Abstract

Primary care physicians are at the forefront of patient care and often are the first clinicians to triage and diagnose any eye-related complaints. They must be able to quickly identify vision-threatening pathologies, as delay in treatment of an ocular emergency can result in permanent vision loss. This concise review describes the definition, presentation, examination, and management of various ophthalmic emergencies including blunt ocular trauma, chemical ocular injury, orbital cellulitis, endophthalmitis, acute angle closure glaucoma, optic neuritis, giant cell arteritis, central retinal artery occlusion, retinal detachment, and homonymous hemianopia in a succinct manner.

Prompt recognition and treatment of ophthalmic emergencies can improve systemic and ocular outcomes. Therefore, it is vital for primary care providers to be well-versed in triaging eye-related complaints (Table 1). We discuss presentation, evaluation, and next steps for common ophthalmic emergencies that may be encountered by the clinician (Table 2).

It is crucial to triage a patient with vision or ocular complaints and assess visual function and ocular integrity. The first step involves visual acuity testing for each eye, ideally with the patient wearing their
eyeglasses or contact lenses. This is followed by inspection of both eyes and the surrounding periorbital tissue including the eyelids, globes, orbits, and cheeks for evident erythema, hematomas, lacerations, proptosis, hemorrhage, corneal clouding, or hyphema (blood in the anterior chamber). Next, examine the pupils for any mydriasis, miosis, asymmetry, response to direct illumination, and relative afferent pupillary defect. This is followed by confrontational visual field testing, extraocular muscle (EOM) mobility assessment, slit-lamp examination (if available), and a direct ophthalmoscopic fundus exam.

**OCULAR INJURIES**

Initial evaluation for any ophthalmologic trauma involves overall assessment of the patient and addressing any life-threatening injuries. Directed questions regarding the mechanism and circumstances of injury, any foreign body involvement, ocular history, and tetanus immunization status should be asked.

**Blunt Injury**

An open globe injury involves loss of integrity and compromise of the cornea or sclera. Signs that may indicate such an injury include moderate to severe pain, reduced vision, hyphema, and pupillary distortion toward the wound. On slit-lamp examination, a decrease in the depth of the anterior chamber may be evident.\(^1\) A Seidel test should also be performed to assess for a full thickness laceration or perforation. Anesthetic eye drops such as proparacaine hydrochloride 0.5% are used to numb the eye, followed by applying a moistened fluorescein dye strip to the site of injury; the eye can then be examined under cobalt-blue light with a slit lamp. A positive Seidel test suggesting full thickness corneal or scleral injury would show fluorescent green dye dilution around the defect demonstrated by a color change of fluorescein from orange to lighter yellow, and flow of intraocular fluid on Cobalt blue examination.\(^2\)

In the case of a possible open globe rupture, a protective shield should be taped over the injured eye without further disturbance of the surrounding tissue (Table 2). An ophthalmologist should be urgently consulted. Broad-spectrum antibiotics, antiemetics, and analgesics should also be administered and the patient should be instructed to eat or drink nothing per mouth in case emergent surgery is needed. Tetanus

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**TABLE 1. Characteristic Pertinent History and Findings for Different Ophthalmic Emergencies**\(^a\)

<table>
<thead>
<tr>
<th>Condition</th>
<th>History and Findings</th>
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<tr>
<td><strong>Open globe injury</strong></td>
<td>Distorted pupil, hyphema, corneal/scleral laceration</td>
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<td><strong>Chemical injury</strong></td>
<td>History of recent chemical splash exposure, conjunctival injection with a blanched patch</td>
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<tr>
<td><strong>Orbital cellulitis</strong></td>
<td>Pain, decreased vision, periorbital swelling and erythema, limited eye movement with our without constitutional symptoms, afferent pupillary defect, elevated intraocular pressure</td>
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<tr>
<td><strong>Endophthalmitis</strong></td>
<td>Recent surgery, pain, decreased vision, hypopyon.</td>
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<tr>
<td><strong>Acute angle closure glaucoma</strong></td>
<td>Severe pain, decreased vision (colored halos around lights, foggy vision), headache (often unilateral), nausea, vomiting</td>
</tr>
<tr>
<td><strong>Optic neuritis</strong></td>
<td>Young woman, pain with eye movement, sudden decrease vision in one eye (change in color vision, presence of scotoma)</td>
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<tr>
<td><strong>Giant cell arteritis</strong></td>
<td>Temporal tenderness to palpation and unilateral headache, jaw claudication, sudden painful vision loss, and possible constitutional symptoms of fever, myalgias, night sweats, weight loss.</td>
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<tr>
<td><strong>Central retinal artery occlusion</strong></td>
<td>Sudden, painless vision loss, history of cardiovascular risk factors (ie, hypertension, type II diabetes, coronary artery disease, prior stroke)</td>
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<tr>
<td><strong>Retinal detachment</strong></td>
<td>Sudden onset of persistent flashes and floaters, peripheral “shower of black dots” or “swarm of bees,” painless vision loss with a dark curtain-like shadow (from any direction—sides, top, or bottom)</td>
</tr>
<tr>
<td><strong>Homonymous hemianopia</strong></td>
<td>Sudden, painless, complete or partial loss of half the visual field in both eyes, other neurological deficits including aphasia, motor weakness, sensory deficits</td>
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</table>

\(^a\)Presence of these characteristics may indicate the corresponding ophthalmic condition, and further workup and management should be performed accordingly. The signs listed for each condition are not pathognomonic and should only be used to help guide the differential diagnoses in relation to a patient.
<table>
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<tr>
<th>Condition</th>
<th>Presentation</th>
<th>Diagnosis and findings</th>
<th>Management</th>
</tr>
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<tbody>
<tr>
<td>Blunt ocular/open globe injury</td>
<td>Based on history</td>
<td>Hyphemia, subconjunctival hemorrhage, pupil irregularity, corneal or scleral laceration, protrusion of intraocular contents, flat or shallow anterior chamber, positive Seidel test.</td>
<td>Place protective shield over the injured eye. Administer broad-spectrum antibiotics, antiemetics, and analgesics. Tetanus prophylaxis in case of foreign body injury. Emergent ophthalmologic consultation. NPO status for patient in case of emergent surgery.</td>
</tr>
<tr>
<td>Chemical ocular injury</td>
<td>Based on history</td>
<td>Abnormal pH of the eye on presentation (acidic or basic depending on the chemical exposure).</td>
<td>Emergent irrigation with sterile isotonic saline or lactated ringers until a neutral pH is achieved is the most crucial step. If no pH strips are available, 10 to 15 L of fluid irrigation is recommended. Once the eye is stabilized, lubricants (i.e., erythromycin ointment) and cycloplegic eye drops (0.25% scopolamine) should be applied. Refer to ophthalmology after stabilization.</td>
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<tr>
<td>Orbital cellulitis</td>
<td>Reduced vision, periorbital erythema, and swelling. Systemic symptoms such as fever and malaise are also possible.</td>
<td>On exam, conjunctival injection and edema, proptosis, decreased vision, ophthalmoplegia (restrictive eye movement), and diplopia, afferent pupillary defect.</td>
<td>Emergent ophthalmologic evaluation to rule out orbital compartment syndrome, abscess, or optic neuropathy. Empiric antibiotic coverage with vancomycin plus cefotaxime and metronidazole or clindamycin.</td>
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<tr>
<td>Endophthalmitis</td>
<td>Usually in patients with recent eye surgery or procedure. Rapidly progressive pain and redness, ocular discharge, vision loss, and eyelid swelling.</td>
<td>On exam, conjunctival edema, hypopyon (layered collection of inflammatory cells in the anterior chamber of the eye), and decreased visual acuity.</td>
<td>Emergent ophthalmologic consultation for intravitreal antibiotic administration.</td>
</tr>
<tr>
<td>Acute angle closure glaucoma</td>
<td>Severe nausea and vomiting, ocular pain, headache, and reduced vision.</td>
<td>On exam, conjunctival injection, corneal haziness, mid-dilated pupil, reduced vision, and severely high intraocular pressure (normal: 12-22 mm Hg). If no tonometry is available, assess tactile pressure of the globe; if harder on palpation than the examiner’s globe, it may suggest acute angle closure glaucoma.</td>
<td>Urgent referral to ophthalmologist for intraocular pressure—lowering medications and surgical intervention.</td>
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<tr>
<td>Optic neuritis</td>
<td>Acute monocular vision loss (possible color vision changes) and pain with eye movement.</td>
<td>On exam, possible RAPD and optic disc swelling.</td>
<td>Refer for ophthalmologic evaluation. Refer to neurology for MRI of the brain and spine to rule out demyelinating lesions. Treat with 3-day intravenous methylprednisolone followed by an oral prednisone (1 mg/kg per day) taper.</td>
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<tr>
<td>Giant cell arteritis</td>
<td>Temporal redness and headache, jaw claudication, sudden painful vision loss,</td>
<td>Presence of three or more of the following: age older than 50 y, new-onset headache,</td>
<td>Immediate steroids (40-60 mg oral if no visual/neurologic symptoms and intravenous methylprednisolone if visual/neurologic symptoms present). Treatment should not be delayed for temporal artery biopsy. Urgent ophthalmologic evaluation.</td>
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<td></td>
<td>constitutional symptoms of fever, myalgias, night sweats, and weight loss.</td>
<td>temporal tenderness, ESR &gt; 50 mm/h, elevated CRP, or abnormal temporal-artery biopsy.</td>
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<tr>
<td>Central retinal artery occlusion</td>
<td>Sudden painless vision loss and history of cardiovascular risk factors.</td>
<td>On funduscopic exam, blanched, grayish retina; cherry-red spot at macula; and box-car appearance of retinal vessels may be present.</td>
<td>Full-dose aspirin and referral to emergency department for stroke evaluation, carotid ultrasound, MRI of brain, echocardiography, and 48-h Holter monitoring. No proven therapies currently.</td>
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<tr>
<td>Retinal detachment (rhegmatogenous type)</td>
<td>Persistent flashes and floaters, peripheral “shower of black dots” or “swarm of bees,” dark curtain-like shadow, and painless vision loss.</td>
<td>Pigmented granules in the anterior vitreous on slit-lamp exam. Using direct ophthalmoscope, funduscopic exam may reveal lack of red reflex, and wrinkled and pale retina if detachment near the macula. If the detachment is peripheral, it may not be visualized with the direct ophthalmoscope.</td>
<td>Emergent referral to an ophthalmologist.</td>
</tr>
<tr>
<td>Homonymous hemianopia</td>
<td>Sudden painless partial loss of the same side of visual field in both eyes.</td>
<td>Comprehensive history for comorbid conditions to ascertain stroke risk. Confrontational visual field exam to evaluate for other neurologic deficits that may suggest a stroke.</td>
<td>Referral to the emergency department if there is a high suspicion for a stroke based on complete neurologic exam.</td>
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*ESR = erythrocyte sedimentation rate; MRI = magnetic resonance imaging; NPO = nothing by mouth; RAPD = relative afferent pupillary defect.*
prophylaxis is also advised if the patient's immune status is unknown or inadequate. A computed tomography (CT) scan of the brain and orbits may be necessary to rule out a retained intraocular foreign body.

**Chemical Injury**
Ocular chemical injuries often result from splashes of products such as household cleaners, industrial chemicals, cement, or automotive fluids. Immediately upon presentation, the first step in treatment is continuous irrigation with sterile isotonic saline/lactated ringers (preferred) or tap water. Irrigation after chemical splash should be performed before official check-in to the emergency department and before other ocular evaluation and ophthalmologic consultation. If available, a Morgan device can be used to provide eye irrigation. Topical anesthetic drops such as proparacaine 1% can be used to reduce pain and facilitate proper irrigation. Using pH indicator strips or litmus paper, test the pH of the eye by gently touching the inside of the lower eyelid. Continued irrigation is recommended until a neutral pH (7.0) is achieved. Up to 10 to 15 L of fluid may be necessary to achieve pH neutralization; if litmus paper is not available to measure the pH, it is recommended that the eye be irrigated for at least 30 min. Overall, alkali burns are more harmful than acid burns due to their lipophilic nature allowing them to penetrate the eye more rapidly and thus accumulate in tissues.

Once adequate irrigation is completed, a detailed ophthalmologic examination should be performed to assess the degree of damage. Topical lubricants, steroids, antibiotic and cycloplegic eye drops (1% cyclopentolate) are helpful in healing and symptomatic management. Once the patient's eye is stabilized, an emergent ophthalmologic referral is recommended (Table 2). Complications from chemical burns include opacification of the cornea leading to visual deficits and chronic dry eye disease, glaucoma, and cataracts.

**PRESEPTAL VERSUS ORBITAL CELLULITIS**
Periorbital cellulitis or preseptal cellulitis results from extension of rhinosinusitis, infection, superficial skin infection, or facial trauma. It involves infection of the eyelids and surrounding soft tissues anterior to the orbital septum. Orbital cellulitis, or postseptal cellulitis, is more invasive and involves orbital soft tissues beyond the orbital septum, including fat, connective tissues, and muscles. It is crucial to distinguish the two types of cellulitis as management differs based on diagnosis, and in rare cases, orbital cellulitis can be vision- and life-threatening due to an orbital compartment syndrome, cavernous sinus thrombosis, meningitis, or brain abscess.

Patients with pre-septal cellulitis present with peri orbital erythema, edema, and eyelid swelling. However, vision, eye motility, or intraocular pressures are not affected. Orbital cellulitis may present with conjunctival injection and edema, proptosis, ophthalmoplegia with diplopia, pain and reduction in EOM motility, and decreased vision in addition to the periorbital erythema and swelling seen with pre-septal cellulitis. Systemic signs such as fever and malaise are more commonly associated with orbital cellulitis, although they can be occasionally present with pre-septal cellulitis (Table 2).

For both entities, a comprehensive ophthalmologic exam is recommended, including visual acuity testing, pupillary response, eye motility, and tonometry. However, in cases where it is difficult to distinguish the two, a CT scan of the orbits and sinuses is necessary. Inflammation of the EOM, fat stranding, and anterior displacement of the globe may be evident on the CT scan in patients with orbital cellulitis. If orbital cellulitis is diagnosed, the patient should have emergent ophthalmologic evaluation for presence of orbital compartment syndrome, optic neuropathy, or abscesses, which would warrant an emergent lateral canthotomy/cantholysis or surgical drainage of the abscess.

Antibiotic treatment should provide coverage against Staphylococcus, Streptococcus, and anaerobic species. Preseptal cellulitis can be managed in the outpatient setting and should be treated with trimethoprim-sulfamethoxazole or clindamycin plus amoxicillin, amoxicillin-
clavulanic acid, or cefdinir. Most cases of orbital cellulitis require hospital admission and treatment should include empiric coverage with intravenous vancomycin plus cefotaxime or ceftriaxone and metronidazole or clindamycin until intracranial involvement or sinusitis is ruled out. Response to therapy should be evident within 24 to 48 h of initiation of appropriate therapy, with continuation of treatment until all signs of cellulitis have resolved, and for at least 2 to 3 wks.7 In cases of pre-septal cellulitis where the patient worsens or does not improve within 24 to 48 h of treatment, a CT scan and possible hospitalization is warranted to evaluate for orbital cellulitis and complications.

**ENDOPHTHALMITIS**

Endophthalmitis refers to a bacterial or fungal eye infection involving the vitreous, retina, choroid and/or the anterior chamber. Most commonly, it is due to exogenous introduction of organisms after recent procedures such as intravitreal/intraocular injections and eye surgeries. Endogenous endophthalmitis is also possible due to seeding of an organism via the bloodstream. These cases are considered medical emergencies with delay in treatment possibly resulting in permanent vision loss.8

Patients present usually within 1 wk of a recent ocular procedure with rapidly progressive pain, red eye, ocular discharge, and vision loss. On examination, eyelid swelling, conjunctival or corneal edema, or hypopyon (collection of inflammatory cells in the anterior chamber of the eye) may be evident (Table 2). Emergent ophthalmic consultation is advised so that a more comprehensive ophthalmologic examination can be conducted, intravitreal samples can be obtained for culture and speciation, and intravitreal antibiotics can be administered.9,10

**ACUTE ANGLE CLOSURE GLAUCOMA**

Acute angle closure glaucoma results from narrowing of the anterior chamber angle, leading to decreased aqueous drainage from the posterior to the anterior chamber of the eye. This pressure differential leads to anatomical alterations in the iris and trabecular meshwork causing an elevation of intraocular pressure. The condition tends to be more common in women, patients older than 50 y of age, and those with a family history of angle closure glaucoma.11 An affected patient may present with severe nausea/vomiting, ocular pain, blurred vision, and headache, causing a migraine to be a differential diagnosis. However, signs of monocular symptoms, conjunctival injection, corneal haziness due to edema, mid-dilated pupil, and a sudden decrease in vision can help distinguish this from mimickers (Table 2). An urgent referral to an ophthalmologist is recommended so the patient can be treated with medications or surgery.12,13 Diagnosis is confirmed by measuring the intraocular pressure and performing gonioscopy, an examination of the angle between the iris and cornea.

**ACUTE PAINFUL LOSS OF VISION**

**Optic Neuritis**

Optic neuritis is often associated with multiple sclerosis but can also be infectious, inflammatory, para-vaccination immunological response, or due to autoimmune disease. The condition is most common in young adults and patients who are white.14

Patients often present with acute monocular vision loss that progresses over hours or days, pain with eye movement, and dyschromatopsia (reduced contrast in colors). On examination, a relative afferent pupillary defect (RAPD) may also be seen, although involvement of the other eye may mask this. On direct ophthalmoscopy, diffuse optic disc edema/swelling can be seen in one-third of cases. The diagnosis of optic neuritis is clinical based on the presence of the above symptoms. Patients should be referred for further neurologic follow-up and gadolinium-enhanced magnetic resonance imaging (MRI) of the brain and spine to rule out presence of demyelinating lesions and determine risk of developing clinical multiple sclerosis (Table 2).15

Treatment of optic neuritis has been most successful with intravenous corticosteroids. The Optic Neuritis Treatment Trial...
showed that patients receiving 3 d of high-dose intravenous methylprednisolone followed by an oral prednisone taper recovered vision faster compared with those treated with 14 d of oral prednisone (1 mg/kg per day). However, this difference equalized within 1 mo of treatment. Patients with oral regimen did have a two-fold greater rate of recurrent optic neuritis. Hence, initial treatment with oral prednisone 1 mg/kg per day alone is contraindicated in patients with optic neuritis.16

**Giant Cell Arteritis**

Giant cell arteritis (GCA), or temporal arteritis, occurs in individuals usually older than the age of 50 y. It is a granulomatous vasculitis that affects large and medium-sized arteries such as the ophthalmic artery. It requires urgent diagnosis as lack of early treatment and intervention could mean permanent vision loss (up to 15% to 25% of cases) from ischemic complications of the disease in the affected eye. Urgent treatment is also necessary to protect the unaffected eye, as it is at an elevated risk of disease involvement.17

In a classic case, the patient may present with temporal headache, jaw claudication, temporal tenderness, sudden/severe vision loss, low-grade fever, and other constitutional symptoms such as fatigue, myalgias, night sweats, and weight loss (Table 2).18 However, up to 20% of patients with GCA-related vision loss do not have systemic symptoms, making it difficult to make the diagnosis.17 Some patients may also have had symptoms of temporary vision loss, eye pain, or diplopia in the preceding weeks before presentation due to transient ischemia of the optic nerve head.

Per the American College of Rheumatology, diagnostic criteria for GCA include three or more of the following: age older than 50 y, new-onset headache, tenderness to temporal artery palpation or decreased pulsation, elevated erythrocyte sedimentation rate (ESR) greater than 50 mm/h, or abnormal temporal-artery biopsy. However, if the suspicion for GCA is high, treatment should not be delayed while awaiting temporal-artery biopsy.16 If lab testing or invasive biopsy cannot be obtained emergently in the outpatient setting, the patient should be referred to the emergency department for prompt evaluation. Workup should include obtaining a complete blood count, ESR, and C-reactive protein (CRP) level. Treatment involves initial oral prednisone dosing of 40 to 60 mg (approximately 0.75 mg/kg) per day if the patient is not having any visual or neurologic symptoms. In the presence of such symptoms, higher doses of 1 to 1.5 mg/kg of oral prednisone or intravenous methylprednisolone should be started.19

**ACUTE PAINLESS LOSS OF VISION**

**Central Retinal Artery Occlusion**

Central retinal artery occlusion (CRAO) is an ophthalmic emergency because irreversible ischemic damage to the retina can occur in as little as 90 min.2 It is considered to be an “ocular stroke” and the risk of an ischemic stroke with large vessel involvement is particularly increased in the first 1 to 4 wks after diagnosis of a CRAO.20 Patients may present with sudden, painless decrease in vision in one eye over a matter of seconds. A patient may have partial vision loss if one of the smaller distal arteriolar branches is affected. Further history may reveal prior episodes of intermittent transient, painless vision loss. Risk factors for CRAO are similar to those for other cardiovascular events and include cigarette smoking, hypertension, high body mass index, high serum lipid levels, diabetes, and cardiac disease. Additionally, hypercoagulable conditions such as antiphospholipid antibody syndrome and autoimmune conditions can also increase one’s risk.21 On examination, RAPD, blanched, non-perfused retina, and a “cherry-red spot” due to bright red appearance of the fovea on funduscopic examination may be evident. However, if the patient presents in the early stages, these signs may not be present and may take several hours to develop. A “box-car” pattern can also be seen in the retinal arterioles and veins due to the slow segmental blood
there are no proven therapies for retinal arterial occlusions. digital massage to dislodge the clot to a distal branch and reduce the ischemic area, anterior chamber paracentesis, and carbogen inhalation (4% to 7% carbon dioxide, 93% to 96% oxygen) have all been suggested as treatment options, although visual outcomes are generally poor. patients with an acute CRAO should be initiated on full dose of aspirin and referred to the emergency department for carotid imaging and neurology evaluation. They should undergo an MRI of the brain, echocardiography, and 48-h Holter monitoring within 1 to 2 wks of presentation. Additionally, all patients with CRAO and age older than 60 y, or age older than 50 y with headache, scalp tenderness, or jaw claudication should also be evaluated for GCA with lab workup including complete blood count, CRP, and ESR. Recommendations for patients with CRAO include diet alterations, smoking cessation, and initiation of a statin in the case of dyslipidemia.

Retinal Detachment

Retinal detachment is defined as separation of the neurosensory retina from the attached retinal pigment epithelium underneath. Although it is a rare condition, it is considered an emergency given that early diagnosis and treatment can prevent ischemic degeneration of photoreceptors and permanent visual impairment.

Patients may present with bright flashes peripherally in one eye, followed by a “shower of black dots” or a “swarm of bees,” and decreased visual acuity (Table 2). Others may complain of brief, recurrent flashes and floaters or a dark curtain or shadow moving from their peripheral to central vision. If the macula is affected, patients may also lose central vision or (rarely) total light perception. Risk factors for developing a retinal detachment include age (due to age-related posterior vitreous detachment [PVD]), high myopia, trauma, diabetes (specifically a risk factor for tractional retinal detachment), lattice degeneration, previous cataract surgery, family history of retinal detachments, and a personal history of previous detachment. Congenital-developmental anomalies such as Stickler syndrome, Marfan syndrome, and Ehlers-Danlos syndrome are also associated with retinal detachments.

On examination, visual acuity should be measured before pupillary dilation. If a slit lamp is available, the appearance of pigmented granules in the anterior vitreous (seen on an oblique slit beam) is suggestive of a possible detachment. A direct ophthalmoscope can be used to visualize a red reflex, lack of which may indicate a retinal detachment or vitreous hemorrhage. Given the narrow view of the direct ophthalmoscope, a detachment near the macula (area surrounding the fovea that is responsible for producing detailed vision) will be wrinkled, opaque, and pale. However, indirect ophthalmoscopy is necessary to fully evaluate the central and peripheral retina for a break. If a retinal detachment is suspected, it is crucial for the patient to be referred to a surgical vitreoretinal specialist so it can be repaired urgently.

PVD, which is an age-related process of degeneration and shrinkage of the vitreous, is physiologic and common in individuals after the age of 40 y. PVD is more likely to be the cause of flashes and floaters, but a patient with these symptoms should still be referred to an ophthalmologist for a dilated eye exam.

Homonymous Hemianopia

Homonymous hemianopia (HH) refers to a visual field defect with same-sided vision loss in both eyes. Depending on the area of the neuro-ophthalmic tract that has been affected, a partial or complete HH can occur. Sudden visual loss in this pattern can be a result of many different etiologies including a tumor and seizure; however, stroke is the most common cause and thus, a prompt evaluation is necessary. In high-risk elderly or post-stroke patients, a confrontational visual field exam and a general neurologic exam should be performed to assess for a possible stroke (Table 2). A good history and evaluation of comorbid conditions that increase stroke risk can help ascertain the possibility of a neurologic event. If the HH...
or any other type of visual field defect is acute or subacute, the patient should be promptly referred to the emergency room for workup of a neurologic event.26

CONCLUSION
Early recognition of ocular emergencies is crucial to preserve visual acuity and achieve the best overall ocular and systemic outcomes. By providing a concise summary and approach to common ophthalmic emergencies, we hope that clinicians can triage and manage patients with acute eye complaints in the primary care setting with ease.

SUPPLEMENTAL ONLINE MATERIAL
Supplemental material can be found online at: http://www.mayoclinicproceedings.org. Supplemental material attached to journal articles has not been edited, and the authors take responsibility for the accuracy of all data.

Abbreviations and Acronyms: CRAO = central retinal artery occlusion; CRP = C-reactive protein; CT = computed tomography; EOM = extraocular muscle; ESR = erythrocyte sedimentation rate; GCA = giant cell arteritis; HH = homonymous hemianopia; MRI = magnetic resonance imaging; PVD = posterior vitreous detachment; RAPD = relative afferent pupillary defect

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REFERENCES