A Review of the Ophthalmic findings in Stroke Patients.

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ABSTRACT

Visual loss or impairment, in the midst of other bodily weakness or dysfunction from stroke, could prove unbearable even for the brave. Measures to prevent eventual visual loss from a systemic condition would give a lot of satisfaction to the patient, the ophthalmologist and other healthcare givers. Ocular involvement in stroke could arise from damage to parts of the brain that subserve ocular function (motor, visual and others) and/or changes in the physiology of the eye and surrounding structures due to the prevailing risk factors for stroke. Changes in the adnexae include ptosis, difficulty with initiating eye lid closure, difficulty with initiating eye lid opening and inability to keep the eye lids open. Strabismic changes include ocular motor nerve palsies (combined or isolated), conjugate eye deviation (partial and forced), dorsal midbrain syndrome, binocular horizontal, vertical and torsional diplopia, intermittent ophthalmoplegia, wrong-way deviation (contralateral conjugate eye deviation), wall-eyed bilateral internuclear ophthalmoplegia, one-and-a-half syndrome and eight-and-a-half syndrome. Anterior segment changes include mild iris atrophy. Posterior segment changes include arteriolar tortuosity, enhanced light reflex of the arterioles, increased venular tortuosity, narrower arteriolar calibre, wider venular caliber, arteriovenous nicking, microanuerysms and dot and blot haemorrhages.

Key words: Stroke, ophthalmic changes, risk factors, posterior segment changes

INTRODUCTION

Stroke or Cerebrovascular accident (CVA) refers to a clinical syndrome of presumed vascular origin, characterized by rapidly developing focal or global disturbances of cerebral function, lasting more than 24 hours or leading to death.[1] A stroke is a medical emergency and can cause permanent neurological damage and death[1]. Risk factors for stroke include old age, high blood pressure, previous stroke or transient ischemic attack (TIA), diabetes mellitus (DM) and high cholesterol; high blood pressure is the most modifiable factor.[2] Prevention of recurrence may involve the administration of antiplatelet medications such as aspirin and dipyridamole, control and reduction of high blood pressure, and the use of statins[1]. Stroke can be classified into two major categories: ischemic and hemorrhagic. Ischemic strokes are those that are caused by interruption of the blood supply, while hemorrhagic strokes are the ones which result from rupture of a blood vessel or an abnormal vascular structure[1]. Stroke is the second leading cause of death worldwide.[3] Stroke can affect people physically, mentally, emotionally, or a combination of the three, with the results of stroke varying widely, depending on size and location of the lesion.[1] Disabilities which affect 75% of stroke survivors are enough to decrease their employability.[4] The presence of visual impairment would make an already physically and mentally challenged patient depressed and frustrated.[5-7] Ocular involvement in stroke could be seen when it (stroke) affects areas of the brain

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that control eye functions e.g the brainstem and the parts of the cerebral cortex that are in charge of vision.[1] On the other hand, risk factors and causes of stroke are known to directly affect the eyes. The eye is often referred to as the "window" of the body. Early detection of ocular signs associated with stroke and/or its risk factors pointing towards future development of stroke could provide a rationale for medical intervention for the patient.[8]

Adnexal problems

Eye lid disorders are generally known to be found in the ocular motor nerve palsies. Generally, ptosis due to stroke is of neurologic origin, and could be unilateral or bilateral. Ptosis is also seen to be more common in patients with hemispheric cortical infarction from ischemic stroke (confirmed by cranial CT scan) than in patients with hemorrhagic stroke.[9] The prevalence of ptosis among stroke patients could range from 2.9% to 37.5%,[9,10,11] and it is noted that a higher prevalence is found with shorter time interval between the incidence of stroke and the time of ophthalmic examination. Amongst patients with stroke and hemiparesis with neurogenic ptosis, it is seen to be bilateral in 15.6% and unilateral in 21.9%.[10] Studies on extracranial dissection of the internal carotid artery show that up to 12% of these patients have cranial nerve palsies, with 5.2% having a syndrome of lower cranial nerve palsies, 3.7% having cranial nerve V palsy, and 2.6% having oculomotor nerve palsy.[11,12] Difficulty with initiating eye lid closure due to weakness of the orbicularis oculi (10%), difficulty with initiating eye lid opening due to levator palpebrae superioris weakness (2%) and inability to keep the eye lids open for more than 1-3 seconds were seen amongst patients with recent unilateral hemispheric stroke.[13]

Strabismus

Strabismus, when found in patients with stroke, are of neurological origin. Studies have shown that the prevalence of strabismus amongst stroke patients is 17.5% - 37%, with higher prevalence seen in newly diagnosed stroke and in stroke due to head injury.[14,15,16]This neurological feature resolves with time, and the prevalence could get as low as 0.6% in old cases of stroke.[9] These patients commonly complain of diplopia. Although strabismus occurs in stroke without brainstem involvement,[16] small strokes of the brainstem area are frequently associated with isolated ocular motor nerve (cranial nerve III, IV and VI) palsies.[17-19] Hence, brain stem stroke should be included in the differential diagnosis of isolated ocular motor palsies. Some strabismic conditions found in stroke patients include conjugate eye deviation (partial and forced), dorsal midbrain syndrome, binocular vertical and torsional diplopia and intermittent ophthalmoplegia.[20-23] Studies have also described rare ocular movement disorders associated with stroke which include wrong-way deviation (contralateral conjugate eye deviation) subsequent to supratentorial stroke and in the presence of huge intracranial hemorrhage, wall-eyed bilateral internuclear ophthalmoplegia (WEBINO) in atrophy of the midbrain tegmentum (revealed by MRI), and one-and-a-half syndrome in lacunar infarction in the paramedian pontine tegmentum (revealed by MRI)[24,25]as well as the eight-and-a-half syndrome if the seventh cranial nerve is also involved..

Anterior Segment

To the best of my knowledge, not much has been done in examining the anterior segment in patients with stroke. In a study, mild iris atrophy was found in 15% of patients with cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy (CADASIL). Among these patients with CADASIL, various degrees of lens opacities were also seen.[26,27] Another study showed cataract as a mere co-morbid ocular finding in stroke patients.[28]Some findings unrelated to stroke such as nasal pyerygium, arcus senilis and old keratic precipitates have been found amongst stroke patients.[9]

Posterior segment

Examination of the posterior segment of the eye, especially the optic disc, retinal vessels, retinal

background, could reveal systemic abnormalities in the body. Wider retinal venular caliber are known to predicted stroke,[29] and they are also known to be associated with marked progression of cerebral small vessel disease.[29,30,31] The calibre of retinal arterioles is not associated with prediction of stroke.[29] However, there is no association between ipsilateral retinal arteriolar/venular calibre, focal arteriolar narrowing or arteriovenous nicking and intracranial large artery disease.[32] Arteriolar tortuosity, increased venular tortuosity, narrower arteriolar calibre, and wider venular calibre were associated with ischemic stroke in another study.[33]. Yet in another study among patients with acute ischemic stroke, it was seen that patients with severe arteriovenous nicking were more likely to have a recurrent cerebrovascular event compared to those without atreriovenous nicking.[34] MRI-defined subclinical cerebral infarcts independent of stroke risk factors are known to be associated with retinal microvascular abnormalities, which include arteriovenous (A-V) nipping, focal arteriolar narrowing and crossings, blot hemorrhages, soft exudates, microaneurysm, sclerosis and tortuosity.[35,36] In patients with carotid artery stenosis (a major precursor of ischemic stroke), it was seen that those with hemodynamically significant carotid artery stenosis of >50% were 1.8 times more likely to have retinal vascular occlusions, 1.9 times more likely to have normotensive glaucoma and 2.4 limes more likely to have peripheral retinal haemorrhages.[37]Retinal ischemic symptoms are also found in patients with cerebral ischemic symptoms.[38] A study on cerebral amyloid angiopathy (CAA) confirmed on MRI reports 100% prevalence of bilateral multiple dot and blot retinal haemorrhages and multiple microanuerysm. It was concluded that microanuerysms and dot and blot haemorrhages seen fundoscopically mirror the histopathology of CAA.[39]In acute lacunar stroke retinal microvessel signs such as focal attenuated narrowing, arteriovenous nipping, enhanced light reflex of the arterioles, generalised arteriolar narrowing, small retinal arteriole, small retinal arteriole: venule ratio and retinal venular widening are seen.[40,41] The presence of cotton wool spots, hard exudates and flame-shaped hemorrhages in stroke patients are likely to be features and complications of the risk factors for stroke[9]. Other features in like extensive chorioretinal sclerosis and pigmentary changes of the macular are routine features found in the elderly.[9]

Conclusion

Most of the ophthalmic changes associated with stroke were neurological (as seen in adnexal changes and squint) and vascular (as seen on retinal examination). Routine ophthalmic examinations for all patients with risk factors for stroke could be important in predicting the occurrence of stroke before they occur.

REFERENCES

1. Alien CMC, Lueck CJ, Dennis M, Colledge NR, Walker BR, Ralston SH. Davidson's Principles and Practice of Medicine. 21st ed: Churchhill-Livingstone; 2010. 1180-1189.
2. Donnan GA, Fisher M, Macleod M, Davis SM. Stroke. Lancet 2008; 371: 1612-1623.
3. Mathers CD, Boerma T, Ma Fat D. Global and regional causes of death. Br Med Bull 2009; 92: 7-32.
4. Edward CC, Cuinmings JL, Sergio S, Robert R, Stroke - the American Psychiatric Press Textbook of Geriatric Neuropsychiatry (Second ed.). Washington DC: American Psychiatric Press 2000; 601-617.
5. Vahlberg B, Cederholm T, Lindmark B, Zetterberg L, Hellstrom K. Factors related to performance-based mobility and self-reported physical activity in individuals 1-3 Years after Stroke. J Stroke Cerebrovasc Dis 2013; 22: 426-434.
6. Jacova C, Pearce LA, Costello R, McClure. LA, Holliday SL. Hart RG, Benavente OR.Cognitive impairment in lacunar strokes. Ann Neurol 2012; 72: 351-362.
7. Fatoye FO, Mosaku SK, Komolafe MA, Eegunrati BA, Adebayo RA, Komolafe EO, Adewuya AO. Depressive symptoms and

associated factors following cerebrovascular accident among Nigerians. J Ment Health 2009; 18: 224-232.
8. Dubal FN, Hokke PE, Wardlaw JM. Retinal microvascular abnormalities and stroke. J Neurol Neurosurg Psychiatry 2009; 80: 158-165.
9. Olubor OJ, Uhumwangho OM, Omoti AE. Ocular disorders in stroke patients in a tertiary hospital in Nigeria. Niger J Clin Pract 2016; 19: 397-400.
10. Averbuch-Heller L, Leigh RJ, Mermelstein V, Zagalsky L, Streifler JY. Ptosis in patients with hemispheric strokes. Neurology 2002: 58: 620-624.
11. Shresthaa GS, Upadhyayaa S, Sharmaa AK, Gajurelb BP. Ocular-visual defect and visual neglect in stroke patients. J Optom 2011; 11:1.
12. Mokri B, Silbert PL, Schievink WI, Piepgras DC. Cranial nerve palsy in spontaneous dissection of the extra cranial internal carotid artery. Neurology 1996; 46: 356-359.
13. SchievinkWl, Mokri B, Garrity JA, Nichols DA. Piepgras DG. Ocular motor nerve palsies in spontaneous dissections of the cervical internal carotid artery. Neurology 1993; 43: 1938-1941.
14. Shresthaa GS, Upadhyayaa S, Sharmaa AK, Gajurelb BP. Ocular-visual defect and visual neglect in stroke patients. J Optom 2011; 11:1.
15. Rowe F. The profile of strabismus in stroke survivors. Eye (London) 2010; 24: 682-685.
16. Fowler MS, Wade DT, Richardson AJ, Stein JF. Squints and diplopia seen after brain damage. J Neurol 1996; 243: 86-90.
17. Patel SV, Mutyala S, Leske DA, Hodge DO, Holmes JM. Incidence, associations, and evaluation of sixth nerve palsy using a population-based method. Ophthalmology 2004; 111:369-375.
18. Kim JS, Kang JK, Lee SA, Lee MC. Isolated or predominant ocular motor nerve palsy as a manifestation of brain stem stroke. Stroke 1993; 24: 581-586.
19. Lee SH, Park SW, Kim BC, Kim MK, Cho KH, Kim JS. Isolated trochlear nerve palsy due to midbrain stroke. Clin Neurol Neurosurg. 2010: 112: 68-71.
20. Singer OC, Humpich MC, Laufs H, Lanfermann H, Steinmetz H, Neumann-Haefelin T. Conjugate eye deviation in acute stroke: incidence, hemispheric asymmetry, and lesion pattern. Stroke 2006; 37: 2726-2732.
21. Bhola R. Olson RJ. Dorsal Midbrain Syndrome with bilateral superior oblique palsy following brainstem hemorrhage. Arch Ophthalmol 2006; 124: 1786-1788.
22. Gregory ME, Rahman MQ, Cleary M, Weir CR. Dorsal midbrain syndrome with loss of motor fusion. Strabismus 2011; 19: 17-20.
23. Kim JS. Internuclear ophthalmoplegia as an isolated or predominant symptom of brainstem infarction. Neurology 2004; 62: 1491-1496.
24. Johkura K. Nakae Y, Yamamoto R, Mitomi M, Kudo Y. Wrong-way deviation: contralateral conjugate eye deviation in acute supratentorial stroke. J NeurolSci 2011; 308: 165-167.
25. Ushio M, iwasaki S, Chihara Y, Murofushi T. Walled-eyed bilateral internuclear ophthalmoplegia in a patient with progressive supranuclear palsy. J Neuroophthamol 2008; 28: 93-96.
26. Ilniczky S, Kamondi A, Varallyay G, Gaal B, Palasti A, Gulyas S, Szirmai I. One-and-a-half syndrome-two cases. IdeggyogySz 2007; 60: 489-93.
27. Roine S, Harju M, Kivela TT, Poyhonen M, Nikoskelainen E, Tuisku S. Ophthalmologic findings in cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy: a cross-sectional study. Ophthalmology 2006; 113: 1411-1417.
28. Garg BP, DeMyer WE. Ischemic thalamic infarction in children. Pediatr Neurol 1995; 13:46-49.

29. McGeechan K, Liew G, Macaskill P, Irwig L, Klein R, Klein BE. Prediction of incident stroke events based on retinal vessel caliber. Am J Epidemic 2009; 170: 1323-1332.
30. Ikram MK, De Jong FJ, Van Dijk EJ, Prins ND, Hofman A, ;Breteler MM. Retinal vessel diameters and cerebral small vessel disease: the Rotterdam Scan Study Brain 2006; 129: 182-188.
31. Ikram MK, De Jong FJ, Bos MJ, Vingerling JR, Hofman A, Koudstaal PJ. Retina vessel diameters and risk of stroke. Neurology 2006; 66: 1339-1343.
32. De Silva DA, Manzano JJF, Liu EY. Woon F, Wong VVX. Associations of retinal microvascular signs and intracranial large artery disease. Stroke 2011; 42: 812-816.
33. Ong YT, De Silva DA, Cheung CY. Chang HM, Chen CP, Wong TY, Ikram MK. Microvascular structure and network in the retina of patients with ischemic stroke.Stroke2013; 44: 2121-2127.
34. De Silva DA, Manzano JJ, Liu EY,;Woon FP, Wong WX, Chang HM. Retinal microvascular changes and subsequent vascular events after ischemic stroke. Neurology 2011; 77: 896-903.
35. Cooper LS, Wong TY, Klein R, Sharrett AR, Bryan RN, Hubbard LD, Couper DJ, Heiss G, Sorlie PD. Retinal Microvascular Abnormalities and MRI-Defined Subclinical Cerebral Infarction. Stroke 2006; 37: 82-86.
36. Kwa VIH, van der Sande JJ, Stam J, Tijmes N, Vrooland JL. Retinal arterial changes correlate with cerebral small-vessel disease. Neurology 2002; 59: 1536-1540.
37. Lyons-Wait VA, Anderson SF, Towasend JC, De Land P. Ocular and systemic findings and their correlation with hemodynamically significant carotid artery stenosis. Optom Vis Sci 2002; 79: 353-362.
38. Persoon S, Klijn CJM, Algra A, Kappelle LJ. Bilateral carotid artery occlusion with transient or moderately disabling ischaemic stroke: clinical features and long-term outcome. J Neurol 2009; 256: 1728-1735.
39. Lee A, Rudkin A, Agzarian M, Patel S, Lake S, Chen C, Retinal vascular abnormalities in patients with cerebral amyloid angiopathy. Cerebrovasc Dis 2009; 28: 618-22.
40. Lindley RI, Wang JJ, Wong MC, Mitchell P, Liew G, Hang P. Retinal microvasculature in acute lacunar stroke: a cross-sectional study. Lancet Neurol 2009; 8: 628-634.
41. Liew G, Baker ML. Wong TY, Hand PJ, Wang JJ, Mitchell P. Differing associations of white matter lesions and lacunar infarction with retinal microvascular signs. Int J Stroke 2012; 18: 1747-4949.