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## **Case Report**

# Balint syndrome (chronic visual-spatial disorder) presenting without known cause

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#### ARTICLE INFO

Article history:
Received 7 July 2018
Revised 18 August 2018
Accepted 26 August 2018
Available online 20 September 2018

Keywords:
Balint's syndrome
Visual-spatial disorder
Optic apraxia
Parieto-occipital atrophy
Simultanagnosia

#### ABSTRACT

Balint's syndrome is a rare disorder characterized by a triad of simultanagnosia, optic apraxia, and ocular apraxia. The syndrome manifests when there is an injury to the posterior parietal and occipital lobes and is often bilateral. Several causes of this syndrome were published in the literature, such as trauma, infarctions, infections, tumors, and preeclampsia. It can also be the presenting feature of several neurodegenerative disorders, such as atypical Alzheimer's disease. We report a case of a 62-year-old lady who presented with simultanagnosia, optic apraxia, and ocular ataxia which are the typical signs and symptoms of this syndrome. Neuropsychological evaluation revealed severe affection of the visual-spatial function with intact memory, language, and cognition. Brain imaging confirmed atrophy and decreased perfusion in the posterior parietal and occipital lobes. No underlying cause could be identified to explain the brain parenchymal atrophy. The follow-up neuropsychological assessment and brain imaging did not show any progression confirming the static course of the disease.

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#### Introduction

Balint's syndrome is a rare disease resulting in poor spatial awareness and eye movement in response to multiple visual stimuli. The disorder affects the parietal and occipital lobes causing altered perception, interpretation, and optic apraxia in response to complex visual scenes [1]. It was first described in 1909 by Reszo Balint, a Hungarian clinician, who noticed this complex neurologic disorder in one of his patients who presented with bilateral posterior parietal lobe lesions [2]. Since then, many researchers have studied this condition

Acknowledgments: none to declare.

Written informed consent for the case to be published (incl. images, case history and data) was obtained from the patient for publication of this case report, including accompanying images.

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https://doi.org/10.1016/j.radcr.2018.08.026

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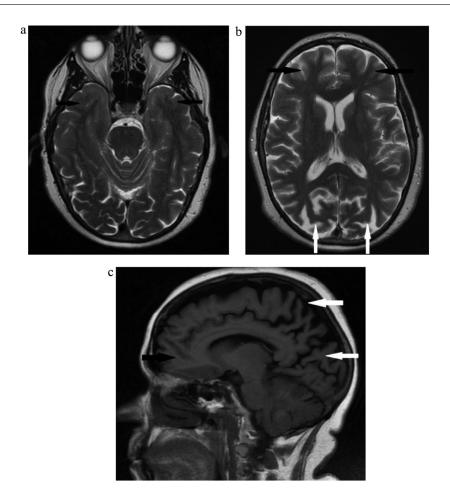


Fig. 1 – Axial T2 and sagittal T1-weighted MRI showing moderate bilateral occipital lobes and posterior parietal lobes atrophy (white arrows) with preserved temporal and frontal lobes volume (black arrows)

in order to shine a light at this rare visual-spatial disorder. Several causes have been proposed, with bilateral watershed infarcts being the most common cause of complete Balint's syndrome [3]. Other causes include traumatic brain injury, pre-eclampsia, embolic stroke, and infections. Progressive Balint's have also been described in patients with Alzheimer's disease [4]. No definitive imaging criteria are available to confirm this condition. However, the presence of bilateral parieto-occipital atrophy on CT or MRI imaging, as well as decreased cerebral perfusion on single photon emission computed tomography (SPECT) scans, is suggestive. Few case reports have been published in the literature describing the different causes and presentations of this disease [1,4-7]. We present an unusual case of chronic Balint's syndrome, with typical clinical and imaging findings of visual-spatial disorder, in which no apparent underlying cause could be identified.

#### **Case report**

Our patient is a 62-year-old female office worker, who was asymptomatic until 4 years prior to her presentation. She described seeing numbers and letters reversed. She was also un-

able to perform tasks that required complex visual orientation, such as pouring water from a kettle or going to a specific place despite clearly seeing the objects and knowing exactly where she wants to go. Also, she repeatedly fell due to poor visual-spatial awareness. She had no history of memory or language deficits, head trauma, seizures or stroke and there was no relevant family history. Initial assessment, using the repeatable battery for the assessment of neuropsychological status version A, showed intact immediate memory, language function (receptive and expressive speech), and praxis. Attention and concentration were affected at the part which required visual ability in order to transfer visual symbols and numbers. The visual-spatial function was extremely low. She was unable to copy complex figure drawing and showed no organization in the performance of complex visual-motor functions. She had difficulty forming movements to visual stimuli and was unable to identify more than one object at a time in a series of events (simultanagnosia). As a result of skipping letters and words, and the inability to scan end to end, she demonstrated great difficulty in reading. Unable to continue working, she developed depression and anxiety after a few months.

Initial brain MRI showed bilateral posterior parietal and occipital atrophy, right slightly more than left (Fig. 1). There was

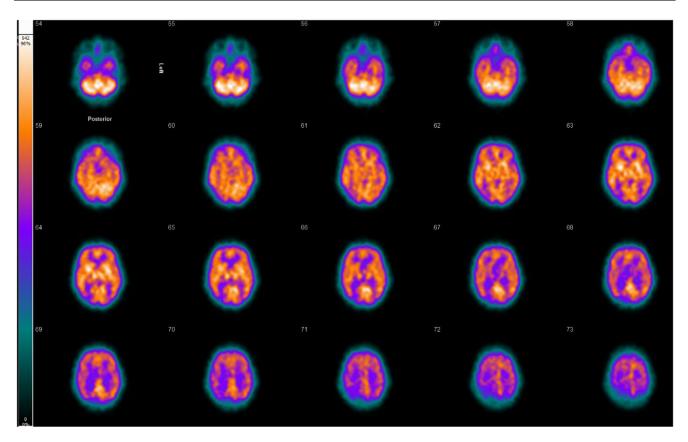


Fig. 2 – Nuclear medicine SPECT perfusion scan showing severely reduced perfusion involving bilateral occipital and posterior parietal lobes with normal perfusion of the frontal and temporal lobes.

no underlying parenchymal signal change or other findings to suggest damage from vascular disease/trauma. The temporal and frontal lobe volume was preserved.

Nuclear medicine perfusion SPECT showed moderate to marked decreased perfusion involving the bilateral posterior parietal and adjacent occipital lobes more so on the right (Fig. 2). The temporal and frontal lobe perfusion was normal with no background features to suggest Alzheimer's disease.

The patient was diagnosed to have Balint's syndrome and therefore was not given any medications other than a selective serotonin-norepinephrine reuptake inhibitor within the initial 2 months to relieve her anxiety and depression. She was also offered a few sessions of physiotherapy/rehabilitation to help her improve vision-movement orientation. Upon follow-up, the repeated clinical assessment after 4 years using repeatable battery for the assessment of neuropsychological status showed no significant changes as well as the brain MRI which showed no progression in the degree of atrophy. These findings suggest a static course of the disease.

### Discussion

Balint's syndrome is an acquired disability that affects the perception of the visual field leading to the inability to perceive and recognize several objects at the same time (simultanag-

nosia), impaired targeting objects under vision guidance (optic apraxia), and loss of control over purposeful eye movement (ocular apraxia) [8]. The complete form of the disease manifests when there is involvement of bilateral posterolateral occipital lobes (visual association area), the parieto-occipital junction, and posterior aspects of both parietal lobes [9].

The most common cause of the acute form of this syndrome is watershed infarctions from sudden hypotension [3]. Other conditions that could cause structural damage bilaterally such as embolic strokes, trauma, pre-eclampsia, and infections have also been reported.

Balint's syndrome can also be caused by neurodegenerative disorders particularly posterior cortical atrophy (PCA) and atypical Alzheimer's disease. PCA is a progressive dementia presenting with a predominant visual-spatial affection which ultimately affects memory and results in global dementia. It was first thought to be a separate entity from atypical Alzheimer's, but it is now known that PCA can be caused by several neurodegenerative disorders particularly atypical Alzheimer's [9].

Our patient presented with signs and symptoms that fulfilled all the three aspects of Balint's syndrome. The results of her imaging also confirmed the parenchymal atrophy. The unusual part of our case is that there was no evident underlying parenchymal injury that would explain the atrophy. Moreover, the patient had intact memory and cognition, and the course of the disease was nonprogressive making it less likely to be due to a neurodegenerative disorder.

There are many cases published in the literature of this syndrome highlighting the varied etiologies [1,4–7]. To our knowledge, this is the only report of a case of Balint's syndrome in which no apparent cause could be identified.

#### Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2018.08.026.

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