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Cortical Blindness- case report

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Abstract: *Cortical blindness is defined as the total loss of vision in the presence of normal pupillary reflexes and in the absence of ophthalmological disease.We report a case of cortical blindness as a result of diffusion restriction in left parieto-occipital-lobe-Acute infarct in left posterior cerebral arteries (PCA) territory and gliosis in right occipital,right temporal lobe. Cerebrovascular disease could be the cause of cortical blindness in our patient.We discuss the possible aetiologies, prognosis and reviewed with relevant literature regarding our patient's condition.*

Key words: *Cortical blindness, Posterior cerebral arteries, Gliosis.*

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I. Introduction

Cortical blindness, a particular type of cerebral blindness, is defined as the total loss of vision in the presence of normal pupillary reflexes and in the absence of ophthalmological disease [1], resulting from bilateral lesions of the striate cortex in the occipital lobes [2].

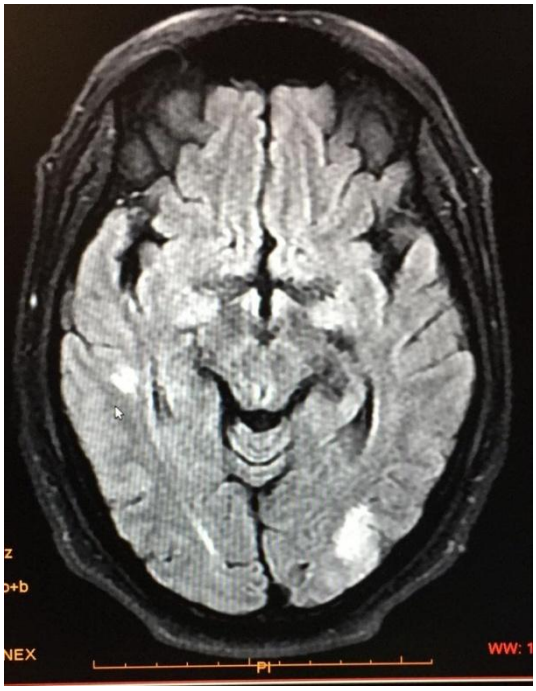
The most common cause of cortical blindness is bilateral occipital lobe infarctions in the vascular territory of the posterior cerebral arteries (PCA). The most frequent finding in patients with PCA infarction is a hemianopia.PCA infarction is most often secondary to emboli from the heart or vertebrobasilar circulation, is less frequently due to prolonged hypotension or hypoxia and leads to homonymous hemianopia, often without other neurologic deficits. Simultaneous bilateral infarctions can be seen in a variety of clinical settings, such as hypertensive crisis, cerebral hypoperfusion and cardiac embolism to the basilar artery or transtentorial herniation.The overall prognosis of bilateral occipital lobe infarcts is poor[1]

II. Case report:

We report a case of 61 years old male with complaint of sudden painless loss of vision. He experienced a headache in the occipital region when he bent down to pick up an object.Patient is a known case of hypertension with depression and is on regular medication for both conditions. Patient's vision were CFCF in both eyes.Pupillary reflexes were intact and fundoscopy showed no obvious abnormality.Ocular malingering test like Schmidt-rimpler test,menace test and finger-finger test were performed but all the test results were unremarkable. Visual field examination could not be done due to poor vision.No sensory or motor deficient in any limbs. His blood pressure 150/90mmHg,pulses 86/m and RBS 106mg/dl

We referred the patient to a neurologist. MRI was done which showed diffusion restriction in left parieto-occipital-lobe-Acute infarct in left posterior cerebral arteries (PCA) territory and gliosis in right occipital,right temporal lobe. Patient was treated with tab Strocit 500mg (citicoline),tab Clopilet A 150mg,tab Aztor 40mg,tab Nexito plus, tab Hemisure,tab Pantocid.Patient came for follow-up in our OPD after 2 months and his visual acuity improved to 6/6 bilaterally.

Figure: MRI (FLAIR) showing hyperintense lesion in left parieto-occipital lobe and gliosis in right temporal lobe



III. Discussion

The most common cause of cortical blindness is occlusion of the posterior cerebral arteries (embolic or thrombus) [3,4]. Hypoxic-ischaemic encephalopathy,Progressive multifocal leucoencephalopathy, other leucodystrophies and bilateral gliomas are other causes [5].

Cortical blindness is a consequence of dysfunction or destruction of brodmann area 17 in both occipital lobes.Unilateral occipital lobe lesion produce contralateral homonymous hemianopia.In our patient,profound diminution of vision were noted bilaterally where visual field could not be elicited though there was infacrtion only in the left parieto-occipital lobe in the left PCA territory. However,gliosis was noted in right occipital and right temporal lobe.Therefore,gliosis could be the additional factor to infarction that causes bilateral poor vision in our case.Rutledge et al[6] reported a case which showed that profound gliosis can cause cortical blindness. An alternative explanation for bilateral cortical involvement with a unilateral structural lesion would be that of diaschisis. Von Monakow[7] initially proposed the concept of diaschisis in 1905, suggesting that after focal brain injury neurons in regions of morphologically intact brain, remote from but anatomically connected to the damaged area, become functionally depressed because of a loss of excitatory input from the injured area.Transhemispheric diaschisis, manifested by decreased cerebral blood flow in the opposite hemisphere to the lesion, has also been described in patients with unilateral stroke.[8] Other authors[9] have also reported bihemispheric decreased blood flow and metabolism in patients with unilateral cerebral infarctions and have attributed this finding to diaschisis. Additionally, clinical evidence of transhemispheric dysfunction in our patient was manifested by severe bilateral visual impairment.

The prognosis for cortical blindness patients depends on the age, medical history, cause, severity, and duration as well as the speed of initial recovery[10,11,13].Good recovery of visual function has been noted in conditions such as hypertensive encephalopathy, cardiac surgery, cerebral angiography, and infective endocarditis [10, 12, 14]. Aldrich et al. [10] mentioned that better visual outcome was observed in (i) young patient (<40 years old), (ii) no history of hypertension and diabetes, (iii) no cognitive, language, or memory impairment, and (iv) CVA is not the causative factor. Our patient had recovered with good vision after few months of attack.The reason could be gliosis was followed by restoration of disrupted sensory and motor function of the brain and also unilateral infarction showed better prognosis than bilateral infarction which was supported by the case study done by J.Bogousslavsky et al.[15] where they found a clear cut association between the occurance of bilateral occipital infarction and a lack of improvement of the initial visual field defect and also found that patients with unilateral occipital infarction has non or mildly disabled functional classes.

There were cases that have reported cortical blindness with Anton syndrome secondary to cerebrovascular accident but our patient has no such symptom.

IV. Conclusion

A suspicion of cortical blindness should be raised in patients with atypical visual loss and evidence of occipital lobe injury.Most of the cortical blindness cases are reversible regardless of aetiology and have no long term sequelae. Since cerebrovascular disease is one of the most common causes of cortical blindness ,as reported in our case herein,a careful follow-up is indicated in these patients so cortical blindness can be prevented with an early detection and institution of an adequate therapy.

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