Imaging of Optic Neuropathy and Chiasmal Syndromes

Bundhit Tantiwongkosi, MD[a,b,c,*], Mahmood F. Mafee, MD[d]

KEYWORDS

• Vision loss • MR imaging • Optic neuropathy • Chiasm • Visual field defect

KEY POINTS

- When clinical presentations of optic neuropathy and chiasm disorders are nonspecific, imaging plays a critical role in detecting, localizing, and differentiating the wide variety of causes.
- Some key imaging features are characteristic, leading to appropriate treatment and preventing unnecessary tissue biopsies.
- The following entities have characteristic imaging findings: optic neuritis, optic nerve sheath meningioma, optic nerve glioma, pituitary adenoma, aneurysm, etc.

INTRODUCTION

Optic neuropathy is characterized by loss of visual acuity, color vision (dyschromatopsia), and visual field defect. Fundoscopic examination may reveal a swollen, pale, anomalous, or normal optic disc.[1] Chiasmal disorders classically present with gradual onset of vision loss, bitemporal hemianopsia, and occasionally, endocrinopathy if the pituitary gland and/or hypothalamus are the causes or are involved.[2]

NORMAL ANATOMY

Optic Nerve

Retinal ganglion cell axons form the optic nerves, which are myelinated by oligodendrocytes and supported by astrocytes.[3] The optic nerve is divided into 4 segments: intraocular, intraorbital, intracanalicular, and intracranial (**Fig. 1**).[4] The optic nerve is isointense to cerebral white matter on T1-weighted and T2-weighted images. The optic nerve is surrounded by the optic nerve sheath, which contains pia, cerebrospinal fluid, arachnoid, and dura.[1]

Optic Chiasm

The nasal retinal fibers of the optic nerve cross at the optic chiasm, whereas the temporal fibers remain uncrossed (**Fig. 2**). The chiasm is surrounded by multiple critical structures including the pituitary gland inferiorly, infundibulum posteriorly, third ventricle and hypothalamus superiorly, inferior frontal lobe anteriorly, and supraclinoid internal carotid artery laterally.[2] The chiasm (**Fig. 3**) is typically located directly above the pituitary gland; in a minority of cases, it may be in prefixed (above the tuberculum sella) or postfixed positions (above the dorsum sella).[5] Mass lesions of the pituitary gland typically compress the central portion of chiasm resulting in classic bitemporal hemianopsia, which may not present in cases of prefixed or postfixed chiasms.[2]

Disclosures: None.

[a] Division of Neuroradiology, Department of Radiology, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, Mail Code 7800, San Antonio, TX 78229, USA; [b] Department of Otolaryngology–Head Neck Surgery, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, Mail Code 7800, San Antonio, TX 78229, USA; [c] Imaging Service, South Texas Veterans, 7400 Merton Minter, San Antonio, TX 78229, USA; [d] Division of Neuroradiology, Department of Radiology, University of California, San Diego, 200 West Arbor Drive, San Diego, CA 92103, USA

* Corresponding author. Division of Neuroradiology, Department of Radiology, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, Mail Code 7800, San Antonio, TX 78229.
E-mail address: tantiwongkos@uthscsa.edu

Neuroimag Clin N Am 25 (2015) 395–410
http://dx.doi.org/10.1016/j.nic.2015.05.004
1052-5149/15/$ – see front matter Published by Elsevier Inc.

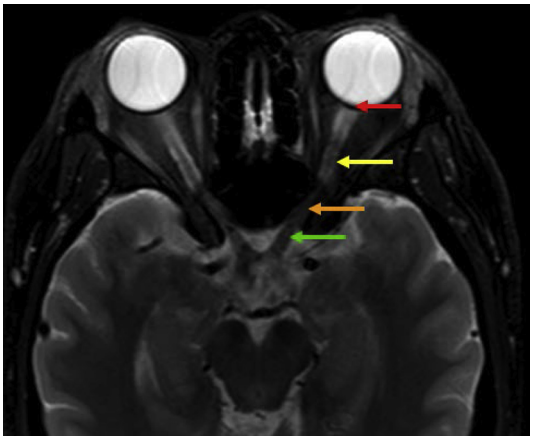

Fig. 1. The 4 segments of optic nerves. Axial T2-weighted image shows intraocular (*red arrow*), intraorbital (*yellow arrow*), intracanalicular (*orange arrow*), and intracranial (*green arrow*) segments.

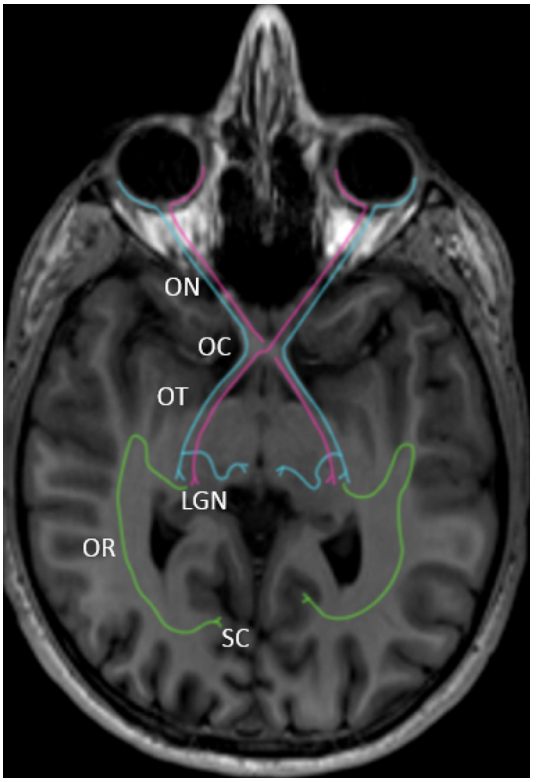

Fig. 2. Afferent visual pathways. Lines are drawn superimposed on an axial T1-weighted image showing expected location of the optic nerves (ON), optic chiasm (OC), optic tract (OT), lateral geniculate nucleus (LGN), optic radiation (OR), and striate cortex (SC).

IMAGING PROTOCOL

Computed Tomography Versus MR Imaging

Computed tomography (CT) and MR imaging are complementary to each other in identifying anatomy and pathology of optic nerve and chiasm.[6] MR imaging provides exquisite details of optic nerve and chiasm because of high tissue contrast differentiation.[7] CT is valuable in the evaluation of bony structures (optic canals), calcified lesions, and metallic foreign bodies when MR imaging is contraindicated.[6]

MR Imaging Protocol

Optic nerve and sella MR imaging protocols are suggested in **Box 1**. Fat saturation sequences with intravenous gadolinium are helpful in detecting optic neuritis, neoplasms, postoperative changes, and optic nerve infarction.[8,9]

PATHOLOGY

Optic Neuropathy

Optic neuritis

Optic neuritis is a clinical diagnosis of optic nerve inflammation secondary to demyelination. A typical clinical scenario is a young white woman presenting with rapid onset of painful vision loss with optic disc swelling seen on fundoscopy.[1] In cases with typical presentations, MR imaging does not aid in the diagnosis and does not alter the clinical course, management, or final visual outcome.[10] If performed, MR imaging may show enlargement and enhancement of the nerve with hyperintensity on T2-weighted images in over 90% of cases (**Fig. 4**A–D).[11] The findings are often similar to those of other inflammatory (eg, sarcoidosis) and infectious causes of optic neuropathy. However, involvement of the optic nerve, optic nerve sheath, and ciliary body favors the diagnosis of sarcoidosis over optic neuritis associated with multiple sclerosis (MS). Visual outcome is worse with longitudinally extensive lesions and with involvement of the intracanalicular segment (rigid bony canal).[12] Eye pain correlates with intraorbital segment involvement, which is the most common segment to be involved.[12] Enhancement suggests active disease, while resolution of enhancement is seen in the recovery phase.[1]

Patients with optic neuritis are at increased risk for developing MS; it can occur as the first presentation of MS or develop later on.[13] The presence of abnormal cerebral white matter lesions on MR imaging is the most important predictor for the development of MS in the future.[1] It was found that 60% to 90% of patients with optic neuritis with abnormal cerebral white matter eventually develop MS. Only 20% to 25% of patients with normal-appearing MR