Diplopia: What to Double Check in Radiographic Imaging of Double Vision

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KEYWORDS

• Diplopia • MR imaging • Computed axial tomography

KEY POINTS

- Binocular diplopia may be caused by life-threatening causes requiring careful neuroimaging in patients who have new onset, progressive symptoms, more than one symptom, or history of neoplasm.
- In patients with a new onset ptosis and binocular diplopia, a careful assessment of the vasculature adjacent to cranial nerves III, IV, and VI is needed to exclude an aneurysm.
- An awareness of the radiographic anatomy of cranial nerves III, IV, and VI from their respective nuclei, cisternal and cavernous segments, terminal innervation, and connective pathways is helpful in assessing imaging for binocular diplopia.

Diplopia or "double vision" comes from the Greek terms "diplous" meaning double and "ops" for eye. Diplopia is distressing for patients and may occur from an extensive list of causes. Because certain causes may be life threatening, patients with diplopia require an accurate clinical physical assessment, and in certain cases, a careful radiographic review. Patients with diplopia are often first evaluated by a neurologist or ophthalmologist, who determines whether the diplopia is "monocular" or "binocular." If the patient has "monocular" diplopia, this means they see double with only one eye open. In monocular diplopia, doctors and patients can usually breathe a sigh of relief because causes are often related to eye issues from refractive difficulties, poor glasses, dry eyes, uveitis, or cornea warping, and radiographic imaging may not be required.[1–3]

However, in "binocular" diplopia, the patients see double with both eyes open. Binocular diplopia requires physicians and radiologists assessing these patients to be on high alert and double check everything, including history and images, because these life-threatening causes need to be excluded in the myriad of possible causes. So, when should radiographic studies be obtained in patients with binocular diplopia? Previous general guidelines for imaging patients with binocular diplopia included new onset diplopia in a patient less than 50 years old, presence of more than one neurologic symptom, or a progressive course or history of cancer[1,2]; an easy way to remember it is the rhyme, "In diplopia, if the patient is young, and symptoms are progressing more than one, then neuroimaging should be done!"

Disclosures: Consultant Primal Pictures 3D Anatomy; Grant Funding: Idiopathic Intracranial Hypertension Foundation, RTOG: Radiation Therapy Oncology Group 3504 (C.F.E. Kirsch). K. Black has nothing to disclosure.
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Radiol Clin N Am 55 (2017) 69–81
http://dx.doi.org/10.1016/j.rcl.2016.08.008

Recent studies, however, have shown that the guidelines for not imaging a patient regardless of age, elderly or not, with a monocular palsy of cranial nerve (CN) III, IV, or VI, are less clear and controversial. These publications advocate that imaging, including a contrast-enhanced MR imaging, may be necessary for all patients presenting with an oculomotor neuropathy resulting in a diplopia.[3] Therefore, a better rhyme to remember is, "If a patient is binocular seeing more than one, than accurate neuroimaging should be done!" Before imaging, a thorough physical examination is essential, especially if patients complain of tiredness at rest and have diplopia. In these cases, myasthenia gravis needs to be excluded, and a good second rule of thumb is, "If the diplopia occurs at rest, also do a tensilon test."

Because an accurate imaging assessment is imperative in certain life-threatening causes of diplopia, understanding the pertinent anatomic pathways of CN III, IV, and VI, including the paramedian pontine reticular formation (PPRF) and medical longitudinal fasiculus (MLF) for lateral gaze, is invaluable.[4] This article presents a few key examples of critical anatomy, abnormality, and radiographic findings affecting these nerves from the cranial nuclei to their distal innervation. A complete list of causes and radiographic findings for binocular diplopia is extensive and beyond the scope of this article.

This article's main focus (pardon the pun) is to present the pertinent anatomy and critical abnormality radiologists should double check on imaging using the acronym, VISION - including the *V*essels, *I*nfection or Inflammation, *S*kull base, *S*uperior orbital fissure, and not forgetting the *S*calp for giant cell temporal arteritis, *I*ncreased *I*ntracranial pressure, *O*nset of new or worst headaches of life, or *O*nset new psychosis, and *N*eoplastic, all of which may cause binocular diplopia and need to be excluded to reduce morbidity and mortality.

ORBITAL ANATOMY

Double checking the course of the nerves involved in orbital imaging requires an awareness of the radiographic course of the cranial nerves, CN III, CN IV, and CN VI, from the brainstem, subarachnoid space, cavernous sinus, superior orbital fissure, and orbit. In addition, a lack of coordinated eye movements may cause diplopia if there is abnormality affecting thePPRF coordinating CN III and CN VI via ascending fibers of the MLF for lateral gaze.[4] Therefore, disruptions of the medial longitudinal fasciculus by upper motor neurons or any other cause can cause diplopia.[1–4] Remembering the orbital cranial nerve muscle innervation is easy with the chemical formula LR_6 SO_4 and all the rest are 3, meaning the lateral rectus muscle is innervated by CN VI the abducens nerve, the superior oblique by CN IV, the trochlear nerve, and remaining orbital musculature by CN III the oculomotor nerve.[5,6] **Fig. 1** is a sagittal T1 MR image, delineating the location of CN III, IV, and IV in the brainstem.

The oculomotor nerve or CN III is a somatic motor nerve with efferent fibers supplying the levator palpebrae superioris, superior, inferior, medial, and lateral rectus and inferior oblique muscles, and a visceral motor efferent with parasympathetic supply constricting the pupil and ciliary muscles, via the ciliary ganglion.[3,5,6] The combined somatic motor fibers and parasympathetic fibers form the CN III oculomotor nerve as it leaves the brainstem.

As demonstrated in **Fig. 2**A, the CN III nucleus somatic motor component is "V" shaped and is located in the midbrain at the level of the superior colliculus, just anterior to the cerebral aqueduct, with the medial longitudinal fasciculus as its neighbor laterally and inferiorly. In the brainstem, the oculomotor complex is composed of lateral subnuclei with the posterior component supplying the *ipsilateral* inferior rectus, the intermediate nucleus supplying the inferior oblique, and the anterior ventral nuclei supplying the medial rectus muscles. The medial subnucleus gives supply to the *contralateral* superior rectus, and the central

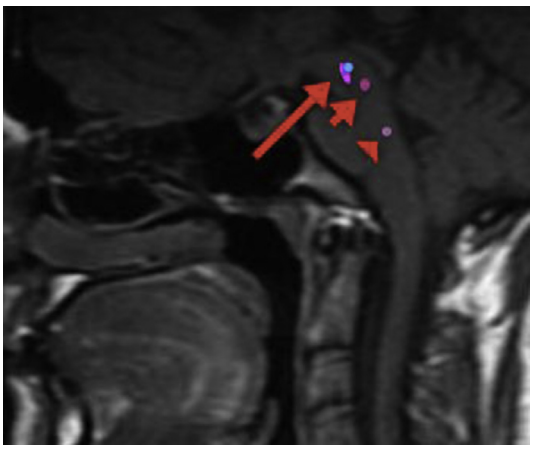

Fig. 1. Sagittal T1-weighted MR imaging with the large arrow showing the location of the nucleus for the oculomotor nerve CN III, including both the visceral motor Edinger-Westphal nucleus posteriorly in blue that innervates the parasympathetics for the pupil constrictor muscles and ciliary muscles, and the anterior pink somatic nucleus. The short arrow demarcates the trochlear CN IV nucleus located below the CN III nucleus, and the arrowhead demarcates the lateral abducens CN IV nucleus.