Case Report

ISOLATED HOMONYMOUS HEMIANOPIA: A RARE PRESENTATION POSTERIOR CEREBRAL ARTERY INFARCT

Tikambari Etthirajan[1,2], Mohammad Aziz Salowi[1] & Fazliana Ismail[2]

[1]Department of Ophthalmology, Hospital Selayang, Lebuhraya Selayang-Kepong, 68100 Batu Caves, Selangor, Malaysia.
[2]Department of Ophthalmology, Faculty of Medicine, University Malaya, Jalan Profesor Diraja Ungku Aziz, 50603 Kuala Lumpur, Malaysia.

ARTICLE INFO

Corresponding author:
Dr. Tikambari Etthirajan

Email address:
tigam2s@gmail.com

Received:
September 2022
Accepted for publication:
September 2022

Keywords:
visual disturbances;
isolated homonymous hemianopia;
posterior cerebral artery infarct

ABSTRACT

Ischemic stroke presents with various physical malfunctions. Visual disturbances are commonly related to limbs or facial weakness, on rare occasions could be the only presenting symptom. We report a 49-year-old man with uncontrolled co-morbidities, presented with sudden bilateral blurring of vision, specifically the loss of his peripheral field of vision. Ocular examination showed normal anterior and posterior segments in both eyes. Confrontation test showed right sided visual field defect, confirmed to be right homonymous hemianopia with automated Humphrey visual field analyser. Apart from visual field defect there were no other central nervous system signs and symptoms. Computed Tomography (CT) of brain revealed left posterior cerebral artery infarct. Visual symptoms may not always accompany other neurological manifestations of stroke but may present as isolated sign in rare occasions. Detailed history, examination and targeted investigation aid the diagnosis.

INTRODUCTION

Ischemic strokes reach around 150 000 every year in the United Kingdom alone, with the common associated etiology being atherosclerosis, small artery diseases and embolisms. About 20-25% of these numbers are posterior circulation infarct affecting structures such as the brainstem, cerebellum, midbrain, thalamus, and areas of the temporal and occipital cortex. These structures are supplied by the vertebrobasilar arterial system [1].

Posterior cerebral artery (PCA) is part of the posterior circulation. Infarction of this artery contributes to 5-10% of ischemic strokes [2]. They commonly present with symptoms such as hemisensory loss and hemi-body pain which is usually burning in nature due to thalamic infarction [2]. In bilateral PCA infarct, often there is reduced visual-motor coordination. Whereas, the most common signs were found to be unilateral limb weakness (38%), gait ataxia (31%), unilateral limb ataxia (30%), dysarthria (28%), and nystagmus (24%). (3) Signs and symptoms may vary due to the location and severity of occlusion, and availability of collaterals [4].

Some other presentations of PCA infarct are visual field defects, visual dysfunction, and cognitive and behavioural dysfunction [4]. Posterior cerebral artery supplies the lower part of optic radiations while the upper part receives blood from the deep branches of the middle cerebral artery (MCA). Therefore, in visual field defects, contralateral homonymous hemianopia (HH) with macular sparing can be caused by unilateral infarctions of the occipital lobe. In limited defects (unilateral superior or inferior optic radiation infarct), quadrantanopia results. Occlusion of the posterior choroidal artery too leads to visual field defects (hemianopia, quadrantanopia, sectoranopia), hemisensory deficit, and neuropsychological dys-function (transcortical aphasia, memory disturbances). Bilateral occipital lobes infarction can give rise to cortical blindness and anosognosia [3,4].

Few more rare presentations have also been studied. Left large PCA stroke may cause visual agnosia which can be divided into apperceptive and associative, meanwhile right PCA stroke may cause prosopagnosia which is difficulty recognizing familiar faces. A pathological lesion in dominant occipital lobe and splenium of the corpus callosum leads to alexia often accompanied by right homonymous hemianopia. Infarction of the ventral occipital cortex with or without infracalcrine involvement causes achromatopsia which refers to difficulty perceiving colours [3,4].

Large left parietal or temporal lobe infarction causes aphasia and infarction of the hippocampus and parahippocampus leads to memory impairment.

Aggressive behaviours have been reported in PCA strokes as well. Other uncommon symptoms are hallucinations and palinopsia especially seen in lingual and fusiform gyri infarctions [4].

Syndromes related to PCA infarct include Balint syndrome and Anton syndrome [4]. Balint syndrome occurs when bilateral occipito-parietal border infarctions happens and it has a triad of optic ataxia, oculomotor apraxia and simultagnosia. Anton syndrome is when there is sudden onset bilateral occipital stroke, leading to cortical blindness.

We report a case of isolated right homonymous hemianopia in a patient with PCA territory infarct.

CASE REPORT

A 49-year-old man presented to the Ophthalmology clinic with the complaint of sudden bilateral blurring of vision for five days. He had diabetes mellitus, hypertension and dyslipidaemia however he defaulted medical follow up for 2 years. The patient noticed that he was unable to see words in the periphery of the pages he was reading. when driving he was unable to see vehicles on his his right side. He denied having floaters, flashes of light or scotoma. There was no metamorphopsia. His symptoms were persistent. He experienced no limb, body or facial weakness, neither was there head-ache, nausea and vomiting.

One week prior to the visual disturbances, the patient was admitted for myocardial infarction (Killip I) and was successfully thrombolysed upon admission.

Ocular examination revealed bilateral normal anterior and posterior segments with intraocular pressure of 14mmHg. His best corrected visual acuity in both eyes was 6/12 There was no relative afferent pupillary defect (RAPD). Other optic nerve function tests were normal too. Extraocular muscle movements were full. However, the confrontation test showed a right sided visual field defect, confirmed right homonymous hemianopia by Automated Humphrey Visual field analyser (Figure1). Power, sensation and coordination were normal. Cerebellar signs were negative. He did not have memory loss, neither cognitive nor behavioural changes. Higher cortical functions were normal too. Contrast-enhanced computed tomography of the brain was done in view of possible space-occupying lesion or cerebral infarction. As per suspicion, it revealed a left posterior cerebral artery infarct. The patient was then referred to the Medical team for the management of his co-morbidities and recent stroke.

DISCUSSION

Stroke related contralateral HH usually occurs together with other neurological manifestations especially when striate cortex, optic radiations, or lateral geniculate bodies (LGB) are involved [5]. Isolated HH is a rare incident with diagnostic difficulties. Interestingly, some patients are not aware of their visual field defects and are still able to drive around [6]. Few case reports have been published and a retrospective study concluded that usually HH is accompanied by other neurological signs and symptoms as mentioned earlier [5,6].

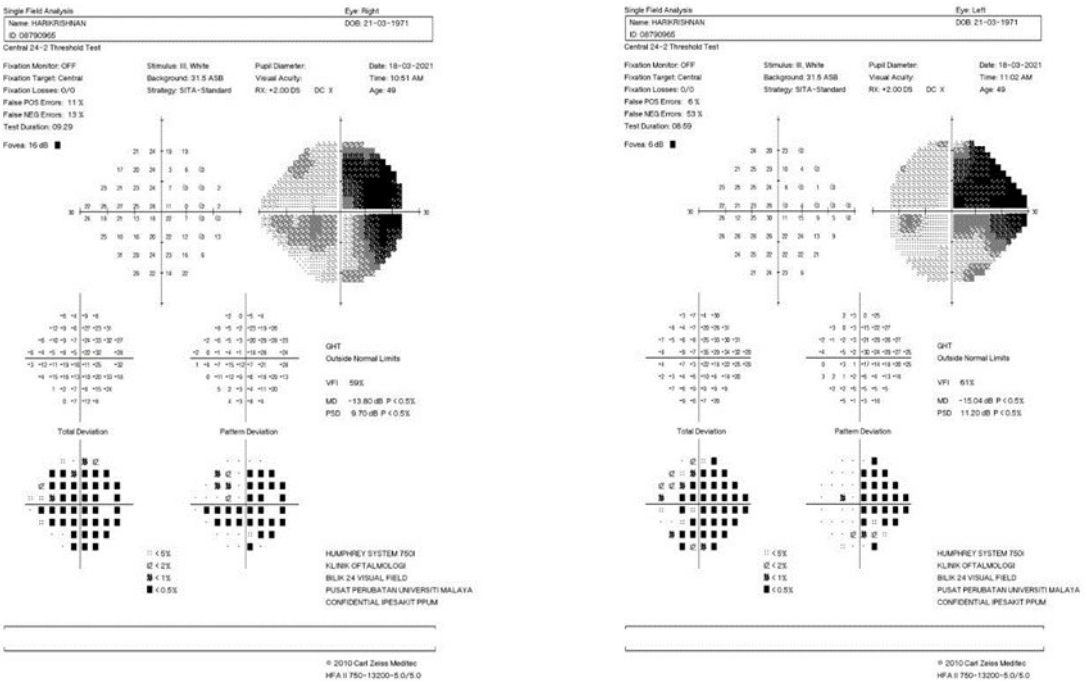

Figure 1: The Humphrey Visual Fields: incomplete right homonymous hemianopia.

Right HH in our patient most probably be caused by left posterior cerebral artery infarct. Further imaging, especially cerebral angiography or magnetic resonance angiography (MRA) would have been helpful to identify the specific branches and regions involved in the ischemic stroke, to justify the isolated HH. Isolated HH has been reported in association with lateral geniculate body infarction. Shibata K et al have reported a case with lateral posterior choroidal artery (LPChA) infarction which presented with isolated incomplete HH [5].

LPChA is a branch of PCA and assault to this artery usually leads to horizontal homonymous sectoranopia or wedge-shaped visual field defects. It arises from the initial part of the second segment of the PCA, immediately adjacent to the thalamogeniculate arteries. The choroid plexus of the lateral ventricle, pulvinar, posterior part of the dorsolateral nucleus, LGB, hippocampus, and mesial temporal lobe are supplied by this artery [6,7,8].

A study by Neau J-P (1996) found that isolated posterior choroidal artery infarction was estimated to be 1.5%, 10 patients out of 740 patients who had posterior circulation infarct [9]. Out of these, nine infarcts involved the LPChA and one was in both lateral and medial posterior choroidal artery. Isolated visual field defect without hemisensory and neuropsychological dysfunctions including transcortical aphasia and memory disturbances, has been associated with an LPChA infarct. Due to variable collaterals, symptoms and signs may vary (eg: incomplete HH) and sometimes be transient [5,6,7].

CONCLUSION

A variety of visual symptoms are associated with intracranial pathologies. However, on rare occasions visual symptoms may be the only manifested symptom mimicking a local pathology. Thus, detailed history, examination and selection of targeted investigation is essential to achieve the correct diagnosis. Anatomical variations must also be given important consideration as not to overlook certain unusual clinical presentations.

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