

LSD Flashbacks – The Appearance of New Visual Imagery Not Experienced During Initial Intoxication: Two Case Reports

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ABSTRACT

A side effect associated with the use of synthetic hallucinogens such as lysergic acid diethylamide-(LSD) is the partial or total recurrence of perceptual disturbances which previously appeared during intoxication, despite absence of recent use. These are commonly referred to as “flashbacks” or Hallucinogen Persisting Perception Disorder (HPPD). Here we present two cases of patients with a prior history of LSD use who turned to psychiatric consultation following brief episodes of HPPD. Surprisingly, in both cases new visual imagery appeared during episodes of flashbacks which was not experienced during primary LSD use. Both subjects reported the ability to discern between LSD-associated visual disturbances and new visual imagery. This phenomenon did not cause functional impairment and in both cases caused gradual concern due to its persistence. Both patients refused medical treatment and continued psychiatric follow-up. At one year follow-up both patients reported almost complete spontaneous remission.

To the best of our knowledge these are the first reported cases of LSD-related benign flashbacks in which new imagery is experienced. Reasons for this reversible and apparently harmless side effect are proposed. Conclusions from case reports should be taken with caution.

INTRODUCTION

Hallucinogens encompass a family of natural-occurring and synthetic substances (1) which may induce states of generally short-term reversible intoxications principally characterized by the presence of visual disturbances popularly named “trips” (2). These phenomena may be manifested as pleasant “good trips” and unpleasant “bad trips.” These “trips” may generally exhibit recurrent single-visual disturbances (i.e., the emergence of the same visual disturbance when intoxicated) or recurrent multiple-visual disturbances (i.e., the emergence of different visual disturbances when intoxicated).

A poorly understood side effect mainly associated with the use of synthetic hallucinogens such as lysergic acid diethylamide (LSD) and LSD-like substances is the partial or total recurrence of visual disturbances which previously appeared during intoxication, despite absence of recent use. These are commonly described as identical to the primarily single or multiple visual disturbances that appeared during prior substance use. Single visual disturbances during intoxication implicate the continuous return of the same single-visual disturbance meaning total recapitulation of the only perceptual disturbance experienced during hallucinogen use. Multiple visual disturbances during intoxication implicate the continuous return of a single or some of the multiple visual disturbances experienced during hallucinogen use (3).

As defined in DSM-5 perceptual recurrences in HPPD recapitulate the prior substance intoxication reflecting the primary perceptual experience (4), namely, visual imagery experienced under hallucinogen intoxication should be re-experienced during recurrent perceptual disturbances.

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We present the cases of two patients with a prior history of LSD recreational use, who sought professional consultation for the presence of visual disturbances after totally stopping substance use. They interestingly reported that following substance discontinuation, they experienced the emergence of new visual disturbances which had not appeared during primary LSD intoxication. To the best of our knowledge this is the first report on the matter in the professional literature. Both patients gave informed consent for the publication of their cases.

CASE 1

Mr. R is a 24 year-old single male university student who completed compulsory military service and did not have any previous police or criminal records. He had a three years past history of occasional “social” cannabis use during weekends and holidays. He additionally reported social alcohol drinking and sporadic MDMA, cocaine and LSD use. He fulfilled DSM-5 full criteria for tobacco use disorder (4). During LSD intoxication he reported a variety of visual disturbances including **halos** (*a circular band of slight colored light around a light source*), **color intensification** (*more intensified color*), **flashes of colors** (*a patch of bright color that is apparent only during motion*) and **distorted perception of distance** (*objects were seen closer or distant*). He attributed the psychedelic experience to LSD use. One week after totally suspending substance use he noted some new returning visual imagery not previously experienced, in the form of **increased brightness** (*static or moving source appears to be radiating or reflecting light*) and **illusions of movement** (*slow motion movement of stationary objects*).

These recurring visual reminiscences were experienced as benign “free trips.” Mr. R reported the ability to discern between his original LSD-associated visual disturbances and the “new” visual imagery. As the condition persisted Mr. R began expressing concerns regarding these perceptual disturbances and feelings of discomfort when they appeared. He then turned to psychiatric consultation. On examination there was no previous history of acute or chronic neurological, ophthalmological or other comorbid medical diseases or co-occurring psychiatric disorders. A complete physical, neurological (including EEG) and laboratory examination were without abnormal findings.

Mr. R was not interested in “chemical” pharmacological treatment and agreed to psychiatric follow-up. After approximately one year these benign visual disturbances completely disappeared.

CASE 2

Ms. B is a 25-year-old female student university student who finished compulsory military service and did not have any prior police or criminal records. She had a four year past history of occasional “social” cannabis use. She also drank alcohol socially and sporadically used MDMA and LSD. She fulfilled DSM-5 full criteria for tobacco use disorder (4). During LSD intoxication she reported a variety of visual disturbances including **positive afterimages** (*an image continuing to emerge after the exposure to the original image has ceased*), **color intensification** (*more intensified color*), **flashes of colors** (*a patch of bright color that is apparent only during motion*) and **trailing phenomena** (*slight afterimage-like trail or perception of a series of slow-movement discrete positive afterimages in the wake of moving objects*). She referred the psychedelic experience to LSD use. Four days after complete substance discontinuation she began to experience recurrent visual imagery she had not previously experienced, mainly in the form of **visual distortions** (*slightly blurred forms and shapes of stationary objects*).

As in the previous case, these recurring visual reminiscences were experienced as benign “free trips.” Ms. B reported the capacity to differentiate between her original LSD-associated visual disturbances and the “new” visual imagery. After two months during which this condition continued Ms. B expressed concern and sought psychiatric evaluation. As in the case of R described above, all examinations revealed no abnormalities, treatment was not requested, and the phenomena had disappeared at one-year follow up. Trailing phenomena continued to appear intermittently.

DISCUSSION

We report two cases in which patients reported the appearance of visual disturbances that were not originally experienced during LSD-intoxication. HPPD has been comprehensively reviewed (5). Despite extensive research, understanding of these complex multifaceted conditions remains obscure (3).

The proposed biological platform which intends to explain these phenomena may encompass partial or total changes in the expression of genes implicated in synaptic plasticity in prefrontal cortical neurons; kindling, reverse tolerance or sensitization; chronic potentiation or depression of brain specific areas; and temporary or permanent impairment in areas involved in visual perception like Lateral Geniculate Nucleus (2, 6).

From a clinical point of view visual recurrences emerge as a result of the interaction between patients with a past history of use of hallucinogenic substances, vulnerability or predisposition (1, 2), co-occurring psychiatric disorders and recognized or unrecognized triggers (4). The cornerstone of this syndrome is the return of experienced visual imagery during hallucinogen use after cessation of hallucinogen use. But novel additional flashes, not experienced during previous hallucinogen use, may suggest the existence of a new subtype or an entirely different disorder.

The suggested mechanism underlying recapitulation of visual disturbances originally experienced during LSD intoxication focuses on continued visual central processing (2). An LSD-generated intense current (7) mediated through 5-HT₂ postsynaptic partial agonist activity (8) may provoke the reversible or irreversible destruction or dysfunction (3) of cortical serotonergic inhibitory interneurons with GABA-ergic outputs. This condition may lead to the persistence of the visual imagery due to pervasive progressive or benign unprogressive sustained disinhibition of visual processors (3). There is an inadequate inhibitory activity of the visual pathway allowing the visual stimulus to persist and giving place to the numerous visual disturbances which may reflect the failure of the each respective visual function (2).

In the above described cases in which flashes not originally experienced during LSD intoxication appeared, we speculate that it could be an additional onward transient functional impairment of the Lateral Geniculate Nucleus (LGN). The LGN, located inside the thalamus, appears to be of cardinal importance in the development of recurrent visual disturbances (2). LGN is the primary retransmitting center for visual information received from the retina and also receives data straight from the ascending retinal ganglion cells via the optic tract and from the reticular activating system. The LGN remits its axons throughout the optic radiation, a straight pathway to the primary visual cortex. Additionally the LGN may collect solid feedback linkages from the primary visual cortex (9, 10). Without profoundly describing structure, layers and cells of the LGN which are beyond the scope of this clinical manuscript, it could be shortly mentioned the existence of *magnocellular cells* which are necessary and responsible for the *perception of movement and brightness* and the *parvocellular cells* which are necessary and responsible for the *perception of forms and shapes* (10). Previous research showed that neurons of the LGN may be afflicted by LSD toxic effects (11, 12).

So, it could be hypothesized and speculated that LSD ingestion leading to ongoing progressive short-term reversible and limited impairment of the LGN affecting *magnocellular and parvocellular cells* could, at least partly, explain the temporary and transient presence of *illusions of movement, increased brightness and visual distortions* in both described patients. Continuation of *trailing phenomenon* may suggest a more permanent impairment and deserves further investigation.

These clinical case presentations may have some limitations. The basic premise of this presentation is that subjects are able to recall what they experienced when taking LSD and the current phenomena were not included. It remains a possibility that they occurred but were not recalled. Research on the matter may increase our understanding of the mechanism of action of LSD and related phenomena. The clinical value of the above described cases in everyday psychiatric clinical practice needs to be elucidated.

Conclusions from uncontrolled case reports should be taken with appropriate caution.

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