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Mubashira Hashmi
Liaquat National Hospital

Ashfaq A. Sharif
Liaquat National Hospital

Shaista A. Siddiqui
Liaquat National Hospital

Muhammad Shahid Mustafa
Liaquat National Hospital

CHANGING PATTERNS OF VISUAL HALLUCINATIONS

Mubashira Hashmi, Ashfaq A. Sharif, Shaista A. Siddiqui, and **Muhammad Shahid Mustafa**
Department of Neurology, Liaquat National Hospital and Institute for Post Graduate Medical Studies and Health Sciences, Karachi, Pakistan

Correspondence to: Dr. Hashmi, Liaquat National Hospital, Stadium Road, Karachi-74800, Pakistan. Tel: +92-21-441-2492 & 441-2531. Fax: 92-21-414-0005. Email: dr_mhashmi@hotmail.com

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ABSTRACT

Visual hallucinations comprise some of the most vivid and sometimes bizarre symptoms in neuro-ophthalmology. This case highlights the under-recognized symptomatology of occipital lobe infarction. Interestingly, in the reported patient, visual hallucinations changed their character during the course of illness, probably because of a change in pathophysiology.

Visual hallucinations are associated with various neurological disorders including epilepsy and stroke.1 Seizures (simple partial, complex partial and generalized) have long been associated with usually unformed visual hallucinations, attributable to irritative focus in primary or associated visual cortex.[1,2,3] Very little, however, has been reported about release visual hallucinations, i.e. visual hallucinations with loss of vision arising from a defect in the afferent visual pathway from the retina up to striate visual cortex.[1,4] Recognition of release visual hallucinations is important to avoid unwarranted treatment with anticonvulsants and their potential side effects.

CASE REPORT

A 45-year-old right handed woman, with adult-onset diabetes for the last two years, presented to the emergency room with a ten-day history of stereotyped episodes of decreased vision in both eyes, left more than right, followed by flashes and zigzag lights of red and blue color in the left eye, gaze deviation towards the left, and a brief period of altered sensorium during which she was unresponsive to verbal commands. These episodes were very frequent, recurring every 15 to 20 minutes and each episode was preceded by a prodrome of fear and a sense of apprehension. Except for the decreased vision in both eyes during the interictal period, she had no other focal symptoms.

On examination, she had a left homonymous hemianopia and a maximum visual acuity of finger perception in both eyes. Routine hematological and biochemical tests were normal. On cranial MRI, the right occipital lobe was slightly edematous, with effaced sulci; post-contrast gyriform enhancement was noted in the vascular territory of the right posterior cerebral artery, consistent with infarction (Figures A, B and C). An EEG showed slowing in the right occipital area without any epileptiform discharges.

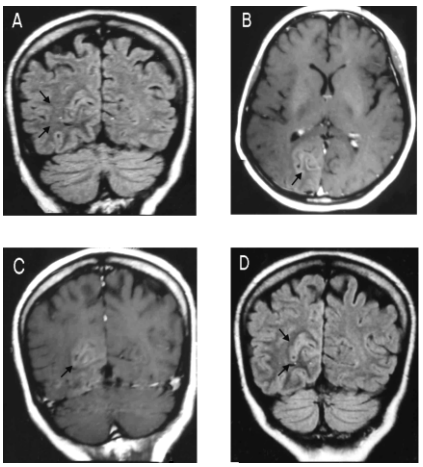

Figure A *Coronal FLAIR MRI showing effacement of sulci and gyri in right occipital lobe.*

Figures B & C *T1-weighted axial and coronal (post-gadolinium) images showing gyriform enhancement in right occipital lobe consistent with subacute ischemic stroke.*

Figure D *Coronal FLAIR MRI repeated after 1 week demonstrates effacement of sulci and a hyperintense rim still localized to the occipital region.*

She was treated with intravenous phenytoin and valproic acid. Symptoms responded with cessation of stereotyped episodes. Two days later she complained of seeing a woman in a veil, a woman wearing red or green clothes imitating the patient's actions, and scary human faces without torsos. These hallucinations were seen mainly in the left field of vision, accentuated in dark environments. They were emotionally disturbing, making her fearful and causing difficulty with sleep. She had insight into their unreal nature. No stereotypy or epileptiform activity was noted on EEG.

On repeat cranial MRI, the lesion was localized to the occipital lobe, with no extension to either the temporal or the posterior parietal region (Figure D). At this point, risperidone (6 mg in divided doses) and clonazepam (1.5 mg in divided doses) were begun. Her symptoms improved but did not disappear completely. Diagnostic work-up for stroke etiology was unremarkable.

Two weeks after admission, the patient was discharged. On the first follow-up visit about a week after discharge, her visual hallucinations had stopped and visual acuity had improved to almost normal with slightly impaired left peripheral vision, although she still complained of difficulty in going to sleep.

DISCUSSION

Visual hallucinations have been described in patients with occipital, posterior parietal and temporal infarcts.[1,2,4,5,6] Peduncular hallucinations are vivid and lifelike images of concrete objects, associated with ventral midbrain and pontine stroke.[7] Nakajima reported visual hallucinations in two patients with anterior cerebral artery infarcts.[8]

In the posterior circulation stroke, visual hallucinations may be explained either by seizures or release phenomena.[1,2] They are most commonly reported with occipital lobe epilepsy, where it can be the sole manifestation of seizure. Simple visual hallucinations were the most common presentation in patients with occipital seizures in multiple case series, whereas complex visual hallucinations represent extension of pathology to association visual cortices in the temporal and posterior parietal lobes. In seizures, visual hallucinations are brief, fragmentary, multicolored, stereotyped, associated with other motor or sensory phenomena, automatisms, and altered awareness, which are useful in differentiation from migraine and release phenomena. Ictal and postictal visual loss have also been reported with seizures. Visual deficits are almost always binocular. Patients may complain of homonymous hemianopia and even complete blindness.[1,2] Interestingly, our patient presented with both homonymous hemianopia and binocular decreased vision, postictally as well as at the start of ictal phenomena.

Release visual hallucinations are visual hallucinations associated with loss of vision, arising if there is any defect in the afferent visual pathway. Proposed mechanisms are either release of inhibition in the higher visual cortex or release of previously recorded percepts due to sensory deprivation in the defective field of vision.[1,2] There is usually a delay of days to weeks between the ischemic event and the onset of hallucinations. The patient reported here first experienced release visual hallucinations almost two weeks after her initial symptoms. In contrast to epilepsy with release visual hallucinations, the complexity of hallucinations in this setting have no localizing value.1 Release visual hallucinations are usually within the defective field of vision, continuous, non-stereotyped and increased in the dark.[1] However, there is a report in which visual hallucinations in a patient with left occipital infarction exacerbated on exposure to light.[9] The hallucinations are usually transient, lasting days or weeks, but they may be more persistent.

Advent of newer imaging techniques such as functional MRI has led to better localization of visual hallucinations and differentiation of seizures from release phenomena. However, these techniques are still not widely available, and in these circumstances clinical features and repeated or long EEGs are the main diagnostic tools. It is important to recognize release visual phenomena, to avoid unwarranted treatment with anticonvulsants and their potential side effects, as well as providing patients with reassurance and counseling. If needed, judicious use of drugs such as atypical antipsychotics is a useful measure in managing these patients.[1]

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