The Cerebellum: A review of Normal anatomy, pathologic conditions and imaging features.

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Learning objectives

To review the normal anatomy of Cerebellum and illustrate pictures and US, CT and MR images.

To discuss the imaging diagnostic features of cerebellar lesions based on the etiologic classification

Fig 1. on page 2

Images for this section:

The Cerebellum: A review of normal Anatomy, Pathologic conditions and Imaging features

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Fig. 1: title
Background

The *cerebellum* plays an important role in motor control it is also involved in some cognitive functions. The knowledge of basic embryology, normal and functional anatomy of the cerebellum allows understanding clinicopathologic manifestations of cerebellar diseases.

In order to assess the normal anatomy and abnormal conditions of cerebellum various imaging techniques can be use, including US, CT and MR imaging. We describe the salient imaging features of the most commons pathologic conditions of cerebellum

**Imaging findings OR Procedure details**

**Embryology**: The formation of the central nervous system starts at the beginning of the *third week* with the appearance of the neural plaque in the ectoderm(fig 1c). The folds of the neural plaque fuse to form the neural tube (fig 1d), which in the fourth week is composed by three encephalic vesicles; the prosencephalon, mesencephalon, and rhombencephalon (fig2), and the rest of the neural tube will compose the spinal cord.

In the *fifth week*, one of the encephalics vesicles, the rhombencephalon, splits into the metencephalon which then will be the cerebellum and the protuberance, and the myelencephalon which will originate the medulla oblongata (fig 2).

Like all the structures of the neural tube, the metencephalon is formed by alares plaques and basales plaques fig (3a).

In the *sixth week* the differentiation of the cerebellum starts with the thickening of the alares plaques which form the rhombic lips (fig3b). These lips get compressed and form the cerebellar plaque which at the end of the third month is built by the vermis in the middle line and the cerebellar hemispheres on the sides (fig 3e). Short time after begins the formation of the fissures which come across between hemispheres through the vermis splitting them in lobes and folias. The first one to show up is the posterolateral fissure which separates from the rest of the cerebellum the most ancestral region, the floculonodular lobulo (fig 4a). In the *fourth month* a new fissure can be seen, the prima fissure (fig 4a), which is followed by a series of creases and fissures which are completed around the seventh month (fig 4d).

The cerebellar plaques consist of three layers: marginal, manto and neuroepitelial. The last one is the producer of the cerebellar cells which migrate to the external surface to form the cortex and some other to the mid part of the vermis and from the hemispheres to form the cerebellar nucleus (fig 4d).
Simultaneously the cerebellar conncetions develop with all the different parts of the neural tube. The higher cerebellar peduncle which connects them with the mesencephalon, the middle ones which joins them with the bridge and the lower ones which connects them with the bulb (fig 5). In its path these fascicles strengthen the lateral walls of the fourth ventricle. Between both higher peduncles the roof of the fourth ventricle is strengthened by a thin layer of nerve fibers called the front medular veil.

**Cerebellum generalities:** The adult human cerebellum weighs 150 qm, and its surface area is about 1000m²; it is situated in the posterior cranial fossa, anatomical areas are in the midline vermis and cerebellar hemispheres. The crust is symmetrical and is composed of three layers (molecular, Purkinje cell and granular layer), the cerebellum mostly related to motor functions and some cognitive functions.

The higher face is flat and is separated from both occipitales lobes by the dura also called the cerebellum tent or tentorium, the vermis stands out in the middle line like a bump called the mound. The lower surface of the cerebellar hemispheres are covered by dura, it is convex and is located in the cerebellosa pit. It shows a wide crease in the middle line called vallecula or middle crack and hosts the cerebellum sickle. (vertical fold of the dura). The anterior face: it is closely related with the posterior face of the trunk of the encephalo (floor of IV ventricle). It shows a central depression that corresponds with the roof of the IV ventricle and it is bound up by the peduncles of both sides and by the higher and lower medular veils.

The study of the cerebellum can be divided in three stages: morphologic, functional and filogenetic.

**Morphologic division:** is a purely descriptive division, without functional basis, ontogenics or with clinic application.

In anatomic terms, two transverse fissures (postero lateral fissure and prima fissure) divide the cerebellum in three lobes: frontal (rostral to the prima fissure), posterior (between the prima and the posterolateral fissure) and flocculonodular (caudal) The demarcation of the three lobes could be better appreciated in mediosagitales cuts (fig 6 a).

Each lobe includes part of the vermis and part of the cerebellar hemispheres, moreover each lobe is divided in little convolutions or cerebellar folia, by less deeper fissures. (fig 6 a-b) Image 4. on page 14

1. **Anterior lobe:** is located in front of the prima fissure y and it is divided in: lingula, central lobulillo and culmen.

2. **Posterior lobe:** is located between the prima fissure and the lateral posterolateral fissure, and it is divided in: declive, folium, tuber, pyramid and uvula. (fig 6)
**3. Floculonodular lobe:** is located in front of the lateral back fissure and it is composed by the nodule and the flocculus. (fig 6).

**Functional division:** it is a secondary division due to the connections that establish and to its main function. Spinocerebellum, cerebrocerebellum and vestibulocerebellum (fig 7). The cerebellum is composed of an outer layer of gray matter (cerebella cortex), internal white matter and three pairs of deep nuclei: the fastigial, interposed (consisting of two nuclei, the spherical and emboliform) and dentate, fig 8. The cerebellum has afferents and efferents through the cerebellar peduncles. The cerebellum efferents originate from cell bodies in the deep nuclei, the exception is a relatively small part of the cerebellar cortex floculonodular lobe.

**1. Vestibulocerebellum:** corresponds to the floculonodular lobe, cerebellar cortex receives its information directly from the vestibular afferent fibers projecting to the lateral vestibular nuclei (fig 8). Its function is to control balance and eye movements.

**2. Spinocerebellum:** Corresponds to the vermis and paravermis region and is so named because they are the only regions that receive somatosensory afferents in the spinal cord. The vermis receives visual, auditory and vestibular and somatic sensory information from the head and proximal part of body. Is projected through the fastigial nucleus to the cortical regions and the brain that give leading to downstream systems that control the proximal muscles of the body and limbs, posture, locomotion, and the look. The middle part of the hemispheres receive somato sensory information of members, this region through the nucleus sends efferents brought through the rubrospinal and corticospinal tract, controlling the distal muscles of limbs fig 8.

**3. Cerebrocerebellum:** The sides of the hemispheres receive input exclusively from the cerebral cortex and this is called cerebrocerebellum. Its efferents are mediated by the dentate nucleus, which projects to the motor area, premotor and prefrontal cortex, and is intimately involved in planning, mental rehearsal of complex motor actions in the conscious evaluation of the errors of movement and cognitive functions Fig 8. Image 5 on page 15

**Phylogenetic Division:** this division allows functional identity and clinical approach. In humans, it have been clearly demonstrated two cerebellar syndromes: midline syndrome, or vermic (arquicerebelo involvement and/or paleocerebellum) and cerebellar hemisphere syndrome, or a combination of the two previous syndrome, syndrome pancerebeloso (fig 9).
1. **Arquicerebelo**: the oldest corresponding to the floculonodular lobe and is identified with the vestibulocerebellum, his involvement causes vermian syndrome: trunk ataxia and nystagmus.

2. **Paleocerebellum**: from subsequent splits the anterior lobe is formed and a small part of the posterior lobe, which is identified with spinocerebellum and its effect causes the vermian syndrome. The clinic is unsteady gait and increased stretch reflexes in lower limbs.

3. **Neocerebellum**: is the most modern part that corresponds to the posterior lobe except the pyramid, most receiving afferents from the cerebral cortex and is identified with the cerebrocerebellum, and its effect causes cerebellar hemisphere syndrome, the clinical is: ataxia, dysmetria, decomposition of movement, adiadococinesia, intention tremor, muscular hypotonia, dysarthria and nystagmus. *Image 6. on page 16*

- The cerebellum gets its blood flow from three long circumferential arteries which come from the vertebro basilar system (fig10) *Image 7. on page 17*

- Posterior inferior cerebellar artery (PICA): Arises from the vertebral artery, supplies most of the lower surface of the cerebellum, including cerebellar hemispheres, the inferior vermis and tonsils.

- Anterior inferior cerebellar artery (AICA): Arise from the basilar artery, due to its regular small size, irrigates a small area from the anterolateral part of the cerebellum lower surface when there is a big AICA, the ipsilateral PICA is almost everytime hipoplasica and in these cases the territory of the AICA includes all the lower frontal surface of the cerebellum.

- Superior cerebellar artery (SCA): Is more regular on its caliber and area of irrigation, it perfunds most of the upper surface of the hemispheres and the vermis and the deepest nucleus of the cerebellum.

The three circumferential arteries and their branches are connected by multiple cortical free anastomosis that help to limit infarct size.

- Venous drainage: The cerebellum is drained by three veins:

  - Superior vein: drains the upper surface of the cerebellum and it flows into the galeno vein; posterior vein: drains the posterior part of the lower surface and flows into the sinus recto and Petrosa or front vein: drains the front lower surface of the cerebellum and ends in the upper and lower petrosos sinus.

The cerebellum is affected by: congenital malformations, hereditary, metabolic, infectious, toxic, vascular, demyelinating, and neoplastic.
DISORDERS OF NEURAL TUBE CLOSURE

Arnold-Chiari: **Type I**: In this type, the inferior pole of the cerebellar hemispheres protrude through the foramen magnum. Most patients are asymptomatic, and the malformation is often found incidentally on MRI. Occasionally, patients present with headache, usually associated with occipital and neck pain and exacerbated by coughing and straining. Hydromyelia or syringomyelia may be associated with (Fig 11).

Type II. In this type, in addition to the protrusion of the cerebellum, the medulla oblongata protrudes through the foramen magnum, resulting in kinking of the cervical medullary junction. Part of the fourth ventricle is also displaced caudally. The foramina of Magendie and Luschka are occluded. The malformation is frequently associated with hydromyelia or syringomyelia, hydrocephalus, and meningomyelocele (fig 12) Image 8. on page 18

Type III: Is rare, this malformation has features of types I and II as well as herniation of the entire cerebellum into a high cervical meningocele. Hydrocephalus is a constant finding.

DISORDER OF FURROW FORMATION AND CELL MIGRATION

Cerebellar dysplasia: is a failure in cell migration and cortical organization, resulting in distortion of normal morphology and fissures and folia.

Diffuse cortical dysplasia. The process of crust formation cerebellar starts at embryonic week 10 and not acquires its adult appearance until after the 8th month of postnatal life. cerebellar cortical dysplasia may be associated with various brain malformations, which suggests an embryonic disorder. In MRI can see change in the direction of the fissures that acquire a vertical, irregular union between substance gray / white matter loss and abnormal arborization of white matter heterotopias in the cerebellar hemispheres, cortical thickening or cortical pseudocysts inclusions (fig13).

Venous drainage anomaly: Developmental venous anomalies (DVA), is believed to be the result of abnormal development of the venous system, by which the nervous tissue venous drainage occurs through abnormal veins enlarge and change direction to radial. DVAS malformations are frequent, its prevalence is about 0.7%-0.3%. A large number of patients with cerebellar DVAS (CDVAs) are diagnosed in incidental findings on CT and MRI images. Cerebellar venous malformations are associated to pontine venous malformations as both drainage systems derived therefrom primitive venous plexus (fig14). Image 9 on page 19

MALFORMATIONS AND CYSTS OF THE POSTERIOR FOSSA :
**Dandy Walker:** Theory Spectrum DW: Obstruction of Magendie and Luschka holes, bulging IV ventricle, cerebellar hemispheres difficulty fusion and hypoplasia / aplasia vermis, changing the configuration of the posterior fossa (fig 15-16) [Image 10 on page 20.

<table>
<thead>
<tr>
<th>VERMIS</th>
<th>MALFORMATION DW</th>
<th>VARIANT DW</th>
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<tr>
<td></td>
<td>ABSENT OR VERY</td>
<td>HYPOPLASIC</td>
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<tr>
<td>HYDROCEPHALUS</td>
<td>75%</td>
<td>25%</td>
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<td>SUPRATENTORIAL ANOMALIES</td>
<td>68%</td>
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<tr>
<td>TORCULAR INVESTMENT</td>
<td>YES</td>
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<tr>
<td>HYPOPLASIC HEMISF CEREBELLAR</td>
<td>YES</td>
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<tr>
<td>CEREBELLAR FALX</td>
<td>ABSENT</td>
<td>32%</td>
</tr>
<tr>
<td>IV VENTRICLE</td>
<td>OPEN CYST</td>
<td>CYST</td>
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**Joubert syndrome:** autosomal recessive, developmental delay, oculo-motor disorder, coloboma, abnormal breathing (tachypnea or neonatal apnea). It can associate corpus callosum dysgenesis, retinal dysplasia and polycystic kidney diseases, which worsens the prognosis. Aplasia / Hypoplasia vermis (vermis split into "spanking"). Absence of the primary fissure.

Superior cerebellar peduncles match, that may be increased or not on their thickness, horizontal orientation, which together with the elongation of the ponto-mesencephalic junction produce the image of "molar teeth”

Contact between hemispheres without nodule.

IV ventricle in "bat wings" or "open umbrella" (alteration stems development, higher and decussation of pyramids). Fastigial point away (ventricle homer)(fig17)

Arachnoid cysts: sporadically, and have higher incidence in patient with autosomal dominant polycystic kindney disease, in rare cases in which cyst produce symptoms, the physical and neurological signs and symptoms reflect their anatomical distribution and their effect on CSF flow (fig 18).[Image 11 on page 21

**VASCULAR SYNDROMES:**(fig19) [Image 12 on page 22
Superior Cerebellar Artery (SCA) Syndrome: This is the most frequently encountered vascular syndrome of the cerebellum. Clinical signs include ipsilateral dysmetria, limb ataxia, and Horner's syndrome, contralateral pain and thermal sensory loss, and contralateral trochlear nerve palsy. Horner's syndrome, the pain and thermal sensory deficits, and trochlear nerve palsy are due to involvement of the brain stem tegmentum. Dysarthria is common and is characteristic of rostral cerebellar lesions, whereas vertigo is not as common in SCA infarcts and is more characteristic of the posterior inferior cerebellar artery (PICA) syndrome. Isolated dysarthria (without other cerebellar signs) has been reported in occlusion of the medial branch of the superior cerebellar artery with an infarct limited to the paravermal area. Prognosis for recovery in SCA syndrome is usually good.

Anterior Inferior Cerebellar Artery (AICA) Syndrome: Occlusion of the anterior inferior cerebellar artery (AICA) is uncommon, and often is misdiagnosed as the lateral medullary syndrome (PICA syndrome). It is characterized by ipsilateral dysmetria, vestibular signs, Horner's syndrome, facial sensory impairment, contralateral pain and thermal sensory loss in the limbs, and at times, dysphagia. Other signs seen in this syndrome and unusual in the lateral medullary syndrome include ipsilateral severe facial motor palsy, deafness, lateral gaze palsy, and multimodal sensory impairment over the face due to involvement of facial, cochleovestibular, abducens, and trigeminal nerves and/or nuclei, respectively. AICA occlusion also can be manifest by purely cerebellar signs.

Posterior Inferior Cerebellar Artery (PICA) Syndrome

This syndrome is as frequent as the superior cerebellar artery (SCA) syndrome. Clinical features of the syndrome are described in the chapter on clinical correlates of the medulla oblongata.

Occlusion of the medial branch of the PICA may be clinically silent or may present with one of the following three patterns: isolated vertigo often misdiagnosed as inner ear disease. Vertigo, ipsilateral axial lateropulsion, and dysmetria or unsteadiness, or classic lateral medullary syndrome when the medulla is also involved in the lesion.

Cerebellar tumors: A frequent location of tumors, especially during childhood, the CT and MRI are the methods most important preoperative imaging to help determine the location, extent and the most likely diagnosis.

Cerebellar astrocytoma: The highest incidence of this tumor occurs during the first two decades of life, 85% are pilocytic type and have a good prognosis, the remaining 15% corresponds to anaplastic. In imaging findings( MRI) appears as a cystic cerebellar mass with enhancing mural nodule that enhances with contrast medium. May have little or no surrounding edema and the solid component is hypo- to isodense; often cause obstructive hydrocephalus.(fig 20)
Fibrillary astrocytoma grade I and II on CT and MRI are seen as a focal lobar lesion without edema or mass effect, homogeneous enhancement with contrast medium (fig 21) Image 13 on page 23

**Medulloblastoma**: corresponds to 25% of all intracranial tumors in childhood, the necrosis is an ominous sign. It is a malignant, invasive, highly cellular embryonal tumor. Commonly is spherical and pushes brain away on all sides.

**IMAGING FINDINGS**: solid mass in 4th ventricle or in the cerebellar hemisphere (in older children and adults). Hydrocephalus is common (95%); >90% enhance.

(Fig.22) MRI showed a lesion in the roof of the fourth ventricle (vermis) that tends to invade the leptomeninges spreading through the CSF CT corresponds to a tumor in the midline hyperdense, homogeneously enhancing and MRI is isointense on T2 sequences

**Ependymoma**: It is 10% of CNS neoplasms in childhood with a peak incidence between 10 and 15 years, usually originates in the floor of the fourth ventricle and extend through the Luschka and Magendie holes, and may invade the bulb oblongata (fig23) in imaging findings Irregular mass, 50% ca, cyst, hemoglobin, heterogeneous enhancement, originates in the IV ventricle and extends through the Luschka and Magendie holes. Image 14 on page 24.

**Cerebellum parenchymal metastases**: Parenchymal metastases: Parenchymal tumors that originate from, but are discontinuous with, other CNS primary or extracranial systemic neoplasms.

15% cerebellum in imaging findings in NECT iso-hypodense mass at gray-white interface, peritumoral edema and hemorrhage variable, in CECT intense, punctate, nodular or ring enhancement. (Fig 24) Image 15

**INFECTIONOUS**:

**Listeria rhomboencephalitis**: rare form of encephalitis due to Listeria that affects the brain stem, usually affects healthy adults. The typical clinical picture is a biphasic disease prodrome of fever, headache, nausea and vomiting that last for about 4 days, followed by a sudden onset of asymmetric cranial nerve deficits and cerebellar signs, CSF findings are only slightly abnormal and positive cultures by 40%, almost two thirds have bacteremia. MRI is superior to CT for detecting encephalitis (fig25). Image 16 on page 26

**Cerebellar atrophy**: Parenchymatous cortical cerebellar atrophy, a disease characterized by cerebellar development in middle-aged individuals with symptoms such
as cerebellar ataxia; is hereditary and has been associated with chronic alcoholism and neoplastic diseases (e.g. lung cancer), being the two most frequent etiologic factors. It narrows the molecular and granular layers and white matter is reduced. Imaging findings: On sagittal images flat ventral pons, diminution pons (normal signal), cerebellar vermis/hemispheres atrophy, on axial images: diminution anteroposterior diameter of pons, midbrain, width of superior and middle peduncles, enlargement of 4th ventricle and cerebro pontine angle. (26) Image 17 on page 27

**Multiple Sclerosis (EM):** EM affects the cerebellum in about three quarters of patients, producing tremors, incoordination and ataxia that last sign may be presented in a paroxysmal and may last seconds, with multiple replays throughout the day (fig 27) Image 18 on page 28

**Guillain Mollaret syndrome (Ocular-palatal myoclonus):** It consists of a pendular vertical nystagmus synchronous with palatal tremor and occurs after damage of the cerebellar dentate nucleus, contralateral red nucleus, inferior olivary nucleus or the tracts between these structures (fig 28). It occurs more frequently after damage of the dentate nucleus. The MRI shows the dentate lesion and a secondary hypertrophy of the contralateral inferior bulbar olivary nucleus. Image 19. on page 29

Symptomatic palatal tremor, in contrast to essential palatal tremor, persists during sleep and is often associated with oscillopsia and cerebellar signs, unilateral or bilateral. In some cases pharyngeal, facial and extraocular muscles, diaphragm, vocal cords and neck muscles can also take part in the rhythmic movements.

**Wallerian degeneration:** Is the irreversible axonal and myelin damage after the injury to the proximal portion of the axon or its cell body. The most frequent cause of Wallerian Degeneration in the central nervous system is ischemic stroke, paraneoplastic syndrome and inflammatory process (fig 29). Imaging findings: CT not sensitive for wallerian degeneration acute/subacute stages; detects atrophy of pyramidal tracts in chronic stage with dissmination of the size of corresponding aspect of brainstem. MRI findings are time-dependent.

**MENINGIOANGIOMATOSIS:** Rare, hamartomatous cortical/leptomeningeal cortical meningo-vascular proliferation with CA+, generally solitary but may be multiple, usually in cortex (frontal and temporal lobes), rarely in 3rd ventricle, thalami, brainstem and cerebellum, often extends into cortex via perivascular space, Etiology uncertain, principally in children and young adults. It has been associated with neurofibromatosis in 50% of patients. In imaging findings NECT solitary or multiple cortical mass and CA+, occasional hemorrhage and cyst, and no or little mass effect. CECT: homogeneous
enhancement. MRI: T1: isointense and hypointense cysts. T2: hyperintense. T1 C+: homogeneous enhancement. (fig 30) **Image 20** on page 30

**Images for this section:**

![Embriology of the Cerebellum](image)

**Fig. 1:** Image 1.
Fig. 2: Image 2.
Fig. 3: Image 3.
Fig. 4: Image 4.
Fig. 5: Image 5.
Fig. 6: Image 6.
Fig. 7: Image 7.
Fig. 8: Image 8.

Fig 11. Sagittal T1 and T2 show inferior displacement of the cerebellar tonsils 16mm below the foramen magnum.

Fig 12. Midsagittal T1 view of a newborn with Chiari II, associated with myelomeningocele.
Fig. 9: Image 9.
Fig. 15. In MRI sagittal T2, axial T1. Cyst in posterior fossa (direct connection between the IV ventricle and cisterna magna), absence of vermis, hypoplastic cerebellar hemispheres, increased posterior fossa, with elevated Tórulo (tentorium torcular-lamblid).

Fig. 16. RM in sequences T1 sagittal and axial T2, showing extensive contact between the fourth ventricle and mega cisterna magna with mild vermis hypoplasia in relation to dandy-walker variant. Prominence and deformity of the fourth ventricle that is associated with mild hypoplasia of most caudal aspect of vermix.

**Fig. 10: Image 10**
**PATHOLOGIC CONDITIONS**

**SINDROME DE JOUBERT**

Fig 17. In MRI axial T2 and sagittal T1.
Horizontality, rectification and thickened superior
cerebellar peduncles that alteras the morphology of
the bain stem-like in "molar tooth"

In coronal FLAIR, and US. IV
ventricle in "bat wings" or "open
umbrella" (alteration stems
development, higher and decussation
of pyramids) Fastigial point away
(ventricle homer).

**QUISTE ARACNOIDEO EN FOSA POSTERIOR**

Fig 18. In MR axial sagittal T1 T2 shows a
posterior fossa arachnoid
cyst of 74 mm with mass effect on the
cerebellum and fourth ventricle.

Fig. 11: Image 11.
PATHOLOGIC CONDITIONS

STROKE IN THE TERRITORY OF THE RIGHT SCA

MRI coronal FLAIR, axial T2, and diffusion that displays an image strongly suggestive of acute/subacute ischemic stroke partially affecting territory, depending on the right SCA.

CEREBELLAR INFARCTION BOTH SCA WITH HEMORRHAGIC COMPONENT

FLAIR coronal, sagittal and T1, and gradient where there are two very suggestive images of infarction in evolution, in areas depending on both superior cerebellar arteries, which, on the left side, shows hemorhagic transformation causing no significant mass effect on fourth ventricle.

ATYPICAL CEREBELLAR INFARCTION

In MRI, axial FLAIR, diffusion, coronal and axial T2 shows a triangular hyperintense area in both cerebellar hemispheres, that suggests cytotoxic edema although the vascular territory is atypical, borderline between PICA and superior cerebellar arteries.

Fig. 12: Image 12.
**Fig. 13:** Image 13.
**PATHOLOGIC CONDITIONS**

**MEDULLOBLASTOMA**

![Images of MRI scans showing medulloblastoma](image)

Fig. 14. Vermian rounded solid mass, originating from the roof of the fourth ventricle. Hyperdense on CT, with relatively homogeneous enhancement, a tendency to be isointense in T1 and T1 + c.

**EPENDYMOMA OF THE IV VENTRICLE**

![Images of MRI scans showing ependymoma](image)

Fig 23. Irregular mass, 50% ca, cyst, hemoglobin, heterogeneous enhancement, originates in the IV ventricle and extends through the Luschka and Magendie holes.

**Fig. 14:** Image 14.
Pathologic Conditions

Metastases from a Lung Cancer

Fig. 15: Image 15.

Fig 24. Patient with a history of lung cancer. Lesions in the cerebellum, with ring enhancement, central necrosis and edema, which determines the partial collapse of the fourth ventricle.
Fig 25. Hyperintensity on T2, FLAIR and diffusion, observing in gradients sequences, small foci suggestive of petechial hemorrhage and minimal edema.

**Fig. 16:** Image 16.
Fig. 17: Image 17.

Fig 26.
MRI coronal T2, Flair and sagittal T1, show cerebellar atrophy predominantly in the Cerebellar hemispheres with involvement of Vermis.

47 year old female patient with lithium intoxication and ataxia. Sagittal T1 and coronal Flair, showing predominantly vermian cerebellar atrophy probably related with the lithium intoxication.
Fig. 18: Image 18.

**MULTIPLE SCLEROSIS AFFECTING THE CEREBELLUM**

Fig 27. Patient with a history of multiple sclerosis on MRI follow-up presents two tenuous new images located in the right cerebellar hemisphere (arrow) and cerebellar peduncle (arrow).
PATHOLOGIC CONDITIONS

LESION IN GUILLAIN-MOLLARET`S TRIANGLE

Fig 28. In MRI gradient T2, axial T2 and coronal FLAIR Old hemorrhage involving the right dentate nucleus observing a very important right hemicerebellum atrophy, increased signal intensity of left medullary olivary nuclei (arrows).

Fig. 19: Image 19.
Fig. 20: Image 20
Conclusion

The Cerebellum can be affected by a wide variety of pathologic processes. Specific location, imaging features and some clinical aspects must be integrated to develop an accurate diagnose.

Personal Information

References


