Lesions of the corpus callosum: MRI findings and differential diagnosis

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Learning objectives

1. To assess the radiological MRI findings of diseases affecting the corpus callosum.

2. To review and illustrate these diseases in order to establish the correct differential diagnosis.
Background

The corpus callosum plays a fundamental role in the transfer of information and integration between the hemispheres. Different pathologies affect the corpus callosum, including in large groups: tumoural diseases, inflammatory-demyelinising events, vascular processes, traumatic injuries, endocrine and metabolic causes, infectious agents and toxic insults, among others. Radiologist should know their imaging features in order to establish the correct differential diagnosis. This exhibit reviews the radiological MRI findings in patients scanned at our centre demonstrating involvement of the corpus callosum.
Imaging findings OR Procedure details

The corpus callosum is the principal supratentorial cerebral commissure (white matter tract) connecting symmetrical areas of cerebral hemispheres (between the parietal lobes, posterior frontal and superior temporal regions). It is divided in four segments: rostrum, genu, body and splenium. It forms during embryogenesis, between the 8th and 20th week of life. It develops in an anterior-posterior direction except for the rostrum, which is the last to form. It contains very compacted myelinated axonal fibres, which hinder the diffusion of interstitial oedema and tumour extension. However, its high density also makes it more susceptible to shear injuries in a traumatic context.

Magnetic resonance imaging (MRI) is the most sensitive technique for the detection of lesions in the corpus callosum, considering both conventional and advanced techniques. This exhibit describes the radiological MRI findings of a variety of pathologies affecting the corpus callosum, including tumoural diseases, inflammatory-demyelinating events, vascular processes, traumatic injuries, endocrine and metabolic causes, infectious agents and toxic insults, among others.

1. CONGENITAL DISORDERS

1a) Agenesis and dysgenesis

There is a complete (agenesia) or partial (dysgenesia) absence of the corpus callosum (Fig. 1).

- The white matter tracts, which usually cross the midline, migrate ipsilaterally and go over the supero-internal region of the lateral ventricles forming the so called Probst bundles.

- Radial guidance and racing car sign configuration of the gyri.

- Parallel lateral ventricles and high-riding third ventricle adjoining the interhemispheric fissure.

- Colpocephaly: dilatation of the trigone and posterior and occipital temporal horns of the lateral ventricles.

Congenital malformations of the corpus callosum may occur in isolation although are frequently associated to other brain abnormalities, such as:

- Interhemispheric cyst: in 7% of patients with agenesis of the corpus callosum. It appears as a thin walled-unilocular cyst, usually hyperintense on T2W images and isointense on T1W images, although it varies depending on its protein content.
- **Lipoma**: 50% of lipomas are associated with different degrees of dysgenesis of the corpus callosum. It displays the characteristic short-T1 and T2 signal of fat (Fig. 2).

- **Dandy-Walker malformation**: agenesis or hypoplasia of the cerebellar vermis, cystic dilatation of the fourth ventricle and enlargement of the posterior fossa. It may be accompanied by supratentorial abnormalities such as agenesis or dysgenesis of the corpus callosum, polymicrogyria, cortical heterotopias and hydrocephalus (Fig 3).

- **Septo optic dysplasia (Morsier Syndrome)**: partial or total absence of the septum pellucidum and hypoplasia of the optic nerves. 50% have schizencephaly or heterotopias and 66% have hypothalamic-pituitary dysfunction (Fig 4).

- **Holoprosencephaly**: incomplete or absent division of the embryonic forebrain (prosencephalon) into distinct lateral cerebral hemispheres. Distinctive midline facial malformations occur in most cases. It is categorized into three types:
  - A-lobar: is the most severe. There is a complete absence of midline forebrain division, resulting in a monoventricle and fused cerebral hemispheres. There are remnants of normal cortical tissue in the anterior zone (horseshoe aspect) without identifying the falx cerebri, the corpus callosum and the olfactory nerves. The roof of the third ventricle can dilate and result in a large dorsal cyst.
  - Semi-lobar: there is an incomplete division of the cerebral hemispheres with H-shaped single ventricle with rudimentary occipital and temporal horns and a partial separation of the thalamus.
  - Lobar: is the least severe. It is not accompanied by craniofacial abnormalities. There is complete ventricular separation with focal areas of incomplete cortical division. Absent posterior falx cerebri and septum pellucidum with fusion of thalami. The corpus callosum is often incomplete or dysplastic (Fig 5).

- **Chiari II malformation**: more common in females (2:1). Downward displacement through the foramen magnum of the cerebellum, tonsils, brainstem and fourth ventricle into the posterior cervical canal. This condition is associated with a variety of findings:

  **Infratentorial malformations**:
  - Posterior fossa hypoplasia with a low tentorial attachment and elongation of the fourth ventricle.
  - Tethered cord mieloecele with lumbar or sacral myelomeningocele, possibly associated with a lipoma of the filum terminale.

  **Supratentorial malformations**:
  - Hydrocephalus due to obstruction in the IV ventricle pathway.
Agenesis or dysgenesis of the corpus callosum (in 75-90% of cases) and distortion due to hydrocephalus.

Enlargement of the heads of caudate nuclei and intermediate mass.

Interdigititation of gyri across the interhemispheric fissure by a fenestrated falx.

Stenogyria: brain sulcation disorder with packaging of small gyri with shallow sulci and preservation of the general sulcal pattern.

Variable degrees of fusion of the colliculi and tectum resulting in prominent breaking and inferior displacement of the tectal plate.

- Chiari III malformation: is the most serious and relatively uncommon. It consists of a posterior cervico-occipital encephalocele containing cerebellar dysplastic tissue, fourth ventricle and sometimes brainstem.

1b) Atrophy due to perinatal brain injury:

It is related to agenesis, dysgenesis or hypoplasia of the corpus callosum caused by an injury to the grey or white matter when it is not fully formed, that is, in the weeks 18 to 20 of gestation. Disorders that could lead to a damage include a viral infection, maternal drug or alcohol use during pregnancy or for example a hypoplasia of the corpus callosum due to severe anoxia (Fig 6), or hypoplasia secondary to hydrocephalus caused by Silvi's aqueduct stenosis (Fig 7).

2. TUMOURAL DISEASES

2a) Glioma

Glioblastoma multiforme (GBM) are the most common primary brain tumours in adults. They are the most aggressive type of glioma, usually supratentorial, which usually spread via direct extension along the white matter tracts, including the corpus callosum, resulting in a "butterfly pattern" by bihemispheric involvement. Hematogenous, subependymal and cerebrospinal fluid spread can also be seen. On MR imaging are characterized by being poorly demarcated and infiltrative lesions, with intratumoral necrosis and intense enhancement of the solid portion, although occasionally no enhancement is seen. Diffusion-weighted imaging shows restriction (lowest ADC) within the solid tumour component. There may be intratumoral bleeding including the development of pathologically deformed vessels (neoangiogenesis), fast tumour growth and direct invasion of the vessel wall. This explains the increase of relative cerebral blood volume (rCBV) values in dynamic T2*-weighted perfusion MR imaging. GBM associate a
moderate peritumoral oedema contributing to mass effect (Fig. 8). The MR spectroscopy study highlights higher levels in Cho/Cr and NAA/Cho ratios and the presence of lactate and lipids.

2b) Lymphoma

Primary central nervous system lymphomas are rare aggressive neoplasms of the brain, accounting for less than 2% of malignant primary brain tumours. They are almost always of the B-cell non-Hodgkin's type. Common locations include the corpus callosum, deep gray matter structures, and the periventricular region. Lymphomas differ from glioblastoma multiformes because they usually have less peritumoral oedema, are more commonly multiple, are less commonly necrotic, are highly radiosensitive, and frequently temporarily respond dramatically to steroid administration producing "vanishing lesions." These lesions are usually iso- or hypointense on T1W images and iso or hyperintense on T2W images. Lymphomas occur commonly in immunocompromised patients, showing a ring-shaped pattern contrast enhancement, whereas it is usually homogeneous and intense in immunocompetent patients. Diffusion-weighted imaging shows restriction within the tumour given its high cellularity (Fig. 9). The MR spectroscopy study includes elevated signal of lipid, lactate, and choline and reduced NAA, which cannot be differentiated from GBM.

2c) Juvenile pilocytic astrocytoma

They are a low-grade variant of astrocytoma. They are usually a well-defined unencapsulated mixed cystic-solid appearance mass. They are usually well-circumscribed unencapsulated masses, with frequent cyst formation, either microscopic or macroscopic. Most lesions commonly involve the cerebellar vermis, cerebellar hemispheres, optic chiasm, hypothalamus, or floor of the third ventricle. The corpus callosum is an uncommon location. On MR imaging they are hypo or isointense on T1W images and hyperintense on T2W images, in contrast to most low-grade infiltrative astrocytomas, which tend not to enhance.

2d) Intraventricular tumours with extension to the corpus callosum

2e) Metastasis (affected directly / indirectly by oedema)

Intracranial metastases represent the most common brain tumours in adults, occurring in 25-50% of all cancer patients. They are often supratentorial and multiple. In adults, lung cancer is the main cause of BMs (50-60%), followed by breast cancer (15-20%) and melanoma (5-10%) respectively, while tumours of the gastrointestinal tract and renal
cell carcinomas are less common origins of metastases to the brain. They are usually well-defined, in contrast to gliomas, with disproportionate vasogenic oedema relative to the size of metastases. They are located in the gray and white matter junction, following a vascular distribution. They also appear in the basal ganglia, the cerebellum, and are rarely involve the corpus callosum.

On MRI lesions are isointense to mildly hypointense on T1W images and hyperintense on T2W images. However, hemorrhagic metastases or melanoma lesions may be hypointense on T1W and hyperintense on T2W images, owing to the chronic breakdown of blood products. Following administering of intravenous contrast agent, different enhancement patterns may be seen: solid, ring-pattern, irregular, homogeneous or heterogeneous (Fig 10).

3. INFLAMMATORY-DEMYELINISING EVENTS

3a) Multiple Sclerosis (MS)

MS is an acquired demyelinating disease that affects young women more than men. Although MS plaques can be found in any region of the brain parenchyma, typical locations are the periventricular region, corpus callosum, centrum semiovale, and deep white matter structures and basal ganglia. According to the literature, 93% of MS patients show lesions in the corpus callosum, characteristically involving the callososeptal surface (Fig. 11). Eventually lesions may coexist with corpus callosum atrophy. These plaques typically are hyperintense on the proton density, T2, and fluid-attenuated inversion recovery (FLAIR) sequences of MRI. They appear hypointense on T1 weighted images without gadolinium and show enhancement during the acute phase. Plaques may also exhibit a reversible restriction of diffusion due to exocytotoxic intramyelinic oedema. Diffusion tensor imaging can demonstrate and measure lesional damage in white matter tracts and gray matter and in areas of normal-appearing white matter (Fig 12.).

3b) Susac Syndrome

Susac syndrome is a rare disease attributed to a microangiopathy affecting the arterioles of the brain, retina, and cochlea, characterized by mono-phase fluctuating episodes of the classic clinical triad of subacute encephalopathy, visual loss secondary to retinal branch occlusions, and sensorineural hearing loss. MRI demonstrates multiple white matter lesions, usually smaller than the MS lesions, and more prone to affect basal ganglia and thalamus. There may also be enhancement of leptomeninges, better shown in postcontrast fluid-attenuated inversion recovery sequences (Fig. 13).

3c) Acute disseminated encephalomyelitis (ADEM)
ADEM is an autoimmune disease with demyelisation of the CNS, which usually occurs after a viral infection, a vaccine, or less frequently associated with rheumatic fever or idiopathic form. The MRI shows multiple large T2W-hyperintenses lesions, located in the periventricular white matter, cerebral cortex, brainstem, cerebellum, spinal cord and optic nerves. Lesions may show nodular or ring enhancement (Fig 14). Differential diagnosis between ADEM and MS may be difficult although ADEM has usually a monophasic clinical course and no new lesions usually appear after 6 months from the onset.

**3d) Progressive multifocal leukoencephalopathy (PML)**

It is a rare AIDS-related demyelinising disease of the brain resulting from opportunistic infection with the JC virus. It only causes disease in immunocompromised conditions. MRI shows multifocal and asymmetric lesions that may coalesce into large lesions. They may occur anywhere although injuries predominate in subcortical white matter (especially with parieto-occipital and frontal lobes U-fibres involvement) and in the corpus callosum. This predilection accounts for the scalloped margins of the lesions. The lesions are typically hypointenses on T1W images and hyperintenses on T2W images. They are not usually associated with mass effect or enhancement after administration of intravenous contrast, although in some cases a slight enhancement of the periphery of the lesion and mass effect can be seen. These characteristics allow differentiation of PML from lesions such as GBM and lymphoma.

**4. VASCULAR PROCESSES**

**4a) Infarcts**

Isolated Ischemic lesions of the corpus callosum are rare so it is usually involved as a part of a large vascular distribution in a large-vessel ischemic event. Vascularisation of the corpus callosum is assured by the anterior communicating and pericallosal arteries for the rostrum and genu, the anterior cerebral arteries for the body and the posterior cerebral arteries for the splenium. Imaging characteristics of infarcts are the same as strokes in other locations. Ischemic lesions usually affect the splenium, followed by the body and genu, with preservation of the dorsal and ventral surface. Patients commonly have a history of hypertension, diabetes or other cardiovascular risk factors (Fig 15). Diffusion tensor imaging can evaluate the anisotropic changes of cerebral white matter tracks in patients with ischemic stroke (Fig.16).

**4b) Periventricular leukomalacia**
Periventricular leukomalacia is the most common ischemic brain injury in premature infants. The ischemia occurs in the border zone at the end of arterial vascular distributions. Characteristically occurs in the periventricular white matter adjacent to the lateral ventricles. There is atrophy and irregularity of the corpus callosum in advanced stages (Fig. 17).

4c) Arteriovenous malformation, aneurysm rupture or cavernoma

Vascular malformations may occur elsewhere in the brain parenchyma including to the corpus callosum. They are prone to bleed, causing a bruise or intraventricular haemorrhage. Most receive blood supply from anterior and posterior cerebral arteries, possibly bilateral, and drain into the internal cerebral vein or superficial interhemispherical veins.

4d) Traumatic injuries

- Callosotomy: in cases of refractory generalized epilepsy.

- Diffuse axonal injury (DAI): is a frequent result of traumatic acceleration/deceleration or rotational injuries that causes a loss of consciousness. DAI typically consists of several focal white-matter lesions measuring 1-15 mm. MRI demonstrates more lesions than CT-scan, especially magnetic susceptibility techniques, which are superior in detecting chronic haemoglobin degradation products because of the susceptibility effects of hemosiderin. Keeping in mind that these lesions may be non-bleeding, they are best visualized on sagittal T2W-FLAIR sequences. Otherwise, for the detection of hemorrhagic lesions in the acute phase T2W images are recommended, while after the third day stands on the usefulness of the T1W images for the detection of subacute-chronic microhaemorrhages. (Fig 18)

Grading is described according to the anatomic distribution of injury, which correlates with outcome:

- **Grade I**: involves grey-white matter interfaces
  - most commonly: parasagittal regions of frontal lobes, periventricular temporal lobes
  - less commonly: parietal and occipital lobes, internal and external capsules, and cerebellum
- **Grade II**: involves corpus callosum (frequently unilateral) in addition to stage I locations
  - most commonly: eccentric, posterior body and splenium but does advance anteriorly with increasing severity of injury
- **Grade III**: involves brainstem in addition to stage I and II locations
• most commonly: rostral midbrain, superior cerebellar peduncles, medial lemnisci and corticospinal tracts

5. ENDOCRINE AND METABOLIC CAUSES

5a) Metachromatic leukodystrophy

It is an autosomal recessive familial disorder with an enzymatic defect that causes demyelination. At T2-weighted MR imaging, metachromatic leukodystrophy manifests as symmetric confluent areas of high signal intensity on T2W images in the periventricular white matter with sparing of the subcortical U-fibers. The cerebellar white matter may appear hyperintense at T2W imaging. No enhancement is evident at MR imaging. The corpus callosum, internal capsule, and corticospinal tracts are also frequently involved. In the later stage of metachromatic leukodystrophy, corticosubcortical atrophy often occurs, particularly when the subcortical white matter is involved.

5b) Adrenoleukodystrophy

It is an autosomal recessive X-linked chromosomal disorder of childhood characterized by an enzymatic defect of peroxisome that produces demyelination of the CNS, adrenal cortex and testes. In the early stages symmetric white matter demyelination occurs in the parietooccipital (peritrogonal), sparing the subcortical U fibers and extending across the corpus callosum splenium. The affected cerebral white matter typically has three different zones. The inner zone appears moderately hypointense on T1W images and markedly hyperintense on T2W images, corresponding to irreversible gliosis. The intermediate zone represents active inflammation and breakdown in the blood-brain barrier. On T2W images it appears isointense or slightly hypointense and readily enhances after intravenous administration of contrast material. The outer zone represents the leading edge of active demyelination and it appears moderately hyperintense on T2W images and demonstrates no enhancement. Symmetrical abnormal areas of hyperintensity along the descending pyramidal tract are common at T2W imaging. There may also be calcifications in trigones and around the frontal horns of the lateral ventricles.

5c) Inherited metabolic disorders

There is a long list of hereditary metabolic disorders including mucopolisacaridosis and Kearns-Sayre syndrome. They can be distinguished by the distribution of lesions through the cerebral and cerebellar parenchyma affecting the gray matter (cortex and basal ganglia), the white matter (periventricular, deep and subcortical) and the cerebellum (nuclei and white matter).
**5d) Wernicke’s encephalopathy**

Wernicke's encephalopathy is an acute neurological syndrome resulting from thiamine (vitamin B1) deficiency. It is characterized by confusion, ataxia, abnormal eye movements, and loss of visual acuity. Typical MRI findings are represented by symmetric hyperintensity on T2W images in the thalami, mamillary bodies, tectal plate, and periaqueductal. Atrophy of these structures, the corpus callosum and supratentorial cortex may be found only in alcoholic patients. These lesions may show a restriction of diffusion because of excitotoxic oedema that may be reversible if treated appropriately.

**6. INFECTIOUS AGENTS**

Viruses are the most common pathogens causing encephalitis, mainly by the herpes virus infection. However, other pathogens such as bacteria (e.g., tuberculosis, Whipple's disease (Fig. 19), Lyme disease), rickettsia, fungi or parasites (e.g., toxoplasmosis, cysticercosis) may cause encephalitis and involve the corpus callosum. HIV also affects the corpus callosum splenium and fornix in patients with cognitive impairment, although there is a more common involvement of the white matter (spared the subcortical U fibers), and basal ganglia (usually sparing the cortical gray matter), without evidence of mass effect or enhancement. Otherwise, you should consider the possibility of HIV infection with other pathogens or coexistence of tumours such as lymphoma.

The infection of the brain parenchyma appears as ill-defined large areas of hyperintensity on T2W images predominantly affecting the cortical gray matter, although it can also affect, in addition, the white matter (corpus callosum included) or the deep gray nuclei. Mottled haemorrhagic areas as well as contrast enhancement may be seen. Restriction of diffusion may be often found as the unique and earlier finding, being unapparent on the conventional MRI sequences. Diffusion tensor imaging can demonstrate and measure lesional damage of the corpus callosum (as well as in the ipsilateral occipital lobe) and white and gray matter tracts in areas of normal-appearing white matter (Fig. 20). MRI findings are not specific and can mimic other diseases, so it is crucial a good clinical context to establish an accurate diagnosis and an early treatment in order to reduce mortality.

**7. TOXIC INSULTS**

**7a) Marchiafava-Bignami**

Marchiafava-Bignami is a rare disease associated with alcoholism though rarely also seen in patients without alcoholism. It is characterized by demyelination and necrosis.
of the corpus callosum with involvement of the central layers with relative sparing of the dorsal and ventral surfaces (which may be seen as a sandwich sign on sagittal MRI imaging). Since clinical symptoms can vary from cognitive impairment, gait disturbance, and hemiparesis to stupor, coma, and death, early recognition of neuroimaging characteristics is crucial for diagnosis and treatment.

There are different temporal clinical presentations. The corpus callosum (mainly the genu and splenium) appears enlarged because of oedema during the acute phase, demonstrating hyperintensity on T2 and proton density-weighted MR images with possible T2-hyperintense foci in the centrum semiovale, and hypointense on T1W images. During the subacute phase, cystic lesions and small foci of T2 hypointensity can develop mainly in the body of the corpus callosum, most likely because of hemosiderin, and often exhibit enhancement (Fig 21). After a few months, signal intensity alterations become less evident but residual atrophy of the involved structure (and, in some cases, also of the cortex) usually is present. Diffusion tensor imaging can demonstrate and measure lesional damage of white matter tracts as the corpus callosum (Fig. 22).

7b) Disseminated necrotizing leukoencephalopathy

Disseminated necrotizing leukoencephalopathy is a rare syndrome of progressive neurologic deterioration seen most often in patients who have received central nervous system irradiation combined with intrathecal or systemic chemotherapy in the treatment or prophylaxis of various malignancies. Diffuse white matter changes are explained because of microvascular occlusion and subsequent ischemia or excititotoxic oedema. Leukoencephalopathy is seen as diffuse or multifocal white matter hyperintensities on T2-weighted MR imaging, predominantly in periventricular regions with sparing of the subcortical U-fibers. However, these injuries can be demonstrated in the early stage only in the diffusion sequences, showing restriction. Although most signal changes are subclinical and usually transient, some rare patients have extensive signal changes and contrast-enhancing lesions accompanying rapid neurological deterioration after treatment with MTX, a condition known as disseminated necrotizing leukoencephalopathy. Other drugs, such as BCNU, melphalan, fludarabine, cytarabine, 5-fluorouracil, levamisole and cisplatin, have also been implicated.

7c) Changes in radiation therapy

MRI can detect demyelination associated with radiotherapy, which is called radionecrosis. As expected, radiation necrosis occurs most commonly at the site of maximum radiation delivery. It manifests as hyperintense areas on T2W images and can closely resemble recurrent tumour at imaging because of the following shared characteristics: origin at or close to the original tumour site, contrast enhancement, growth over time, oedema, and exertion of mass effect. With respect to the MR imaging
characteristics of radiation necrosis, most lesions consist of an enhancing mass with a central area of necrosis. The contrast enhancement of these lesions is secondary to radiation-induced endothelial damage, which leads to the breakdown of the blood-brain barrier. On T2-weighted images, the solid portion of the radiation-induced necrotic mass has low signal intensity, and the central necrotic component shows increased signal intensity.

These changes can occur in the early phase (during treatment), in the early delayed phase (in the first 3 months) or in the late delayed phase (starting from 3 months). It is also in this last phase where calcifications and microhaemorrhages are commonly found (because of vasculitis, formation of telangiectasia or other vascular malformations) in basal ganglia, dentate nuclei of the cerebellum. These microhaemorrhages are better depicted in magnetic susceptibility sequences (T2*W GRE sequence).

The use of platinum-based chemotherapy drugs, such as cisplatin and carboplatin, combined with radiation therapy may contribute to the development of radiation-induced necrosis.

8. TRANSIENT LESIONS

Transient lesions of the corpus callosum are probably associated with oedematous and/or inflammatory changes. Acute withdrawal of antiepileptics (carbamazepine, phenytoin, vigabatrina), a state of hypoglycemia (Fig. 23), epileptic status (Fig. 24), Wernicke, Marchiafava-Bignami, the use of sympathomimetics drugs (cocaine, fenilciclidina, hidroclorat, amphetamines), or virus infection (such as influenza, rotavirus, E. Coli, adenovirus and mumps), hemolytic uremic syndrome, syndrome of brain damage associated with the altitude are reported as risky factors related to this entity. MRI usually reveals an oval-shaped lesion of 1-2cm in the corpus callosum splenium, hypointense on T1W images and hyperintense on T2W images. These injuries show restriction of diffusion, which is usually reversible due to exocitotoxic oedema. These lesions usually disappear within a few weeks following adequate therapy.

9. MISCELLANEOUS

9a) After drainage cystic lesions of the adult chronic hydrocephalus

In patients with long-standing hydrocephalus after shunting, focal and diffuse lesions have been described in the corpus callosum, being hypointense on T1W images and hyperintense on T2W images, with sparing of the splenium. The mechanism responsible for the production of these lesions is unknown although it is postulated that may be the result of ischemia with subsequent demyelination caused by prolonged sever stretching...
of the corpus callosum from ventriculomegaly, followed by a rapid decompression of the ventricles.

9b) Chronic adult hydrocephalus

It is characterized by the following radiological findings (Fig 25):

- Enlargement of the temporal horns of the lateral ventricles > 2mm.

- Enlargement of both the Silvi’s fissures and convexity sulci, which helps differentiation from other causes of hydrocephalus.

- Rounding of the frontal horns of the lateral ventricles and third ventricle (biconvex morphology), with a measured Evans index of more than 0:33.

- "CSF Flow void sign" at the Aqueduct of Sylvius caused by an increase of cerebrospinal fluid peak velocity.

- Increased curvature of the corpus callosum.
Fig. 1: Axial (a) and sagittal (b) fluid-attenuated inversion recovery images show a complete agenesis of the corpus callosum with parallel orientation of the lateral ventricles (a) and radial direction of giry and sulci on the inner surface of the cerebral hemispheres around the third ventricle (b)

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Fig. 2: Sagittal fluid-attenuated inversion recovery image. Pericallosal lipoma in a patient without morphological alterations of the corpus callosum

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**Fig. 3:** Axial (a) and sagittal (b) fluid-attenuated inversion recovery image. Dandy-Walker malformation with complete agenesis of the corpus callosum with parallel orientation of the lateral ventricles (a). There is hypoplasia of the cerebellar vermis with a retrocerebellar cyst that communicates with the fourth ventricle and enlargement of the posterior fossa (b)

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Fig. 4: Axial T2-weighted (a) and sagittal fluid-attenuated inversion recovery (b) images.
Septooptic dysplasia (Morsier Syndrome). Total absence of the septum pellucidum (a) associated to schizencephaly, polymicrogyria and dysgenesis of the corpus callosum (b). Left parietal subdural cystic collection in a patient with a ventriculo-peritoneal shunting

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Fig. 5: Axial T2-weighted (a) and sagittal fluid-attenuated inversion recovery (b) images. Lobar holoprosencephaly. Lack of posterior falx with posterior expansion of the unique ventricle due to absence of the septum pellucidum and corpus callosum partial agenesis

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**Fig. 6:** Axial T1-weighted (a) and sagittal (b) images. Corpus callosum splenium hypoplasia due to a prenatal noxa (left parietal ischemic stroke) with ex-vacuo dilatation of the ventricular horn

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Fig. 7: Axial T2-weighted (a) and sagittal (b) images. Corpus callosum hypoplasia due to a hydrocephalus caused by Silvi's aqueduct stenosis and secondary supratentorial ventriculomegaly

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Fig. 8: MRI studies of two different patients (a and b) and (c and d) that show the radiological findings of GBM with involvement of the corpus callosum. Axial T2-weighted image shows a cystic-solid component with marked perilesional edema/infiltration (a) and a postcontrast axial T1-weighted image with heterogeneous enhancement of the solid portion (b). Axial perfusion study with an increase of rCBV (c), and an ADC map (d) with a diffusion restriction in the solid parasagittal portion of the lesion.

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**Fig. 9:** Axial T1-weighted image (a) and ADC map (b) that show the behaviour of a CNS lymphoma with involvement of the corpus callosum. Note the homogeneous contrast enhancement (a) and the diffusion restriction because of its high cellularity (b)

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Fig. 10: Axial T2*-weighted conventional GRE sequence (a) and fluid-attenuated inversion recovery (b) images. There are two metastatic lesions of lung cancer with hemorrhagic component (a) in the right parietal gray-white matter junction and adjacent to the left parietal side wall of the third ventricle, as well as extensive vasogenic perilesional edema with corpus callosum involvement (b)

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Fig. 11: MRI images of two different patients (a and b) and (c, d and e) with MS compatible lesions. Axial fluid-attenuated inversion recovery (a) and sagittal (b) images with multiple demyelinating plaques in the periventricular, subcortical and juxtacortical white matter (a) infratentorial lesions (b) and lesions on the calloseptal surface of corpus callosum (a and b). Axial fluid-attenuated inversion recovery (c) and double inversion-recovery sequence (d) and sagittal fluid-attenuated inversion recovery (e) showing multiple periventricular lesions (c and d) and involvement of the corpus callosum with atrophy (e)

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**Fig. 12:** A diffusion tensor tractography image overlaying a sagittal FLAIR MRI scan of the brain is shown. Patient with MS without crossing fibers in the genu of corpus callosum

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Fig. 13: Axial T2-weighted MRI (a), axial (b) and sagittal (c) fluid-attenuated inversion recovery images and axial T1-weighted postcontrast (d) in a patient with Susac syndrome. There are multiple small lesions in the basal ganglia (a), in the juxtacortical white matter (b), corpus callosum (red arrow in b and c), infratentorial lesions with enhancement (d) and leptomenengitis signs

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**Fig. 14:** Axial FLAIR (a), sagittal FLAIR (b) and axial T1-weighted postcontrast (c) images that show an ADEM. Big lesions can be observed in periventricular, subcortical and juxtacortical white matter, and corpus callosum, with nodular enhancement (c)

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Fig. 15: Axial diffusion with b1000 factor (a) and corresponding ADC map (b) where there is a focal lesion with restricted diffusion in the corpus callosum splenium corresponding to an ischemic infarction of the left posterior cerebral artery territory

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Fig. 16: A diffusion tensor tractography image overlaying an axial diffusion weighted image with b=1000 sec/mm². Patient with an infarction of the right posterior cerebral artery. Absence of crossing fibers in the splenium of corpus callosum and ipsilateral occipital lobe

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Fig. 17: Axial fluid-attenuated inversion recovery (a), axial T2-weighted (b) and sagittal fluid-attenuated inversion recovery (c) images. Periventricular leukomalacia in a preterm infant. Posterior periventricular hyperintense signal (more evident in a), associated with corpus callosum splenium hypoplasia compared with the genu and secondary mild dilatation of the posterior ventricular horns (b and c)

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Fig. 18: Axial venBOLD imaging (a), axial T2*-weighted conventional GRE sequence (b), and sagittal (c) and axial (d) fluid-attenuated inversion recovery images with patient movement artifacts (shaking). There is a hemorrhagic right frontal contusion and a small left injury in the corpus callosum splenium consistent with a DAI grade II, best demonstrated in venBOLD study related to T2*-weighted conventional GRE sequence (red arrows). Perilesional edema is shown in the fluid-attenuated inversion recovery (d) images.
Fig. 19: Axial T2-weighted (a), axial fluid-attenuated inversion recovery (b) and sagittal (c) and T1 postcontrast (d) images. Whipple's disease. Involvement of the thalami, deep white matter, brainstem and corpus callosum (in a, b and c) with a contrast enhancement area (shown in d)

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Fig. 20: A diffusion tensor tractography image overlaying a sagittal FLAIR MRI scan of the brain is shown. Previous patient with Whipple's disease. There is affectation of corpus callosum (abscese of crossing fibers), deep white matter and brainstem

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Fig. 21: Axial diffusion image with $b=1000$ sec/mm² (a) and axial fluid-attenuated inversion recovery images. Marchiafava-Bignami disease. There is a diffusion restriction predominantly in the corpus callosum splenium (a) also demonstrated in the fluid-attenuated inversion recovery sequence (b)

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Fig. 22: A diffusion tensor tractography image overlaying a sagittal diffusion image with $b=1000$ sec/mm$^2$ (a) and a color-coded maps diffusion tensor tractography of the corpus callosum in the same subject (b) are shown in a patient with an acute Marchiafava-Bignami disease. A decrease of number of crossing fibers within the body of corpus callosum is seen

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**Fig. 23:** Axial diffusion image with $b=1000$ sec/mm$^2$ (a) and corresponding ADC map (b) and fluid-attenuated inversion recovery (c) images. Severe hypoglycemia was demonstrated a diffusion restriction of the corpus callosum splenium, subcortical white matter and cerebral cortex.

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Fig. 24: A diffusion tensor tractography image overlaying an axial fluid-attenuated inversion recovery image with $b=1000$ sec/mm$^2$ (left) and an axial fluid-attenuated inversion recovery image (right). Patient with a metabolic encephalopathy after status epilepticus. On the left image there are both qualitative (colors on image) and quantitative measurements of fractional anisotropy (FA) and apparent diffusion coefficient (ADC). There is a decrease in FA and increase in ADC in the splenium relative to the rostrum, indicating lesion of splenium fibers. On the right image there is a subtle hyperintensity of the splenium due to oedema which is expected to be transient.

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**Fig. 25:** Coronal (a) and axial T2-weighted image (b), sagittal fluid-attenuated inversion recovery image (c) and sagittal phase-contrast flow quantification (d). Rounding of the frontal horns of the lateral ventricles and biconvex third ventricle (a) with dilatation of the ventricular system and increased Evans index (b), increased curvature of the corpus callosum (c) and aqueductal "CSF Flow void sign" (red arrow in d); findings in relation to chronic adult hydrocephalus

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Conclusion

This exhibit has revised and illustrated different pathologies affecting the corpus callosum based on radiological MRI findings, in order to establish a correct differential diagnosis.
References

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