Abnormalities of the corpus callosum - expect the unexpected: pictorial essay

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Learning objectives

To illustrate and discuss the spectrum of magnetic resonance imaging (MRI) features of both congenital and acquired most common pathologies of corpus callosum in fetuses, children and adults.
Background

Corpus callosum is one of the three interhemispheric commissures (anterior commissure, hippocampal commissure and corpus callosum) and the greatest of them. [1]. Its role is interhemispheric connection and coordination, as well as cognitive functions, social skills, problem solving and attention. [2]

Corpus callosum development is a very quick process and takes place in 13th week of gestational life. In its embryological development, corpus callosum is formed by two separate parts: the anterior one, consisting of the rostrum, genu, and body and the posterior one-splenium. The place of the fusion is the isthmus. It reaches its final shape in midgestation (week 20) but is still small and grows, initially by addition of fibers and later by myelination. The target volume is reached at the age of 6-9 years. [3]

Various pathologies can affect the corpus callosum and their characteristic appearances on MRI. Pathologies of the corpus callosum result in typical symptoms of interhemispheric discoordination. [2]
Findings and procedure details

This pictorial essay reviews the radiological MRI findings in patients scanned at our centre, demonstrating involvement of the corpus callosum of fetuses, children and adults.

The wide spectrum of lesions in the corpus callosum, both congenital and acquired, were illustrated.

1. Developmental Abnormalities

The most extreme form of developmental malformation of the corpus callosum is **agenesis**. Agenesis of the corpus callosum can be **complete (agenesis)** (Fig. 1 on page 11, Fig. 2 on page 11) or **partial (dysgenesis)** (Fig. 5 on page 13) [4]. It is an anatomically defined condition that result from disruption of the early stages of fetal callosal development. The diagnosis is based on a finding of absent callosal fibers as visualized through neuroimaging. Agenesis of the corpus callosum is typically accompanied by a characteristic dilatation of posterior lateral ventricles (colpocephaly) and often the presence of atypical fiber bundles (Probst Bundles) that run anterior to posterior just lateral to the interhemispheric fissure. [1] Suspected defects of the corpus callosum should be confirmed by MRI because in 80% of cases they coexist with other CNS pathologies [3].

Radiological terminology to describe corpus callosum abnormalities used in literature is rather confusing and heterogeneous. [4]

**Hypoplasia** consists of uniformly thin or partially underdeveloped corpus callosum in the posterior region. There are 3 subtypes of hypoplasia: hypoplasia without dysplasia (generalized hypoplasia but intact morphology) (Fig. 3 on page 12), apple core corpus callosum abnormality (hypoplasia of posterior corpus callosum) (Fig. 4 on page 12), and anterior remnant of corpus callosum (agenesis of the mid and posterior corpus callosum with an anterior remnant), based upon the physical appearance. [5]

**Dysplasia** without hypoplasia encompasses cases in which corpus callosum is morphologically abnormal but has no evidence of hypoplasia, and is represented by a case with hump-shaped corpus callosum. In hypoplasia with dysplasia of corpus callosum, there are 2 subtypes, stripe corpus callosum (uniformly thinned corpus callosum, with dysplasia) and kinked corpus callosum (hypoplasia and kinked). [5]

2. Pericallosal lipoma (corpus callosal lipoma) (Fig. 5 on page 13)
Intracranial lipomas are rare callosal or pericallosal lesions associated with persistence and maldifferentiation of the meninx primitiva, which is the embryonic precursor of the meninges. The morphology may be tubulonodular or curvilinear. Tubulonodular lipomas have a round or cylindrical shape, are larger in diameter, and are usually located in the anterior brain. There is a high association with frontal lobe anomalies, encephaloceles, and callosal dysgenesis. Curvilinear lipomas are thin lesions that curve posteriorly around the splenium. Associated callosal malformations are rarer and less severe [6].

On MRI, lesions are homogeneously T1 hyperintense and T2 hyperintense, following the signal of subcutaneous fat, and without contrast enhancement. Loss of signal on fat-suppressed or STIR images confirms the presence of internal fat.

3. Phakomatoses (Fig. 6 on page 14)

Phakomatoses belong to congenital diseases in which callosal abnormalities are observed. Neurofibromatosis type 1 or von Recklinghausen disease is the most frequent of them, with the estimated incidence of 1:3000.

Neurofibromatosis bright objects (UNO), called formerly unidentified bright objects (UBO), are T2 hyperintense and appear most often in the basal ganglia, brainstem, and posterior fossa. They are also found in the corpus callosum, mainly in the splenium. UNO are rare before the age of 4 years; they increase in number and volume till the age of 10-12 years and tend to resolve thereafter, so that after the age of 20 they are almost never seen. Usually they do not undergo malignant transformation but they can, so follow-up MRI studies are very important in NF1 patients [7]. Besides it has been shown that NF1 children have a significantly larger corpus callosum while their IQ is significantly lower than in control subjects [8].

4. Dysmyelinating disorders - Leukodystrophy (Fig. 7 on page 14, Fig. 8 on page 15)

Dysmyelinating disorders are a subset of white matter disorders characterised by abnormal myelination [9]. They are also known by the term leukodystrophy and are composed of a group of inherited conditions that are characterized by a defective structure and function of the myelin sheath [10].

They typically, although not invariably, affect children, and may affect the corpus callosum.
Metachromatic leukodystrophy is the most common form and is caused by arylsulfatase A deficiency. Other hereditary leukoencephalopathies include X-Linked Adrenoleukodystrophy, Metachromatic leukodystrophy, Alexander disease, Krabbe disease, Sudanophilic leukodystrophy and others.

MR imaging is highly sensitive in determining the presence and assessing the severity of underlying white matter abnormalities in patients with leukodystrophy. Although the findings are often nonspecific, systematic analysis of the finer details of disease involvement may permit a narrower differential diagnosis, which the clinician can then further refine with knowledge of patient history, clinical testing, and metabolic analysis. MR imaging has also been extensively used to monitor the natural progression of various white matter disorders and the response to therapy. [11]

5. Demyelinating diseases

5.1. Multiple sclerosis (MS) (Fig. 9 on page 16, Fig. 10 on page 17)

Multiple sclerosis (MS) is a relatively common acquired chronic relapsing demyelinating disease involving the central nervous system, and is the second most common cause of neurological impairment in young adults, after trauma [12]

Callosal involvement is typical of MS although it has never been included in the evolving diagnostic criteria of this disease [13].

On MRI, the disease is T2 and FLAIR hyperintense, with contrast enhancement in the acute stage. The presence of subcallosal striations or callosal-septal interface lesions, which are thin bands along the undersurface of the corpus callosum, is highly sensitive and specific (Fig. 6A). Dawson fingers are ovoid lesions that radiate perpendicularly from the lateral ventricles in a pattern thought to reflect perivenular demyelination (Fig. 6B). In the chronic phase, T1-hypointense lesions (dark spots or black holes) reflect increased water content secondary to extreme demyelination and axonal loss. Susceptibility artifact on gradient recalled-echo/susceptibility-weighted imaging sequences indicates macrophage and iron deposition in areas of permanent tissue destruction [14, 15]

5.2 Marchiafava-Bignami Disease (Fig. 11 on page 17)

Marchiafava-Bignami disease refers to a disorder resulting in demyelination of the corpus callosum. It was first described by two Italian pathologists who identified it in the autopsies of three patients who presented in status epilepticus and subsequently developed coma [16]
On MR images, patients with Marchiafava-Bignami Disease show areas of low T1 signal intensity and high T2 and FLAIR signal intensity in the body of the corpus callosum at times extending into the genu and the adjacent white matter. These lesions do not have mass effect and may show peripheral contrast enhancement during the acute phase. Eventually, the lesions cavitate and become well marginated. [17-20]

The lesions are difficult to visualize on CT scans, where they appear as hypoattenuated areas. [21]

Other lesions involving the corpus callosum that may have a similar appearance include infarctions, shearing injuries, and demyelination process. [18, 22]

6. Vascular lesions

6.1. Infarcts (Fig. 12 on page 18, Fig. 13 on page 19)

Because of a rich collateral blood supply, corpus callosal infarcts are rare and associated with systemic vasculitides, shower emboli, major ischemic stroke, or subfalcine herniation with mass effect. The splenium is most commonly affected, followed by the body and genu. [23]

CT lacks the necessary contrast resolution to identify mild edema associated with callosal infarcts.

On MRI, reduced diffusivity on diffusion-weighted imaging is the earliest sign, followed by edema with T2 hyperintensity and T1 hypointensity. Contrast enhancement is variable but more likely in the acute phase. In the subacute to chronic stages, edema evolves into gliosis or atrophy, with corresponding normalization of diffusivity. Hemorrhagic transformation may manifest as magnetic susceptibility and chronic hemosiderin on gradient recalled-echo and susceptibility-weighted imaging sequences.

6.2. Cavernous haemangioma (Fig. 14 on page 20)

Cerebral cavernous venous malformations, commonly known as cavernous haemangioma or cavernoma, are common cerebral vascular malformations, usually with characteristic appearances on MRI. It is a "mulberry-like" vascular malformation that is defined in histologic terms by blood cavities surrounded by a single layer of endothelium without muscular tissue or intervening brain parenchyma. This lesion may occur throughout the central nervous system but is more frequently demonstrated in the cerebral hemispheres. Callosal localization is less common. [24]
MRI is the modality of choice, demonstrating a characteristic "popcorn" or "berry" appearance with a rim of signal loss due to hemosiderin, which demonstrates prominent blooming on susceptibility weighted sequences.

T1 and T2 signal is varied internally depending on the age of the blood produces and small fluid-fluid levels may be evident.

Gradient echo or T2* sequences are able to delineate these lesions better than T1 or T2 weighted images. In patients with familial or multiple cavernous angiomas GRE T2* sequences are very important in identifying the number of lesions missed by conventional Spin echo sequences.

Susceptibility weighted imaging (SWI) may have sensitivity equal to that of GRE in detecting these capillary telangiectasias in the brain. SWI is also highly sensitive in detecting calcification as compared to T1 and T2 images. [25]

If a recent bleed has occurred, then surrounding oedema may be present. The lesions generally do not enhance, although enhancement is possible. [26].

6.3. Dilated perivascular spaces (Virchow-Robin spaces) (Fig. 15 on page 21, Fig. 16 on page 22)

Dilated perivascular spaces can occur throughout the cerebral white matter, including the corpus callosum.

They appear as well-circumscribed ovoid lesions isointense to CSF on all sequences. These lesions have no known clinical significance but increase in size and frequency with age. There is an association with mucopolysaccharidosis, in which accumulation of glycosaminoglycans dilates the Virchow-Robin spaces and produces a cribriform (état criblé) appearance in the white matter, corpus callosum, and basal ganglia. [23]

7. Neoplastic Lesions

7.1. Primary neoplastic lesions

7.1.1. Glioma

Glioblastoma multiforme (WHO grade IV astrocytoma) (Fig. 17 on page 23) is the most common primary brain malignancy, accounting for 12%-15% of all intracranial neoplasms. The prognosis is uniformly grim.
Glioblastoma occurs most frequently in the cerebral hemisphere of adults between 45 and 70 years of age. It is rare in the cerebellum and spinal cord, and fewer than 10% of cases are found in children, in whom the brainstem is affected more commonly than in adults [27].

Callosal GBM, in addition to the corpus callosum, affects also both cerebral hemispheres resulting in the typical "butterfly glioma". [28].

Conventional gadolinium-enhanced MR imaging is the standard technique for the evaluation of glioblastoma and typically demonstrates a large, heterogeneous mass in the cerebral hemisphere exhibiting necrosis, hemorrhage, and enhancement.

MR spectroscopy and perfusion MR imaging have been shown to be useful in prospective determination of tumor grade. At spectroscopy, elevation of choline and depression of NAA suggest tumor; metabolite ratios (choline-creatine, choline-NAA, NAA-creatine, myoinositol-creatine) exhibit relationships to tumor grade. At perfusion MR imaging, relative cerebral blood volume is increased in higher-grade astrocytomas [29, 30]

**Gliomatosis cerebri** has been deleted from the 2016 CNS WHO classification as a distinct entity, rather being considered a growth pattern found in many gliomas, including IDH-mutant astrocytic and oligodendrogial tumors as well as IDH-wildtype glioblastomas. [31, 32] *(Fig. 18 on page 24)*

Thus, widespread brain invasion involving three or more cerebral lobes, frequent bilateral growth and regular extension to infratentorial structures is now mentioned as a special pattern of spread within the discussion of several diffuse glioma subtypes. [33]

The tumour may be primary (de novo) or secondary, with the latter as a result from the spreading of a more focal glioma [34]. Gliomatosis cerebri can contain areas of WHO grade II or III tumours and rarely grade IV. [35, 36]

**7.1.2. Lymphoma (Fig. 19 on page 25)**

Primary CNS lymphoma accounts for approximately 16% of primary brain tumors. They preferentially affects immunocompromised patients and can involve or extend through the corpus callosum. Most of them are non-Hodgkin's and represent B-cell type.

The hypercellular histology manifests with increased density on unenhanced CT. Lesions are multifocal and nodular, tending to show less mass effect and peritumoral edema than expected for the size. They are most often isointense-hypointense on T1-weighted...
images, hypointense on T2-weighted images. Most lesions enhance in a solid or ring fashion, although nonenhancing lymphoma has been reported. DWI often shows restricted diffusion.

In patients with immunodeficiency lymphoma is more often multifocal, irregular, and heterogeneous in terms of signal intensity and ring enhancing.

Lymphoma is highly radiosensitive and regresses rapidly with steroid treatment (so-called "vanishing lesions") [37].

7.2. Metastases (Fig. 20 on page 26, Fig. 21 on page 27)

Corpus callosum may also be affected by metastases although it is reported to be rare. Callosal involvement is more frequent in case of infiltration by a lesion from the adjacent structures [38].

8. Trauma - traumatic brain injury (Fig. 22 on page 28, Fig. 23 on page 29)

Traumatic brain injury is a primary cause of neurologic deficits in patients with severe head trauma, most commonly high-speed motor vehicle accidents. The corpus callosum, particularly the posterior body and splenium, are preferentially involved because of their fixation to the overlying dura, with resulting torque injury.

CT is not sensitive for TBI, but shows hyperdense petechial foci of hemorrhage in 20% of cases.

MRI is a much more sensitive technique, with microhemorrhage detected as foci of magnetic susceptibility on T2*-weighted gradient-recalled echo or susceptibility-weighted imaging sequences (SWI). Nonhemorrhagic lesions show reduced diffusivity on diffusion-weighted imaging, possibly with surrounding edema. Chronic lesions are associated with hemosiderin and encephalomalacia. Diffusion-tensor imaging with tractography also holds promise for detailed assessment of axonal injury. [23]

9. Iatrogenic Injuries (Fig. 24 on page 30, Fig. 25 on page 31)

Corpus callosum may be injured as a result of shunting procedures in patients with hydrocephalus [6] as well as a result of various neurosurgical interventions.
Fig. 1: Complete agenesis of corpus callosum - a 17-year-old girl with history of fine motor and sensory perception impairments, as well as visuo-spatial organization and verbal memory impairments. MRI shows complete absent corpus callosum, with colpocephaly (dilatation of the occipital horns), ventricles run parallel (rather than the normal "bow-tie" configuration) and narrow frontal horns on T1W sagittal (A), T2W axial (B, D) and T2W coronal (C) image. Accidental finding: pineal cyst (well circumscribed fluid intensity cystic lesion within epiphysis).

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**Fig. 2:** Complete agenesis of corpus callosum (fetal MRI) - antenatal MRI scan at 24/25 weeks of gestation, shows complete absent corpus callosum on T2W sagittal (A) coronal (B) and axial (C) image.

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**Fig. 3:** Hypoplasia without dysplasia - a 2-year-old boy with 47,XY, +mar karyotype. MRI shows hypoplasia without dysplasia (generalized hypoplasia but intact morphology) of corpus callosum on T1W sagittal (A) and T2W (B) coronal images.

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**Fig. 4:** Apple core corpus callosum abnormality (hypoplasia of posterior corpus callosum) - a 48-year-old man with history of epilepsy. MRI shows hypoplasia of posterior corpus callosum (apple core corpus callosum abnormality), on T1W sagittal (A) and T2W axial (B) and coronal (C) images, associated with schizencephaly.

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**Fig. 5:** Pericallosal lipoma (corpus callosal lipoma) with partial agenesis of the corpus callosum - a 17-year-old boy with history of epileptic seizures. MRI shows partial absent corpus callosum with large tubulonodular pericallosal lipoma - T1W sagittal (A), coronal (C) and axial (F) hyperintense, T2W coronal (B) and axial (E) hyperintense, peripheral...
blooming on susceptibility-weighted imaging - SWI (D), loss of signal on fat-suppressed T1W images (G) and no contrast enhancement (H).

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**Fig. 6:** Phakomatoses in NF1 patient - a 30-year-old man with neurofibromatosis type 1 and history of epileptic seizures. MRI shows focal T2W (B, D) and FLAIR (A, C) hyperintense lesion in splenium of the corpus callosum, isointense to hyperintense on T1W (E) and without contrast enhancement (F).

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Fig. 7: Dysmyelinating disorders - a 10 months old boy with leukodistrophy. MRI shows bilateral T2W (B, D) and FLAIR (A, C) high signal intensity of white matter with callosal involvement.

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Fig. 8: Dysmyelinating disorders - a 2-year-old boy with metachromatic leukodistrophy. MRI shows bilateral T2W (A, B) and FLAIR (C) high signal intensity of the periventricular white matter with callosal involvement.

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**Fig. 9:** Multiple sclerosis (MS) - a 30-year-old man with multiple sclerosis. MRI shows bilateral, multifocal T2W (B, D) and FLAIR (A, C) hyperintense lesions, particularly periventricular, with callosal involvement and no restricted diffusion (E, F) - chronic lesions.

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**Fig. 10:** Multiple sclerosis (MS) - a 38-year-old man with multiple sclerosis. MRI shows bilateral, multifocal T2W (B, D) and FLAIR (A, C) hyperintense, T1W hypointense lesions (E), particularly periventricular, with callosal involvement and without contrast enhancement (F) - chronic lesions.

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Fig. 11: Marchiafava-Bignami Disease - a 38-year-old chronic alcoholic man with headaches. MRI shows areas of T2W (A, D) and FLAIR (B, C) hyperintense signal intensity in the body and genu of the corpus callosum without mass effect.

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**Fig. 12:** Infarct of corpus callosum - a 62-year-old woman. MRI shows focal T1W (A) hypointensity, T2W (B, D) and FLAIR (C) hyperintensity with small zone of restricted diffusion (E, F) - focal chronic/subacute infarct in splenium of corpus callosum.

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**Fig. 13:** Infarct of corpus callosum - a 61-year-old woman. MRI shows focal T1W (A) hypointensity, T2W (B, D) and FLAIR (C) hyperintensity without restricted diffusion (E, F) - chronic pericallosal infarct with affecting the body of corpus callosum. Additional finding: infarct in pons.

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Fig. 14: Cavernous haemangioma - a 37-year-old woman (history of operated meningioma) MRI shows two small focal T1W (A), T2W (C, D) and FLAIR (B, E) "popcorn" or "berry" like lesions in genu of corpus callosum, with blooming artefact on susceptibility weighted imaging - SWI (F) - cavernous haemangioma or cavernoma.

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Fig. 15: Dilated perivascular spaces (Virchow-Robin spaces) - a 10-year-old boy with headaches. MRI shows multiple linear and small tubular/ovoid lesions in corpus callosum, isointense to CSF on all sequences T1W (E), T2W (A, B, C, D, E, F, G) and FLAIR (H).

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**Fig. 16:** Dilated perivascular spaces (Virchow-Robin spaces) - 49-year-old woman with headaches. MRI shows single small tubular/ovoid lesion in splenium of corpus callosum, isointense to CSF on all sequences T1W (A), T2W (B, D) and FLAIR (C), with no restricted diffusion (E, F).

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Fig. 17: Glioblastoma multiforme (WHO gr. IV) - a 53-year-old man. MRI shows infiltrative lesion of both frontal lobes and centrum semiovale with transcallosal spread (typical "butterfly glioma"), on axial T2W (A), FLAIR (B) and T1W with a gadolinium contrast agent images, and with restricted diffusion (D and E). ASL PI (F) shows hypervascular lesion consistent with glioblastoma multiforme (WHO gr. IV).

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Fig. 18: Diffuse glioma (former gliomatosis cerebri) - a 49-year-old man. MRI shows infiltrative lesion of both frontal lobes, centrum semiovale, left temporal and partially left parieto-occipital lobe, with transcallosal spread on T2W (A, C) and FLAIR (B) images, and with minimal zones of restricted diffusion on ADC map (E) and contrast enhancement (G). Spectroscopy (D, H) shows areas of WHO grade II-IV.

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Fig. 19: CNS lymphoma - a 31-year-old woman with epileptic seizure. MRI shows infiltrative T2W (A, E) and FLAIR (B) hyperintense lesion, affecting corpus callosum, with partially restricted diffusion - high signal intensity on DWI (F) with corresponding low signal intensity on the ADC map (G), postcontrast enhancement (C, D), and spectroscopy (H) consistent with CNS lymphoma.

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Fig. 20: A 27-year-old man with brain metastasis from testicular germ cell tumour. MRI shows infiltrative T2W (A) and FLAIR (B) hyperintense lesion affecting corpus callosum, with restricted diffusion - high signal intensity on DWI (C) with corresponding low signal intensity on the ADC map (D), and postcontrast enhancement (E, F, G) and increased FDG uptake on PET/CT (H).

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Fig. 21: A 72-year-old man with lung cancer. MRI shows focal, partially haemorrhagic lesion affecting corpus callosum on T1W (A, G), T2W (B, D), FLAIR (C), DWI and ADC (E, F) and on postcontrast T1W (H) image - lung cancer metastasis with perifocal edema.

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Fig. 22: Traumatic brain injury - a 20-year-old man, two years after car accident and coma. MRI shows multiple foci of microhemorrhage - T2W (A, B, D) and FLAIR (C) hypointense, with blooming artefact on susceptibility weighted imaging - SWI (E, F), affecting corpus callosum - consistent with traumatic brain injury. Additional finding: cavernoma in left frontal lobe.

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**Fig. 23:** Traumatic brain injury - a 24-year-old woman, ten years after bicycle accident and head trauma. MRI shows foci of microhemorrhage - T1W (A), T2W (B, D) and FLAIR (C) hypointense, with blooming artefact on susceptibility weighted imaging - SWI (F), affecting splenium of corpus callosum - consistent with traumatic brain injury.

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Fig. 24: Postoperative defect - a 35-year-old man, tree years after operation of glioma. MRI shows postoperative defect of left frontal lobe, affecting partially genu and rostrum of corpus callosum - T2W (A), FLAIR (B), T1W (C) and postcontrast (D) image.

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Fig. 25: Iatrogenic injury - a 35-year-old man, five years after operation of meningioma front-parietal left. MRI shows tip of the CSF shunt stabbed in body of corpus callosum - T1W (A, B), T2W (C, D), SWI (E) images. Additional finding: cavernoma in left frontal lobe T2W (F) image.

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Conclusion

MRI is a method of choice in the assessment of the corpus callosum and its congenital and acquired pathological lesions.

Visualization of corpus callosum involvement helps to establish diagnosis in certain disease entities.
References


3. Raybaud C. The corpus callosum, the other great forebrain commissures, and the septum pellucidum: anatomy, development, and malformation. Neuroradiology. 2010:52(6);447-77.


