Transient Recurrent Acquired Delayed Eidetic Memory: TRADEM Syndrome-Remembering the Present

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Abstract

A seventy eight year old psychiatrist developed recurrent transient episodes of sectoral visual colorful and detailed memories, as the actual identical events that have occurred in real life and registered by him in previous hours, resurrecting the actual original experience in a clear sensorium. He had no additional symptoms. In contrast to a previously reported patient with multiple sclerosis, these “remembered hallucinations” were not under volitional control and were mute. They differed from palinopsia because its complexity, its vivid detailed overtones, its delayed presentation, their logical evolving concatenation, and because they were not precipitated by external visual cues but originated from within. They could be suppressed by eye movement and by eye closure but not by blinking. They were also clinically distinct from the hallucinations of Charles Bonnet syndrome. Symptoms followed influenza vaccination and dissipated after four months, to never return. Extensive neurological investigations revealed only the presence of bilateral retro-bulbar optic neuropathy, suggesting a peripheral visual pathogenic mechanism for the replayed eidetic memories.

Introduction

Eidetic memory refers to a normally inherited ability to recall in vivid detail visual information, with or without other senses participation. Eidetic memory can be subdivided: When vivid recall is solely visual, is more strictly labeled as “photographic memory” or “eidetic imagery”. If other senses participate, it constitutes eidetic memory proper, in where the actual recollection conforms in its entirety to the original experience [1]. Modifying factors of the eidetic phenomenon includes duration, repetition, intensity and affective salience of the original life experience that is later recalled in great or greater detail, as “déjà-vécu” [1]. Some overlapping exists between synesthesia and eidetic memory proper. Synesthesia may be inherited as a unique ability or be acquired, as in the case of bimodal synesthesia secondary to central pontine myelinolysis (CPM) [2]. In synesthesia the simultaneous engagement of the senses is normally induced by external stimuli, with the exception of ictal epileptic synesthesia [3]. Although never submitted to rigorous scientific validation, there are similarities between synesthesia and psychotic behavior. The psychotic experience has “a life of its own” or even exists as alter ego, albeit detached from reality and internally accepted as tangible-real, suggesting the presence of an internal generator for dissociation and “synesthetic re-association”. A unique case of volitional miniature (“lilliputian”) monocular sectoral synesthesia in a patient with multiple sclerosis was reported by this author [4]. The patient could voluntary recall and replayed images of past experiences or faces with sound, over the right eye only. Yet, no dialogue or direct interaction was established with the false images.

A patient with intact cognition, bilateral post-vaccination retrobulbar optic neuropathy and recurrent episodes of eidetic imagery occurring in a clear sensorium is described. He exhibited no signs of psychosis and had no history of migraine.

Case Report

A seventy year old retired psychiatrist was seen in neurological consultation for multiple recurrent visual hallucinations in a clear sensorium, in the absence of additional concurrent symptoms of three months duration. The episodes lasted from three to five minutes. Although the hallucinations were colorful, they were not mobile or shifted, yet, “they were not frozen in time”. They tended to occupy the upper visual fields on both eyes and simulated the playback of a silent movie already watched, in luxury of detail. There was a delay of up to a few hours in the appearance of the hallucinations that spontaneously reproduced a living scene that took place earlier. For instance, the national news watched on television hours earlier, would appeared silently again in front of his eyes. Closing either eye did not suppress the visual phenomena but shifting his gaze or closing both eyes simultaneously suppressed them. Head position changes made no difference in either inducing or
abolishing the hallucinations. They began after experiencing severe flu symptoms following routine annual influenza vaccination. He never had similar symptoms in the past. He had no headache, nausea, double vision, unilateral weakness and numbness or a near syncope sensation along with the visual symptoms. His partner did not observe any changes in his demeanor, speech fluency and comprehension during the reported episodes. He had no droopy eyelids. He was never confused or acted inappropriate. He had no warnings including slurred speech or tunnel vision. Although he had mild depression it was stable and it was never accompanied of any hallucinations. He took bupropion 50 mg and escitalopram 20 mg a day reporting no side effects. He also took atorvastatin, levethyroxine, valsartan, amlodipine, allopurinol, hydrochlorothiazide and metoprolol at standard doses. Additional history included hypertension, hypothyroidism, mild renal failure following successful complete resection of renal cell carcinoma of the left kidney, localized prostate cancer, prostate hypertrophy, gout, obstructive sleep apnea, coronary artery disease, compensated chronic obstructive lung disease and coronary artery disease. One daughter was bipolar and there was family history of hypertension, heart disease, hypertension, obstructive lung disease and coronary artery disease. His general physical examination was not contributory. His blood pressure was 132/64 mm Hg, heart rate 54, regular and he was afebrile. His weight was normal. His visual acuity corrected was 20/20. Color perception was accurate. There was no tilting of the horizon or defects in depth perception. Pupils were round, equal and reactive to light and near and far. There were no afferent or efferent defects. He had no optic atrophy, papilledema or optic disc pallor. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus. He had no ptosis or lid retraction. He had no nystagmus.

Discussions and conclusion

Visual hallucinations arising from abnormal peripheral visual pathways are not uncommon. Lepore reported fifty seven patients with visual loss of peripheral origin that had experienced spontaneous visual phenomena [5]. In 21% of his series the hallucinations were complex in nature. Palinopsia is a visual phenomenon of perseveration. It may be secondary to cerebral lesions or may originate in the most peripheral visual pathways including the optic nerves. To illustrate this clinical occurrence in greater detail, refer to this author’s description of a patient with MS, palinopsia and visual extinction on fixation due to retrobulbar ON, and to Pomeranz and Lessell review on the subject [6, 7]. The latter authors reported palinopsia in addition to polyopia, in eight patients lacking cerebral lesions. Two patients of the latter group had optic nerve involvement (one had optic neuritis and a second Leber’s hereditary optic neuropathy). A third patient had macular edema [7]. Palinopsia in patients with peripheral visual pathways compromise is explained on basis of occipital cortical release or “deafferentation” [5, 6, 7]. The hallucinations of Charles Bonnet syndrome are also complex but somewhat capricious, may be mobile or stationary and include metamorphic details or figures lacking internal cohesion, i.e., “having a story within the story” [8]. Conversely, they may present as simple hallucinations, i.e., periodic monocular achromatopsia (personal observation, unpublished). They develop in a clear sensorium in subjects with visual defects, more often due to macular degeneration and less frequently following optic neuritis [8]. A patient with sectoral, “TV-like”, or “picture within a picture” spontaneous visual hallucinations involving the left inferior visual field was reported by Benegas, et, al [9]. She had a right parieto-occipital lobe stroke and a left inferior quadrantanopia [9]. Of relevance, the
patient herein described had no volitonal control on his hallucinations as in the case of lilliputian monocular hallucinations mentioned above, yet, eye movements (shifting gaze and refocusing) and bilateral eye closure extinguished the “re-lived memories”. Visual hallucinations only during eye closure, hence suppressed by opening of the eyes, may occur following general anesthesia, and constitutes a rare, transient, post-operative complication of cardiovascular and orthopedic surgery [10, 11]. The opposite is even more exotic; one patient with monocular quadrantanopic complex visual hallucinations due to non-arteritic anterior ischemic optic neuropathy, abolished by closure of the affected eye and gaze aversion, was reported by Toosy, et al. [12]. His hallucinations were not related to immediate or recent events and posed no logic or recognizable context. He had visual loss congruous with the location of his hallucinations [12]. The authors equated the effect of closure of the eye with poor vision, as the restorative mechanism for normal occipital cortical activation, once the afferent defective information was excluded from central computation [12].

Blinking on the patient been discussed, did not interrupt his displayed sectoral visual memories, even that blinking, albeit briefly, may interfere with visual recognition in normal circumstances [13]. Disappearance of epileptic occipital hallucinations with “fixation off” (changing the target of fixation) is the hallmark of epileptic “fixation off sensitivity” (FOS) but this patient had no occipital lesions, no history of seizures and his EEG was normal [14]. Although he had history of depression he was never psychotic, his hallucinations were not associated or followed by headache [17, 18]. It is unclear why the hallucinations were sectoral on this case, and not assigned to a fixed quadrantanopic defect, as is in the highly unusual example of sectoral palinopsia [19]. In the absence of hemianopsia his hallucinations did not constitute visual “mirror” allesthesa, where objects visualized in the normal field transferred, albeit incompletely, to the quadrantanopic “blind” visual field [20].

The diagnosis of non-inflammatory retrobulbar optic neuropathy may be missed in patient with normal or near normal visual acuity, or when it develops in isolation. Its diagnosis is best established by performing visual evoked potentials (VEPs) since MRI of the orbits will fail to show acute demyelination.

Uremia rarely causes ON [21]. Several categories of uremic related ON may be identified: 1. Anterior ischemic ON due to anemia and transient recurrent hypotension sometimes precipitated by dialysis. 2. Demyelinating ON. 3. Non inflammatory “toxic” axonal ON due to uremia or medication adverse effects. 4. Atrophic ON secondary to uremia related chronic idiopathic intracranial hypertension and papilledema [21]. It is very unlikely that uremic encephalopathy caused this patient’s visual hallucination given he had only a mild elevation of his BUN and creatinine, exhibited no clinical cognitive impairment and had a normal EEG. Furthermore, there was electrophysiological evidence of abnormal signal conduction through his optic nerves. Given his history, I propose this patient represents a clinical example of post influenza vaccination optic neuropathy, first described by Hull and Bates in a patient with bilateral ON recurring twice, within two weeks of exposure to the annual standard vaccination [22].

Recently a construct in the formation, sustenance, modification (“editing”) and expression of memories into the conscious real was presented by Meyer [23]. According to his hypothesis, the present is the past in process of actualization, where memories contained in the “converging-diverging zone”, are played back into “the screen” of the special senses, i.e., the retina and optic nerves. Following this reasoning, a defective peripheral sensor occasionally could result in bizarre recreations, when the retrieved information from the cortical convergent-divergent zones supersedes and masks the current life images been observed by the individual. Finally, of speculative philosophical significance (i.e., time is an illusion created by the brain), the rare clinical phenomenon of TRADEM, may represent a neuro-chronological disorder of time flow, in where past is also the present but only kept “undercover” until it becomes unmasked at the wrong time and for the wrong reasons [24].

References

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