Visual anosognosia on a patient due to bilateral ischemic occipital lobe strokes

Anosognosia visual en una paciente con lesión isquémica occipital bilateral

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Abstract

A case report of a 60 year old female brought by her family to medical consultation because they noticed decreased visual function 2 days prior to medical evaluation. The patient denies having altered visual function. Her visual acuity was of hand motion on both eyes with no ocular findings to justify this vision. The patient was told she needed a Goldmann perimetry test and brain magnetic resonance imaging and to be evaluated in 24 hours, nevertheless, the patient returns one week later with a visual capacity of 20/250 in both eyes. By that time, the Goldmann perimetry shows complete left homonymous hemianopsia and peripheral reduction of temporal 30° on the right eye and nasal 10° on the left eye with partial preservation of fixation point. Magnetic resonance imaging shows right occipito-temporal ischemic lesion with bilateral small vessel occipital ischemia. Treatment is indicated by neuro-ophthalmology service and the patients were diagnosed with visual anosognosia associated to occipital ischemia, with gradual improvement of visual function by reperfusion of central nervous system.

Key words: Occipital ischemia. Visual anosognosia. Anton’s Syndrome.

Resumen

Presentación de un caso clínico que trata de una paciente de 60 años de edad que acude a la consulta oftalmológica por disminución de la función visual observada por familiares dos días previos a la consulta; sin embargo, la paciente niega tener alteración de la función visual. A la exploración se encuentra con agudeza visual de movimiento de manos en ambos ojos, sin alteraciones en las estructuras oculares que lo justifiquen. Se recomienda nueva valoración en 24 horas y se solicitan estudios complementarios; sin embargo, la paciente acude una semana después ya con capacidad visual de 20/250 en ambos ojos y con campos visuales manuales, los cuales muestran hemianopsia homónima izquierda y reducción de 30° periféricos temporales en el ojo derecho y 10° periféricos nasales en el ojo izquierdo, con conservación parcial del punto de fijación y resonancia magnética de cráneo que muestra lesión isquémica occipitotemporal derecha con isquemia de pequeño vaso occipital bilateral. Se indica tratamiento a cargo de neurooftalmología. Se integra diagnóstico de anosognosia visual asociado a daño isquémico occipital, con mejoría paulatina de la función visual por probable reperfusión del sistema nervioso central.

Palabras clave: Isquemia occipital. Anosognosia visual. Síndrome de Anton.

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Introduction

There are causes of visual loss unrelated to ocular pathologies that are not evident in a routine ophthalmological exploration. In such cases, it is important to be aware of the possible regions of the visual pathway that can be compromised, as well as the different manifestations that can be found in the physical examination to suspect a possible area of affections and, thus, establish a more accurate suspected diagnosis.

When there is visual loss due to visual pathway alterations, usually there are other manifestations that may not be evident until the patient is interviewed. They can include specific changes in the visual field, alterations in the perception of movements, variations in the ability to observe several objects simultaneously, inability to acknowledge the visual loss, inability to recognize or understand visual stimuli, among others.

Approximately in 70% of cases, the posterior cerebral arteries originate from the basilar trunk, in 20-25% one of them originates from the carotid artery and the other one from the basilar trunk, and in the remaining cases both arise from the carotid system. This anatomical variability justifies the variable clinical expressions that this vascular syndrome shows. Atheromatous occlusion of the posterior cerebral artery is rare, as is complete infarction. Distal branches are more frequently affected, especially the calcarine artery. The most frequent mechanism is embolic. Occlusion of temporal-occipital branches determines visual alterations and gnosia disorders. Homonymous hemianopia occurs in a third of cases of posterior cerebral artery occlusion, due to calcarine/striatum cortex and/or geniculocalcarine fibers involvement. In lesions of the dominant hemisphere, color dysnomia and amnesic aphasia are more frequent than dyslexia without agraphia. Visual neglige can be the clinical manifestation of large right temporoparietal infarctions. It is not uncommon to observe visual perseverations associated with hemianopia. Amnesia is associated with medial lesions of the left temporal lobe, often including the hippocampus and the adjacent white matter. Transcortical aphasia can occur in large infarcts of the left posterior temporal-occipital region.

An aspect of extraordinary interest from a clinical point of view, is the bilateral cortical syndromes that may occur as a result of complete occlusion of the upper portion of the basilar trunk, especially if the posterior communicating arteries are non-functioning. Occipital bilateral lesions cause cortical blindness or bilateral homonymous hemianopia associated with visual hallucinations and anosognosia, amnesia and delusional disorder.

Balint syndrome manifests with spatial attention alteration with simultanagnosia, optic ataxia and difficulty to execute movements under visual control, as well as deficit when generating eye movements towards objectives located within the visual field, similar to an oculomotor apraxia. Reflex ocular movements to vestibular, auditory and unexpected visual stimuli are preserved. This symptomatology is the result of calcarine and bilateral parietal-occipital infarctions. Anton syndrome refers to cortical blindness and anosognosia without dementia or confusion.

Prosopagnosia is a disorder characterized by the inability to recognize faces. It results from bilateral lesions, but the participation of the right occipital hemisphere seems essential for its development.

Inverted or tilted perception disorder. The illusion of inversion of visual images consists of an altered perception of objects’ situation that is produced by complete rotations in the different spatial planes. It is more frequent in the vertical plane and especially in cases of vertebrobasilar ischemia. The pathophysiological mechanism is due to posterior parietal cortex dysfunction.

Several patients who suffer a stroke present visual-spatial alterations with difficulty to perceive drawings of overlapping objects and to correctly identify the hands of the clock. These alterations are due to parietal-occipital damage. Pure alexia is the inability to read, with writing and dictate preserved. Copying is also altered. The topography of the injury is usually paraventricular to the occipital horn of the left lateral ventricle. Visual agnosia is produced by bilateral injuries of the subcalcarine region. Lesions of the inferior occipital lobe or occipital-temporal lesions affect structures of the ventral region of the visual association cortex.

Clinical case

This is a 60-year-old patient from Mexico City, dedicated to housework, who is taken to the emergency ophthalmology consultation by her son, who observed that the patient collided with objects when walking, starting two days before the assessment. The patient denies visual symptoms. When interviewing her directly, she refers having good vision and even tries, unsuccessfully, to describe the doctor’s clothes to show her visual capacity. Also, the patient looks convinced that she described the clothes correctly.
In her family history, she refers parents with a diagnosis of diabetes mellitus. She also refers a 20-year history of hypertension treated with an unknown dose of losartan.

Neurological examination showed conserved mental functions with correct location in space, time and person. Cranial nerves without alterations, and normal motor and sensory systems.

Ophthalmologic examination showed right eye with hands motion vision, intraocular pressure of 12 mmHg, normal eyelids and annexes, normal conjunctiva, clear cornea, reactive pupil, with normal pupillary light reflexes, lens with NO2 NC2 opacity, applied retina with arteriovenous crossings and decrease in arteriolar caliber, macula with normal appearance and optic disc with a 5/10 excavation (Fig. 1). Left eye with hands motion vision, intraocular pressure of 13 mmHg, normal eyelids and annexes, normal conjunctiva, clear cornea, reactive pupil, with normal pupillary light reflexes, lens with NO2 NC2 opacity, applied retina with arteriovenous crossings and decrease in arteriolar caliber, macula with normal appearance and optic disc with a 5/10 excavation (Fig. 2).

Due to low vision a refraction was performed, showing in right eye -2.25 -0.50 x 15° with a visual capacity of counting fingers at 10 cm, and in the left eye -2.00 -1.75 x 15° with a visual capacity of hands motion.

The patient was appointed 24 hours later, requesting manual visual fields and brain nuclear magnetic resonance to complement the assessment. One week later, the patient completed the requested tests and attended the assessment. The patient showed a visual capacity of 20/250 in both eyes, without changes in physical examination.

Manual visual fields showed a complete left homonymous hemianopsia and a 30° temporal peripheral reduction in the right eye and a 10° nasal peripheral reduction in the left eye, with partial preservation of the fixation point (Fig. 3).

By nuclear magnetic resonance imaging, axial sections showed a right occipital-temporal ischemic lesion, with bilateral small vessel occipital ischemia (Fig. 4) (Fig. 5) and coronal sections with a cortical defect of the right occipital pole (Fig. 6).

The neuro-ophthalmology department started treatment with atenolol 50 mg every 24 h, spironolactone 50 mg every 24 h, amlodipine 5 mg every 24 h, atorvastatin 20 mg every 24 h and referred the patient to neurological assessment.

Two months after admission, she had a visual capacity of 20/100 in both eyes without changes on ocular examination or symptoms. Her relatives informed that she still collides with objects.

**DISCUSSION**

At the time of the first contact with the patient, it is striking that she refers no visual problems. She was brought by her relatives because they observed that the patient lost the ability to see and recognize objects. Based on this, we deducted that the patient had a neurological disorder, classified as visual anosognosia or Anton syndrome.

The poor visual capacity of the patient cannot be explained by any of the ocular findings on examination.
That is why we decided to perform complementary tests, such as visual fields and imaging studies of the central nervous system. The visual fields showed changes that correlated with the MRI findings of occipital cortex ischemic damage.

The poor visual capacity observed at the first contact with the patient contrasts with an apparent conservation of the fixation point one week later when the visual fields were performed. This can be explained by reperfusion of the macular integration area by collateral circulation. This possibility of reperfusion is based on the clinical evolution towards improvement; however, in order to confirm this phenomenon, a second MRI with a diffusion sequence should have been performed. In our patient, it was not possible to perform it due to economic reasons.

Figure 3. Perimetry.

Figure 4. Nuclear magnetic resonance, axial sections.

Figure 5. Nuclear magnetic resonance with ischemia due to small vessel disease in the left parietal-temporal-occipital region (arrow).
Conclusion

The visual pathway can be affected in any of its regions by ischemic damage that causes variable manifestations that must be taken into consideration when evaluating a case of visual loss of cortical origin. It is important to be aware that not all cases of visual loss originate in the eyeball. The early recognition of pathologies with other associated visual manifestations allows a timely diagnosis of central nervous system disorders. It is also important to take into consideration the potential comorbidities in patients with this type of disorders, since most of the treatment is focused on strict metabolic control and close monitoring with an interdisciplinary team for comprehensive assessment and treatment.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

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Conflicts of interest

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