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Charles Bonnet' syndrome triggered by brimonidine in a patient with Leber's hereditary optic neuropathy☆

Síndrome de Charles Bonnet desencadenado por brimonidina en paciente con neuropatía óptica hereditaria de Leber

Dear Editor:

Charles Bonnet syndrome is a complex clinical entity consisting of the appearance of simple or complex visual hallucinations in patients preserving their cognitive state but evidencing great deterioration of eyesight.[1–3] Its incidence is increasing in our environment because of the growth of ocular pathologies such as age-related macular degeneration. Other pathologies (such as Leber hereditary optic neuropathy) that develop with serious sight deficits can present visual hallucinations. In the case we present, these problems were set off by the use of topical brimonidine (Alphagan®, Allergan, Madrid, Spain) for the treatment of ocular hypertension.

We present the case of a 30-year-old male, referred to the neuro-ophthalmology unit at our centre for visual hallucinations occurring during the previous month. The hallucinations consisted of people and faces that stared at him without speaking, with movement and in colour, with a history of a month; these coincided with starting treatment using topical brimonidine (1 drop every 12 h in both eyes [BE]) for ocular hypertension ocular diagnosed in his centre a month earlier. The patient specifically indicated that the hallucinations appeared 1 week after initiating treatment with the topical drug. He did not report any special pattern in the appearance of hallucinations, which were sporadic but on a daily basis, lasting an average of 15 min, and he did not present any other type of hallucinations. The patient had been diagnosed with Leber hereditary optic neuropathy in another centre. He did not report any other relevant personal antecedents or any known allergies.

In the examination, the patient presented visual sharpness in counting fingers at 1 m in BE, with normal anterior pole in BE. The intraocular pressure was 25 mm Hg in BE and 2 whitish papillae that looked atrophied could be seen in BE. The automated perimetry performed (OCTOPUS 1-2-3) revealed a terminal field of visual in BE. In the optical coherence tomography (Cirrus® HD-OCT, Carl Zeiss Meditec, USA), atrophy could be seen in the 4 quadrants of the papillae in BE. The patient was checked in the neuro-ophthalmology unit, carrying out a complete analysis and imaging tests, and other causes of hallucinations were ruled out. The treatment with brimonidine was suspended, due to poor tension control, and prostaglandin was substituted. The hallucinations disappeared partially after 72 h and totally at 1 week. The patient was diagnosed with Charles Bonnet syndrome secondary to treatment with brimonidine.

Brimonidine is a liposoluble α 2 agonist drug with the capacity to cross over the blood–brain barrier. Consequently, it can affect the central nervous system, producing symptoms such as somnolence, confusion and depression, and it can even produce coma in children. In patients having serious vision deficits, it has been described as the cause of visual hallucinations[4] that can be simple or complex, as in the case we present. Although the mechanism responsible is unknown, direct action of the drug on deafferented neurons as it passes through the blood–brain barrier could produce alterations in neuron stability that would trigger the hallucinations.

Although the cause is unknown, it is believed that the theory of deafferentation would be responsible for the development of the hallucinations. According to this theory, the loss of stimulation of the nerve cells in the retina from any ocular pathology would produce a loss of stimulation of the occipital cortex. The residual afferents would trigger the phenomenon of deafferentation, with anatomical, biochemical and histological changes in the synapses in an attempt to compensate for the lack of stimulation, being transformed into hyperexcitable.[1–4] In the face of specific stimuli (such as glare or darkness), different pathologies (anaemia or occipital stroke) or treatments (estrogens, tramadol or brimonidine), these hyperexcitable neurones would be stimulated, triggering the visual hallucinations in patients with serious visual deficits.[1–4]

In conclusion, we emphasise the side effects of brimonidine, which can produce visual hallucinations in patients with greatly deteriorated vision such as our patient affected by Leber optic neuropathy. The condition should not be confused with psychiatric pathology by ophthalmologists, neurologists, psychiatrists and family doctors, whose joint work is fundamental for the appropriate diagnosis and treatment of our patients.

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