MRI of the normal and abnormal brainstem.

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Learning objectives

+ Familiarity with normal brainstem anatomy as shown on MR images is essential for proper interpretation of pathology MR images of the brainstem.

+ Establish a differential diagnosis of the disorders most frequently on the brainstem depending on the patient's age, their location and their characteristics in MRI.

Background

Introduction

MR imaging is more sensitive and specific technique in the diagnosis of lesions of brainstem because it has an anatomic resolution allows us to accurately locate lesions.

The brainstem is best evaluated at MR imaging, which allows visualization of anatomic detail such as white matter-gray matter differentiation. More important, however, unlike CT, MR imaging is not limited by beam-hardening artifact in the evaluation of the posterior fossa.

It is therefore essential to know the anatomy of the brainstem and the characteristic morphological and normal signal in different sequences MRI.

MRI anatomy of brainstem:

The brainstem, or encephalic trunk, is subdivided into three sections: the medulla oblongata (elongated spinal cord), the pons (bridge), and the mesencephalon (midbrain). (Fig. 1.)

The brainstem is the lower extension of the brain where it connects to the spinal cord. Neurological functions located in the brainstem include those necessary for survival (breathing, digestion, heart rate, blood pressure) and for arousal (being awake and alert). Most of the cranial nerves come from the brainstem. The pons forms a broad arching bulge with prominent transverse fibers. Here, descending pathways from the brain are relayed to neurons extending to the cerebellum by the cerebellar peduncles.
The medulla oblongata functions primarily as a relay station for the crossing of motor tracts between the spinal cord and the brain. It also contains the respiratory, vasomotor and cardiac centers, as well as many mechanisms for controlling reflex activities such as coughing, gagging, swallowing and vomiting.

The longitudinal organization of the neural tube can still be recognized in the brain stem, where the somatomotor zone lies medially, next comes the visceromotor zone; the viscerosensory and somatosensory zones are localized laterally. (Fig.2.)

The anterior median fissure, which is interrupted by the pyramidal decussation, and the anterolateral sulcus on each side extend up to the pons. The anterior funiculi thicken below the pons to form the pyramids. Lateral to them on each side bulge the olives.

The posterior funiculi thicken on both sides to form the tubercle of the cuneate nucleus and the tubercle of the gracile nucleus.

The fourth ventricle forms on each side the lateral recess which opens to the subarachnoid space by the foramen of Luschka. (Fig.3.)

The floor of the rhomboid fossa shows bulges near the median sulcus because of the medial eminence, the facial colliculus, the trigono of the hypoglossal nerve, the trigon of the vagus nerve, and the vestibular area.

The rhomboid fossa is crossed by myelinated nerve fibers, the medullary striae, and the pigmented nerve cells of the locus ceruleus shine blueish through the floor of the rhomboid fossa.

The midbrain serves as the nerve pathway of the cerebral hemispheres and contains auditory and visual reflex centers.

The anterior surface of the mesencephalon, is formed by the cerebral peduncles (descending cerebral pathways). (Fig.4.) Between them lies the interpeduncular fossa; its floor is perforated by numerous vessels and is known as the posterior perforated substance.

At the posterior surface of the midbrain lies the tectal plate or quadrigeminal plate with the two superior colliculi, that are the station of the optic system, and the inferior colliculi, that are the station of the acoustic system.

Cranial nerves

According to classical anatomical nomenclature, there are 12 pairs of cranial nerves,
although the first two pairs are not really peripheral nerves, the brain stem contains the nuclei of cranial nerves (Fig. 5-6.). Their functions are resumed in the next table. (Table 1.1-1.2).

The olfactory nerve (I) consists of the olfactory fibers, the bundled processes of sensory cells in the olfactory epithelium which enter the olfactory bulb.

The optical nerve (II) is a cerebral pathway of the optical.

The oculomotor nerve (III) leaves the brain on the floor of the interpeduncular fossa.

The trochlear nerve (IV) emerges at the dorsal surface of the midbrain and extends around the cerebral peduncles to the basal surface and the abducens nerve (VI) emerges from the lower border of the pons.

The trigeminal nerve (V) emerges from the lateral part of the pons. Its sensory root extends to the trigeminal ganglion (semilunar ganglion, Gasser's ganglion).

The facial nerve (VII) and the vestibulocochlear nerve (VIII) leave the medulla oblongata at the cerebellopontine angle. The taste fibers of the facial nerve emerge as an independent nerve, called the intermediate nerve.

The glossopharyngeal nerve (IX) and the vagus nerve (X) emerge dorsal to the olive. The cervical roots of the accessory nerve (XI) unite to form the spinal root.

The hypoglossal nerve (XII) is a somatomotor nerve.

Cranial Nerve Nuclei

As in the spinal cord, where the anterior horn represents the area of origin of motor fibers and the posterior horn the area of termination of sensory fibers, the medulla oblongata contains the nuclei of origin (with the cell bodies of efferent fibers) and the nuclei of termination (for the axon terminals of afferent fibers) of different fibers (Fig.6.)

The somatomotor nuclei lie close to the midline and are:

The nucleus of the hypoglossal nerve (tongue muscles)

The nucleus of the abducens nerve

The nucleus of the trochlear nerve
The nucleus of the oculomotor nerve (*eye muscles*)

The visceromotor nuclei (*parasympathetic*) follow laterally, includes:

The dorsal nucleus of vagus nerve (*viscera*)

The inferior salivatory nucleus (preganglionic fibers for the *parotid gland*)

The superior salivatory nucleus (preganglionic fibers for the *submandibular and sublingual glands*)

The Edinger-Westphal nucleus (accessory nucleus of oculomotor nerve) They are the preganglionic fibers for the *sphincter muscle of pupil* and the *ciliary muscle*.

The series of *motor nuclei* of the branchial arch nerves lies deep:

The spinal nucleus of the accessory nerve.

The ambiguous nucleus, which is the motor nucleus of the vagus nerve, the glossopharyngeal nerve, and the nucleus of the facial nerve.

The most cranial nucleus of the branchial arch nerves is the motor nucleus of the trigeminal nerve

The sensory nuclei are located medially:

The sensory fibers of the vagus nerve and the glossopharyngeal nerve, (solitary nucleus), as well as all taste fibers.

The trigeminal nerve, which has the largest expanse of all cranial nerve: has three nucleus: pontine, mesencephalic and spinal.

Finally, most laterally lies the area of the vestibular nucleus and the cochlear nucleus.

Images for this section:
This image illustrates the brainstem, showing the midbrain and the pons. The midbrain is a section of the brainstem located near the posterior of the thalamus. The pons is a part of the brainstem that connects the cerebrum with the medulla oblongata. Fig. 1
Longitudinal organization of the medulla oblongata
-somatomotor zone.
-visceromotor zone
-viscerosensory / somatosensory.

Olive
Pyramid
Ventral roots of 1st spinal nerve (C1)
Decussation of pyramids

Vestibulocochlear nerve (VIII)
Glossopharyngeal nerve (IX)
Vagus nerve (X)
Hypoglossal nerve (XII)
Accessory nerve (XI)

Fig. 2
**Fig. 4**

**Median sagittal section**

- Pulvinars of thalami
- Pineal body
- Superior colliculi
- Inferior colliculi
- Cerebral peduncle
- Posterior commissure
- Quadrigeminal plate

**Posterolateral view**

- Trochlear nerve (IV)
- Somatomotor nucleus: II, VI, IV, III.
- Visceromotor (parasympathetic) nucleus
- Motor nuclei: XI, XII, VII, V.
- Sensory nuclei: solitary nucleus, V, VIII
- Vestibular and cochlear nucleus.
- Red nucleus
- Olive.

Fig. 6
Fig. 7
Table 1.1. Cranial nerves and principal clinical functions.

- I Olfactory Sensory Smell
- II Optic Sensory Vision
- III Oculomotor Motor Movements of eyeball: most orbital muscles. Parasympathetic: ciliary muscle, accommodation of lens, etc.; iris muscle, pupilloconstriction
- IV Trochlear Motor Movements of eyeball: superior oblique muscle.
- V Trigeminal
  - Va: Ophthalmic Sensory Sensation from eyeball, anterior scalp, upper face
  - Vb: Maxillary Sensory Sensation from nasal cavity and sinuses, palate, mid face, maxillary teeth
  - Vc: Mandibular Mixed Muscles of mastication, tensor tympani sensation from chin, temple, oral cavity, tongue, temporomandibular joint (TMJ), mandibular teeth, ear, proprioception from muscles of mastication
- VI Abducens Motor Movements of eyeball: lateral rectus muscle.

Fig. 8
Table 1.2. Cranial nerves and principal clinical functions.

- VII Facial Mixed Muscles of facial expression, stapedius (middle ear) (parasympathetic: lacrimal, nasal, palatine, submandibular, sublingual glands) (taste: anterior tongue)

- VIII Vestibulocochlear Sensory Hearing, balance

- IX Glossopharyngeal Mixed Sensation from oropharynx, posterior tongue, carotid body and sinus (taste: posterior tongue) (muscle: stylopharyngeus) (parasympathetic: parotid gland)

- X Vagus Mixed Muscles of larynx, pharynx (phonation, swallowing) Sensation from larynx, hypopharynx, heart, lungs, abdominal viscera (taste: epiglottic region, hypopharynx) (parasympathetic: cardiac muscle; muscles and glands of foregut and midgut: intestinal activity)

- XI Accessory Motor Muscles: sternocleidomastoid, trapezius

- XII Hypoglossal Motor Tongue muscles and movements

Fig. 9
Imaging findings OR Procedure details

After recalling the normal anatomy of brainstem, we focus on three points to establish the differential diagnosis and can thus provide more accurate final diagnosis.

**Differential diagnosis based on location:**

Based on the location of brainstem lesions can be divided into:

1. The intraaxial lesions.
2. The extraaxial lesions.

1. The intraaxial lesions.

Intra-axial lesions can be located in:

- Medulla.
- Pons.
- Midbrain.

Lesions that we found in both medulla, pons and midbrain can be divided into metabolic-congenital disorder, traumatic injury, infectious- inflammatory, degenerative, infiltrating diseases, vascular and tumor.

Within the pathology of metabolic-congenital origin that affects to medulla, highlighted in order of frequency:

1. Syringobulbia
2. Vitamin B12 and copper deficiency.
3. Nonketotic hyperglycinemia.
4. Leukodystrophies, X-linked adrenoleukodystrophy, Alexander’s Disease or Leigh Syndrome
Within the pathology of metabolic-congenital origin that affects to pons, highlighted in order of frequency:

1. Central pontine myelinolysis.
2. Vitamin B12 and copper deficiency.
5. Leukodystrophies, X-linked adrenoleukodystrophy, Alexander’s Disease or Leigh Syndrome

Within the pathology of metabolic-congenital origin that affects to midbrain, highlighted in order of frequency:

1. Vitamin B12 and copper deficiency.
2. Maple syrup urine disease.
3. Nonketotic hyperglycinemia.

Within the pathology of traumatic injury that affects to medulla, highlighted in order of frequency:

Contusions or diffuse axonal injury.

Within the pathology of traumatic injury that affects to pons, highlighted in order of frequency:

Contusions or diffuse axonal injury.

Within the pathology of traumatic injury that affects to midbrain, highlighted in order of frequency:

Contusions or diffuse axonal injury.
Within the pathology of infectious-inflammatory origin that affects the medulla, highlighted in order of frequency:

1. Multiple sclerosis or acute disseminated encephalomyelitis and Devic's neuromyelitis optica
2. Encephalitis, pyogenic abscesses, neurocysticercosis, or tuberculomas.
4. Behçet disease and primary angiitis of the central nervous system.

Within the pathology of infectious-inflammatory origin that affects the pons, highlighted in order of frequency:

1. Multiple sclerosis, acute disseminated encephalomyelitis and Devic's neuromyelitis optica
2. Encephalitis, pyogenic abscesses, neurocysticercosis, or tuberculomas.
4. Behçet disease and primary angiitis of the central nervous system

Within the pathology of infectious-inflammatory origin that affects the midbrain, highlighted in order of frequency:

1. Multiple sclerosis, acute disseminated encephalomyelitis and Devic
2. Encephalitis, pyogenic abscesses, neurocysticercosis, or tuberculomas.
4. Behçet disease and primary angiitis of the central nervous system

Within the pathology of degenerative origin that affects the medulla, highlighted in order of frequency:

1. Encephalomalacia.
2. Wallerian degeneration of the corticospinal tracts.

4. Olive degeneration.

Within the pathology of degenerative origin that affects to pons, highlighted in order of frequency:

1. Encephalomalacia.

2. Wallerian degeneration of the corticospinal tracts.


Within the pathology of degenerative origin that affects to midbrain, highlighted in order of frequency:

1. Encephalomalacia.

2. Wallerian degeneration of the corticospinal tracts.

Within the pathology of infiltrating disease that affects to medulla, highlighted in order of frequency:

Sarcoidosis or Langerhans cell histiocytosis

Within the pathology of infiltrating disease that affects to pons, highlighted in order of frequency:

Sarcoidosis or Langerhans cell histiocytosis

Within the pathology of infiltrating disease that affects to midbrain, highlighted in order of frequency:

Sarcoidosis or Langerhans cell histiocytosis

Within the pathology of vascular origin that affects to medulla,
highlighted in order of frequency:
Infarction, resulting in edema, hemorrhage, or vascular malformations.

Within the pathology of **vascular origin** that affects to **pons**, highlighted in order of frequency:
Infarction, resulting in edema, hemorrhage, or vascular malformations.

Within the pathology of **vascular origin** that affects to **midbrain**, highlighted in order of frequency:
Infarction, resulting in edema, hemorrhage, or vascular malformations.

Within the pathology of **tumoral origin** that affects to **medulla**, highlighted in order of frequency:
Gliomas or metastatic lesions.

Within the pathology of **tumoral origin** that affects to **pons**, highlighted in order of frequency:
Gliomas or metastatic lesions

Within the pathology of **tumoral origin** that affects to **midbrain**, highlighted in order of frequency:
Gliomas or metastatic lesions

2. The extraaxial lesions.

The extraaxial lesions can be divided into infectious, vascular and tumor.

Within the pathology of infectious **origin**, highlighted in order of frequency:
1. Inflammatory infiltration of the basal meninge.

Within the pathology of vascular origin, highlighted in order of frequency:

1. Hemorrhage can be seen in the cisternal spaces around the brainstem.

2. Aneurysms.

Within the pathology of tumoral origin, highlighted in order of frequency:

1. Meningiomas and schwannomas, particularly in the cerebellopontine angle (epidermoid cysts; arachnoid cysts).

2. Neoplastic infiltration of the basal meninges.

**Differential diagnosis according to age:**

Depending on the age the pathology of the brainstem can be divided into two groups:

1. The most common pathology in children:

   Congenital or tumoral origin and infectious-inflammatory.

**POSTERIOR FOSSA MASS - CHILD**

Cerebellum/IVth Ventricle

- Medulloblastoma - midline, vermian or roof - usually hyperdense on plain CT - often enhance homogeneously - Astrocytoma
- usually PILOCYTIC ASTROCYTOMA - 2/3 are cystic with mural nodule - cyst fluid denser than CSF due to protein
- Ependymoma - INTRA-ventricular - "cast" of lumen - 50% are calcified

Brainstem - Brainstem glioma - expands brainstem (infiltration w/o destruction) - hydrocephalus (may be late)
Extraaxial fluid collection
- Large cisterna magna ("Mega Cisterna Magna")
- Epidermoid inclusion cyst
- Arachnoid cyst (may bevel inner table of skull) - Dandy Walker cyst of 4th ventricle (look for vermian abnormalities)
- Vermian agenesis.
- Chronic subdural hematoma.

2. The most common pathology in adults:

Vascular, inflammatory and tumoral.

**POSTERIOR FOSSA MASS - ADULT**

**Extraaxial:**
- Vestibular Schwannoma (CPA)
- Meningioma
- Ependymoma

**Intraaxial:**
Metastasis - most common intraaxial neoplastic post fossa mass in adult
Hemangioblastoma - cystic or solid - angio shows hypervascularity & stain
Astrocytoma - usually not vascular on angio
Medulloblastoma - often more lateral in adults Lymphoma Abscess Infarct.

**Differential diagnosis based on the characteristics of lesions in different sequences MRI:**

1. The intraaxial lesions.

Within the pathology of metabolic-congenital origin that affects to brainstem:

**Central pontine myelinolysis (CPM)** occurs in the setting of rapidly corrected hyponatremia, especially in chronically debilitated patients. Conventional CT and MR imaging findings lag the clinical manifestations of CPM. We present a case in which restricted diffusion was identified within the central pons by using MR diffusion-weighted imaging within 24 hours of onset of patient tetraplegia and before findings were conspicuous with conventional MR imaging sequences (T1, T2, and fluid-attenuated inversion recovery).

Central pontine myelinolysis (CPM) is an osmolar disturbance resulting in demyelination that is initially difficult to detect with convention CT and MR imaging. The literature includes several cases in which the temporal evolution of CPM is followed with serial imaging. There have been a few reports describing findings of diffusion-weighted imaging (DWI) in CPM and extrapontine myelinolysis (EPM); however, we are unaware of published cases in which DWI demonstrated changes of CPM before development of conventional MR imaging signal intensity changes. This case also helps elucidate the
temporal relationship of DWI with clinical symptoms and serum sodium levels, which are well documented for this patient. (Fig 1, 2 and 3).

**B12 deficiency and copper**, plus the subsequent degeneration of the spinal cord, can be associated with bilateral degeneration of the cortico-spinal tracts.

**Maple syrup urine disease.** The most severe, classic neonatal form of this disease is characterized by early postnatal onset and rapidly progressive neurologic deterioration. The MR imaging appearance is highly characteristic, with restricted water diffusion in the cerebellar white matter, dorsal brainstem, cerebral peduncles, posterior limb of the internal capsule, and posterior centrum semiovale. Recognition of this imaging pattern is important to allow early treatment.

**Nonketotic hyperglycinemia** is an autosomal recessive inborn error of metabolism caused by a defect in the glycine cleavage system. Resultant elevated levels of glycine in the brain and cerebrospinal fluid cause neurologic impairment, which usually manifests soon after delivery. MR imaging findings include ventriculomegaly; absent corpus callosum; restricted water diffusion in the pyramidal tracts, middle cerebellar peduncles, and dentate nuclei; and an abnormal glycine peak at 3.55 ppm in MR spectra.

Within the pathology of infectious-inflammatory origin that affects to brainstem:

**Multiple sclerosis** is a disease Idiopathic inflammatory-demyelinating CNS that predominantly affects women between 20 and 40 years. It is characterized by multiple demyelinating plaques in the subcortical white matter and periventricular juxtaposition, with special predilection for the corpus callosum and infratentorial brain parenchyma. (Fig. 4)

Patients presenting with clinically isolated demyelinating syndromes do not necessarily have multiple sclerosis, although they may be at risk of developing this disease. It could be important for treatment and prognosis to determine the patient’s likelihood of developing clinically definite multiple sclerosis after the first episode. The main paraclinical indicators associated with an increased risk of progression to multiple sclerosis are the presence of CSF oligoclonal bands and multifocal white matter abnormalities on MR images. These MR imaging abnormalities are indistinguishable from multiple sclerosis in 40% to 80% of patients presenting with isolated syndromes, such as unilateral optic neuritis, internuclear ophthalmoplegia, or partial myelopathy. Even when such lesions are present, it is still not possible to diagnose definite multiple sclerosis, because the criterion of dissemination in time has not been fulfilled. It has, however, been shown that the presence of white matter abnormalities on MR images at the time of
presentation with a clinically isolated syndrome suggestive of central nervous system demyelination is predictive of the long-term risk for subsequent development of multiple sclerosis, type of disease, and extent of disability.

Several criteria have been proposed to classify MR imaging findings as being suggestive or not suggestive of multiple sclerosis. In 1988, Paty et al. established that images showing either four or more lesions or three lesions, one of which is in a periventricular location, are indicative of multiple sclerosis. These criteria have been evaluated prospectively in cases of patients who presented with isolated syndromes suggestive of multiple sclerosis, and they have shown high sensitivity but relatively low specificity. The most widely used diagnostic criteria for this purpose have been established by Fazekas et al., who defined abnormal images as those that show three or more lesions with two of the following characteristics: infratentorial location, periventricular location, or size larger than 6 mm. This model showed high sensitivity and specificity when evaluated retrospectively in cases of established multiple sclerosis, but it performed less well when prospectively applied to patients presenting with isolated syndromes suggestive of multiple sclerosis. Recently, Barkhof et al. developed a four-parameter dichotomized MR imaging model based on logistic regression analysis that requires the presence of at least one enhancing lesion (or nine lesions visible on T2-weighted image), one juxtacortical lesion, one infratentorial lesion, and three periventricular lesions. This model predicts conversion to clinically definite multiple sclerosis better than do the criteria proposed by Paty et al. or Fazekas et al. The purpose of this study was to compare the effectiveness of the MR imaging criteria established by Barkhof et al., Paty et al., and Fazekas et al. for predicting conversion of isolated demyelinating syndromes to clinically definite multiple sclerosis in a cohort of patients followed up prospectively for a minimum of 18 months. (Fig. 5).

Acute disseminated encephalomyelitis (ADEM) is a severe, acute, demyelinating disease of the CNS. It is usually triggered by an inflammatory response to viral infections and vaccinations. A hemorrhagic, hyperacute variant of ADEM (AHEM/AHLE or Hurst disease) has also been described.

The course of ADEM is usually monophasic and affects children more commonly than adults. The main symptoms are decreased level of consciousness varying from lethargy to coma, convulsions, and multifocal neurologic symptoms such as hemi-, para-, and tetraparesis, cranial nerve palsies, and movement disorders.

In MR imaging, ADEM causes multiple sclerosis (MS)-like, but more asymmetrical, white matter lesions. In several reports, these lesions have been documented to show up in the first MR imaging scans performed shortly after the first symptoms. Disappearance of these lesions has been suggested to be associated with clinical recovery. To better characterize when the ADEM-associated lesions actually appear on MR scans and how the evolution of these lesions is associated with the patients’ clinical condition, we performed serial MR imaging on four previously healthy adults with ADEM. (Fig. 6).
Bickerstaff's brainstem encephalitis: Bickerstaff reported eight patients who, in addition to acute ophthalmoplegia and ataxia, showed drowsiness, extensor plantar responses or hemisensory loss. This condition has been named Bickerstaff's brainstem encephalitis (BBE). One patient had gross flaccid weakness in the four limbs. Presumably because of the rarity of this disorder, there has been no reported study on a large number of patients with BBE. To clarify its clinical features, we reviewed detailed clinical profiles and laboratory findings for 62 cases of BBE diagnosed by the strict criteria of progressive, relatively symmetrical external ophthalmoplegia and ataxia by 4 weeks, and disturbance of consciousness or hyperreflexia. Ninety-two per cent of the patients involved had an antecedent illness. Besides ophthalmoplegia and ataxia, disturbance of consciousness was frequent (74%), and facial diplegia (45%), Babinski's sign (40%) and pupillary abnormality and bulbar palsy (34%) were present. Almost all the patients had a monophasic remitting course and generally a good outcome. Serum anti-GQ1b IgG antibody was positive in 66%, and MRI showed brain abnormality in 30% of the patients. Another striking feature was the association with flaccid symmetrical tetraparesis, seen in 60% of the patients. An autopsy study of a BBE patient clearly showed the presence of definite inflammatory changes in the brainstem: there was perivascular lymphocytic infiltration with oedema and glial nodules. Electrodiagnostic study results suggested peripheal motor axonal degeneration. Limb weakness in the BBE cases studied was considered the result of overlap with the axonal subtype of Guillain-Barré syndrome. These findings confirm that BBE constitutes a clinical entity and provide additional clinical and laboratory features of BBE. A considerable number of BBE patients have associated axonal Guillain-Barré syndrome, indicative that the two disorders are closely related and form a continuous spectrum.

Abnormal lesions (high-intensity areas on T2-weighted images of the brainstem, thalamus, cerebellum and cerebrum) on MRI findings were present in about one-third of the BBE patients. Normal MRIs have been reported for some BBE patients, whereas in others high-signal lesions, documented on T2-weighted images of the upper mesencephalon, cerebellum, thalamus or brainstem, may move and regress with the clinical course of the illness. (Fig. 7).

Neurobehçet disease: it most often involves the brainstem, especially around the cerebral peduncles and the pons.

The thalamus and basal ganglia are the next most common sites of involvement, and similar foci can be noted in the cerebral hemispheres.

Well-known magnetic resonance (MR) findings in neurobehçet disease are small foci of high signal intensity on T2-weighted images; these foci are iso- or hypointense relative to brain parenchyma on T1-weighted images. Lesions may be circular, linear, crescent-shaped, or irregular. (Fig 8, 9 and 10). The development and disappearance
of lesions at CT and MR imaging correlate with the course of clinical neurologic deficits. Differential diagnoses include multiple sclerosis, brainstem infarction, and dilated perivenular spaces.

Within the pathology of degenerative origin that affects to brainstem:

**Wallerian degeneration** (WD) is the process of progressive demyelination and disintegration of the distal axonal segment following the transection of the axon or damage to the neuron. Although this term originally referred to lesions of peripheral nerves, today it can also refer to the CNS when the degeneration affects a fiber bundle or tract through the same mechanism.

The most commonly recognizable cause of WD is cerebral infarction. WD can also result from a variety of conditions including hemorrhage, trauma, necrosis, and focal demyelination.

MR imaging may depict WD when it involves a sufficiently large bundle of fibers. The most common observations regard the corticospinal tract. If attention is paid to the course of fibers that may be affected by a certain lesion, however, degeneration of fibers crossing the corpus callosum, fibers of the optic radiations, fornices, and cerebellar peduncles may be recognized. Knowledge of the course of fibers is essential in recognizing WD. An excellent review of the lesions that may affect the middle cerebellar peduncles (MCPs) was recently published. This review by Okamoto et al quotes an article that, to our knowledge, is the only current report of WD of pontocerebellar fibers caused by an acute vascular lesion in the pons. Very few other reports illustrate or mention MCP abnormalities in WD. The course of the pontocerebellar fibers in humans is still not widely known. To understand how pontine infarcts may affect these fibers, Schmahmann et al recently carried out a very interesting study in monkeys.

WD is a process that develops through different stages. In stage 1, no signal intensity abnormalities are usually recognizable. From 20 days to 2-4 months after stroke (stage 2), the tissue becomes more hydrophobic because of the myelin-protein breakdown: the high lipid-protein ratio results in hypointense signal intensity in proton density-and T2-weighted images. Hypointense signal changes are usually recognizable earlier in proton density-versus T2-weighted images because of the different conspicuity of the low signal intensity of the degenerated fibers versus the background. Stage 3 results from subsequent myelin lipid breakdown, gliosis, and changes in water content and structure; the tissue becomes hydrophilic and there is hyperintense signal on T2-weighted and FLAIR images and hypointense signal on T1-weighted images. After several years, the end stage (stage 4) is characterized by volume loss from atrophy, which, for instance, may be recognized in the brain stem as unilateral shrinkage following WD of the corticospinal tract. These temporal relationships between the MR imaging findings and the stages
of WD are usually recognized; however, there is a great variability, and signal intensity abnormalities may be visible many years after stroke. *(Fig. 11 and 12).*

**Multiple system atrophy** is a progressive neurodegenerative disorder of adult-onset and unknown etiology, combining cerebellar disorders, pyramidal, extrapyramidal and autonomic. There are two variants-striatal degeneration nigricans (without bulbar involvement) and atrophy olivo-ponto-cerebellar. In the latter entity is observed atrophy of the pons, the inferior olives and cerebellum, which are accompanied by a high signal on T2-extrusion of the superior cerebellar peduncles and olives (for fiber degeneration pons transverse, fibers ponto-cerebellar and inferior olivary nuclei).

**Olive degeneration** is a secondary degeneration of inferior olivary nucleus, usually caused by a primary lesion in the gastrointestinal dento-rubro-olive (Guillain-Mollaret triangle). There are three distinct patterns, whichever is the location of the primary lesion, if the primary lesion is confined to the trunk (central tegmental tract) will ipsilateral olive degeneration, if the primary lesion is located in the cerebellum (dentate nucleus or superior cerebellar peduncle) injury is contralateral involvement if stem and cerebellum, olive degeneration is bilateral. On MRI, what we will see a signal on T2 sequences of the olive, which is accompanied by an olive-size (if the injury is acute, subacute), hypertrophic (if the primary lesion occurred between 6 months and 3 years ago) or atrophic (if more than 3 years was the primary lesion tract dento-rubro-olive).

Within the pathology of **infiltrating disease** that affects to brainstem:

**Neurosarcoidosis** has a variable expression, which tends to mimic other neurologic diseases. The diagnosis is based on the documentation of systemic **sarcoidosis** in the absence of other neurologic disease. The histopathologic hallmarks include epithelioid granulomas without caseation or staining for infectious agents. These granulomas often incorporate multinucleated giant cells and lymphocytes. Compared with **sarcoidosis** granulomas elsewhere in the body, these tend to be smaller, have fewer multinucleated giant cells, and be associated with blood vessels. The frequent simultaneous expression of new and old granulomas suggests that the disease process waxes and wanes.

Although no part of the CNS is immune to **sarcoidosis**, frequently affected brain parenchymal locations include the hypothalamus, brain stem, cerebral hemispheres, and cerebellar hemispheres. Granulomatous infiltration into the subependymal layers of the ventricular system is thought to be responsible for hydrocephalus associated with **neurosarcoidosis**. Involvement of the pituitary is less common.
**Langerhans cell histiocytosis (LCH)** is a rare systemic granulomatous disease of the dendritic system with a variable clinical course that may be encountered at any age. The annual incidence of LCH in children younger than 10 years was reported to be 0.2-2 patients per 100,000 children. The incidence in adults remains to be determined. Almost all organs of the body can be affected by the disease. The typical LCH lesion is composed of a granulomatous infiltrate of a variable number of monoclonal Langerhans cells, T cells, and eosinophils.

Central nervous system (CNS) involvement in LCH most frequently manifests itself in the hypothalamic pituitary region with the key symptom of diabetes insipidus. Less frequently, granulomatous lesions in the meninges, the choroid plexus, the pineal gland, or the cerebral parenchyma are encountered. In addition to granulomatous lesions, LCH-associated MR imaging signal intensity abnormalities of variable intensity in the cerebellum, the basal ganglia, the pons, and the supratentorial white matter have been described in a number of reports. The signal intensity abnormalities in the cerebellum were composed of symmetric hyperintense signal intensity alterations on T2-weighted images and hypointense or hyperintense signals on T1-weighted images involving the gray matter only or extending to the surrounding white matter, eventually resulting in CSF-intense "holes" on T1-weighted images. Pontine lesions have been reported as T2 hyperintense signal intensity abnormalities in the pontine tegmentum or symmetrical T2 hyperintensities in the pontine pyramidal tracts. In the basal ganglia, the abnormalities consisted of hyperintensities on T1-weighted images and variable signal intensities on T2-weighted images. These lesions did not show contrast enhancement or mass effect and inconsistently displayed calcifications. (Fig. 13).

Within the pathology of vascular origin that affects to brainstem:

**Vascular disease** is responsible for most of the pathology of the brainstem. The vascularization of the **medulla** depends on branches of the anterior spinal artery and vertebral artery branches. According to this topography, we divide the lesions into three provinces: paramedian territory (perforating branches of the vertebro-basilar territory or branches of the anterior spinal artery), lateral territory (depending on short circumflex branches of the vertebral artery) and posterior territory lateral (dependent on the long circumflex branches of the vertebral artery, the posterior inferior cerebellar artery). (Fig. 14, 15 and 16).

The vascularization of **pons** depends on anteromedial and anterolateral groups arising from the basilar artery, entering the foramen cecum, the basilar sulcus, and the interpeduncular fossa. Lateral group arising from the anterior inferior cerebellar artery (entering the parenchyma in the pontomedullary sulcus) and arising from the lateral pontine arteries (entering the brachium pontis). Posterior group arising from the superior cerebellar artery (medial and lateral branches). Probably due to the large development of the ventral part of the pons in humans, the arterial supply of the pontine tegmentum
can be divided into three levels. At the lowest tegmental level the artery entering the foramen cecum and the pontomedullary sulcus (superior rami of the lateral medullary fossa) supplies the tegmentum along its ascending path. At the middle tegmental level the arteries reach the tegmentum by a direct and straight path. At the upper tegmental level, the arteries entering the interpeduncular fossa (inferior rami) reach the tegmentum through a descending path. (Fig. 17 and 18).

Five arterial trunks supply the arterial midbrain groups, from below to above, the superior cerebellar artery (mainly the medial branch), the collicular artery, the posteromedial choroidal artery, the posterior cerebral artery, and the anterior choroidal artery. Due to their anatomic relationship with the anterior, lateral, and posterior aspects of the midbrain, these arterial trunks have specific involvement in the anteromedial, anterolateral, lateral, and posterior groups of midbrain arteries. The posterior cerebral artery provides the only supply to the anteromedial group (middle rami of the interpeduncular fossa). The collicular and posteromedial choroidal arteries are the main source of the anterolateral and lateral groups; the posterior group is supplied by the superior cerebellar, collicular, and posteromedial choroidal arteries. Note that the anterior choroidal and posterior cerebral arteries may participate in the upper anterolateral group. (Fig. 19).

**Posterior reversible encephalopathy** syndrome (PRES) is a neurotoxic state coupled with a unique CT or MR imaging appearance. Recognized in the setting of a number of complex conditions (preeclampsia/eclampsia, allogeneic bone marrow transplantation, organ transplantation, autoimmune disease and high dose chemotherapy) the imaging, clinical and laboratory features of this toxic state are becoming better elucidated. This review summarizes the basic and advanced imaging features of PRES, along with pertinent features of the clinical and laboratory presentation and available histopathology. Many common imaging/clinical/laboratory observations are present among these patients, despite the perception of widely different associated clinical conditions.

The brain typically demonstrates focal regions of symmetric hemispheric edema. The parietal and occipital lobes are most commonly affected, followed by the frontal lobes, the inferior temporal-occipital junction, and the cerebellum. Lesion confluence may develop as the extent of edema increases. MR diffusion-weighted imaging (DWI) was instrumental in establishing and consistently demonstrating that the areas of abnormality represent vasogenic edema. The edema usually completely reverses. (Fig. 20).

Within the pathology of tumoral origin that affects to brainstem:

Tumor pathology may be primary (glioma, lymphoma) or secondary (metastases or meningeal carcinomatosis).
The brainstem glioma constitutes approximately 15% of tumors in children and adolescents, being a rare entity in adults. There are two main types: diffuse infiltrative glioma and focal glioma, and while the former are variable grade infiltrating astrocytomas, the latter are usually pilocytic. MRI is the most sensitive technique for detecting, displaying a high signal on T2 sequences diffusely affecting the brainstem, often in the absence of significant mass effect on the tanks peritronculares. It is usually accompanied by anatomical distortion of the morphology, and enhancement after contrast administration is variable (approximately 50%). (Fig. 21 and 22).

2. The extraaxial lesions.

Within the pathology of tumoral origin:

**Cerebellopontine angle masses** are a common pathology, with approximately 10% of intracranial tumors in adults are located in this anatomical area. Because, overall, the three most common injuries at this location (neurinoma, meningioma and squamous cell) are between 95 and 98% of the total knowledge of the typical radiological findings of each of them and their distinctive features allow the radiologist to make a proper diagnosis in the vast majority of cases.

Tumors that originate as such in the cerebellopontine angle are the neurinoma, meningioma, epidermoid cyst, an aneurysm of the arteries of the posterior fossa, lipomas, arachnoid cyst, dermoid cyst, the cyst and cancer neuroentérico melanocytic.

The most common tumor in the cerebellopontine angle **neurinoma** is that in 95% of cases depends on the eighth cranial nerve, but can also be caused by the V or VII cranial nerve, and, although less frequent, in any the stop scranuales IX-XII. The existence of bilateral acoustic neuromas most frequently associated with neurofibromatosis type 2. The neuromas are slow-growing benign tumors. They have a round or oval morphology, are homogeneous and well defined margins, as it is an encapsulated tumor. If you are small, are entirely within the rock, but as they grow, emerge from the internal auditory canal, channel widening in expanding posteriorly. When large may have a heterogeneous morphology secondary to cystic degeneration or fatty degeneration, and bleeding inside. In the TC are isodense or slightly hypodense compared to brain parenchyma and enhanced after contrast administration. RM appears isointense or slightly hypointense on T1, hyperintense on T2 and shows an intense enhancement after gadolinium administration. (Fig. 23).

The **epidermoid cyst** is the third most common cancer. Derived from the ectoderm and looks unilocular cyst, or rarely multilocular, containing keratin and cholesterol are shed from the lining of the cyst. It grows slowly around the vascular bundles and nervous, it’s
rare that move. CT appears as a hypodense mass, almost isodense to cerebrospinal fluid (CSF) and has jagged edges and lobed, no reaction occurs in the adjacent bone and rarely calcify the cyst wall or enhanced after contrast administration. RM is presented as a heterogeneous mass with a layer of onion morphology, slightly hyperintense to CSF on T1 and T2, but sometimes looks very similar to the CRL so it can be difficult to distinguish from an arachnoid cyst. These cases are very useful in diagnosing the investment recovery sequences in which signal $s$ esuprime CSF in arachnoid cysts, and dissemination sde sequence in which the epidermoid cyst shows high signal intensity (restricted diffusion). Not enhance after gadolinium administration. (Fig. 24).

**Meningeal carcinomatosis** is a relatively rare disorder that manifests clinically as a meningeal syndrome with cranial nerve palsies. Apart from the primary malignant CNS tumors such as gliomas, adenocarcinomas are the main cell type that affects leptomeninges.

**Images for this section:**
Fig. 1

**Pontine and Extrapontine myelinolysis**

**FIGURE 2:** Pontine and extrapontine myelinolysis in a 69 year-old man. Serial T2 FLAIR weighted MR images, show the classic indent-shaped pontine signal intensity abnormality and extrapontine signal intensity abnormality.
FIGURE 3: Pontine and extrapontine myelinolysis in a 60-year-old man. Serial T2 weighted MR images show the classic trident-shaped pontine signal intensity abnormality and extrapontine signal intensity abnormality.

Fig. 3
Fig. 4

**FIGURE 4.** Multiple sclerosis in a 42-year-old woman. Serial T2 and T2 FLAIR weighted MR images show multiple demyelinating plaques in the subcortical white matter and periventricular juxtaposition, with special predilection for the corpus callosum and infratentorial brain parenchyma.
FIGURE 5. Left lateral medullary stroke in a 55 year-old woman. Serial T2 and T2 FLAIR weighted MR images and DWI, show hyperintense lesion, left lateral medullary.
Fig. 6

**FIGURE 17.** Sagittal section of the pons showing the paths of different pontine arteries. A = pontine ventral (basilar) part; B = pontine tegmentum; 1 = basilar artery; 2 = the lower part of the pontine tegmentum, vascularized by ascending arteries; 3 = the middle part of the pontine tegmentum, vascularized by arteries with a straight path; 4 = the upper part of the pontine tegmentum, vascularized by descending arteries.
**Left paramedian pontine stroke**

**Fig. 7**

*Fig. 18. Left paramedian pontine stroke in a 72 year-old man: Serial T1 and T2 FLAIR weighted MR images and DWI show hyperintense lesion, left lateral pons.*
The vascularization of midbrain

**FIGURE 10.** Right lateral view of the midbrain showing the general arrangement of the midbrain arteries (Modified from D'Arcey MO). A = interpeduncular fossa; B = mamillary body; C = cerebral peduncle; D = lateral surface of the midbrain; E = superior colliculus; F = inferior colliculus; G = superior cerebellar peduncle; 1 = basilar artery; 2 = superior cerebellar artery, 2 prime = medial branch, 2 double prime = lateral branch; 3 = posterior cerebral artery; 4 = collicular artery; 5 = posteromedial choroidal artery; 6 = anterior choroidal artery; 7 = anteromedial group of midbrain arteries (middle group of interpeduncular arteries); 8 = anterolateral group of midbrain arteries; 9 = lateral group of midbrain arteries; 10 = posterior group of midbrain arteries; 11 = posterior group of pontine arteries (arteries of the posterior cerebellar peduncle).

Fig. 8
FIGURE 9. Posterior reversible encephalopathy in a 32 year-old man. Serial T2 and T2 FLAIR weighted MR images show diffuse hyperintense lesion in cerebellum, both cerebellar peduncles and white matter occipital. Right down to study the same patient a week later where the findings have been resolved.
Fig. 10

**FIGURE 10.** Diffuse glioma of brainstem in a 7 year-old child. Serial T1, T2 and T2 FLAIR weighted MR images, show hyperintense diffuse lesion in T2 images and hypointense diffuse lesion in T1 image.
**Diffuse glioma of brainstem**

**Fig. 11**

*Fig. 11. Diffuse glioma of brainstem in a 7 year-old child. Serial T1 with contrast weighted MR images, DWI and spectroscopy with T$\text{E}$ short and long, show several foci of uptake in the ring; no restrictions on diffusion and spectroscopy clearly shows a tumor.*
**Fig. 12**

**NEURINOMA**

*FIGURE 23. Neurinoma in a 37 year-old woman. Serial T2 and T2 FLAIR weighted MR images and T1 with contrast show a tumor in the right cerebellopontine angle. It is hyperintense on T2 and shows an intense enhancement after gadolinium administration.*
Hemibulbar stroke. Syndrome Babinski-Nagot

**Fig. 13**

Figure 13. Hemibulbar stroke. Syndrome Babinski-Nagot in a 60 year-old woman. Serial T2 and T2 FLAIR weighted MR images and DWI show hyperintense lesion, right hemibulbar.
Fig. 14
Langerhans cell histiocytosis (LCH)

**Fig. 15**

*Figure 15: Langerhans cell histiocytosis (LCH) in a 3-year-old child. Serial T2 and T2 FLAIR weighted MR images show hyperintense lesion, dentate nucleus millimeter in left cerebellar hemisphere. Patient with a history of LCH.*
Fig. 16
Bickerstaff’s brainstem encephalitis

Fig. 17

FIGURE 17. Bickerstaff’s brainstem encephalitis in a 62-year-old man. Serial T2 and T2 FLAIR weighted MR images show MRI scan demonstrated high-intensity abnormalities on T2-weighted images. Anti-GQ1b IgG antibody is in serum.
Fig. 18

**Figure 8.** Typical neurobehcet lesions in a 52-year-old man. Serial T2 FLAIR weighted MR images showed ill-defined high-signal-intensity areas involving the right thalamus, crus cerebri, cerebral peduncle, pons and midbrain.
Typical Neurobehcet Disease

Figure 9. Typical neurobehcet lesions in a 52-year-old man. Serial T2 weighted MR images show ill-defined high-signal-intensity areas involving the right thalamus, crus cerebri, cerebral peduncle, pons and midbrain.

Fig. 19
Typical Neurobehcet Disease

Figure 20. Typical neurobehcet lesions in a 52-year-old man. Serial T1 weighted MR images with contrast and DWI show minimal contrast enhancement and mild restriction.
Wallerian degeneration of the corticospinal tracts

Fig. 21
Wallerian degeneration of the corticospinal tracts

Fig. 22
Fig. 23

**Epidermoid cyst**

**FIGURE 23.** Epidermoid cyst in a 45-year-old man. Serial T2 and T2 FLAIR weighted MR images. T2 with contrast and DWI show cystic mass in tanks preoptic and invading the right cavernous sinus. Restricted in DWI and does not enhance after contrast.
Figure 24: In a 22-year-old woman, serial T2 and T2 FLAIR weighted MR images show multiple demyelinating plaques in the periventricular white matter and brainstem. Acute clinical.
Conclusion

**Magnetic resonance imaging** is the technique most used in the diagnosis of brainstem injury. Knowledge of imaging characteristics of lesions in MRI, the patient's age and location of the lesions are the most important criteria to establish a correct differential diagnosis.

**Personal Information**

**References**


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