

Management of Acute Vision Loss

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ABSTRACT

Acute visual loss is a mutual complaint with variable presentations amid patients of different ages. The degree of difference diagnoses of vision loss is immense. Generally, monocular vision loss regularly specifies an ocular problem. Binocular vision loss is commonly cerebral in origin. Monocular vision loss can respect the horizontal midline. Binocular vision loss can respect the vertical midline. Many diverse causes of sudden visual loss are recognized; though, the most common cause for painless visual loss is ischemia. Vision loss with positive scotoma may be seen with migraine. Vision loss with a negative scotoma may be seen with amaurosis fugax. Ischemia, often via mechanical obstruction, can affect any aspect of the visual system. Those who develop ischemia of the eye often have other evidence of atherosclerotic disease, such as coronary artery disease and peripheral vascular disease, which increases their susceptibility to ischemic events in other parts of the body. Risk factors include smoking, hypercholesterolemia, and hypertension.

Keywords: Vision Loss, Migraine, Retinal detachment, retinal vein occlusion.

INTRODUCTION

Acute visual loss is a mutual complaint with variable presentations amid patients of different ages. The degree of difference diagnoses of vision loss is immense. Generally, monocular vision loss regularly specifies an ocular problem. Binocular vision loss is commonly cerebral in origin^[1]. Monocular vision loss can respect the horizontal midline. Binocular vision loss can respect the vertical midline. Some patients define their symptoms as a progressively descending gray-black curtain or as clouding, fogging, or dimming of vision. Symptoms commonly last a few minutes but can persist for hours. Variation in frequency ranges from a single episode to many episodes per day; recurrences may continue for years but more frequently occur over seconds to hours. Several diverse causes of sudden visual loss are recognized; though, the most common cause for painless visual loss is ischemia^[2].

Vision loss with positive scotoma may be seen with migraine. Vision loss with a negative scotoma might be seen with amaurosis fugax. Ischemia, often via mechanical obstruction, can affect any aspect of the visual system. Those who develop ischemia of the eye often have other indication of atherosclerotic disease, for example, coronary artery disease and peripheral vascular disease, which increases their susceptibility to ischemic events in other parts of the body. Risk factors contain smoking, hypercholesterolemia, and hypertension. Other etiologies of sudden

visual loss include infection, inflammation, trauma, vasculitis, mechanical dysfunction, and idiopathic causes^[3].

For any patient with sudden visual loss, the following information should be obtained:

- Age
- History of trauma
- Whether one eye or both eyes affected
- Symptoms - Photophobia, headache, pain
- Duration of visual loss or changes
- Prior episodes/ophthalmologic history

It is important to ask about comorbid conditions for example, hypercholesterolemia arrhythmia, hypertension, cancer, collagen vascular disease, hematological disorders, or medication use^[4].

Funduscopy and visual field testing can be challenging and, when negative, cannot completely rule out retinal detachment, as the retina is only partly visualized with these approaches. If accessible, ultrasound is a valuable adjunct to the physical examination of the eye. When the fundus cannot be visualized, ocular ultrasonography can reveal retinal detachment, vitreous hemorrhage, vitreous detachment, ocular tumors, increased intracranial pressure, retrobulbar hematoma, and intraocular foreign bodies^[5]. Retinal detachment is evident by a taut, linear opacity seen in the vitreous chamber that moves in conjunction with eye movement. Vitreous detachment appears as an opaque line separated from the retina that floats in the vitreous humor.

Vitreous hemorrhage appears as curved strands connecting with the retina as the eye moves. Severe vitreous hemorrhage causes complete opacification of the vitreous chamber. The examination must also contain complete cardiac and neurologic evaluation, including murmurs and carotid bruits.

The study was done after approval of ethical board of Northern Border university.

Pathophysiology

Ischemia compromises cell metabolism by decreasing delivery of oxygen and other essential nutrients to tissues. The consequential functional shortage can be temporary or permanent, depending on the degree of injury. Terminology of eye ischemia as given by Hedges and others (Table 1) ^[1,6,7] :

Table 1: Terminology of eye ischemia

Transient monocular visual loss (TMVL) or transient monocular blindness (TMB) - A more persistent vision loss that lasts minutes or longer
Ocular infarction - Persistent ischemic damage to the eye, resulting in permanent vision loss
Transient visual obscuration (TVO) - Episodes lasting seconds that are associated with papilledema and increased intracranial pressure
Transient bilateral visual loss (TBVL) - Episodes affecting one or both eyes or both cerebral hemispheres and causing visual loss
Amaurosis fugax - Brief, fleeting attack of monocular partial or total blindness that lasts seconds to minutes

Management of Acute Vision Loss

• **Surgical Management**

Carotid artery stenosis expands the danger of hemispheric stroke. This hazard is more prominent after hemispheric ischemic symptoms than after retinal ischemic symptoms. Amaurosis fugax with a carotid stenosis of 70% or more certainly increases a person's danger of stroke, but with fewer hazards than if the ischemic symptoms were cerebral. Carotid endarterectomy following to episodes of transient cerebral or retinal ischemia is known to decrease the danger of cerebral infarction. This impact is seen after cerebral ischemia with stenosis more prominent than half. It is seen after retinal ischemia given that stenosis is 70% or more noteworthy. Thus, endarterectomy is supported in the retinal patient only if the

stenosis is 70% or more whereas supported for hemispheric events with stenosis of 50% or greater. Suggestions for this technique must be individualized. It ought to be considered for patients with TMB or amaurosis fugax only if the surgical complication rate is less than 2%. For patients with cerebral transient ischemic attacks (TIAs), a complication rate of 3% or less is tolerable ^[8,9].

Nonarteritic-Ischemic Optic Neuropathy

No great surgical alternative or helpful treatment for non arteritic ischemic optic neuropathy has yet been explained ^[10]. In an examination by **Dickersin et al.** ^[11] an optic nerve decompression surgery including cutting at least 2 openings inside the tissue around the optic nerve with the expectation to enable CSF to escape and decrease weight around the nerve was halted ahead of schedule for vanity ^[11]. Surgical patients experienced both intraoperative and postoperative antagonistic occasions, including CRAO amid surgery and light observation vision at a half year. There was likewise quick loss of light discernment following surgery and loss of vision that continued to the year visit.

Central Retinal Vein Occlusion

Surgical selections for central retinal vein occlusion contain radial optic vitrectomy, chorioretinal venous anastomosis, neurotomy, and retinal vein injection with tPA. Nothing of these surgical managements has been confirmed to be more effective than nonsurgical approaches for improving vision loss and is still experimental at this time ^[12].

Local arterial fibrinolysis for the treatment of central retinal artery occlusion (CRAO)

A nonrandomized, single center, interventional examine by Aldrich et al. ^[13] shown enhanced visual perception in patients who established local intra-arterial aliquots of tissue plasminogen activator (tPA). In this little investigation, 21 patients got 3 mg aliquots of intra-arterial vessel tPA and 76% of these patients had enhanced visual acuity compared with 33% of the patients in the standard treatment group. The authors advised that on account of the nonrandomized idea of this and past investigations, local arterial fibrinolysis can't be suggested as standard treatment in day by day clinical work on pending the publication of randomized clinical trials. In a later study, outcomes from the first interim analysis of the first randomized clinical trial comparing efficacy of conservative treatment to local arterial fibrinolysis (the European Assessment Group for Lysis in the Eye [EAGLE] study) found no change in

effectiveness between the treatment groups ^[14]. Furthermore, even though having comparable visual improvements in groups, local intra-arterial fibrinolysis (57%) and conservative treatment (60%), outcomes presented higher incidences of adverse events in the local intra-arterial fibrinolysis group; therefore, the study was discontinued.

• **Medical Management**

Aspirin is thought to be useful in patients with no hemodynamically significant illness of the carotid artery (i.e., more than 1 mm residual lumen) or in those who are poor surgical applicants ^[15]. Generally, aspirin together with modification of risk factors (e.g., diminishing serum cholesterol level, controlling systemic hypertension) decreases the possibility of myocardial infarction. It is likewise very operational in decreasing the danger of stroke. Aspirin was once supposed to be most effective in high doses, but new proof has shown that similar benefits can be reached with low-dose aspirin at 81 mg/day. Counsel patients with regular or severe headaches to stop smoking. Women who smoke and take birth control pills are at greater risk for stroke.

Clopidogrel (Plavix) has been presented to be effective in decreasing the danger of stroke and in a study comparing its efficacy to aspirin, was shown to be only minimally better. It can be used easily in patients who are aspirin intolerant. Whether the combination of clopidogrel plus aspirin is better than either medication alone is presently unknown.

Aggrenox (aspirin plus dipyridamole) has been appeared to be powerful in diminishing stroke hazard. In a comparison with either agent alone, it was observed to be significantly more effective. The recent outcomes of the PROFESS trial demonstrated that aspirin plus dipyridamole and clopidogrel were proportional in efficacy. Either medication is an adequate beginning medication for the patient at danger for future stroke.

Inferior retinal detachment is treated with the patient sitting up. Superior detachment is treated with the patient lying prone, so to abstain worsening of the detachment by gravity.

Current guidelines for optic neuritis are depend on one randomized control trial (Optic Neuritis Treatment Trial) and propose either high-dose intravenous methylprednisolone or no treatment. In a review article reporting on 750 participants across 6 randomized trials observing low-dose, high-dose, intravenous steroids, and oral for optic neuritis, there was no indication of benefit in

regards of recovery of visual field, visual acuity, or contrast sensitivity with either oral or intravenous corticosteroids compared with placebo at 6 months^[16]. Nevertheless, sign that treating with steroids hastened the rate of return of vision to normal compared with placebo. When selecting this treatment, oral steroids need to be preceded by intravenous steroids, as oral steroids only caused in fewer patients attaining normal visual acuity compared with controls and might actually be related with increased return rates.

In cases of acute central retinal artery occlusion, conservative therapy possibly will include the following (Table 2) ^[17]:

Table 2: Conservative therapy for acute central retinal artery occlusion.

Ocular massage	Mannitol
Acetylcholine	Hemodilution
Acetazolamide	Hyperbaric oxygen
Pentoxifylline	Heparin IV
Calcium channel blockers	Glycerol
Prostaglandin E1	Paracentesis
Carbogen inhalation	Lidocaine
Methylprednisolone	hydrochloride
	Topical glaucoma medications

None of these approaches has been proven more effective than any other. A randomized study by **Schumacher *et al.*** ⁽¹⁸⁾ (EAGLE study) compared these conservative managements with a more invasive technique called local intra-arterial fibrinolysis.

For patients with non ischemic central retinal vein occlusion (CRVO), there has been considerably examination in the last few years into effective managements to both correct vision and avert progression to ischemic CRVO. Some managements to aid with the result of the disease comprise lowering intraocular pressure, panretinal laser photocoagulation, treating underlying medical conditions^[19], intravitreal anti-VEGF, laser-induced chorioretinal venous anastomosis (L-CRA)^[20], and intravitreal triamcinolone treatments. The latter 2 approaches are suggested to reduce subsequent macular edema from CRVO, while L-CRA is anticipated to directly treat the venous occlusion. Limited studies have assessed the efficacy of triamcinolone injections, and it has been establish to have only a temporary influence with danger of significant adverse effects^[20].

Though, anti-VEGF injections have revive the forefront inbranch retinal vein occlusion (BRVO)

and CRVO therapy, further studies are required to target treatment groups that would benefit most from these therapies, as well as to determine specific dosing regimens and window for treatment initiation. A retrospective, single-center study by Ferrara *et al.*^[21] established better visual acuity and reduced macular edema in patients with CRVO of less than 3 months who were given bevacizumab. In this study, 5 patients (6 eyes) who received intravitreal bevacizumab were tested for visual acuity and retinal appearance before and after treatment. Though, small sample size and the nonrandomized nature of the study and other studies assessing this process limits its use as standard treatment at this time.

Later analysis described probable risk of bias as a result of incomplete result data and found that it was not possible to exclude selective reporting^[22]. The small sample size resulted in insufficient power to investigate result variances between the treatment doses, and the absence of protracted observation allows only for speculation of short term treatment with pegaptanib. Intravitreal anti-VEGF injection with ranibizumab (approved by the FDA for treatment neovascular age-related macular degeneration in 2006) has presented promise in the short-term management of non ischemic CRVO-related macular edema, with lately published follow-up study data and FDA approval of ranibizumab for retinal vein occlusion. In the CRUISE trial, a phase III randomized, double-masked, multicenter, injection-controlled trial by **Campochiaro et al.**^[22], 392 patients with macular edema after CRVO were randomized to get monthly 0.3-mg, 0.5-mg, or sham intravitreal injections of ranibizumab over 6 months. Those patients who established ranibizumab injections were revealed to have significantly enhanced visual acuity (46.3-47.7%) compared with study controls (16.9%), in addition to reduced central foveal thickening.

In a similar prospective, double-masked, randomized, sham injection-controlled, multicenter clinical trial, BRAVO, by Campochiaro et al.^[23], intravitreal ranibizumab was

likewise found to develop visual acuity (55.2-61.1%) in patients with BRVO compared with controls (28.8%), as well as reduced central foveal thickness. Follow up data from both trials, BRAVO and CRUISE, for the subsequent 6 months presented that patients with both conditions continued to progress with recurrent injections with no increase in opposing events^[24, 25].

The HORIZON trial, carried by **Heier et al.**^[21], which comprised a cohort of patients who finished the BRAVO and CRUISE trials, found no different adversarial safety events after an further year of treatment with ranibizumab. It did, though, find that throughout the second year of management, the clinical development of patients with BRVO was tenacious, while CRVO patients inclined to have a weakening in vision, possibly allied to a reduced frequency of injections or the opposing degrees in retinal injury^[27].

Even though the HORIZON study was completed early secondary to FDA approval of ranibizumab for RVO, numerous limitations of the aforesaid studies occur and questions persist concerning the effectiveness of ranibizumab for RVO. Follow-up data at the 6-month time point eradicated the control group of sham-injection patients and delivered rescue laser management for all patients^[28], neither the BRAVO nor CRUISE trials had sufficient power to examine variances between the 2 treatment doses, ischemic CRVO was debarred in these trials, and optimal timing of early management has not yet been determined, which is correspondingly limited by the small amount of data concerning the disease progression and prognosis of untreated CRVO-related macular edema.

The aims of pharmacotherapy in acute visual loss are to decrease morbidity and avert complications. Inhibit platelet function possibly by blocking cyclooxygenase and subsequent aggregation. Antiplatelet treatment has been presented to decrease mortality by decreasing the danger of fatal strokes, fatal myocardial infarctions, and vascular death in patients at risk.

Table 3: Medication for vision loss

Drug	Drug information
Aspirin (Ascriptin, Aspirtab, Aspercin, Bayer Aspirin, Buffinol)	Irreversibly inhibits the formation of cyclooxygenase, thus preventing the formation of thromboxane A2, a platelet aggregator and vasoconstrictor. Platelet inhibition lasts for the life of the cell (approximately 10 d).
Clopidogrel (Plavix)	Selectively inhibits ADP binding to platelet receptor and subsequent ADP-mediated activation of glycoprotein GPIIb/IIIa complex, thereby inhibiting platelet aggregation.
Aspirin and dipyridamole (Aggrenox)	Aspirin irreversibly inhibits formation of cyclooxygenase, thus preventing formation of thromboxane A2, a platelet aggregator and vasoconstrictor. Platelet-inhibition lasts for life of cell (approximately 10 d).
	Dipyridamole is a platelet adhesion inhibitor that possibly inhibits RBC uptake of adenosine, itself an inhibitor of platelet reactivity. In addition, may inhibit phosphodiesterase activity leading to increased cyclic-3', 5'-adenosine monophosphate within platelets and formation of the potent platelet activator thromboxane A2.
	Each tablet contains 25 mg aspirin and 200 mg dipyridamole for total of 50 mg aspirin and 400 mg dipyridamole per day.

CONCLUSION

Managing vision loss starts with a focused history and examination. Once a preliminary diagnosis is completed, appraisal and treatment depend on the findings. Amaurosis fugax needs workup with carotid and cardiac imaging. Hypercoagulable testing and angiography may be helpful in certain cases. Carotid surgery or medications can prevent future stroke. Retinal vasospasm could be treated with aspirin or calcium channel blockers. Retinal migraine responds to standard migraine treatments. Inferior retinal detachment is treated with the patient sitting up. Superior detachment is treated with the patient lying prone, so to abstain worsening of the detachment by gravity. Ocular causes such angle closure are treated accordingly. First and foremost, however, the patient must get evaluated. Thus, we must continue to educate the public and our medical colleagues about the importance of getting an ophthalmologic evaluation in patients with acute vision loss.

REFERENCES

- Hedges TR(1984):**The terminology of transient visual loss due to vascular insufficiency. *Stroke*, 15(5):907-8.
- Rosamond W, Flegal K, Friday Get al.(2007):**Heart disease and stroke statistics--2007 update: a report from the American Heart Association statistics committee and stroke statistics subcommittee. *Circulation*,115:69–171.
- Rodriguez A, Lozano JA, del Pozo Det al.(1993):** Post-traumatic transient cortical blindness. *Int Ophthalmol.*, 17(5):277-83.
- Wong TY, Larsen EK, Klein R, Mitchell P, Couper DJ, Klein BE, Hubbard LD, Siscovick DS, Sharrett AR(2005):**The Atherosclerosis Risk in Communities & Cardiovascular Health Studies. Cardiovascular Risk Factors for Retinal Vein Occlusion and Arteriolar Emboli. *Ophthalmology*,112:540–547.
- Shinar Z, Chan L, Orlinsky M(2011):** Use of ocular ultrasound for the evaluation of retinal detachment. *J Emerg Med.*, 40(1):53-7.
- Amick A, Caplan LR(2007):** Transient monocular visual loss. *Compr Ophthalmol Update*, 8(2):91-8.
- Brodsky MC(2006):** Contractile morning glory disc causing transient monocular blindness in a child. *Arch Ophthalmol.*,124(8):1199-201.
- Kunkel RS(2005):**Migraine aura without headache: benign, but a diagnosis of exclusion. *Cleve Clin J Med.*,72(6):529-34.
- Ng RH(2016):**Post-traumatic transient cortical blindness in a child with occipital bone fracture. *J Clin Neurosci.*,34:225-227.
- Rootman DB, Gill HS, Margolin EA(2013):** Intravitreal bevacizumab for the treatment of nonarteritic anterior ischemic optic neuropathy: a prospective trial. *Eye (Lond)*,27(4):538-44.
- Dickersin K, Manheimer E, Li T(2012):** Surgery for nonarteritic anterior ischemic optic neuropathy. *Cochrane Database Syst Rev.*,1:CD001538.

- 12.Berker N, Batman C(2008):** Surgical treatment of central retinal vein occlusion. *Acta Ophthalmol.*,86(3):245-52.
- 13.Aldrich EM, Lee AW, Chen CSet al.(2008):** Local intra-arterial fibrinolysis administered in aliquots for the treatment of central retinal artery occlusion: the Johns Hopkins Hospital experience. *Stroke*, 39(6):1746-50.
- 14.Schumacher M, Schmidt D, Jurklies Bet al.(2010):**Central retinal artery occlusion: local intra-arterial fibrinolysis versus conservative treatment, a multicenter randomized trial. *Ophthalmology*, 117(7):1367-75.e1.
- 15.**<https://www.ncbi.nlm.nih.gov/pubmed/9593407>
- 16.Gal RL, Vedula SS, Beck R(2012):** Corticosteroids for treating optic neuritis. *Cochrane Database Syst Rev.*,4:CD001430.
- 17.Biousse V, Calvetti O, Bruce BB, Newman NJ(2007):** Thrombolysis for central retinal artery occlusion. *J Neuroophthalmol.*,27(3):215-30.
- 18.Schumacher M, Schmidt D, Jurklies Bet al.(2010):**Central retinal artery occlusion: local intra-arterial fibrinolysis versus conservative treatment, a multicenter randomized trial. *Ophthalmology*, 117(7):1367-75.e1.
- 19.Morley MG, Heier JS(2008):** Venous obstructive diseases of the retina. Yanoff M, Duker Js. *Ophthalmology*, Mosby.
- 20.McAllister IL, Gillies ME, Smithies LAet al.(2010):** The Central Retinal Vein Bypass Study: a trial of laser-induced chorioretinal venous anastomosis for central retinal vein occlusion. *Ophthalmology*, 117(5):954-65.
- 21.Ferrara DC, Koizumi H, Spaide RF(2007):** Early bevacizumab treatment of central retinal vein occlusion. *Am J Ophthalmol.*,144(6):864-71.
- 22.Braithwaite T, Nanji AA, Greenberg PB(2010):**Anti-vascular endothelial growth factor for macular edema secondary to central retinal vein occlusion. *Cochrane Database Syst Rev.* ,1(5):CD007325.
- 23.Campochiaro PA, Heier JS, Feiner L, Gray S, Saroj N, Rundle ACet al.(2010):** Ranibizumab for macular edema following branch retinal vein occlusion: six-month primary end point results of a phase III study. *Ophthalmology*, 117(6):1102-12.
- 24.Brown DM, Campochiaro PA, Bhisitkul RB, Ho AC, Gray S, Saroj Net al.(2011):** Sustained benefits from ranibizumab for macular edema following branch retinal vein occlusion: 12-month outcomes of a phase III study. *Ophthalmology*, 117(6):1594-602.
- 25.Campochiaro PA, Brown DM, Awh CCet al.(2011):** Sustained benefits from ranibizumab for macular edema following central retinal vein occlusion: twelve-month outcomes of a phase III study. *Ophthalmology*, 118(10):2041-9.
- 26.Heier JS, Campochiaro PA, Yau L, Li Z, Saroj N, Rubio RGet al.(2012):** Ranibizumab for macular edema due to retinal vein occlusions: long-term follow-up in the HORIZON trial. *Ophthalmology*, 119(4):802-9.
- 27.Varma R, Bressler NM, Suñer Iet al.(2012):** Improved vision-related function after ranibizumab for macular edema after retinal vein occlusion: results from the BRAVO and CRUISE trials. *Ophthalmology*, 119(10):2108-18.
- 28.Mitry D, Bunce C, Charteris D(2013):** Anti-vascular endothelial growth factor for macular oedema secondary to branch retinal vein occlusion. *Cochrane Database Syst Rev.*,1:CD009510.