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Case Report

Intracerebral Hemorrhage Manifesting as Optic Aphasia: A Case Report

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Abstract

Optic aphasia is a rare neurological disorder that affects the visual-semantic ability of patients with normal vision and is caused by a lesion in the left occipital lobe. The signs and symptoms of optic aphasia are similar to those of associative visual agnosia, where patients have difficulty recognizing objects both in shape and function, resulting in challenges performing daily tasks. The transformation to optic aphasia or associative visual agnosia is closely related to the degree of damage to the corpus callosum, with some studies hypothetically suggesting that complete damage to the corpus callosum leads to optic aphasia, whereas incomplete damage causes associative visual agnosia. We present a case of a 60-year-old man with a history of intracerebral hemorrhage in the left occipitotemporoparietal lobe. The patient complained of intermittent episodes of painless, blurry vision. Upon examination, we observed that the patient was unable to read the Snellen chart, although he could draw the letter. Furthermore, we discovered that the patient had difficulty naming objects and instruments, even though he was able to express their shape and function through gestures and mimicry. The signs and symptoms of the patient, along with the result of the multi-slice non-contrast CT scan, suggest that he had optic aphasia rather than associative visual agnosia. A comprehensive neuropsychological and aphasia examination needs to be performed to further assess the condition of our patient and establish the diagnosis.

Keywords: Intracerebral hemorrhage, optic aphasia, visual agnosia.

Introduction

Optic aphasia is a rare neurological disorder first described by Freud in 1889 in which patients cannot name visually presented objects with the ability to identify them by sight and name those objects by other sensory means (1). It is different from agnosia, where there is a complete inability to recognize objects by visual, verbal, and tactile stimulus (2). Patients with optic aphasia commonly have good visual perception but have difficulties pairing visual stimuli with their corresponding names or verbal labels. They may replicate the object's sound or function through pantomimes, something that is absent in patients with visual agnosia (2).

The underlying cause of optic aphasia is damage to the posterior region of the brain, which is vital for visual processing. Traditionally, it is thought to be the result of left occipital lobe damage, depriving left visual canters of direct input and interrupting interhemispheric connection by the corpus callosum, preventing visual information from reaching the semantic area in the left hemisphere (3).

The symptoms of both optic aphasia and visual agnosia are usually hard to differentiate since they both share simi-

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lar pathological mechanisms. A complete neuropsychological assessment should be conducted to evaluate both visual and verbal functions, allowing for differentiation between patients with optic aphasia and those with visual agnosia (4).

Case Report

A 60-year-old right-handed man came to the ophthalmologist clinic with the chief complaint of intermittent episodes of painless blurry vision on both eyes for the last 6 months. The symptom appeared unpredictably without any discernible pattern, and he did not notice any aggravating factors. He is highly myopic and usually uses −8.00 D contact lenses on both eyes. Prior to this, he had no history of any ocular diseases and had never undergone any ocular surgery.

He had a history of decompressive craniotomy with hematoma evacuation performed 2 years ago due to a spontaneous intracerebral hemorrhage in the left occipito temporo parietal lobe, which resulted in weakness in his right extremities and gait problems. He routinely used insulin injections and metformin to manage his type II diabetes mellitus, in addition to taking Tanapress® and Bisoprolol for his hypertension.

On initial examination, the patient was fully conscious with slight difficulty in speech and an abnormally slow gait. He spoke in incomplete, non-fluid sentences but was able to express his symptoms and respond accordingly. His vital signs were within the normal limits. No blood glucose examination was performed.

No abnormalities were found during the external eye examination, as both eyes demonstrated a normal gaze and unrestricted eye movements. The pupils displayed normal light reflexes. The conjunctiva, cornea, anterior chamber, and lens appeared normal on slit-lamp examination. Retinal fundoscopy revealed mild vitreous opacities in both eyes, with no signs of infection or abnormalities in the retina or optic nerve head.

On a visual acuity examination, the patient had difficulty identifying individual letters but was able to reproduce the shapes of the letters through writing. The best-corrected visual acuity of both eyes was 6/6 (equivalent to 20/20) with the use of a −10.0 spherical lens for both eyes. Due to the presence of apparent agnosia, further history was obtained. The patient demonstrated the ability to perform basic daily tasks, scoring 90/100 on the Barthel Index (requiring slight assistance with mobility and stairs). He was capable of using common tools (such as a television remote control, a house key, etc.), although he experienced difficulty recalling the names of these tools.

No neuropsychological tests were performed to assess the visual and verbal disturbances due to the unavailability of the required tools at the ophthalmologist's clinic. Supporting ophthalmologic examinations such as optical coherence tomography, visual evoked potentials, and electroretinography were not available at our hospital during that time. Referring the patient to another hospital was necessary for these specific tests, which the patient and his family refused at the time. We prescribed Vitrolenta® eye drops for the patient's vitreous opacities, to be administered twice daily in both eyes. We referred the patient to the neurology department for further examination of the agnosia symptoms, but the patient declined for undisclosed reasons.

Discussion

Inability to recognize visual objects is encompassed under the umbrella term of visual agnosia, a rare disorder in which a patient cannot identify objects despite having normal visual function. Visual agnosia is further classified into two subtypes: apperceptive visual agnosia, where abnormalities in visual perception and discriminative processes result in the patient's inability to apprehend, draw, or copy figures, even though their knowledge of the object remains intact; and associative visual agnosia, in which the patient is unable to link visual stimulus to prior experience, hindering their ability to recognize the stimulus. The patient correctly perceives the form when tested with verbal or tactile information but cannot identify the objects (5).

On the other hand, optic aphasia, another rare neuropsychological disorder first described by Freud in 1889, is an anomaly in which the patient has difficulty naming objects presented visually but is able to reproduce the form and function of the object through gestures or mimicking. Optic aphasia is often associated with agnosia, with a difference in the capacity to copy shapes, which is intact in optic aphasia. The patient can recognize objects through other forms of stimulus, whose function is impaired in associative agnosia, and has no compromise in their daily lives, with the patient being able to use instruments in the correct manner, albeit not knowing the names of those objects. Optic aphasia can also be distinguished from anomia by the incapability to name objects based on definition, tactile, and auditory means (6).

In optic aphasia, there is minimal disturbance of recognition, and a purely naming defect predominates, whereas in associative visual agnosia, the problem of recognition is prevalent. Optic aphasia patients are capable of identifying the shape of the object, demonstrating a preserved structural representation, which is not present in associative visual agnosia (4-6).

The location of the lesion also contributes to the symptoms demonstrated in patients with optic aphasia and visual agnosia. Both disorders are associated with left unilateral lesions, while bilateral lesions cause a classical form of visual associative agnosia with greater severity than unilateral ones.

Unilateral lesions produce symptoms of impairment of recognition and semantic knowledge, visual anomia, and naming errors, with these symptoms overlapping in both optic aphasia and visual agnosia (2). Lesions located in the left occipital and posterior temporal areas have been associated with visual associative agnosia, whereas involvement of the splenium at the corpus callosum is observed in patients with optic aphasia, showing a difference in degree of callosal disconnection and semantic identification by the non-dominant hemisphere between visual agnosia and optic aphasia (4). Based on the findings of multiple case studies, it is observed that if the damaged left inferior temporo-occipital area is disconnected from the right hemisphere's visual input, the right hemisphere may process stimuli similarly to split-brain patients, leading to optic aphasia. On the other hand, if some visual information can access the damaged left inferior temporo-occipital area, more visual processing occurs in the damaged left hemisphere, resulting in associative visual agnosia (2,4).

Our patient displayed normal vision function, apart from high-grade myopia and mild vitreous opacities. He had difficulty reading the letters on the Snellen chart but was able to draw the letters on paper. In addition, he experienced challenges in naming certain objects, although he could convey their shapes through gestures. While he could use common instruments in his daily activities, he had difficulty recalling the names of those instruments.

During a simple examination at the ophthalmologist clinic, he could accurately portray objects through mimes when given verbal instructions, indicating intact recognition but compromised semantic function. Along with speech difficulties, our patient exhibited signs and symptoms consistent with optic aphasia rather than associative visual agnosia or anomia.

We obtained the multi-slice non-contrast CT scan of a previous incidence of cerebrovascular accident in October 2021, which demonstrated intracerebral hemorrhage in the left occipito temporo parietal lobe, reaching the splenium of the corpus callosum, and intraventricular hemorrhage in the left 3rd and lateral ventricles. A decompressive craniotomy and evacuation of the hematoma were performed with no surgery complications and a good result, achieving full consciousness on the 2nd post-operative day. A second CT scan was done 6 days after surgery, showing improvement. The patient then proceeds to attend a physiotherapy session and reach full motoric function and grade 5 muscle strength on four extremities (Figures 1 and 2).

Another CT scan was performed in March 2023, revealing infarcts on the medial side of the left and right temporal lobes, as well as the lateral side of the left temporo-occipital lobe and left hydrocephalus. No invasive treatment was planned, and the patient underwent monthly check-ups with both the neurologist and internist for his condition (Fig. 3).

The brain lesion in the left temporal and occipital lobes bears a similar resemblance to previous case reports of optic aphasia and visual agnosia (2-4,7). Unilateral involvement, along with symptoms of slight speech impairment, further marks this patient's case as optic aphasia. A standardized test for memory, cognition, and aphasia, such as the Mini-Mental State Examination, Montreal Cognitive Assessment, Alzheimer's Disease Assessment Scale, Boston Naming Test, and Western Aphasia Battery, needs to be performed to further differentiate this patient's optic aphasia from visual agnosia or other similar disorders (2,4,5).

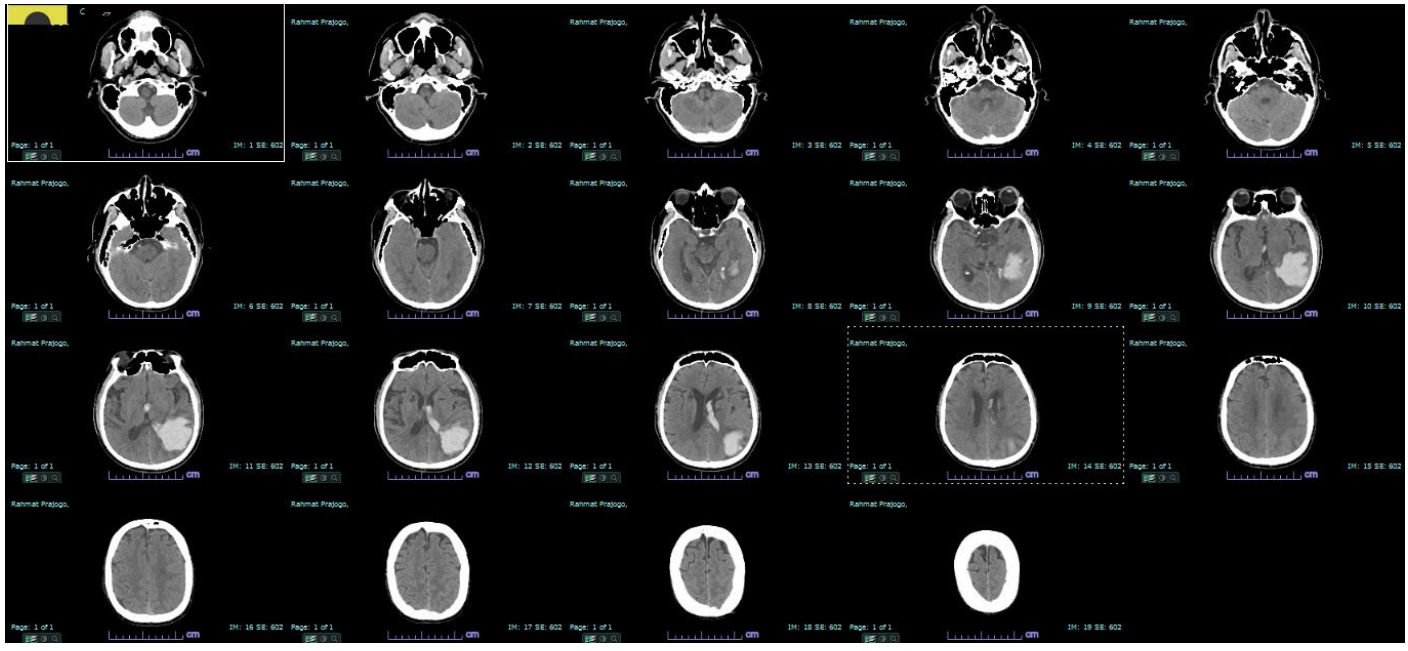

Figure 1. Computerized-Tomography scan of pre-decompressive craniotomy and hematoma evacuation.

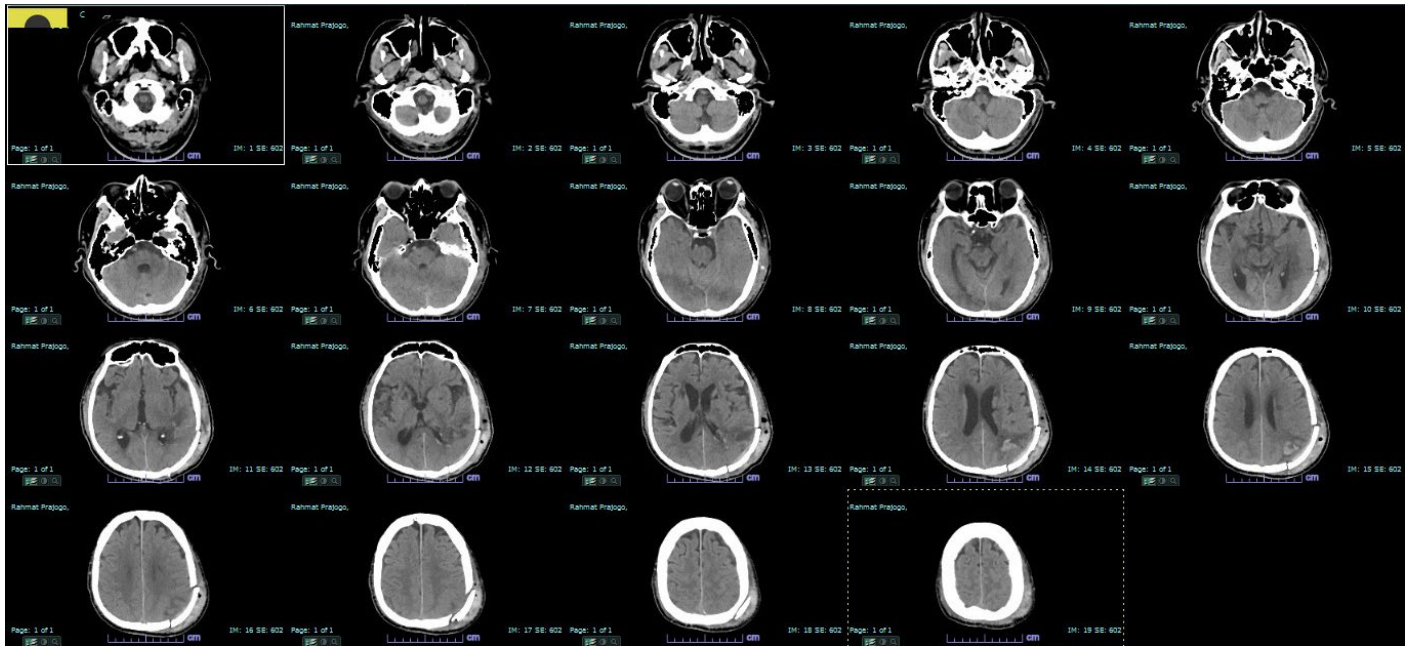

Figure 2. Brain CT scan 6 days post-operative, showing a decrease in intracerebral hemorrhage.

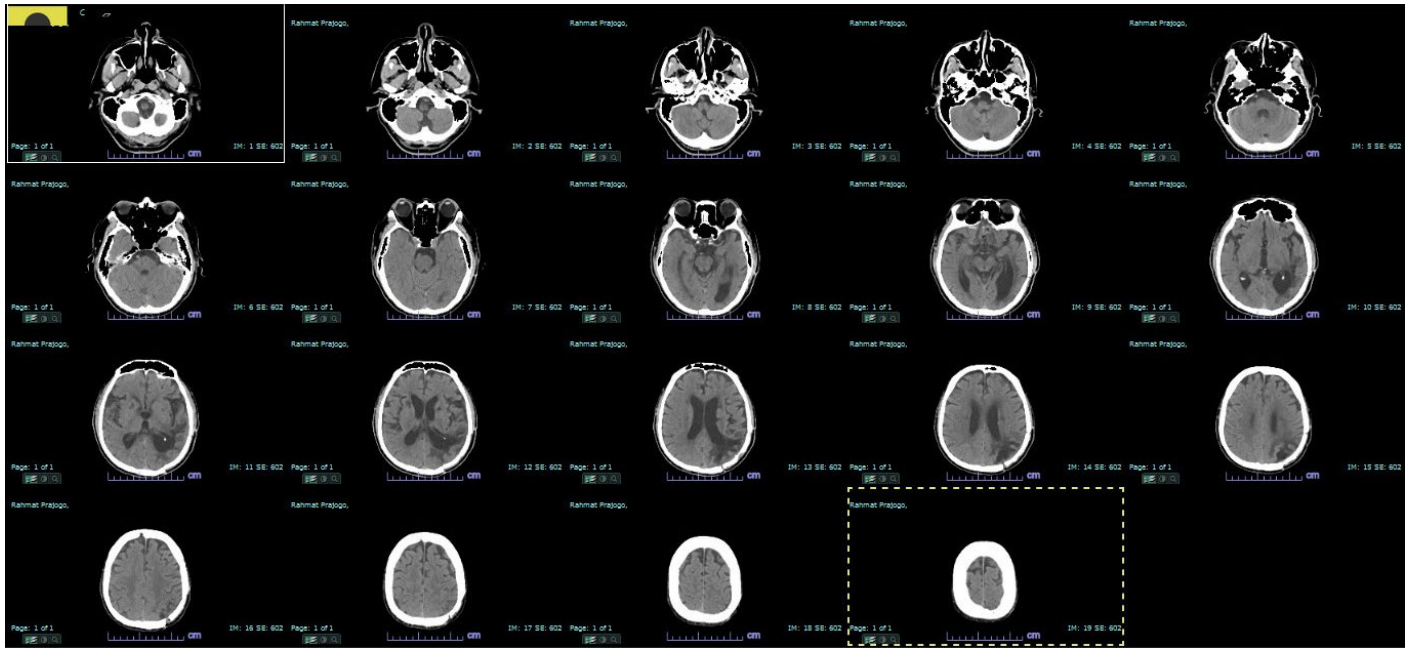

Figure 3. Brain CT scan in March 2023 showing signs of multiple infraction lesions at both side of temporal lobe and left temporooccipital lobe.

Conclusion

Optic aphasia is a rare disorder that shares similar signs and symptoms with visual associative agnosia, although both syndromes possess different pathophysiology. While optic aphasia demonstrates impairment in semantic function, visual associative agnosia shows signs of dysfunction in the recognition canter, further limiting the ability to perform basic daily activities. In our case, the brain condition closely aligns with previous reports of optic aphasia, indicating an intracerebral hemorrhage in the left temporo-occipital lobe with damage to the splenium of the corpus callosum. Our hypothesis suggests that the damage to the splenium results in callosal disconnection, making the right hemisphere the exclusive processor of visual input. This condition is responsible for the semantic problem experienced by the patient.

Conversely, if the callosal system remains intact, some visual input may still be processed by the impaired left occipital lobe, leading to recognition difficulties in patients with associative visual agnosia. The signs and symptoms of the patient, along with the results of the CT scan, are consistent with optic aphasia rather than associative visual agnosia. A comprehensive neuropsychological and aphasia examination needs to be performed to further assess the condition of our patient.

Disclosures

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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