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Case Report Open Access

Hallucinogen Persisting Perception Disorder (HPPD) and Flashback-are they Identical?

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Abstract

Background: Despite a multitude of etiological and therapeutic approaches, the exact pathophysiological mechanisms underlying Hallucinogen persisting perception disorder (HPPD) remain elusive. Rather, in each individual case, specific risk factors and different vulnerabilities form part of a multifactorial origin of this rare but highly debilitating psychiatric disorder.

Case: The following case report describes the history of a 36 year old male who has been suffering from a visual perception disorder for the last 18 years. At the age of 17 he used LSD for the first time, having consumed cannabis and alcohol on a regular basis since a year earlier.

Descriptions: After one particular LSD trip at age 18, the patient suddenly developed persistent visual disturbances including small-sized, colour-intensive, flickering, geometrical patterns; intermittent after images of objects in the visual fields, and trailing phenomena of moving objects. Results from the Early Trauma Inventory (ETI) questionnaire indicated significant mental trauma in childhood and adolescence. Brain MRI and electrophysiological investigations (median nerve SEPs) revealed a few disseminated subcortical lesions.

Conclusion: Upon experimental treatment with lamotrigine, the patient experienced partial to complete remission of the various visual disturbances. With its potentially neuroprotective and mood-stabilising properties, lamotrigine may offer a promising new therapeutic approach for the treatment of HPPD.

Keywords: Hallucinogen persisting perception disorder (Flashbacks); Mental disorders

Introduction

From 1955 onwards, there have been numerous case reports of reocurring or prolonged persistent visual disturbances following hallucinogen use [1-3]. With the publication of the revised third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R) in 1987 operational diagnostic criteria were established under the diagnosis of "Posthallucinogen Perception Disorder". The DSM-IV-TR recognizes this syndrome as "292.89 Hallucinogen Persisting Perception Disorder (Flashbacks)" [4]. HPPD as defined in DSM-IV-TR is based entirely on observations from Abraham's cohort of habitual LSD-users [5]. Abraham interviewed 123 LSD users, who had consumed the substance in illegal settings. He reported the subsequent development of 16 persistent visual disturbances in these individuals [5]. The clinical relevance of these findings and their distinction from "flashbacks" and "HPPD", however, remains unclear and awaits further clarification [3-7]. Current knowledge with regard to risk factors, etiology and therapeutic options is thus limited and must be interpreted with caution [3,8].

According to DSM-IV-TR (292.89) [4], the main characteristic of HPPD is a permanent disturbance of visual perception. Such disturbances may take the following shapes or forms: geometric hallucinations, false perceptions of movement in the peripheral-field images, flashes of color, intensified colors, trails of images of moving objects as seen in stroboscopic photography, positive afterimages, halos around objects, macropsia and micropsia. In contrast to genuine psychosis there is no paranoid misinterpretation of these abnormal perceptions in HPPD sufferers. Visual disturbances may last for years and can cause severe mental distress.

In order to positively identify HPPD in DSM-IV-TR, other causes of visual disturbances such as anatomical lesions and infection of the brain, epilepsy, schizophrenia, delirious state or hynopompic hallucinations must be excluded first.

In contrast to DSM-IV-TR [4], ICD-10: F16.70 [6] flashbacks may be distinguished from psychotic state partly by their episodic nature, frequently of very short duration (seconds to minutes), and by their duplication of previous psychoactive substance-related experiences. Furthermore, the experience of typical flashbacks according ICD-10 are more often agreeable to the individual, because spontanous recurrences of altered states of conciousness that have occurred during previous intoxications with hallucinogens or cannabis are enjoyed and tolerated by some [9,10]. The short duration and transient nature of flashbacks, however, are likely to hamper research into this phenomenon [8].

Prevalence

The terms "Flashback" and "HPPD" are used interchangeable in the literature and has been defined in so many different ways that the concept of "flashback" is no longer considered a useful diagnostic entity [3]. Numerous reports of "flashbacks" date back for nearly 60 years in the scientific literature. Flashback seems to be a benign, non-distressing condition, sometimes accompanied by a pleasant feeling and tend to be fade out in short period of time. In contrast, HPPD causes pervasive distress and has been reported to occur either slowly reversible or irreversible on a permanent daily basis for months or years [11]. The classification of HPPD in DSM-IV-TR as a sequela to hallucinogen use exclusively as well as its postulated equivalence with flashback phenomena are not without problems. For example, Abraham [6,12] in

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his initial description of the condition, highlighted the persistence and stability of the visual abnormalities in the exclusive context of prior LSD use. Administration of the substance in a poorly controlled, illegal setting and the prolonged duration of its acute effects together with substance-induced altered self conciousness could combine to produce HPPD. Information with regard to the prevalence of flashbacks in the wake of hallucinogen use differs widely in literature. 5-50% of hallucinogen users are supposed to have experienced flashbacks on one or several occasions [13,14]. In contrast - the probability of developing HPPD after consuming a hallucinogenic agent is not known - the prevalence seems to be very low [3]. In a recent study using an online questionaire to document unusual visual phenomena in hallucinogen users 2679 individuals were included. Of these 224 had unrelated diagnoses and were excluded. 1487 (60.6%) of the remaining 2455 individuals reported at least one of the nine drug-free visual experiences. Although visual symptoms were common, 104 (4.2%) out of 2455 hallucinogen users found them distressing enough to consider seeking treatment. Thus, visual changes in hallucinogen users may be more commonthan previously suspected [15].

HPPD has been associated with a broader range of substances than only natural or synthetic occurring serotonergic 5-HT_{2A}-receptor hallucinogens. For example, methylenedioxymethamphetamine (MDMA) [16], cannabis [9,17], alcohol [18] and psychostimulants [19] have also been associated with HPPD-like syndroms.

In a study conducted on 9.400 participants, who had consumed LSD for research or therapeutic purposes, not a single case of HPPD was documented [20,21]. In an interview of over 500 Navajo members of the Native American Church -a legal mescaline using religious community- no signs of HPPD were reported [2,3]. These carefully controlled prospective and retrospective studies underline the importance of a protective setting to counter later anxiety and loss of self-control.

Closely related to mescaline in its psychotropic actions is psilocybin, albeit with a much shorter duration of the intoxicated state. Interestingly, only one case of HPPD after ingestion of *psilocybe semilanceata* mushrooms is mentioned in psychiatric scientific literature, despite the nowadays widespread use of psilocybin [22].

Methods

Relevant literature was identified by means of a computerized MEDLINE search from 1994 – present. As key-words "hallucinogen persisting perception disorder, (flashbacks)" and "HPPD" were used.

Case Report

History

The first administration of LSD as per the patient's own account occurred at the age of 17. Prior to this, he had been consuming alcohol and cannabis on a regular basis. During the first year of LSD use, the patient consumed 1-2 "blotters" with unknown dosage every other month. At the end of that year, after one particular "trip", the patient was afflicted with a sudden onset of several abnormal visual disturbances, which bear resemblance to visual symptoms of LSD intoxication. These included small intensely coloured, flickering, geometric shapes within the entire visual field; intermittent trailing phenomena following moving objects as seen in strobosopic photography, and after images of objects he had seen shortly before. No micropsia or macropsia was reported.

The symptoms were aggravated by mental stress, lack of sleep and after drinking caffeine containing beverages. These visuals disturbances severely impaired the patient's reading capacity. His own attempts to relieve the disorder by continued LSD use failed and the symptoms persisted unabated even during acute intoxication. The patient's LSD abuse continued for another 6 years followed by a 5 year period of abstinence. In 2005 he resumed his consumption of LSD once a month. Alcohol and cannabis addiction had been a constant feature in his life from the age of 16. From 2000-2005 the patient was also cocaine-dependent.

On several occasions, acute alcohol intoxication required emergency hospitalisation. In 2005 he needed intensive care treatment for acute pancreatitis and a peptic ulcer of the oesophagus. In 2010 he suffered from an upper gastrointestinal bleed. In October 2010 he quit alcohol and cannabis and moved into a therapeutic long-term institution for former addicts. Despite of his strict abstinence from polysubstance use the symptoms of HPPD persisted. Following the sudden deaths of his parents, a few weeks prior to his most recent appointment in our hospital, he had a brief relapse of his alcohol addiction, in the course of which he suffered another bout of acute pancreatitis.

Psychological assesssment

Following the patient's first admission to our hospital on 31.01.2012, psychological tests were conducted covering the following parameters: We used Beck Depression Inventory II (BDI II) for measuring the severity of depression [23]. To evaluate psychological symptoms and personality disorders the SCL-90-R, the SCID-II and the Essen Trauma Inventory (ETI) were used [24-26]. The patient's self-assessment showed slightly elevated scores for phobic anxiety (SCL-90 R). BDI result gave no indication of clinical depression, whereas SCID-II outcome scores were above cutoff for emotional instability, narcissistic and antisocial personality traits. Results from Essen Trauma Inventory (ETI) were indicative of mental trauma during childhood and adolescence.

During a follow-up evaluation 7 months later (8/2012) the patient scored higher (SCL-90 R) in the areas of somatization, depression and anxiety (probably as a response to the recent loss of his parents). Overall, his personality appeared to be more stable (SCID-II), specifically, scores for narcissism and emotional instability being clearly below cut off. However, antisocial traits remained unchanged and there was a marginal increase in insecurity and obsessive-compulsiveness (Table 1).

Treatment course

Between 2008 and 2010, the patient spent several weeks each in specialised institutions, as an inpatient, for the treatment of his multiple addictions. In February 2012, he underwent cognitive behavioural therapy for the very first time. Simultaneously, psychopharmacological therapy in the form of the antiepileptic lamotrigine was initiated – with weekly increments of 25 mg to reach a target dose of 200 mg per day. Prior to this the patient had been three months on clonidine (0.025 mg twice daily), which was stopped.

Lamotrigine is a widely used mood-stabilizing drug which acts by blocking sodium and voltage-gated calcium channels and inhibiting glutamate-mediated excitatory neurotransmission. Additionally, there are data supporting a neuroprotective effect and reduce symptoms of depersonalisation and derealisation [27,28]. Thus, we thought lamotrigine may offer a promising new medication for HPPD.

Following 7 months of regular medication with lamotrigine, the patient reported a steady improvement of his debilitating perception

Sensitivities	Initial evaluation (January 2012)	Follow-up evaluation (August 2012)
Beck Depression Inventory (BDI-II) [47]	Score 20	Score 10
Self-report Symptom somatisation	normal	T 69
Inventory 90 Items- depression	normal	T 70
Revised (SCL-90R) [48] obsessive-compulsive	normal	T 62
interpersonal sensitivity	normal	T 65
anxiety	T 60	T 68
phobic anxiety	T 65	T 71
aggression	normal	T 63
Psychoticism	normal	T 61
Global Severity Index, GSI	T 60	T 69
Positive Symptom Distress Index, PSDI	normal	normal
Positive Symptom Total, PST	T 63	T 71
Essen Trauma Inventory (ETI)		distinct indices for traumatisation and trauma sequelae
PERSONALITY		
Structured Clinical Avoident Personality Disorder, APD	4	5
Interview II Obsessive-Compulsive Personality Disorder, OCPD	7	6
(SCID-II) [49] Negativistic Personalitiy Disorder, NegPD	4	below cut-off
Narcisstic Personality Disorder, NPD	9	below cut-off
Borderline Personality Disorder, BPD	10	below cut-off
Antisocial Personality Disorder, ASPD	7	7

Table 1: Psychological tests.

disorder. The after images had disappeared completely and the colour-intensive geometric flickering patterns had grown fainter, less bright and more transparent. His reading capacity had improved significantly. In a particularly telling description of the unfolding perception changes, the patient reported that the "dirty screen" in front of his eyes through which he was forced to gaze for 18 years, was slowly beginning to clear.

Additional investigations

Ophthalmologic examination: Except for convergent strabismus, unremarkable.

Brain magnetic resonance imaging (MRI): A few very small subcortical gliotic scars in both hemispheres, most probably of vascular origin.

Median nerve-SEP: Significant reduction in amplitude on the left, latencies within normal range – a sign of somatosensory fibre damage.

VEP: Normal P100 amplitudes and latencies for both eyes.

Other medical causes of visual disturbances were excluded by careful clinical examination.

Discussion

Our patient, now 36 years old and currently in an apprenticeship in a tree nursery, had been experiencing persistent visual disturbances from the age of 18 onwards following a yearlong recreational use of LSD, cannabis, alcohol and cocaine. These disturbances manifested themselves variously as photopsia (included small intensely coloured, flickering, geometric shapes within the entire visual field), afterimages (of an object he had seen shortly before), and trailing phenomena (following in the path of moving objects) – all of which seriously compromised his overall quality of life.

Upon admission to our hospital, routine investigations yielded either completely normal results (EEG, VEP, negative blood and urinary drug screen) or minor abnormalities (Brain MRI, median nerve SEP). Psychopathologically, there were no signs of a major affective, psychotic or cognitive disorder. Psychological assessment (Table 1) did show relevant impairment of the self-report inventories: The structured personality interview (SCID-II) [25] yielded high scores for antisocial, avoidant, narcissistic, negative, obsessive-compulsive and borderline personality disorders. In addition to social phobic tendencies (SCL-90-R) [24], the patient's self-assessment in the ETI was indicative of mental trauma suffered during childhood and adolescence [26].

A three month course of the α_2 agonist clonidine was abandoned after it did not result in any benefit to the patient. Within 7 months of lamotrigine treatment (200 mg daily) the patient noticed significant improvement of his visual disorder as well as overall mental well-being. Complete to partial remission of the after images and flickering patterns occurred. The photopsias were judged to be less colour intensive and more translucent. Being able to read again was a source of great relief to the patient.

Despite 60 years of research into HPPD, a unifying pathophysiological model has yet to emerge. Case-specific environmental influences, such as type of setting during drug use, as well as individual vulnerabilities warrant a multifactorial etiopathological approach to this distressing disorder. As demonstrated in this case report, co-morbid major psychiatric illnesses (in our case polysubstance use, mixed personality disorder, mental trauma in childhood/adolescence) may constitute such

variables [7,8]. In this context it is noteworthy that a whole range of non-hallucinogenic psychotropic drugs are also known to elicit complex visual perception disorders. For example, palinopsia and visual trails were reported in young adults, with no history of prior substance abuse, after treatment with trazodone [29], nefazodone [30], risperidone [31], mirtazapine [32] and topiramate [33,34]. In most cases, however, the visual disturbances resolved fully after discontinuation of the respective drugs. One can only speculate about the underlying pharmacodynamic mechanisms. For instance, all the above-mentioned compounds - with the exception of topiramate, share a partial serotonergic receptor profile. Although no report on serotonergic receptor activity of topiramate has been performed to date, it has been suggested that the weight loss associated with topiramate may be ascribed to an action of the 5-HT_{2C} receptors [35]. On the other hand, short duration of visual symptoms and heterogeneity of the pharmacological triggers argue against a distinct substance or receptor specific etiology.

In contrast our patient displayed a very long-lasting form of HPPD. As indicated in criterion C of DSM-IV-TR, alternative causes must be considered before diagnosing HPPD. It cannot rule out that medical and psychological co-factors may have combined with the effects of LSD to produce HPPD. In our case brain MRI and median nerve SEPs revealed a few disseminated subcortical lesions. Indeed, several case-reports have noted that the abuse of cannabis, alcohol and psychostimulants may trigger or worsen HPPD [9,10,18,19]. The so-called "bad trips", i.e. acute intoxications producing intense fear and dysphoria in the user, often result from multiple drug use in an unfavourable setting, frequently culminating in severe mental disorders and occasionally HPPD. It is also difficult to rule out other psychiatric disorders such as posttraumatic stress disorder (PTSD), because some of its diagnostic criteria resemble the symptoms of HPPD [3]. Despite of these restrictions, it seems right that some individuals who have used LSD, experience persistent symptoms of HPPD for years, are not better attributable to another medical or psychiatric condition.

Abraham & Duffy (1996) hypothesized that HPPD is a disinhibition of visual processing related to a loss of 5-HT-receptors on inhibitory interneurons [36]. This suggests that the circuitry responsible for HPPD is not a higher brain area, but a lower primary first cortical area (V1) which is responsible for geometric processing of visual input.

Treatment

Pharmacological approaches to HPPD are currently based on a few uncontrolled single case observations with sometimes contradictory recommendations as to the clinical usefulness of clonidine, SSRIs, benzodiazepines, risperidone, olanzapine and naltrexone (Table 2). Thus, no sound clinical guidelines exist for a rational pharmacological treatment of HPPD [3,7,37,38].

Author	Year of publication	Drug	Sample size	Study design	Major results
Moskowitz et al. [38]	1971	Haloperidol	8	case reports	8 military prisoners were sucessfully treated with haloperidol. The case description suggest that several subjects suffering from an underlying chronic psychotic disorder.
Abraham [6]	1983	Benzodiaepines, Phenothiazines	21	Observational study	8 of the 9 subjects receiving benzodiaepines reported a reduced intensity and frequency of visual disturbances, whereas 11 of 12 sujects receiving phenothiazines reported exacerbation of HPPD
Abraham [39]	1996	Risperidone	3	Case reports	3 HPPD patients treated with risperidone reported an exacerbation of LSD – like panic and visual symptoms.
Young [40]	1997	Sertraline	1	Case report	A 22 old male patient had stopped using LSD after an 8-month history of abuse. Despite his abstinence he developed HPPD. sertraline appeared to have exerbated HPPD initially, but attenuated symptoms after 1 month administration (100 mg/daily).
Lerner and Crayton [41]	1997	Naltrexone	2	Case reports	Dramatic improvement with naltrexone (50 mg daily) was reported in two young men with LSD-induced HPPD. The remission was sustained as it was possible to discontinue the naltrexone after 2 months without precipitating a relapse.
Lerner and Bard Ermentrout [43]	2000	Clonidine	8	Observational study	6 of the 8 subjects (2 dropped out) received clonidine (0.025 mg, three times a day) for 2 months alleviated LSD-related HPPD.
Lerner et al. [46]	2001	Olanzapine and Fluoxetine	1	Case report	A 17-year-old boy with abuse of LSD weekly for 4.5 years developed HPPD after 5 months of abstinence. The patient showed a marked exacerbation in symptoms while taking risperidone and showed an attenuation of HPPD with a combination of fluoxetine and olanzapine.
Moskowitz [44]	2002	Reboxetine	1	Case report	During a 6-month follow-up period on reboxetin (6mg/day) no exacerbation of visual disturbance were reported.
Abraham and Mamen [45]	2003	Clonazepam	16	Observational study	16 patients received clonazepam 2mg/day for 2 months. Patients reported significant relief during the clonazepam administration. This improvement persisted during a 6 months follow-up period.
Batzer et al. [18]	2005	Olanzapine Risperidone Sertraline	1	Case report	A case of a young man presenting HPPD after a mixed intoxication with psilocybin and cannabis. Olanzapine (5mg) exacerbated symtoms and was replaced by risperidone (2mg/day) and sertraline (150 mg/day). After 6 months of this treatment HPPD disapeared.
Fontenelle [34]	2012	Lamotrigine	1	Case report	A case of a young woman, who displayed over 13 years HPPD received a year-long tial of lamotrigine (200 mg/daily) and experienced a significant relief from her HPPD symptoms.
Abraham HD [unpublished data]	2012	COMT-inhibitor Tolcapone Levodopa augmentation	20	Observational study	20 Patients with HPPD received in an open label design tolcapone (200 mg/day), carbidopa (25 mg/daily), and levodopa (100mg/day). The treatment resulted in a medication effect size of 0.2

Table 2: Case reports on HPPD treatment.

Moreover, it remains unclear whether the "successful" treatments listed in Table 2 are the result of actual therapeutic efficacy or spontaneous remissions. The latter, according to Abraham, occur in approximately half of all cases of HPPD within a few months of the onset of symptoms [39]. Patients with HPPD treated with SSRIs and atypical antipsychotics (e.g. risperidone, olanzapine) reported an initial exacerbation of their symptoms with a subsequent gradual improvement over time [22,40,41]. Patients with HPPD treated with benzodiazepines reported experience with an overall reduction in the intensity of visual disturbances [5]. Lerner and colleagues used clonazepam, which is known to have serotonergic properties [42]. Other successful pharmacologial treatments include clonidin and reboxetine, which act on adrenergic receptors.

An open-label treatment study was presented by HD Abraham at the Annual Meeting of the Biological Psychiatry Society in 2012. 20 patients with HPPD received in an open label design the COMT inhibitor tolcapone (200 mg/day) and levodopa augmentation. This study is the largest controlled treatment study of HPPD to date. The treatment improved HPPD symptoms in a third of the patients (unpublished data). Thus, it seems there are different pathways reducing the symptoms of visual disturbances, so HPPD may arise from either excitation or inhibition of visual networks [43-49].

In the present case, as well as a recently reported case of a 33 year old female with a 14 year long disease course, the observed improvement was at least partly a result of lamotrigine treatment [11].

Implications for clinical care

In clinical practice HPPD may be observed as a syndrome with individual psychological and somatic comorbidity.

According to Halpern and Pope [3], Passie and Holland [8] current knowledge about risk factors, etiopathologic mechanisms, and available treatments must be interpreted with caution. In available literature, information about co-existing medical conditions, use of alcohol and other illicit drugs is limited and not sufficiently evaluated.

HPPD should not only associate with hallucinogen use and should recognize for the multifactorial origin of the condition. In particular, non-substance related variables such as prior mental trauma and concomitant psychiatric illnesses may combine with alcohol or other illicit substances to produce a clinical HPPD syndrome.

Lamotrigine may offer a new treatment for HPPD. Future research into the treatment of the condition will, however, require randomised controlled trials especially for patients with chronic form of the disorder [3].

Treatment of HPPD should also involve abstinence from all substances of abuse, stress reduction and treatment of psychiatric comorbidities.

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Conflict of Interest statement

The authors declare no conflict of interest in preparing this article.

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