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CASE REPORT

Balint syndrome: an unusual triad

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The paper investigates a case of sudden loss of vision in a patient with recent history of blurred vision of right eye diagnosed with Central retinal artery occlusion (CRAO). The point of interest of this case report is that the clinical features are something different from those of a CRAO and revealed cardinal triad of simultanagnosia, optic ataxia, and oculomotor apraxia which are conclusive of a rare clinical entity known as Balint syndrome.

Keywords: Balint Syndrome, optic, ataxia

Introduction

A rare cause of visual impairment described by Rudolf Balint in 1909, characterized by triad of symptoms consisting of simultanagnosia, optic ataxia, and oculomotor ataxia (1). From literature review it was found that Balint syndrome exists mostly in the form of case reports (2). The exact incidence or prevalence of Balint syndrome is not known (3). The aim of this case report is to describe the symptom complex in a patient admitted with sudden loss of vision and to explain how it differs from Central Retinal artery occlusion co-existing in the same patient.

Objective

(1) To describe the clinical triad of Balint syndrome (4).

(2) To differentiate the scenario from Central Retinal Artery Occlusion.

(3) To aid in early diagnosis and prompt intervention, thereby increasing the quality of life of the patient.

Clinical scenario

A 67-year-old gentleman, a known case of recent cerebrovascular accident 2 weeks back, presented with a history of tachybrady syndrome and permanent pacemaker *in situ* and a recent history of blurring of vision in right eye 1 week back, which was evaluated and diagnosed as Central Retinal Artery Occlusion (CRAO) (5) (**Figure 1**) (6); however he subsequently developed a sudden visual impairment of both eyes within the previous 3 days. A detailed ophthalmological evaluation was done, which showed features of resolving CRAO right eye (**Figure 2**) (7). No ophthalmological abnormalities were detected in the left eye. Laboratory investigations were unremarkable except for mild alteration in Lipid profile. However, there was a disproportionate visual impairment in both eyes of the patient than what was expected in resolving CRAO. Normal Bilateral direct pupillary reflex and absence of Relative afferent pupillary defect ruled out the possibility of CRAO alone and the possibility of cortical involvement was suspected (8).

Bed side examination of the patient revealed the following findings:

1. Patient found it difficult to move his hand to a specific object with his eyes open, a phenomenon called optic ataxia (9). It was demonstrated as patient aiming for food outside the plate.
2. He had difficulty in identifying a whole familiar picture shown to him; however, he was able to identify certain parts within the same picture such as eyes, lips, eyebrows, etc., suggestive of simultanagnosia (9). Here

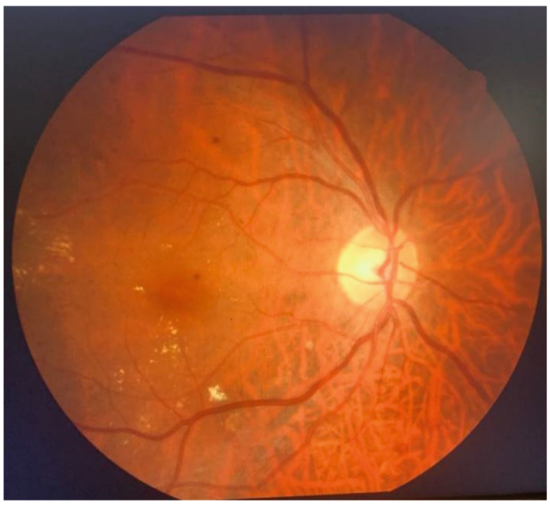

FIGURE 1 | (6): Initial fundus picture of the patient's right eye.

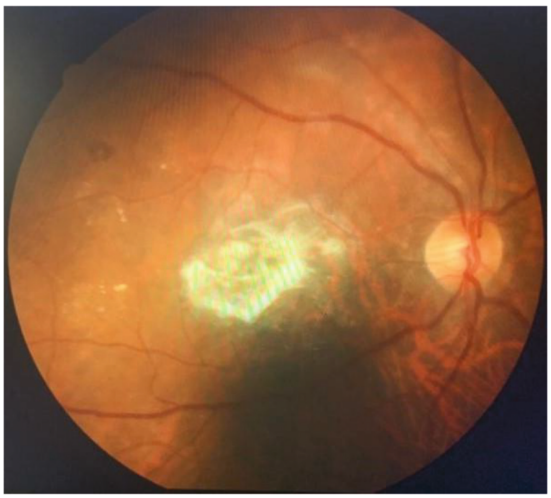

FIGURE 2 | (7): Fundus picture at admission (right eye).

the patient had difficulty in identifying his own family member; however, he could recognize his voice.

3. He had no control over his eye movement and he turned his head to follow objects coming from periphery, suggestive of oculomotor apraxia (9).

Etiology

Balint syndrome is usually caused by bilateral parietal and occipital lobe infarct resulting from bilateral posterior cerebral artery occlusion (10).

Rarely Balint syndrome is seen Alzheimer's disease, Creutzfeldt-Jakob syndrome, cortico bulbar degeneration, progressive multifocal encephalopathy, brain metastasis, trauma, following conditions was excluded with symptomatology and brain imaging (11).

Epidemiology

Exact details regarding incidence and prevalence of Balint syndrome are not available. Literature regarding Balint syndrome mostly exists in the form of case reports in adult population even though case reports in pediatric population do exist.

Pathophysiology

Balint syndrome is probably caused by systemic hypo perfusion of the bilateral parieto occipital region. Watershed infarcts are the most common cause. Bilateral dysfunction of the posterior portions of the cerebral hemisphere that is, embolization of rostral basilar artery causing bilateral tempero-occipital lobe infarct in the posterior cerebral artery (PCA) territory, could cause features of Balint syndrome. Alternatively, hypoperfusion of same region from unrecognized prolonged hypotension may also result in a similar clinical picture (12). Visual abnormalities can be more severe in either right or left visual field. Occasionally patients with Alzheimer's disease (13) Creutzfeldt Jakob disease have features of Balint syndrome but findings develop gradually and insidiously. Memory dysfunction and agitation can also be associated with later. When hypoperfusion is more severe, lesion can spread to the anterior border zone between the anterior and middle cerebral artery and even to the ventricle. Postmortem findings of patients with prosopagnosia (commonly associated with Balint syndrome) show both fusiform gyri destruction (14), suggesting that this structure functions as a visual association area for the recognition of specific faces (15).

Patients with visual agnosia usually have bilateral lesion (16) but this can also occur with unilateral left posterior parietal lesion. Some patients with Balint may also experience altitudinal neglect, suggesting bilateral parietal damage since the termination of the optic radiation is topographically arranged with lower retinal fibers terminating in the cortex below the calcarine fissure. Hemianopia with macular sparing is also seen in posterior cerebral artery occlusion (17).

Background and risk factors

1. Pre-existing cardiac arrythmia-tachy brady syndrome with permanent pacemaker *in situ* – strong predisposing factor for a cardio embolic event.
2. Longstanding diabetes mellitus
3. Recent history of central retinal artery occlusion (probable embolic event)

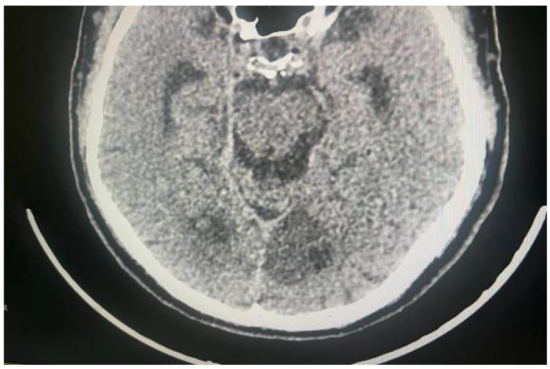

FIGURE 3 | (18): CT Brain on admission.

Confirmation

CT BRAIN with CT CEREBRAL ANGIOGRAPHY showed bilateral parieto occipital infarct with bilateral posterior cerebral artery (PCA) occlusion (**Figure 3**) (18).

Discussion and conclusion

Balint syndrome was clinically diagnosed after excluding the possibility of CRAO causing blindness even though the patient had CRAO within the last one week, further ophthalmological evaluation revealed features of CRAO resolved (19). In this patient the classic triad of optic ataxia, simultanagnosia, and ocular apraxia was evident along with landmark agnosia (patient also suffered difficulty in finding his way around).

Sudden onset and progression of symptoms ruled out possibility of other differential diagnoses such as Alzheimer's disease, Creutzfeldt-Jakob syndrome, cortico bulbar degeneration, and progressive multifocal encephalopathy, which are gradually progressive and are insidious in nature.

The etiology was confirmed using CT Cerebral angiography showing bilateral posterior cerebral artery occlusion with bilateral parieto occipital infarct (12).

Treatment and outcome

The patient was put on Dual Anti platelets- ECOSPIRIN 150 MG per day and CLOPIDOGREL 75 MG per day and high-dose statin- ATORVASTATIN 40MG per day, and was reviewed after 7 days and drugs modified as Single anti platelet (ECOSPIRIN 150MG per day) along with novel oral anticoagulant(DABIGATRAN 110MG twice daily) considering the background risk factors. Over the course there was an improvement in visual perception and quality of life. The patient was followed up for a period of 30 days after discharge with repeat CT angiography showing resolution of Bilateral posterior cerebral artery infarction. The patient and his family were counselled regarding the need for adherence to probable lifelong therapy with anticoagulant and antiplatelets.

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