Review Article

Eye Stroke – A Warning Sign of Concurrent or Future Brain Stroke

Dr. Virendra Singh Lodha
Professor, Department of Ophthalmology
Pacific Medical College and Hospital
Bhillo Ka Bedla, Udaipur

ABSTRACT

The aim of this review is to discuss vascular etiological factors causing visual loss and emphasizing in recognizing their importance as a warning signal of future or concurrent brain stroke because risk and etiological factors are common for both conditions. Patients often do not take visual loss as seriously as a brain stroke and often delay seeking consultation or are unaware of the similarity of risk factors responsible for these two events. Sometimes ophthalmologists also do not realize the importance and urgency to be considered in referring the patient for stroke protocol workup at neurology and cardiac centre so that timely and appropriate remedial measures can be undertaken for treating and controlling modifiable risk factors to prevent future brain strokes.

Keywords : Eye Stroke, Ocular Ischemic Syndrome, Central Retinal Artery Occlusion.

INTRODUCTION

Sudden loss of vision in an eye may be caused by acute compromised blood supply to the ocular tissues especially retina and optic nerve and the term eye stroke may be used similar to brain stroke. Using the term eye stroke emphasises the importance of the gravity of the condition and its proper management because factors which are responsible for reduction in blood supply to the eye are similar to the factors responsible for causing brain stroke. Similarly amaurosis fugax (transient loss of vision) may be compared to transient ischemic attack (TIA) of brain and caused by same etiological factors. Similar to TIA of brain transient loss of vision may be caused by reversible blockage of blood supply to the eye by occlusion or narrowing of blood vessels i.e. arteries supplying the blood to the retina and optic nerve. Incidence and mortality was observed to be higher due to Ischemic heart disease and stroke in persons who have suffered amaurosis fugax.[1] Hence early and thorough evaluation and management of conditions responsible for an eye stroke may help in preventing future brain stroke.[2] Eye stroke and brain stroke (non-hemorrhagic), both are caused by reduction in blood supply and have same risk factors.

Causes of eye stroke and amaurosis fugax may be following:

Central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO)

Central retinal vein occlusion (CRVO) and branch retinal vein occlusion

Carotid artery disease

Ischemic optic neuropathy

Papilloedema

Brain tumour

Migraine

Address for Correspondence
Dr. Virendra Singh Lodha
vl901161@gmail.com

Multiple sclerosis

Head injury

Systemic lupus erythmatosus

In the present review only the diminution of vision, permanent or temporary caused by vascular pathologies are discussed which may be a warning signal of future neurological sequelae or stroke.

PREDISPOSING FACTORS FOR ISCHEMIC EYE AND BRAIN STROKE ARE –

Hypertension

Diabetes mellitus

Hyperlipidemia

Hyperviscocity of blood

Aging

Substance (cocaine) and alcohol abuse

Vasospasm

Smoking

CENTRAL RETINAL ARTERY OCCLUSION (CRAO) AND BRANCH RETINAL ARTERY OCCLUSION (BRAO)

CRAO was first described by von Graefe in 1859 who attributed the cause to be embolism.[3] CRAO is characterized by acute, severe, painless monocular loss of vision. In some patients presence of cilioretinal artery may help preserving central vision. But the condition is not often taken as seriously as brain stroke by patients resulting in delay in treatment and hence usually poor visual prognosis or the occlusion occurred during sleep at night and visual loss observed only in the morning. CRAO may be nonateritic CRVO (NA-CRAO), NA-CRAO with cilioretinal artery sparing, transient NA-CRAO or artertic CRAO (A-CRAO).4 Majority of cases are of NA-CRAO type, 96%) with or without presence of cilioretinal artery or transient NA-CRAO causing amuorosis fugax and only 4% patients have A-CRAO.[4] The most common cause of NA-CRAO is embolism, embolus arising from carotid artery or heart and associated stenosis of internal carotid artery (18%).[5] Amaurosis fugax may be caused by transient constriction of central retinal artery or a small embolus which migrated distally. Other associated conditions like Diabetes Mellitus, Hypertension and hyperlipidemia, crotid artery disease, hyperuricemia and chronic smoking also contribute significantly for plaque formation in heart and carotid arteries leading to embolism.[6,7]The incidence of developing ischemic stroke after CRAO is highest during first week after occurrence of CRVO, hence every patient of CRVO needs urgent stroke workup so that preventive measures can be taken in time.[8] It has been reported that 89% of brain strokes developing after CRAO, branch retinal artery occlusion or amaurosis due to retinal TIA are silent.[9]

CAROTID ARTERY DISEASE

Narrowing or stenosis of common or internal carotid artery is the most cause of reduced ocular blood supply. The most common cause of carotid artery narrowing is atheroma formation, less common causes include dissection, arteriris or external compression. Release of emboli from an atheromatous plaque which may get lodged in the central retinal artery, its branches or other cerebral arteries. Reduced blood supply to the eye leads to stasis of outflow of blood and retinal ischemia, a condition which was earlier named "Venous stasis retinopathy"but was later changed to ocular ischemic syndrome (OIS).[10] The most common cause of stenosis of carotid artery responsible for producing OIS is formation of atheromatous plaque.[11,12]

GIANT CELL ARTERITIS (ARTERITIC CRAO)

Giant cell arteritis (GCA) has also been reported to cause OIS by affecting and narrowing internal carotid artery.[13] Stroke though rare but can occur in 3-4% patients of GCA due to stenosis of carotid, vertebral or basilar arteries with high morbidity and mortality.[14]

NON-ARTERITIC ANTERIOR ISCHEMIC OPTIC NEUROPATHY

Persons who have suffered from nonarteritic anterior ischemic optic neuropathy (NAION) have been found to have an increased risk of developing ischemic stroke.[15] All such patients should have systemic evaluation for vasculopathy and control of risk factors to prevent brain stroke in future.

CONCLUSION

In a study it was observed that 25% patients who had retinal ischemia were found to have asymptomatic strokes discovered on magnetic resonance imaging (MRI) performed with diffusion weighted imaging (DWI). In another study it was found that patients suffering from monocular visual loss due to vascular aetiology had a 19.5% risk of developing ischemic stroke simultaneously which may be silent.[16,17] From this review of literature it may be emphasized that whenever a patient presents to an ophthalmologist for visual loss and diagnosed to have a vascular aetiology for the event, the patient should promptly be referred for neurological evaluation for a complete workup (stroke protocol) including complete cardiovascular studies to diagnose concurrent silent stroke and to prevent brain stroke in future. Hence it is to be emphasized that the ophthalmologist should consider referring a patient of visual loss due to vascular pathology with utmost urgency to a neurological centre for complete workup under stroke protocol so that precise etiological factor can be identified and measures can be undertaken to prevent serious neurological events like stroke in future.

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REFERENCES

1. Poole CJ, Ross Russell RW. Mortality and stroke after amaurosis fugax. J Neurol Neurosurg Psychiatry.1985 Sep;48(9):902-5 Doi: 10.1136/jnnp.48.9.902. PMID:4045484;PMCID: PMC1028491
2. Fisher CM. Transient monocular blindness associated with hemiplegia. Am Arch Ophthalmol 1952;47:167-203
3. Von Graefe A. Central retinal artery embolism as a cause of sudden blindness. Arch Ophthalmol. 1859;5: 136-57

4. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: Visual outcome. Am J Ophthalmol 2005;140:376-91

5. Hayreh SS, Podhajsky PA, Zimmerman MB. Retinal artery occlusion: Associated systemic and ophthalmic abnormalities. Ophthalmology 2009;116: 1928-36.

6. Schmidt D.,Hetzel A, Geibel-Zehender A, Schulte-Monting J. Systemic diseases in noinflammatory Branch and central retinal artery occlusion – an overview of 416 patients. Eur J Med Res. 2007 Dec 14; 12(12):595-603.

7. Hayreh SS.Ocular Vascular Occlusive Disorders.Heidelberg:Springer Verlag; 2015. P 31-53, 239-305

8. Park SJ, Choi NK, Yang BR et al. Risk and Risk periods for Stroke and acute Myocardial Infarction in patients with Central Retinal Artery Occlusion. Ophthalmology. 2015Nov;122(11):2336-2343.

9. Lauda F, Neugebauer H, Reiber L et al. Acute Silent Brain Infarction in Monocular Visual Loss of Ischaemic Origin.Cerebrovasc Dis. 2015;40:151-156.

10. Kearns TP, Hollenhorst RW. Venous stasis retinopathy of occlusive disease of the carotid artery. Mayo Clin Proc.1963;38:304-12.

11. Brown GC, Magargal LE, Simone FA et al. Arterial obstruction and ocular neovascularisation. Ophthalmology 1982;89:139-46.

12. Brwon GC, Magargal LE. The ocular ischaemic syndrome: Clinical, fluorescein angiographic and carotid angiographic features. Int Ophthalmol 1988;11:239-51.

13. Hamed LM, Guy JR, Moster MI, Bosley T. Giant cell arteritis in oclular aschaemic syndrome. AM J Ophthalmol 1992;113:702-05

14. Cox BC, Fulgham JR, Klaas JP. Recurrent Stroke in Giant Cell Arteritis Despite Immunotherapy. Neurologist. 2019 July; 24(4):139-141

15. Lee YC, Wang JH, Huang TL, Tsai RK. Increased risk of Stroke in Patients With Nonarteritic Anterior Ischemic Optic Neuropathy: A nationwide Retrospective Cohort Study. Am J Ophthalmol. 2016 Oct;170:183-189.

16. Helenius J, Arsava EM, Goldstein JN et al. Concurrent acute brain infarcts in patients with monocular visual loss. Ann Neurol. 2012 Aug;72(2):286-93

17. Zhang LY, Zhang J, Kim RK et al. Risk of Acute Ischemic Stroke in Patients with Monocular visual Loss of Vascular Etiology. J Neuroophthalmol. 2018 Sep;38(3):328-333