# Managing Patients With Acute Visual Loss

Jonathan A. Edlow, MD*; Beatrice Hoffmann, MD

*Corresponding Author. E-mail: jedlow@bidmc.harvard.edu.

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## INTRODUCTION

Acute visual loss is a frightening symptom for patients. Timely, accurate diagnosis and early treatment can preserve or restore vision. This article focuses on the emergency department (ED) management of adult patients presenting with spontaneous (not traumatic or postsurgical), acute-onset (minutes to days), isolated (no other obvious neurological symptoms) visual loss. Acute visual loss may be transient or persistent. Etiologies fall into 4 categories (Table 1):

- Ocular or orbital pathology
- Ocular neurovascular dysfunction
- Brain pathology (optic chiasm or retrochiasmal)
- Functional neurological disease

To clarify some terms, optic neuropathy is a general term for optic nerve dysfunction, which can be ischemic, inflammatory, infectious, toxic, or compressive. Inflammatory optic neuritis is most commonly caused by multiple sclerosis and less commonly by neuromyelitis optica. Methanol causes bilateral, toxic optic neuritis.[1] Central retinal artery occlusion causes retinal (not optic nerve) ischemia.

A management algorithm (Figure 1) emphasizes diagnoses and emergency treatments that maximize visual outcomes.

## HISTORY

Key historical elements (Table 2) include the rapidity of onset, transient or persistent nature of the acute visual loss, presence or absence of ocular pain or redness, and localization of the acute visual loss (monocular or binocular). Importantly, many patients with a hemianoptic field cut incorrectly report the deficit as unilateral vision loss to the eye on the side of the cut. Therefore, assessing visual fields is intrinsic to localizing the pathology.[2] Retro-orbital pain with eye movement suggests optic neuritis or orbital compression. Optic neuritis reduces color perception.[3] Contact lens use suggests corneal problems. Methanol poisoning causes dyspnea and gastrointestinal symptoms with the acute visual loss. Coincident seizure or headache suggests posterior reversible encephalopathy syndrome.

Vascular risk factors and prior cardiovascular disease suggest brain or eye ischemia. Other neurological symptoms (weakness, sensory symptoms, dizziness) suggest brain pathology. The review of systems should investigate new headache, nausea, and vomiting. In older patients, a history of shoulder or hip pain and stiffness, jaw claudication, scalp tenderness, or new headaches suggests giant cell arteritis, an important diagnostic consideration, since treatment with corticosteroids can prevent subsequent visual loss and occasionally reverse existing loss.[4-7]

In patients with preexisting monocular visual problems, new symptoms in the "good eye" add a greater sense of urgency to the evaluation.

For asymptomatic patients with recent transient visual loss, define the number, nature, frequency, and duration of the spells. Patients who instinctively cover one eye, then the other, can often distinguish monocular from binocular visual loss. Nearly 70% of patients with idiopathic intracranial hypertension have brief (seconds to a minute) transient visual obscurations, episodes of monocular or binocular visual "fogginess," black or gray out, sometimes with flashes or sparkles of light (photopsia).[8] Fewer than 25% of patients with transient monocular visual loss due to retinal emboli endorse brief episodes of the classic "curtain dropping," but its presence suggests ipsilateral carotid disease.[9-11] Although a third of patients with giant cell arteritis have episodes of transient monocular visual loss prior to their diagnosis, giant cell arteritis is an uncommon but important cause of transient monocular visual loss.[12,13] Be very hesitant to diagnose "retinal migraine" in patients with monocular acute visual loss; it is rare.[14]

## PHYSICAL EXAMINATION

Systematic ophthalmological and neurological examinations help to sort through diagnostic hypotheses generated by the history (Table 2).

**Table 1.** Differential diagnosis of acute visual loss by category.*

| Category | Causes | Notes |
|---|---|---|
| **Ocular pathology** | Corneal abrasion, edema or keratitis | Slit lamp examination with fluorescein important; consult ophthalmology except for some small corneal abrasions |
| | ANAG | Red painful eye with corneal edema and a midposition fixed pupil; consult ophthalmology |
| | Iritis and uveitis | Slit lamp examination important; consult ophthalmology |
| | Vitreous hemorrhage | Can be diagnosed by ocular POCUS; consult ophthalmology |
| | Vitreous detachment | Can be diagnosed by ocular POCUS; consult ophthalmology |
| | Retinal detachment | Can be diagnosed by ocular POCUS; consult ophthalmology |
| | Endophthalmitis | IV antibiotics; consult ophthalmology, can be evaluated with ocular POCUS |
| | Orbital mass effect | From cellulitis, tumor, inflammation or hematoma; may need orbital thin-cut CT; look for proptosis and EOM abnormalities; consult ophthalmology, can be evaluated with ocular POCUS |
| **Neurovascular supply to eye** | Optic neuritis (nonischemic) | This can be due to inflammation, demyelination, toxins (such as methanol) and various medications |
| | Retrobulbar optic neuritis | |
| | AION (nonarteritic) | |
| | AION (arteritic—GCA) | Nearly always in patients >50 years of age with other symptoms and signs (see text) and increased inflammatory markers |
| | CRAO | Unilateral visual loss, can be partial (from a branch occlusion) or complete |
| | CRVO | |
| | Increased IOP/papilledema | An important cause is IIH (pseudotumor cerebri), can be diagnosed by ocular POCUS |
| **Brain** | | |
| Optic chiasm | Pituitary apoplexy | Usually associated with abnormalities of extraocular muscle movement; often have a bitemporal field cut |
| Retrochiasmal | Strokes involving the occipital cortex and retrochiasmal visual pathways | Patients with a homonymous hemianopsia often do perceive unilateral visual loss, and not a field cut; some patients may be tPA candidates |
| | PRES | Usually associated with seizures and headache; often associated with underlying medications, diseases or conditions. MRI for diagnosis |
| | Retinal migraine | Be hesitant to make this as a new diagnosis in the absence of a well-established prior history; true "retinal migraine" is rare |
| **Functional neurological disease** | Functional visual loss | Be very hesitant to make this diagnosis in the ED without extensive evaluation and consultation |

*ANAG*, acute narrow angle glaucoma; *POCUS*, point-of-care ultrasound; *IV*, intravenous; *CT*, computed tomography; *EOM*, extraocular muscles; *AION*, acute ischemic optic neuropathy; *GCA*, giant cell arteritis; *CRAO*, central retinal artery occlusion; *CRVO*, central retinal vein occlusion; *IOP*, intraocular pressure; *IIH*, idiopathic intracranial hypertension; *PRES*, posterior reversible encephalopathy syndrome; *MRI*, magnetic resonance imaging.
*This list is not meant to be encyclopedic but, rather, includes the more common serious causes of acute visual loss. As in the text, this table does not include patients with posttraumatic or postophthalmic surgical visual loss.

Always test visual acuity. Smartphone apps allow this to be done easily at the bedside. For patients without their glasses (or who do not know they need glasses), looking through a pin hole (or a tiny hole punched into a stiff piece of paper) will normalize any refractive error. Both proptosis and enophthalmos suggest orbital disease.[15] Proceed with a front-to-back examination, including slit lamp evaluation with fluorescein. Measure intraocular pressure in patients with red, painful eyes.

Ophthalmoscopy should be performed, although physician skill in performing and interpreting the results is declining.[16] Dilating the pupil improves the view but is not commonly done by emergency physicians without ophthalmology consultation and, then, only after excluding acute narrow angle closure glaucoma. In patients with transient monocular visual loss, visualization of refractile cholesterol emboli (Hollenhorst plaques) suggests ipsilateral carotid disease.[17] Papilledema, especially in overweight, young women, suggests idiopathic intracranial hypertension. In central retinal vein occlusion, the fundus shows "blood and thunder" (hemorrhages and cotton wool spots), whereas it is pale, classically with a cherry-red spot, in central retinal artery occlusion.

Patients with mucormycosis are often febrile and show signs of nasal, palatal, or orbital inflammation.[18] If the problem is not clearly ocular/orbital, perform a systematic neurological examination. Visual field testing is essential to

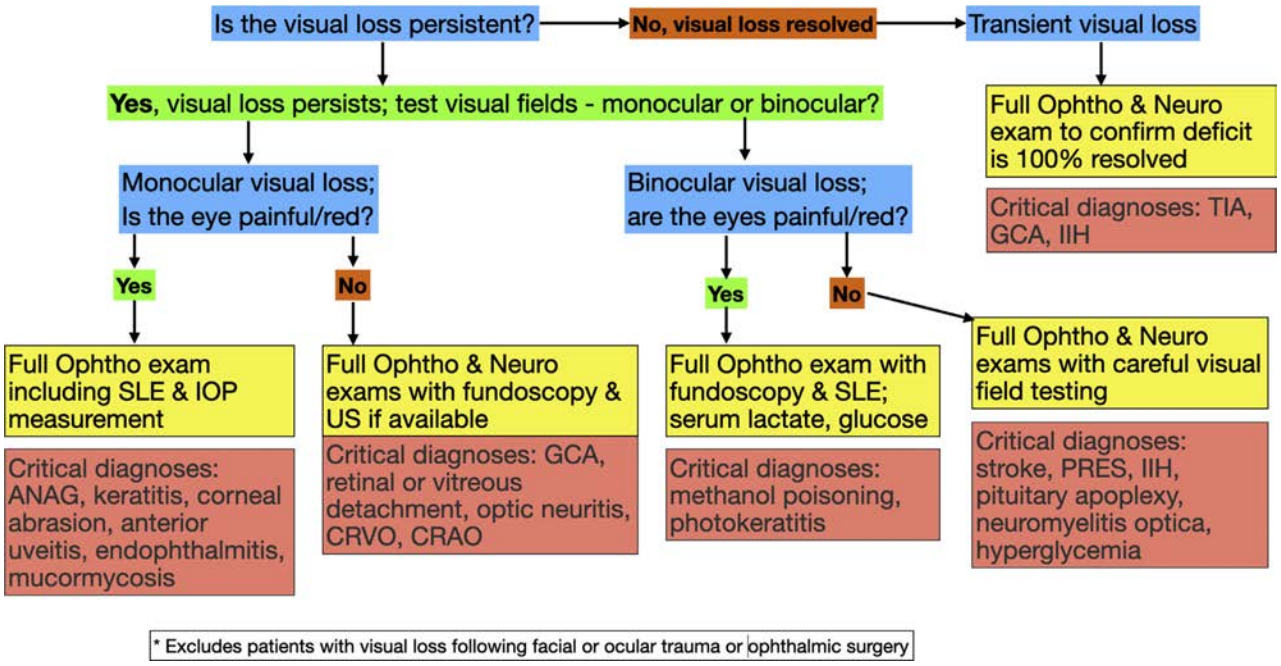


**Figure 1.** Algorithmic approach to patients with acute visual loss. *IIH*, idiopathic intracranial hypertension; *TIA*, transient ischemic attack.

differentiate prechiasmal, chiasmal, or retrochiasmal pathology. For bedside confrontation field testing, the patient covers 1 eye and either counts the number (using 1, 2, or 5 fingers to maximize the patient's ability to discriminate between them) or movement of the examiner's fingers.[2]

Patients with unilateral or asymmetric optic nerve dysfunction will have a relative afferent pupillary defect on the swinging flashlight test. A relative afferent pupillary defect does not occur in brain-related acute visual loss or with functional visual loss. Temporal artery redness, tenderness, swelling, nodules, and reduced pulses all suggest giant cell arteritis.

## LABORATORY TESTING

Laboratory testing is generally unhelpful. Exceptions include point-of-care glucose for hyperglycemia (blurred vision, mucormycosis) or hypoglycemia (any neurological deficit) and serum lactate (methanol poisoning), and if giant cell arteritis is suspected, a complete blood count (for anemia or thrombocytosis), an erythrocyte sedimentation rate (ESR), and C-reactive protein. Measuring both increases diagnostic sensitivity.[19] One study of 167 patients with biopsy-proven giant cell arteritis found that 11% had ESRs of less than 50 mm per hour and 5% had ESRs of less than 40 mm per hour.[20] Rarely, patients' inflammatory markers are normal.[21] Conversely, a meta-analysis showed that an ESR of less than 40 reduced the likelihood of giant cell arteritis.[22] If either marker is increased, consult an ophthalmologist.

## IMAGING

Orbital computed tomography is indicated if orbital compression is suspected. Brain imaging, preferably magnetic resonance imaging, will help diagnose strokes and neuromyelitis optica.

## POINT-OF-CARE OCULAR ULTRASOUND

Emergency-physician-performed ocular point-of-care ultrasound is a diagnostic, bedside examination that visualizes all chambers of the eye and retrobulbar structures, including the optic nerve sheath diameter. It is especially helpful when direct visualization is difficult or equivocal and allows for a dynamic evaluation of the eye. Emergency physicians with the appropriate training can diagnose a variety of eye conditions with high accuracy.[23] The level of clinician training probably correlates with the diagnostic accuracy.[24]

### Technique

The patient, placed in a supine or semireclining position, closes their eyes. For ocular point-of-care

**Table 2.** Important elements of the clinical examination in patients with acute visual loss.

| Diagnosis | History | Physical Examination |
|---|---|---|
| **Ocular & orbital** | | |
| Corneal abrasion/keratitis | Severe eye pain, headache, possible contact lens use | Red eye with corneal defect most easily seen on SLE using fluorescein |
| Spontaneous hyphema | May be a history of ocular trauma | Blood visible in the anterior chamber on direct inspection and SLE |
| Uveitis, iritis | Pain worse in bright light | Pain in affected eye when light shined on good eye. Flare from WBCs in anterior chamber on SLE, sometimes with hypopyon |
| ANAG | Severe eye pain, headache, vomiting, seeing colored halos around lights | Red eye with corneal edema, midposition pupil, shallow anterior chamber, increased IOP |
| Vitreous hemorrhage | Painless floaters and flashes of light | Difficulty seeing retina on ophthalmoscopy. POCUS helpful, might be associated with acute vitreous detachment |
| Endophthalmitis | Often postoperative or posttrauma. Subacute onset of eye pain and decreasing vision | Fever uncommon. Hypopyon. View of retina is often hazy. POCUS helpful |
| Retinal detachment | Monocular painless variable AVL. Sudden onset of floaters and dark spots in vision. Photopsias | Occasional APD present. Ophthalmoscopy may show irregular retinal folds. POCUS helpful |
| Orbital compression | Pain and variable AVL, possible diplopia | May have proptosis, enophthalmos and restriction of one or more EOMs. Can cause an APD |
| **Neurovascular** | | |
| CRAO | Sudden onset of painless AVL (partial or complete depending upon vessels involved). May have been preceded by TMVL | Retinal pallor and optic disc swelling (if anterior, but normal if retrobulbar), usually with a APD. Look for findings of GCA (see text) |
| CRVO | More subacute onset of painless AVL and blurred vision | Retina shows "blood and thunder" (retinal hemorrhages and cotton wool spots) |
| Optic neuritis | Pain on eye movement and decreased color perception | Optic disc is often swollen. APD present |
| IIH | May have transient visual obscurations, headaches worse lying flat or with Valsalva and pulsatile tinnitus. Most common in overweight women | Bilateral (but can be asymmetric) papilledema. May have a sixth nerve palsy. POCUS helpful |
| Nonarteritic AION | Sudden onset of painless AVL | APD present |
| Arteritic AION | Sudden onset AVL often preceded by transient episodes and accompanied by other symptoms (see text) | APD present. Temporal artery tenderness, swelling, nodularity or redness |
| Methanol poisoning | Blurred vision or visual loss (plus other nonvisual symptoms) | Poorly or unreactive pupils, optic disc hyperemia, retinal edema, dilated pupils |
| **Brain** | | |
| Homonymous hemianopsia | May have headache and feels like one eye is the problem. Bumping into things or being startled by objects appearing from the hemianoptic side; note that rarely, GCA can cause stroke | Homonymous field cut. Sometimes problems with reading words, other neurological deficits depending on exact location of infarct, ICH or mass |
| Pituitary apoplexy | Thunderclap headache, ophthalmoplegia, and altered mental status | Variable deficits of EOMs, altered mental status. May have a bitemporal hemianopsia |
| Cortical blindness | Bilateral blindness with relative unawareness of the visual loss (Anton syndrome). This can also occur with PRES | Normal ophthalmic examination. Opticokinetic nystagmus if tested |
| PRES | Variable visual symptoms usually associated with high blood pressure, seizures and headache | Increased blood pressure and variable visual field cuts |

**Table 2.** Continued.

| Diagnosis | History | Physical Examination |
|---|---|---|
| **Functional** | | |
| Functional visual loss | Variable; may have prior psychiatric disorders and/or an acute stressor | Normal or nonanatomic examination. Never have APD |

*ANAG*, acute angle closure glaucoma; *APD*, afferent pupillary defect; *AVL*, acute visual loss; *EOM*, extraocular muscles; *AION*, anterior ischemic optic neuropathy; *ICH*, intracerebral hemorrhage; *PRES*, posterior reversible encephalopathy syndrome.

ultrasound, a linear, high-frequency transducer is used with the appropriate machine settings, assuring that low mechanical and thermal indices are used with the shortest exposure time and as low as reasonably achievable acoustic output. Current national, international, and FDA recommendations postulate that the mechanical index should be below 0.23 and the thermal index below 1.0 for ocular sonography.[25,26] Once proper patient positioning and machine settings are assured, some sonographers apply Tegaderm over the closed eyelids as a protective barrier, followed by a copious amount of gel. The transducer should not directly touch the patient's eyelids; no external pressure is applied to the globe.

The transducer is held suspended in this gel "pillow" and fanned and rotated to obtain the appropriate views in the axial and sagittal planes. The ocular structures can be evaluated in a systematic, anterior-to-posterior manner, with depth adjustments to eventually incorporate retrobulbar structures. For better accuracy, both static and dynamic examination components should be employed. During the static component, instruct the patient to hold their eyes still as the sonographer moves the transducer. During the dynamic component, hold the probe still while the patient is asked to move their eyes. This maneuver will improve the detection of vitreous or retinal detachments and is used with increased gain settings. The optic nerve sheath and disk are examined with increased depth settings and normal gain.[25]

## Normal Findings

Ocular point-of-care ultrasound findings can improve diagnostic accuracy (Figure 2 and https://www.acep.org/sonoguide/advanced/ocular-emergencies/). Eyelids are seen closed underneath the gel pillow; the cornea appears as a small hyperechoic layer running in parallel underneath the eyelids. Next is the anterior chamber, which is separated from the much larger posterior chamber by the iris, ciliary body, and lens. Further posterior is the vitreous body. In young patients, the lens should appear echo-free and with echogenic capsule. The chambers and vitreous body should be mostly anechoic. With advancing age, usually, benign vitreous opacities are common and more apparent with increased gain settings.[27] The normal, nondetached retina is found posterior to the vitreous chamber. Most posteriorly, retrobulbar structures, such as the optic nerve with nerve sheath, orbital fossa soft tissue, and muscles surrounded by orbital bones, can be visualized (Figure 2).[28]

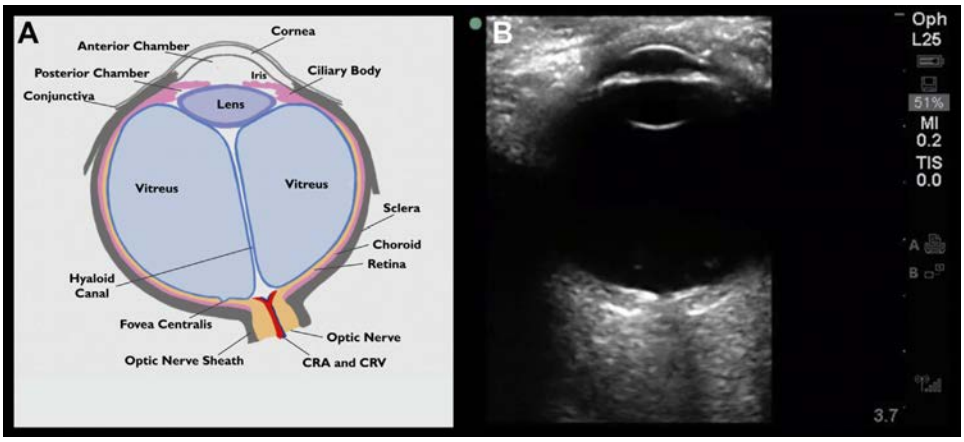


**Figure 2.** *A*, Anatomical illustration of the eye. Reprinted with permission.[28] *B*, Corresponding normal point-of-care ocular ultrasound image showing anterior and posterior chamber, iris, lens, and vitreous chamber with retina.

### Abnormal Ultrasound Findings

Figures 3 and 4 show a selection of common pathologies detected with ocular point-of-care ultrasound and their appearances on ophthalmoscopy.

## INITIAL MANAGEMENT

Table 3 lists treatable conditions causing acute visual loss; Figure 5 lists clinical clues. Patients with acute visual loss caused by ocular and neurovascular conditions usually need ophthalmology consultation. If consultation is unavailable, tele-ophthalmology with or without nonmydriatic retinal photography may help to identify patients with serious diagnoses.[29,30] Consult neurology for patients with brain or neurovascular problems. This section emphasizes emergency treatments that should take place in the ED.

For patients with transient monocular visual loss not likely to be idiopathic intracranial hypertension, distinguish between nonarteritic and arteritic causes, as described above. For nonarteritic transient monocular visual loss, treat the patient as if they had a transient ischemic attack with antiplatelet therapy and a same-day evaluation for large vessel disease and cardioembolic sources.[31,32] If the transient visual loss is thought to be due to giant cell arteritis, treat with steroids (see below).

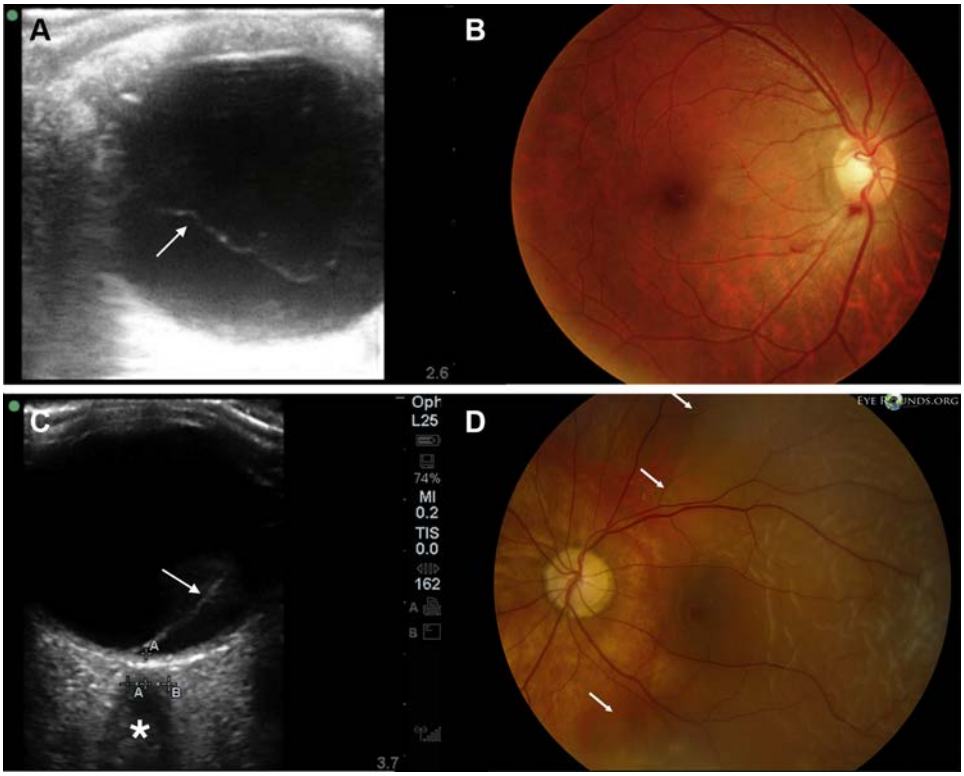


**Figure 3.** *A*, Ultrasound image of posterior vitreous detachment (arrow). *B*, Ophthalmoscopic image of a vitreous detachment. In posterior vitreous detachment (PVD), a thin linear membrane that will cross the intersection of the optic nerve sheath and posterior wall of the globe is seen. The gain should be increased to improve visualization, and the patient is asked to move the closed eyes. This "oculokinetic ultrasonography" approach will show the freely mobile detached vitreous membrane with its characteristic and prominent aftermovements. Over time, the membrane stiffens, decreasing its mobility, making it more difficult to distinguish a PVD from other, less-mobile globe pathologies, such as a retinal detachment (RD). A PVD may be tethered near the ora serrata and can be associated with a RD and vitreous hemorrhage (VH). Reprinted with permission. (From Chong SY, Fhun LC, Tai E, et al. Posterior vitreous detachment precipitated by yoga. *Cureus*. 2018;10:e2109.) *C*, Showing a retinal detachment (arrow) that is tethered to the optic nerve (*); and *D*, ophthalmoscopic image of retinal detachment. An RD appears as a bright echogenic membrane, anchored to the optic nerve sheath (*). In the acute phase, the membrane appears thin and has some mobility but progressively stiffens and thickens over time. A total retinal detachment anchored at the optic nerve and ora serrata is commonly visualized as a triangular or funnel-shaped figure in the vitreous chamber. Reprinted with permission. (From Rhegmatogenous retinal detachment. Mortensen ZQ, Coussa RG, Folk JC, et al. Accessed July 20, 2021. http://webeye.ophth.uiowa.edu/eyeforum/atlas/pages/Rhegmatogenous-ret-detach.htm)

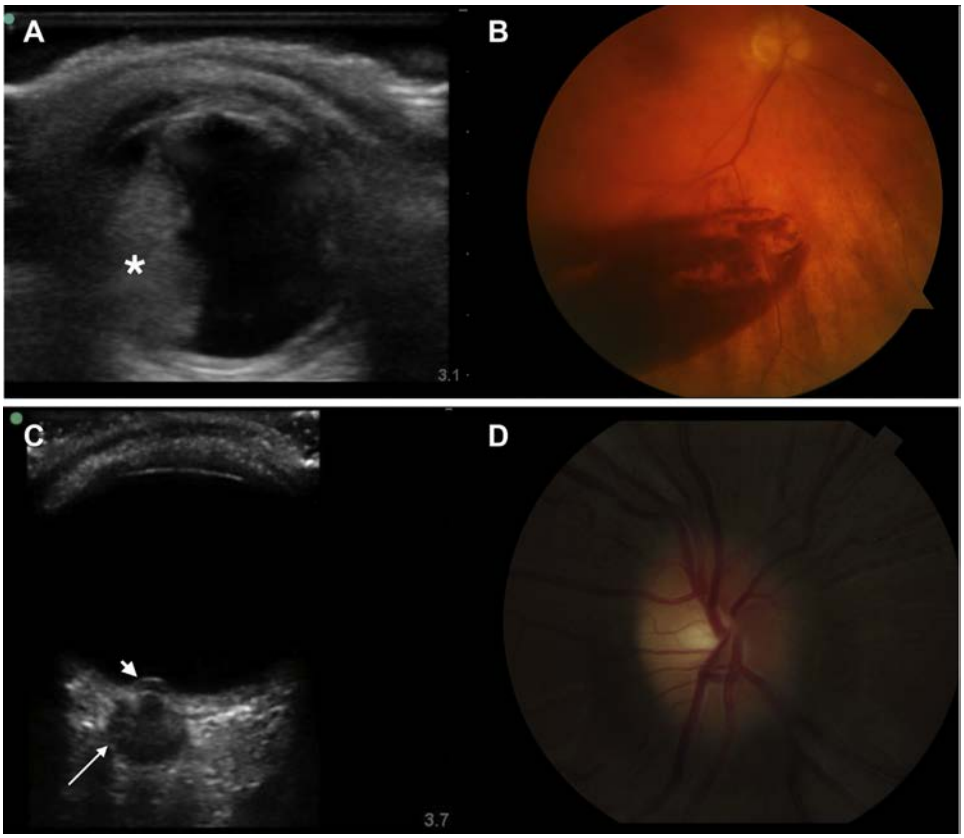


**Figure 4.** *A*, Vitreous hemorrhage (*). *B*, Ophthalmoscopic image of a VH. Sonographic appearance of acute VH shows slightly echogenic material that is poorly defined. Small bleeds can be visualized as scattered particles; large bleeds can fill the entire vitreous chamber. A significant increase in the gain setting will reveal the true extend of the bleeding. Hematomas from acute bleeds should be mobile with oculokinetic testing. Over time, hematomas will organize and appear more echogenic and less mobile. Reprinted with permission. From Moran CORE, Winter T. et al. Vitreous Hemorrhage. [Zachary Q. et al. Rhegmatogenous retinal detachment.] *C*, Ultrasound image of a papilledema with increased optic nerve sheath diameter (ONSD, *) and "crescent sign" (arrow). The optic disc of a patient with idiopathic intracranial hypertension is seen bulging into the vitreous chamber. *D*, Ophthalmoscopic image of a patient with mild papilledema. The ONSD is measured 3 mm below the vitreous chamber (Figure 2C and *). The widened nerve sheath appears darker than the optic nerve. This finding is called the "crescent sign" and contributes to increased subarachnoid fluid surrounding the optic nerve. The arrow shows papilledema. Reprinted with permission. Images provided courtesy of Dr. Nurhan Torin, MD.

There is no specific emergency treatment for patients with ongoing acute visual loss due to nonarteritic optic neuropathy. However, arteritic acute ischemic optic neuropathy is a treatable, time-critical diagnosis. In patients with giant cell arteritis presenting with visual symptoms, second-eye involvement occurs within 24 hours in 36% of cases and within a week in an additional 36%.[33] These data, coupled with the variability in inflammatory marker levels and the fact that approximately 20% of patients have visual loss without systemic symptoms or signs, justify having a very low threshold for giving an initial dose of steroids in the ED.[34] Although controversy exists about the dose and route of administration, many experts recommend a first dose of 500 to 1000 mg of intravenous methylprednisolone.[6,35-38] Importantly, temporal artery biopsy results remain diagnostic for up to 2 weeks of steroids.[39,40]

Because the retina is central nervous system tissue, a central retinal artery occlusion is a stroke, and a "code stroke" should be activated to facilitate rapid decisions. Historical treatments, including intravenous acetazolamide, carbogen inhalation, ocular massage, and anterior chamber paracentesis, may be harmful.[41] In a meta-analysis of patients with central retinal artery occlusion treated with tissue plasminogen activator (tPA), the 67 tPA-treated patients within 4.5 hours had a 20% absolute risk reduction (37.3 treated versus 17.7% not treated) of visual recovery, defined as a visual acuity of 20/100 or better.[42]

**Table 3.** ED interventions to be considered in patients with acute visual loss.*

| Diagnosis | Intervention | Comments |
|---|---|---|
| **Ocular** | | |
| ANAG | Drops, carbonic anhydrase inhibitors, rarely anterior chamber paracentesis | If ophthalmology consultation not immediately available, administer:<br>• Timolol 0.5% 1 drop, then 1 minute later,<br>• 1 drop of 1% apraclonidine, then 1 minute later,<br>• 1 drop of 2% pilocarpine, followed by 500 mg of IV acetazolamide.<br>Should also treat pain and give antiemetics as needed. |
| Endophthalmitis | Antibiotics | Intravitreal antibiotics are most important; systemic IV antibiotics are given, but their efficacy is not proven |
| **Neurovascular** | | |
| CRAO | Fibrinolytics | Basically, a stroke of the retina that should be approached like any other stroke |
| CRVO | No specific treatment | Consult ophthalmology, but there is no immediate treatment |
| GCA (arteritic AION) | Steroids | Patients with AVL and suspected GCA should be treated with steroids as soon as possible and always receive a first dose in the ED |
| Inflammatory optic neuritis | Steroids | Steroids are often given, but not with the same urgency as with some other diagnoses, and the evidence for their efficacy is weak |
| Nonarteritic AION | No specific treatment | Exclude GCA with inflammatory markers since not all patients have the usual clues in the history and physical examination |
| TMVL thought to be TIA | Antiplatelet agents and imaging for LVO | Many of these patients have carotid stenosis. Workup should occur the same day as the patient presents. Consider GCA in these patients also |
| Methanol poisoning | Fomepizole | IV fomepizole (sometimes IV ethanol and hemodialysis); toxicology consultation |
| **Cerebral** | | |
| Ischemic stroke | IV fibrinolytics | Patients with retrochiasmal stroke are treated like any other patient with a stroke, with fibrinolytics if they qualify, and potentially with endovascular therapy; consult neurology |
| PRES | BP control | Control hypertension; definitive treatment depends on the context. For example, if due to eclampsia, delivery and magnesium would be given. Consult neurology (OB-GYN if pregnant or postpartum) |
| Pituitary apoplexy | Hormone replacement and potential surgical decompression | One of the few causes of acute visual loss when a neurosurgical consultation should be done in the ED |
| Pseudotumor cerebri | IIH/LP and acetazolamide | Other treatments can be done on a nonurgent basis as recommended by neurology consultant |
| **Functional** | | |
| Functional visual loss | No specific acute treatment | May need psychiatry evaluation but ONLY after a thorough medical evaluation |

*TMVL*, transient monocular visual loss; *LVO*, large vessel occlusion; *BP*, blood pressure; *LP*, lumbar puncture; *OB-GYN*, obstetrics and gynecology.
*Barring exceptional circumstances, all patients in the ED presenting with acute visual loss should have consultation with a specialist, usually an ophthalmologist or neurologist, depending on the target diagnosis. Other potential consult services include neurosurgery (pituitary apoplexy), obstetrics (eclampsia with PRES), and psychiatry (functional visual loss, after thorough exclusion of other possibilities and consultation).

Patients treated between 4.5 and 6 hours had no benefit. Two additional screening steps beyond the usual ones prior to tPA use are a (preferably dilated) ophthalmoscopic examination to exclude intraocular hemorrhage and an attempt to identify patients with likely giant cell arteritis. Current guidelines state that intravenous tPA should "be considered" in patients with central retinal artery occlusion with disabling acute visual loss.[43]

Acute narrow angle closure glaucoma requires emergency treatment. Consult ophthalmology emergently, and start pharmacotherapy (Table 3).[44] Definitive treatment is surgical. Although optic neuritis is

Ocular pathology

- Patients with optic neuritis often notice loss of color vision and may have a field loss and a relative afferent pupillary defect.
- Rhino-orbital mucormycosis usually occurs in the setting of diabetes and ketoacidosis. Orbital imaging should be done, and consult an infectious disease specialist and an otorhinolaryngologist.
- If ANAG is suspected (pain, redness, and halos) and formal tonometry is unavailable, one can palpate the eyes (sequentially, not simultaneously); the symptomatic eye will feel harder than the normal eye. For patients with increased IOP, emergency clinicians should begin pharmacotherapy when an ophthalmologist is not rapidly available.

Neurovascular pathology

- In patients with CRAO being considered for thrombolytic treatment, 2 management steps (in addition to the usual inclusion and exclusion criteria) are a ophthalmoscopic examination to exclude ocular hemorrhage and gathering information by history, physical examination, and inflammatory markers to reduce the likelihood of GCA.
- GCA is rare in patients younger than 50 years of age but is a VERY important condition to consider in older patients with new visual symptoms (even in the absence of headache). Because GCA can be "occult" (no clinical findings other than visual symptoms), emergency clinicians should send ESR and CRP in elderly patients with AVL.
- Although uncommon, patients with GCA can have low ESR and/or CRP. In these patients, associated symptoms (fatigue, fevers, shoulder or hip pain and stiffness, jaw claudication, and scalp tenderness, in addition to visual ones) should raise the suspicion of GCA.
- In patients whose evaluations suggest GCA, high-dose steroids should be started in the ED. For patients with normal vision (have had an episode of TMVL), 60 mg of oral prednisone is often recommended, and for patients with persistent AVL, many authorities recommend 500–1000 mg of IV methylprednisolone. A KEY POINT is that steroids will not affect temporal artery biopsy results for at least 2 weeks.
- Although the evidence for emergency therapy for optic neuritis is weak, when optic neuritis is bilateral or associated with visual acuity of 20/100 or worse, the cause is more likely to be neuromyelitis optica, which is treated more aggressively. Therefore, consult ophthalmology for all cases of optic neuritis.
- Test for methanol poisoning in inebriated patients with acute visual symptoms, as it both is treatable and may be fatal untreated. Look for coexistent dyspnea, metabolic acidosis, an APD, and disc hyperemia.

Brain pathology

- Patients with a homonymous hemianopsia frequently experience their visual field deficit as decreased vision in one eye (on the side of the field cut) and, thus, incorrectly report a monocular problem.
- Stroke, including CRAO, can occur in young people without classic vascular risk factors.
- Although IIH is uncommon in the general population, it is far more common in female patients of childbearing age who are significantly overweight. It is often associated with a sixth nerve palsy.
- Although the concept of "retinal migraine" is firmly entrenched in medical nomenclature, true retinal migraine is rare. Emergency clinicians should be hesitant to diagnose it in the ED in patients who do not have a well-established diagnosis by an ophthalmologist or neurologist and who have had multiple prior episodes.

Functional pathology

- Functional visual loss is a diagnosis of exclusion that should only be made after thorough ophthalmological and neurological evaluations.

Miscellaneous

- Patients with mild diplopia often endorse blurred vision rather than double vision.
- Bedside visual field examination is extremely important to localize the site of pathology. Although not as accurate as formal testing, stand in front of the patient and have the patient close one eye (and the examiner the opposite one, to compare the patient's visual field with the examiner's "normal"). Test the patient's ability to see movement of the examiner's fingers or to count fingers. For the latter, use either 1, 2, or all 5 fingers to maximize the patient's ability to distinguish between the numbers.

**Figure 5.** Clinical pearls and pitfalls.

often treated with steroids, the supporting evidence is weak, and the decision is not an emergency.[3,45] Ophthalmology should be always consulted for their recommendations since neuromyelitis optica is treated more aggressively.

Ophthalmologists treat endophthalmitis with intravitreal antibiotics; most cases are postsurgical or posttraumatic. Pituitary apoplexy, usually from a hemorrhage into a previously undiagnosed pituitary adenoma, requires emergency hormone replacement and,

often, neurosurgical decompression of the sella. Patients with methanol poisoning are treated with intravenous fomepizole.

Ischemic strokes involving the retrochiasmal visual pathways are treated the same as other strokes. Treatment decisions are not based on a specific NIHSS score but rather on the degree of disability for that individual patient.[46] Patients with minor strokes occasionally have large vessel occlusions for which endovascular treatment should be considered.[47] Finally, nearly a third of patients with "mild" strokes end up dependent.[48] Rarely, giant cell arteritis can also causes strokes from large vessel inflammation.

Emergency clinicians should be hesitant to diagnose functional visual loss without a detailed evaluation including specialist consultation.

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*Author affiliations:* From the Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA.

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## IMAGES IN EMERGENCY MEDICINE

*(continued from p. 451)*

**DIAGNOSIS:**

*Esophageal foreign body impaction.* Esophageal foreign body is a common complaint.[1] Point-of-care ultrasound is a useful tool for detecting foreign bodies in the cervical esophagus.[1,2] Emergency physicians must be careful not to mistake the innermost hyperechoic layer (superficial mucosa or the interface between the lumen and mucosa) of the esophagus as a foreign body.[3] Sonographers can distinguish a foreign body from normal esophageal layers (Figure E1) based on acoustic shadowing. The echogenicity of a foreign body varies with posterior acoustic shadowing or ring-down artifact.[4]

The patient underwent esophagogastroduodenoscopy, which revealed duck bone in the middle third of the esophagus. After retrieval (Figure 3), the patient was uneventfully discharged.

*Author affiliations:* From the Department of Emergency Medicine (Huang, Chu, Chang, Chiu, Sun), Far Eastern Memorial Hospital, New Taipei City, the Department of Emergency Medicine (Chiang, Ma), National Taiwan University Hospital, Taipei, and the Department of Emergency Medicine (Chiang, Ma), National Taiwan University Hospital Yun-Lin Branch, Douliu City, Taiwan.

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