



PROFILE OF STROKE PATIENTS WITH CORTICAL VISUAL IMPAIRMENT IN A TERTIARY CARE HOSPITAL.

Dr. Parvezalam M Tank*

Final Year postgraduate resident, Dept. of Ophthalmology, Yenepoya Medical College, Derlakatte, Mangalore, Karnataka- 575018 *Corresponding Author

Dr. Sithara Sara Oomman

Final Year postgraduate resident, Dept. of Ophthalmology, Yenepoya Medical College, Derlakatte, Mangalore, Karnataka- 575018

ABSTRACT

Cortical visual impairment is a rare neurological disorder characterized by loss of vision due to damage to occipital cortex. Ischemic cerebrovascular disease is the most common etiology. There may be partial or complete recovery of visual function depending on the underlying damage. Possibility of cortical visual impairment should be kept in mind, in case of any atypical visual loss. All stroke patients should undergo a mandatory ophthalmological examination irrespective of whether they complain of vision loss or not.

KEYWORDS : Cortical visual impairment, Cerebrovascular disease

Introduction:

Cortical blindness is a rare complication of posterior circulation stroke. However, its complex presentation with sensory, physical, cognitive and behavioral impairments makes it one of the most challenging entity for diagnosis and management. (Gaber, 2010) The term cortical blindness has been in use conventionally; however the term 'cortical visual impairment' would be a better terminology because the amount of vision loss in each case is variable. Cortical visual impairment is total or partial loss of vision in a normal eye, due to damage to occipital cortex of the brain. It can be acquired or congenital, and may also be transient or permanent. Acquired cortical visual impairment is most commonly caused by caused by cerebrovascular accidents. Here, the impaired blood flow to the occipital cortex can occur from either unilateral or bilateral posterior cerebral artery blockage (ischemic stroke). In most cases the complete loss of vision is not permanent and patient may recover some of their vision. Congenital or neonatal cortical visual impairment is most often caused by perinatal brain damage due to impaired blood/oxygen supply, sepsis, encephalitis or meningitis.

Cerebrovascular disease is the most common cause of cortical vision loss in adults. Stroke, whether ischemic or hemorrhagic in nature, has the ability to culminate in devastating clinical outcomes. The mechanisms of ischemic stroke are variable, complex and multifactorial in nature. These include thromboembolic stroke secondary to atherosclerosis in the elderly patient population and structural cardiac or vasculopathic/metabolic etiologies in younger patients.

Strokes have a high likelihood of involving vision in some way. Vision loss can be the most disabling residual effect after a cerebral infarction. (Pula JH, Yuen CA, 2017) Criteria for Cortical blindness includes loss of all visual sensations, loss of menace reflex, preservation of pupillary reflexes, a normal fundoscopic examination and preservation of ocular movements. Depending on the severity of brain damage, patients may have some amount of visual recovery over a period of few weeks to months.

Methodology:

This study is an analysis of patients who were admitted with stroke in a tertiary care hospital and were seen by ophthalmologists and were diagnosed to have cortical visual impairment. All patients seen during the period of one year from August 2016 to September 2017 were included. Stroke patient with normal vision and patients having low vision due to ocular problems were excluded from the study. Consent was obtained from all the patients for reporting their case and all data were de-linked from patient details to maintain confidentiality and anonymity. Clinical profile of each patient was retrospectively studied. All the patients were admitted and were evaluated and treated by the physician and/or neurologist. MRI was done for all patients who had clinical and radiological evidence of posterior circulation stroke. All patients were seen by ophthalmologist (referred or directly presented). Vision testing, anterior segment examination and fundus evaluation was done for all patients.

Results:

Out of eight cases, five patients were males and three were females. Age of the patients ranged from 50- 85 years. The cause of cortical visual impairment in all cases was cerebrovascular accident. All the eight patients were hypertensive and four patients were diabetics. Anterior segment examination was unremarkable in all (apart from senile immature cataract in two eyes). Pupillary reaction was brisk. On fundus examination two patients had evidence of mild non proliferative diabetic retinopathy. However none of these accounted for the amount of visual loss. Seven out of eight patients complained of visual loss and were aware of their visual impairment. Visual acuity at the time of presentation ranged from counting fingers at three meters to perception of light. However patients had poor comprehension at this point. Visual acuity at the time of follow up visit (at one month) ranged from 6/12 on Snellen chart to perception of light. One patient had denial of blindness and even during follow up visit he exhibited confabulation regarding his vision.

Discussion:

Cortical blindness, a rare neurological disorder is characterized by loss of vision caused by unilateral or bilateral lesions in visual cortex and presence of intact anterior visual pathways. The lesions are most often binocular with preserved papillary light reflexes. They are results of an insult in the occipital lobe cortex.

Cortical vision loss has been described in association with cerebral venous thrombosis, pulmonary embolus, pregnancy (preeclampsia and eclampsia), hepatic encephalopathy, post cardiac surgery and cerebral vascular accidents. The common pathologic component is ischemia of the occipital cortex, either as a result of local event (hemorrhage and embolism) or more commonly as a result of global process. Alternate etiologies include migraine headache, occipital trauma (head injury with subdural or epidural hematoma), meningitis, carbon monoxide or other poisoning and neoplasm. Cortical blindness is sometimes difficult to differentiate from hysterical blindness since the pupil may still react to light. The diagnosis of cortical blindness is based on the exclusion of disease in the anterior visual tract by history and complete neuro-ophthalmologic evaluation. Pupillary response to light and corneal reflexes are intact because these functions are independent of cortical integrity. Functions dependent on the optic cortex, such as the blink response to threat and opocinetic nystagmus are absent.

Magnetic resonance imaging is recommended as the diagnostic imaging technique of choice in these patients. It useful in the exclusion of hemorrhage or neoplastic process. Also it helps in detection of subtle vasogenic edema in the brain (characteristic of vasoconstriction and ishemia) as well as detailed evaluation of the venous sinuses and anterior visual tracts.

The prognosis for patients with cortical blindness depends on the cause, severity, duration, speed of initial recovery, age, and medical history. (Galetović, Karlica, Bojić, & Znaor, 2005) The mode of recovery from cortical blindness has been studied carefully by Gloning

and colleagues, who describe a regular progression from cortical blindness through visual agnosia and partially impaired perceptual function to recovery. Even with recovery, the patient may complain of visual fatigue and difficulties in fixation and fusion.(Srikant, Deepa, Murthy, & Dhar, 2012) In our study, we found a variable degree of visual recovery when examined the patients during their follow up visits at the end of one month. Out of eight cases, there was remarkable improvement in visual acuity in three patients, in three patients there was some improvement and two patients showed no recovery at all.

Bilateral infarction in the distal PCAs produces cortical blindness and also damage to visual association areas of the cortex. In such cases, the patient is often unaware of the blindness or may even deny it. Cortical blindness associated with denial of vision loss and confabulation is known as Anton's syndrome (Anton-Babinski syndrome or Visual anosognosia). Tiny islands of vision may persist, and the patient may report that vision fluctuates as images are captured in the preserved portions. (Pula JH at al., 2017) Although cerebrovascular disease is the most common cause; surgery, particularly cardiac surgery and cerebral angiography are also major risk factors.(Galetović et al., 2005) In our study we have assessed cortical visual impairment in patients with cerebrovascular accident only. All our eight patients had infarcts in the PCA territory. This was seen on imaging (MRI). Only one patient in our series showed features consistent with denial of blindness. The clinical presentation included confabulations. Damage of the visual association cortex has been thought as one of the main causes explaining the loss of awareness of the visual deficit. (Maddula, Lutton, & Keegan, 2009) Limitation of our study is that we could include only a few patients with cortical visual impairment as most of the stroke patients were lost to follow up. We measured only visual acuity; however measuring colour, contrast and field of vision would be more informative.

Conclusion:

Ischemic cerebrovascular disease is the most common etiology of cortical visual impairment. There may be partial or complete recovery of visual function depending on the underlying damage. Diagnosis of this condition is based on the exclusion of disease in the anterior visual tract, and by detailed history and complete neuro-ophthalmological and radiological evaluation. Possibility of cortical visual impairment should be kept in mind, in case of any atypical visual loss. All stroke patients should undergo a mandatory ophthalmological examination irrespective of whether they complain of vision loss or not. Follow up visits are important to assess the prognosis. Stroke patients need careful visual assessment and visual rehabilitation.

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