Imaging of Central Nervous System Diseases that Affect Vision

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KEYWORDS

• Visual pathway imaging • Magnetic resonance imaging • Optical nerve • Lateral geniculate nucleus • Optical radiations • Occipital lobe • Visual field defects • Pupillary light reflex

KEY POINTS

- Visual dysfunction in central nervous system disease reflects lesions anywhere along the visual pathway, from the retina and optical nerve to the occipital cortex.
- Lesion localization along the visual pathway strongly predicts the pattern of visual field deficits and pupillary abnormalities.
- A wide spectrum of vascular, inflammatory, infectious, neoplastic, metabolic, and neurodegenerative disorders can involve the visual pathways.
- Advanced imaging techniques, including diffusion and tractography, enhance assessment of optical radiations and occipital lobe involvement.

INTRODUCTION

Central nervous system (CNS) diseases encompass a wide spectrum of pathologies capable of impairing vision and significantly reducing quality of life. Visual dysfunction in these diseases may arise from direct involvement of the optical nerve, optical chiasm, optical tract, lateral geniculate nucleus (LGN), optical radiations, or visual cortex, as well as from secondary processes such as increased intracranial pressure (ICP) or vascular compromise. CNS pathologies that affect vision can be broadly classified into infectious, inflammatory, neoplastic, vascular, metabolic, neurodegenerative, congenital, traumatic, and idiopathic categories, with each class affecting distinct components of the visual pathway and manifesting with characteristic clinical and imaging findings (**Table 1**).

Infectious Diseases

Infectious CNS diseases can involve the visual pathway at multiple levels, leading to optical neuritis, uveitis, retinitis, or encephalitis.[1] Bacterial infections, such as tuberculosis, may directly affect the optical nerve, optical chiasm, or occipital lobe. Cat scratch disease caused by *Bartonella henselae* can result in optical neuropathy and neuroretinitis. Viral infections also pose significant risk; cytomegalovirus (CMV) can cause retinitis, particularly in immunocompromised patients such as those with HIV/AIDS, transplant recipients, or chemotherapy patients, at times leading to blindness.[2] Herpes viruses, including Herpes Simplex Virus (HSV) (**Fig. 1**) and varicella-zoster virus, can cause acute retinal necrosis and/or cerebritis.[3] Congenital infections, as part of the TORCH complex (Toxoplasmosis, CMV, Rubella, Syphilis, HSV), can produce visual impairment by damaging

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Abbreviations

CMV	Cytomegalovirus
CNS	central nervous system
CT	Computed tomography
CZS	Congenital Zika syndrome
DWI	diffusion-weighted imaging
EW	Edinger-Westphal
HSV	Herpes Simplex Virus
ICP	intracranial pressure
IIH	idiopathic intracranial hypertension
LGN	lateral geniculate nucleus
MCA	middle cerebral artery
MLF	medial longitudinal fasciculus
MS	multiple sclerosis
NMOSD	neuromyelitis optica spectrum disorder
PCA	posterior cerebral artery
RGC	retinal ganglion cell
SC	superior colliculus
TBI	traumatic brain injury
TORCH	Toxoplasmosis, CMV, Rubella, Syphilis, HSV

the developing brain and visual cortex, as well as the retina and choroid. Congenital Zika syndrome (CZS) is associated with microcephaly, cortical malformations, and profound visual deficits. Computed tomography (CT) and MR imaging studies have demonstrated occipital cortical volume loss in 95% of affected infants, even in cases where the ocular examination is normal, highlighting the importance of neuroimaging in identifying cortical visual impairment.

Inflammatory Diseases

Inflammatory disorders frequently target the optical nerve and optical pathway. Multiple sclerosis (MS) (**Fig. 2**) and neuromyelitis optica spectrum disorder (NMOSD) are prototypical demyelinating diseases that produce optical nerve inflammation and white matter lesions.[4] Myelin oligodendrocyte glycoprotein antibody-associated disease frequently affects vision through acute optical neuritis with prominent inflammatory signs. Visual recovery after high-dose corticosteroids is often favorable, however, relapses are common and can lead to cumulative optical nerve damage and permanent visual impairment.[5] Neurosarcoidosis is characterized by granulomatous lesions that may affect the optical nerves, meninges, or brain parenchyma, at times leading to irreversible visual impairment.[6] Imaging modalities, including contrast-enhanced MR imaging and PET-CT, are critical for detecting inflammatory lesions and monitoring response to therapy.

Neoplastic Diseases

Neoplasms affecting the visual pathway can be primary or secondary and involve structures from the optical nerve to the occipital cortex. Optical nerve gliomas, commonly associated with neurofibromatosis type 1, are slowly growing tumors that can compromise visual function.[7] Pituitary macroadenomas (**Fig. 3**) and craniopharyngiomas frequently exert mass effect on the optical chiasm, producing bitemporal hemianopia. Meningiomas arising from the dura mater near the optical nerve or chiasm may similarly cause progressive visual loss. Occipital lobe tumors interfere with visual processing and may result in homonymous visual field defects. Lymphomas and brain metastases may infiltrate visual pathway structures, depending on their location, resulting in variable visual symptoms. Additionally, tumors can increase ICP, leading to papilledema and secondary optical nerve damage. Early recognition using MR imaging and CT is vital, as timely intervention may preserve visual function.

Vascular Diseases

Vascular disorders are a major cause of vision loss, affecting both the eye and the brain. Local vascular compromise in the retina can result in retinal artery or vein occlusion, ischemic optical neuropathy, or retinopathy secondary to systemic diseases such as diabetes or hypertension.[8] Large vessel occlusions involving the posterior cerebral artery (PCA), which supplies the occipital lobe and visual cortex, can cause homonymous hemianopia or quadrantanopia.[9] The clinical spectrum of vascular injury extends beyond field defects to include visual hallucinations, visual agnosia, neglect, prosopagnosia, and cortical blindness.[10–12] Imaging modalities such as diffusion-weighted MR imaging and CT angiography are crucial for identifying ischemic injury and guiding intervention.

Metabolic Diseases

Metabolic disorders can disrupt vision through direct effects on the optical nerve, retina, or visual cortex. X-linked adrenoleukodystrophy causes demyelination, predominantly involving the parietal and occipital white matter that frequently compromises visual pathways (**Fig. 4**).[13] Mitochondrial disorders, such as MELAS and Leigh syndrome, impair energy metabolism, leading to optical neuropathy and cortical visual impairment.[14] Thiamine deficiency, hepatic encephalopathy, and storage diseases, such as Batten disease, may similarly produce vision loss by damaging neuronal tissue,[15] particularly through involvement of the retina,

Table 1
CNS diseases affecting vision: classification, anatomic location, imaging findings, and clinical features

Disease Category	Example Disorders	Anatomical Location	Key Imaging Findings	Clinical Features
Infectious	Tuberculosis, CMV retinitis, Herpes retinitis, TORCH, Congenital Zika Syndrome (CZS)	Optical nerve, chiasm, occipital lobe, retina	MR imaging: T2/FLAIR hyperintensity, contrast enhancement of optical nerve/chiasm; CT: cortical calcifications (in CZS)	Vision loss, visual field defects, cortical visual impairment, retinal necrosis
Inflammatory	Multiple sclerosis, NMOSD, neurosarcoidosis	Optical nerve, optical tract, brain parenchyma, meninges	MR imaging: T2 hyperintense demyelinating lesions, optical nerve enhancement, granulomas (sarcoidosis)	Optical neuritis, monocular or bilateral visual loss, visual field deficits
Neoplastic	Optical nerve glioma, pituitary adenoma, craniopharyngioma, meningioma, lymphoma, brain metastases	Optical nerve, chiasm, occipital lobe	MR imaging: Mass lesion, contrast enhancement, edema, possible optical nerve compression; CT: mass effect, calcifications	Visual field defects (bitemporal hemianopia, homonymous hemianopia), progressive vision loss, papilledema
Vascular	Retinal artery/vein occlusion, ischemic optical neuropathy, PCA infarct, temporal arteritis	Retina, optical nerve, visual cortex	MR imaging: Diffusion restriction (stroke), T2/FLAIR hyperintensity; CT angiography: vessel occlusion	Hemianopia, quadrantanopia, cortical blindness, visual hallucinations, visual agnosia, prosopagnosia
Metabolic	Adrenoleukodystrophy, MELAS, Leigh syndrome, thiamine deficiency, Batten disease, small vessel disease	Optical nerve, optical radiations, visual cortex	MR imaging: White matter abnormalities, cortical atrophy, basal ganglia lesions; MR spectroscopy: lactate peaks	Visual loss, optical neuropathy, cortical visual impairment, diplopia
Neurodegenerative	Alzheimer's disease, Parkinson's disease, Lewy body dementia, posterior cortical atrophy, Creutzfeldt–Jakob disease	Visual cortex, parietal/occipital association areas, retina	MR imaging: Cortical and subcortical atrophy, occipital/parietal cortical thinning; FDG-PET: hypometabolism	Visual hallucinations, visuospatial deficits, cortical visual syndrome, rapid cortical blindness, impaired eye movement control

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Table 1
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Disease Category	Example Disorders	Anatomical Location	Key Imaging Findings	Clinical Features
Congenital	Holoprosencephaly, septo-optical dysplasia, periventricular leukomalacia, congenital hydrocephalus, perinatal stroke	Optical nerve, optical radiations, visual cortex, white matter	MR imaging: Malformations (absent septum pellucidum, polymicrogyria, agenesis of corpus callosum), white matter injury, cortical thinning	Congenital visual impairment, cortical visual deficits, visual field abnormalities
Traumatic	TBI, shearing injury, posterior circulation stroke post-trauma	Optical nerve, chiasm, optical tract, visual cortex	MR imaging: Susceptibility-weighted imaging for microhemorrhages, DTI for axonal injury, CT for acute hemorrhage	Cortical blindness, hemianopia, visual neglect, motion perception loss, visual attention deficits
Idiopathic	Idiopathic intracranial hypertension, functional/psychogenic visual loss, idiopathic occipital atrophy	Optical nerve, visual cortex	MR imaging: Normal or subtle cortical atrophy, empty sella (IIH)	Visual field loss, papilledema (IIH), inconsistent exam (functional), progressive cortical visual loss (idiopathic occipital atrophy)

Abbreviations: CMV, cytomegalovirus; CT, computed tomography; CZS, Congenital Zika syndrome; DTI, diffusion tensor imaging; FDG - PET, fluorodeoxyglucose positron emission tomography; FLAIR, fluid-attenuated inversion recovery; IIH, idiopathic intracranial hypertension; MELAS, mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes; MR, magnetic resonance; MR imaging, magnetic resonance imaging; NMOSD, neuromyelitis optica spectrum disorder; PCA, posterior cerebral artery; T2, T2-weighted imaging; TORCH, toxoplasmosis, other infections [including syphilis], rubella, cytomegalovirus, and Herpes Simplex Virus.

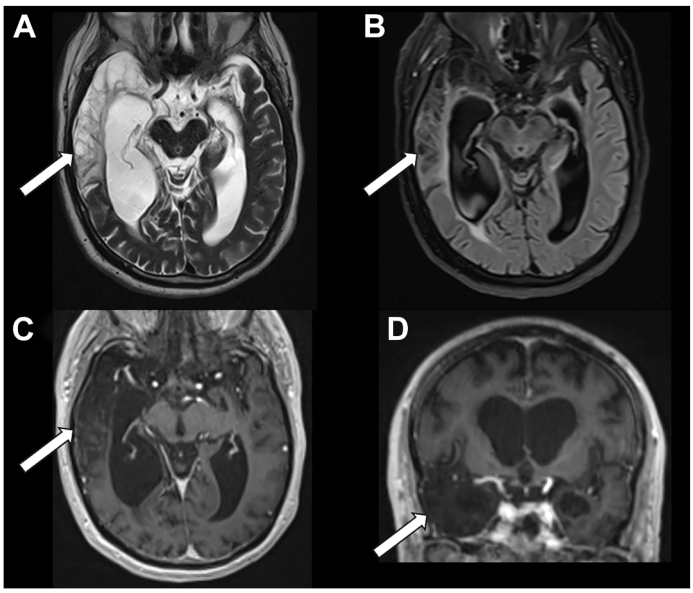

Fig. 1. Sequela of HSV encephalitis involving the optical radiations within the right temporal lobe. Axial T2-weighted (*A*), axial FLAIR (*B*), axial postcontrast T1 (*C–D*), and coronal postcontrast T1 showing encephalomalacia (*white arrows*) involving the right temporal lobe, including portions of the optical radiations (Meyer's Loop) in a patient with sequela of HSV encephalitis. HSV, Herpes Simplex Virus.

optical nerve, optical radiations, and visual cortex. Systemic metabolic diseases, including diabetes and hypertension, can compromise the vasa nervorum of cranial nerves, resulting in diplopia or other ocular motor deficits.[16,17] Neuroimaging, including MR spectroscopy, may help identify metabolic abnormalities affecting visual pathways.

Neurodegenerative Diseases

Neurodegenerative disorders can affect vision through ocular involvement or cortical degeneration. Disorders such as age-related macular degeneration, glaucoma, and retinitis pigmentosa primarily involve the retina or optical nerve, while diseases like Alzheimer's and Parkinson's disease affect visual perception and processing through cortical and subcortical degeneration.[18] Posterior cortical atrophy, a variant of Alzheimer's disease, presents with cortical visual syndrome, including visuospatial and perceptual deficits. The Heidenhain variant of Creutzfeldt–Jakob disease produces rapid cortical blindness. Lewy body dementia is characterized by visual hallucinations and perceptual errors, while Parkinsonian syndromes may cause subtle visuospatial and ocular motor deficits. Neuroimaging, particularly volumetric MR imaging and functional imaging, aids in assessing structural and functional changes underlying visual dysfunction.

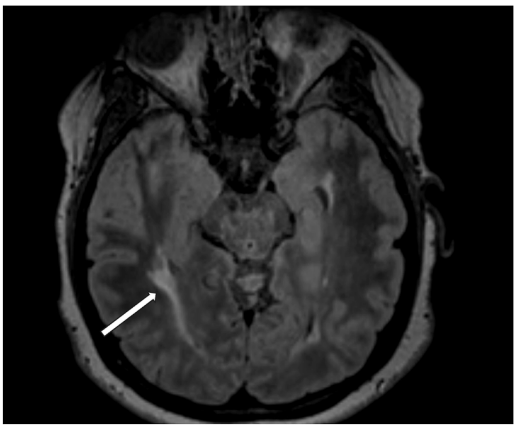

Fig. 2. Multiple sclerosis lesions involving the optical radiations. Axial FLAIR imaging in a patient with long-standing history of multiple sclerosis, including old demyelination lesions involving the optical radiations, with an example on the right noted by a white arrow.

Congenital Disorders

Congenital cerebral visual impairment arises from maldevelopment or early damage to visual pathways. Structural brain malformations, such as holoprosencephaly, septo-optical dysplasia (**Fig. 5**), lissencephaly, pachygyria, polymicrogyria, schizencephaly, and corpus callosum agenesis, can all affect optical nerves, optical radiations, or the visual cortex. Posterior fossa malformations, including Chiari and Dandy–Walker malformations, may distort visual pathways. White matter abnormalities (such as periventricular leukomalacia), congenital hydrocephalus, or arachnoid cysts, can compromise the optical radiations. Perinatal hypoxic-ischemic injury is the most common cause of congenital visual impairment worldwide,

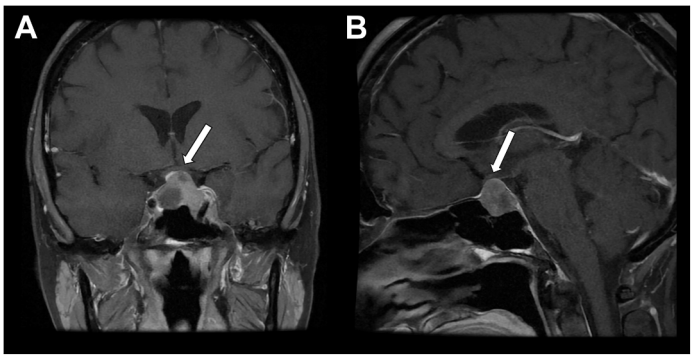

Fig. 3. Pituitary macroadenoma extending into the suprasellar cistern and exerting mass effect on the inferior aspect of the optical chiasm. Coronal (*A*) and sagittal (*B*) post-contrast T1-weighted imaging showing a heterogeneously enhancing mass lesion contacting and elevating the left aspect of the optical chiasm (*white arrow*).

while congenital stroke or intracranial hemorrhage affecting visual structures further contributes to early-life vision loss. Genetic, metabolic, infectious, and inflammatory disorders may also manifest as congenital visual pathway deficits, highlighting the importance of neuroimaging for early detection and intervention, particularly when reversible causes can be identified.

Traumatic Disorders

Traumatic brain injury (TBI), brainstem injury, or cranial nerve trauma can disrupt vision. Focal cortical injury may cause immediate cortical blindness or hemianopia, while diffuse axonal injury can produce delayed visual field deficits and impaired visual attention. Traumatic ischemia involving the PCA territory may result in occipital lobe stroke with homonymous hemianopia. Injury to the optical chiasm or optical tracts may produce specific visual field defects, and higher-order cortical injury may result in visual neglect, agnosia, or impaired motion perception. MR imaging, particularly with susceptibility-weighted imaging and diffusion tensor imaging, plays a central role in assessing post-traumatic visual pathway injury.[19]

Idiopathic Disorders

Idiopathic visual loss encompasses conditions in which no structural, infectious, inflammatory, traumatic, or metabolic cause can be identified. Idiopathic intracranial hypertension (IIH) is characterized by elevated ICP of unknown etiology, producing papilledema and visual field loss. Functional or psychogenic visual disorders may present with apparent blindness in the setting of normal imaging and inconsistent examination findings. Idiopathic occipital atrophy is a rare condition associated with slowly progressive cortical visual loss. Neuroimaging may reveal subtle cortical atrophy or be entirely normal, necessitating careful correlation with clinical findings.

In summary, CNS diseases affecting vision encompass a broad range of etiologies, each with unique mechanisms of injury, clinical manifestations, and imaging characteristics. Infectious, inflammatory, neoplastic, vascular, metabolic, neurodegenerative, congenital, traumatic, and idiopathic disorders may impact any level of the visual pathway, from the retina to the occipital cortex. Advanced imaging modalities, particularly MR imaging and CT, are critical in identifying the

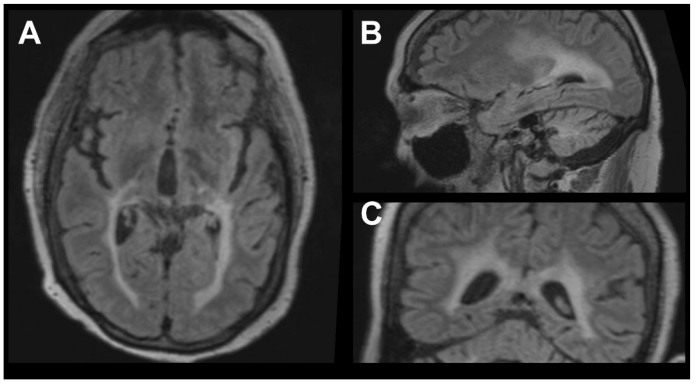

Fig. 4. Adrenoleukodystrophy involving the optical radiations. Axial (*A*), sagittal (*B*), and coronal (*C*) FLAIR imaging in a case of adrenoleukodystrophy showing extensive involvement of the white matter tracts of the optical radiations.

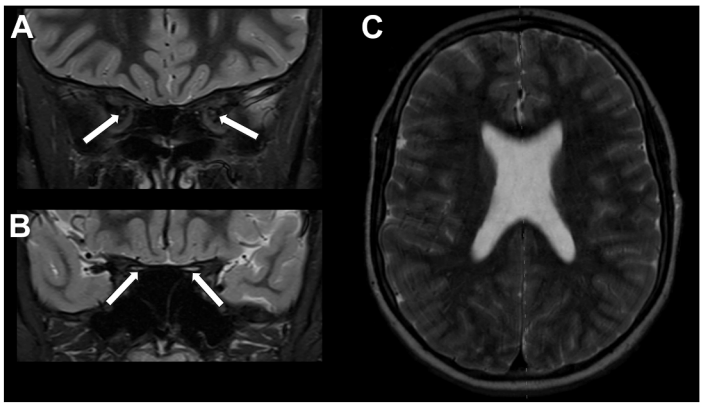

Fig. 5. Septo-optical dysplasia. (*A*) Coronal STIR showing diminutive bilateral optical nerves (*white arrows*). (*B*) Small optical canal bilaterally with diminutive canalicular segment of the bilateral optical nerves. (*C*) Axial T2 weighted imaging showing the lateral ventricles with an absent septum pellucidum.

location, extent, and nature of these lesions. Clinicians must integrate clinical examination, neuroimaging, and functional assessments to accurately localize pathology, guide treatment, and predict visual outcomes. Understanding the diverse causes of CNS-related visual impairment is essential for timely diagnosis, intervention, and preservation of visual function.

VISUAL PATHWAY ANATOMY OVERVIEW

The visual system is a highly specialized and complex neural network responsible for the reception, transmission, and processing of visual information. The pathway begins in the retina, where light stimuli are converted into electrical signals through a process called phototransduction. Photoreceptors—comprising rods and cones—are specialized neurons that detect light and color. Rods are highly sensitive to dim light and facilitate scotopic vision, whereas cones are responsible for color perception and high-acuity photopic vision. Upon stimulation, photoreceptors release neurotransmitters that activate bipolar cells, which, in turn, transmit signals to retinal ganglion cells (RGCs).[20]

The axons of RGCs converge in the retinal nerve fiber layer to form the optical nerve head. The optical nerve is a collection of over one million axons that exit the globe via the lamina cribrosa and extend posteriorly toward the brain (**Fig. 6**). At the optical chiasm, fibers from the nasal half of each retina cross to the contralateral optical tract, while temporal fibers remain ipsilateral. This partial decussation enables the visual cortex to integrate binocular visual input, providing depth perception and a cohesive visual field.

After the chiasm, the fibers continue as the optical tracts, which terminate primarily in the LGN of the thalamus. The LGN acts as a relay and processing hub, where information is organized and sent via the optical radiations to the primary visual cortex (V1) in the occipital lobe. The optical radiations follow a precise trajectory: fibers representing the superior visual field pass through the parietal lobe, whereas fibers representing the inferior visual field travel via Meyer's loop in the temporal lobe.[20] This organization is clinically significant, as lesions along specific portions of the pathway result in predictable visual field deficits, such as homonymous hemianopia, quadrantanopia, or scotomas.

The visual cortex receives its blood supply predominantly from the posterior cerebral arteries and their branches, including the calcarine, posterior temporal, and parieto-occipital arteries. Notably, the occipital pole, which mediates central vision, may receive dual blood supply from anastomoses between PCA branches and the superior temporo-occipital branch of the middle cerebral artery. This dual vascularization is clinically relevant because ischemic or hemorrhagic events affecting one arterial territory may produce selective visual field deficits while sparing central vision.[20]

Visual field defects correlate with the anatomic location of lesions along the pathway. For instance, retinal or optical nerve injuries typically produce monocular visual loss, whereas chiasmal lesions—such as pituitary adenomas—classically result in bitemporal hemianopia. Lesions of the optical tract, LGN, or optical radiations often cause homonymous hemianopia, with the location of quadrantic or hemianopic defects determined by the portion of the visual field represented by the affected fibers. Lesions affecting the occipital cortex may produce cortical blindness or scotomas, which are often congruous between both eyes due to the precise retinotopic organization of the primary visual cortex (V1) (**Fig. 7**).[20]

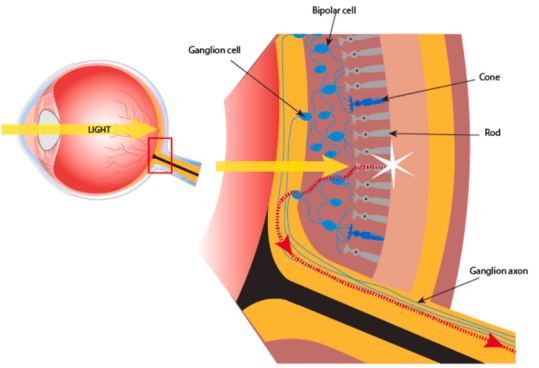

Fig. 6. Retinal neural elements and optical nerve formation. The retinal photoreceptors, cones and rods, receive light information and pass an electrical signal to the bipolar cells, which synapse with ganglion cells and transmit the information. The ganglion cells have long axons that extend to form the optical nerve at the optical papilla (optical nerve head), subsequently continuing toward the brain.

LIGHT REFLEX PATHWAY OVERVIEW

The pupillary light reflex is an essential autonomic function that adjusts pupil diameter in response to changes in ambient light, thereby optimizing retinal illumination and protecting photoreceptors from excessive light exposure. This reflex is mediated by a well-defined neural circuit consisting of an afferent (sensory) limb, which detects light, and an efferent (motor) limb, which mediates pupillary constriction.

AFFERENT LIMB

The afferent limb begins in the retina, where photoreceptor cells respond to light stimuli and activate bipolar cells and retinal ganglion cells. The axons of RGCs form the optical nerve, which transmits visual information, including light intensity, toward the CNS. At the optical chiasm, nasal fibers decussate to the contralateral side, while temporal fibers remain ipsilateral, allowing bilateral processing of light stimuli. These axons continue through the optical tracts, where a subset of fibers diverges to synapse in the pretectal nuclei of the midbrain, a crucial relay station for the pupillary light reflex.[20]

EFFERENT LIMB

The pretectal nucleus projects bilaterally to the Edinger-Westphal (EW) nuclei, which are part of the oculomotor nerve complex (cranial nerve III). Preganglionic parasympathetic fibers from the EW nucleus travel along the oculomotor nerve to synapse in the ciliary ganglion. Postganglionic fibers, known as short ciliary nerves, innervate the

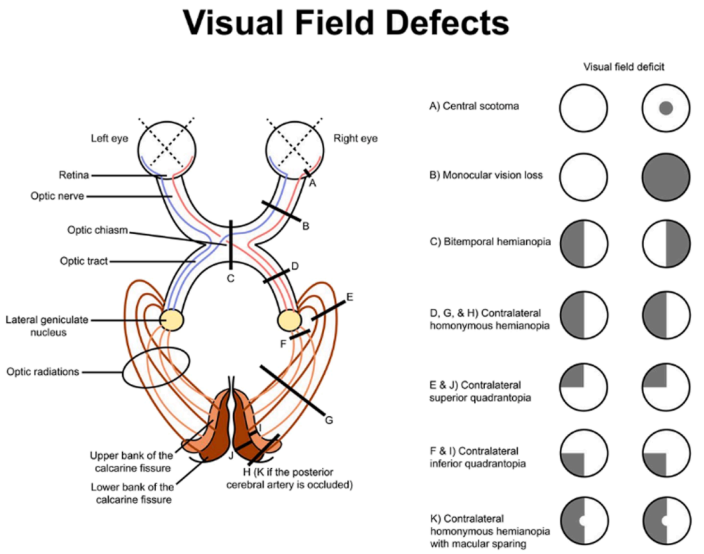

Fig. 7. Visual field defects resulting from injury to different areas of the visual pathway.

sphincter pupillae muscle of the iris. Activation of this muscle induces pupillary constriction, reducing the amount of light entering the eye and protecting the retina (**Fig. 8**).[20,21]

CONSENSUAL RESPONSE

An important feature of the pupillary light reflex is its bilateral response. Because the pretectal nucleus communicates with both EW nuclei, illumination of one eye leads to constriction in both the ipsilateral eye (direct response) and the contralateral eye (consensual response) (see **Fig. 8**). This mechanism ensures synchronized pupil size and highlights the integration of visual and autonomic pathways. Clinical evaluation of the light reflex can reveal localized lesions in the retina, optical nerve, midbrain, or oculomotor pathway, making it a valuable diagnostic tool in neuro-ophthalmology.[20,21]

CLINICAL IMPLICATIONS

Understanding the anatomy and physiology of the visual and pupillary pathways is essential in both clinical and imaging contexts. Precise localization of lesions based on visual field defects or abnormal pupillary responses can guide diagnostic imaging, surgical planning, and prognosis. For example, lesions at the optical chiasm often present with bitemporal field defects, whereas cortical lesions may produce homonymous congruous hemianopia. Similarly, afferent pupillary defects can indicate optical nerve dysfunction, while efferent deficits suggest involvement of the oculomotor nerve or its parasympathetic components. Radiologic imaging, including MR imaging and CT, plays a crucial role in identifying the etiology and extent of these lesions.[20,21]

In summary, the visual pathway and pupillary light reflex pathway represent intricate neural networks with distinct anatomic and physiologic characteristics. From the retina to the visual cortex, and from photoreceptor activation to pupillary constriction, each component plays a critical role in vision and ocular protection. An understanding of these pathways provides a framework for clinical assessment, interpretation of visual field deficits, guidance of necessary imaging studies and targeted management of neurologic and ophthalmic disorders.

ANATOMIC BRAIN REGIONS AFFECTING VISION

Lateral Geniculate Nucleus

Background

The LGN is a key thalamic relay nucleus involved in the transmission of visual information from the retina to the primary visual cortex. As a sensory projection nucleus, it serves as an essential hub in the visual processing pathway, integrating input from both retinas and transmitting organized visual signals to cortical regions.[22] The LGN is strategically located in the dorsal posterolateral thalamus, adjacent to the pulvinar and posterior to the inferior choroidal point of the choroid plexus. Its nomenclature derives from its lateral position relative to the medial geniculate nucleus and the sharp bend or "geniculum" of its laminae.[23]

Anatomically, the LGN consists of 6 layers, with parvocellular layers processing high-resolution visual input, particularly color and fine detail, and magnocellular layers mediating motion and luminance contrast.[24] The LGN receives input not only from retinal ganglion cells but also from feedback projections of the visual cortex and other brainstem structures, allowing modulation of sensory input in response to attention and other cognitive states.

Pathology and clinical syndromes

Lesions affecting the LGN disrupt the normal flow of visual information from the retina to the primary

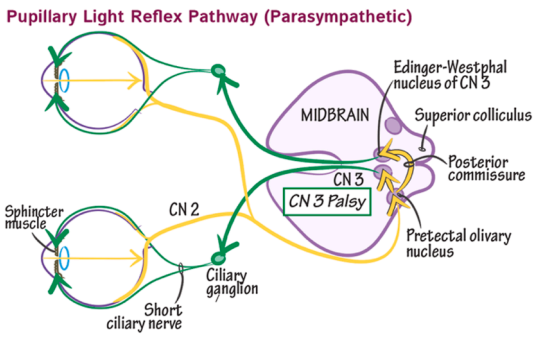

Fig. 8. Pupillary light reflex pathway: schematic of the anatomy and function of the pupillary light reflex pathway. Neuroanatomy Glossary: Dilated Pupil | ditki medical & biological sciences. Retrieved from: https://ditki.com/course/neuroanatomy/glossary/physical-exam/dilated-pupil.

visual cortex, often resulting in *homonymous visual field defects*. Thalamic infarcts, particularly in the territory of the anterior choroidal artery and the lateral posterior choroidal artery, represent a common vascular cause.[25] Tumors extending from surrounding structures such as the temporal lobe, thalamus, or posterior fossa may also involve the LGN.[26] MS plaques may involve thalamic pathways, leading to selective visual deficits.[27] Infectious causes such as viral encephalitis can rarely produce LGN involvement via thalamic inflammation.

Visual implications

LGN lesions classically produce contralateral homonymous hemianopia or quadrantanopia. The precise pattern of visual field loss correlates with which LGN layers are involved and whether the lesion affects fibers from the superior or inferior retina. For example, infarction in the lateral portion of the LGN may result in superior quadrantanopia, whereas medial LGN involvement may produce inferior quadrantanopia (**Fig. 9**).[26]

Imaging pearl: High-resolution MR imaging, particularly with diffusion-weighted imaging (DWI), is crucial for identifying acute thalamic infarcts affecting the LGN. Structural lesions such as tumors are well visualized with T1/T2-weighted sequences and contrast enhancement.

Optical Radiations

Background

The optical radiations, also known as the geniculocalcarine tracts, represent essential white matter pathways that transmit visual information from the LGN of the thalamus to the primary visual cortex (V1) in the occipital lobe. These tracts are integral to normal visual perception, ensuring the accurate relay of visual signals from the retina to the cortical regions responsible for conscious visual processing. The fibers of the optical radiations are organized in a broad, fan-like distribution as they course through the temporal, parietal, and occipital lobes, ultimately terminating in the calcarine cortex, corresponding to Broadman area 17. Anatomical studies have delineated the optical radiations into 3 primary bundles based on their cortical terminations and the regions of the visual field they convey. The anterior or temporal portion, known as Meyer's loop, carries fibers associated with the superior visual field, which correspond to the inferior retinal quadrants. The central bundle, traversing the parietal lobe, contains macular fibers essential for central vision and high-acuity perception. The posterior or parietal portion, sometimes referred to as Baum's loop, transmits fibers from the superior retinal quadrants responsible for the inferior visual field.[28]

Pathology and clinical syndromes

The optical radiations are particularly susceptible to a wide spectrum of pathologic processes, each with characteristic clinical consequences. Ischemic events affecting the middle cerebral artery (MCA) territory frequently involve the temporal and parietal portions of the optical radiations. Primary brain tumors, such as gliomas, as well as metastatic lesions and CNS lymphomas, can disrupt these tracts either through direct invasion or by inducing peritumoral edema, resulting in progressive visual disturbances. Demyelinating diseases, most notably MS, can produce focal lesions within the optical radiations, which manifest clinically as intermittent visual symptoms that may fluctuate in severity and often precede more widespread neurological dysfunction. Traumatic injury, including diffuse axonal injury, as well as iatrogenic disruption from surgical

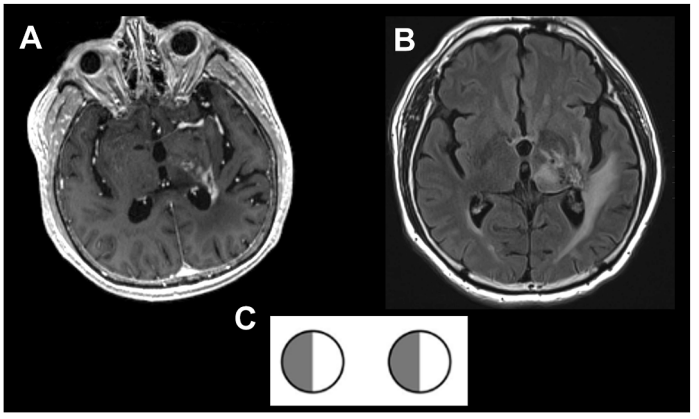

Fig. 9. Treated glioblastoma involving the LGN in the thalamus and schematic of expected associated visual field defect. Axial post-contrast T1-weighted imaging (*A*) showing heterogeneously enhancement involving the left thalamus, internal capsule, and periatrial white matter and axial FLAIR imaging (*B*) showing areas of surrounding edema. Example of visual field defect resulting from a lesion in the LGN, with a schematic displaying contralateral homonymous hemianopia (*C*), indicating visual field defect in the gray area of the representative visual field. LGN, lateral geniculate nucleus.

interventions such as temporal lobectomy or parietal craniotomy, can also damage optical radiation fibers. In contemporary neurosurgical practice, preoperative mapping of the optical radiations using diffusion tensor imaging (DTI) and tractography has become a critical tool to minimize postoperative visual deficits and to preserve high-acuity vision.[28]

Visual implications

Visual field defects from optical radiation injury are topographically specific:

- *Meyer's loop lesions:* Contralateral superior quadrantanopia ("pie in the sky").
- *Central bundle lesions:* Central scotomas affecting macular vision.
- *Baum's loop lesions:* Contralateral inferior quadrantanopia ("pie on the floor").

The clinical manifestations of optical radiation pathology are closely tied to the anatomical distribution of the damaged fibers. Lesions affecting Meyer's loop typically produce contralateral superior quadrantanopia, colloquially referred to as the "pie in the sky" defect (**Fig. 10**), reflecting the disruption of fibers carrying inferior retinal input. Involvement of the central bundle predominantly results in central scotomas, which can severely impair macular vision and consequently impact activities requiring fine visual discrimination, such as reading or recognizing faces. Damage to Baum's loop fibers results in contralateral inferior quadrantanopia, sometimes described as the "pie on the floor" defect, due to interruption of superior retinal fibers. The precise mapping of visual field deficits to specific portions of the optical radiations not only aids clinicians in localizing lesions within the white matter but also informs surgical planning, prognostic evaluation, and rehabilitation strategies.[28]

Imaging pearl: Advancements in neuroimaging have enhanced our ability to visualize and evaluate the integrity of the optical radiations in vivo. Techniques such as DTI allow for the quantification of fractional anisotropy and mean diffusivity along the tract, providing insight into microstructural changes associated with demyelination, edema, or axonal loss. Functional MR imaging, when combined with tractography, can also facilitate the identification of functional boundaries in proximity to lesions, improving the safety of surgical interventions and guiding neurorehabilitative approaches.

Occipital Cortex

Background

The *occipital cortex* is the cerebral hub for visual processing. It is located in the posterior cerebrum, bounded by the parieto-occipital sulcus superiorly and the pre-occipital notch inferiorly. The calcarine sulcus on the medial surface represents a crucial landmark separating upper and lower visual field representations.[29–31]

The occipital cortex is organized into Brodmann areas 17, 18 and 19 (V3, V4, V5/MT). The primary visual cortex, also known as area 17 or V1, serves as the initial cortical recipient of visual information from the retina via the LGN. It is organized retinotopically, allowing for a spatially precise representation of the visual field, and plays a critical role in encoding fundamental visual features such as edges, orientation, motion, and color. Area 18, corresponding to the secondary visual cortex (V2), receives input from V1 and is responsible for higher-order integration, contributing to the perception of depth, contour delineation, and

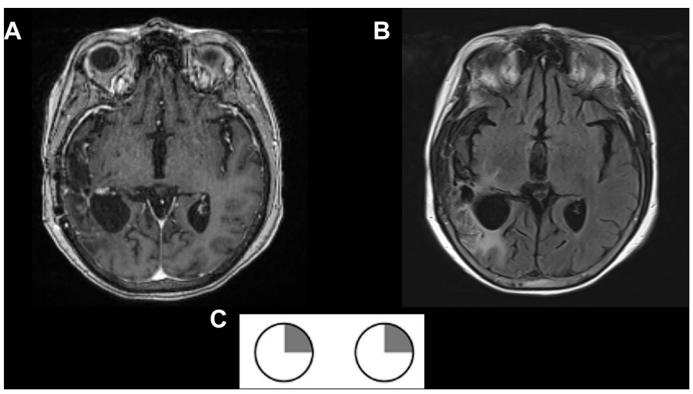

Fig. 10. Treated tumor involving Meyer's Loop and associated visual field defect schematic. Axial postcontrast T1-weighted imaging (*A*) showing foci of heterogeneous enhancement and axial FLAIR imaging (*B*) showing areas of surrounding hyperintense signal in a case of glioblastoma multiforme with extensive post-treatment changes, including surgical debulking and post-radiation changes involving the right temporal lobe, including the white matter tracts that form Meyer's Loop. Example of visual field defect resulting from a lesion involving Meyers Loop, with a schematic displaying contralateral superior quadrantanopia, known as "pie in the sky" visual field defect (*C*), indicating visual field defect in the gray area of the representative visual field.

binocular vision. Area 19, which encompasses the tertiary visual cortical regions V3, V4, and V5/MT, mediates specialized aspects of visual processing. Specifically, V3 is implicated in the analysis of form and shape, V4 is critical for color perception, and V5/MT is specialized for motion detection, facilitating the interpretation of dynamic visual stimuli. Together, these hierarchical cortical areas support the complex processing required for coherent visual perception, enabling the brain to construct a detailed and dynamic representation of the surrounding environment (**Table 2**).

PATHOLOGY AND SYNDROMES

The occipital cortex is vulnerable to a broad spectrum of CNS pathologies, including vascular, neoplastic, infectious, inflammatory, demyelinating, and neurodegenerative conditions.[32] Vascular insults affecting the occipital cortex most commonly arise from infarctions in the territory of the PCA (**Fig. 11**). Such infarcts frequently produce contralateral homonymous hemianopia, which may be accompanied by relative macular sparing due to collateral perfusion from the MCA. In addition, watershed infarcts can lead to patchy or sectoral visual field defects.

Neoplastic processes involving the occipital cortex also have profound visual consequences. Primary cortical gliomas, metastatic lesions, and CNS lymphomas can disrupt normal cortical processing and lead to complex visual disturbances. Depending on the lesion's location and extent, patients may experience homonymous visual field deficits, distortions in color perception, or more subtle disruptions of higher-order visual cognition. The infiltrative nature of some neoplasms, particularly high-grade gliomas, further complicates the clinical presentation, as adjacent white matter tracts—including the optical radiations—may be involved, magnifying the severity of visual dysfunction.

Neurodegenerative disorders provide another pathway for occipital cortical dysfunction. Posterior cortical atrophy, an atypical variant of Alzheimer's disease, selectively targets parieto-occipital and occipito-temporal regions, leading to progressive visuospatial disorientation, difficulty with object and face recognition, and deficits in complex visual processing. Patients with posterior cortical atrophy often retain memory and language functions early in the disease, which highlights the specificity of cortical involvement. Classical Alzheimer's disease may also involve the occipital lobes in later stages, producing visuoperceptual deficits that compound the cognitive impairment.[32]

Demyelinating conditions, most notably MS, can generate focal cortical lesions within the occipital lobes, leading to transient or permanent visual field deficits. These plaques disrupt the normal transmission of visual signals and may manifest clinically as acute visual loss or subtle alterations in visual perception. In addition to demyelination of the optical nerves, involvement of the occipital cortex underscores the widespread impact that CNS demyelinating processes can have on vision.

VISUAL IMPLICATIONS

- *V1 lesions:* Contralateral homonymous hemianopia; bilateral lesions → cortical blindness (Anton syndrome).
- *V4 lesions:* Central achromatopsia (color perception deficits).
- *V5/MT lesions:* Akinetopsia (motion perception loss).
- *Dorsal stream (parietal-occipital):* Optical ataxia, impaired spatial orientation.
- *Ventral stream (temporal-occipital):* Visual agnosia, prosopagnosia (face blindness).[29–33]

The functional implications of occipital cortex lesions are highly dependent on the specific cortical area affected. Lesions involving the primary visual cortex (V1) typically result in contralateral homonymous hemianopia, reflecting the precise retinotopic organization of this region. Bilateral V1 lesions may lead to cortical blindness, a profound loss of conscious vision, which can be accompanied by

Table 2
Occipital cortex organization and function

Area	Name	Function
17	Primary visual cortex (V1)	Retinotopic mapping; encodes edges, orientation, motion, and color.
18	Secondary visual cortex (V2)	Integration of V1 input; depth, contour, binocular vision.
19 (V3, V4, V5/MT)	Tertiary visual cortex	Higher-order processing: V4 → color, V5/MT → motion, V3 → form/shape.

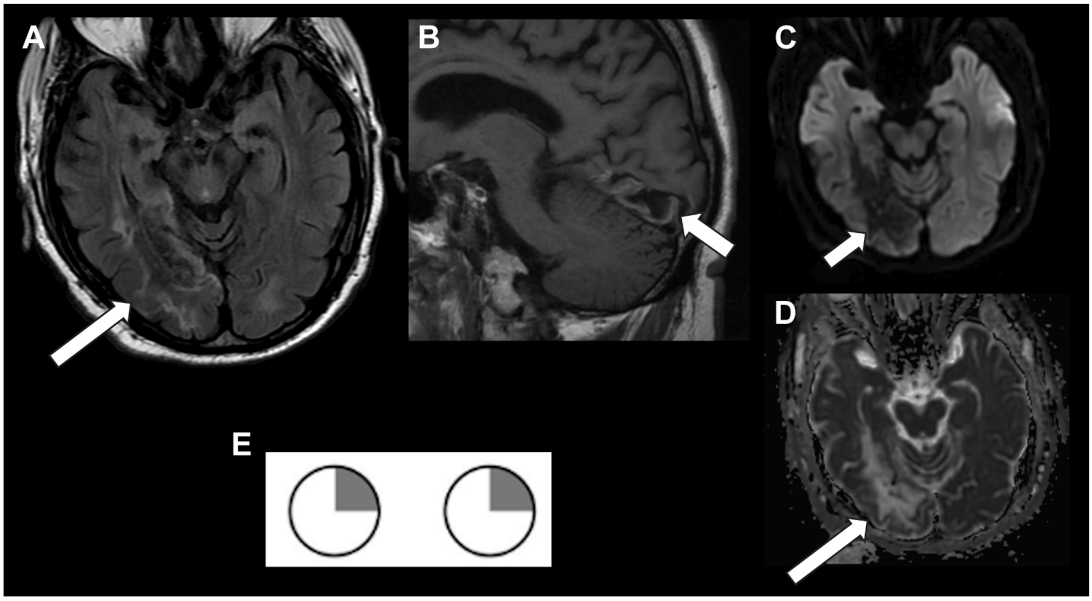

Fig. 11. Chronic infarct involving the right occipital lobe and associated visual field schematic. Axial FLAIR (*A*), Sagittal T1 (*B*), DWI (*C*), and ADC (*D*) images of a chronic right occipital infarct involving areas inferior to the calcarine fissure including the lingual and resulting in contralateral superior quadrantanopia (*E*). DWI, diffusion-weighted imaging.

Anton syndrome, a rare condition in which patients are unaware of their blindness and may confabulate visual experiences.[34,35] Lesions affecting the V4 area, responsible for color perception, result in central achromatopsia, a deficit in color discrimination that may leave motion and form perception largely intact. Damage to the V5/MT area, which is specialized for motion processing, produces akinetopsia, a rare condition characterized by the inability to perceive smooth motion, making moving objects appear to jump or blur.

Beyond these specialized regions, dysfunction in the higher-order visual pathways produces distinct cognitive and perceptual syndromes. Lesions in the dorsal stream, which extends from the occipital to the parietal cortex, can produce optical ataxia, impaired spatial orientation, and difficulties with visually guided movements, reflecting disruption of the brain's "where" pathway. Conversely, lesions in the ventral stream, projecting toward the temporal cortex, are associated with visual agnosia, including prosopagnosia, the inability to recognize familiar faces, and impairments in object recognition, reflecting damage to the "what" pathway.[29–33] These clinical syndromes not only illustrate the functional specialization of the occipital and adjacent cortical areas but also provide a critical framework for correlating lesion location with observed deficits in patients, enhancing both diagnostic precision and targeted therapeutic strategies.

Brainstem Visual Structures

- *Superior Colliculus (SC)*
  The SC is a midbrain structure mediating reflexive eye movements, visual attention, and saccades. It integrates retinal input and cortical feedback to coordinate rapid orienting responses.[36]
- *EW Nucleus*
  The EW nucleus is involved in parasympathetic control of the pupil and lens accommodation, forming part of the pupillary light reflex pathway.[37]
- *Pretectal Olivary Nucleus*
  The pretectal olivary nucleus plays a pivotal role in pupillary light reflexes. It receives direct retinal input and projects to the EW nucleus, mediating pupillary constriction.[38]

PATHOLOGY AND SYNDROMES

Infectious: Rhombencephalitis (commonly caused by Listeria monocytogenes), viral brainstem encephalitis, and fungal abscesses can affect cranial nerve nuclei and the medial longitudinal fasciculus (MLF), causing internuclear ophthalmoplegia, diplopia, and nystagmus.

Inflammatory: MS, neurosarcoidosis, and autoimmune encephalitis can produce demyelinating lesions of the MLF or cranial nerve nuclei,

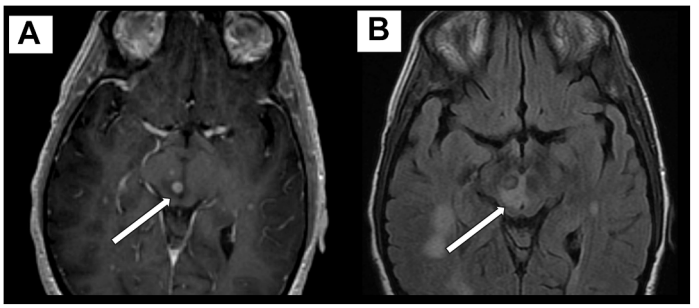

Fig. 12. Mid-brain metastasis. Axial T1-weighted imaging (*A*) showing enhancing right midbrain lesion (*white arrow*) and axial FLAIR imaging (*B*) showing surrounding edema (*white arrow*) involving areas of pupillary light reflex processing.

causing characteristic supranuclear ocular motor deficits.[27]

Neoplastic: Brainstem gliomas, metastases (**Fig. 12**), pineal region tumors, and posterior fossa masses may compress ocular motor centers, leading to Parinaud syndrome (upward gaze palsy, light–near dissociation, convergence-retraction nystagmus) or optical nerve dysfunction due to raised ICP.

Vascular: Brainstem infarcts (paramedian midbrain, pontine, medullary) and basilar artery thrombosis produce gaze palsies, diplopia, and occasionally cortical blindness if combined with occipital involvement.[39]

Metabolic: Diabetic microvascular ischemia may cause CN III palsy with *pupil-sparing*, while Wernicke's encephalopathy leads to ophthalmoplegia and nystagmus.

Neurodegenerative: Progressive supranuclear palsy and multiple system atrophy produce vertical gaze palsy, impaired smooth pursuit, and blepharospasm.[40]

VISUAL IMPLICATIONS

- *Pupillary reflexes:* EW nucleus lesions result in efferent defects; optical nerve lesions result in relative afferent pupillary defect.
- *Light–near dissociation*: Pretectal/dorsal midbrain lesions (Parinaud) demonstrate impaired light reflex but preserved near response.
- *Field defects*: Brainstem lesions rarely cause direct homonymous hemianopia but can produce secondary optical atrophy via chronic papilledema.

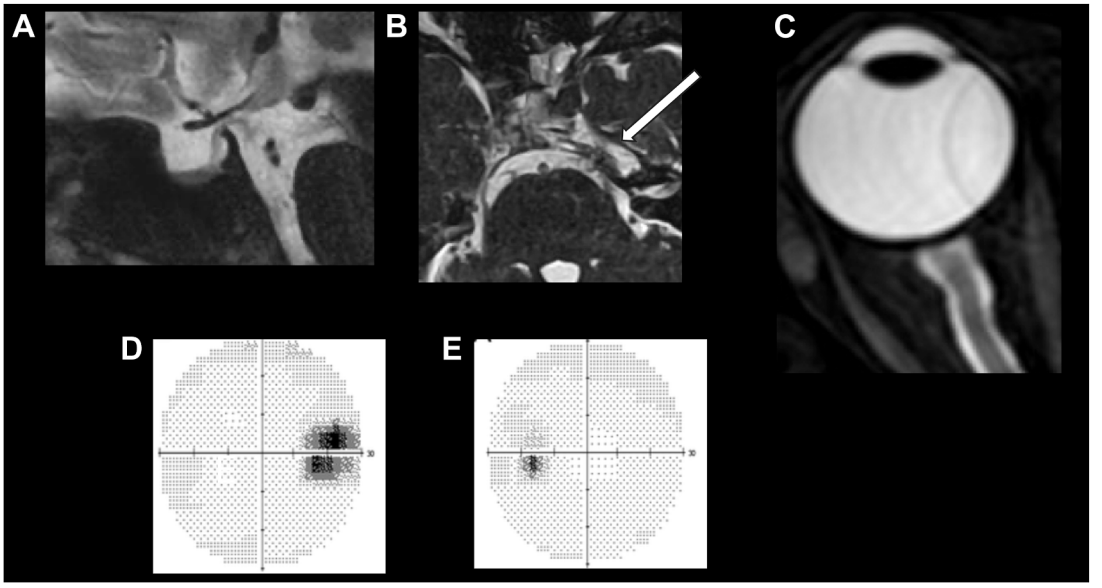

Fig. 13. IIH characteristic imaging findings and example of early visual field defect on perimetry. Enlarged partially empty sella (*A*), prominent Meckel's cave (*B*) and tortuous optical nerve with prominent CSF along the optical nerve sheath (*C*) are imaging features of IIH. Example of initial visual field defect in IIH with an enlarged blind spot on perimetry (*D*) in comparison with a normal sized blind spot on the contralateral eye (*E*). IIH, idiopathic intracranial hypertension.

Multiple Intracranial Regions: Idiopathic Intracranial Hypertension

Background

IIH predominantly affects women of childbearing age and is characterized by elevated ICP without mass lesions. Multiple intracranial compartments are affected, including orbital structures, the optical nerve, skull base foramina, and venous sinuses.[41]

Mechanism of vision loss

- *Axoplasmic flow stasis:* Elevated ICP leads to papilledema and retinal ganglion cell injury.
- *Chronic* optical *nerve damage:* Inferonasal and inferotemporal optical nerve fiber loss leads to visual field constriction.
- *Visual field patterns:* Initial defects include enlarged blind spot and inferior nasal step; later stages may show arcuate defects and generalized constriction, ultimately leading to central scotomas or total blindness if untreated.[42]

Clinical correlation

Early recognition of IIH is critical to prevent irreversible vision loss. Imaging may reveal empty or partially empty sella, optical nerve sheath enlargement, protrusion of the optical nerve head into the globe, Meckel's cave enlargement and transverse sinus stenosis (**Fig. 13**).[43,44]

SUMMARY AND FUTURE DIRECTIONS

Visual dysfunction in CNS disease is highly dependent on lesion location, extent, and underlying pathophysiology. A precise understanding of neuroanatomy allows clinicians to correlate clinical visual findings with neuroimaging, thereby guiding urgent interventions and surgical planning. Advances in imaging modalities, including high-resolution MR imaging and diffusion tensor imaging, enhance the detection and mapping of visual pathways, improving patient outcomes.

Future research may focus on integrating *functional neuroimaging* with structural mapping to predict visual recovery, optimize surgical approaches, and develop targeted therapies for neurodegenerative and demyelinating diseases affecting vision.

CLINICS CARE POINTS

- Visual field defects provide critical clues to lesion localization along the visual pathway, and correlation with imaging findings helps narrow the differential diagnosis.
- NMRI is the imaging modality of choice for evaluating visual pathway pathology, with high-resolution sequences enabling assessment of the optic nerves, chiasm, optic radiations, and occipital cortex.
- Diffusion-weighted imaging and diffusion tensor tractography can improve detection and characterization of optic radiation involvement, particularly in infiltrative, demyelinating, or surgical planning contexts.
- A wide spectrum of diseases—including infectious, inflammatory, neoplastic, vascular, metabolic, neurodegenerative, congenital, and traumatic disorders—can affect the visual pathways and should be systematically considered when interpreting imaging findings in patients with visual symptoms.
- Early radiologic recognition of visual pathway abnormalities can guide timely clinical management and help prevent permanent visual deficits.

DISCLOSURE

The authors have nothing to disclose.

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