Original Research Paper Ophthalmology

A study of the ocular manifestations in patients with Cerebrovascular Accidents.

Dr Gargi A Shah	MS Ophthalmology, DOMS, FCPS, Fellow(Paediatric Ophthalmology and Strabismus)
Dr Anamika H. Agrawal	DNB (Ophthalmology), DO, DOMS, FICO. Associate Professor, TNMC & BYL Nair Hospital, Mumbai. - Corresponding author
Dr Chanchal Gadodiya	MS Ophthalmology , FICO SMO, Municipal Eye Hospital, Mumbai
Dr Anjali D Nicholson	MS Ophthalmology, DNB, ICO (Vitreoretinal Surgeon) Ex- Prof & HOD TNMC & BYL Nair Hospital, Mumbai.

ABSTRACT

A study of the ocular manifestations in patients with Cerebrovascular Accidents.

Purpose: To study the ocular manifestations in patients with cerebrovascular accidents and to co-relate clinically.

Methods: A prospective, observational study was conducted on 50 patients diagnosed as stroke at tertiary care hospital. Patients with head or ocular injury, CNS infection or pre-existing ocular motility disorder were excluded. Visual assessment, pupillary reaction, anterior segment and fundus examination, ocular motility, visual fields & neurological examination were done. Neuro imaging findings were noted.

Results: Of 50 patients, 3 were venous, 37 were ischaemic and 10 were haemorrhagic arterial stroke. 14% had pupillary abnormalities, 14% had papilloedema, 46% had visual field defects commonest being contralateral homonymous hemianopia and 48% had ocular motility defects. MCA was affected most commonly.

Conclusion: Studying ocular manifestations in CVA can help in localising the level of lesion in brain.

KEYWORDS : Stroke, motility defect, hemianopia, pupil, papilloedema

Introduction

A cerebrovascular accident(CVA)also known as stroke is characterized by sudden loss of blood circulation to an area of brain, resulting in a corresponding loss of neurologic function.1 It may be haemorrhagic or ischemic. Acute ischemic stroke is caused by thrombosis or embolism and is commoner than haemorrhagic stroke.[2,3] Within seconds to minutes of the loss of perfusion to a portion of the brain, an ischemic cascade is unleashed & if left unchecked, causes a central area of irreversible infarction surrounded by an area of potentially reversible ischemic penumbra. CVA have associated ophthalmologic findings, which are important to recognize & can be of great help in localizing the site of lesion. However, many patients with stroke do not receive detailed eye examination. Our study highlights ophthalmologic manifestations associated with CVA & evaluates the relationship between ocular–visual defect and stroke thus emphasizing need for visual assessment of such patients.

Materials and Methods

This prospective, observational study was conducted at tertiary care hospital on 50 patients diagnosed with CVA. The study was approved by Institutional Ethics Committee. Patients with history of head or ocular injury, tuberculous or pyogenic meningitis were excluded. Written informed consent was taken. History about presenting complaints, risk factors like smoking or any medications & systemic illness like hypertension or diabetes was taken. Visual disturbances, diplopia, field defect or altered colour perception if any were enquired. Radiological findings were noted.

On examination, visual acuity, any deviation of eyes, gaze preferences, nystagmus & extraocular movements were assessed wherever possible. Adnexa and anterior segment examination was done and pupillary reaction was checked. Exposure keratitis if present was treated with lid taping, lubricants and antibiotic ointment. Fundus examination was done and hypertensive or diabetic retinopathy changes, papillodema were noted. Visual field was done by confrontation test or perimetry. Neurological evaluation was done. Radiological findings on CT scan/ MRI were noted including presence of ischemia or haemorrhage, area of brain affected and the blood vessel involved.

Results

In our study, 92% patients were of age >50 years. Out of 50 patients, 47(94%) had arterial while 3(6%) had venous stroke. Out of 47 arterial stroke, 37 had ischemic &10 had haemorrhagic stroke. In 62% of ischemic and 60% of haemorrhagic stroke MCA was involved.

Ischemic Arterial Stroke

Blood vessel involved	Area of brain affected	No. Of patients Affected	Ocular manifestations
PCA (10)	Occipital infarct (Dominant lobe)	5	Contralateral Homonymous Hemianopia with macular sparing 4 of them had altered colour perception
	Occipital+ Thalamic infract	2	Contralateral homonymous hemianopia with macular sparing
	Thalamic	1	Vertical gaze palsy
	Mid brain	2	Ipsilateral III N Palsy, Pupil fixed and dilated, ptosis
Basilar(2)	Right inferior pons	1	Right 6th and 7th nerve palsy
	Right paramedian basal tegmental infarct	1	Right INO

MCA & PCA (2)	Right parieto-occipital infarct (non dominant lobe)	1	Contralateral(left) homonymous hemianopia Contralateral visual neglect.
	Left parieto occipital infarct with bilateral frontal horn of lateral ventricle	1	Left eye:visual acuity is perception to light :negative Left eye:pupil:rapd Left eye showed central retinal artery occlusion Right eye:visual acuity:perception and projection to light present
MCA (23)	gangliocapsular infarct	15	4 had Gaze preference(conjugate deviation) to the side of lesion 6 had contralateral homonymous hemianopia
	gangliocapsular and frontoparietal infarct	5	Gaze preference(conjugate deviation) to the side of lesion contralateral homonymous hemianopia
	parieto temporal infarct	2	contralateral homonymous hemianopia
	parieto temporal and frontal infarct	1	contralateral homonymous hemianopia

Hemorrhagic Arterial Stroke			
MCA(6)	Gangliocapsular	5	All had ocular deviation to the same side 2 of them had ipsilateral dilated pupil
	Parieto temporal lobar	1	gaze preference to the side of lesion
PCOM (1)	Aneurysm rupture with subarachnoid hemorrhage.	1	Complete 3rd nerve palsy Papilloedema
PCA (1)	right Posterolateral thalamic	1	ocular skew deviation with left eye being downward and medially unreactive pupils
Vertebro basilar artery (2)	Pontine	1	pinpoint pupils reacting to light
	Cerebellar	1	ocular bobbing papilloedema

Venous Stroke

Superior sagittal sinus thrombosis(3)	Raised ICT	2	Papilloedema
		1	6th nerve palsy & Papillodema

Discussion

Following stroke, impairments in the visual system such as loss of vision, visual field defect, extra ocular muscles paralysis, diplopia, and visual perception deficits are well documented. Documenting ophthalmic features in stroke patients will not only help physician to localize the site of lesion but also help in rehabilitation of patients.

In our study, 47 patients(94%) had arterial stroke and 3(6%) had venous stroke. Results were similar to study by Ellekjær et al, suggesting arterial stroke are more common than venous stroke.[4] Majority of patients (57.44%) were in an age group of 51-60 years. As per study by 'National Institute of Neurological Disorders and Stroke'(1999) advanced age is one of the most significant stroke risk factors.[3] 95% of strokes occur in people age 45 and older. On other side all 3 patients of venous stroke were between 31-40 years with risk factors being homocysteinemia postpartum state and intake of oral contraceptives (OC).[5] Our findings are consistent with Canu et al who found association of cerebral venous sinus thrombosis with pregnancy, puerperium & use of OC pills as risk factors.[6]

In our study, 64% were males and 36% were females. According to Peter Appelros et al, there was sex differences in stroke epidemiology; male stroke incidence & prevalence rate was 33% & 41% higher than female.[7,3]

Visual acuity of our patients ranged from 6/6 to No perception of light (PL) depending on area of brain affected. 5 patients with ischemic arterial stroke reported history of transient vision loss consistent with thromboembolic phenomenon. One patient had No PL due to ischemic CRAO with ipsilateral carotid artery stenosis.[8] Four of 5 patients with dominant (left)occipital lobe involvement showed altered colour perception. Similar findings were noted by Mohr JP et al.[9]

Pupils were abnormal in 14% . Ipsilateral dilated fixed pupil were found in 2 patients with midbrain infarct of PCA territory, 4 with haemorragic stroke & 1 with PCOM artery aneurysm rupture related haemorrhagic stroke. PCA infarct causes damage to midbrain parasympathetic centre leading to unopposed sympathetic action and hemorrhagic stroke results into raised intra cranial tension (ICT) causing uncal herniation pressing on 3rd nerve thus leading to pupillary dilatation.

Papillodema was seen in 14% with majority having haemorrhagic arterial stroke with raised ICT. All patients with venous stroke had papilloedema due to superior sagittal sinus thrombosis causing inadequate CSF drainage and raised ICT. Similar findings were noted by Hossein Azin et al.10Thus fundus examination recommended in all stroke patients.

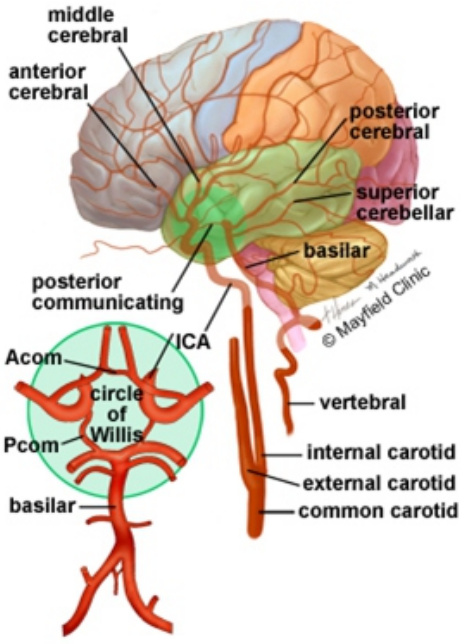
Visual field defects were found to be affected in 46%. Contralateral homonymous hemianopia was commonest field defect observed, associated with Occipital/ Parieto-occipital infarct.[11] Ocular motility was found to be affected in 48%. Most common motility defect was gaze preference i.e conjugate deviation of eye towards the site of lesion seen in 15 patients with gangliocapsular area involved due to MCA ischemia or haemorrhage. Study by Hier DB et al, on patients with putaminal haemorrhage showed homonymous hemianopia and gaze preference as common manifestations.[12]

3rd nerve palsy was seen in 3 patients, 2 had mid brain ischemia due to MCA involvement. While one had ruptured aneurysm of PCOM leading to subarachnoid haemorrhage and raised ICT resulting in brain stem herniation and 3rd nerve compression. [13] 6th nerve palsy was observed in 2 patients, one of them had associated 7th nerve palsy due to Basilar artery ischemia involving inferior pons. Similar finding were noted by Satoshi Kataoka, MD et al.[14] Other patient had superior sagittal sinus thrombosis leading to raised ICT and bilateral 6th nerve palsy as false localizing sign.

Internuclear Ophthalmoplegia(INO) was observed in patient with basal tegmental paramedian pontine infraction.[15] The cardinal findings in INO are impaired adduction and abducting nystagmus during conjugate version movements occurs due to dysfunction of the medial longitudinal fasciculus (MLF), a tract that contains axons projecting from VI nucleus to medial rectus subnuclei of the contralateral III nuclear complex. Ocular skew deviation was observed in one patient who had PCA hemorrhagic stroke causing right posterolateral thalamic hematoma. Study by Kumral E et al, showed that all such patients had severe sensorimotor deficit. Several variants of vertical gaze dysfunction, skew ocular deviation, gaze preference toward the site of the lesion, and miotic pupils were frequent in posterolateral thalamic haemorrhage, particularly in the large type.[17] Vertical gaze palsy was observed in one patient with bilateral paramedian thalamic infarct. Similar finding was found by Massimo Gentilini et al. [16]

To summarize ocular findings roughly guide us towards the location of CVA.

	Eye Manifestation	Localization
1	Vision No PL	Cortical Blindness Ischemic CRAO
2	Abnormal Color Vn	Dominant lobe (Occipital & Temporal)
3	Pupils	
	Pin point	Pontine Haemorrhage
	Dilated	Stroke involving ventral Midbrain Damage to Parasympathetic fibers in Putamen bleed
	Not reacting to light	Posterolateral Thalamic haemorrhage
4	Papillodema	Raised ICT in case of haemorrhage, Venous stroke
5	Visual field defect Contra lateral Homonymous hemianopia	Occipital/ Parieto occipital infarct
6	III N Palsy	Midbrain
7	VI N palsy	Papillodema – False localising sign
8	VI & VII N palsy	Ventral & Inferior Pons
9	INO	Pons
10	One & half Syndrome	PPRF & MLF
11	Horizontal gaze palsy	Along with INO Upper Dorsal Pons
12	Vertical gaze palsy With CRN & Skew deviation	Thalamus Midbrain (Basilar Artery Occlusion)
13	Skew dev	Along with INO
14	Eyes deviation to opp side	Postero-lateral Thalamic hemorrhage
15	Eyes deviation to same side	MCA Stroke Putamen/ Ganglio-Capsular hemorrhage
16	Horners Syndrome	Vertebro Basilar Artery infarct Ipsilateral Lateral Medullary Synrome
17	Nystagmus	
	Vertical	Vertebro Basilar Artery infarct



Conclusion:

Stroke is a common condition, with significant effects on a patient's ability to live an active and independent life. Ocular problems are common in patients with stroke, it is important that their implications are understood. Careful clinical observation of the neurological & ophthalmological features of affected patients may guide to the site of lesion in the brain which may facilitate early diagnosis & treatment.

References:

1. World Health Organisation, (1978). Cerebrovascular Disorders- Clinical and research Classification, Offset Publication Geneva: World Health Organization. ISBN 9241700432. OCLC 4757533.
2. "Brain Basics: Preventing Stroke". National Institute of Neurological Disorders and Stroke. Retrived 2009-10-24
3. National Institute of Neurological Disorders and Stroke (1999). "Stroke: Hope Through Research". National Institutes of Health.
4. Ellekjær, H; Holmen J, Indredavik B. "Epidemiology of Stroke in Innherred, Norway, 1994 to 1996 : Incidence and 30-Day Case-Fatality Rate". Stroke 28 (11): 2180–2184.
5. Martinelli I, Battaglioli T, Pedotti P. Hyperhomocysteinemia in cerebral vein thrombosis. Blood 2003;102:1363-6.
6. Canu C, Barinagarrementeria F. Cerebral venous thrombosis associated with pregnancy and puerperium: a review of 67 cases. Stroke1993; 24:1880–4.
7. Peter Appelros; Birgitta Stegmayr, Andreas Terént, Sex Differences in Stroke Epidemiology,Stroke. 2009;40:1082
8. Hollenhorst RW , "Significance of bright plaques in the retinal arterioles". Jama PMID 1961. 178: 23–29.
9. Mohr JP, Leicester J, Stoddard LT. Right hemianopia with memory and color deficits in circumscribed left posterior cerebral artery territory infarction. Neurology. Nov 1971;21(11):1104-13.
10. Hossein Azin, Nahid Ashjazadeh. Cerebral Venous Sinus Thrombosis-Clinical Features, Predisposing and Prognostic Factor. Acta Neurol Taiwan. 2008 Jun;17(2):82-7
11. Zhang X, Kedar S, Lynn MJ, Newman. Homonymous hemianopia in stroke.Neuroophthalmol. 2006 Sep;26(3):180-3.
12. Hier DB, Davis KR, Richardson EP Jr. Hypertensive putaminal hemorrhage.Ann Neurol. 1977 Feb;1(2):152-9.
13. Yamana T, Murakami N, Itoh E, A Weber's syndrome of ischemic vascular origin--a clinical and neuroradiologic study, No To Shinkei, 1993 Apr;45(4):349-54.
14. Satoshi Kataoka, ; Ariyuki Hori, ; Tomoyasu Shirakawa, Genjiro Hirose, Paramedian Pontine Infarction Neurological/Topographical Correlation. Stroke, Aha Journals 1997 ;28:809-815
15. Emre Kumral, ; Tülay Kocae, Özgen Ertübey, Thalamic Hemorrhage A Prospective Study of 100 Patients. Stroke, 1995;26:964-970
16. Massimo Gentilini, Ennio De Renzi, Bilateral paramedian thalamic artery infarcts: report of eight cases. Journal of Neurology, Neurosurgery , and Psychiatry 1987;50:900-909