433 Teams

OPHTHALMOLOGY

3

Acute Visual Loss

Color index:

432 Team – **Important** – **433 Notes** – **Not important**

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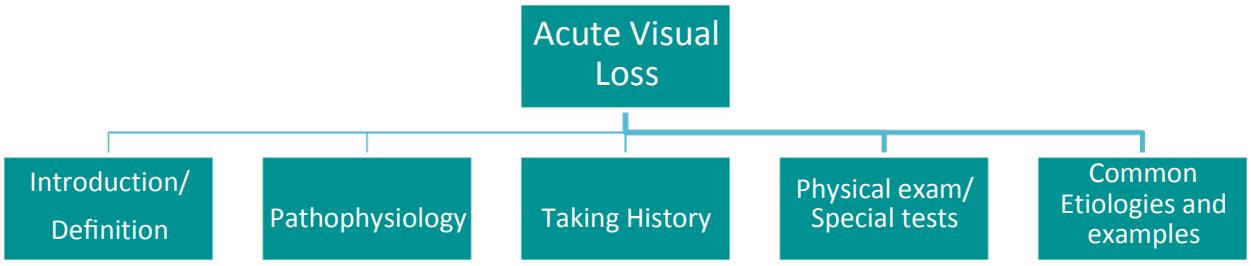
Objectives:

The student should be able to:

1. Properly screen and evaluate patients presenting with acute visual loss
2. Understand the pathophysiology and identify common causes of acute visual loss.
3. Recognize situations requiring urgent ophthalmic care to prevent permanent visual loss.



Acute Visual Loss

Definition:

Loss of vision is usually considered acute if it develops within a few minutes to a couple of days (up to a week)

--- It may affect one or both eyes.

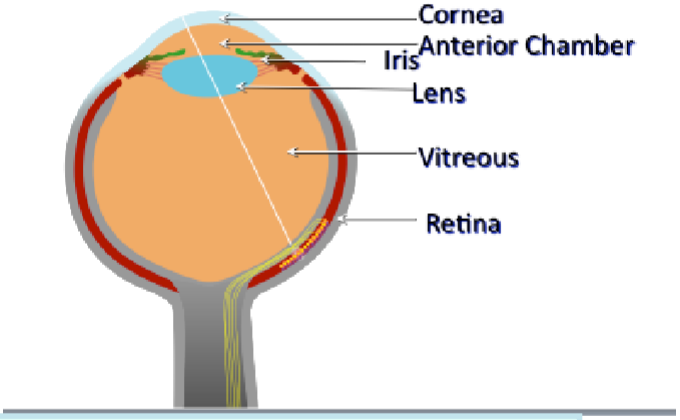
--- All or part of the visual field.

--- Arise from pathology of any part of the visual pathway

430 Team:
--- Chronic visual loss > slowly progress
--- Acute visual loss > sudden (seconds or minutes to few days)
--- Acute visual loss not necessary means complete loss.

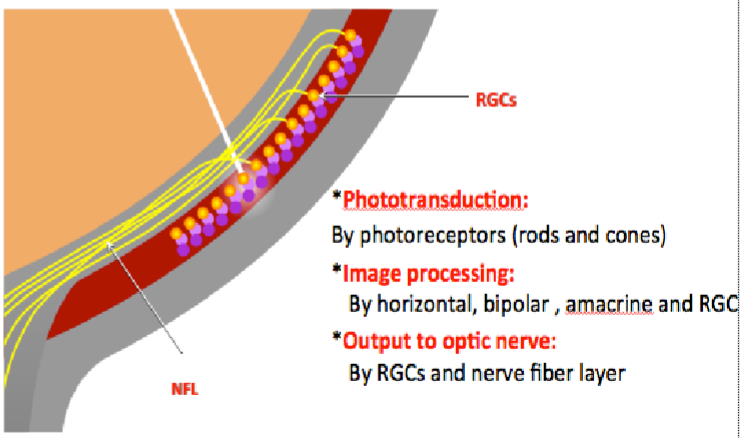
The Visual Pathway:

Light enters the eye via the refractive media, namely the cornea, anterior chamber, lens, and vitreous, and stimulates the retina posteriorly.

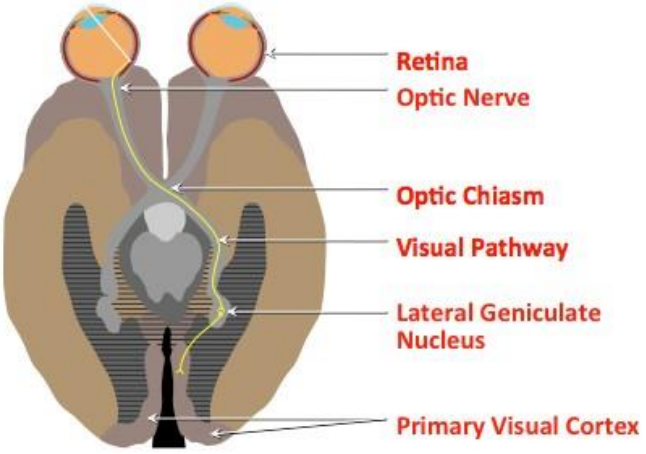


The incident must be through the pupil to the fovea (the smallest part of the macula & the most sensitive part in the retina), so anything that can affect any part of visual pathway can lead to visual loss (which can be mild, moderate, severe, or even complete)

Light stimulates the photoreceptors, ie., the rods and cones. Through a series of other retinal nerve cells, the end result is that the RGC is stimulated. The RGC sends its axon, or fiber, in the nerve fiber layer to the optic disc and then down the optic nerve.



From the optic nerve, about half of the fibers cross over at the chiasm to the opposite optic tract, and the other half remains on the same side. The fibers in the optic tract synapse in the lateral geniculate nucleus of the thalamus. Neurons in the lateral geniculate nucleus then project to the occipital lobe, to the primary visual cortex. From there, there is further processing with projections to other cells in the visual cortex and elsewhere, resulting in conscious visual perception.



The light is absorbed by the photoreceptors (rods & cons) >> chemical reaction in the photoreceptors which creates neuro-electrical impulse that is carried by the bipolar & amacrine cells to the retinal-ganglion cells >> then through their axons "nerve fiber layer" (inner most layer of the retina) from all the parts of the retina >> these fibers conjoint at the posterior part of the eye through the lamina cribrosa and exit as optic nerve head >> transmit these impulses to the brain (to the visual cortex)

(Very important)

What are the important questions to ask in history for a patient with acute visual loss?

1. Is the visual loss transient or persistent? (If transient you can exclude retinal detachment & central retinal artery occlusion)

2. Is the visual loss monocular or binocular? (If monocular; the cause is not cortical "but something before the cortex")

3. Did the visual loss occur suddenly or it developed over hours, days or weeks?

4. What is the patient's age and general medical condition?

5.Did the patient have normal vision in the past and when was vision last tested (because it could be a chronic loss of vision but the patient just discovered it)

6. Some people will only realize loss of vision from one eye; when they cover the good eye.

7. Is it painful or painless? (extremely important)

Transient or persistent:	Migraine, TIA vs. Retinal detachment
Monocular or binocular:	Optic neuritis vs. Cortical blindness
Hours, days or weeks:	CRAO, stroke vs. Retinal detachment, glaucoma
Patient's age:	Acute Glaucoma (if old) vs. Corneal abrasion, optic neuritis (if young)
Contact lens use:	Corneal ulcer
Painless visual loss:	Vitreous hemorrhage, retinal detachment, CRAO
Painful visual loss:	Acute glaucoma, Keratitis

Causes of Acute Visual Loss:

PAINFUL	PAINLESS
Acute (congestive) Glaucoma	Vitreous Hemorrhage (it can be painful if it was traumatic)
Uveitis	Retinal Detachment (the patient may have it & not discover it unless he covers one eye)
Keratitis	Retinal vascular occlusions (arteries/veins)
Hyphema (Traumatic) (Hyphema is a collection of blood in the anterior champer)	Optic neuritis + (Ischemic optic neuritis is usually associated with temporal arteritis)
	Ischemic optic neuropathy
	CVA
	Functional

Physical Exam and Special Tests:

1) **Visual acuity testing** (in CRAO there will be NO light perception)
2) **Confrontation visual fields test** (to differentiate between neurological "respect midline" & non-neurological causes)
3) **Pupillary reactions** *extremely important (will help to decide at what level is the pathology) – Afferent: optic nerve. Efferent: occulomotor

4) **External examination of the eye with a pen light**
5) **Slit lamp examination**
6) **Direct Ophthalmoscopy exam** "especially red reflex" (will help in limiting the possible causes) – if you can't appreciate a red reflex, the cause is either vitreous hemorrhage or retinal detachment
7) **Tonometry to measure the intraocular pressure**

--- Any lesion affect optic nerve from the retina until the lateral geniculate body should have afferent pupillary defect
-Tonometry: normal= 10-21 mm Hg, ocular HTN= 22-29, glaucoma: ≥ 30)

Causes of Acute Visual Loss:

Common causes

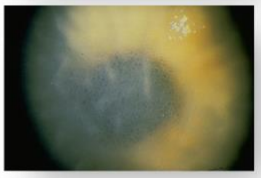
1) Corneal ulcer
2) Acute angle closure glaucoma
3) Central retinal artery occlusion
4) Central retinal vein occlusion
5) Retinal detachment
6) Vitreous hemorrhage
7) Optic neuritis
8) Occipital stroke

Common causes based on patient's age

1) Wet, age related macular degeneration
2) Commotio retinae (edema of the macula caused by blunt trauma) – seen in young patients
3) Rupture globe (seen in young patients)
4) Orbital cellulitis (seen in both young & old patients)

1) Media opacities. The problem could be in the cornea, anterior champer, lens "unlikely unless it ruptures", and vitreous

A. Corneal edema:

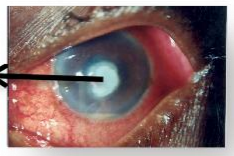


- **When the cornea appears like ground glass** rather than its normal clear appearance. (The cornea normally appears crystal clear unless there is a pathology)
- The most common cause of corneal edema is increased intraocular pressure typically in angle closure glaucoma. (most commonly in acute congestive glaucoma)
- Increased intraocular pressure >> dysfunction of corneal endothelium >> fluid leaks into the stroma >> edema >> hazy cornea
- ⁉️ Any acute infection of the cornea resulting in a corneal ulcer
  - may mimic corneal edema
- ⁉️ We don't see corneal edema in open angle glaucoma

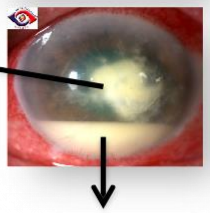
B. **Corneal ulcer:** (painful)

- When there is a corneal opacity due to destruction of tissue by infiltration of microorganisms and WBCs.
- Could be viral, bacterial, fungal, protozoal
  - or neurotrophic in etiology

Ulcer + loss of clarity of the cornea

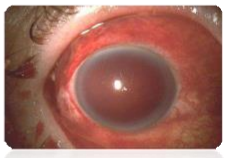


Infiltration deep in the cornea (not penetrating the cornea)



Fluid level (hypopyon). Content: WBC + pus accumulating in the anterior champer

C. **Hyphema:** (painful, especially total hyphema)



- **Hyphema is blood in the anterior chamber** not in the
  - cornea (beyond the cornea)
- The hyphema is a direct consequence of blunt trauma to a
  - normal eye.
- However, it can occur with tumors, diabetes,
  - intraocular surgery and chronic inflammation
  - which all cause neovascularization of the anterior segment.
- In proliferative diabetic retinopathy they may have vessels

--- Less than total "up to 2/3rd" (benign condition)

--- very minimal increase in intraocular pressure

on the iris (rubeosis iridis) abnormal fragile vessels can break and cause hemorrhage > acute visual loss. (430 team)

Usually resolve spontaneously within a few days

Management: observe the patient & monitor the intraocular pressure (IOP)

We can tolerate an IOP of 50 mmHg in a patient with no sickle cell disease, but if the patient has a sickle cell disease, an IOP of 25 mmHg is considered risky to leave them without intervention because they can develop a central retinal artery or vein occlusion

If the IOP is elevated in patient with sickle cell disease >> you give anti-glaucoma medications & monitor the pressure (shouldn't exceed 30 mmHg)

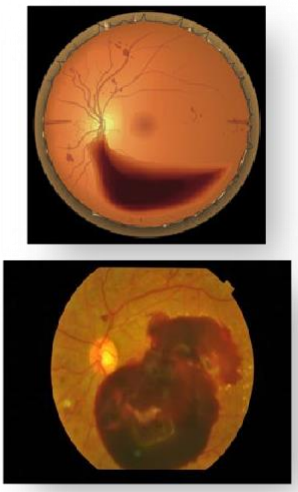
In patients with no sickle cell disease >> you can wait for 1-2 weeks & only monitor the pressure

Total hyphema + high IOP >> the patient may have corneal blood staining >> very dangerous; develops very quickly (within hours to a day) and take years to resolve (2-3 years) and may end up with a corneal scar.
In children (less than 10 years) if this happens >> you need to do penetrating cranioplasty, otherwise the child will have deep amblyopia (non-organic drop of vision = lazy eye)

D. **Vitreous hemorrhage:** (painless)

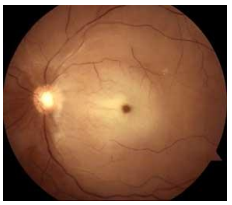
Any bleeding into vitreous cavity will reduce visual acuity. Depends on the density of the vitreous hemorrhage
Can result from: Trauma, Diabetic retinopathy (most common cause in our population) or Retinal vascular occlusion.
Rarely, can accompany subarachnoid hemorrhage.
If you cannot appreciate a red reflex with an ophthalmoscope
B scan ultrasound is important to know the etiology.

Pre-vitreous hemorrhage



2) Retinal diseases

A. Retinal vascular occlusions:

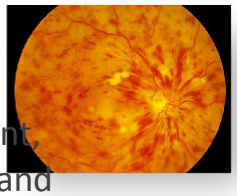


I. Central Retinal artery occlusion:

(Most serious one)

- A sudden, painless and often complete visual loss may indicate central retinal artery occlusion.
- Several hours after a central retinal artery occlusion, the inner layer of the retina becomes opalescent (white).
- A cherry red spot is seen due to the pallor of the perifoveal retina in contrast to the normal color of the fovea (the thinnest part in the retina & it has no nerve fiber layer & it reflects the choroid vasculature).
- A chronic cherry red spot is also a feature of the storage diseases such as Tay-Sachs disease and Niemann-Pick disease.
- There is no generally accepted acute management. (Retina dies in 90 mins, we can only do a workup for the patient to look for the cause)

II. Central Retinal vein occlusion:



(not ocular emergency, never cause absent light perception)

- Ophthalmoscopes picture of disc swelling, venous engorgement, cotton wool spots and diffuse retinal hemorrhages like blood and thunder.
- Afferent pupillary defect (++++) > in ischemic type
- Loss of vision may be moderate to severe. (In non-ischemic type
  - with no macular edema it could be asymptomatic)
- Treatment should be directed at reducing associated macular edema by injecting anti-vascular endothelial growth factor agents. If ischemic, you can also do panretinal photocoagulation
- Visual prognosis depends on degree of associated retinal
  - ischemia.
- It can be: ischemic (more severe & more permanent acute visual
  - loss) or non-ischemic

B. Retinal detachment:

It is retinal splitting, and it happens between 2 layers, the neurosensory retina and retinal-pigmented epithelium. In normal retina there is no actual connection or junction between them. It is a potential space, it is firm and adherent. When retina gets break, fluid come between the 2 layers and separates them. (430 team)

- **Could be macula on** (very good prognosis) **or macula off** (if intervention within 10 days > good prognosis. And if delayed intervention > poor prognosis)
- Complain of flashing lights, large number of floaters, shade or
- curtain covering the visual field.
- An afferent pupillary defect (pupil examination is very
- important here)
- The diagnosis is confirmed by ophthalmoscopy through a dilated pupil, and retina appears elevated with folds and the choroid background behind the retina is indistinct.
- It has 2 types:
  - Rhegmatogenous: there is a tear in the retina (due to trauma or vitreous detachment)
  - Non-rhegmatogenous: no tear in the retina (due to vitreous hemorrhage)

3) Optic nerve disease

Optic Neuritis: (most common cause)

- Optic Neuritis is inflammation of the optic nerve.
- It is usually associated with multiple sclerosis and could be the first clinical manifestation.
- Visual acuity and color vision are markedly reduced with a positive afferent pupillary defect. (It's a MUST to have afferent pupillary defect to diagnose optic neuritis)
- Associated with pain on extra-ocular muscle movement in 90% of patients.
- The optic disc could be hyperemic and swollen (Optic papillitis), but usually appears normal.
- The visual acuity usually recovers.
- However, repeated episodes of optic neuritis may lead to permanent loss of vision.

► 99% of the time patients recover completely even without treatment

430 Team:
Inflation of the optic nerve is called **optic neuritis.** One of optic neuritis symptoms that it is painless BUT retrobulbar neuritis could be painful with ocular motility.
Inflammation of the optic nerve head is called **papillitis**

How to differentiate between *papilitis* and *papillary edema*?
Both have fuzzy margins and engorgements, but in *papilitis* there will be a decrease of vision while the vision in *papillary edema* is not affected.

4) Visual pathway disorders

A. Homonymous hemianopia: is loss of vision on one side of both visual fields

- ► May result from occlusion of one of the posterior cerebral arteries with infarction of the occipital lobe.
- ► Other vascular abnormalities occurring in the middle cerebral artery distribution may produce a hemianopia, but usually other neurological signs are prominent.
- ► Any patient with a hemianopia needs at CT or MRI to localize and identify the cause.

430 Team:
Visual pathway: optic nerve optic chiasm tract radiation then cortex. Studying the visual failed will help to localize the site of lesion.
For example: in pituitary adenoma there will be compression on the chiasm ⁉bilateral temporal hemianopia.
Affected optic nerve visual field affected in one eye.
Affected optic tract visual field affected in both eyes And so on, each lesion has its visual failed impact.

B. **Cortical Blindness:** (very rare condition)

- ► **A rare bilateral extensive damage to the cortical visual** pathways results in complete loss of Vision.
- ► This condition is referred to as cortical, central or cerebral blindness.
- ► As the pathways serving the pupillary lights reflex are spared, the patient who is cortically blind has normal pupillary reactions.

► Therefore, a patient with normal fundus examination along with normal pupillary reactions, most likely has cortical blindness..

5) Functional Visual loss

- Functional visual loss describes vision loss due to hysterical or malingering reasons. i.e.: not explained by organic basis. (malingering reasons can be picked up by examination)
- A patient may report complete blindness in one eye and normal vision in the other eye, and have no relative afferent pupillary defect.
- Various techniques exist to confirm functional visual loss.

Summary

Loss of vision is usually considered acute if it develops within a few minutes to a couple of days.

1. It may affect one or both eyes.
2. All or part of the visual field.
3. Arise from pathology of any part of the visual pathway
4. Taking good history and considering the anatomy of the visual pathway is the key for proper evaluation of the patient with acute visual loss.

MCQs:

1. A 69-year-old woman presents with acute onset of ocular pain, decreased vision, and halos around lights in the right eye associated with nausea and vomiting. The most likely diagnosis is:

A) Primary open-angle glaucoma
B) Lens induced glaucoma
C) Pigmentary glaucoma
D) Acute primary angle-closure glaucoma

2. A 30 -year-old woman presents with sudden vision loss of the right eye and mild pain on upgaze movement. Examination reveals that vision is 20/50 on the right and 20/20 on the left. There is a +RAPD on the right and a Visual field testing showed an inferior altitudinal defect on the same side. The left side is normal. Optic discs and fundi are normal in both eyes. What is the most likely diagnosis?

A) Branch retinal vein occlusion
B) Anterior ischemic optic neuropathy
C) Retrobulbar optic neuritis
D) Compressive optic neuropathy

3. A 23 --- year --- old female presents with loss of vision in the right eye over 3 days, she also complains that the right eye is painful when she moves it. She is otherwise fit and well, with no past ocular or medical history. Examination reveals acuity of counting fingers in the right eye, 6/6 in the left. The eye is white, the pupils equal and reactive to light, but a right relative afferent pupillary defect is present. Examination of the fundus is normal. What is the most likely diagnosis?

A) Central retinal vein occlusion
B) Acute glaucoma
C) Optic neuritis
D) Posterior cerebral artery occlusion

4. A 72 --- year --- old man with a previous diagnosis of glaucoma presents with a sudden loss of vision in the right eye. There is no pain. He is hypertensive. There is a family history of macular degeneration. Examination reveals a visual acuity of counting fingers in the right eye, 6/6 in the left. The eye is white, intraocular pressure is not raised. The pupils are equal and no relative afferent pupillary defect is present. Dilated fundoscopy reveals a swollen optic disc and multiple hemorrhages scattered over the retina. The retinal veins appear dilated and tortuous. What is the most likely diagnosis?

A) Central retinal artery occlusion
B) Central retinal vein occlusion
C) Retinal detachment
D) Giant cell arteritis

Answers

Q1: D
Q2: C
Q3: C
Q4: B

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