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Original Research Article

Etiological Classification and Prognostic Outcomes of Sudden Vision Loss: A Prospective Observational Study

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Abstract

Introduction: Sudden vision loss is a clinical emergency with a broad etiological spectrum, ranging from ischemic and non-ischemic medical causes to trauma. Understanding the underlying cause is essential for prognosis and timely intervention. This study aimed to evaluate the etiological distribution and prognostic outcomes in patients presenting with sudden vision loss.

Materials and Methods: This prospective observational study was conducted at Tirunelveli Medical College Hospital from January 2015 to July 2016. A total of 65 patients presenting with sudden vision loss lasting more than 24 hours were included. Clinical evaluation, ophthalmic investigations, imaging, and laboratory workup were performed as indicated. Patients were followed for six months to assess visual outcomes. Statistical analysis was performed using SPSS v20, with p-values <0.05 considered significant.

Results: Of 65 patients, 66.2% had medical causes (30.8% ischemic, 35.4% non-ischemic), and 33.8% had trauma-related vision loss. NA-AION and CRVO were the most common ischemic causes, while traumatic optic neuropathy (TON) was the leading traumatic etiology. Visual improvement at six months was highest in ischemic cases (65%), followed by non-ischemic (43.5%) and traumatic cases (27.3%) ($p = 0.048$). Hypercholesterolemia showed a significant association with poor visual outcome in ischemic cases ($p = 0.028$). Most trauma cases occurred in young adult males.

Conclusion: Ischemic etiologies, especially NA-AION and CRVO, were most frequent and had better visual outcomes than traumatic causes. Hypercholesterolemia was a significant predictor of poor prognosis in ischemic vision loss. Early diagnosis, risk factor management, and multidisciplinary care are crucial to preserving vision.

Keywords: sudden vision loss, NA-AION, CRVO, traumatic optic neuropathy, ischemic vision loss, visual outcome, hypercholesterolemia, risk factors, prospective study, ocular trauma.

Introduction

Sudden loss of vision is a medical emergency with diverse etiologies, presenting either as transient visual loss lasting less than 24 hours or as persistent loss lasting beyond 24 hours [1]. It is a frequently encountered symptom in ophthalmology outpatient departments and emergency settings, requiring urgent evaluation to identify potentially reversible causes.[2,3]

Patients often describe the symptom subjectively—as dimming, fogging, or a curtain-like obscuration of vision—and the onset may be painless or painful depending on the underlying pathology. The etiology of sudden vision loss spans a wide spectrum, broadly classified into ischemic, non-ischemic, inflammatory, infectious, traumatic, neuro-ophthalmic, drug- or toxin-induced, idiopathic, and functional causes [4]. Vascular and ischemic events are among the most critical etiologies and often reflect systemic associations such as atherosclerotic disease, carotid artery stenosis, cardiac disorders, and hypercoagulable states [5,6].Common ischemic causes include central retinal artery occlusion (CRAO), branch retinal artery occlusion (BRAO), anterior ischemic optic neuropathy (AION), and ocular ischemic syndrome (OIS) [6,8].

Cardiac sources of emboli (e.g., atrial myxomas, valvular disease), internal carotid artery atherosclerosis, and hematologic disorders can contribute to retinal ischemia and embolic events, leading to either transient monocular visual loss (amaurosis fugax) or permanent vision deficits [5,6].Non-ischemic etiologies such as optic neuritis, retinal detachment, vitreous hemorrhage, traumatic optic neuropathy, and angle-closure glaucoma also contribute significantly to the

clinical spectrum of sudden vision loss. Optic neuritis is particularly common in younger individuals and may serve as a sentinel manifestation of demyelinating disorders such as multiple sclerosis [11]. Retinal detachment, on the other hand, poses a time-sensitive threat to vision and often presents with photopsia, floaters, or a curtain-like shadow in the visual field [12].Certain life-threatening systemic conditions such as posterior reversible encephalopathy syndrome (PRES) and cortical blindness also manifest as acute visual disturbances and necessitate neuroimaging to exclude intracranial pathology [7]. Moreover, toxic substances (e.g., methanol, quinine), phosphodiesterase inhibitors, and trauma—including globe rupture and compressive injuries—constitute other important but less common causes of visual loss [4,9].

The evaluation of a patient with sudden vision loss must be systematic, starting with a detailed history, visual acuity testing, pupillary reactions, slit-lamp examination, intraocular pressure measurement, and fundus examination. Additional investigations such as optical coherence tomography (OCT), fluorescein angiography, magnetic resonance imaging (MRI), visual evoked potentials, and blood workup (ESR, CRP, ANA, antiphospholipid antibodies) are often crucial in determining the etiology [6,10].Despite the high clinical importance of this presentation, there is limited consolidated data on the relative frequencies of different etiologies and their prognostic outcomes. Early diagnosis is critical, as the visual prognosis can vary significantly across etiologies—from reversible (e.g., optic neuritis) to irreversible (e.g., CRAO) [1,6,11].This study was conducted to understand the etiologies of sudden vision loss and to evaluate the associated clinical and prognostic outcomes in a prospective manner**.** Through this, we aim to enhance the understanding of etiological distribution and guide better clinical decision-making for optimal visual rehabilitation.

Materials and Method

This prospective observational case series was conducted in the Department of Ophthalmology at Tirunelveli Medical College Hospital between January 2015 and July 2016. A total of 65 patients presenting with sudden loss of vision were included. Patients were recruited from the outpatient department, referrals from other departments, and the emergency unit, including trauma cases. Follow-up was conducted over a six-month period to assess visual outcomes and systemic associations.

Inclusion and Exclusion Criteria: Inclusion criteria were patients of any age and sex with sudden visual loss persisting for more than 24 hours, who were conscious and cooperative. Exclusion criteria included patients with congenital ocular anomalies, gradual vision loss, unconsciousness, or inability to cooperate for examination.

Clinical Evaluation: A structured questionnaire was used to collect demographic and clinical data. History regarding onset, duration, progression, laterality, associated symptoms, trauma, systemic illnesses (e.g., diabetes, hypertension), and lifestyle factors (e.g., smoking, alcohol) was obtained. General and systemic examinations were followed by detailed ophthalmic evaluation, including visual acuity (Snellen's chart), color vision (Ishihara plates), visual fields (Octopus 300), intraocular pressure (Goldmann applanation tonometer), and slit-lamp biomicroscopy. Fundus evaluation was done using a +90D lens and indirect ophthalmoscopy.

Investigations: All patients underwent B-scan ultrasonography. Fundus fluorescein angiography and optical coherence tomography were performed in indicated retinal cases. Visual evoked potentials were used in optic neuritis. Relevant blood tests (ESR, CRP, lipid profile) and neuroimaging (CT/MRI of brain/orbit) were conducted based on clinical suspicion.

Management Protocol: Patients were managed based on the underlying diagnosis. Intravenous methylprednisolone followed by oral corticosteroids was administered in optic neuritis and ischemic optic neuropathy. CRVO cases with macular edema underwent grid laser. Retinal detachment and vitreous hemorrhage were treated surgically. Cortical blindness was managed with antiplatelets and statins. Other conditions such as fungal keratitis, uveitis, globe rupture, traumatic cataract, and acute angle closure glaucoma were treated with standard medical or surgical protocols.

Follow-up and Outcome Assessment: Patients were reviewed daily in the first week, and at 1, 3, and 6 months. Visual acuity was reassessed and recorded in LogMAR units. Improvement was defined as ≥1 line gain.

PL vision improving to HM/CFCF and HM/CFCF improving to 1/60 were considered clinically significant. Fundus, color vision, and field assessments were repeated. Neuroimaging was repeated when necessary.

Statistical Analysis: Data were analyzed using IBM SPSS Statistics v20. Continuous variables were analyzed using Student's t-test and categorical variables with Chi-square or Fisher's exact test. Correlations were evaluated using Spearman's rho. A p-value <0.05 was considered statistically significant. Graphs were plotted using MS Excel.

Results

A total of 65 patients with sudden vision loss were included in the study. Sudden vision loss was more common in males, especially in the traumatic group. Among males, traumatic causes predominated in the 10–40 year age group, whereas non-traumatic causes were more prevalent in patients above 40 years. Trauma was a rare cause of sudden vision loss in females.The left eye was most commonly affected overall. Bilateral involvement was noted exclusively in cases of medical etiology (particularly ischemic). The difference in laterality was statistically significant ($p = 0.001$) .Of the total 65 cases, 43 (66.2%) were attributed to medical (non-traumatic) causes, and 22 (33.8%) to trauma. Among medical cases, 20 patients (30.8%) had ischemic causes and 23 patients (35.4%) had non-ischemic causes (Table 1). The most common ischemic cause was Non-Arteritic Anterior Ischemic Optic Neuropathy (NA-AION; n=8), followed by Central Retinal Vein Occlusion (CRVO; n=6). Other ischemic causes included Central Retinal Artery Occlusion (CRAO; n=2), Cortical blindness (n=2), and Posterior Reversible Encephalopathy Syndrome (PRES; n=2).In the non-ischemic group, Advanced Diabetic Eye Disease (ADED) with vitreous hemorrhage was the most frequent (n=5), followed by Retrobulbar neuritis (n=4) and Rhegmatogenous Retinal Detachment (RRD; n=3). Other causes included cavernous sinus thrombosis, superior orbital fissure syndrome, acute angle closure glaucoma, central serous chorioretinopathy, fungal keratitis, infiltrative optic neuropathy, multifocal choroiditis, and panuveitis. Among traumatic cases (n=22), Traumatic Optic Neuropathy (TON) was the most common cause (n=13), followed by vitreous hemorrhage with traumatic mydriasis (n=3), globe rupture (n=2), and isolated cases of traumatic cataract, Berlin's edema, choroidal rupture, and corneal tear.

Risk factors including diabetes mellitus (DM), hypertension (HTN), hypercholesterolemia, ischemic heart disease (IHD), cerebrovascular accidents (CVA), smoking, and alcohol intake were evaluated. There was no statistically significant difference in the distribution of risk factors between ischemic and non-ischemic groups ($p = 0.762$) .Out of 65 patients, 29 (44.6%) showed improvement in visual acuity at 6-month follow-up, while 36 (55.4%) did not. Visual improvement was most frequent in the ischemic group (13/20), followed by non-ischemic (10/23), and was least in traumatic causes (6/22). The difference in visual outcome across the three groups was statistically significant ($p = 0.048$). Among the 20 patients with ischemic causes, 75% (n=15) were male. Seventeen of the 20 patients were over 40 years old, showing age predilection for vascular etiologies. Visual outcomes varied by diagnosis. NA-AION showed good response to treatment, with 6 out of 8 patients improving. CRVO had a mixed outcome (3 improved, 3 unchanged), while CRAO showed no improvement. All patients with cortical blindness (n=2) and PRES (n=2) had favorable visual recovery. While DM and HTN did not significantly correlate with visual prognosis, hypercholesterolemia was significantly associated with poor visual outcome ($p = 0.028$). A moderate negative correlation was observed between systolic blood pressure and visual improvement ($r = –0.432$, $p = 0.057$), suggesting better perfusion leads to favorable outcomes. Patients with multiple risk factors had poorer prognosis, although this was not statistically significant three ischemic patients had a history of TIA. One patient with ischemic CRVO progressed to neovascular glaucoma and was found to have 80–95% ICA stenosis on Doppler imaging.

NA-AION was the most common ischemic cause (n=8). It showed no significant association with diabetes ($p = 0.582$) or smoking ($p = 0.582$). Among NA-AION patients, those with diabetes had relatively poorer visual outcomes .Systolic and diastolic blood pressure were negatively correlated with visual improvement, with diastolic BP showing significant association ($r = –0.97$, $p = 0.0023$).Hypercholesterolemia was associated with poor outcomes but without statistical significance.

CRVO was the second most frequent ischemic cause (n=6). Ischemic CRVO was noted in 3 cases, hemi-retinal CRVO in 2, and non-ischemic in 1. No significant association was found between CRVO and DM ($p = 0.492$), HTN ($p = 0.141$), hypercholesterolemia ($p = 0.202$), glaucoma ($p = 0.303$), or IHD ($p = 0.131$). Increasing number of risk factors correlated with worse visual outcomes. Diastolic BP showed a strong negative correlation with vision improvement ($r = –0.71$, $p = 0.1069$) .In comparison with NA-AION, patients with CRVO more commonly had HTN, glaucoma, and hypercholesterolemia, whereas NA-AION cases were more strongly associated with DM, smoking, and alcohol. A mild negative correlation was seen between presenting vision and improvement for both NA-AION and CRVO.The most frequent non-ischemic cause was ADED with vitreous hemorrhage (n=5), followed by retrobulbar neuritis (n=4), and RRD (n=3).

Vision improved in all patients with acute angle closure glaucoma and fungal keratitis, and partially in RBN and RRD cases. Poor visual outcome was noted in cases of CST, SOF syndrome, panuveitis, and infiltrative optic neuropathy. Trauma-related sudden vision loss was more common in young adult males. Both eyes were equally affected (n=11 each). TON was the most common traumatic cause (n=13), followed by VH with traumatic mydriasis (n=3), and globe rupture (n=2). Best visual

outcomes were seen in patients with traumatic cataract and VH with mydriasis. Among patients with isolated TON, only 2 (15.4%) showed improvement, both of whom had indirect TON with presenting vision of PL or better.(Figure 1)

Table 1: Distribution of Etiologies of Sudden Vision Loss (n = 65)

Etiology	Subcategory	Number of Patients (n)	Percentage (%)
Medical (Non-Traumatic)	**Ischemic (n = 20)**		
	NA-AION	8	12.3%
	CRVO	6	9.2%
	CRAO	2	3.1%
	Cortical Blindness	2	3.1%
	PRES	2	3.1%
	Non-Ischemic (n = 23)		
	ADED with VH	5	7.7%
	Retrobulbar Neuritis	4	6.2%
	RRD	3	4.6%
	CST, SOF Syndrome, ACG, etc.	11	16.9%
Trauma (n = 22)	TON	13	20.0%
	VH with Traumatic Mydriasis	3	4.6%
	Globe Rupture, Cataract, Others	6	9.2%
Total		**65**	**100.0%**

Table 2: Visual Outcome in Different Etiological Groups (n = 65)

Etiological Group	Improved Vision (n)	%	No Improvement (n)	%	Total	p-value
Ischemic (n = 20)	13	65.0%	7	35.0%	20	**0.048**
Non-Ischemic (n = 23)	10	43.5%	13	56.5%	23	
Trauma (n = 22)	6	27.3%	16	72.7%	22	

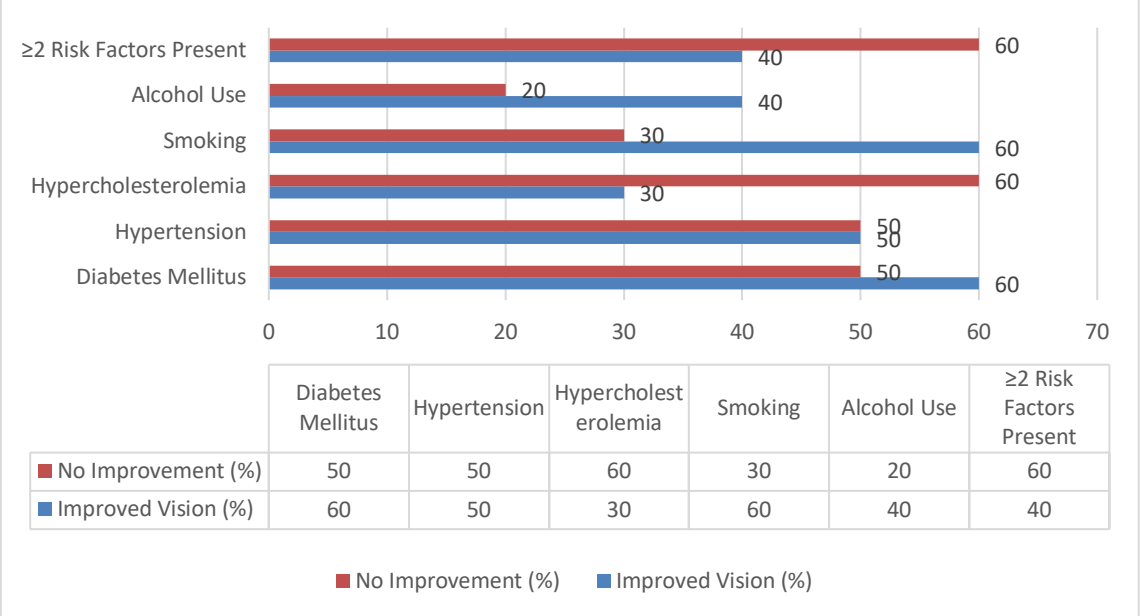

Figure 1: Association between Risk Factors and Visual Outcome in Ischemic Group (n = 20)

Discussion

This prospective study analyzed 65 patients presenting with sudden vision loss, assessing their demographic patterns, etiological distribution, risk factors, and visual outcomes. In our study, 69% of patients were males and 31% females. Traumatic causes were more common in young males aged 10–40 years, whereas non-traumatic causes were predominant in patients above 40 years, with a peak in the seventh decade.

This age distribution aligns with the higher incidence of systemic comorbidities in older individuals. Adhikari et al. [13] also reported trauma-related vision loss being more prevalent in males in the younger age group, supporting our findings. Left eye involvement was more common

(n=34), with bilateral involvement noted only in medical causes, especially ischemic cases. The laterality difference was statistically significant (p = 0.001). Among 65 patients, 66.2% had non-traumatic (medical) causes, further categorized as ischemic (30.8%) and non-ischemic (35.4%), while 33.8% were trauma-related. NA-AION (12.3%) and CRVO (9.2%) were the most common ischemic causes. Among non-ischemic causes, ADED with vitreous hemorrhage (7.7%) and retrobulbar neuritis (6.2%) were frequent. Traumatic optic neuropathy (TON) (20%) was the leading traumatic cause.

Although diabetes mellitus, hypertension, and smoking were prevalent across all groups, hypercholesterolemia showed a significant association with poor visual outcome in ischemic cases (p = 0.028). This supports the hypothesis by Hayreh et al. [14], who emphasized that ocular perfusion pressure is a key determinant of optic nerve head blood flow and ischemia.NA-AION was the most frequent ischemic etiology (n=8; 12.3%). In our study, 75% of NA-AION cases were under 50 years of age, differing from Hayreh et al. [15], who reported that NA-AION typically affects patients over 50. However, Jacobson et al. [16] documented that 11–23% of NA-AION cases can occur below 50 years. Diabetes mellitus was seen in 65% of our NA-AION patients, consistent with the meta-analysis by Chen et al. [17], which showed a positive association between DM and NA-AION.

Smoking was seen in 37.5% of NA-AION patients but was not statistically significant (p = 0.582), similar to findings by Hayreh et al. [15].Visual improvement was noted in 75% of NA-AION cases at 6 months, higher than the 25–40% improvement rates reported by Arnold et al. [18] and Yee et al. [19].CRVO accounted for 9.2% of all cases and 30% of ischemic causes. Ischemic CRVO was noted in 3 patients, hemiretinal in 2, and non-ischemic in 1.

Among CRVO patients, 66.6% had diabetes (p = 0.492), 83.3% had hypertension (p = 0.141), and 66.6% had hypercholesterolemia (p = 0.202). These values are similar to McIntosh et al. [31], who reported hypertension in 65% of CRVO patients and diabetes in 25–30%. Sodi et al. [20] emphasized the role of hypercholesterolemia in ischemic CRVO, which was also evident in our study. Glaucoma was present in 50% of our CRVO cases (p = 0.303), in line with Hayreh et al. [21], who found elevated IOP (>22 mmHg) in 22% of CRVO patients. Sperduto et al. [22] recommended cardiovascular evaluation in such cases, which our findings support. Visual improvement was seen in 33.3% of CRVO cases, limited to those with non-ischemic and hemiretinal variants. This supports Hayreh SS [23], who reported poor prognosis in ischemic CRVO.CRAO was noted in 2 cases (3.1%), both with hypercholesterolemia and a smoking history. Hayreh et al. [15] identified both as independent thrombophilic risk factors for CRAO. No patient showed vision improvement, likely due to delayed presentation. Cortical blindness and PRES (each 3.1%) showed complete visual recovery, reflecting their known reversible nature. Non-ischemic causes (35.4%) included ADED with VH (7.7%), retrobulbar neuritis (6.2%), and RRD (4.6%). Good outcomes were seen in cases of acute angle closure glaucoma and fungal keratitis. Poor outcomes were observed in CST, SOF syndrome, infiltrative optic neuropathy, and panuveitis.

These cases highlight the heterogeneity in prognosis and need for systemic evaluation. Trauma-related sudden vision loss was observed in 22 patients (33.8%), predominantly young males (90%), consistent with findings by Adhikari et al. [13] and Singh et al. [24]. Bilateral involvement was not observed, supporting Adhikari et al.'s observation that bilateral trauma is rare.Road traffic accidents accounted for 72% of trauma cases, with two-wheeler accidents under alcohol influence contributing to 62.5%, matching De Juan et al. [25].Traumatic optic neuropathy was the most frequent cause (n = 13; 59%). Only 15.4% of isolated TON patients showed vision improvement. This is lower than the 40–60% improvement seen with steroid therapy in indirect TON in the study by Patrick Yu-Wai-Man [26]. Delayed presentation in our cohort may explain this difference. Visual improvement at 6 months was seen in 29 patients (44.6%). Improvement was highest in ischemic etiologies (65%), followed by non-ischemic (43.5%) and trauma (27.3%) . These findings underscore the importance of early identification and risk factor control, particularly in ischemic vision loss.

Conclusion

This study underscores the varied etiologies of sudden vision loss, with ischemic causes—especially NA-AION and CRVO—being most common, followed by traumatic optic neuropathy (TON). Systemic risk factors like diabetes, hypertension, and hypercholesterolemia were prevalent, with hypercholesterolemia significantly associated with poor visual outcomes in ischemic cases. Visual improvement was highest in ischemic cases (65%) compared to non-ischemic (43.5%) and traumatic (27.3%) causes. Early recognition and management of risk factors are crucial, especially in ischemic vision loss. Traumatic cases, though more frequent in younger males, showed the poorest outcomes, highlighting the need for prompt, multidisciplinary care to prevent permanent visual disability.

Limitations

This study had a relatively small sample size (n = 65) and was conducted at a single tertiary care center, limiting generalizability and introducing potential selection bias. Delayed presentation, especially in CRAO and TON cases, may have impacted visual outcomes. Follow-up was limited to six months, restricting assessment of long-term prognosis. Additionally, lack of access to advanced imaging (e.g., OCTA) and detailed coagulation profiles in all patients limited further evaluation of vascular causes.

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