Lesions of the Corpus Callosum

This article reviews the causes and imaging appearances of lesions involving the corpus callosum (Table 1). The corpus callosum consists of densely bundled white matter tracts connecting the two cerebral hemispheres, with a compact structure that largely blocks interstitial edema and tumor spread. Isolated lesions of the corpus callosum are rare and may represent transient responses to injury or myelination abnormalities. More common butterfly lesions involve the corpus callosum and both cerebral hemispheres—a pattern associated with aggressive tumors, demyelination, and traumatic brain injury.

Unenhanced CT is a first-line neuroimaging modality. Although soft-tissue contrast enhancement is limited, CT can help characterize hemorrhage, edema, mass effect, calcification, and necrosis. MRI provides more detailed information regarding tissue structure and composition, using various pulse excitation sequences, such as FLAIR, to distinguish abnormal signal in the corpus callosum from adjacent CSF in the lateral ventricles. Administration of IV contrast material is useful for characterization of neoplastic and vascular lesions, which may show characteristic patterns of enhancement.

Neoplastic Lesions

Glioma

Glioblastoma multiforme (GBM) is the most common and aggressive primary brain tumor in adults, classified histologically as World Health Organization (WHO) grade IV, with a median survival time of 14 months. Tumors can involve both cerebral hemispheres, with extension across the corpus callosum producing a classic butterfly appearance. On CT, GBM appears as an irregular, heterogeneous mass with peritumoral vasogenic edema or mass effect and possible internal areas of necrosis, calcification, and hemorrhage (Fig. 1A). On MRI, tumor is heterogeneously T1 hypointense and T2 hyperintense, with possible foci of susceptibility, vascular flow voids, and irregular contrast enhancement (Fig. 1B).

Gliomatosis cerebri is a slow-growing, diffuse form of glioma, classified as WHO grade III. By definition, it infiltrates two or more lobes and shows minimal contrast enhancement (Fig. 2). The prognosis is slightly better than GBM, with a median survival time of 12 months.
Primary CNS lymphoma is a rare and aggressive neoplasm that preferentially affects immunocompromised patients and can involve or extend through the corpus callosum. 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 [1, 2] (Fig. 3A). MR signal is generally T1 iso- to hypointense and T2 iso- to hyperintense to gray matter (Fig. 3B). There is usually slow internal diffusion and mild homogeneous contrast enhancement (Fig. 3C). An important variant is HIV-associated lymphoma, which is more likely to produce significant surrounding edema as well as central necrosis with characteristic ring enhancement. Lymphoma is highly radiosensitive and regresses rapidly with steroid treatment (so-called “vanishing lesions”) [3].

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Meningioma

Meningioma is the second most common primary CNS neoplasm and the most common nonglial tumor. In less than 10% of cases, lesions arise from midline and parasagittal dural surfaces, and can secondarily involve the corpus callosum. On CT, there is increased attenuation due to hypercellularity as well as coarse internal calcifications. On MRI, contrast enhancement is avid and generally homogeneous, although hemorrhage, cavitation, and necrosis may be seen in larger lesions (Fig. 4).

Metastasis

Metastasis to the corpus callosum is rare. More frequently, there is contiguous extension of tumor from adjacent structures. Imaging characteristics depend largely on the primary malignancy but generally involve mass effect, vasogenic edema, and heterogeneous contrast enhancement (Fig. 5).

Neurodystrophic Demyelinating Diseases

Demyelinating diseases are acquired disorders of adulthood in which formerly normal white matter is progressively destroyed by infectious, inflammatory, autoimmune, genetic, toxic, or metabolic causes.
Fig. 5—Intracranial metastases from primary lung cancer.
A and B, Contrast-enhanced CT (A) and contrast-enhanced T1-weighted MR (B) images show heterogeneously enhancing dominant metastasis in left frontal lobe (asterisk). Additional enhancing foci are noted in splenium (S), genu (G), and periventricular regions (arrows).

Fig. 4—Meningioma.
A, Unenhanced CT image shows round well-circumscribed hyperdense mass (asterisk) centered in falx cerebri (arrow) and extending across midline. There is subtle surrounding edema that involves bilateral temporooccipital regions.
B, T1-weighted contrast-enhanced MR image shows relatively homogeneous contrast enhancement within mass, which deviates surrounding pial vessels (arrows).
C, FLAIR MR image shows vasogenic edema extending into both parietal lobes, creating butterfly pattern (arrows).
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Multiple sclerosis—Multiple sclerosis (MS) is the most common demyelinating disorder, with presumed autoimmune and inflammatory causes. CT has a low positive predictive value for detection of MS lesions. 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 [4, 5].

Progressive multifocal leukoencephalopathy—Progressive multifocal leukoencephalopathy is a rare demyelinating disease caused by John Cunningham virus replication in immunocompromised patients. There is resulting destruction of subcortical white matter, showing T1 hypointensity and T2 hyperintensity on MRI. Lesions are typically multifocal and asymmetric, with progressive enlargement and confluence over time. Corpus callosal involvement has been described in 10–15% of published studies [1, 6–8]. There is no or minimal peripheral contrast enhancement because of the patient’s inability to mount an adequate inflammatory response.

Hereditary Leukoencephalopathies

Hereditary leukoencephalopathies are congenital disorders of myelin formation or maintenance, which may affect the corpus callosum. Metachromatic leukodystrophy is the most common form and is caused by arylsulfatase A deficiency. Diffuse symmetric demyelination results, with increased T2/FLAIR signal and global cerebral atrophy. Adrenoleukodystrophy is a peroxisomal disorder due to acyl-CoA synthetase deficiency, which produces cerebral and adrenal cortical dysfunction. Demyelination typically progresses from the parietooccipital to frontotemporal lobes and involves the corpus callosum from posterior to anterior. Affected regions appear hypointense on T2-weighted FLAIR images and hypointense on T1-weighted images with variable enhancement (Fig. 7). In certain cases, lesions may calcify or produce mass effect. Other hereditary leukoencephalopathies include Alexander disease, Krabbe disease, and Sudanophilic leukodystrophy.

Wallerian Degeneration

Wallerian degeneration is a process of antegrade neural disintegration that develops after injury to the proximal axon or cell body. For example, bilateral cerebral infarction can produce atrophy of the intervening corpus callosum due to Wallerian degeneration of the commissural fibers. Diffusion-weighted imaging is sensitive for early detection and quantification of callosal degeneration [9–12] (Fig. 8).

Fig. 6—Multiple sclerosis (MS).
A, Midline sagittal FLAIR MR image shows hyperintensity at interface between posteroinferior corpus callosum (C) and septum pellucidum of lateral ventricles (S). Callosal-septal interface lesions are highly sensitive and specific for MS.
B, Parasagittal FLAIR image shows radiating periventricular hyperintensities (subcallosal striations or Dawson fingers), consistent with perivenular demyelination. Scattered nodular foci in deep white matter (arrows) are also reflective of MS plaques.
Traumatic brain injury (TBI) 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 (Figs. 9A and 9B). 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 (Figs. 9C and 9D). 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 [13–17].

**Hypoxic-ischemic encephalopathy**—Hypoxic-ischemic encephalopathy is a pattern of brain injury resulting from partial oxygen deprivation due to hypoxic, hypoxemic, ischemic,
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or histotoxic causes. Lesions appear hypodense on CT and T1-hypointense and T2-hyperintense on MRI (Fig. 10A) because of associated edema and encephalomalacia. Reduced diffusivity (Fig. 10B) and contrast enhancement vary with the stage of injury. The corpus callosum is affected in severe or advanced cases.

Marchiafava-Bignami disease—Marchiafava-Bignami disease is primary demyelination of the corpus callosum after severe vitamin B₁₂ deficiency. Usually seen in chronic alcoholic patients, the clinical course may be acute and fulminant, affecting the genu and splenium; or subacute to chronic, involving the body of the corpus callosum. In the early phase, affected areas are edematous with T₂ FLAIR hyperintensity, T₁ hypointensity, reduced diffusivity, and variable contrast enhancement (Fig. 11). There is selective involvement of the central layer of the corpus callosum, resulting in a sandwich-like appearance. In the chronic stages, affected regions become necrotic and atrophic, with minimal contrast enhancement. There may be associated hemorrhage with susceptibility on gradient recalled-echo or susceptibility-weighted imaging sequences.

Fig. 9—Traumatic brain injury. A and B, Unenhanced CT images at level of lateral (A) and third (B) ventricles show hyperdense areas of acute hemorrhage in splenium (S) and genu (G). C and D, T₂* gradient recalled-echo MR images at same levels confirm magnetic susceptibility in these areas. There are multiple additional foci of microhemorrhage throughout gray-white mater junction and basal ganglia (arrows).
In patients with chronic hydrocephalus, placement of a new ventriculostomy catheter may yield T1-hypointense and T2-hyperintense MR signal abnormalities in the corpus callosum. The mechanism is incompletely understood and may involve biomechanical compression, edema, ischemia, or demyelination or a combination. This entity should be considered when new enlargement and signal changes are seen in the body of the corpus callosum. Often, there is a scalloped appearance of the dorsal surface due to segmental tethering by pericallosal artery branches (Fig. 12). No associated clinical symptoms have been reported [18].

**Congenital Callosal Malformations**

Embryologically, the corpus callosum develops between the eighth and 20th weeks of gestation. Growth is primarily from anterior to posterior with the genu forming first, followed by the anterior body, posterior body, and splenium. The rostrum is the last to form at 18–20 weeks [19].
Embryonic insults at different stages of development result in varying degrees of callosal dysgenesis. On midline sagittal T1-weighted MRI, a radiating sunburst gyral pattern may be seen because of nonconvergence of the major fissures. Absence of the splenium is associated with posterior cerebral hypoplasia and ventricular colpocephaly. Complete agenesis produces parallel “racecar” lateral ventricles and white matter tracts known as “Probst bundles.” The third ventricle is also high riding and may form an interhemispheric arachnoid cyst at the level of the absent corpus callosum (Fig. 13).
Lipoma
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 [20].

On CT, lesions are homogeneous and well circumscribed, showing internal fat density (less than −30 HU) and occasional rim calcification. On MRI, lesions are homogeneously T1 hyperintense and T2 hyperintense, following the signal of subcutaneous fat, and without appreciable contrast enhancement (Fig. 14). Opposed-phase images may reveal chemical-shift artifact due to macroscopic (bulk) fat. Loss of signal on fat-suppressed or STIR images confirms the presence of internal fat.

Vascular
The corpus callosum is supplied by multiple small penetrating vessels arising from the anterior and posterior pericallosal arteries, which branch respectively from the anterior and posterior cerebral arteries. Additional branches may arise from the anterior communicating arteries, including subcallosal and median callosal arteries.

Infarct
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. 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 (Fig. 15). 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.

Aneurysm
Midline and parasagittal cerebral aneurysms can occasionally rupture into the corpus callosum. These usually arise from the distal anterior cerebral (Fig. 16A) or anterior communicating arteries (Figs. 16B–16E). Unenhanced CT is useful for localizing areas of hemorrhage, but
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patients should be promptly evaluated with CT or MR angiography to assess the status of the intracranial circulation. Treatment depends on aneurysm size and location and may involve transarterial coil embolization or surgical craniotomy with clipping of the aneurysm neck. Cerebral vasospasm, a feared complication of aneurysm rupture, may produce additional ischemia or infarction.

Arteriovenous malformation—Cerebral arteriovenous malformations (AVMs) involve the corpus callosum in 10% of cases, with vascular involvement of both cerebral hemispheres. Unenhanced CT is useful for identifying callosal hemorrhage, edema, mass effect, and calcified phleboliths. Contrast-enhanced CT angiography or time-of-flight MR angiography can help delineate the feeding arteries, central nidus, and draining veins. MRI may also show flow-associated signal voids and magnetic susceptibility artifact in regions of calcification.

Small asymptomatic AVMs can be managed expectantly. Therapies for symptomatic lesions include endovascular coil embolization, surgical resection, and stereotactic radiosurgery directed at the central nidus. Outcomes correlate with the Spetzler-Martin grading system, which includes

Fig. 15—Callosal infarcts.
A, Left anterior cerebral artery infarct (thick arrow) with extension into genu (thin arrow) is seen on diffusion-weighted (left) and apparent diffusion coefficient (ADC) (right) images, which confirm reduced diffusivity in these areas.
B, Splenial infarct after evacuation of left epidural hematoma. Susceptibility artifact is noted in left occipital lobe. There is postoperative subdural hygroma along left cerebral convexity (asterisk), resulting in mild rightward subfalcine herniation. Diffusion-weighted (left) and ADC (right) images show acute splenial infarct (arrow) secondary to compression of corpus callosum during herniation.
Fig. 16—Aneurysms.
A, CT angiogram of head shows right pericallosal artery aneurysm (arrow) abutting anterior corpus callosum, with coarse peripheral calcification.
B, Unenhanced CT image in different patient who had ruptured anterior communicating artery aneurysm. Lobulated hyperdense collection (asterisk) suggestive of acute hematoma is visualized in midfrontal region, involving genu of corpus callosum.
C, T2* gradient recalled-echo MR image verifies magnetic susceptibility in region.
D, Sagittal contrast-enhanced magnetization-prepared 180° radiofrequency pulses and rapid gradient-echo (MP RAGE) image show lobulated rim-enhancing collection (asterisk) involving genu.
E, Time-of-flight maximum-intensity-projection image of circle of Willis depicts T1-hyperintense hematoma (asterisk) surrounding ruptured anterior communicating artery aneurysm.
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the size of the nidus, eloquence (degree of functional importance) of adjacent brain, and depth of venous drainage.

Callosal gliosis—Focally increased T2 signal in the anteroinferior portion of the splenium commonly occurs in patients with small-vessel ischemic disease secondary to aging, subcortical arteriosclerotic encephalopathy (Binswanger disease), or radiation therapy. Best appreciated on FLAIR MRI, this condition corresponds pathologically to subependymal gliosis. There is an association with periventricular white matter disease (leukoaraiosis), which appears hypodense on CT and hyperintense on T2-weighted MRI (Fig. 17).

Periventricular leukomalacia—Periventricular leukomalacia frequently affects premature infants and is thought to represent watershed infarction in the setting of low flow or decreased oxygen states. Severe cases may involve the corpus callosum, with cystic changes or hemorrhage reflecting ischemic damage to hemispheric white matter tracts. MRI is useful for identifying abnormal areas of signal change, magnetic susceptibility, and reduced diffusivity (Fig. 18).

Virchow-Robin spaces—Virchow-Robin spaces are dilated perivascular spaces that 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 (Fig. 19).

Fig. 17—Callosal gliosis. Axial FLAIR image in patient with left frontotemporal glioma after radiation shows vasogenic edema surrounding lesion as well as white matter hyperintensities in splenium (arrow) and periventricular regions.

Fig. 18—Periventricular leukomalacia. A, T2-weighted MR image shows periventricular white matter abnormalities, with increased signal in both frontal and parietal lobes as well as genu (G) and splenium (S) of corpus callosum. Susceptibility artifact within choroid plexus of occipital horns (asterisk) confirms presence of hemorrhage. B, Diffusion-weighted MR image identifies reduced diffusivity within splenium (arrows) and multiple other areas.
Transient splenial signal changes have been reported in a wide variety of conditions, including seizures, antiepileptic drug toxicity or withdrawal, sympathomimetic-induced kaleidoscopic visual illusions, viral encephalitis, bacterial and parasitic meningitis, hypoglycemia, hypernatremia, osmotic myelinolysis, Wernicke encephalopathy, and hemolytic-uremic syndrome. Lesions are well circumscribed and located in the central splenium, with T2 and FLAIR hyperintense and T1 hypointense signal. In the acute phase, contrast enhancement and reduced diffusivity may be seen (Fig. 20). After disease recovery or withdrawal of the offending agent, imaging abnormalities resolve over several weeks to months. The pathophysiology is poorly understood.

**Infection and Inflammation**

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**Fig. 19**—Virchow-Robin spaces in Hunter syndrome (mucopolysaccharidosis type 2). (Courtesy of Prabhu SP, Boston Children’s Hospital, Boston, MA)

**A**, Axial FLAIR image shows diffusely enlarged perivascular spaces, which are isointense to CSF and involve body of corpus callosum (arrows).

**B**, Sagittal T1 FLAIR contrast-enhanced image shows large Virchow-Robin spaces within corpus callosum (arrow) and brain parenchyma.

**Fig. 20**—Transient splenial intensity changes.

**A–C**, Example images in migraine and visual scotomas (**A**), adenovirus encephalitis (**B**), and metronidazole toxicity (**C**) show FLAIR hyperintensity and reduced diffusivity in splenium (arrows), which resolved on follow-up imaging.

(Fig. 20 continues on next page)
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Fig. 20 (continued)—Transient splenial intensity changes. A–C, Example images in migraine and visual scotomas (A), adenovirus encephalitis (B), and metronidazole toxicity (C) show FLAIR hyperintensity and reduced diffusivity in splenium (arrows), which resolved on follow-up imaging.

understood but is thought to reflect reversible demyelination and intracellular edema secondary to infectious, inflammatory, or metabolic disturbances.

References

Ho et al.


**Suggested Reading**