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LETTERS TO THE EDITOR

Transient improvement in visual hallucinations with 'eye closing' technique

Background: Giant cell arteritis (GCA) is a chronic form of vasculitis of large and medium vessels, mostly involving the external branches of the carotid arteries, especially the temporal artery when it is referred to as Temporal Arteritis.[1] Many people are diagnosed with GCA after presenting with visual problems. Unusually GCA may involve smell and hearing alteration which may therefore be of diagnostic relevance.[2] GCA is treated with steroids and may result in steroid-induced psychosis that in turn can occur at lower doses in the older adult population due to pharmacodynamic and pharmacokinetic effects. We report here on visual hallucinations in the context of an exacerbation of GCA treated with high-dose steroids and with comorbid cataracts. and an intervention that proved effective in transiently alleviating symptoms.

Case description: A 65-year-old lady presented to the emergency department with suicidal ideation and anxiety. The past medical history included post-polio syndrome, atrial fibrillation, giant cell arteritis, newly diagnosed cataracts and hypertension. Subsequently, she was referred to our older adult home treatment team, which provides intensive treatment in the community. None of the medication was theoretically associated with visual hallucinations other than Prednisolone.

She reported visual hallucinations that were in the form of animals, a man and faces. An example of the animals were rats that were sometimes the size of beavers. The rats were seen peripherally running across the floor. There were also more general descriptions of animals and these were both stationary and moving. There was also a man seen standing inside the house. She also reported seeing faces on her bed or on the wall looking at her approximately once a week. The faces would mainly happen in poorly lit conditions, would be clear and in focus and would contort. Early childhood experiences of living on a farm may have influenced the content. Steroid titration was associated with olfactory hallucinations that were described as a burning smell and were reported less frequently. There was also a report of auditory hallucinations such as crashing sounds.

We monitored the response to a reduction in the steroid dosage which was gradual and associated with a slight improvement in symptoms although still distressing. An exploration of aggravating and relieving factors by one of the authors (AA) resulted in a request for the patient to assess the effects of eye closing. In the next appointment, she reported that whenever she closed and then opened her eyes, she would no longer experience the visual hallucinations of animals and faces and was consequently less distressed at those times.

Discussion: Visual hallucinations are an important clinical feature of Temporal Arteritis and may precede ophthalmologic emergencies. In one case report, visual hallucinations improved after the initiation of systemic steroids.[3] Although the mechanism of steroid-induced psychosis is unclear, synthetic steroids mediate mood disorders by disrupting the balance in the hypothalamo-pituitary-adrenal axis, by activating glucocorticoid receptors and suppressing steroid production from the adrenals[4] According to one study, psychiatric disturbances can occur at any point during corticosteroid treatment, including almost immediately after initiation as well as after cessation. In a review of 70 case reports, Lewis and Smith found a median time to onset of 11.5 days, with 39% of disturbances occurring during the first week, 62% within 2 weeks, and 83% within 6 weeks of treatment initiation[5](6). In a report of 14 cases and review of the literature, Lewis and Smith found that severe psychiatric reactions occur in approximately 5% of steroid-treated patients and that a large proportion of these patients have affective and/or psychotic symptoms.[6]

In a review with a case report, an 85-year-old widowed patient with temporal arteritis started to use Prednisolone 60 mg/day due to permanent loss of vision in her left eye. While reducing the Prednisolone, she started to experience depression, anhedonia, apathy, poor concentration, suicidal ideation, and hallucinations. She was hospitalised due to aggressive behaviour toward her 24 h carer.[7] Cataracts resulting in visual impairment can lead to the well-described Charles-Bonnet syndrome that may be of relevance in our case.

We suggest five possible mechanisms that may account for the hallucinations.

1. Release phenomenon: According to the theory of release phenomenon, visual deprivation removes inhibition of

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the visual association cortex. The lack of inhibition will result in inappropriate excitation[8,9] resulting in visual hallucination.[8,10,11]

2. Deafferentation: In deafferentation theory, loss of visual input to the cortex increases the excitability within the visual association cortex resulting in spontaneous neuronal activity and visual hallucinations[8,11–13]
3. Conscious Effect: Absence of Conscious Inhibition of hallucinations: Eye closing by interrupting the sensory input and misperception may support a conscious overriding of subconscious perception. There is evidence that dysfunctional inhibition of memory may have a role in auditory hallucinations in schizophrenia[14]
4. Gestalt Psychology: With the Gestalt phenomenon, the brain derives meaning from all of the sensory input. In our case, incomplete sensory input due to cataracts may result in unpredictable pattern completion resulting in visual hallucinations. Closing and reopening the eyes with a slightly altered head position may result in distinct but still incomplete sensory input with different results for pattern completion. In a more complex example with phantom limb syndrome, the manipulation of visual information resulted in resolution of pain perhaps using a similar pattern completion mechanism but this time integrating nociceptive and visual information perhaps at the level of the association cortex[15]
5. Ischaemic changes: The temporal arteries supply the retina and optic nerve. Ischaemia may result in visual dysfunction leading to hallucinations. Eye closing may significantly reduce the demands on the optic nerve and retina and provide an opportunity for recovery in the context of a compromised blood supply. In one case report, a 68-year old patient with Charles Bonnet Syndrome was diagnosed with nonarteritic anterior ischemic optic neuropathy and had similar visual hallucinations to our patient. This patient experienced monocular visual hallucinations in the left inferior visual field and this resolved with covering of the affected eye allowing the other eye to compensate.[16]

It is difficult to differentiate between steroid-induced psychosis and hallucinations due to the progression of GCA. Hallucinations seen in the later stages of the disease may precede permanent visual loss. An ophthalmologic review two months prior to the initial presentation to our services revealed that the GCA was stabilised with Prednisolone at that point. The alleviation of the visual hallucinations with eye closing merits further investigation and with further case reports it may be possible to more definitively investigate the impact of eye closing on visual hallucinations in GCA, steroid-induced psychosis, Charles-Bonnet syndrome or all.

Ethical considerations

We have obtained written consent from the patient and approval from the local research and innovation committee of our trust.

Declaration of Competing Interest

None.

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