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Macropsia, micropsia, allesthesia, and dyschromatopsia after occipital intraparenchymal haemorrhage

Macropsia, micropsia, alestesia y discromatopsia tras hemorragia intraparenquimatosa occipital

Dear Editor,

Optical illusions are alterations in visual perception that can be characterized by an apparent modification of the size and shape of objects (dysmetropsia or metamorphopsia), seeing multiple images in the presence of a single object (polyopsia), preservation of visual images once the object that has caused the images has disappeared (palinopsia), and transposition of an object from one visual field to another (allesthesia). They may present temporarily during epileptic seizures, migraine, encephalitis, poisoning, and in psychiatric illnesses.[1] In most cases, focal brain lesions cause visual field defects. Optical illusions resulting from focal brain lesions are unusual[2]. In the review carried out, we have not found any previous case reports of combined visual illusions without campimetric alterations due to a vascular brain lesion.

A sixty-four year old female presented at the Emergency Department complaining of visual disturbances over the course of the previous 36 hours, of sudden onset, preceded by intense headache. The patient reported a mono and binocular visual syndrome consisting of three types of alterations. Firstly, and most strikingly, she reported constant variations in the shape of objects and people or metamorphopsia, such that they appeared to be extremely long and thin (macropsia), short (micropsia), wide, etc. Secondly, she reported altered colour perception (dyschromatopsia), such that objects changed in colour or even in intensity, and, finally, she described transposition of objects from one side to the other (allesthesia). The rest of the neurological examination failed to reveal any kind of focality or campimetric disturbance. A cerebral CT was performed (fig. 1) and revealed the existence of a right occipital intraparenchymal haemorrhage. Underlying lesions were ruled out by cerebral MR (fig. 2) and angio-MR. In the light of the possibility of occipital lobe epileptic seizures and despite the fact that the waking electroencephalogram failed to reveal any alterations, a decision was made to initiate treatment with levetiracetam 1000 mg/day. After 24 hours of treatment, the visual symptoms remitted and the patient was asymptomatic.

Cortical visual disorders may arise as a consequence of injury to the calcarin cortex that alters primary visual function or of lesions in associative visual areas. Permanent optical radiation and primary visual defects affect the visual field. Patients with lesions in this area may report visual

Figure 1 Cerebral CT revealing the presence of an on-going right occipital haematoma.

Figure 2 T1 sequence of the cerebral MR in which a hyperintense lesion can be seen located in the right occipital region, presenting a maximum diameter of 42 mm and very discreet perilesional oedema, compatible with parenchymal haemorrhage.

distortions and hallucinations, generally in the anopsic visual. Associative visual cortex syndromes (Brodman areas 18 and 19) differ depending on whether the lesion affects the temporo-occipital projections (fundamental for visual object identification) or the parieto-occipital projections (involved in the visual location of objects). Likewise, it appears that injuries to the most medial portion of the temporo-occipital pathway would give rise to macropsic phenomena, whereas those affecting the more lateral portion would cause micropsias.[2] In the case we present, it would seem reasonable to think that the lesion involves both parieto-occipital and temporo-occipital pathways, including the fusiform gyrus that plays an essential role in visual identification.

The exact pathophysiological mechanism of visual hallucinations secondary to focal lesions has yet to be elucidated. By means of SPECT (single-photon emission computedtomographyusing^{99}mTc-hexamethylpropyleneamine oxime [HMPAO]) the existence of occipital hyperperfusion has been demonstrated, suggesting an excitatory mechanism in visual hallucination in cases of subcortical haemorrhage.[3] Another aspect attributes optical illusions secondary to stroke to epileptogenic mechanisms of the occipital lobe. In our patient, the waking EEG coinciding with the optical illusions failed to yield findings of interest; nonetheless, it is worth mentioning that the symptoms resolved after 24 hours with anti-convulsion treatment without any residual visual field alterations.

There are cases in the literature that advocate a temporary nature of metamorphopsias, that rarely last for more than 24 hours, despite being due to a focal lesion as in the case of an established cerebral infarction.[4] However, episodes have been reported in which the metamorphopsias persisted for several years.[5] It is also a well-known fact that in certain cases, the phenomenon improves or resolves momentarily with different stimuli, such as hand movements, changes in posture, closing one's eyes, looking at an object close by, or holding on to a fixed object.[6] Attempts have been made to explain this phenomenon as being due to neuronal plasticity and the multi-sensory nature of the posterior parietal cortex. Thus, the intensification of certain stimuli by means of the afferent pathways in the posterior parietal cortex and the integrating capacity of the neural networks in charge of visual-spatial representation in this area of the cortex would be able to compensate for the deficit established by the lesion and the visual defect would be restored.

We underline the importance of a comprehensive aetiological study when dealing with optical illusions in the Emergency Department, since, as in the case presented here, they may be due to a focal brain lesion without campimetric alterations or other associated neurological focality that would make it possible to distinguish them from visual illusions due to ophthalmologic or psychiatric causes.

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