BRAIN CME

In and Around the Optic Chiasm: A Pictorial Review of Neuroimaging

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ABSTRACT

The optic chiasm is a key anatomic structure along the visual pathway, situated at the crossroads between the optic nerves and tracts. A wide range of diseases can affect the optic chiasm and its surrounding strategic region at the base of the brain. Management strategies for optic chiasm abnormalities vary substantially, depending on the abnormalities revealed on neuroimaging. Scant attention has been paid to date to the comprehensive classification of neuroimaging manifestations of optic chiasm abnormalities. We comprehensively reviewed and presented the imaging findings in a wide spectrum of pathologies that originate from or involve the optic chiasm. This review will aid in differentiating the many neuroimaging appearances of lesions in this region.

Learning Objective: List the lesions that involve the optic chiasm and classify them according to their etiology.

INTRODUCTION

The optic chiasm is a key anatomic structure along the visual pathway, situated at the crossroads between the optic nerves and tracts. As is the case for many anatomic names, its structure defines its name: "chiasma" is indeed derived from the Greek *χιάζω*, meaning "to mark with an X" (after the Greek letter *χ*), which is the shape of the chiasm when looked at from above or below. The optic nerves come together within the chiasma to allow the crossing of fibers from the nasal retina to the contralateral optic tract; this enables visual inputs from the nasal half of the eye to be processed by the contralateral occipital lobe. It is estimated that approximately 53% of fibers cross within the optic chiasm.[1] The intracranial course of the optic tracts arises from the posterior aspect of the chiasm, with an angle between 15° and 45°.

When looking at the regional anatomy (Fig 1), the chiasm sits just below the hypothalamus, overlies the tuberculum sellae of the sphenoid bone and the pituitary gland, and lies within the wall of the third ventricle; it is surrounded by CSF, with the chiasmatic cistern being just anterior. The infundibulum of the pituitary lies immediately posteriorly, and the mammillary bodies are behind this, medial to the 2 optic tracts. The optic chiasm also lies at a crossroads of blood vessels; the internal carotid arteries lie on either side, and the anterior communicating artery is just directly above

ABBREVIATION KEY

AIDS = acquired immunodeficiency syndrome
CCF = carotid-cavernous sinus fistula
GBM = glioblastoma
NCC = neurocysticercosis
TB = tuberculosis

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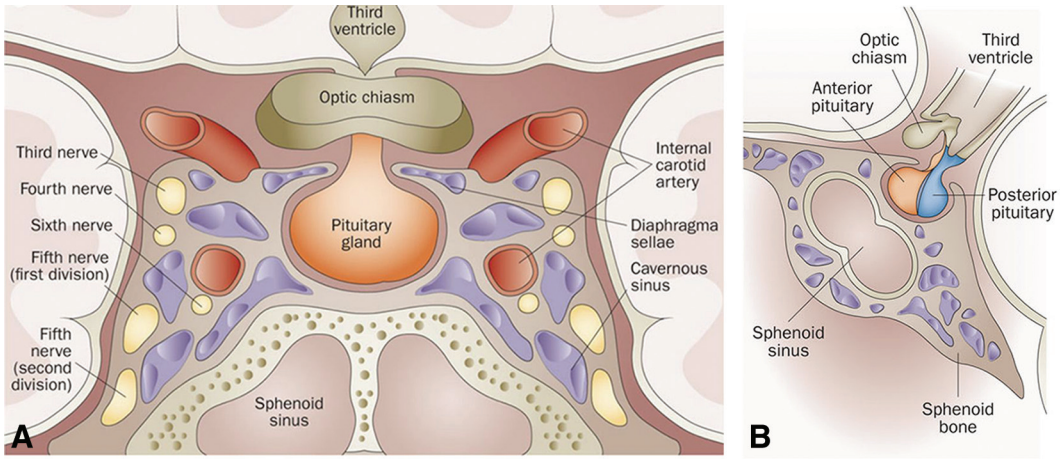

Fig 1. Anatomy of the optic chiasm. Coronal (*A*) and sagittal (*B*) views of the optic chiasm and the surrounding structures. (Reproduced with permission from Di Ieva A, Rotondo F, Syro LV, et al. Aggressive pituitary adenomas–diagnosis and emerging treatments. *Nat Rev Endocrinol* 2014;10:423–35.)

the chiasm. The blood supply of the chiasm can be quite variable. In most cases, the feeder vessels are branches of the anterior communicating artery, anterior cerebral, posterior communicating, posterior cerebral, and basilar arteries. However, there are many collateral vessels, so infarction of the chiasm is extremely rare. In view of the complex anatomic and topographic landmarks adjacent to the chiasm, a wide range of diseases can affect this structure. Scant attention has been paid to the comprehensive classification of the neuroimaging manifestations of optic chiasm abnormalities. In this review, we addressed the neuroimaging findings of lesions that involve the chiasm and classify them according to their etiology.

CONGENITAL AND IATROGENIC LESIONS

Intrinsic Lesions

Ocular Albinism. Albinism is a rare congenital disorder characterized by a defective synthesis of melanin from tyrosine or its complete absence, which results from the mutation of 1 or more associated genes. The consequence of such mutations is a partial or complete absence of pigment in the skin, hair, and eyes. Varying degrees of hypopigmentation occur, owing to different mutation types, which give rise to phenotypic heterogeneity.[2] Ocular albinism, an X-chromosome-linked disorder, results in the reduction or absence of melanin only in the eyes, with the melanocytic system offering normal pigmentation to the rest of the body.[3] It is associated with several visual defects, such as decreased visual acuity, monocular vision and photophobia, foveal hypoplasia, and congenital misrouting of fibers within the chiasm. Quantitative testing of visual evoked potentials usually demonstrates this misrouting at the chiasmatic level. Patients with albinism show an asymmetry of visual evoked potentials between the 2 eyes, secondary to misrouting of the optic pathways. Indeed, achiasma (see *Nondecussating Retinal–Fugal Fiber Syndrome*) has rarely been reported in patients with albinism.[2] MR imaging has a limited potential in assessing this misrouting; however, it has shown that humans with albinism have significantly smaller chiasmatic widths, smaller optic nerves and tracts, and wider angles between nerves and tracts. The size and configuration of the optic chiasm are distinctly different from those in healthy subjects.[4]

Nondecussating Retinal–Fugal Fiber Syndrome. Nondecussating retinal–fugal fiber syndrome, also called achiasma, is extremely rare and may occur with or without nystagmus. The optic chiasm is absent, with normal appearances of the remaining structures of the visual pathway. In achiasma, there is a disruption of retinal-fugal projections, as well as diminished organization and function throughout the visual pathways. Because of the absence of the optic chiasm, all nasal fibers fail to decussate, thereby misprojecting and malaligning with projections of the temporal retina of the same eye. Monocular visual evoked potentials reveal "mirror reversely" interocular ipsilateral asymmetry.[5] Besides nondecussating retinal–fugal fiber syndrome, achiasma or hypochiasmia has been reported in patients with congenital anophthalmos and with midline anomalies, and in patients with albinism.[5] MR imaging shows a complete absence (or partial absence in patients with hypochiasmia) of the optic chiasm, with the optic nerves transitioning into the optic tracts. Functional MR imaging confirms electrophysiologic observations of crossed asymmetry, with each ocular cortex receiving a complete but monocular visual field. Despite these abnormalities, vision is normally preserved because of a reorganization of intracortical connections.[5]

Radiation Necrosis. Radiation necrosis of the optic chiasm is extremely rare; however, it may be caused by radiation therapy for pituitary adenomas or other regional malignant lesions. It may involve normal tissues and may mimic recurrent disease on both the clinical presentation and follow-up imaging studies.[6] Visual loss is the main symptom and, when it occurs in a patient who has undergone surgery for a sellar, parasellar, or skull base tumor with adjuvant radiation therapy, the clinical presentation may be helpful for the

differential diagnosis. Visual loss owing to radiation-induced optic neuropathy, in fact, causes a rapid deterioration in days to weeks, whereas recurrent or progressive tumors or arachnoidal adhesions around the chiasm, which can cause visual symptoms as well, produce more slowly progressive visual impairment. After radiation therapy, peak occurrence of radiation necrosis is between 12 and 18 months, with most cases occurring within 36 months.[6] Contrast-enhanced MR imaging of the orbits, optic nerves, and chiasm is indispensable for the early diagnosis of radiation necrosis, which is not visualized on CT. MR imaging can demonstrate widespread gadolinium-enhanced lesions in the optic chiasm, optic tract, and hypothalamus. A thickening of the chiasm can also be seen.[7] Optic nerve enhancement in a patient with visual loss and a history of radiation therapy to the orbits or optic nerves indicates radiation optic neuropathy.[6]

Extrinsic Lesions

Mucoceles. A mucocele is an accumulation of mucoid secretion through the sphenoid sinus (eg, either congenital, posttraumatic, or idiopathic). Fewer than 3% of all paranasal sinus lesions occur in the sphenoid sinus, and an isolated sphenoid sinus mucocele is extremely rare.[8] Symptoms are nonspecific, which thus results in a delay in diagnosis and may arise from mechanical pressure on neighboring structures and/or the involvement of nerves and the chiasm in the inflammatory process. The most common symptoms include headache, visual loss, and palsies of cranial nerves III and IV.[9-11] However, oculomotor nerve palsy is rare.[12] This is thought to be due to the direct compressive effects of the expansible mucocele on the nerves. Current treatment is by endoscopic sphenoidotomy and drainage of the mucocele.[9,10] Rapidly treated disease usually allows the return of ocular nerve function. However, recurrence is not uncommon, and, therefore, strict follow-up of patients is recommended.[8] Mucoceles may have variable densities on CT as well as intensities on MR imaging, depending on their protein content and possible superinfection.[12] On CT images, they appear as an opacification of the affected sphenoid sinus, with bony expansion, with or without erosion. MR imaging findings include the following: on T1WIs, they are hypointense to hyperintense according to their proportion of water, mucus, and protein (if predominantly water: hypointense; if predominantly protein: hyperintense). On T2WIs, signal intensity is opposite that of T1WIs. Postgadolinium enhancement can be seen at the periphery.

NON NEOPLASTIC LESIONS

Intrinsic Lesions

Choristomas. Choristomas are uncommon cystic lesions composed of adipose tissue and smooth muscle that involve the optic nerve. To date, only a few cases have been reported in the literature. Choristomas can rarely affect the optic chiasm or tracts and the pituitary gland. This lesion is most likely a non neoplastic malformation, and it can cause progressive visual loss.[13] Because of the high adipose tissue content in the lesion, pathologic confirmation is often required for the diagnosis, even though MR imaging may be highly suggestive.[13] On MR imaging, these are well-defined masses, with signal intensity of the tissue contained within the lesion being quite nonspecific; there is vivid enhancement after gadolinium administration. Even if imaging studies are highly suggestive of the diagnosis, then pathologic confirmation is required because of the high adipose tissue content in most cases.

Extrinsic Lesions

Suprasellar Arachnoid Cysts. Suprasellar arachnoid cysts are benign lesions that share all the features of other arachnoid cysts. They are not infrequent and present with signs of compression of the surrounding structures (including the chiasm), which may lead to hypothalamic dysfunction and hydrocephalus.[14] It can be difficult with imaging to differentiate these lesions from other cystic lesions, for example, Rathke cleft cysts,[15] and the diagnosis is often made only at surgery. Treatment involves incision and fenestration of the cysts, with a low risk of recurrence. On MR imaging, they present as cystic lesions with thin walls, no solid component, and no enhancement after gadolinium administration. However, they may be challenging to diagnose when located in the suprasellar region.

Rathke Cleft Cysts. Rathke cleft cysts are benign epithelium-lined intrasellar cysts that contain mucoid material.[16] They are thought to originate from the invagination of the Rathke pouch, which is the precursor of the anterior and posterior lobes of the pituitary gland. Most are sellar lesions but may extend to, or arise from, the suprasellar region. They are twice as common in women than in men and are usually incidentally diagnosed in adults between 40 and 60 years old.[15] Most are asymptomatic and do not enlarge. Occasionally, they become large enough to cause symptoms that include visual loss owing to chiasmal compression, hypopituitarism, and headache.[16] The treatment of choice is the endoscopic transsphenoidal drainage and marsupialization of the cyst.[14] Imaging characteristics are variable, and they are usually included as a differential diagnosis for suprasellar cystic lesions. Unenhanced CT demonstrates a noncalcified cyst with low attenuation, with no enhancement after contrast medium administration, though some sporadic cases of contrast enhancement have been reported. On MR imaging, the signal intensity varies according to the composition of the fluid within the cyst (Fig 2). Cysts with low protein content are isointense with CSF on all sequences; however, as the protein content rises, these cysts become hyperintense on T1 images[15]; on T2, they are usually hyperintense, and there is no enhancement after gadolinium administration; however, it is possible to see a rim of en-

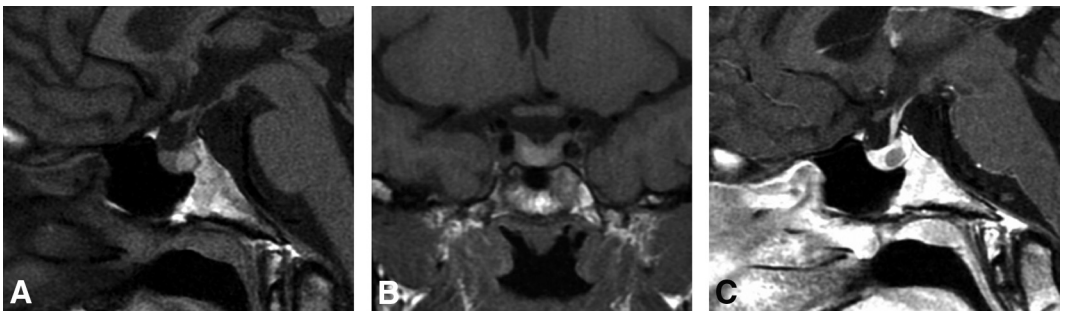

Fig 2. Rathke cleft cyst. Sagittal (*A*) and coronal (*B*) T1WI and sagittal (*C*) postgadolinium T1WI of a Rathke cleft cyst. A 37-year-old woman with headache. The MR imaging demonstrates a T1-hyperintense, nonenhancing lesion in the pituitary gland, consistent with an incidental Rathke cyst.

hancement that represents the pituitary tissue displaced and compressed by the cyst. In contrast with craniopharyngiomas, Rathke cleft cysts do not calcify.

INFLAMMATORY DISEASES

Intrinsic Lesions

Multiple Sclerosis. MS is a chronic inflammatory demyelinating disease of the CNS of unknown etiology that usually affects young and middle-aged adults. MS presents with clinical manifestations, such as impaired sensory and motor functions, and cognitive, urogenital, visual, and mental disorders that may alternate with periods of remission and exacerbation. Acute optic neuritis occurs at some time during the disease in 50% of patients, though it rarely affects the chiasm.[17] Other ocular or visual findings include the following: retinitis, uveitis, peripheral vasculitis, decreased visual function, nystagmus, internuclear ophthalmoplegia, diplopia, optic papillitis, and Marcus Gunn pupil.[18] Optic neuritis, the most important ocular manifestation of MS, is an inflammatory injury of the optic nerves and represents the initial symptom in 20% of patients with the disease. Diagnosis is essentially clinical, though MR imaging, CSF analysis, and evoked potential studies are routinely used for confirmation. MR imaging is the method of choice for the diagnosis because it can show multiple lesions (dissemination in space) and new lesions in follow-up scans (dissemination in time), according to the McDonald criteria for MS and their revisions[19,20] and the Magnetic Resonance Imaging in Multiple Sclerosis consensus guidelines.[21] MR imaging can reveal swelling and enhancement of the optic chiasm in the acute phase; there may be atrophy in patients with long-standing disease. In optic neuritis, the optic nerves appear hyperintense and swollen on T2-weighted sequences, and enhance on T1-weighted sequences after gadolinium administration.

Sarcoidosis. Sarcoidosis is an autoimmune disorder of uncertain etiology in which granulomatous inflammation develops and leads to tissue destruction and fibrosis.[14] It can spread to the CNS through infiltration of meningeal spaces and may involve the anterior visual pathway at any level. Indeed, visual system abnormalities are the most common extrathoracic manifestations of sarcoidosis, with extraocular muscles, retrobulbar space, optic nerves, the chiasm, and optic radiations that may be affected.[22] Granulomas may arise within the disc, and the optic nerve is commonly involved, where an intrinsic lesion may develop and lead to the clinical syndrome of optic neuritis.[23] Compression by an inflammatory mass at the orbital apex or, less frequently, at the cavernous sinus, may occur[24] and can be associated with ophthalmoparesis and trigeminal neuropathy. Corticosteroids are the mainstay of treatment, but additional immunosuppressive therapy is required in some patients.[23] MR imaging is the technique of choice and gadolinium-enhanced T1-weighted sequences are the most important (Fig 3). Imaging features are variable according to the different locations of involvement. Invasion of the leptomeninges is seen as contrast enhancement, the dura mater can be thickened, and involvement of the parenchyma can mimic a pseudotumor lesion. In many patients, some combination of neural optic or perineural enhancement is noted after gadolinium administration.[22] The coexistence of multiple locations is suggestive of sarcoidosis.

Idiopathic Optic Chiasmitis. Chiasmal neuritis is a clinical syndrome that consists of acute visual loss with a chiasmal visual field pattern and/or radiographic demonstration of chiasmal inflammation.[25] Occasional cases of chiasmal neuritis are due to a systemic inflammatory disease, such as tuberculosis (TB), sarcoidosis, systemic lupus erythematosus, Epstein-Barr disease, or Lyme disease. It is rare, often diagnosed only after having ruled out other pathologies. However, more commonly, chiasmal disease occurs as an idiopathic event. In such cases, it is a rare disorder whose clinical profile is not well characterized, even if, most commonly, patients present with painless monocular visual loss or bitemporal hemianopia.[25] Treatment includes oral and/or IV corticosteroids. MR imaging reveals chiasm swelling in most cases, with or without enhancement, and with lesions in the white matter as well.

Extrinsic Lesions

Antineutrophil Cytoplasmic Antibody Positive Vasculitis. Antineutrophil cytoplasmic antibody positive vasculitis is a granulomatous inflammatory disorder with features of venular and arteriolar perivasculitis, and it affects the lungs,

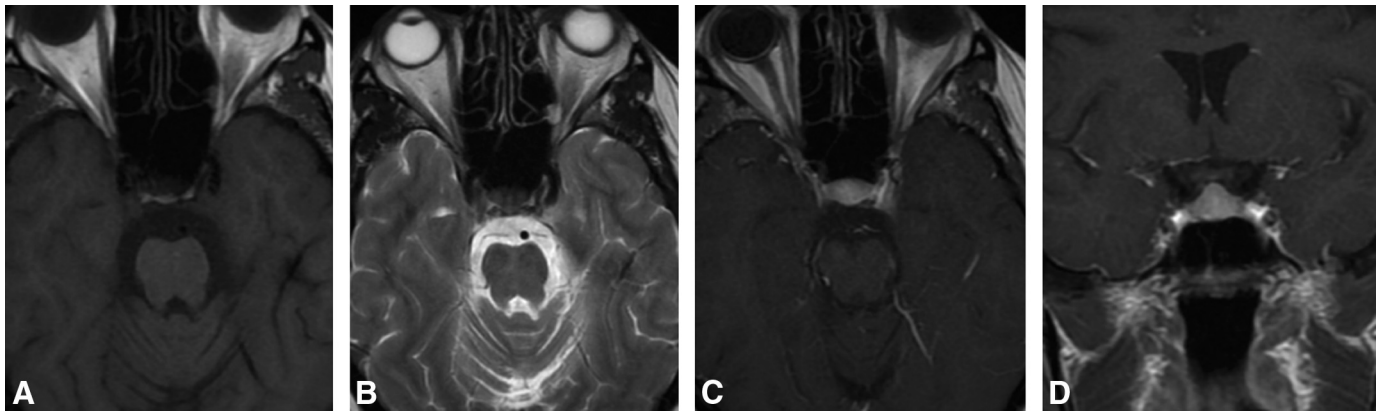

Fig 3. Sarcoid hypophysitis. Axial T1WI (*A*) and T2WI (*B*) and postgadolinium axial (*C*) and coronal T1WI (*D*) of a patient with hypophysitis. A 49-year-old woman with a history of progressive dizziness. MR imaging demonstrates an enlargement of the pituitary, with upward bulging into the suprasellar cistern, touching the optic chiasm; appearances may be the result of hypophysitis due to involvement by sarcoidosis.

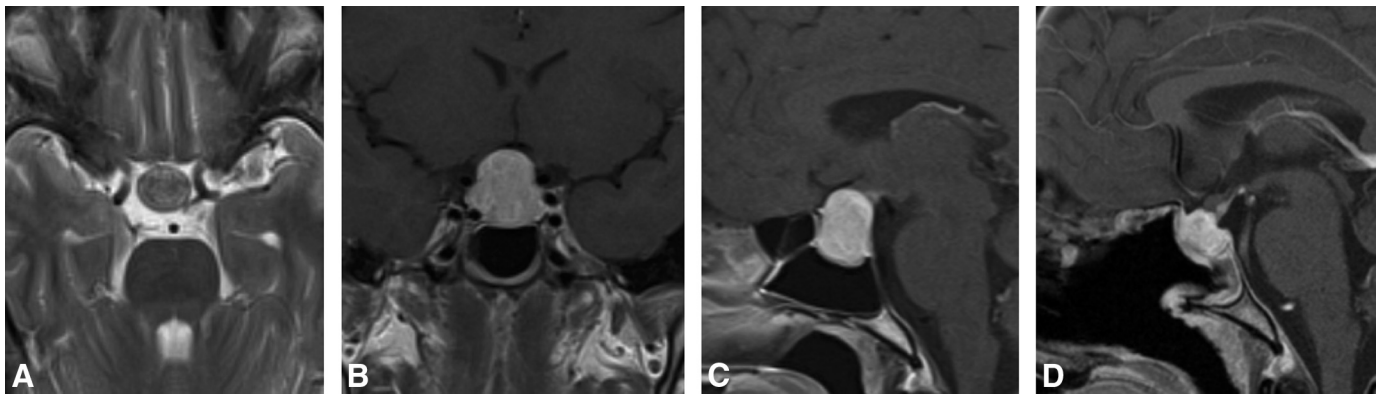

Fig 4. Lymphocytic hypophysitis. Axial T2WI (*A*) and coronal (*B*) and sagittal (*C* and *D*) postgadolinium T1WI of a patient with lymphocytic hypophysitis. A 31-year-old postpartum woman with headache, fatigue, and vision loss. Axial T2 (*A*), coronal T1 (*B*), and sagittal T1 postcontrast (*C*) images demonstrate a large sellar-suprasellar mass, with superior displacement of the optic chiasm—features that mimic a pituitary macroadenoma. During surgery, the appearance of the gland was inflammatory and a biopsy was obtained, which demonstrated attenuated infiltrate of the B and T lymphocytes, consistent with the diagnosis of lymphocytic hypophysitis. Sagittal T1 postgadolinium image (*D*) obtained as part of follow-up MR imaging 3 weeks after starting steroid treatment demonstrates an interval decrease in size of the sellar-suprasellar mass, as well as postsurgical changes related to the trans-sphenoidal approach.

skin, eyes, and kidneys. Included in this disease group are granulomatosis with polyangiitis, formerly known as Wegener granulomatosis, microscopic polyangiitis, and eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome) that involve small vessels (both arteries and veins). Neurologic involvement arises as a hypertrophic pachymeningitis, as an isolated inflammatory mass, or as a central or peripheral manifestation of inflammatory perivasculitis.[26] In Wegener granulomatosis, for example, it is thought that granulomatous tissue may spread out from the nasal or paranasal cavities, which are mainly involved, and invade the adjacent structures, such as the orbit, optic nerves, chiasm, and/or pituitary gland.[26] MR imaging can detect an enlarged and gadolinium-enhancing pituitary gland, a thickening of the pituitary stalk and, inconstantly, the loss of the normal posterior lobe hyperintensity on T1-weighted sequences.[26]

Lymphocytic Hypophysitis. Lymphocytic hypophysitis is a rare autoimmune disease of the pituitary gland, which can present with varying grades of pituitary hormonal impairment and/or with symptoms related to pituitary enlargement.[27] It affects women more frequently than men, with a reported ratio of 5:1, and occurs in the later stages of pregnancy or early postpartum.[27] Some patients in the early stages of the disease may present with clinical signs and symptoms due to pituitary enlargement, with possible extrasellar extension, including headache, visual field impairment, and, more rarely, diplopia, with or without hypopituitarism.[27] Hyperprolactinemia is a usual finding, even though sometimes prolactin levels can be normal or even reduced.[28] Anti-inflammatory and/or immunosuppressive drugs can be effective to reduce the size of the pituitary mass. In patients with lymphocytic hypophysitis and with signs and symptoms related to pituitary enlargement, MR imaging is particularly important to differentiate this disorder from adenoma, even if imaging findings sometimes tend to overlap (Fig 4). Patients with lymphocytic hypophysitis usually have pituitary enlargement, with a symmetric suprasellar extension, which can displace the optic chiasm, whereas patients with adenoma have asymmetric pituitary

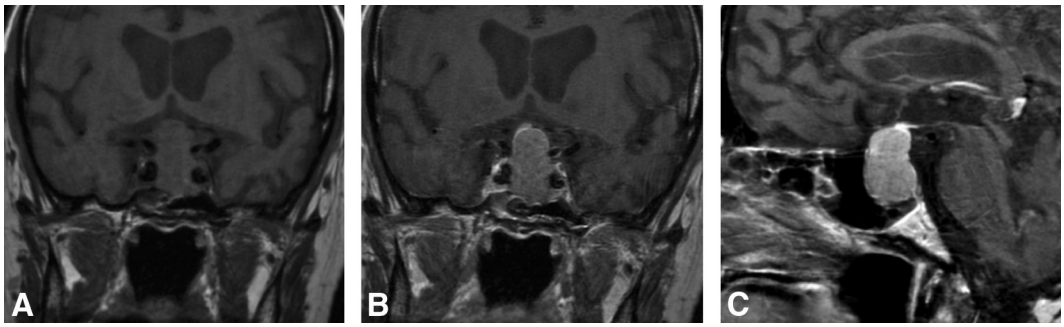

Fig 5. Pituitary macroadenoma. Coronal (*A* and *B*) and sagittal (*C*) T1WIs of a pituitary macroadenoma before (*A*) and after (*B* and *C*) gadolinium administration. An 85-year-old man with a history of bitemporal hemianopia. Images demonstrate a homogeneously enhancing anterior pituitary mass with suprasellar extension, with superior deviation of the optic chiasm. The pituitary infundibulum is posteriorly displaced.

enlargement with a deviation in the stalk, which is thickened but usually not deviated in lymphocytic hypophysitis. After gadolinium administration, pituitary enhancement is homogeneous in lymphocytic hypophysitis, with a rim of enhancing tissue along the dura mater, the so-called dural tail. However, adenomas have delayed and poor enhancement, usually without the dural tail.[27]

Xanthomatous Hypophysitis. This is a rare inflammatory disorder of the pituitary gland owing to lymphocytic and histiocytic infiltration. Some investigators believe this to be an incomplete form of Erdheim– Chester disease, in which a histiocytic infiltration of the bones, lungs, and numerous other tissues may arise.[29] Neurologic complications are mostly due to the involvement of the orbits, optic nerves, and chiasm, but other areas, such as the skin, heart, kidney, and skeletal muscle, may be affected as well.[30] Imaging shows a cystic lesion within the pituitary gland, which predominantly contains fluid.

Benign Neoplastic Extrinsic Lesions

Pituitary Adenomas. Pituitary adenomas are the most common type of pituitary disorder. They are benign neoplasms that account for 10%–15% of all intracranial masses.[31] The incidence of pituitary adenomas within the general population is as high as 16.7%.[32] They typically arise from the epithelial cells of the anterior part of the gland and are classified according to their size: microadenomas are defined as neoplasms <1 cm contained in the sella turcica, whereas macroadenomas (Fig 5) are neoplasms ≥1 cm that may be contained in the sella turcica but often extend to the superior, inferior, or lateral extrasellar space. Furthermore, pituitary adenomas may also be categorized as functional or nonfunctional; functional lesions present with clinical symptoms due to increased hormonal secretion. Nonfunctional adenomas, however, usually present with mass effect and are often incidental findings.[33] Prolactinomas comprise 40%–57% of all pituitary adenomas, followed by nonfunctioning adenomas (28%–37%), growth hormone-secreting adenomas (11%–13%), and adrenocorticotropic hormone-secreting adenomas (1%–2%).[31]

Pituitary adenomas that secrete follicle-stimulating hormone, luteinizing hormone, or thyroid-stimulating hormone are rare.[34] Pituitary adenomas may present clinically in 3 different ways: syndromes of hormone hypersecretion or deficiency, neurologic manifestations due to mass effect, or incidental findings on imaging performed for an unrelated issue. They are the most common extrinsic lesions responsible for chiasmal syndromes.[31] Prolactinomas usually present with galactorrhea and amenorrhea; adrenocorticotropic hormone– and growth hormone–secreting tumors produce Cushing disease and acromegaly, respectively. Headache may be the only prominent feature in up to 34% of patients.[35] The main treatment goals are to reduce hormone hypersecretion and its clinical manifestation, decrease tumor size to improve symptoms due to mass effect, and correct hormone deficiency. Most prolactinomas can be managed medically with dopamine agonists (bromocriptine and cabergoline), whereas medical management of growth hormone– and adrenocorticotropic hormone–secreting tumors is less effective than for prolactinomas, and surgical resection through a trans-sphenoidal approach is the treatment of choice. Nonfunctioning microadenomas and macroprolactinomas in patients who are asymptomatic do not require immediate treatment and may be observed with follow-up MR imaging.

MR imaging is the mainstay of imaging for both micro- and macroadenomas (Fig 5), and dedicated pituitary sequences (thin sections, small field of view) are used. Contrast-enhanced MRIs have a sensitivity of 90%.[36] On unenhanced images, some features include asymmetric bulkiness of the gland and remodeling of the floor of the sella, and deviation of the infundibulum away from the lesion can suggest the presence of an adenoma.[36] Postgadolinium images typically show an area of delayed enhancement compared with the rest of the gland, which enhances vividly. Dynamic acquisition may help in identifying a small area of delayed enhancement as per microadenomas. Macroadenomas may behave differently due to areas of cystic changes, necrosis, or hemorrhage. In particular, the solid components may demonstrate vivid enhancement similar to the normal gland.

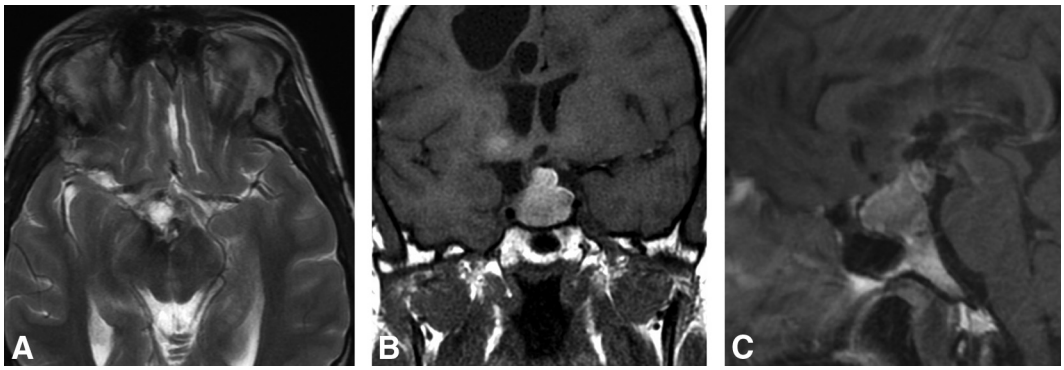

Fig 6. Craniopharyngioma. Axial T2WI (*A*) and coronal (*B*) and sagittal (*C*) postgadolinium T1WI, showing a craniopharyngioma. A 30-year-old man with a history of craniopharyngioma. Images show a sellar-suprasellar lesion displacing the optic chiasm and the infundibulum.

Craniopharyngiomas. Craniopharyngiomas account for approximately 3% of all primary intracranial tumors.[37] They are benign lesions (World Health Organization grade I) that arise from the squamous epithelium at the junction between the infundibulum and the anterior part of the gland; according to some investigators, these are remnants of the Rathke pouch.[14] There is a bimodal incidence, in childhood and again in middle-to-late life, with clinical features of a sellar or hypothalamic lesion. They usually lie above or below the chiasm and are classified as adamantinomatous, papillary, or mixed, with the first being the most common.[38] The lesion is usually cystic, with a viscid oily fluid (referred to as "engine oil"). It is possible to also demonstrate cholesterol clefts, calcium, and keratin within the lesion. The most common symptoms are visual loss, visual field deficits, and hypothalamic impairment. The surgical approach depends on the size and the extent of the tumor; some lesions can be accessed via a trans-sphenoidal approach, whereas others require a craniotomy. Postoperative radiation therapy is especially useful in the case of incomplete resection and is associated with tumor regression or lack of recurrence.[37] CT can demonstrate lesions with different characteristics according to their main components. Cystic lesions have attenuation similar to CSF[39]; the solid component shows a soft-tissue attenuation with vivid enhancement, and calcification can be seen in 90% of patients, the adamantinomatous type. Of note, calcification and cystic changes are not usually seen in the papillary type. At MR imaging (Fig 6), the cystic component is iso- to hyperintense to brain parenchyma on T1WI, with high signal intensity on T2WI. The solid component exhibits vivid enhancement after gadolinium administration, and calcification can be seen with gradient-echo images or SWIs.

Dermoid and Epidermoid Cysts. Dermoid and epidermoid cysts are dysembryogenic tumors that represent 1% of all intracranial tumors.[40] They are characterized by slow growth and a benign course. Dermoid cysts are rare benign lesions of the CNS that result from the sequestration of totipotent cutaneous ectodermal cells into the developing neural tube during neurulation.[40,41] Unlike their slightly more common epidermoid counterparts, which are often laterally placed, dermoid cysts tend to occur in the midline.[14,40] Both dermoid and epidermoid cysts can develop anywhere in the CNS; however, in the suprasellar region, they usually present in the same way as a pituitary adenoma or a craniopharyngioma, with visual loss due to optic nerves and chiasm compression and hypothalamic dysfunction. Despite the benign nature of dermoid cysts, their rupture is associated with chemical meningitis and seizures.[14] Dermoids are hyperintense on T1WI and T2WI, which may be secondary to intralesional fat (Fig 7). Epidermoids typically have fluid signal intensity on both T1-weighted and T2-weighted sequences, but they show restricted diffusion.

Meningiomas. Meningiomas are benign lesions that arise from the arachnoid cap cells.[42] They are twice as common in women as in men and arise more frequently in the second half of life.[14] They tend to be benign and slow-growing, but some may show an infiltrative behavior with respect to the surrounding tissues. Because of their locations, meningiomas of the clivus, sphenoid wings, and the olfactory groove and optic nerve sheath can involve the chiasm,[43] and primary optic nerve sheath meningiomas may grow backward to involve the chiasm.[42] Tuberculum sellae meningioma is a distinct clinical entity that represents 5%–10% of all intracranial meningiomas.[14,44] Tuberculum sellae meningiomas displace the optic pathways upward and laterally, and thus occupy a subchiasmal location. Therefore, they must be distinguished from the olfactory groove, sphenoid planum, and anterior clinoid process lesions, which are often considered suprasellar tumors.[44] They may present with progressive loss of vision and visual field defects. Treatment is surgical, with the transcranial approach still considered the criterion standard for suprasellar tumors. Despite this, the extended endonasal trans-sphenoidal route has recently been developed for some select cases (small tumors, no lateral extension, no vascular encasement).[44] On unenhanced CT, meningiomas are usually hyperattenuated compared with the normal brain parenchyma and show some calcification; the enhancement is typically avid. CT can easily highlight the hyperostosis in the underlying bone, which is quite characteristic. At MR imaging (Fig 8), the lesion is

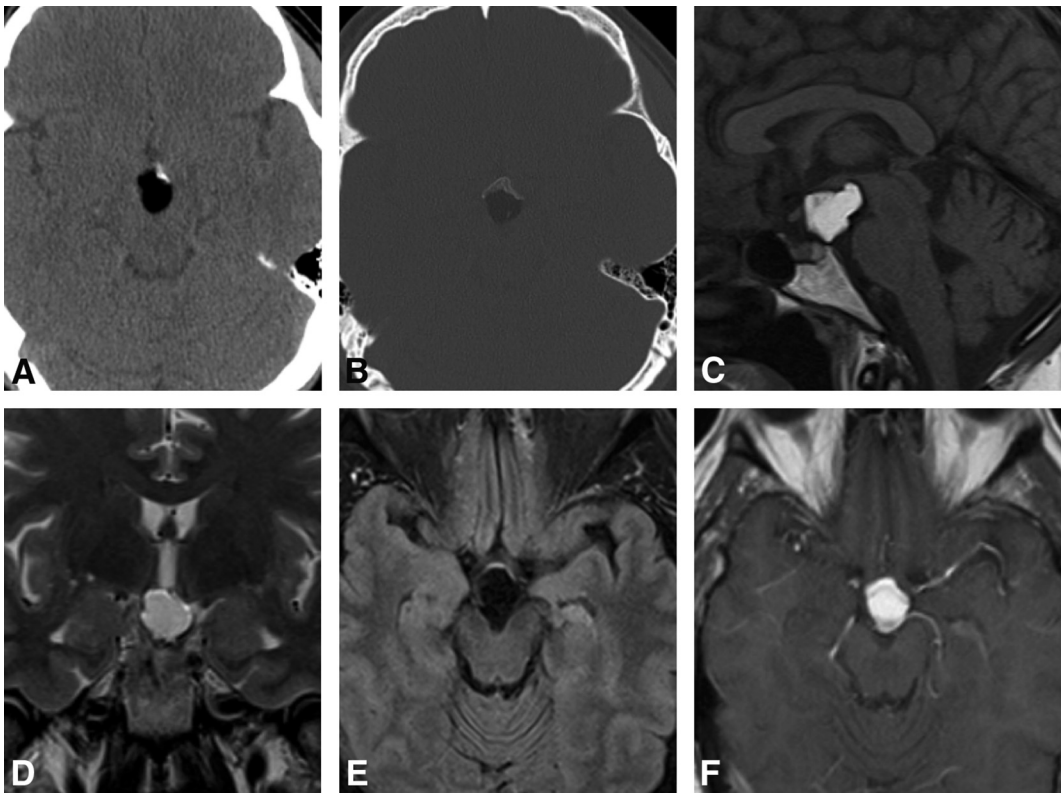

Fig 7. Dermoid cyst. Axial soft-tissue (*A*) and bone (*B*) algorithm CT reconstructions; sagittal T1-weighted (*C*), coronal T2-weighted (*D*), axial fat-suppressed T2W FLAIR (*E*), and postgadolinium T1-weighted (*F*) images of a dermoid cyst. A 35-year-old patient with an incidental finding of a pituitary infundibular lesion on a CT of the head performed for trauma. CT of the head in soft-tissue (*A*) and bone algorithm (*B*) reconstructions demonstrates a hypoattenuated midline lesion centered in the suprasellar cistern, with associated anterior calcifications. The lesion demonstrates fat attenuation. MR imaging of the brain was then obtained, which demonstrated the lesion to be centered in the suprasellar cistern along the infundibulum and demonstrated hyperintense signal intensity on T1WI (*C*) and T2WI (*D*), hypointense signal intensity on fat-suppressed T2 FLAIR (*E*), and no contrast enhancement after gadolinium administration (*F*). Note the chemical shift artifact along the frequency-encoded direction.

isointense to gray matter in most patients, both in T1WI and in T2WI. As on CT, the postgadolinium administration enhancement is quite vivid, and it is possible to appreciate a dural tail, thickening, and enhancement of the dura adjacent to the lesion, which, although could also be seen in other pathologies, is quite suggestive of meningiomas.

MALIGNANT NEOPLASTIC LESIONS

Intrinsic Lesions

Gliomas. Optic nerve gliomas represent approximately 2% of all brain tumors.[45] These tumors are categorized into benign optic gliomas (pilocytic astrocytomas, neurofibromatosis type 1) that typically occur in childhood and malignant optic gliomas that occur in adulthood. Malignant optic and chiasmatic gliomas are extremely rare and are classified pathologically as anaplastic astrocytomas and glioblastomas (GBMs).[46] Approximately 40 cases of anterior optic pathway GBM in adults have been reported in the literature, and only 5 of them were described to originate from the optic chiasm.[46] GBMs may involve the chiasm, either primarily or by infiltration from the adjacent structures. A peculiar subset of GBM is hypothalamic-optochiasmatic GBM, which is associated with neurofibromatosis type 1 and involves the optic nerves, chiasm, and tracts. Early symptoms include blurred vision and progressive loss of bitemporal visual fields; headache and periorbital pain may also occur.[47] Standard treatment for GBM is combined chemoradiation therapy in most patients. Because optic GBM infiltrates extensively and grows rapidly, the prognosis is unfavorable. Reported survival ranges from 6 to 14 months after diagnosis.[46] Dinh et al[47] published a case of chiasmatic GBM, with the patient surviving 14 months after the onset of the symptoms, which is the longest survival of such patients in the literature. MR imaging can show the lesion with subsequent enlargement of the chiasm, as clearly shown in Fig 9. GBMs are usually hypointense on T1WI and hyperintense on T2WI and FLAIR, with enhancement of the solid component after gadolinium administration.

Extrinsic Lesions

Chordomas. Chordomas are intracranial tumors, especially in children, that originate from the remnants of primary notochord and account for <1% of all intracranial tumors.[48] They arise most commonly in the clivus, but extension upward may lead to optic tracts or chiasmal involvement and hypothalamic dysfunction.[14] They are slow-

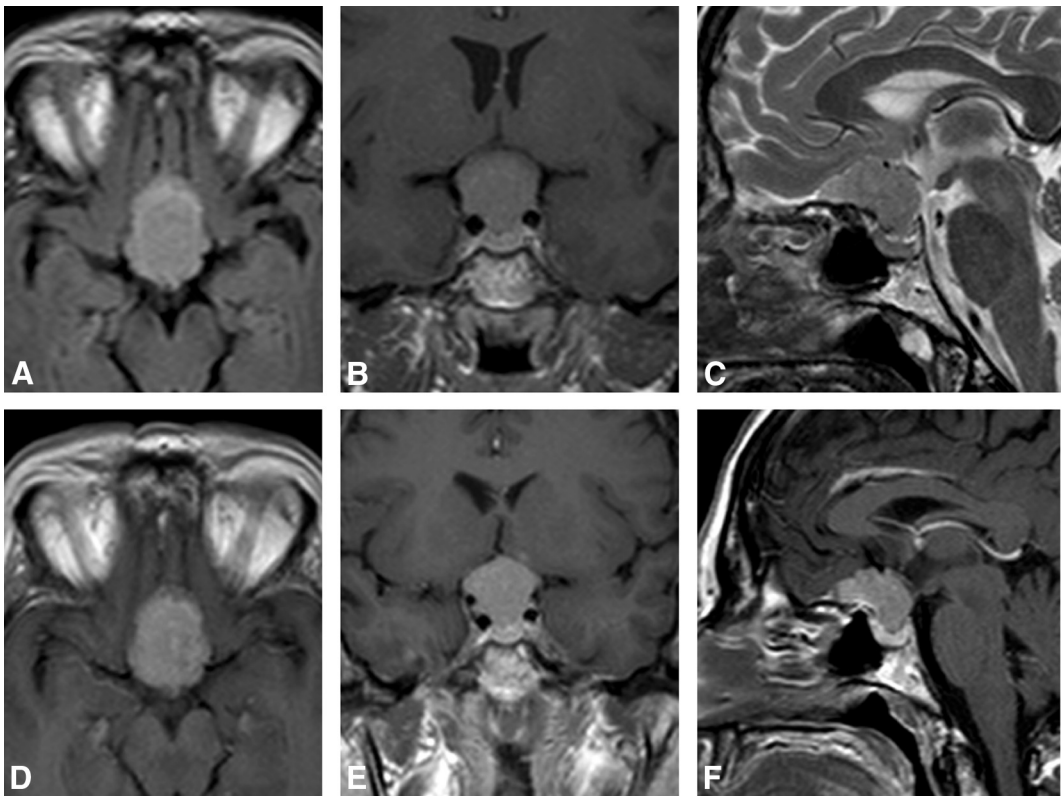

Fig 8. Planum sphenoidale meningioma. Axial T2 FLAIR (*A*); coronal T1WI (*B*); sagittal T2WI (*C*); and axial (*D*), coronal (*E*), and sagittal (*F*) postgadolinium T1WI of a planum sphenoidale meningioma. A 37-year-old man with headache and bitemporal hemianopia. The MR imaging shows a lesion centered on the planum sphenoidale, with compression of the pituitary gland and displacement of the optic chiasm.

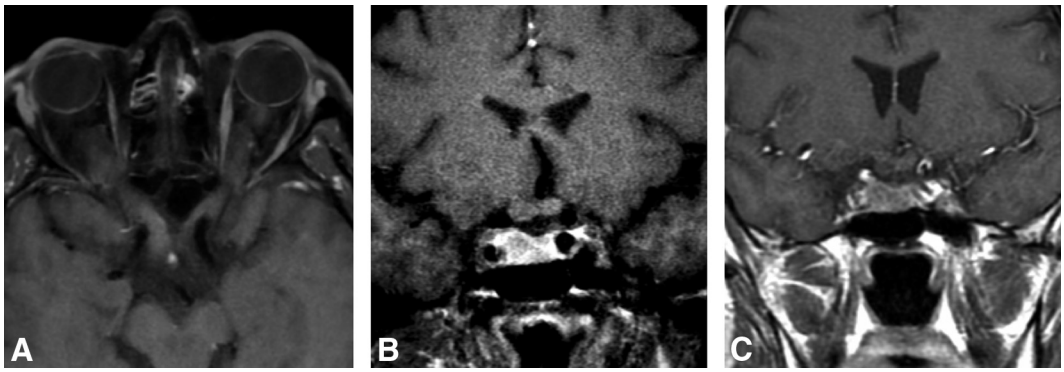

Fig 9. Bilateral optic nerve glioma. Postgadolinium axial (*A*) and coronal (*B*) T1WIs with fat saturation, and coronal (*C*) postgadolinium T1WI in a patient with a bilateral optic nerve and chiasm glioma. A 53-year-old man with a history of vision deterioration. MR imaging demonstrates a bilateral optic nerve glioma with retro-orbital extension and involvement of the optic chiasm.

growing, locally aggressive tumors. The treatment of choice is surgery, and endoscopic techniques in skull base surgery have provided higher rates of tumor removal.[48] Postoperative radiation therapy is useful, though high doses are required. CT may show a soft-tissue mass with bone destruction and, in 50% of patients, ossification. MR imaging exceeds CT in both identifying tumor expansion and the relationship to adjacent structures (Fig 10). On T1WI, signal intensity is variable according to the expansion of the tumor into the bone marrow, which leads to hyperintense signal intensity. On T2WI, lesions are hyperintense as well, with hypointense stripes representing septa of fibrous connective tissue. Contrast enhancement is significant and inhomogeneous both on CT and MR imaging.

Germinomas. Germinomas are rare tumors, predominantly seen in early adult life. Germinomas are also known as dysgerminomas or extragonadal seminomas and tend to arise mostly in the pineal region but may also be seen in the suprasellar region, at the level of the floor of the third ventricle, or from the optic chiasm.[49] Germinomas are locally invasive and can recur or metastasize. Those that arise within the sellar region present with visual disturbances and hypothalamic dysfunction, in particular, diabetes in-

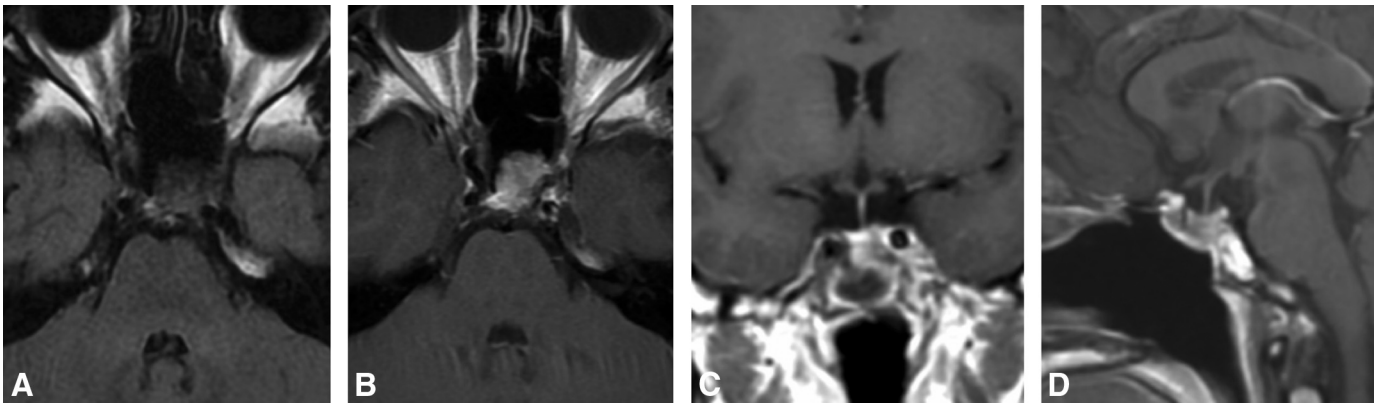

Fig 10. Chordoma. Axial unenhanced T1WI (*A*); and axial (*B*), coronal (*C*), and sagittal (*D*) postgadolinium T1WI of a chordoma. A 19-year-old man with a history of hypothalamic pituitary dysfunction and growth hormone deficiency. MR imaging demonstrates a lesion into the pituitary sellae, with no contact with the optic chiasm.

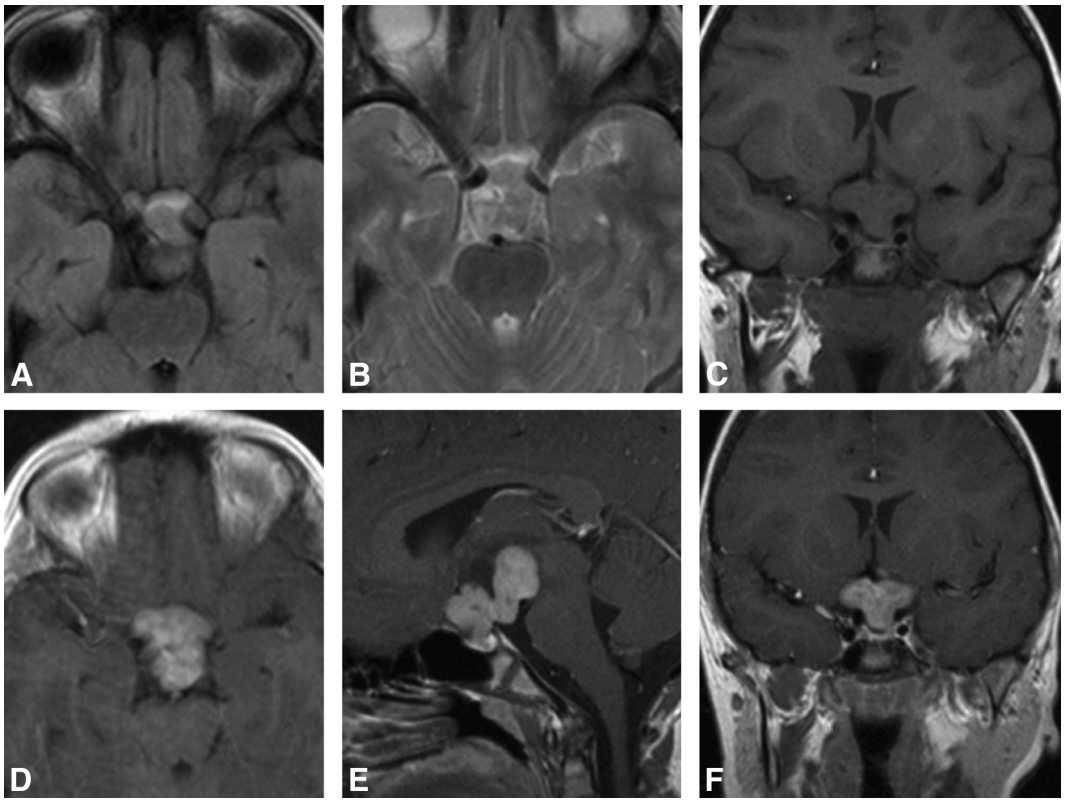

Fig 11. Germinoma. Axial FLAIR (*A*) and T2WI (*B*); coronal T1WI (*C*); and postgadolinium axial (*D*), sagittal (*E*), and coronal (*F*) T1WIs of a germinoma. An 11-year-old child with a history of polyuria, polydipsia, and vision loss. MR imaging demonstrates a suprasellar mass with a lobulated appearance that enhances homogeneously. The optic chiasm is difficult to separate from the mass.

sipidus.[49] Surgical treatment includes biopsy, followed by radiation therapy. CT shows hyperattenuated tissue compared with adjacent normal parenchyma; there is avid enhancement after contrast medium administration. MR imaging (Fig 11) shows a soft-tissue mass that is isointense or slightly hyperintense on T1WI and T2WI compared with the normal adjacent brain. The peritumoral region may show areas of edema or hemorrhage. As on CT, there is avid enhancement after gadolinium administration.

Other Germ Cell Tumors. Although rare, teratoma, embryonal cell carcinoma, yolk sac tumors, and choriocarcinoma can all arise in the optic nerves and chiasm. Intracranial germ cell tumors are often of mixed histologic composition, and only teratomas and germinomas are likely to be pure tumor types. It is thought that these tumors are all associated with chromosomal defects.[50] Some teratomas are benign, while others can be locally invasive. The prognosis is good for benign tumors that are completely removed; embryonal cell carcinoma, however, is a highly malignant tu-

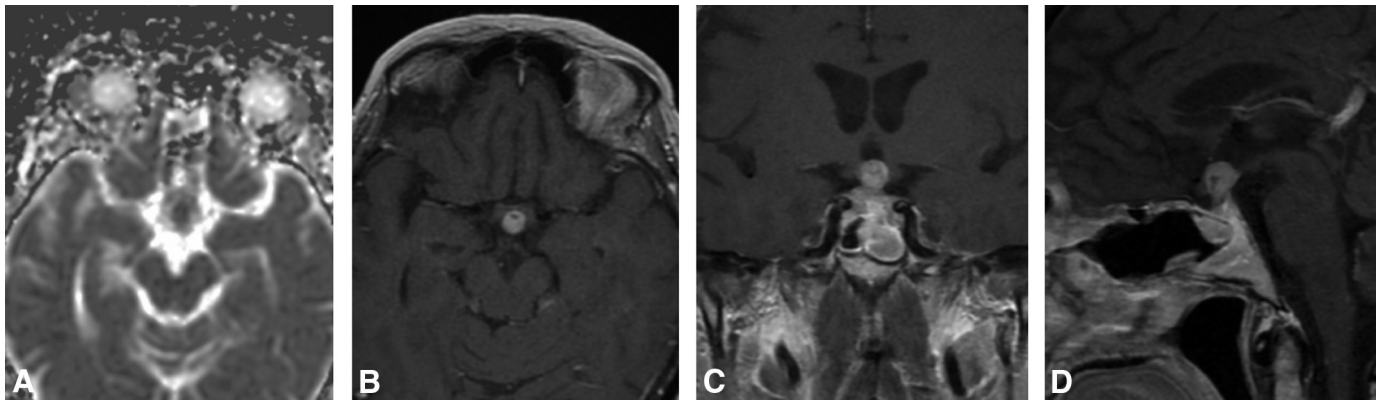

Fig 12. Hemangiopericytoma. Axial ADC (*A*), and axial (*B*), coronal (*C*), and sagittal (*D*) postgadolinium T1WI of a hemangiopericytoma. A 74-year-old woman with a history of bitemporal hemianopia. MR imaging demonstrates an intrasellar hemangiopericytoma, with compression of the optic chiasm and extension into the sphenoid sinus and cavernous sinus.

mor with a poor response to treatment. Yolk sac tumors may also arise in the midline similar to other germ cell tumors and often share pathologic features with choriocarcinoma and embryonal carcinoma. Their prognosis is poor despite surgery and chemoradiation. Choriocarcinoma is rare as an isolated intracranial mass.[14] These tumors have no definite specific characteristics, but teratomas on CT can show fat and calcification, as well as cystic and solid components. On MR imaging, teratomas are quite inhomogeneous due to their different components; indeed, lesions can show hyperintense T1WI areas owing to fat or proteinaceous fluid, intermediate signal intensity owing to soft tissue, and hypointense signal intensity owing to calcification and blood products. The solid tissue components may enhance after gadolinium administration.

Hemangiopericytomas. Hemangiopericytoma is a rare vascular neoplasm that originates in the pericytes in the walls of capillaries. Intracranial hemangiopericytomas are aggressive in nature and can originate in the sellae. The treatment is surgery, often supplemented by radiation therapy. At MR imaging (Fig 12), they can mimic an adenoma and can be misdiagnosed. On postgadolinium images, hemangiopericytomas show rather homogeneous enhancement, though an area of necrosis could be present at the core.

Metastases. Metastases in the pituitary gland are rare and, unless a metastatic systemic disease is already known, they are often preoperatively misdiagnosed as pituitary adenomas. The most common types of malignant tumors seen in the sellar and parasellar regions are breast and lung metastases in women and men, respectively.[51] Despite this, almost every primary cancer could metastasize to those regions (though quite rarely). Clinical presentation may include hormonal dysfunction, such as diabetes insipidus and panhypopituitarism, and symptoms owing to chiasmatic compression. There are no specific criteria to label a lesion in this area as a metastasis; however, metastases exhibit the same characteristics of deposits seen elsewhere in the brain and characteristic patterns of enhancement.

INFECTIVE DISEASES

Intrinsic Lesions

Tuberculosis. TB generally affects the lungs but can spread throughout the body and infiltrate the sellar region as well. The 2 most common presentations in this region are within the pituitary gland (tuberculoma) or optochiasmal arachnoiditis; both can cause visual symptoms and pituitary hypofunction due to chiasmal compression.[52] Tuberculoma masses are difficult to distinguish from other pituitary lesions,[53] even though the thickening of the pituitary stalk seems to be more common in TB than elsewhere. MR imaging can demonstrate a mass lesion within the pituitary gland; however, the mass is indistinguishable from other primary pituitary mass lesions. When TB presents as optochiasmal arachnoiditis, perichiasmal enhancement can be observed after gadolinium administration.[54]

Viruses. Isolated chiasmitis associated with viral infections is rare and has been reported with varicella-zoster virus and Epstein-Barr virus, especially in patients with acquired immunodeficiency syndrome (AIDS) and who are immunocompromised.[55,56] Clinical symptoms are decreased visual acuity and visual field defects. Treatment is by IV administration of antiviral drugs.

Extrinsic Lesions

Pituitary Abscesses. Although extremely rare, pituitary abscesses are considered a life-threatening disease. They account for <1% of all pituitary lesions and have the same frequency in both men and women.[57,58] Often misdiagnosed as tumors, the origin of these abscesses is unclear. Their management is mainly surgical. They may occur de novo or can originate from hematogenous spread or spread from a contiguous focus of infection, such as meningitis or

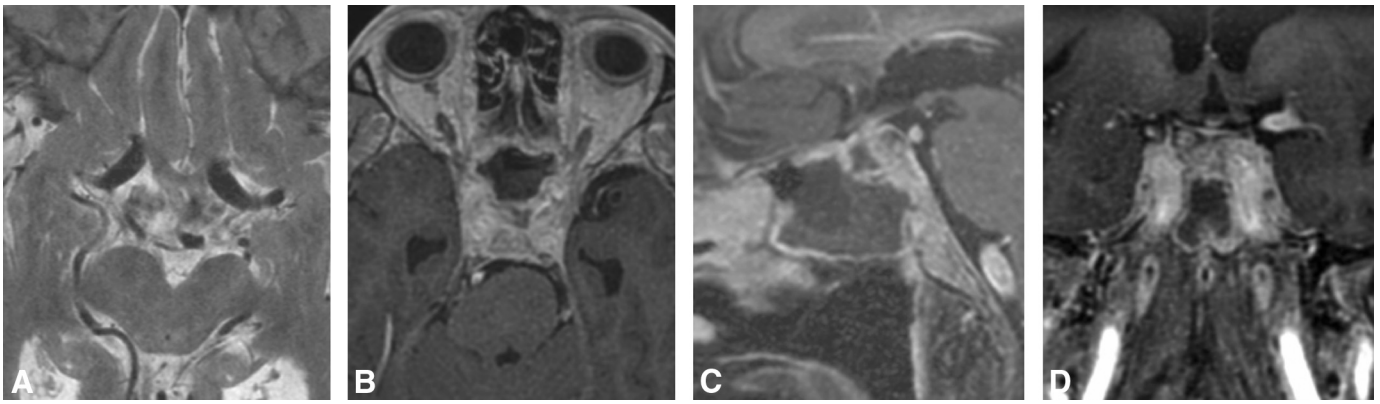

Fig 13. Aspergillosis. Axial T2WI (*A*), and axial (*B*), sagittal (*C*), and coronal (*D*) postgadolinium T1WI in a patient with aspergillosis. A 78-year-old man with a history of severe sinusitis and II, III, VI nerve palsies after bilateral sphenoidotomies for drainage and washout of a mucopyocele in the sphenoid sinuses. MR imaging demonstrates a defect in the anterior wall of the sellae (*C*), through which the postoperative fluid collection in the sphenoid sinus is continuous with a small pocket of fluid either within or compressing the pituitary gland. The optic chiasm shows a tiny area of enhancement in its inferior aspect (*C* and *D*).

sphenoiditis. Clinical symptoms include headache (70%–92%); anterior pituitary dysfunction (54%–85%); central diabetes insipidus (41%–70%); visual disorders due to optic nerves and chiasm involvement (27%–50%); slight or moderate fever (14%–33%); fever with signs of meningeal irritation (25%); and nonspecific symptoms of systemic infection, for example, dizziness (10%).[59] The most commonly isolated pathogens are *Staphylococcus* species and *Streptococcus* species, followed by *Neisseria* species, *Micrococcus, Citrobacter* species, *Escherichia coli, Brucella, Salmonella, Corynebacterium,* and *Mycobacterium.*[60] MR imaging is the examination of choice. A sellar or suprasellar mass is often demonstrated with hypointense signal intensity on T1WI and hyperintense signal intensity on T2WI. Restricted diffusion at DWI may be seen as common for intracranial abscesses. After gadolinium administration, there is ring enhancement, which can mimic the enhancement pattern seen in tumors.

Fungal Infections. Cryptococcosis is the most common opportunistic fungal infection of the CNS in patients with HIV infection and/or AIDS.[61] *Cryptococcus neoformans*, in fact, is a ubiquitous and encapsulated yeast that can affect patients with severely compromised cell-mediated immunity. Apart from patients with AIDS, it can affect, though more rarely, other patients who are compromised, as well as those with prolonged corticosteroid therapy, chemotherapy, and lymphomas.[62] The spread of cryptococcosis to the meninges from the sinuses is life-threatening. The most common clinical form of neurocryptococcosis is meningoencephalitis,[63] a diffuse brain basal meningitis associated with a high rate of morbidity and mortality in patients with AIDS that presents clinically with fever and headache, and less frequently with confusion, seizure, and meningeal signs. Corti et al[63] also reported a case of sudden blindness due to invasion and infection of the optic nerves and chiasm by the fungus in a patient with AIDS. The acute visual loss in this patient was linked to the presence of cryptococcal organisms throughout the basal meninges and the sheaths of the optic nerves and the chiasm.[63]

Kestelyn et al[64] observed papilledema as the most common clinical manifestation of cryptococcal infection in 32.5% of 80 patients with AIDS, but also observed visual loss, ocular cranial nerve palsy (9%), and optic nerve atrophy (2.5%). In most cases, treatment involves antifungal therapy, along with management of raised intracranial pressure. In response to the host immune system attacks on the organism, the cryptococci produce a mucoid material that may enlarge the perivascular spaces of Virchow-Robin, and thus lead to the formation of the cryptococcomas composed of organisms, mucoid material, and inflammatory cells.[65] Involvement of the pituitary gland from other fungi is uncommon; however, there have been a few cases of aspergillosis involvement due to sphenoidal sinusitis (Fig 13). MR imaging demonstrates a sellar or suprasellar lesion that mimics a pituitary adenoma. MR imaging can also demonstrate punctate hyperintensities that correspond with dilated perivascular spaces and cryptococcomas.[65] Both MR imaging and CT tend to underestimate the real extension of the disease.

Cysticercosis. Cases of sellar neurocysticercosis (NCC) are rare. So far, little is known about the mechanisms through which the parasite can attack the optic nerves and pituitary gland, though they do not differ from the mechanisms proposed for NCC in other areas of the CNS. Therefore, the mechanisms of sellar involvement range from direct through indirect invasion to a severe local inflammatory response.[66] Direct invasion of the sella turcica by the NCC vesicles that affects the hypophysis is the most common described form reported in the literature[67,68] and is often confused with other more common lesions of the area (eg, pituitary adenomas and craniopharyngiomas). Clinical symptoms include visual field deficits and endocrine dysfunctions, but patients can also develop hydrocephalus due to arachnoiditis, with compromise of the optic chiasm and

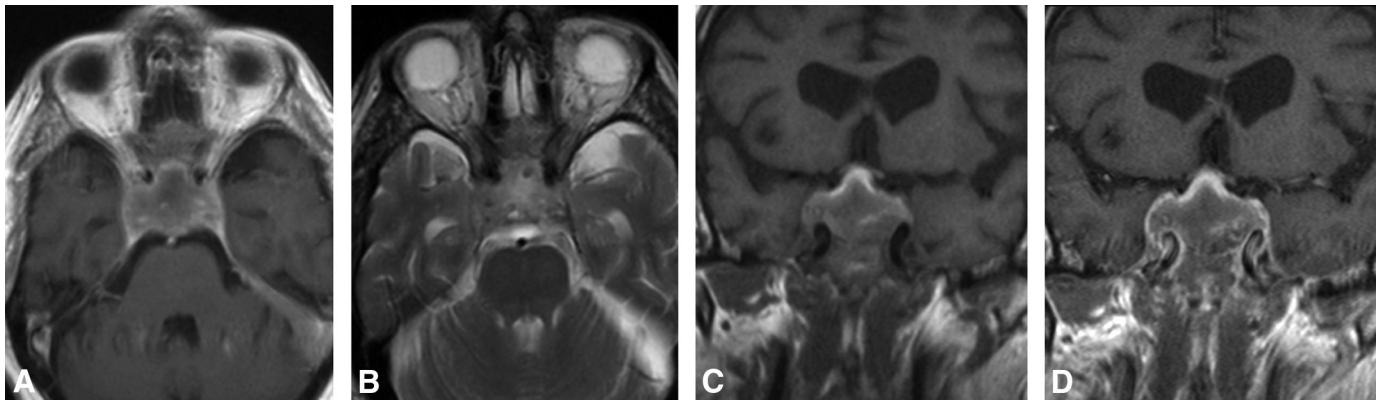

Fig 14. Apoplexy. Axial postgadolinium T1WI (*A*) and T2WI (*B*), and coronal T1WI (*C*) and postgadolinium T1WI (*D*) in a patient with apoplexy. An 82-year-old man with sudden onset of panhypopituitarism. MR imaging of the brain (*A–C*) demonstrates a markedly enlarged pituitary gland with heterogeneous signal intensity; the optic chiasm is cranially displaced. The postgadolinium T1WI (*D*) demonstrates peripheral enhancement, albeit difficult to appreciate due to the intrinsic high signal intensity on T1WI.

pituitary stalk.[66] Treatment usually consists of the surgical excision of the lesion, generally through a trans-sphenoidal approach. On CT, NCC is easily misdiagnosed as a pituitary adenoma with cystic degeneration, a craniopharyngioma, or an arachnoid cyst. MR imaging is more revealing and shows a hypointense cystic lesion that displaces the hypophysis laterally and dorsally to the third ventricle and optic chiasm on postcontrast T1-weighted sequences.[66]

VASCULAR LESIONS

Intrinsic Lesions

Ischemia. Ischemic infarction of the optic chiasm is relatively rare because of its extensive and redundant collateral vascular supply that originates from the circle of Willis.[69] Two cases of embolic chiasmal infarction have been described, one after aortic valve replacement[70] and the other due to atrial fibrillation.[71] In both cases, the patients experienced sudden complete vision loss in one eye with a visual field defect in the contralateral eye, and with an otherwise normal neurologic examination.[70,71] MR imaging (Fig 14), and, in particular, DWI and DTI, plays a major role in detecting ischemia of the visual pathways. DWI may depict larger lesions of the optic chiasm and optic radiations. DTI, which uses multiple diffusion directions of analysis and has a higher signal intensity-to-noise ratio, is sensitive in demonstrating the acute onset and subsequent evolution of visual pathway ischemia.[72] Nevertheless, the necrosis of the chiasm can be appreciated on T1WI and T2WI.

Extrinsic Lesions

AVFs. Cavernous sinus dural AVFs are arteriovenous shunts between dural arteries and the cavernous sinus. They are one of the most common types of intracranial dural arteriovenous shunts and usually present with benign symptoms, such as chemosis and diplopia.[73] Despite this, they may also present with cerebral hemorrhage associated with cortical venous reflux and require urgent treatment for occlusion of the fistula.[73] Carotid-cavernous sinus fistulas (CCFs) are abnormal communications between the cavernous sinus and the internal carotid. They can be classified according to 3 criteria: pathologically into spontaneous or traumatic fistulas, hemodynamically into high-flow or low-flow fistulas, and angiographically into direct or dural fistulas.[74] Traumatic fistulas usually require treatment if there is progressive visual loss or intolerable headache, or if a traumatic venous aneurysm enlarges beyond the cavernous sinus and compresses the optic chiasm.[74] CCFs usually present with diplopia, proptosis, headache, and ocular pain. The treatment of choice for both cavernous sinus dural AVFs and CCFs includes embolization. Cavernous sinus dural AVFs can be seen on CTA as abnormally enlarged and tortuous vessels in the subarachnoid space, which correspond to dilated cortical veins. MR imaging shows dilated pial vessels in the subarachnoid space (Fig 15). There could be regions of white matter edema that may also enhance. These findings are indicative of an aggressive fistula, with a high rate of hemorrhage. DSA remains the definitive imaging technique. CCFs have similar characteristics but the clear connection between the cavernous sinus and the internal carotid is demonstrated.

Cavernous Malformations. Cavernous hemangiomas or cavernous malformations are histologically benign vascular malformations composed of capillary tangles. They can occur anywhere in the brain and spinal cord but have been reported most frequently at subcortical sites in the frontal and temporal lobes.[75] They can be spontaneous, syndromic, or familial, with the familial form often associated with multiple cavernous malformations. The familial form is also reported in association with chiasmal cavernous malformations.[76] Cavernous malformations affect approximately 0.5% of the population, with only a small fraction of these having optochiasmatic cavernous malformations.[77] Cavernous malformations of the cavernous sinus can extend into the sellar or parasellar region. The presenting

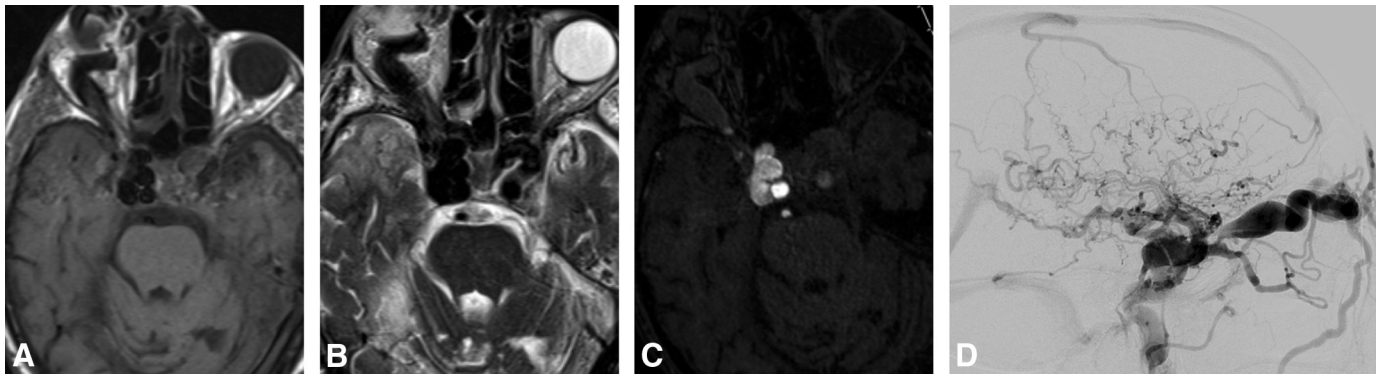

Fig 15. CCF. Axial T1WI (*A*), T2WI (*B*), MRA (*C*), and cerebral angiogram (*D*) of a CCF. An 88-year-old man admitted for status epilepticus. MR imaging of the brain (*A–C*) demonstrates a markedly enlarged right cavernous segment of the internal carotid artery with associated severe enlargement of the right superior ophthalmic vein. There also is arterial flow noted within the right cavernous sinus on the MRA (*C*) of the brain. There is marked venous congestion along the right cortical veins with associated venous hypertension and cortical hyperintensity on T2 (*B*), which is likely the cause of the seizure in this patient. Cerebral angiogram with right internal carotid artery injection with a subtracted view demonstrates early filling of the right cavernous sinus, with antegrade flow to the right superior ophthalmic vein toward the facial vein, along with additional venous reflux to the cortical vein of the right hemisphere, with drainage toward the superior sagittal sinus and the straight sinus.

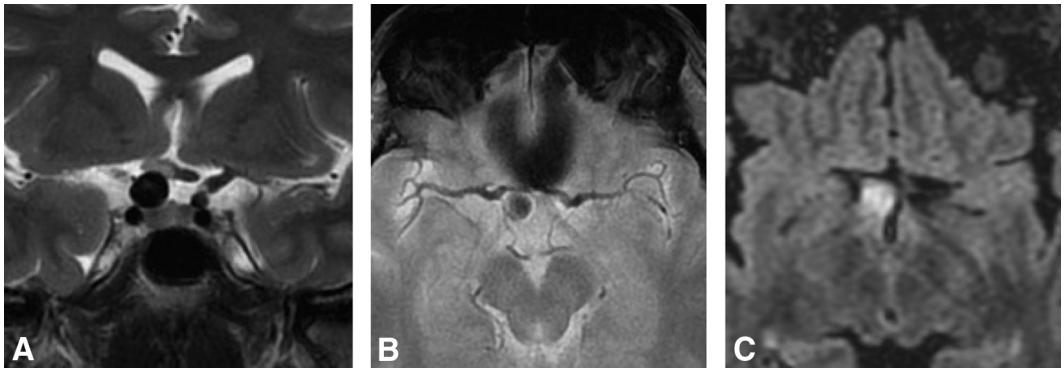

Fig 16. Internal carotid artery aneurysm. Coronal T2WI (*A*), axial gradient recalled-echo T2WI (*B*), and FLAIR (*C*) in a patient with a right internal carotid artery aneurysm. A 45-year-old man with a history of headache. Coronal T2WI (*A*) and axial FLAIR images (*C*) demonstrate an aneurysm of the right internal carotid artery. The optic chiasm is displaced, and there is an area of signal intensity change in the T2WI (*A*) and FLAIR (*C*), which indicates edema.

symptoms in these cases range from patients with asymptomatic lesions to patients with gradually progressive visual loss or pituitary dysfunction, to patients who present acutely with a chiasmatic apoplexy syndrome.[78] The differential diagnosis includes AVMs, aneurysms, optic glioma, craniopharyngioma, and pituitary apoplexy.[77] Treatment for symptomatic cavernous malformations is surgical. MR imaging is the imaging study of choice and demonstrates the typical heterogeneous signal intensities that reflect blood deposits of different ages.[78] It can show a lobulated mass that extends from the cavernous sinus. Signal intensity is usually high on T2WI and low on T1WI; vivid enhancement is demonstrated after gadolinium administration.

Aneurysms. Internal carotid artery–ophthalmic artery aneurysms represent a small proportion of all intracranial aneurysms. They account for 0.3%–1% of all intracranial aneurysms and 0.9%–6.5% of all aneurysms of the internal carotid artery.[79] Yasargil et al[80] reported that 18% of patients with aneurysms of the ophthalmic segment presented with visual symptoms. Ophthalmic artery aneurysms sometimes elevate the optic nerve or optic chiasm, and, in cases of large or giant aneurysms, the optic apparatus can be thinned. There have been only a few cases reported in the literature of complete optic nerve penetration by an unruptured ophthalmic artery aneurysm.[81-84] Takagi and Miyamoto[85] reported the first case of falciform ligament penetration by an ophthalmic artery aneurysm, which thus made anterior clinoidectomy necessary during the surgical procedure to allow the optic canal unroofing and clipping of the aneurysm. Similar to other intracranial aneurysms, those that originate from this segment of the internal carotid artery present with SAH in 20%–70% of patients.[86] In addition, neurologic deficits occur because of the compression of surrounding visual structures, such as the optic nerves and chiasm, which thus leads to blurred vision and visual field impairment.[86] Treatment of this kind of aneurysm is still challenging for neurosurgeons, which results in high rates of morbidity and mortality. Treatment ranges from classic clipping of the neck of the aneurysm through transcranial routes (pterional approach and its variants) to endovascular procedures. MR imaging can easily identify the

aneurysm and provide information on the status of the surrounding parenchyma (Fig 16). Cerebral angiography is the imaging technique of choice. It allows the identification of the aneurysms and measurement of the neck, which are useful in planning the surgical clipping or endovascular treatment.

Compression from Hydrocephalus. Due to the close relationships between the third ventricle and the optic chiasm, optic nerve, and cavernous sinus, different neuro-ophthalmologic disorders may result from ventricle enlargement.[87] Compression of these structures from hydrocephalus may produce proptosis, ophthalmoplegia, or optic nerve dysfunction. Treatment of these symptoms depends on the management of the underlying hydrocephalus. CT is the imaging technique of first choice in the evaluation of enlarged ventricles. However, it may not always show the cause of the obstruction, which may be further investigated with MR imaging.

Traumatic Chiasmal Syndrome. Traumatic damage of the optic chiasm is uncommon because of its protected location at the base of the brain. For this reason, brain injury associated with damage to the anatomically privileged chiasm is necessarily severe, which results in damage of surrounding structures, such as craniofacial bones, the hypothalamic-pituitary axis, carotid arteries, and cranial nerves.[78] Hassan et al[88] reported the largest series of patients with chiasmal trauma, which consisted of 19 patients evaluated at a single institution during a 30-year period. The typical patient was a young adult man after a motor vehicle accident.[88] Two-thirds of the patients had a skull fracture, most commonly a combined frontal and skull base fracture (31% of patients) or a frontal bone fracture (21%).[88] The most common clinical symptom was severe visual loss with bitemporal hemianopia, which represents the hallmark of traumatic chiasmal syndrome.[88] Other deficits related to optic chiasm damage may be represented by diabetes insipidus and/or pituitary dysfunction, anosmia, and oculomotor impairment. Possible mechanisms of chiasmal trauma include direct tearing, contusion and hemorrhage, necrosis, and axonal injury.[86] There is no specific treatment for traumatic chiasmal syndrome, and management of patients is usually focused on the underlying head trauma. CT is useful to identify the skull fractures associated with head trauma. MR imaging in the acute setting, however, is able to detect asymmetries or transections of the optic chiasm and hypothalamic lesions.[88]

SUMMARY

The optic chiasm is a key anatomic structure along the visual pathway that can be affected by many pathologic processes, either intrinsic to the optic chiasm itself or extrinsic and arising in one of the many structures present in this region. We reviewed the imaging findings in a wide range of diseases that originate from or involve the optic chiasm to help differentiate the different neuroimaging features of lesions in this region of the brain.

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