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!"

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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

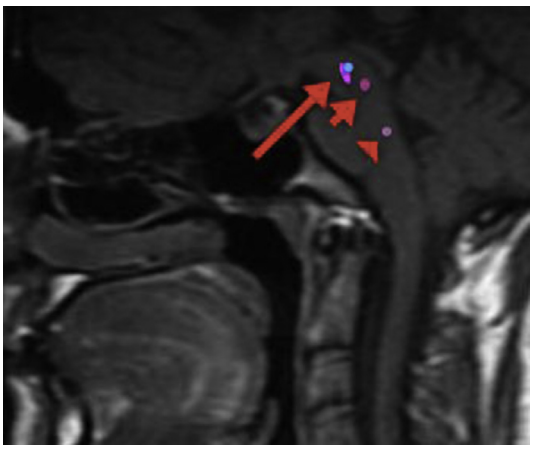


Fig. 1. Sagittal T1-weighted MR imaging with the large arrow showing the location of the nucleus for the oculomotor nerve CN III, including both the visceral motor Edinger-Westphal nucleus posteriorly in blue that innervates the parasympathetics for the pupil constrictor muscles and ciliary muscles, 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.

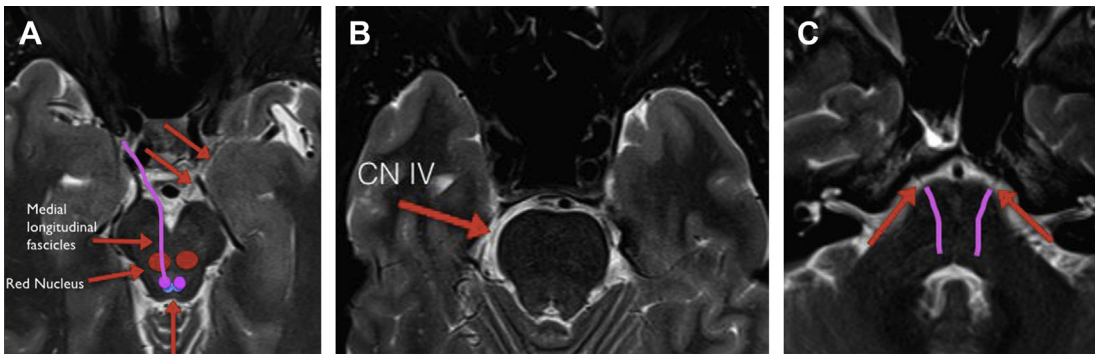


Fig. 2. The course of the cranial nerves. (*A*) Axial T2 MR imaging at the level of the CN III oculomotor nucleus demonstrates the course of CN III from its nucleus, through the medial longitudinal fasciculus, and into the interpeduncular cistern as it extends underneath the posterior communicating artery. Red arrows are the cisternal segment of CN III, pink line is the intracranial course of CN III, red circles are the red nuclei, pink circles are the CN III nuclei, the tiny blue dot is the CN III Edinger-Westphal nucleus. (*B*) Axial T2 MR imaging taken a slice below (*A*), demonstrating the trochlear nerve CN IV, whose nucleus is located at the midbrain tegmentum near the midline anterior to the cerebral aqueduct. CN IV decussates exiting dorsally, going around the cerebral peduncles marked by the red arrow and with CN III going between the PCA and SCA, underneath the posterior communicating artery and then piercing the tentorial free and attached border to enter the cavernous sinus located below CN III. (*C*) Axial T2 MR image of pons at the level of the fourth ventricle. The expected course of CN VI (*pink lines*) is shown with the red arrows pointing to the CN VI cisternal segments in the prepontine cistern, where it then pierces the dura and extends superiorly.

subnucleus gives supply to the bilateral levator palpebrae superiorus muscles, with the Edinger-Westphal (visceral motor) nucleus located posteriorly, giving rise to the parasympathetic fibers.

The CN III axons for the lower motor neurons extend anteriorly through midbrain tegmentum, through the red nucleus, emerging in the medial interpeduncular cistern where the pons and midbrain merge. CN III runs between the superior cerebellar artery (SCA) and posterior cerebral artery (PCA), just below the posterior communicating artery. CN III enters the dura into the cavernous sinus, running along the superior lateral wall above the trochlear nerve. The CN III nerve exits via the superior orbital fissure into the tendinous ring, and entering the orbit, divides into superior and inferior components. The superior nerve of CN III goes upward and lateral to the optic nerve supplying the superior rectus and levator palpebrae. The inferior CN III separates into 3 parts, supplying the inferior rectus, and medial rectus muscles along the medial ocular margin and the inferior oblique muscle along its posterior aspect.[3,5]

The Edinger-Westphal visceral motor neurons course along with the somatic motor axons, from the middle cranial fossa, cavernous sinus, and superior orbital fissure. The nerves separate from the neural components supplying the inferior oblique muscle and terminate in the ciliary ganglion at the apex of the orbital cone. Exiting from the ciliary ganglion are short ciliary nerves that join with sympathetic fibers from the internal carotid artery (ICA), that enter into the globe at the posterior margin near the optic nerve. These nerves control the constrictor pupillae muscles and ciliary muscles and run in between the sclera and choroid of the eye ending up in the ciliary body and iris of the globe where the fibers control pupil size and lens shape.[6]

As noted in **Fig. 2**B, CN IV, the trochlear nerve, is smallest cranial nerve, with the longest intracranial course. CN IV is a somatic motor nerve only innervating the superior oblique muscle, with the nucleus located in the midbrain tegmentum below CN III, in the inferior colliculus.[5,6] Most motor neurons are located medially as is the nucleus, located just ventral to the cerebral aqueduct. CN IV is unique because it is the only nerve exiting from the back (dorsal) brainstem and crossing to the opposite side. Because CN IV decussates or crosses from the brainstem, each superior oblique muscle is innervated by the trochlear nucleus located in the contralateral brainstem. CN IV runs with CN III between the PCA and SCA arteries, along the margin of the free and attached margin of the tentorium cerebelli. CN IV runs below CN III in the cavernous sinus, and through the superior orbital fissure crossing diagonally across the levator palpebrae and superior rectus muscle to the superior oblique muscle.[6] When CN IV is not functioning, the CN III and CN VI take over the globe,

and the globe is rotated outwardly by abduction from CN VI and inferiorly from unopposed action of the muscles innervated by CN III.[3,5] Therefore, this is sometimes referred to as the "bum's muscle" because a lack of a functioning CN IV has you looking down and out.

As shown in **Fig. 2**C, the abducens nerve CN VI is a somatic efferent nerve to the lateral rectus muscle. The CN VI nucleus is in the pontine tegmentum, and like additional somatic motor nuclei, is located closer to the midline anterior to the fourth ventricle. As the seventh cranial nerve (CN VII) circles over the sixth nerve nuclei, it creates a little bump or hill, "colliculus" along the fourth ventricular anterior margin. The CN VI emerges where the pons and medullary pyramid meet, extends through the prepontine cistern entering the dura lateral to the dorsum sella, and then travels through the petroclival confluence (Dorello canal) under the petrosphenoidal "Gruber ligament."[7] It is important to be aware of the actual course of CN VI and the true definition of the "Dorello canal." The course of CN VI is well outlined by Umansky and colleagues,[8] who note 3 curves. The first curve of CN VI is at the dural foramen where CN VI curves upward to lateral to the petrous apex; the second curve of CN VI is over the petrous apex where CN VI must angle inferiorly and laterally to reach the posterior aspect of the cavernous ICA, and the third curve as CN VI extends around the posterior ICA and then runs next to the vessel in the medial cavernous sinus (**Fig. 3**).[7–9] In Dorello's original 1905 paper, he defined the canal as the "Triangular space bordered by Gruber's superior petrosphenoidal ligament (PSL) superolaterally, superior clivus inferiorly, petrous apex inferolaterally. The space at the 2nd bend of the CN VI, with CN VI always below Gruber's ligament and containing the ostium of the inferior petrosal sinus (IPS) within the canal."[7,8] However, this definition has many problems, as modern microneurosurgery and imaging demonstrate. First, there are no bony margins like the optic canal! Second, since 1991, studies demonstrated that CN VI can go above Gruber ligament.[7] On thin-section MR imaging, follow this nerve closely and you may notice this finding as well! Third, in microneurosurgery, it is noted that the ostium of the IPS is outside of this space; therefore, more recently, clinicians have referred to this space as the "petroclivus venous confluence."[7–9]

The extension of the CN VI through the petrovenous confluence (Dorello canal) over the petrous ridge is where CN VI is at highest risk for injury. The risk of injury is greater partly because CN VI is tethered inferiorly entering the dura and

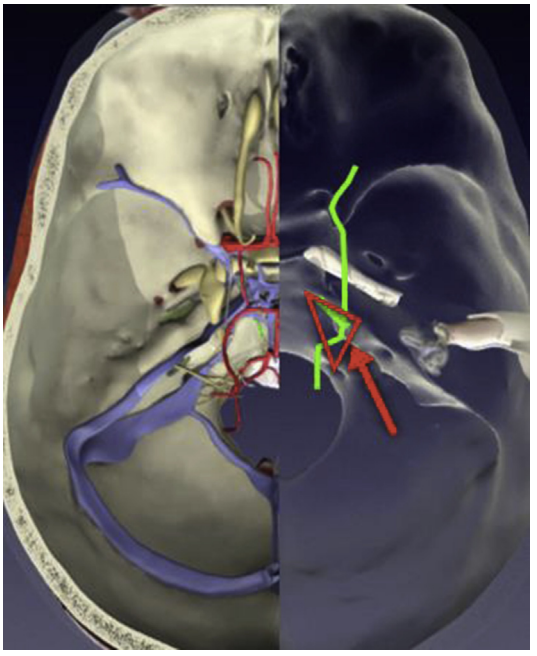


Fig. 3. The course of CN VI (*green line*) through the petroclival confluence or "Dorello canal" marked by the triangle in red. The arrow is pointing to the location of Dorello's canal. (*From* Berkovitz B, Kirsch C, Moxham B, et al. 3D head & neck anatomy with special senses and basic neuroanatomy [DVD]. Primal Pictures; 2007; with permission.)

superiorly as it enters the cavernous sinus. Therefore, the curved segment of the nerve extending over the petrous apex of the temporal bone is the site most at risk to trauma, infection, or increased intracranial pressure.[7–9] After CN VI extends over the petrous apex, CN VI, into the cavernous sinus, it is the most medial of the cranial nerves, next to the ICA, as shown in **Fig. 4**. The CN VI then extends into the superior orbital fissure. In patients with a lower motor neuronal lesion, the lateral rectus muscle is denervated, and the patient cannot abduct the globe laterally; the globe is pulled medially due to the unopposed action of CN III on the medial rectus muscle.

What to Double Check Using the Acronym VISION

In assessing patients with binocular diplopia, it is helpful to have a checklist to rule out the most critical causes. Using the acronym VISION is a good way to double check for abnormality causing double vision: *V*asculature, *I*nfectious or *I*nflammatory, remembering to check the *S*calp for a giant cell arteritis, *S*kull base and *S*uperior orbital fissure in trauma, *I*ncreased *I*ntracranial pressure, *O*nset of new or worst headaches, or *O*nset new psychosis,

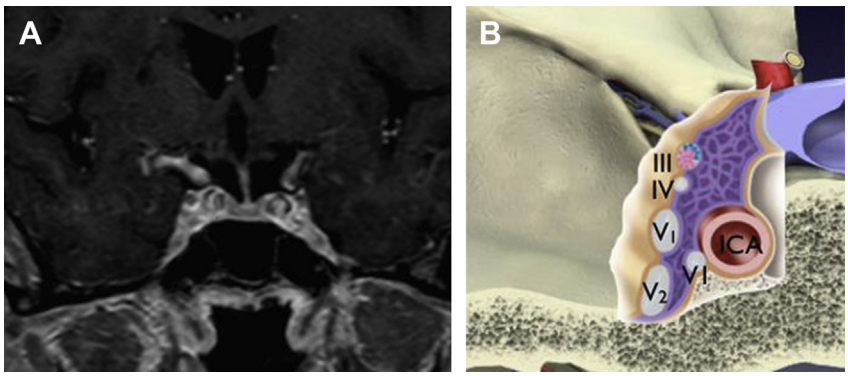


Fig. 4. (*A*) Coronal T1 postgadolinium through the cavernous sinus. The cranial nerves are identified as the foci of nonenhancement within the enhancing cavernous sinus. (*B*). Schematic with cranial nerves labeled lateral to the ICA. (*From* Berkovitz B, Kirsch C, Moxham B, et al. 3D head & neck anatomy with special senses and basic neuroanatomy [DVD]. Primal Pictures; 2007; with permission.)

and *N*eoplastic in determining causes of binocular diplopia.

V: vasculature

In patients who present with a drooping eyelid (ptosis), binocular diplopia, and a poorly reacting pupil, an aneurysm needs to be excluded. A missed aneurysm that may eventually rupture with subarachnoid hemorrhage may have a mortality of up to 50%.[10] The clinical findings of diplopia and ptosis are important clues to the presence of an aneurysm, because CNs III and IV run directly between the PCA and SCA and underneath the posterior communicating artery (Pcom). In addition, these CNs run close together into the cavernous sinus with the ICA located medially to CN VI. Aneurysms compressing the cranial nerves may lead to denervation atrophy of the orbital musculature resulting in a binocular diplopia (**Fig. 5**A–G). Because the pupillary response may be difficult to assess, and loss of CN III results in a ptosis, a good rule to follow is "A new ptosis is a PCom aneurysm until proven otherwise."

In elderly patients or patients with diabetes, strokes or vascular lesions leading to hemorrhage involving the brainstem may present with a diplopia (**Fig. 6**A, B). A computed tomographic (CT) scan is helpful to assess for hemorrhage, and a diffusion weighted imaging (DWI) MR imaging may help delineate new infarcts; vasculature can be assessed either via computed tomographic angiography (CTA) or magnetic resonance angiography (MRA).

In addition to aneurysms at risk for rupture, life-threatening vascular causes that may cause diplopia include vascular malformations, dissections, strokes, and arteritis, such as giant cell arteritis. When viewing the study, double check the course of the CNs III, IV, and VI, and adjacent vessels and nuclei for strokes of the basal midbrain (see **Fig. 6**; **Fig. 7**). Strokes of the midbrain are often associated with other symptoms, including a contralateral hemiplegia due to the adjacent corticospinal tract fibers, or if in the red nucleus, an ipsilateral ophthalmoplegia and contralateral intentional tremor.[3,4]

As a brief review, CNs III, IV, and VI are at risk from an aneurysm from adjacent vessels resulting in a possible denervation atrophy of the corresponding musculature; this means double checking the posterior communicating artery, PCA, SCA, or ICA. In patients with a ptosis with the double vision, pay close attention to the Pcom, as an aneurysm from this vessel may cause a ptosis by the aneurysm compressing the CN III with denervation of the levator palpebrae superiorus and continued action of CN VII on the orbicularis oculi.

In addition to aneurysms, double check the basilar and posterior inferior cerebellar arteries for thrombus or increased attenuation of the vessels on CT and lack of flow on MR imaging, MRA, or CTA. Be on the lookout for dissections, which may lead to a stroke in the pons or midbrain (see **Fig. 7**). If there is abnormal increased attenuation within the basilar artery (thrombus), this may occlude the pontine arterial branches, resulting in a pontine infarct. The visual complex coordination is controlled by thePPRF coordinating CN III and CN VI via ascending fibers of the MLF for lateral gaze. Pontine infarcts in this region may result in paralysis of conjugate lateral gaze. Lesions along the MLF can result in lateral gaze problems and nystagmus due to involvement of vestibular-oculomotor fibers.[2–4,11]

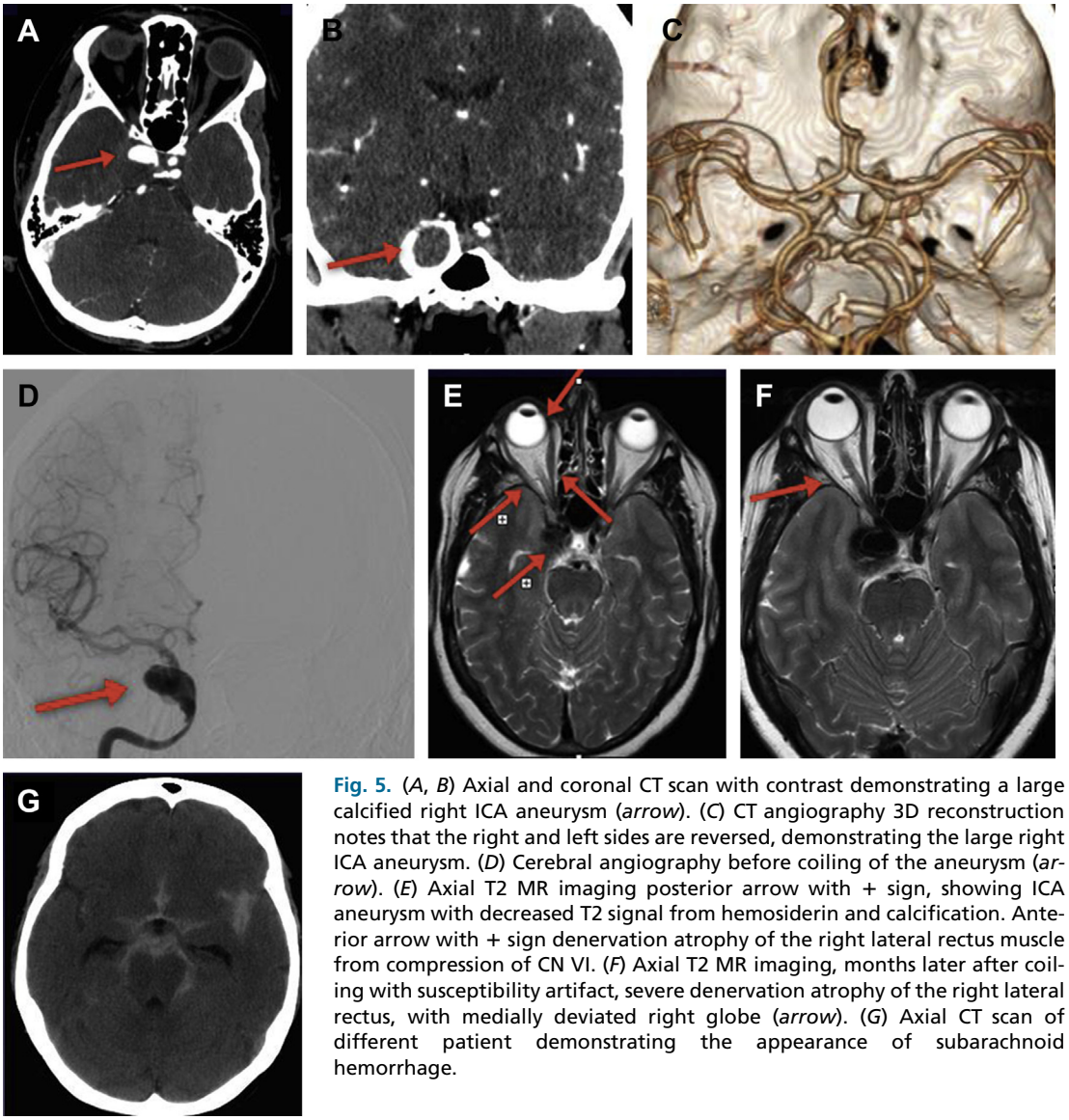


Fig. 5. (*A*, *B*) Axial and coronal CT scan with contrast demonstrating a large calcified right ICA aneurysm (*arrow*). (*C*) CT angiography 3D reconstruction notes that the right and left sides are reversed, demonstrating the large right ICA aneurysm. (*D*) Cerebral angiography before coiling of the aneurysm (*arrow*). (*E*) Axial T2 MR imaging posterior arrow with + sign, showing ICA aneurysm with decreased T2 signal from hemosiderin and calcification. Anterior arrow with + sign denervation atrophy of the right lateral rectus muscle from compression of CN VI. (*F*) Axial T2 MR imaging, months later after coiling with susceptibility artifact, severe denervation atrophy of the right lateral rectus, with medially deviated right globe (*arrow*). (*G*) Axial CT scan of different patient demonstrating the appearance of subarachnoid hemorrhage.

In binocular diplopia with a small pupil (miosis) and ptosis and anhydrosis (lack of sweating on the same side), be on high alert for Horner syndrome and assess the vessels for a dissection (see **Fig. 7**A, B). Diplopia and ptosis are like the old ads for potato chips, stating, "You can't have just one." If there are multiple findings with diplopia, this is a strong indicator to double check for the underlying abnormality.[2–4,11]

Last, in patients who have experienced a rapid alteration in venous pressure affecting the superior orbital venous vasculature or external bleeds, make sure there are no compressive masses along the orbital cone (**Fig. 8**).

I: inflammatory and infectious causes

Infectious, inflammatory, or neoplastic leptomeningeal processes and increased intracranial pressure can affect cranial nerves as well as temporal lobe uncal herniation, which displaces the cerebral peduncle to the contralateral side, distorting CN III along the tentorial notch. Pay careful attenuation to patients who are at increased risk for infections, including embolic causes in patients who are immunocompromised, on dialysis, or at risk for septic emboli. In dialysis patients who cannot have contrast due to elevated creatinine, a noncontrast MR imaging with a DWI may be useful to help identify abscesses, which demonstrate

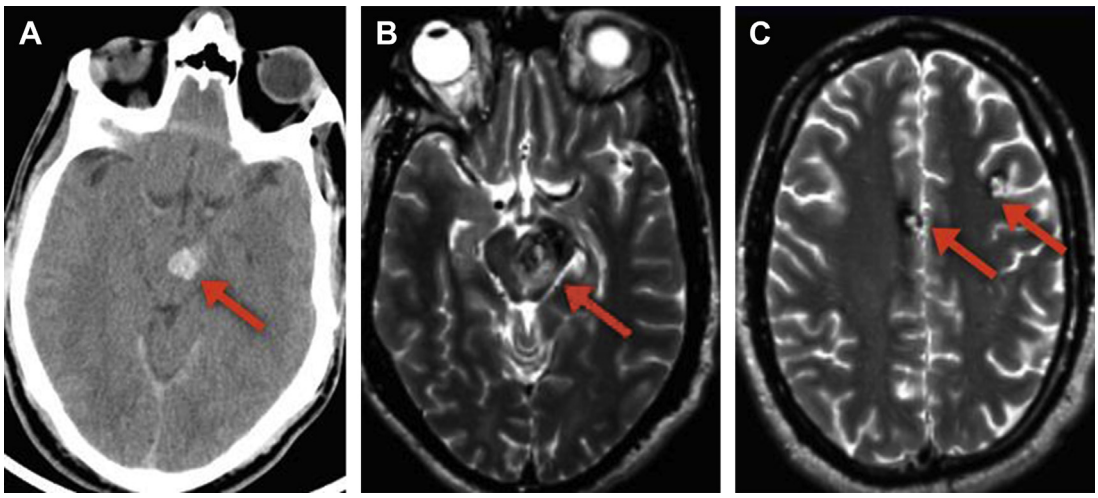
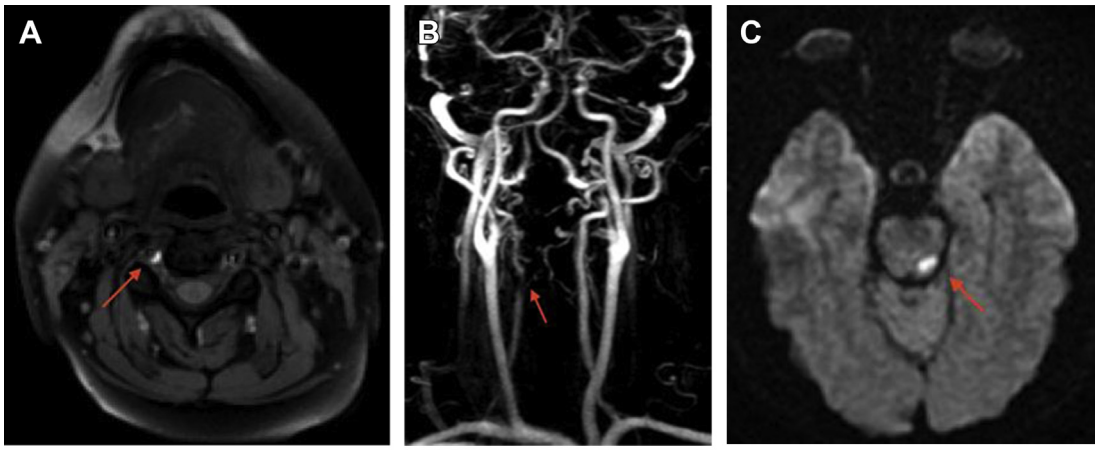


Fig. 6. A 63-year-old man presented with new onset binocular diplopia. (*A*) Axial CT scan with arrow showing intraparenchymal hemorrhage in the midbrain. (*B*) Corresponding axial T2 MR imaging demonstrates lesion with surrounding edema. (*C*) Axial T2 MR imaging of same patient with arrows demonstrating small foci of hyperintense T2 signal with surrounding hemosiderin in patient with multiple cavernous angiomas.

restricted diffusion on the acute diffusion coefficient images (ADC) and bright signal on the DWI, as demonstrated in **Fig. 9**. In patients with sinus infections at risk for extension, look at the cavernous sinus carefully for signs of infection and possible thrombosis, as in **Fig. 10**. Inflammatory causes such as Guillain-Barré or Miller Fisher variants can be life threatening, and additional



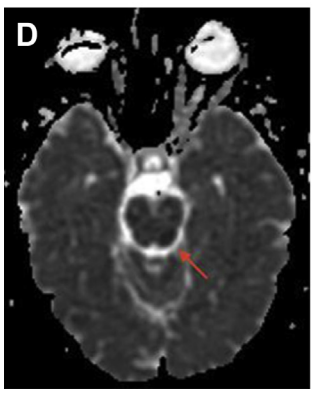


Fig. 7. (*A*) Axial T1 postgadolinium fat-saturated T2 MR imaging with arrow noting right vertebral artery dissection in 42-year-old woman presenting with sudden onset diplopia. (*B*) Postcontrast time-of-flight 3D MRA with arrow pointing to right vertebral artery dissection. (*C*, *D*) Axial DWI and ADC MR imaging with bright DWI signal and restricted diffusion from acute (*arrow*) stroke in the left inferior colliculus.

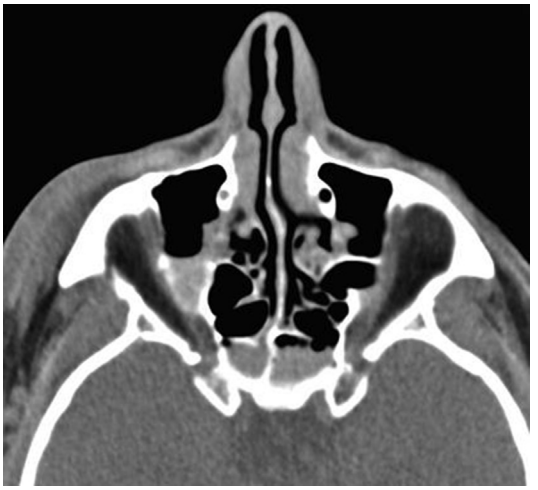


Fig. 8. Traumatic–axial noncontrast CT through the facial bones in a patient status post motor vehicle accident with fracture deformity through the right superior orbital fissure resulting in a binocular diplopia, with hemorrhagic air fluid levels in the sphenoid sinus.

inflammatory changes from demyelination if involving the nuclei will affect the cranial nerves, as in **Fig. 11**. Remember, if ptosis is combined with other deficits, it is like a dangerous bag of potato chips, "you can't have just one"; therefore, look carefully for *I*nvolvement of other ocular findings with the diplopia.[1,3,4,11,12] Demyelinating disease, in particular, may be located at multiple sites, requiring assessment of both the brain and the spinal cord (see **Fig. 11**).

Additional inflammatory causes, including tumefactive inflammatory fibrosis, Tolusa-Hunt, paraneoplastic, or immunoglobulin G (IgG) depositional disease, may involve and affect the cranial nerves with irregular enhancement noted along the cranial nerves on postgadolinium MR imaging, with a resultant binocular diplopia.[13–17]

S: scalp tenderness, skull base, and sphenoid bone

New headache with scalp tenderness in the temporal regions or pain with chewing may be seen with giant cell arteritis. New imaging techniques including CTA and thin postgadolinium contrast MR imaging with fat saturation may demonstrate inflammatory change along the superficial temporal artery, aiding in the diagnosis.[1–3] Because CNs III, IV, and VI extend close together from the cavernous sinus through the superior orbital fissure, trauma through the fissure may damage any of the nerves, resulting in a binocular diplopia. In trauma patients with a binocular diplopia, double check cranial nerves especially at the skull base and superior orbital fissure for adjacent fracture deformities. Skull base fractures may affect CN VI because it lies close to the floor of the posterior cranial fossa. Fractures involving the sphenoid or superior orbital fissure may affect nerves CN III, CN IV, CN IV, or V1[2,5,9,18] (**Fig. 12**).

I: increased intracranial pressure

As the incidence of obesity increases in the general population, so has the prevalence of idiopathic intracranial hypertension or pseudotumor cerebri.[19] Therefore, if a patient with a larger body mass index presents with diplopia, pseudotumor cerebri should be considered because this may cause a binocular diplopia, from a CN IV or CN VI palsy occurring from the nerve being compressed along the petrous temporal ridge. Ancillary radiographic findings suggestive of the diagnosis include a partially empty sella, papilledema that can be assessed on the thin-section T2 images or DWI diffusion sequences, prominent enlarged optic nerve sheathes, and compression and flattening of the transverse sinus on MR imaging as well as skull base erosive changes (see **Fig. 12**). In patients with uncal herniation and severe increased intracranial pressure, this may compress CN III and also result in a blown pupil.[1–3,11]

O: onset of new headache or psychosis

Onset of new headache, that is, worst headache of life, should prompt assessment for subarachnoid hemorrhage (see **Fig. 5**G), or new headaches with scalp tenderness or pain with chewing may be seen with giant cell arteritis, requiring careful double checking of the superficial temporal artery, and may help in aiding in the diagnosis.[1–3] In patients with a new onset psychosis and binocular diplopia, clinicians and radiologists should also exclude NMDA receptor antibody encephalitis, paraneoplastic syndromes, or reactive inflammatory causes, including IgG4 or umefactive fibroinflammatory fibrosis.[11,13,14,20] Remember, a patient does not always need to have teratoma. Therefore, in cases with a negative MR imaging, do not forget about neuroinflammatory syndromes and for clinicians to carefully exclude NMDA receptor antibody encephalitis or any additional immune-related abnormality.[18,20,21]

N: neoplasm

All patients with a history of neoplasm and new onset binocular diplopia should be imaged. Patients with tumors either primary in neurofibromatosis with schwannomas along the nerve or metastatic with leptomeningeal disease, or tumor compressing the nerve, or skull base invasion, require imaging with careful attention to the course of the CN III–VII. *N*eoplasms in the region of the

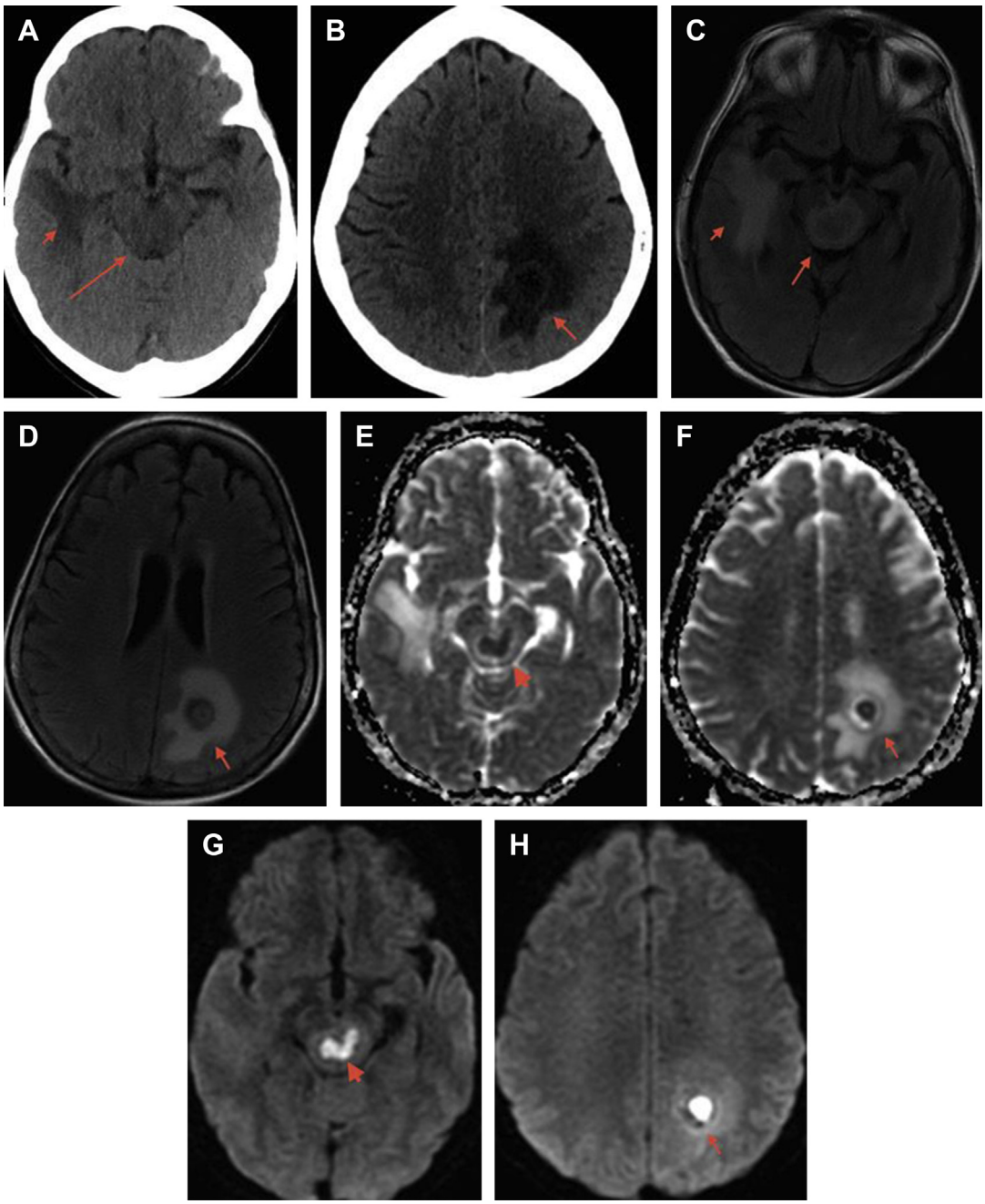


Fig. 9. A 58-year-old woman dialysis patient who presented with binocular diplopia and ophthalmoplegia. Patient could not receive contrast due to elevated creatine and poor renal function. (*A*, *B*) Axial CT scans at level of cerebral peduncles and left parietal lobe with arrowhead and arrows denoting areas of decreased attenuation consistent with edema. Note the faint rim of increased attenuation from abscess rim within the left parietal edema. (*C*, *D*) Corresponding axial fluid-attenuated inversion recovery (FLAIR) MR imaging with hyperintense FLAIR signal (edema) surrounding foci of decreased attenuation (abscesses) (*arrow*). (*E–H*) MR imaging, ADC and DWI. Arrows and arrowhead denote restricted diffusion of the abscess within the brainstem and left parietal lobe with dark ADC and hyperintense DWI signal.

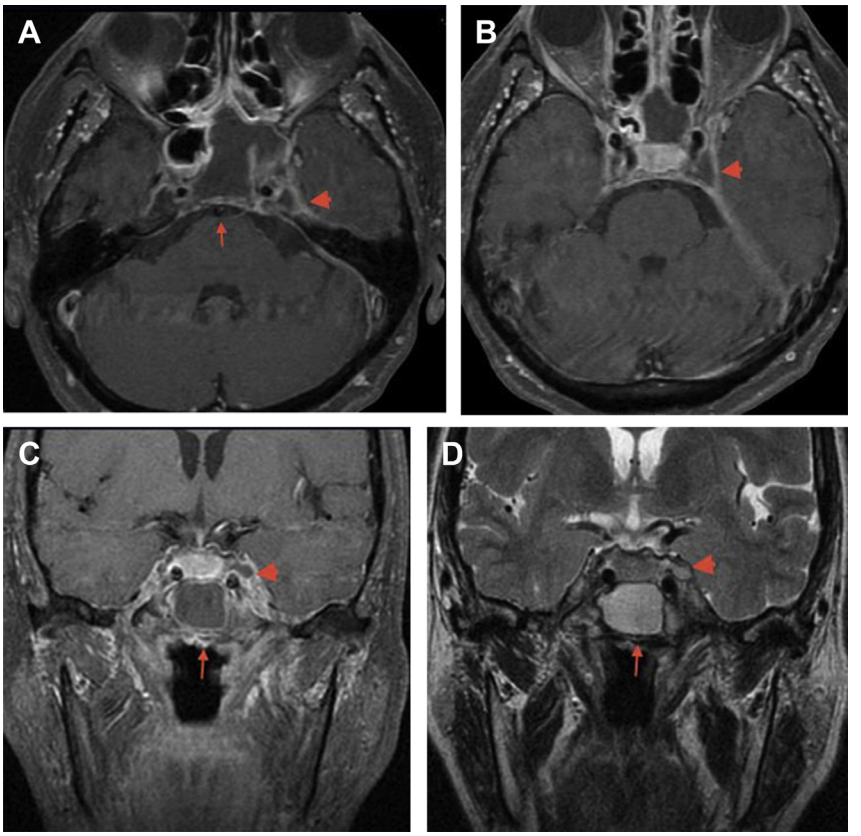


Fig. 10. (*A*, *B*) Patient with infected sphenoid mucocele presenting with diplopia. Axial postcontrast fat-saturation T1 images through the sphenoid sinus and cavernous sinus with small arrow demarcating sphenoid mucocele and arrowhead demonstrating sinus inflammatory change extending into the left cavernous sinus. (*C*) Coronal postcontrast with fat-saturation arrowhead pointing out small "goldfish"-shaped foci of nonenhancement in the left cavernous sinus consistent with abscess (arrow pointing the mucocele in the sphenoid sinus). (*D*) Coronal T2 series through sella and cavernous sinus arrow pointing to opacified sphenoid sinus with arrowhead demarcating extension into the left cavernous sinus and the "goldfish"-shaped abscess.

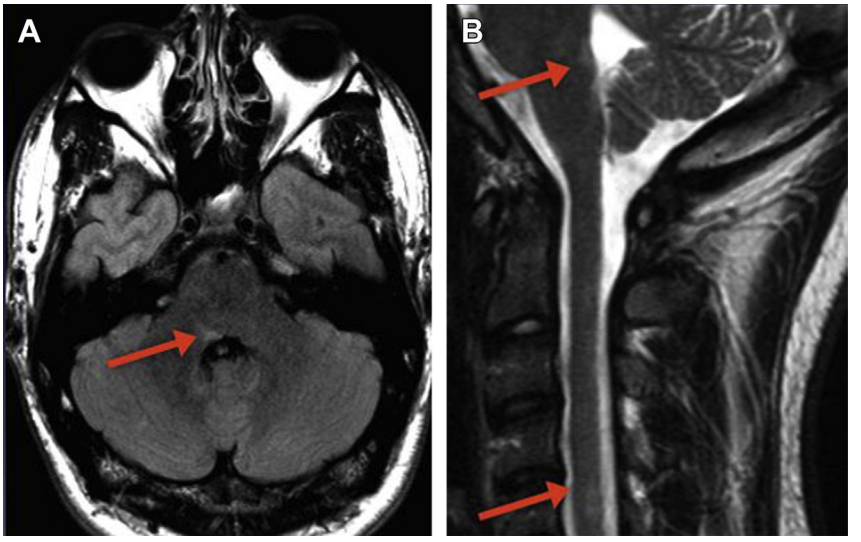


Fig. 11. (*A*) MR imaging brain-axial FLAIR of the brain at the level of the pons. (*B*) Sagittal T2-weighted image of the cervical spine in a 23-year-old man presenting with binocular diplopia involving right CN VI. Arrows denote the FLAIR and T2 hyperintensity at the level of the right CN VI nucleus, just anterior to the fourth ventricle, in patient with demyelinating disease. Second arrow more inferiorly on the T2-weighted MR imaging demonstrates an additional demyelinating plaque in the cervical spinal cord.

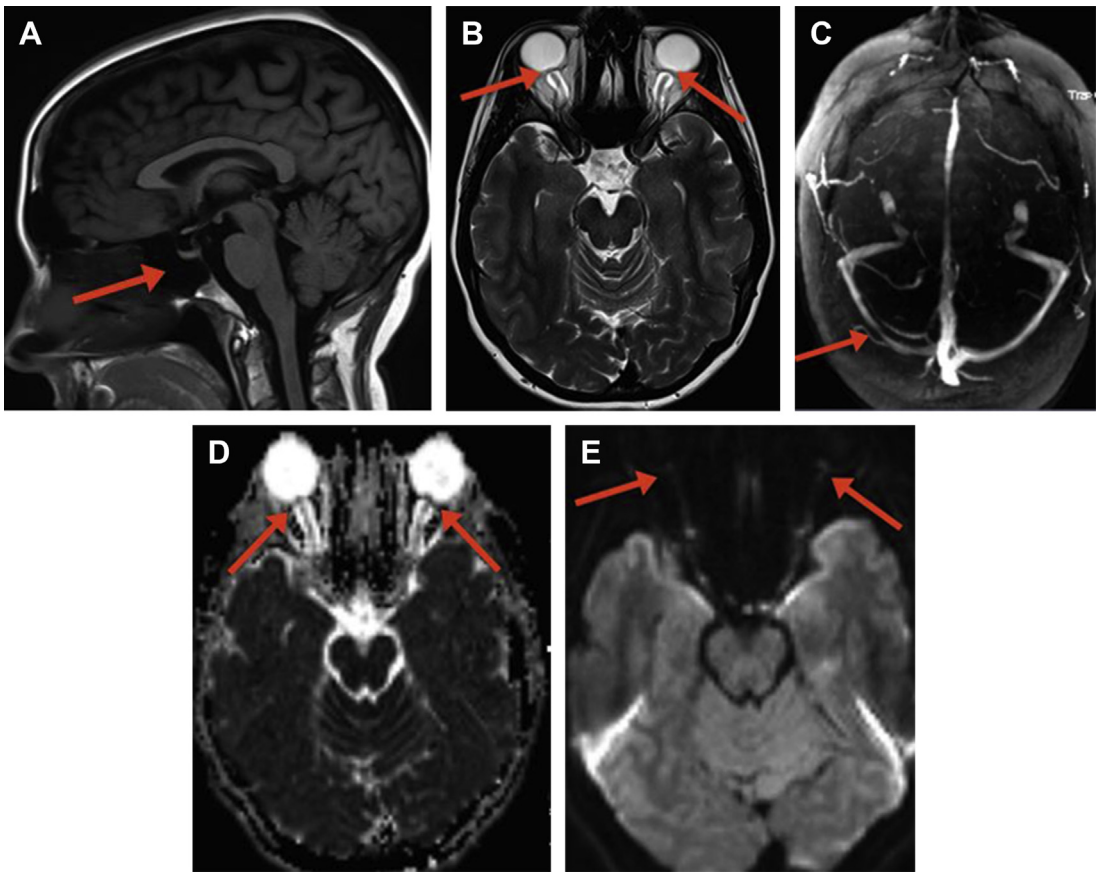


Fig. 12. A 32-year-old woman presented with binocular diplopia, with multiple cranial nerve palsies, with visual loss and headache. Patient underwent a diagnostic lumbar puncture with elevated intracranial pressure diagnosing pseudotumor cerebri or idiopathic increased intracranial hypertension. (*A*) Sagittal T1 MR imaging. Arrow points to a partially empty sella. (*B*) Axial T2 MR imaging. Arrows demarcate enlarged optic nerve sheaths and flattening of the posterior globe margins with papilledema. (*C*) Magnetic resonance venography with arrow pointing to the flattened right transverse sinus. (*D*, *E*) ADC and DWI images with arrows pointing to the restricted diffusion at the optic nerve heads consistent with papilledema.

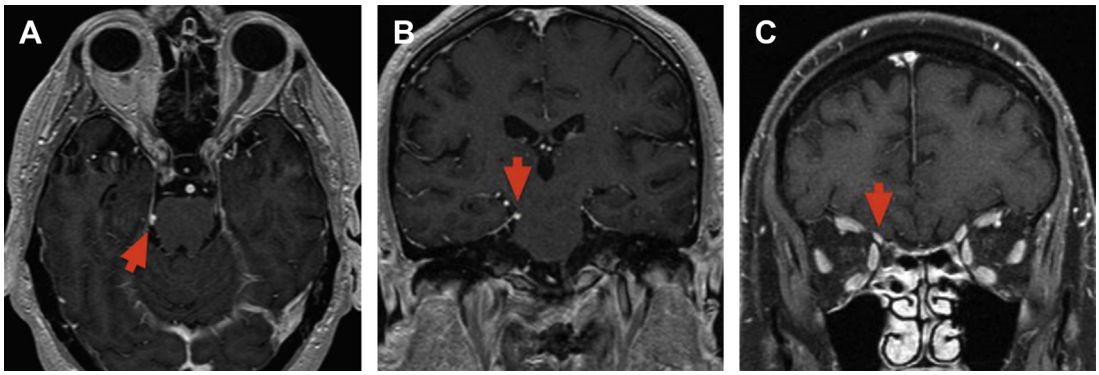


Fig. 13. A 54-year-old man complaining of vertical binocular diplopia, compensating by tilting his head. (*A*, *B*) Axial and coronal T1 postgadolinium MR imaging with small arrow demarcating meningioma at the right tentorial incisura along the course of the distal right CN IV. (*C*) Coronal T1 postgadolinium MR image with arrow pointing to the decreased size of the right superior oblique muscle.

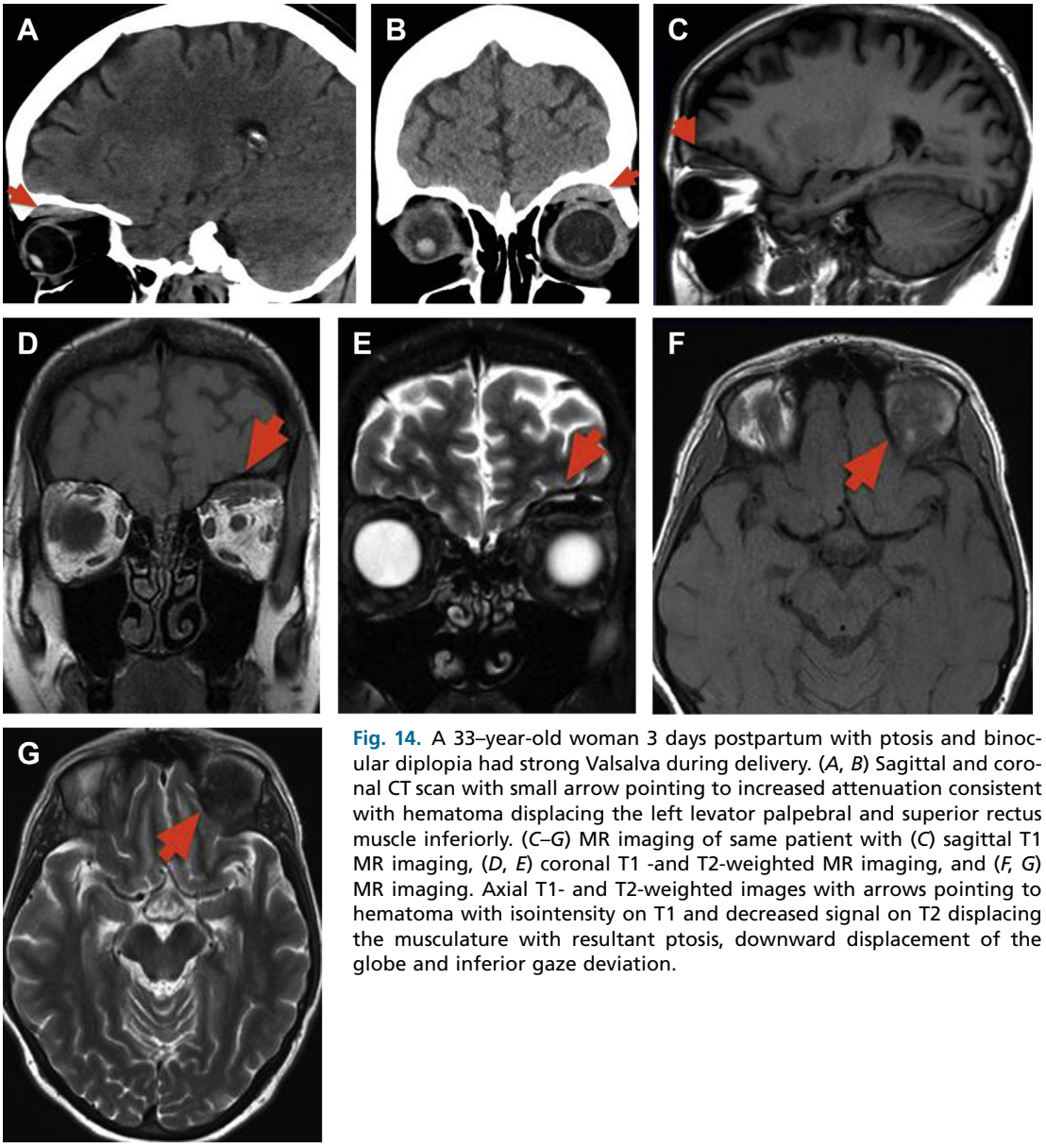


Fig. 14. A 33–year-old woman 3 days postpartum with ptosis and binocular diplopia had strong Valsalva during delivery. (*A*, *B*) Sagittal and coronal CT scan with small arrow pointing to increased attenuation consistent with hematoma displacing the left levator palpebral and superior rectus muscle inferiorly. (*C*–*G*) MR imaging of same patient with (*C*) sagittal T1 MR imaging, (*D*, *E*) coronal T1 -and T2-weighted MR imaging, and (*F*, *G*) MR imaging. Axial T1- and T2-weighted images with arrows pointing to hematoma with isointensity on T1 and decreased signal on T2 displacing the musculature with resultant ptosis, downward displacement of the globe and inferior gaze deviation.

fourth ventricle can also compress CN VI; often this occurs in conjunction with its neighbor CN VI, resulting in both a diplopia with paralysis of lateral gaze and an ipsilateral paralysis of the muscles of facial expression.[1–3] Even a small tumor results in a diplopia if it affects CN III, IV, or VI, as in **Fig. 13**.

In some patients, the mass that is compressing the nerves or muscles resulting in a diplopia may not necessarily be a tumor; therefore, in any patients who may be at risk for a bleed from either trauma, alterations in pressure, or coagulopathies, double check the surrounding periorbital tissue to make sure that there is no compressive causes (**Fig. 14**).

SUMMARY

In binocular diplopia, patients see double with both eyes open. The differential diagnosis for binocular diplopia is extensive, and because this may be a harbinger of life-threatening abnormality, a careful radiographic assessment is critical. This article presents the anatomy and the course of the CN III, CN

IV, and CN VI, and imaging features of abnormality leading to double vision, at either the level of the brainstem, subarachnoid space, cavernous sinus, superior orbital fissure, and orbit. This article uses the acronym VISION as a reminder to double check for causes, including *V*asculature, Infectious or *I*nflammatory, double checking the *S*calp for giant cell arteritis, and the *S*kull base and *S*uperior orbital fissure in trauma, in patients with a larger body mass index, exclude *I*ncreased *I*ntracranial pressure, and in patients with *O*nset of new or worst headaches, double check for subarachnoid hemorrhage. In patients with binocular diplopia and new *O*nset psychosis, this may be a sign of NMDA receptor antibody encephalitis; although these are often associated with teratomas, a teratoma does not always need to be present. In addition, paraneoplastic syndromes or neuroimmune-mediated abnormality should be excluded. All patients with history of *N*eoplasm, primary or metastatic, require careful neuroimaging. A final good rule of thumb, regardless of the age of any patient, is "If diplopia is progressive, or with symptoms of more than 1, then careful neuroimaging should be done!" Patients with binocular diplopia require a careful physical examination, and radiographic images need careful assessment and double check of the brainstem, course of the cranial nerves, and adjacent vessels, to exclude life-threatening critical abnormality.

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