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 during the preceding year.

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 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 reoccurring 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 hypnopompic hallucinations must be excluded first.

In contrast to DSM-IV-TR [4], ICD-10: F16.70 [6] flashbacks may be distinguished from psychotic states 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 spontaneous recurrences of altered states of consciousness 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 interchangeably in the literature and have 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 tends to 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 sequel to hallucinogen use exclusively as well as its postulated equivalence with flashback phenomena are not without problems. For example, Abraham [6,12] in...
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 consciousness 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 questionnaire
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 common than
previously suspected [15].

HPPD has been associated with a broader range of substances
than only natural or synthetic occurring serotonin 5-HT_{3}-receptor
hallucinogens. For example, methylenedioxymethamphetamine
(MDMA) [16], cannabis [9,17], alcohol [18] and psychostimulants [19]
have also been associated with HPPD-like syndromes.
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 “blotter” 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 tailing phenomena following moving objects as seen in stroscopic 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 visual 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
hospitalization. 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 bleeding. 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 assessment
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
specialized 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).
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
depersonalization and derealization [27,28]. Thus, we thought
lamotrigine may offer a promising new medication for HPPD.
Following 7 months of regular medication with lamotrigine, the

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patient reported a steady improvement of his debilitating perception 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 year long 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 stopped 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

### Table 1: Psychological tests.

<table>
<thead>
<tr>
<th>Sensitivities</th>
<th>Initial evaluation (January 2012)</th>
<th>Follow-up evaluation (August 2012)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck Depression Inventory (BDI-II) [47]</td>
<td>Score 20</td>
<td>Score 10</td>
</tr>
<tr>
<td>Self-report Somptom</td>
<td>somatisation normal</td>
<td>T 69</td>
</tr>
<tr>
<td>Inventory 90 Items</td>
<td>normal</td>
<td>T 70</td>
</tr>
<tr>
<td>Revised (SCL-90R) [48]</td>
<td>obsessive-compulsive normal</td>
<td>T 62</td>
</tr>
<tr>
<td>interpersonal sensitivity</td>
<td>normal</td>
<td>T 65</td>
</tr>
<tr>
<td>anxiety</td>
<td>T 60</td>
<td>T 68</td>
</tr>
<tr>
<td>phobic anxiety</td>
<td>T 65</td>
<td>T 71</td>
</tr>
<tr>
<td>aggression</td>
<td>normal</td>
<td>T 63</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>normal</td>
<td>T 61</td>
</tr>
<tr>
<td>Global Severity Index, GSI</td>
<td>T 60</td>
<td>T 69</td>
</tr>
<tr>
<td>Positive Symptom Distress Index, PSDI</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>Positive Symptom Total, PST</td>
<td>T 63</td>
<td>T 71</td>
</tr>
<tr>
<td>Essen Trauma Inventory (ETI)</td>
<td>distinct indices for traumatisation and trauma sequelae</td>
<td></td>
</tr>
</tbody>
</table>

**PERSONALITY**

<table>
<thead>
<tr>
<th>Structured Clinical Interview II</th>
<th>Avoidant Personality Disorder, APD</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obsessive-Compulsive Personality Disorder, OCPD</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Negativistic Personality Disorder, NegPD</td>
<td>4</td>
<td>below cut-off</td>
</tr>
<tr>
<td>Narcissistic Personality Disorder, NPD</td>
<td>9</td>
<td>below cut-off</td>
</tr>
<tr>
<td>Borderline Personality Disorder, BPD</td>
<td>10</td>
<td>below cut-off</td>
</tr>
<tr>
<td>Antisocial Personality Disorder, ASPD</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>
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 published to date, it has been suggested that the weight loss associated with topiramate may be ascribed to an action of the 5-HT₄ 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 these restrictions, it seems evident that some individuals who have used LSD, experience persistent symptoms of HPPD for years, 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-HT₄ 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,

<table>
<thead>
<tr>
<th>Author</th>
<th>Year of publication</th>
<th>Drug</th>
<th>Sample size</th>
<th>Study design</th>
<th>Major results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moskowitz [44]</td>
<td>1971</td>
<td>Haloperidol</td>
<td>8</td>
<td>case reports</td>
<td>8 military prisoners were successfully treated with haloperidol. The case description suggest that several subjects suffered from an underlying chronic psychotic disorder.</td>
</tr>
<tr>
<td>Abraham [5]</td>
<td>1983</td>
<td>Benzodiazepines, Phenothiazines</td>
<td>21</td>
<td>Observational study</td>
<td>8 of the 9 subjects receiving benzodiazepines reported a reduced intensity and frequency of visual disturbances, whereas 11 of 12 subjects receiving phenothiazines reported exacerbation of HPPD</td>
</tr>
<tr>
<td>Abraham and Mamen [45]</td>
<td>1996</td>
<td>Risperidone</td>
<td>3</td>
<td>Case reports</td>
<td>3 HPPD patients treated with risperidone reported an exacerbation of LSD – like panic and visual symptoms</td>
</tr>
<tr>
<td>Young [40]</td>
<td>1997</td>
<td>Sertraline</td>
<td>1</td>
<td>Case report</td>
<td>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 treatment after 2 months without precipitating a relapse</td>
</tr>
<tr>
<td>Lerner et al. [46]</td>
<td>1997</td>
<td>Naltrexone</td>
<td>2</td>
<td>Case reports</td>
<td>6 of the 8 subjects (2 dropped out) received naltrexone (0.025 mg, three times a day) for 2 months alleviated LSD-related HPPD.</td>
</tr>
<tr>
<td>Lerner et al. [50]</td>
<td>2000</td>
<td>Clonidine</td>
<td>8</td>
<td>Observational study</td>
<td>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</td>
</tr>
<tr>
<td>Aldurra and Crayton [41]</td>
<td>2001</td>
<td>Olanzapine and Fluoxetine</td>
<td>1</td>
<td>Case report</td>
<td>During a 6-month follow-up period on reboxetine (6mg/day) no exacerbation of visual disturbance was reported</td>
</tr>
<tr>
<td>Lerner et al. [48]</td>
<td>2002</td>
<td>Reboxetine</td>
<td>1</td>
<td>Case report</td>
<td>During a 6-month follow-up period on reboxetine (6mg/day) no exacerbation of visual disturbance was reported</td>
</tr>
<tr>
<td>Lerner et al. [51]</td>
<td>2003</td>
<td>Clonazepam</td>
<td>16</td>
<td>Observational study</td>
<td>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.</td>
</tr>
<tr>
<td>Espiard et al. [22]</td>
<td>2005</td>
<td>Olanzapine Risperidone Sertraline</td>
<td>1</td>
<td>Case report</td>
<td>A case of a young man presenting HPPD after a mixed intoxication with psilocybin and cannabis. Olanzapine (5mg) exacerbated symptoms and was replaced by risperidone (2mg/day) and sertraline (150 mg/day). After 6 months of this treatment HPPD disappeared.</td>
</tr>
<tr>
<td>Hermle et al. [11]</td>
<td>2012</td>
<td>Lamotrigine</td>
<td>1</td>
<td>Case report</td>
<td>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.</td>
</tr>
<tr>
<td>Abraham [unpublished data]</td>
<td>2012</td>
<td>COMT-inhibitor Tolcapone Levodopa augmentation</td>
<td>20</td>
<td>Observational study</td>
<td>20 Patients with HPPD received tolcapone (200 mg/day), carbidopa (25 mg/daily), and levodopa (100mg/day) in an open label design. The treatment resulted in a medication effect size of 0.2</td>
</tr>
</tbody>
</table>

**Table 2:** Case reports on HPPD treatment.
benzodiazepines, risperidone, olanzapine and naltrexone (Table 2). Thus, no sound clinical guidelines exist for a rational pharmacological treatment of HPPD [3,7,37,38]. 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 serotoninergic properties [42]. Other successful pharmacological treatments include clonidine 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 [43-50].

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 be only associated with hallucinogen use and should be recognized for the multifactorial origin of the condition. In particular, non-substance related variables such as prior mental trauma and comonitant 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.
of Essen Trauma Inventory (ETI). Zeitschrift für Psychotraumatologie, Psychotherapiewissenschaft, Psychologische Medizin. 1: 75-69.


