



Bálint syndrome caused by bilateral medial occipital infarcts

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Received: 18 April 2018 / Accepted: 3 July 2018
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A 78-year-old woman known for treated hypertension presented a 3-day history of spatial disorientation, attention deficit, word-finding difficulties, and diffuse visual impairment. Examination showed mild anomia, verbal anterograde amnesia, partial right homonymous hemianopsia (with preservation of the right inferior quadrant for the right eye), and Bálint syndrome. This consisted of optic ataxia for both hands in her preserved visual field, ocular apraxia with difficulties to shift gaze to targets in her intact visual field, visual simultagnosia with inability to detect more than one out of two or more points simultaneously presented to her visual field during a few seconds. However, she was able to describe single parts of pictures and letters of a word. Additional visual phenomena were palinopsia and “vanishing” objects perception, i.e., the persistence of visual stimuli perception after they left the visual field and, conversely, the sudden perceptible loss of an object which yet remained in the visual field. Brain MRI showed acute ischemic lesions in the left posterior cerebral artery territory, including medial temporal and occipital lobes, but no parietal lesion. In addition, it revealed a previously unknown chronic right medial occipital lobe infarct and diffuse leukoaraiosis (Fig. 1).

While aphasia rapidly resolved, visuospatial deficits only partially improved. The three features of Bálint syndrome persisted for 10 weeks, thus severely impairing the patient’s autonomy. She returned to her home with the help of home care after intensive rehabilitation.

Bálint syndrome was first described by the Reszö Bálint in 1909 as a combination of three complex visual symptoms [1]:

1. Spatial disorder of attention, later called dorsal visual simultagnosia, inducing an inability to simultaneously perceive multiple stimuli in the preserved visual field. It has been attributed to an extreme shrinkage of the attentional visual field, also called “spatial window of attention” [2]. Other authors attribute simultagnosia to an inability of the brain to build a visual spatial frame in which it can compute multiple visual informations, i.e., to “bind” them [3]. Typical lesions are bilateral parietal or parieto-occipital. The present case is exceptional by the absence of parietal damage.
2. Optic ataxia describes the difficulty to reach a target under visual guidance in the absence of any motor, proprioceptive, or primary visual deficits. It can occur independently from simultagnosia and ocular apraxia, with unilateral lesions, and is thought to rely on two potentially impaired pathways: a visual proprioceptive deficit or a visual attentional deficit [4]. Both imply the dorsal visual pathway within the posterior parietal cortex.
3. Psychic paralysis of gaze, or acquired ocular apraxia, describes the difficulty to shift gaze from one target to another—i.e., to visually “grasp” objects in the visual field. It has been described in lesions involving the posterior or frontal eye field. When occurring in Bálint syndrome, a decrease of visual attentional flexibility has been proposed as an explanation. This mechanism may be shared by simultagnosia and ocular apraxia [5].

In addition, many visual phenomena such as palinopsia or *vanishing objects* have been reported in cases of Bálint syndrome [5].

Etiologies are very diverse— ischemic, degenerative, inflammatory, tumoral, or traumatic [6]. This syndrome has been classically described in patients with bilateral dorsolateral parietal lesions, and sometimes with other localizations; bilateral involvement appears to be necessary.

Our patient suffered from a chronic infarct in the right medial occipital lobe. It was not associated with any clinical deficit before the current episode and was revealed by the MRI. Still,

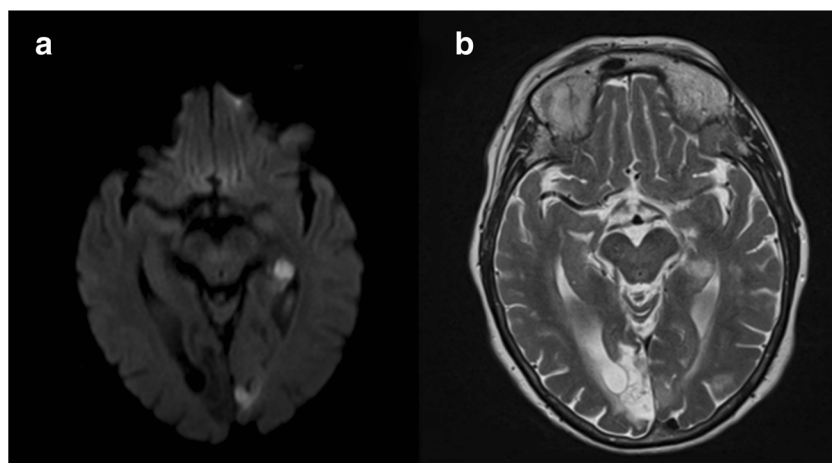
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Fig. 1 **a** Axial diffusion-weighted MRI showing acute ischemia of the left medial temporal and occipital lobes. **b** Axial T2-weighted MRI showing a chronic infarct of the right medial occipital lobe and diffuse leukoaraiosis



its localization is compatible with disconnection of the superior longitudinal fasciculus, a component of the visual attention network potentially implicated in Bálint syndrome. Moreover, the MRI revealed diffuse leukoaraiosis. Thus, our patient potentially already had asymptomatic disruption of attentional pathways before the present episode, and we can hypothesize that, since the first silent stroke, her left hemisphere was assuming the main visual attentional functions through a neural compensation strategy involving a shift in hemispheric lateralization. Then, the second (left medial occipito-temporal) ischemia would have impaired her compensation network and resulted in Bálint syndrome. To support our hypothesis, we can cite the recent works about the impact of cognitive reserves on the symptomatology and recovery of stroke, and more specifically on the attentional deficits [7].

An alternative (but not exclusive) hypothesis [5] posits that with two lesions happening at different timepoints, the first causing left hemineglect and the second right hemianopsia, the combination of both results in Bálint syndrome in the remaining left visual field. In our case, the recent stroke caused a partial right hemianopsia, and it is difficult to predict if her right visual field would also have suffered from severe attentional deficit had it been preserved.

Rehabilitation of Bálint syndrome consists of functional adaptation programs to develop compensatory strategies and visuoperceptive trainings to reduce deficits [8]. Effects of

rehabilitation versus the natural course of the condition are not established.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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