Migrainous Binocular Peripheral Oscillopsia: A typical Persistent Visual Aura Without Infarction

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**Article ID:** WMC003984
**Article Type:** Case Report
**Submitted on:** 01-Feb-2013, 04:39:44 AM GMT  **Published on:** 01-Feb-2013, 06:27:17 AM GMT
**Article URL:** http://www.webmedcentral.com/article_view/3984
**Subject Categories:** NEUROLOGY
**Keywords:** Migraine, Migraine aura, Oscillopsia, Headache, Vertigo, Status epilepticus

**How to cite the article:** Jacome DE. Migrainous Binocular Peripheral Oscillopsia: A typical Persistent Visual Aura Without Infarction. WebmedCentral NEUROLOGY 2013;4(2):WMC003984

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**Source(s) of Funding:**
Self funded

**Competing Interests:**
None
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Abstract

An 18 year old female with history of migraine without aura reported the abrupt onset of continuous, irregular, rapid ("shaking") movements of objects perceived on the periphery of her visual fields over both eyes, when fixating on any given target. Symptoms were not suppressed by monocular eye closure. In some form, she experienced incomplete tunnel vision ("pseudo-tunnel"), since the periphery of her fields was not totally dark, lost coloration or was populated by phosphenes. She had some inconsistent lightheadedness but no vertigo, and reported a moderate continuous mid-facial headache. She denied symptoms referable to any other cranial nerve dysfunction. Her ophthalmologic and repeated neurological examinations were normal showing symmetric eye movements, normal saccades and smooth pursuit, no nystagmus and no ataxia. Her brain MRI and MRA of the extra and intracranial vessels were normal. Her EEG showed a normal background in the absence of a focal slowing, epileptic discharges or periodic complexes. Her visual evoked responses were symmetric with normal latencies. Her visual field testing revealed no deficits, yet, she had initially persistent peripheral movement illusion over both eyes. Her ocular symptoms lasted for several weeks progressively dissipating with the prophylactic administration of topiramate. I suggest that this patient's binocular peripheral oscillopsia represented persistent cortical oscillopsia without nystagmus, as a very rare atypical variant of migraine persistent visual aura without infarction. Cortical oscillopsia without nystagmus constitutes a neuro-ophtalmological syndrome, recently validated in a patient with neuromyelitis optica (NMO) and visual pathways demyelinating lesions. In my belief, this patient’s symptoms probably emanated from sustained cortical occipital hyper-excitability and from reverberating spreading cortical depression (CSD).

Introduction

Visual auras are the hallmark of migraine with aura. Patterns of visual disturbance can be assessed by applying the Visual Aura Rating Scale (VARS) to a population of migraine individuals belonging to the study group. The scale fundamentals call for assessment of duration, rate of progression, lateralization of the aura, and presence of scotoma and scintillation, that allows defining with greater precision if the symptoms experienced by the patient represent a true visual migraine aura or a non-migranous visual disturbance (1). The greater the score assigned on the VARS (i.e., > than 5) the greater diagnostic weight for migraine visual phenomena. The scale also facilitates the distinction between classic visual migraine aura, normally preceding the headache and of longer duration, and “transient visual disturbance”, prevalent in adolescents, of shorter duration and occurring during the headache phase (2). By applying the VARS, Wang, et al, were able to discriminate between the two groups (migraine versus non-migraine) in a total of 29 patients, six of their own (3). Individuals with greater VARS scores approximate migraine and have a better prognosis for short term resolution of their symptoms. Visual auras may occur in the absence of headache (i.e. “acephalgic migraine”) or headache may occur within the context of persistent visual aura with variable features and degree of intensity (2,3,4).

Persistent aura without infarction refers to focal neurological symptoms (i.e., of visual nature in this discussion) persisting for more than 1 week, in the absence of radiographic evidence of ischemic lesions on brain imaging studies (5). If auras are intermittent, occurring twice a day for 5 or more consecutive days as a minimum, the patient is classified as exhibiting “aura status” (5). In my experience these two definitions overlap in clinical practice and commonly are used interchangeably, given the circadian fluctuations on symptoms intensity and the participations of multiple variables in the clinical construct. These variables are among others: stress levels, effect of sleep, effect of medication and the menstrual cycle in women. The actual clinical features of visual auras are variable and often extensively complex to the point of almost achieving individually created outstanding artistic schemes in a significant number of patients, when interrogated in the clinical encounter. In general terms, auras include scintillating scotoma, bilateral central scotoma, tunnel vision, temporal crescent involvement, dyschromatopsia, amaurosis fugax, altitudinal loss of vision, transient...
Variants on the theme are recognized as “visual snow”, “primary persistent visual disturbance”, and “persistent positive visual phenomena” (4,6,7). In addition to imaging studies, electroencephalography (EEG), is indicated to discard “status epilepticus migrainosus” (8). Of pertinent relevance, exceptional patients with occipital lobe epilepsy manifested by ictal symptoms simulating typical lateralized visual aura, may also experience epileptic oscillopsia. Furthermore, as illustrated by Perucca, et al, in a 56 year woman with occipital post-hypoxic perinatal porencephaly, these ictal manifestations may associate with persistent headache on basis of secondary “non-convulsive status epilepticus”, establishing the occasional link between the epileptic process and cortical spreading depression (8). “Status epileptic migrainosus” can be equally observed in patients with “posterior reversible encephalopathy syndrome” (PRES), as described by Palma, et al, in a patient with PRES secondary to cetuximab (9). Finally, patients with persistent visual aura without infarction may show focal cerebral hypo-perfusion solely detected by perfusion MRI, or by single photon emission computed tomography (SPECT), yet, in the absence of clinical deficits or delayed radiographic evidence of cerebral infarction (10).

Case Report

An 18 year old female college freshman was seen in neurological consultation because of the rapid development of “shaky vision”. She described that objects or any background on the periphery of her visual fields were trembling of moving in irregular fashion without specific direction, and that her symptoms were initiated by attempting central fixation on any given target. Closing one eye did not suppress the movement on the open eye. She never had experienced this before. Her visual symptoms were associated initially with lightheadedness, photophobia and mild postural imbalance. In addition she reported a moderately intense mid-facial pain that soon became intermittent and controlled by rescue non-prescription analgesics. Her previous medical history included migraine without aura, mild asthma and occasional dizziness. She used albuterol inhalations rarely for asthma. Her mother had migraine and one aunt died from a ruptured intracranial aneurysm. Her paternal grandfather had Alzheimer’s disease. General physical examination was unremarkable. Her blood pressure was 130/91 mm Hg. Her pulse was 71, regular and she had no fever. Her neurological was normal while experiencing her visual symptoms and included a normal eye examination. Her eye movements were full and symmetric without nystagmus. Her ocular saccades and her smooth pursuit were normal. Formal ophtalmological examination was normal including slit lamp examination, automated visual perimetry and pattern shift visual evoked responses. Her electroencephalogram (EEG), brain MRI and MRI of the intra and extra-cranial circulation were normal. Her peripheral binocular visual oscillations persisted for several weeks, eventually dissipating following the administration of topiramate orally on incremental doses up a final dose of 50 mg twice a day. Eventually, when headache improved topiramate was discontinued without relapse on her oscillopsia.

Discussion

Oscillopsia refers to a visual percept movement disorder in which the objects in the environment move back and forth, up and down or side to side, in a disconcerting oscillation (11). Transient environmental oscillopsia is common as a compensatory corrective counter movement sensation, as when coming down from an elevator ride for instance. Typical recurrent or non-physiological oscillopsia, is a cardinal sign of superior oblique myokimia (SOM) among other conditions (12). Symptoms in SOM arise from irregular involuntary spontaneous contractions of the superior oblique muscle pulling the eye “down and in”. Individuals suffering with this condition describe monocular, intermittent, spontaneous oscillation of perceived targets that is not gaze dependant. The oscillation occupies the entire visual field, rather than only the periphery. Nystagmus is present but maybe subtle, intermittent, or both, therefore it may not be apparent to the clinical observer, unless electro-oculography (EOG) or orbital ultrasound is performed. Patients with SOM have no headache or additional visual symptoms other than periodic diplopia or polypia, while they respond readily to anti-epileptics agents and to surgical microvascular decompression of the homolateral trochlear nerve (12). Oscillopsia and tunnel vision are in the other hand, defining symptoms in patients with hindbrain-related syringomyelia (13). Although visual illusions may be a presenting sign of occipital strokes, the brain imaging studies in this patient did not demonstrate ischemic or hemorrhagic lesions, evidence of reversible cerebral vasoconstriction syndrome (RCVS) or of syringomyelia. Oscillopsia needs to be distinguished from “fixation switch diplopia”
(FSD) in where the patient experiences brief double vision when refocusing on new targets, but without the illusion of movement of the background. FSD is an infrequent condition seen in patients with history of early strabismus and loss of ocular dominance secondary to a change in their refractive error, or to the use of glasses encouraging fixation with the non-dominant eye (14).

The vestibular apparatus and its pathways may be compromised by the migraine process as often patients report dizziness, lightheadedness, vertigo, nausea and vomiting during the acute attacks. When the vestibular involvement becomes the sole expression of migraine, the symptoms are recognized under the rubber of vestibular or vertiginous migraine (15). In fact, individuals with migraine are susceptible to motion sickness, and vice versa, with preferential vulnerability to either movement-induced, or to visual-induced triggers (16). The latter association underscores the anatomic and physiological link between the visual and the vestibular systems, via brain stem trigemo-vestibular connectivity, and to its tendency to periodically become hyperexcitable in migraine sufferers. Of interest, facial pain induced experimentally by applying ice to the temples, aggravate nausea and headache during optokinetic-induced motion sickness in migraine patients (17). The latter finding reflects that accentuating cranial neurovascular reflexes by pain mechanisms or stress, heightens symptoms of motion sickness and increases susceptibility to migraine (17). These investigational subjects however, did not experience oscillopsia or visual disturbance in the form of tunnel vision. The patient herein described reported lightheadedness and vertigo at the onset of her symptoms of binocular persistent peripheral oscillopsia, as well as mid-facial headache.

I suggest that this patient had cortical oscillopsia without nystagmus. This rare, or at least poorly recognized disorder, is precisely defined by its name: these patients experiencing oscillopsia have no nystagmus on their examination, but evidence of occipital cortical dysfunction detected by functional MRI or PET scan (18). One single patient with NMO was reported very recently with the syndrome in question, and was found to harbor demyelinating lesions over the occipital lobes, corpus callosum and cortical lesions involving parietal area V5 (19). It is yet to be elucidated if co-activation of the vestibular cortex constitutes a pre-requisite for the development of oscillopsia, and if it indeed violates the visual physiological principle of “space constancy” that maintains perceptual stability despite gaze displacements (11,19). This principle requires a mechanism of high order visual afferent suppression with cancellation of irrelevant cognitive peripheral elements, during selective attention (11,18,19).

Unfortunately this patient impossibility to access promptly sophisticated testing in a major tertiary institution, did not permit the completion of functional MRI, PET scan and transcranial magnetic stimulation (TMS) studies. In particular, TMS and magnetoencephalography (MEEG) could have determined if she had visual cortex hyper-excitability, as it was demonstrated on six patients with migraine with persistent visual aura without infarction, by Chen, et al. (20). These authors advanced the notion that patients with the latter migraine variant, had reverberating cortical spreading depression (CSD) resulting in sustained excitatory effects. It is appropriate to suspect however, that the underlying occipital cortical hyper-excitability spared this patient’s occipital poles, since she had no central oscillopsia.

“The rotating snakes illusion” is elicited by looking to an arrangement of colored patches of four different luminance, periodically placed along the circumference of concentric circles (21). This layout allows the perception of a spatiotemporal illusory rotational motion triggered by micro-saccades and blinks. A related false percept is the “The pursuit –pursuing illusion” (22). The latter illusion is created by pursuing a circularly traveling small target located in the center of a display containing several peripherally located color disks placed in the center of a radial arrangement of sectors resembling sun flowers. The eye pursuit of the circular travel of the central target while exposed to the experimental background visual display creates the illusion of movement of the peripheral color disks in the direction of the moving central target (22). It appears that the peripheral disks in the radial arrangements are “carried over” with the pursuing eye movements of the rotating central fixation target, as the brain attempts to average or to synthesize the percept, in order to “make sense” of a discordant or illogical happenstance, given its evolutionary design to provide fast and useful responses (21 ). “The pursuit-pursuing illusion” illustrates the existence of a selective supersensitive visual sense of peripheral object motion, especially when following a moving central target. If this patient migrainous spontaneous visual phenomenon in a way simply represents repeats or the unmasking of atavistic pre-conscious abilities of the brain interpretative peripheral perceptual mechanisms, remains totally speculative without further experimental advanced psycho-physiological visual
The fact that this patient could not suppress her peripheral oscillopsia by monocular eye closure seems to indicate that the simultaneous engagement of both occipital cortices by visual stimuli is not essential for the clinical phenomenon to surface, even if sparing the occipital poles, the allocated areas for central vision.

References

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