## Ophthalmology – Acute Visual Loss

**SOAP Note**
**with Sanjay Sharma M.D.**

### INTRODUCTION

Acute visual loss is a medical emergency because there are some conditions that cause this symptom can quickly cause permanent blindness – like retinal detachment and wet macular degeneration – and it is associated with one condition which is potentially lethal – giant cell arteritis.

### BASIC SCIENCE PEARLS FROM A CLINICIAN

- The retina is a 10-layer tissue which functions to absorb light energy, and convert it to electrical impulses – a process called phototransduction. The cells in which phototransduction occur are called photoreceptors. There are two photoreceptors: **rods** and **cones**. Rods, the most prevalent type of photoreceptor, are peripherally located and are responsible for night-time vision. Cones, seen in very high concentration in the macula, are responsible for both colour and the ability to see fine detail. The process of phototransduction is dependent on a series of chemical reactions, which involve **vitamin A**. Deficiencies in vitamin A can lead to a degeneration of the retina which can manifest as pigmentary retinopathy and vascular narrowing.
- Embryologically, the neurosensory retina arises from brain tissue at the **3rd week of gestation** after neural ectoderm comes into contact with surface ectoderm. With this, the optic vesicle is formed, and the neural ectoderm folds back over itself such that the neurosensory retina occupies the inner fold and the retinal pigment epithelium (RPE) the outer. This is very important because there is fundamentally a cleavage point that exists between the photoreceptors and retinal pigment epithelium. In many pathologic processes, including a retinal detachment in which a full thickness break in the neurosensory retina allows fluid from the vitreous to seep under the retina, the photoreceptors can quickly separate from the underlying RPE. This easy to create cleavage plain is also why subretinal fluid (fluid by located between the photoreceptors and the RPE) may also may be seen in wet macular degeneration, retinal vein occlusion and diabetic retinopathy.
- **Vascular Endothelial Growth Factor (VEGF)** is a molecule that is upregulated in many retinal conditions that present with acute visual loss, including wet macular degeneration, diabetic retinopathy, and retinal vein occlusion. It is important to recognize that VEGF is not only involved in the process of angiogenesis (new blood vessel creation arising from either the retina itself of the choroid located just below the RPE), but also vessel permeability. Specifically, when upregulated, vessels can become hyper permeable. Treatment paradigms to block VEGF have become the cornerstone of treating macular edema related to diabetic retinopathy, vein occlusion and macular degeneration. Anti-VEGF agents also cause regression of neovascularization, which is important in managing wet macular degeneration and proliferative retinopathy.

**SUBJECTIVE**

When presented with a patient with acute visual loss, there are some key questions that you must ask. Here is my key list of questions that I ask any patient with acute visual loss, and why I ask them:

- **How profound is the visual loss?** Mild blurring is not as important as an absolute scotoma (or pure black areas in the vision); absolute blackness is worrisome.
- **What was the temporal nature of the visual loss?** If transient visual loss think of things like amaurosis fugax and migraine; if permanent think of retinal detachment, vascular occlusion, macular degeneration or anterior ischemic optic neuropathy.
- **Qualitative nature of visual loss** – if sudden and altitudinal loss, think of an ischemic event to the optic nerve; if it appeared like a "blind going down" for 5 minutes and they rising again, think of amaurosis fugax and rule out a carotid artery lesion; if described as distortion, think of wet macular degeneration, macular edema or macular hole/epiretinal membrane.
- **Was the visual loss monocular or binocular?** If binocular and simultaneous, the visual loss is at or behind the ocular chiasm.
- **Was the visual loss preceded by or associated with any symptom?** If preceded by flashing lights or new onset of floaters, retinal detachment must be ruled out. If associated with weight loss, arthralgias or jaw claudication you must rule out giant cell arteritis. If associated with nausea, vomiting and a brow ache – think of angle closure glaucoma. If associated with scintillating scotomas lasting 5-20 minutes and followed by a headache, think migraine.
- **Any known past medical history?** Diabetics are predisposed to new vessels which can bleed into the vitreous or cause a tractional retinal detachment; hypertensives are at higher risk for the development of retinal artery and vein occlusion; coagulation problems are linked to retinal vein occlusion; carotid artery stenosis, artial fibrillation and cardiac valvular disease can all raise the risk for embolic retinal artery occlusion.

**OBJECTIVE**

On examination, there are a few things that must be done as a primary care physician.

- **Measure visual acuity** – best to formally measure accurately with snellen or visual acuity card, but even crude measures are very useful. Can the person read small newspaper print? large newspaper print? count your fingers? perceive movement? see light?
- **Check pupils and examine for a relative afferent papillary defect.** If an afferent defect is present, this is an optic nerve problem until proven otherwise.
- **Look at the anterior segment** (even if you don't have access to a slit lamp). Is the eye red or white? If red, is a ciliary flush present (if it is think keratitis, iritis or angle-closure glaucoma). Is the globe (eye) intact or is there a rupture (this is important in the setting of trauma). If you see a "peaked" pupil, this is an open eye until proven otherwise.

- **What is the eye pressure?** Even if you don't have access to a tonometer, if the eye is very firm angle closure glaucoma must be ruled out.
- **Is the cornea clear?** When a penlight is shone on a cornea it should reflect a very sharp reflex. In corneal edema (which can be present for a number of reasons including angle closure glaucoma and iritis) the corneal light reflex can be very dull.
- While the anterior segment exam is important most cases of acute visual loss are retinal in origin. **This means that a thorough exam of the retina is critical.**

**ASSESSMENT AND PLAN**

While many conditions can cause acute visual loss, because it can be caused by retinal detachment, angle closure glaucoma, wet AMD and giant cell arteritis, all cases of acute visual loss **MUST** be emergently referred. Here are some key things to remember about the most worrisome causes of acute visual loss, including how these conditions may be managed by an ophthalmologist (these are the A and P in SOAP):

1. **Angle closure glaucoma.** Think of this with sudden visual loss and a red eye, especially if nausea and vomiting are present. It may be precipitated by being in a dark room. Emergent referral is necessary to perform peripheral iridotomy as the optic nerve will be destroyed within hours of having very high eye pressures.[1, 2]
2. **Retinal vein occlusion** – think of this in typical "blood and thunder" appearance (hemorrhage in all 4 quadrants); may have a swollen disc; at high risk for developing neovascular glaucoma; injections of ant-VEGF antibodies lower risk of neovascularization and dry up macular edema. Patients may also require laser if the retinal vein occlusion causes neovascularization (new blood vessel growth on the retina or disc) or if neovascular glaucoma (high pressures in the eye caused by new vessels plugging up the eye's drainage system in the anterior chamber) develops.[3,4]
3. **Central retinal artery occlusion** – think of this with cloudy swelling and a cherry-red spot; may be linked to carotid artery stenosis or cardiac pathology; patients may benefit from sudden lowering of eye pressure (needle into front of eye to suddenly lower pressure by removing aqueous fluid).[5,6,]
4. **Retinal detachment** – most have posterior vitreous detachment and have preceding symptoms of floaters and flashes; need emergent attention – laser or freezing (cryotherapy) to seal hole, injection of gas into eye (pneumatic retinopexy), surgery to outside of eye (scleral buckle) or to inside of eye (vitrectomy).[7]
5. **Wet Macular degeneration** – think about this in an elderly patient new distortion. Patients have new vessels in the macula and present with blood and fluid. Treated with eye injections with anti-VEGF antibodies.[8,9,10]
6. **Giant cell arteritis** – this is a critical diagnosis to consider in all patients with acute visual loss. Especially consider in patients who are elderly, who appear systemically unwell (arthralgias, weight loss, jaw claudication, may have temporal tenderness). Patients need stat ESR and CRP; low threshold to start steroids; definitive diagnosis made on temporal artery biopsy (giant cells and epitheloid cells). This is a systemic disease of medium arteries and can quickly develop blindness in other eye or other vessels. Can be lethal![11]

**SUMMARY**

In summary, acute visual loss is an extremely important condition for you to know about because patients can quickly go blind if not treated and those with GCA can die from their underlying condition.

**References**


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