Dear Editor,

A 50-year-old male was evaluated in our neurology clinic during a follow-up visit due to previous ischemic stroke. An interrogation of his medical history revealed that the patient had been under follow-up due to rheumatic heart disease and related aortic valve insufficiency since childhood. Due to the progression of ventricular dysfunction, a prosthetic aortic valve replacement was performed nine years ago. After this surgery, when the influence of anesthesia was over, a significant weakness in his left upper extremity was noticed. More devastatingly, he was unable to visually perceive any colored or colorless objects. However, the symptoms improved in the following one-year period and the patient was able to perceive the objects, separately. At admission to our clinic, his direct and indirect pupillary light reflexes were evaluated as normal. In addition, the visual acuity was found to be bilaterally normal. However, the patient was unable to make pursuit eye movements, as well as saccadic eye movements, which were totally absent in the left direction and moderately disturbed in right direction. Moreover, during the oculocephalic test, rotation of the eyes to the opposite direction of the head movements were observed. Ability of visual-guided reaching was defective. This was compatible with optic ataxia. In addition, the patient was able to see and read a letter but could not combine the letters to perceive a word, which was compatible with simultanagnosia. The patient could distinguish the objects separately; however, he had difficulty when they were visualized side-by-side or back-to-back (Video 1). Cranial tomography showed bilateral parietooccipital hypodensity, which was prominent in the left hemisphere (Figure 1). The clinical findings of severe oculomotor apraxia, optic ataxia, and simultanagnosia led to the diagnosis of Balint syndrome due to ischemic stroke.

In this letter, a rare case of Balint syndrome is presented through detailed video images of the neurologic examination. Balint syndrome, which was first described by Balint in 1909, can be defined as a clinical presentation including defects consisting of psychic paralysis of gaze, spatial disorder of attention, and optic ataxia (1). In the literature, lesions mainly affecting the bilateral occipitoparietal lobes have been attributed to causing Balint syndrome (1,2). In the author’s opinion, this report gives valuable findings, presenting a smart example of this rare syndrome, which is rarely illustrated in the literature. At first clinical presentation, the patient experienced total blindness, which gradually improved during the following one-year period. In a similar report by Espay and Allen (3), a patient with Balint syndrome who presented with total blindness was described. In that report, the visual acuity of the patient improved within the first 24 hours of clinical symptomatology, which was explained in the setting of posterior reversible encephalopathy syndrome. However, the clinical triad of Balint syndrome was apparent at the follow-up evaluation. In our patient, recovery of total blindness took a much longer time.

Keywords: Balint syndrome, stroke, pathophysiology

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Received/Geliş Tarihi: 29.03.2017 Accepted/Kabul Tarihi: 01.07.2017
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Future reports including long-term follow-up of these patients may provide substantial data for the underlying pathways/mechanisms of these manifestations, as well as their compensatory networks.

**Ethics**

**Peer-review:** Internally peer-reviewed.

**Financial Disclosure:** The author declared that this study received no financial support.

**References**