ systems. Hyperintensity in lentiform nuclie and mesencephalic regions on T1 have been described as most common initial MR abnormality. T2 hyperintensity is also seen typically involving basal ganglia and thalamus. Axial T2 MR at midbrain level can show a "face of the giant panda" sign Fig. 29 on page 35, a characteristic of Wilson disease. This refers to the appearance of the midbrain, when the red nucleus and substantia nigra are surrounded by high T2 signal.

• **Glutaric aciduria:** Glutaric aciduria type 1 is an autosomal recessive disorder of degradation of lysine, hydroxylysine, and tryptophan caused by
deficiency of glutaryl coenzyme-A dehydrogenase. Bilateral abnormalities of the basal ganglia involving the globi pallidi, caudate nuclei, and putamina can be seen, but they usually spare the thalami. The affected deep gray matter can be atrophic. Other gray matter structures can be affected e.g. the substantia nigra and the dentate nuclei Fig. 31 on page 37. Hyperintensity of the tegmental tracts along the fourth ventricle floor has also been described. Most patients have a large head with prominence of the sylvian fissures Fig. 30 on page 36.

- **Ataxia telangiectasia**: Ataxia telangiectasia is an inherited multisystem disease characterized by cerebellar ataxia, oculomucocutaneous telangiectasias, and susceptibility to certain infections and neoplastic processes. MR findings of the brain include cerebellar vermal and hemispheric atrophy Fig. 32 on page 53, Fig. 33 on page 54 and diffuse increased white matter signal changes on T2-weighted images.

5. **Vascular**:

- **Cerebellar infarct**: Cerebellar infarction is a rare but serious neurologic event in children. It may involve any of the three arteries supplying the cerebellum: superior cerebellar artery, anterior inferior cerebellar artery and posterior inferior cerebellar artery. The causes include vasculopathies, disorders of coagulation and cardioembolic events Fig. 34 on page 38, Fig. 35 on page 39.

- **Vasculitis**: Vertebralbasilar circulation can be affected by vertebral dissection, primary CNS vasculitis Fig. 36 on page 40, fibromuscular dysplasia and vascular disease associated with neurofibromatosis which can present as posterior circulation infarcts Fig. 37 on page 41 of children.

- **Dural sinus thrombosis**: Cerebral venous thrombosis is a relatively uncommon but serious neurologic disorder. Causal factors may be classified as local (related to intrinsic or mechanical conditions of the cerebral veins and dural sinuses) or systemic (related to clinical conditions that promote thrombosis). In pediatric population the common causes include dehydration, mastoiditis Fig. 38 on page 42, meningitis, sinusitis, polycythemia, malignancies, autoimmune disorders.

6. **Neoplastic**:

- **Medulloblastoma**: Medulloblastomas are one of the commonest primary tumors of the posterior fossa in children. They account for 12-25 % of all paediatric CNS tumours, and 30-40% of paediatric posterior fossa tumours. These are highly malignant neoplasm which arises within the cerebellum, most often in the vermis Fig. 39 on page 43, Fig. 40 on page 44. Evidence of leptomeningeal metastatic spread is present in 33% of all cases at the time of diagnosis (ZUCKERGUSS).
- **Ependymoma**: Ependymoma represent a relatively broad group of glial tumours arising from differentiated ependymal cells lining the ventricles of the brain or the central canal of the spinal cord. These account for 6-12% of all paediatric brain tumours. Ependymomas are typically heterogeneous masses Fig. 41 on page 45 with areas of necrosis, calcification, cystic change Fig. 42 on page 46 and haemorrhage Fig. 43 on page 47 frequently seen. They usually extend into lateral recesses and through the foramina of Luschka and Magendie into cisterna magna Fig. 44 on page 48 hence the term *Plastic ependymoma*.

- **Pilocytic Astrocytoma**: Pilocytic astrocytoma are low-grade, relatively well-defined astrocytoma (WHO grade I). They tend to occurs in young patients and have a relatively good prognosis. These are the most common primary brain tumour of childhood. In posterior fossa they usually arise in the cerebellum Fig. 45 on page 49 and are seen as a predominantly cystic lesion with mural nodule Fig. 46 on page 50.

- **Vestibular Schwannoma**: Vestibular schwannomas are rare in children. Individuals with NF2 usually develop symptoms in their teens or early adulthood and present with bilateral vestibular schwannomas Fig. 47 on page 51.

- **Brainstem Glioma**: Brainstem gliomas consist of a heterogeneous group which account for approximately 25% of all posterior fossa tumors and are most common in children between 7 and 9 years of age. They are classified based upon the appearance as diffuse, focal, exophytic or cervicomedullary Fig. 48 on page 55, Fig. 49 on page 56.

- **Lymphoma**: Clival and temporal bone masses can project posteriorly and present as posterior fossa mass lesion. Lymphoma involving the skull base can project into posterior fossa to present as a mass Fig. 50 on page 52.
Fig. 1: Dandy Walker variant: Transverse T2 weighted MR image in a child evaluated for meningitis shows prominent retrocerebellar space of cerebrospinal fluid that communicates freely with fourth ventricle.

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**Fig. 2:** Mega Cisterna Magna: Transverse T2 weighted MR image shows prominent retrocerebellar collection of CSF and normal cerebellum.

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Fig. 3: Arnold Chiari (type II) malformations: Sagittal T2 weighted image in a child with meningomyelocele demonstrates inferiorly displaced vermis behind the medulla, medullary kinking, concave clivus and small posterior fossa with low lying torcular herophili.

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**Fig. 4:** Sagittal T1W image demonstrates small posterior fossa with low lying transverse sinuses and towering of cerebellum.

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Fig. 5: Dandy Walker malformations: Axial T2 weighted image shows enlarged posterior fossa and communication of the anterior fourth ventricle with its large retrocerebellar portion. Bilateral temporal horns are dilated due to obstructive hydrocephalus.
Fig. 6: Sagittal Ti-weighted image demonstrates the hypoplastic vermis, which is rotated anterosuperiorly by the markedly enlarged fourth ventricle.

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Fig. 7: Dandy Walker malformations: Transverse SS-FSE T2 weighted image in a 28 gestational week old fetus of twin pregnancy shows cystic dilatation of the fourth ventricle causing enlargement of the posterior fossa.

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Fig. 8: Sagittal SS-FSE T2 weighted image in the second fetus of the same twin pregnancy shows enlarged posterior fossa and hypoplastic displaced vermis.

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Fig. 10: Arachnoid cyst: Transverse T2 weighted image shows the cyst contents following CSF signal. Lesion extends into the basal cisterns and causes hydrocephalus.

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**Fig. 9:** Arachnoid cyst: Sagittal T1W image shows a huge cyst arising from the prepontine cistern causing mass effect on the brain stem.

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Fig. 11: Joubert syndrome: Transverse T2 weighted image in an infant with hypotonia and developmental delay shows classic Molar Tooth sign including thickened, elongated, parallel and horizontally oriented superior cerebellar peduncles.

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**Fig. 12:** Tuberculosis: Axial T2 weighted image at level of mid brain shows a giant tuberculoma involving the vermis which shows typical hypointense signal with hyperintense rim. Vasculitic infarcts are seen in the left basal ganglia and tegmentum.

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Fig. 13: Axial diffusion weighted image in same case shows restricted diffusion in left basal ganglia and tegmentum.

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Fig. 14: Single voxel long TE MR spectroscopy depicts predominant lipid peak at 1.33 ppm. A small Choline peak is also seen to resonate at 3.2 ppm.

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**Fig. 15:** Neurocysticercosis: Transverse T2 MR image of the brain shows cystic lesion with eccentric scolex suggesting vesicular neurocysticercosis in cerebellum. Further cysticerci are also seen in pterygoid muscles.

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**Fig. 16:** Acute Disseminated Encephalomyelitis: Axial T2W image in a child presenting with acute onset loss of sensorium shows multiple hyperintense areas in cerebellum.

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**Fig. 17:** Similar lesions are seen in the bilateral middle cerebellar peduncle of the same child.

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**Fig. 18:** Axial T2W image at the level of thalamus depicts multiple lesions in the left basal ganglia and bilateral cerebral white matter.

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**Fig. 19:** Axial diffusion weighted image shows restriction of diffusion within the lesions.

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**Fig. 20:** Metachromatic Leukodystrophy: Transverse T2W image in a child with loss of milestones and blindness shows bilateral symmetrical confluent areas of signal change in periventricular white matter with sparing of subcortical U fibers.

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Fig. 21: Transverse T2 weighted image at the level of midbrain depicts involvement of cerebellar white matter.

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**Fig. 22:** Krabbe disease: Axial T2 MR image in a child with recurrent seizures and tonic posturing depicts increased signal in periventricular white matter, centrum semiovale and deep gray matter.

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Fig. 23: Axial T2 MR image of same patient at the level of midbrain shows involvement of tegmentum.

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**Fig. 24:** Mitochondrial disorders: Transverse T2W MR image in a child presenting with progressive external ophthalmoplegia demonstrates bilaterally symmetric hyperintense signal in the basal ganglia.

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**Fig. 25:** Axial T2 weighted image in the same child shows signal changes within the midbrain.

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Fig. 26: Maple Syrup Urine Disease: Axial T2 MR image in child with poor feeding, lethargy shows involvement of the posterior internal capsule and globus pallidus.

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Fig. 27: Transverse T2 MR image depicts diffuse edema involving upper brainstem.

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**Fig. 28:** Coronal FLAIR MR image shows increased signal in the cerebellar white matter.

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**Fig. 29:** Wilson's disease: Transverse T2W MR image in child presenting with personality changes and movement disorders show classic "face of the giant panda" sign.

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**Fig. 30:** Glutaric aciduria: Axial T2W MR image in a child presenting with macrocephaly and regression of milestones demonstrates markedly widened sylvian fissures with wide open opercula. There is also diffuse signal abnormality in white matter and lentiform nuclei with atrophy of caudate nucleus.

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Fig. 31: Coronal FLAIR MR image of same patient demonstrates increased signal intensity within dentate nuclei.

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Fig. 34: Cerebellar infarct: Transverse T2 weighted image at the level of midbrain in a child presenting with acute ataxia shows multiple wedge shaped hyperintense areas in bilateral cerebellum.

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Fig. 35: Transverse diffusion weighted image at the same level shows restriction of diffusion within the lesions.

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Fig. 36: Vasculitis: 3D TOF angiogram image in the same child shows non-visualization of the distal basilar artery due to vasculitic involvement.

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**Fig. 37**: Single voxel long TE MR spectroscopy from the infarct depicts inverted doublet lactate peak at 1.3ppm. The NAA peak at 2.0 ppm is reduced.

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**Fig. 38:** Dural sinus thrombosis: Transverse T1 weighted image of a child with mastoiditis reveals hyperintense thrombus within the left tranverse sinus

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Fig. 39: Medulloblastoma: Axial T2W image depicts well defined, lobulated midline mass in posterior fossa arising from the vermis and projecting in 4th ventricle.

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**Fig. 40:** Coronal contrast enhanced T1W image shows intense enhancement within the lesion.

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Fig. 41: Ependymoma: Sagittal T1 weighted image shows an irregular, lobulated tumor arising from the floor of forth ventricle causing obstructive hydropscephalus. Posteriorly mass extends into cistern magna and posterior spinal canal.

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**Fig. 42:** Transverse T2W image shows the heterogeneous appearance of the mass with cystic areas.

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**Fig. 43:** Transverse gradient echo image shows multiple areas of blooming due to hemorrhage/calcification within the mass.

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**Fig. 44:** Coronal post contrast image shows heterogeneous enhancement of the mass which is extending through foramen of Magendie.

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Fig. 45: Pilocytic Astrocytoma: Transverse T2W image of a child presenting with ataxia shows large midline cystic lesion with eccentric mural nodule.

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Fig. 46: Transverse contrast enhanced T1W image shows intense enhancement of the solid mural nodule.

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**Fig. 47:** Vestibular Schwannoma in Neurofibromatosis 2: Post contrast T1 weighted image in a child presenting with hearing loss shows well defined heterogenous enhancing masses in bilateral cerebellopontine angles with extension along IAC giving "ice-cream cone" appearance.

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**Fig. 50:** Lymphoma: Mid sagittal T1 weighted image of brain in a child with Burkitt lymphoma shows heterogeneous clival mass extending into posterior fossa. There is also diffuse involvement of the mandible.

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**Fig. 32:** Ataxia telangiectasia: Axial T2 weighted image in a patient with history of ataxia, headache and conjunctival telangiectasias shows bilateral cerebellar and vermian atrophy.

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**Fig. 33:** Sagittal T1-weighted MR image of same patient shows cerebellar atrophy with normal morphology of spinal cord.

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**Fig. 48:** Axial T2W image shows diffuse expansion of brain stem by ill defined hyperintense mass lesion. The mass is also involving left middle cerebellar peduncle and causing obstrutive hydrocephalus.

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**Fig. 49:** Axial post contrast image shows mild enhancement within the pontine mass.

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Findings and procedure details

MR imaging of the brain was performed on a 1.5 T magnet system. Protocol included sagittal T1, axial T2, coronal FLAIR, diffusion weighted imaging. Contrast enhanced imaging, MR angiography, MR spectroscopy supplemented the study whenever relevant according to the results.
Conclusion

A multitude of pathologies affect the posterior cranial fossa in children. Magnetic resonance imaging is an excellent noninvasive modality for evaluating posterior fossa. The pattern of signal intensity alteration, morphology, and location on MR imaging along with appropriate clinical details can help accurately characterize these lesions imperative for optimal management.
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