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APPROACH TO A PATIENT WITH DIPLOPIA IN THE EMERGENCY DEPARTMENT

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☐ **Abstract—Background: Diplopia can be the result of benign or life-threatening etiologies. It is imperative for the emergency physician to be proficient at assessing diplopia and recognize when urgent referral or neuroimaging is required. Objective: The first part of this review highlights a simple framework to arrive at the appropriate disposition of diplopic patients presenting to the emergency department (ED). The second part of this review provides more detail and further management strategies. Discussion: ED strategies for assessment of diplopia are discussed. Management strategies, such as when to image, what modality of imaging to use, and urgency of referral, are discussed in detail. Conclusions: Unenhanced plain computed tomography (CT) of the head or orbits is largely not useful in the work-up of diplopia. Magnetic resonance imaging is preferred for ocular motor nerve palsies. Due to limited resources in the ED, patients with isolated fourth and sixth nerve palsies with the absence of other neurological signs on examination should be referred to Neurology or Ophthalmology for further work-up. All patients presenting with an acute isolated third nerve palsy should be imaged with CT and CT angiography of the brain to rule out a compressive aneurysm. Contrast-enhanced CT imaging of the brain and orbits would be indicated in suspected orbital apex syndrome or a retro-orbital mass, thyroid eye disease, or ocular trauma. CT and CT venogram should be considered in cases of suspected cavernous sinus thrombosis. In any patient over the age of 60 years presenting with recent (1 month) history of diplopia, inflammatory markers should be obtained to rule out giant cell arteritis.**

☐ **Keywords—diplopia; Emergency Department**

INTRODUCTION

Diplopia or "double vision" represents 0.1% of all presenting complaints to the Emergency Department (ED) (1). Diplopia can be the result of benign causes, such as refractive error, or life-threatening etiologies, such as compressive aneurysms or tumors; therefore, it is important for the assessing physician to be proficient at assessing diplopia and recognize when the patient may require an urgent referral or neuroimaging.

The purpose of this review is to first present a simple framework for the emergency physician to arrive at the appropriate disposition of patients presenting to the ED with diplopia.

The second part of this review provides a more detailed framework for assessing and localizing the location of lesion responsible for diplopia. We will also highlight literature findings that support our belief that the majority of patients presenting with isolated, binocular diplopia do not need to be imaged with plain computed tomography (CT) of the head, and would be better served with referral to an Ophthalmology or Neurology service for assessment and consideration of magnetic resonance imaging (MRI) or other investigations. We will also highlight scenarios where CT should be used in the ED as part of diplopia work-up.

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DISCUSSION

Part 1. Simplified Approach to the Patient with Diplopia to Arrive at Appropriate Disposition in the ED

Six steps described below can be easily utilized by an emergency physician to arrive at the disposition.

Is the diplopia monocular or binocular?. The first and most important question that should be answered in evaluating a patient with diplopia is whether the diplopia is monocular or binocular. Monocular diplopia persists when either eye is closed, in contrast to binocular diplopia, which disappears when one eye is covered.

Monocular diplopia is almost exclusively an eye problem, most commonly caused by benign entities such as dry eyes or refractive error. Monocular diplopia is not due to eye misalignment; therefore, no neuroimaging is needed, and referral to an ophthalmologist is the most appropriate disposition.

In contrast, binocular diplopia will resolve with closure of either eye, implying that the eyes are misaligned. It is important to elicit this during the examination by asking a patient to occlude either eye to see if the diplopia resolves. Ocular misalignment can be due to pathology affecting one or several ocular motor nerves (III, IV, and VI) intracranially or in the orbit, an orbital process causing restriction of movement of extraocular muscles (thyroid orbitopathy or orbital tumors), or pathology affecting the neuromuscular junction (myasthenia gravis).

Is the diplopia isolated or is it associated with other neurological symptoms?. It is very important to assess whether patients presenting with diplopia have any other neurological symptoms, specifically if any "brainstem" symptoms are present. Brainstem symptoms such as acute onset of dizziness or vertigo, aphasia, ataxia, dysphagia, and presence of "crossed signs" (ipsilateral cranial motor signs and contralateral hemiparesis or hemianesthesia) should prompt one to consider the brainstem as a localization for the symptoms of diplopia. An urgent consideration of neuroimaging (preferably an MRI to detect early signs of ischemic stroke) and mobilization of a stroke consult team should be done in all patients with diplopia and brainstem symptoms. All other investigations should be deferred until an acute brainstem stroke has been ruled out.

Is there an obvious abnormality of ocular motility?. If it is determined that the diplopia is binocular, without any other associated neurological symptoms, the next step should be an examination of ocular motility. The examination should be done by slowly moving the examiner's finger to the different cardinal positions of gaze while maintaining the patient's head stationary. In the ED, checking whether the eye can move all the way up, down, right, and left should be sufficient.

The purpose of checking extraocular motility in the ED is to determine whether there is a palsy of the third or sixth cranial nerve (Figure 1).

Is the motility abnormality compatible with sixth or third nerve palsy?. Inability of the eye to abduct should prompt the examiner to consider a sixth nerve palsy (Figure 1A). Adult patients with acute onset of diplopia with an isolated abduction deficit should be referred for further evaluation to an ophthalmologist. Usually these cases are caused by microischemia of the sixth cranial nerve and spontaneously resolve (2). If the palsy persists beyond 3 months, an MRI of the brain should be obtained to rule out a compressive lesion. Bilateral sixth nerve palsies tend to be associated with more ominous etiologies, such as anything causing increased intracranial pressure (intracranial hemorrhage, subdural hematoma) and brainstem infarction, but other neurological abnormalities would be expected in these patients, which would direct the ED work-up (2). In children presenting with a new abduction deficit, MRI of the brain should be obtained immediately, as compression is the most common cause of a new sixth nerve palsy in the pediatric population.

Patients with a complete third nerve palsy present with mydriasis, ptosis, the eye deviated outward, and an inability to look up and medially (Figure 1B). They should be immediately evaluated with CT and CT angiography of the brain, as it could be caused by a compressive lesion (specifically an aneurysm) most commonly arising from the junction of the posterior communicating and internal carotid arteries. Aneurysmal rupture would result in subarachnoid hemorrhage and has a high mortality rate (3).

Patients with a fourth nerve palsy most commonly do not harbor any sinister underlying pathology and are difficult to diagnose by a non-ophthalmologist. They usually

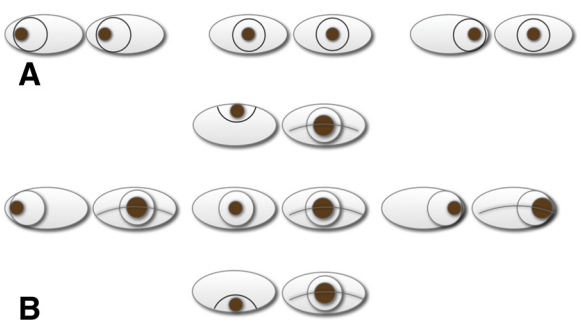

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Figure 1. (A) Complete left sixth nerve palsy. (B) Complete left third nerve palsy. Note left ptosis and pupillary involvement.

present with vertical or oblique diplopia and would have near-normal extraocular motility. The most common etiology of fourth nerve palsy is either trauma or decompensation of a congenital fourth nerve palsy. These patients should be referred to an ophthalmologist.

Are there multiple motility abnormalities that could possibly be due to involvement of more than one oculomotor nerve?. This can be a difficult question to answer for an inexperienced examiner, therefore, all patients whose ocular motility abnormalities do not point to either a sixth or third nerve palsy should be referred to an ophthalmologist or neurologist for further testing.

If the emergency physician believes that ocular motility abnormalities are secondary to the involvement of several ocular motor nerves, it would be appropriate to order a semi-urgent MRI study to evaluate the cavernous sinuses and orbits. In the absence of other neurological symptoms or signs in the vast majority of cases, it is safe for the patients to wait to see a specialist who will be better equipped to perform careful ocular motility and ocular alignment testing, and come up with an appropriate differential diagnosis.

CBC, ESR, and CRP. All patients over the age of 60 years presenting with the new onset of binocular diplopia should have complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) drawn to rule out rare cases of giant cell arteritis (GCA) that present with diplopia (4,5).

Summary (Figure 2): Simplified 6-Step Approach to a Patient Presenting with Diplopia to an ED

1) Is the diplopia monocular or binocular?
2) Are there are any focal neurological symptoms (particularly brain stem symptoms)? If yes, activate stroke protocol.

If there are no other associated neurological symptoms or signs:

3) Perform extraocular motility testing.
4) Is there an obvious sixth or third cranial nerve palsy? All patients with third nerve palsies require urgent CT angiography of the brain to rule out aneurysmal compression.
5) Are the motility abnormalities complex and do not point to an isolated cranial nerve palsy? Consider semi-urgent MRI study to evaluate cavernous sinuses or a semi-urgent referral to the Ophthalmology or Neurology service.

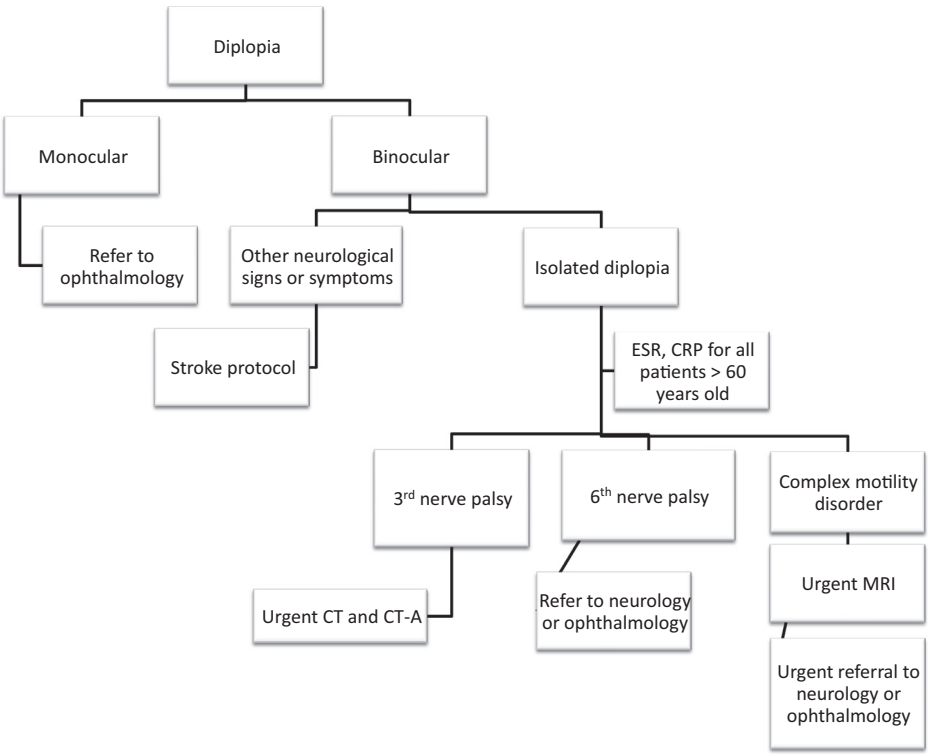

Figure 2. Simplified approach to the patient with diplopia to arrive at the appropriate disposition in the Emergency Department. ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; CT = computed tomography; CT-A = computed tomography angiography; MRI = magnetic resonance imaging.

6) All patients over 60 years of age with the new onset of binocular diplopia—order CBC, ESR, and CRP to rule out GCA.

Part 2. After an Appropriate First Assessment, the Following Management Strategies are Suggested to Help Further Localize the Lesion

Diplopia related to a single isolated cranial nerve palsy. In cases of an isolated fourth or sixth palsy, the literature largely supports MRI of the brain and orbit, usually with contrast, as the imaging of choice (6–8). Urgent MRI in these cases is not necessary (with the exception of children who should receive an urgent MRI study in all cases of new-onset cranial nerve palsies) and referral should be made to Neurology or Ophthalmology for further assessment and decision-making (7,8).

In cases of an isolated third nerve palsy, 6% of patients would harbor a growing compressive aneurysm and thus, all patients presenting with an isolated third nerve palsy require an urgent CT angiography (CT-A) scan of the brain (9).

Other exceptions where CT scan of the brain and facial bones/orbit (thin slices if possible) with contrast in the ED would be helpful are in cases of trauma and suspected orbital or orbital apex pathology, which will be discussed below (10).

Third nerve palsy. The superior branch of the third cranial nerve (CN3) innervates the ipsilateral superior rectus and levator muscle, and the inferior branch innervates the ipsilateral inferior and medial recti, the inferior oblique, and the iris sphincter. A complete CN3 palsy is easy to identify, as the patient presents with the affected eye in the "down and out" position, and complete ptosis on the affected side. The eye presents with an inability to supraduct, infraduct, or adduct. The pupil may or may not be dilated.

A partial CN3 palsy is harder to recognize, and can present as any combination of a supraduction, infraduction, or adduction deficits, ptosis, or pupil dilation. It is important to remember, though, that an isolated, dilated pupil in the absence of extraocular motility deficits is never due to a CN3 palsy, and other causes of anisocoria should be sought (11).

A careful examination should be performed to look for any signs of lid retraction or pupil constriction on adduction or depression of the eye, which would indicate aberrant regeneration of the branches of the third cranial nerve. Any sign of aberrant regeneration suggests a compressive etiology and urgent neuroimaging should be sought (8,12).

In adults over the age of 50 years, the most common cause of a complete, pupil-sparing third nerve palsy is microangiopathic disease, which represents up to 42% of cases (9). In cases where there is any evidence of partial CN3 palsy, pupil involvement, or aberrant regeneration, a compressive lesion should be suspected and must be ruled out with urgent neuroimaging, as 6% of patients would harbor a compressive aneurysm, most commonly at the junction of the posterior communicating artery and the internal carotid artery (11).

Although it is imperative that this subset of patients receive urgent neurovascular imaging, there is a debate among the experts as to whether all patients presenting with an acute third nerve palsy should be imaged. A recent large population-based study performed in the United States found that 36% of compressive third nerve palsies were pupil-sparing, and 22% of compressive aneurysms presented as complete third nerve palsies (9). Therefore, we recommend that all patients presenting with an acute third nerve palsy regardless of pupillary involvement should have an urgent CT and CT-A scan of the brain performed in the ED, and then be referred to an appropriate service based on imaging findings.

Of note, pain is not a distinguishing factor and can be present in both compressive and microangiopathic third nerve palsies (12,13).

Sixth nerve palsy. The sixth cranial nerve (CN6) innervates the ipsilateral lateral rectus muscle. Patients with CN6 palsy typically present with esotropia, or "in-turning" of the affected eye, with limited abduction of the affected eye, and binocular horizontal diplopia that is worse on looking toward the affected side and worse in the distance.

CN6 palsy is the most common isolated ocular motor cranial nerve palsy, representing 50% of all patients (6). Microangiopathic disease has been reported to account for as much as 36% of all patients with CN6 palsies, with other etiologies being trauma, demyelination, and rarely, neoplasms in adults (2). All microangiopathic CN6 palsies should spontaneously resolve within 2–3 months.

Therefore, in patients over the age of 50 years with vascular risk factors, isolated acute (defined as < 3 months in duration) CN6 palsy is most likely caused by microangiopathic disease and can be referred to a Neurology or an Ophthalmology service for further management (2). In all other patients with acute CN6 palsy without history of trauma, MRI at presentation vs. close follow-up is debatable, but would be the imaging of choice over a CT scan, and therefore, the patient should be referred on for further assessment (2,6–8).

An exception to this rule would be a patient with a history of head trauma, when a CT scan with attention to the skull base should be performed given the frequency of CN6 palsies associated with petrous apex fractures (6).

It is also important for an emergency physician to rule out signs of increased intracranial pressure (the most important sign of which is papilledema) as a cause of sixth nerve palsy, which would warrant urgent CT and CT venogram in the ED to rule out an intracranial mass, and dural sinus thrombosis, which can rarely present with sixth nerve palsy and symptoms of increased intracranial pressure. If a physician's confidence in their funduscopic examination is low, a prompt ophthalmological referral to rule out papilledema should be obtained. Idiopathic intracranial hypertension, which can lead to visual loss if not diagnosed and treated promptly, is a common cause of papilledema (and thus can present with sixth nerve palsy) and most of the time will not produce abnormalities on CT scanning. We thus emphasize the importance of obtaining an ophthalmological consultation in all cases where a physician is not confident in their funduscopic examination skills and suspects presence of papilledema.

Bilateral CN6 palsies also tend to be associated with more ominous etiologies, such as clivus pathology, intracranial hemorrhage, subdural hematoma, and brainstem infarction, but other neurological abnormalities would be expected in these patients, which would direct the ED work-up (2).

Fourth nerve palsy. The fourth cranial nerve innervates the superior oblique (SO) muscle, and therefore, patients with SO palsy frequently complain of vertical, or sometimes oblique, diplopia. Most of the SO palsies are actually congenital in etiology and have decompensated over time as the patient's fusional amplitudes, which is the ability to fuse two discrepant images into one, decreases with age. The SO muscle is also responsible for incyclotorsion (inward torsional movement) of the eye, so it is helpful in congenital cases to look for an associated head tilt that patients tend to develop as a response to chronic excyclotorsion (outward torsional movement) of the eye. As the fourth cranial nerve has the longest intracranial course, it is very susceptible to trauma, thus the second most common etiology of fourth nerve palsies is traumatic. When trauma and congenital causes are excluded, the major cause of isolated fourth nerve palsies in patients over the age of 50 years is microangiopathic disease (14). Therefore, in the absence of trauma, patients with fourth nerve palsies should be referred to an Ophthalmology service for further assessment. CT scan of the brain should not be utilized in cases of isolated SO palsy.

Diplopia related to multiple cranial nerve palsies. The presence of multiple extraocular nerve palsies may indicate pathology that affects areas where the nerves run in close proximity to each other. Particular areas of interest would be the cavernous sinus and orbital apex. One should also remember that myasthenia gravis can mimic the presence of multiple extraocular nerve palsies. It is imperative for the emergency physician to remember that in patients with pituitary apoplexy, an acute bleed into the preexisting pituitary adenoma can cause lateral expansion of the pituitary gland, thus affecting cranial nerves in the cavernous sinus, and all patients presenting with headaches and cranial nerve palsies require emergency neuroimaging to rule out this condition.

Orbital apex. The orbital apex is the most posterior part of the orbit. Important structures pass through here, including the all the extraocular rectus muscles, sympathetic fibers, and cranial nerves 3, 4, and 6, the first and second branches of cranial nerve 5, and the optic canal, which contains the optic nerve.

A wide variety of pathology can affect the orbital apex, including infections, multiple inflammatory entities, neoplasms, trauma, and thyroid eye disease (15,16). Findings of ophthalmoplegia with any evidence of decreased vision or numbness in V1 and V2 distribution should raise concerns about pathology in the orbital apex. Additionally, proptosis may signal an extraconal mass. In cases of suspected orbital apex syndrome, CT scan of the orbits with contrast could be an appropriate first imaging choice given that contrast from the orbital fat allows for excellent visualization of intraorbital and bony structures, as well as the sinuses (15,16).

Cavernous sinus. The cavernous sinuses are paired sinuses located in the skull base, located on either side of the pituitary fossa. Cranial nerves 3, 4, and 6, the first and second divisions of cranial nerve 5, the sympathetic nerve, and the internal carotid artery all travel through the cavernous sinuses, which serve as the major drainage passageway for the ophthalmic veins. Of note, orbital apex syndrome can be distinguished from cavernous sinus pathology by its involvement of the optic nerve and thus, decrease in the central visual acuity. Cavernous sinus by contrast lies away from the optic nerve, thus the visual acuity is usually not affected.

A serious and life-threatening complication that can affect the cavernous sinuses is septic cavernous sinus thrombosis (CST). It is usually caused by the direct spread of infection from the adjacent structures and often caused by sinusitis, dental abscesses, otitis media, and periorbital cellulitis (14).

Although the prevalence of septic CST has decreased with the widespread use of antibiotics, mortality is still reported at 30% with significant morbidity (17). Patients are often ill when they present to the ED, and appear septic and febrile. Due to impaired drainage from the ophthalmic veins, patients present with eyelid swelling, proptosis, and

chemosis; ophthalmoplegia is present in 50–80% of cases. Bilateral eye findings are present in most cases because the cavernous sinuses are in direct communication with one another, and in cases of septic CST involvement of the opposite side, occurs usually within 24–48 h of presentation. Thus, signs of bilateral eye involvement in an ill-appearing patient should signal the clinician to direct attention to the cavernous sinus (17).

In cases of suspected CST, CT and CT-venogram of the brain and orbits would be an appropriate imaging choice in the ED, and may show a filling defect in the cavernous sinus (17). Blood cultures and cerebrospinal fluid studies should be obtained in toxic patients and broad-spectrum antibiotics should be initiated empirically. The patient should be co-managed by Neurology and Internal Medicine. High suspicion for fungal infection should be maintained in diabetic and immunocompromised patients.

Diplopia related to other causes. Other causes of diplopia include myasthenia gravis, thyroid eye disease, multiple sclerosis, and GCA. Up to 60% of patients with myasthenia gravis present with ptosis or diplopia, and 20% have a form of localized ocular myasthenia gravis (18). The hallmark of ocular myasthenia gravis is fatigability and variability of symptoms with diplopia or ptosis that worsens near the end of day or with prolonged activity. In cases of suspected myasthenia gravis, referral should be made to a neurologist for further assessment, without the need for a CT scan.

Thyroid eye disease (TED) can cause diplopia as a result of enlargement or fibrosis of the extraocular muscles. Although TED mainly presents in patients with a history of hyperthyroidism, up to 10% of patients can be euthyroid or hypothyroid (19). Patients often present with eyelid retraction, chemosis, conjunctival injection, proptosis, and eye motility disturbances. Findings are frequently bilateral, and often asymmetric. The most common eye motility disturbance is restricted supraduction from fibrosis of the inferior recti, although any of the extraocular muscles can be affected. Although most patients suffer a mild form of TED, 3–7% of patients have sight-threatening complications from either corneal exposure or compressive optic neuropathy (19,20). Therefore, any complaints of worsening visual acuity, especially in cases with marked proptosis, should be urgently assessed for the presence of compressive optic neuropathy. A CT scan of the orbits would allow for assessment of the extraocular muscles, which frequently demonstrate enlargement, as well as optic nerve status.

In patients with multiple sclerosis, the most common ocular motility abnormality is internuclear ophthalmoplegia (INO), which can be found in up to 53% of patients with clinically definite multiple sclerosis at some point throughout their clinical course (18,21). INO is caused by a lesion of the medial longitudinal fasciculus located in the brainstem. INO is responsible for connecting the third nerve nucleus on one side to the sixth nerve nucleus on the other and manifests clinically as inability of both eyes to look to one side, which requires activation of the medial rectus muscle in one eye and the lateral rectus muscle in the other. In patients with demyelinating disease, it is frequently bilateral, and is characterized by slowing or impaired adduction of the ipsilateral eye with or without nystagmus of the contralateral abducting eye. Patients presenting with INO should be referred to Neurology for systemic evaluation and MRI imaging. CT scan is not helpful in evaluation of lesions of the posterior fossa, thus it is not useful in evaluating patients with INO.

GCA causes systemic inflammation and occlusion of the medium- to large-sized vessels, resulting in a host of systemic ischemic complications, including anterior ischemic optic neuropathy, which leads to sudden, irreversible, and bilateral vision loss in up to 60% of cases if untreated (3). It is an uncommon cause of diplopia, with reported rates of 3–8%, but a multicentered, large prospective study looking at etiologies of isolated third, fourth, and sixth nerve palsies in the United States found that GCA was the cause of 2.7% of cases of isolated sixth nerve palsy in patients with no other systemic symptoms or signs (3,4). Given the potential morbidity and mortality associated with missing this diagnosis, screening bloodwork for GCA with CBC, ESR, and CRP should be performed in all patients over the age of 60 years presenting to the ED with a recent (within past month) onset of diplopia.

Diplopia related to trauma. Diplopia related to ocular trauma can be a result of damage along any part of the pathway of the cranial nerves, but is frequently accompanied by other obvious signs of trauma. A large retrospective chart review looking at patients presenting to the ED with closed head trauma found that patients with ocular motor nerve injuries had significantly lower Glasgow Coma Scale scores, higher rates of CT abnormalities (intracranial injury and cranial facial fractures), and more frequent need for inpatient rehabilitation compared with patients without ocular motor nerve injuries (22). Patients with multiple ocular motor nerve palsies, in particular, had the lowest Glasgow Coma Scale scores.

Another recent, large retrospective case series that analyzed the use of CT imaging in the ED for undifferentiated eye complaints found that 68% of patients with triage complaints of ocular trauma who had CT of the brain performed had positive ocular imaging findings, including orbital wall fractures and globe rupture (23).

These findings suggest that CT scans in patients with ocular trauma in the ED are high yield, and they also help in the assessment of associated facial injuries.

WHEN TO IMAGE A PATIENT WITH DIPLOPIA

There are few studies that have looked at the assessment of diplopia in the ED. A recent, large retrospective case series studying the use of CT imaging for undifferentiated vision complaints in a tertiary care ED found that the rate of CT head imaging for eye-specific complaints increased from 4.7% to 7.2% over a period of 4 years (23). Two percent of all patients with eye complaints had diplopia, and more than one-third of diplopic patients were investigated with CT scan of the head (37.5%). Fifty of the 376 patients (13.2%) who were scanned had a primary complaint of diplopia. Of the 50 scans performed, four had positive CT findings (one carotid cavernous fistula, one thyroid eye disease, one thyroid eye disease vs. orbital pseudotumor, and one intracranial mass) (23).

Only one study to date has looked at the frequency of etiologies of diplopia presenting to the ED (1). This prospective study found that in cases of isolated diplopia without associated neurological signs or symptoms, the sensitivity of unenhanced CT scan was 0%. Only 13% of the patients with isolated diplopia were ultimately diagnosed with an underlying organic etiology based on investigations.

Overall, this suggests that unenhanced CT imaging of patients with isolated diplopia in the absence of trauma or neurological signs in the ED is very low yield, and that referral to Neurology or Ophthalmology for further assessment prior to neuroimaging should be strongly considered. The primary exception would be acute-onset, isolated third nerve palsy where CT and CT-A should be performed urgently to rule out a compressive aneurysm.

CONCLUSIONS

Based on the current literature, unenhanced plain CT of the head or orbits is largely not useful in the work-up of patients with isolated diplopia in an emergency setting, with the exception of trauma patients. MRI is the imaging modality of choice in the assessment of ocular motor nerve palsies, and due to limited resources in the ED setting, patients with isolated fourth and sixth nerve palsies with the absence of other associated signs should be referred to Neurology or Ophthalmology for further assessment.

All patients presenting with an acute isolated cranial nerve 3 palsy should have urgent neuroimaging with CT and CT-A of the brain to rule out a compressive aneurysm as part of their ED assessment.

Contrast-enhanced CT imaging of the brain and orbits would be indicated in the ED in cases of suspected orbital apex syndrome or a retro-orbital mass, thyroid eye disease, or cases of ocular trauma.

CT and CT-V should be considered in cases of suspected cavernous sinus thrombosis.

In patients over the age of 60 years, high clinical suspicion for GCA as a rare cause of diplopia should be maintained, and appropriate bloodwork should be ordered.

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ARTICLE SUMMARY

1. Why is this topic important?

Diplopia is a frequent undifferentiated visual complaint of patients presenting to the Emergency Department (ED). The utilization rate of computed tomography (CT) in the ED has been increasing, but a recent study found that in cases of isolated diplopia without associated neurological signs or symptoms, the sensitivity of unenhanced CT scan was 0%.

2. What does this review attempt to show?

This review provides a simple framework for assessing a patient in the ED with diplopia and arriving at the proper disposition. We highlight literature findings and describe clinical scenarios where referral to Ophthalmology or Neurology is helpful and demonstrate that magnetic resonance imaging (MRI) is often the preferred imaging modality.

3. What are the key findings?

The majority of patients presenting with isolated, binocular diplopia do not benefit from plain CT of the head, and would be better served with referral to an Ophthalmology or Neurology service for assessment and consideration of MRI or other investigations.

4. How is patient care impacted?

Proper recognition of the underlying causes of diplopia and the utility of diagnostic imaging in the work-up of patients presenting with diplopia will decrease improper use of plain CT head imaging of these patients in the ED setting and lead to the appropriate disposition and referral.