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Case Report

A Riddle: When you Look at One and See Two, What is it?

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Abstract

An array of frequent misperceptions encountered in clinical practice is presented, highlighting the rarity, among these, of Visual Allesthesia (VA). A clinical case is reported and a review of such published cases seem to indicate a strategic disruption of the occipito-parietal coneccions in the non-dominant hemisphere as the cause of VA.

Keywords: *Visual misperceptions-non-dominant parietal damage*

Introduction

Visual perceptual abnormalities are occasionally encountered in various fields of Medicine, particularly in Neurology, Psychiatry and Ophthalmology. These misperceptions may be caused by a wide variety of pathological conditions [1] and may express themselves as illusions, where the perception of a visual stimulus, present in the external environment, is altered [2] or as hallucinations where a visual stimulus is been perceived when in reality none is present [3].

Other visual misperceptions include palinopsia which consist in the persistence of a visual sensation after its visual stimulus has ceased, and metamorphopsia, where the view of the objective´s size and/or form is altered. On ocasion other forms of altered visual perceptions can occur and among these one rarely found in clinical practice is Visual Allesthesia (VA), a condition where the visual image of a stimulus is transposed from one homonymous visual field to the other [4,5]. Visual Allesthesia, whose pathophysiology is obscure, has been reported in the context of various medical conditions [6-8], the most frequent of which is a right parietal ischemic stroke. In the realm of neurosurgery, VA can be seldom found, as a postoperative complication [9,10].

Case Report

AJ, a 78 year-old righthanded Caucasian male, with no family history of sinistrality, reported that for the previous three years he had been experiencing recurrent episodes of colorless shimmering lights (flicker scotoma) in his right homonymous visual fields. These were not triggered by any particular stimulus, had no color and showed no movement but their light intensity fluctuated in a rhythmic pattern. These were not associated with auditory phenomena, nor with any change in mental status, convulsions, nausea or headache. These lasted a few minutes and cleared spontaneously, only to recur after variable periods of time.

During those years AJ did not notice any deterioration in his visual acuity nor any visual field defect. Only when his relatives observed some personality changes and some gait imbalance a consultation was requested.

His general physical and neurological examination, including visual field testing by confrontation, detected no abnormalities. A contrasted MRI revealed a right intraventricular meningioma with edema in the right parietal lobe (Figure 1).

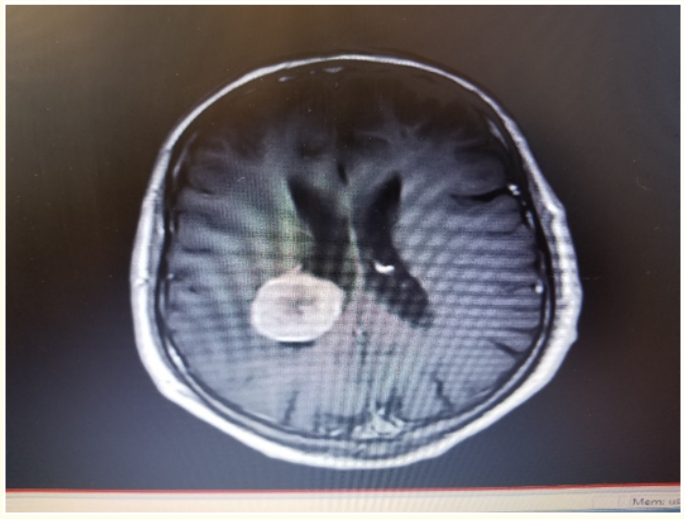

Figure 1: *Enhancing centrally necrotic intraventricular 3.8 x 3 x 4.5 cm mass in the posterior right lateral ventricle.*

AJ was directed to a neurosurgical tertiary referral center where surgery was undertaken via a right posterior parietal transcortical approach to the right lateral ventricle with image guidance assistance. Under the operating microscope the tumor was visualized and a biopsy revealed a grade 2 meningioma. The tumor was debulked by ultrasonic aspiration and it was totaly resected.

Post-operatively AJ reported a left homonymous hemianopia, some increase in his preexisting imbalance, optic ataxia [13,14] which interfered with his playing guitar and piano and caused a difficulty in shifting gaze to the next text line while reading. Through a two-year follow-up, the gait imbalance, and the optic ataxia improved somewhat but the visual field defect and the reading difficulty persisted. The left homonymous hemianopia was confirmed by Goldman field testing.

For the first two months of follow-up the homonymous hemianopsia manifested itself simply by the lack of visión in the afected visual fields, but thereafter some visual perceptions appeared spontaneously and exclusively in the left homonymous visual fields. The patient knew these visions were unreal and caused him no anxiety. Consequently these should be considered as pseudo-hallucinations. These visual phenomena presented themselves in three different formats which seemed to follow each other in a certain sequence, but were not associated with any other symptom. Initially they consisted of the visión of geisers of vapor, constantly changing in their position, their size and their orientation. These were monochromatic, in a light grey tone and lasted for a few seconds at a time, only to recur minutes or hours later. This format persisted for only five days when it was followed by a second type of visual perception consisting of the visión of a small colorless bright dot of light, a phosphene, which would appear at any point within the affected visual fields. During each episode the phosphene did not change in size, color or location, it lasted for a few seconds, only to reappear after variable periods of time at a

different location but with identical characteristics. The occurrence of this format gradually decreased in frequency and it was eventually replaced by a black dot which did not change in size or position and rather than a pseudo hallucination, it seemed to represent a sequelae of the damaged visual pathway. It has not changed its characteristics until the time of this writing. Finally a third visual format presented itself about two months postoperatively and consisted of a simultaneous visual replication, only within the impaired hemi fields of vision, of the image been visualized at the unaffected visual fields. This replicated image (Visual Allesthesia) appeared with no tilt with respect to the original object and it was polychromatic, its colors matching exactly the ones present at the scene been visualized by the intact visual fields. The replicated image would persist for a few seconds only but it would recur at various times during the day. This format persisted for about a month, after which the frequency and intensity of the replicated image gradually decreased, becoming less noticeable to the patient and more difficult to detect by him in the context of a real life scene. For the following two months AJ could still elicit this weakened replicated image by focusing his attention on it, but thereafter it disappeared for good.

Neither of these three formats presented themselves as a a sudden paroxysm nor where they associated with headache, simultaneous auditory phenomena nor with any change in his mental status. The allesthetic image was triggered only by focusing his existing visión on any particular objective.

The apparent sequence of these three formats suggested that they could represent the effects of the ongoing cortical and/or subcortical healing process.

Postoperatively and through this whole clinical course, the long-standing shimmering lights in the right homonymous visual fields persisted but changed somewhat. They would start by locating themselves at the center of the intact visual field, blurring the patient´s visión and gradually expanding as an enlarging circle to the point of involving the whole visual field, forcing the patient to close his eyes. After a couple of minutes the flicker scotoma would disappear and visión would be restored to normalcy in the right homonymous visual fields.

Four months postoperatively a MRI scan with contrast injection revealed the surgical track through the right posterior parietal área into the right lateral ventricle but no evidence of residual or recurrent tumor. An EEG showed a right parietal Delta focus with no epileptiform activity. Despite of it, and because of the obvious epileptic nature of of the flicker scotoma, treatment with Pregabaline 100 mg/day was initiated and shortly thereafter these visual phenomena disapeared not to reapear ever again.

Discussion

An anlysis of the published cases of VA (Table 1) would conclude that it results, in the majority of cases, from damage to the non-dominant visual dorsal stream [11,12] with interruption of the occipito-parietal connections, causing a selective loss of motion visión (Akinetopsia) [13], Balint síndrome, optic ataxia14 and/or VA [4-6].

Table 1

Author	Age (Years)	Sex	Cause	Seizures	Palinopsia
Murakami H.	49	M	Mitochondrial.	yes	Yes
Ardila., *et al.* #1	37	M	Cysticercosis	yes	Yes
Ardila., *et al.* #2	50	M	Stroke	No	Yes
Baumeler	60	M	Hemorrhage	yes	No
Mendez and Chen	57	M	Gunshot	Yes	Yes
Gonzalez mingot	64	F	Hemorrhage	Doubtful	No
Kasten and Poggel	61	F	Hemorrhage	Yes	No
Eretto., *et al.*	59	M	Dural AVM	Yes	Yes
Arai., *et al.*	63	F	Meningioma	Yes	Yes
Nakajima., *et al.*	30	M	Occipital AVM	Yes	Yes
Reptsis., *et al.*	46	M	Glioblastoma	No	No

Although the pathogenesis of this symptoms remains unsolved, the original neural disruption could hypothetically cause VA by a) the actual neural disconnection per se, or b) by the tissue disruption cutting off inhibitory circuitry leading to a focal cortical irritation resulting eventually in VA or c) by clinical or subclinical epileptic activity preceeding or following the original injury which could result in VA.

Depending of the extent of the affected area, which can vary from been very focal, to a more extensive one, its sequelae may be permanent or recover, in a minor or major degree, depending on the capacity for plasticity of the cerebral área affected [15].

While on-going research is focused on detecting and deciphering the underlying mechanisms of such plasticity along the optic pathway, the potential for the future emergence of therapeutic means to control this plasticity provides a more encouraging outlook for the care of these patients.

Conclusion

Visual Allesthesia, rather than to suggest an optical pathology should indicate a neurological condition.

Ethics Statement

Written informed consent from the patient was obtained for the publication of any potentially identifiable images or data included in this article.

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