Balint syndrome

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Abstract

We report a patient who presented with complaints of blindness following stroke and was subsequently diagnosed to have Balint syndrome

Key Words

Balint syndrome, optic ataxia, simultagnosia

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Introduction

Balint syndrome is a rare manifestation of visual and spatial difficulties due to the parietal lobe lesions. We describe one such patient who had bilateral parietal infarcts and briefly discuss the etiopathogenesis of this disabling condition.

Case Report

A 50-year-old laborer presented with blindness for past 3 months. He had sudden onset right hemiparesis 3 months ago, which resolved after 3 days. After 1-week, he developed difficulty in seeing distant objects and required assistance to do any manual activity. He was a hypertensive on regular medication for the past 10 years and was right handed and was not literate.

On examination, he had severe difficulty in reaching out to objects held by the examiner (optic ataxia) [Video 1]. He could not locate his footwear and he had to use his hands to put his footwear on [Video 2]. He could not do any visual task and always required help by his son to carry simple tasks like locating his plate. He could recognize a key and a spoon individually, but could identify only one of these objects when presented simultaneously (simultagnosia). He had normal color vision and stereognosis. His visual field could not be assessed due to oculomotor apraxia. Surprisingly, he could walk without bumping into objects. There were no anomia, finger agnosia, hemineglect, cranial nerve deficits, motor weakness or sensory loss.

Magnetic resonance imaging (MRI) brain showed bilateral parieto occipital infarcts with multiple periventricular white matter lesions [Figure 1].

Based on the triad of optic ataxia, oculomotor apraxia and simultagnosia and on the MRI findings, a diagnosis of Balint syndrome secondary to stroke was made.

Discussion

Balint first described psychic paralysis of gaze in 1909.[1] This rare and disabling condition consists of the triad of

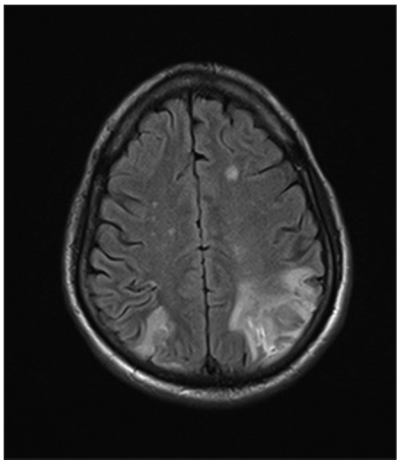

Figure 1: Magnetic resonance imaging - bilateral parietal infarcts

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optic ataxia, oculomotor apraxia and simultagnosia. This is classically seen due to bilateral posterior parietal lobe lesions.

Etiologies[2] include trauma, post-cardiac arrest, near drowning, stroke, metastasis,[3] eclampsia,[4] human immunodeficiency virus encephalitis, posterior reversible encephalopathy, posterior cortical atrophy[5] and Creuzfeldt-Jacob disease.

Mechanism

The superior parietal lobule (SPL) has been shown to be the main center for visual control of movements. SPL is located at the parieto occipital junction. Neurones in the SPL coordinate visual and hand movement required to reach an object based on visual stimulus. Thus damage to SPL has been postulated to be the reason for optic ataxia.[6]

Simultagnosia refers to inability to perceive more than one object at a time. Two types of simultagnosia have been described.[7] In the dorsal type, the patient cannot see more than one object at a time and often collides with other objects while walking. This is seen in bilateral parietal lesions. In the ventral type the patient sees multiple objects at the same time, but cannot visually integrate them together. Therefore, they do not collide against objects while walking. The ventral type is seen in left inferior occipito-temporal lobe lesions. Though, our patient had bilateral parietal infarcts, he could walk without bumping into obstacles.

Conclusion

Balint syndrome is a very disabling condition and patients require dedicated neurological rehabilitation. The exact neural pathways involved in this condition are still being evaluated due to the rarity of this disorder.

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