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A patient with distinct dissociative and hallucinatory fugues

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Summary

A 62-year-old man presented with a history suggesting both dissociative fugue and a distinct fugue-like hallucination. The dissociative fugues included unplanned travel, loss of personal identity, inability to recall his past and amnesia for the fugue interval. The subjective fugues consisted of a stereotyped hallucination wherein he would travel to a social gathering place, meet his 'imaginary friends' and engage with them in conversation. He experienced the subjective fugues as if they were real, recognised them as hallucinations when he was normally conscious, and remembered them in great detail. A hallucinatory fugue episode occurred during video-EEG monitoring. The patient engaged in semipurposeful behaviour for which he had no memory, and the EEG demonstrated waking rhythms. Epilepsy, sleep disorder, factitious disorder and malingering were excluded from the differential diagnosis, leaving a patient with both dissociative and hallucinatory fugues, likely made possible by remote traumatic injury to limbic, arousal and motor circuits.

BACKGROUND

In addition to a diagnosis of dissociative fugue, which itself is very rare, the patient had unique hallucinatory fugue episodes. His detailed and articulate description of these experiences and his cooperation in the diagnostic process, combined with the remarkable video-EEG data narrowed the diagnosis to hallucinatory fugues which he vividly remembered, associated with a behavioural disturbance for which he was amnesic.

CASE PRESENTATION

A 62-year-old highly educated man was referred by his psychiatrist for evaluation of possible epilepsy. He described having had several episodes lasting hours consistent with dissociative fugue. Typically, he would get on the New York City (NYC) subway near his home in Brooklyn, but would never arrive at his destination. Instead, he would regain consciousness hours later, either on the subway or wandering around an unfamiliar neighbourhood in a borough of NYC to which he had not intended to go. He would then determine his location by asking passers by, and would return home without further incident. In one remarkable episode he began a subway ride with a destination in Manhattan, and regained awareness wandering in Boston, Massachusetts, USA nearly 250 miles away. From a ticket stub in his pocket he determined that he had taken the subway to Penn Station in Manhattan, bought a ticket and rode an Amtrak train to Boston, and then began wandering the nearby streets.

He also described distinct episodes that began about 5 years earlier and occurred two to three times per week, usually when he was walking outside or relaxing at home in the evening. These spells consisted of a stereotypical hallucination wherein he would travel to a gathering place such as a bar or coffee house, meet a group of 'imaginary friends' and engage with them in collegial conversation. He experienced the episodes as if they were completely real, yet when normally conscious he recognised them as hallucinations. Unlike the dissociative fugues, he remembered these episodes in great detail, and expressed mild curiosity about what might happen in the next one. When episodes occurred while he was walking outside, he had more than once fallen onto the ground 'perhaps because I was not paying attention to steps or curbs.' He recalled a few instances of lying on the ground feeling agitated and disoriented, but had been able to get up and resume behaviour normal enough not to attract undue attention.

His medical history was significant for a head injury and traumatic brain injury (TBI) 9 years prior to presentation while a passenger in a taxicab involved in a high speed collision. He suffered multiple facial bone fractures and loss of consciousness for about 1 h. He did not require neurosurgery, but underwent a series of reconstructive facial surgeries over the next several years. During this time he was treated for chronic pain, a mood disorder and behavioural difficulties with a variety of medications including ziprasidone and aripiprazole. He developed mild memory impairment and Parkinsonian signs (see below), which were unaffected by a brief trial of levodopa, and neither progressed nor remitted when his antipsychotic medication was switched to quetiapine.

At presentation to our centre, medications included valproate for mood stabilisation, clonazepam, quetiapine, duloxetine and suboxone. He had a several year history of heroin use to treat chronic face and head pain (following the accident and facial surgeries) not adequately relieved by prescription medication. He would carefully dose himself once a day in the morning so that, paradoxically, he could function in an occupation that required considerable concentration and multi-tasking abilities. At presentation to our centre, he had been heroin free for over 6 months.

INVESTIGATIONS

The patient was fully oriented, with articulate and fluent speech. Immediate memory was 3/3 items, but only 1/3 and 2/3 items at 5 min on two separate examinations.

There were several stigmata of Parkinsonism including a resting 4–6 Hz tremor of the right forearm and hand, mild masked facies and abulia. However, glabellar blink reflex was negative, posture was not stooped, and he could turn around in two to three steps. Neuroimaging performed at the time of the accident and in conjunction with the facial surgeries was not available for review.

During diagnostic video-EEG monitoring he had a typical hallucinatory fugue episode (video 1). The event began at 12:28 a.m with the patient in stage 1b sleep. He awoke and said 'Oh boy.' He sat up in bed, grabbed his handbag from the floor, placed it on the bed and began to look through it. He put a shirt and socks in his handbag and pulled out another shirt from another bag. He looked through this bag, then the first bag and then put a shirt on over his hospital gown. He put papers and a book into his handbag and began rearranging items in the bag. He pulled out a candy box and ate from it. He put his pants and shoes on, got up from the bed and walked to the foot of it, then sat back down and pressed the patient event button. He took off his shoes, pressed the nurse call button and laid back on the bed.

Video 1 The video shows the first 4 m, 40 s of the episode, until the patient is pulling on his pants. It then shows when he is describing the episode to the nurse. The final 3–4 min of the episode are not included due to the large size of the file.

When the nurse entered, he said, 'I just had an episode that lasted about 40 min… I was out with friends…at a bar. So it was a real typical event that I have when I dissociate… I was surprised that I was back in the room all of a sudden. I don't remember coming back and getting plugged in, but I obviously am.' In fact, the episode lasted less than 8 min during which the EEG demonstrated the patient's waking rhythms. When he later watched the video, he was astonished to see his actual behaviours, for which he had absolutely no recollection, and was amazed at the episode's short duration compared to his 40 min hallucinatory 'experience.'

Seventy-two hour of EEG was notable for a mildly slow posterior dominant rhythm, diffuse θ slowing and intermittent bilateral temporal δ slowing, right more than left. These findings, while aetiologically non-specific, can be explained by the TBI and medication effect. There were no epileptiform abnormalities.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of stereotypical episodes that involve altered consciousness or awareness is fairly limited, but the resulting potential treatments are very different. Possible neurologic diagnoses include epilepsy (seizures as well as postictal psychosis), sleep disorder, transient ischemic attack and complicated migraine. Patients with Parkinson's disease can have stereotyped visual hallucinations due to either the disease itself, dopamine agonists, or both. Other physiologic diagnostic options are syncope and presyncope. Psychiatric diagnostic possibilities include psychogenic non-epileptic spells (pseudoseizures), somatoform disorder, factitious disorder and malingering. The final diagnosis in this case – hallucinatory fugue – is extremely unusual, although at least one somewhat similar case has been reported. The differential diagnosis can usually be narrowed fairly quickly with a careful and targeted history, for example, if the spells are diurnal, nocturnal, or both, determining provocative factors and spell duration, associated symptoms such as dizziness, vertigo, headache, automatisms and both cardiovascular and psychiatric risk factors. Some medications and recreational drugs can cause hallucinations, but not hallucinations that are stereotyped and repetitive with well-organised content.

TREATMENT

The patient requested to see the video recorded during his hallucinatory fugue episode. He was positively astonished to see his actual behaviours, for which he was completely amnesic, and also expressed amazement at the episode's short duration compared to the 40–50 min hallucinatory 'experience.' He found watching the video a cathartic experience insofar as it indicated the episodes were real, that is, associated with a documented behavioural alteration, and were not seizures. He was informed of his diagnoses of both dissociative and hallucinatory fugues, and was reassured that he had neither epilepsy nor a psychotic disorder and did not require any additional medications. We explained that his previous brain injury was a definite risk factor for the dissociative fugues, and a presumptive risk factor for the hallucinatory fugues.

OUTCOME AND FOLLOW-UP

A few months after this procedure the patient moved to a less populated and more picturesque part of the country where he could 'avoid the stress and bad weather of NYC.' When contacted by telephone 16 months later he reported that since the move he had only one hallucinatory fugue and no dissociative fugues. He attributed this dramatic improvement to the relative lack of psychosocial stress. He was no longer taking quetiapine or suboxone, and remained on valproate, clonazepam and duloxetine. He reported that watching the video and being told of his diagnoses was '… a welcome experience. All of a sudden I felt validated and (the video-EEG data) affirmed what I always knew – something strange was going on, but very few people believed me including physicians.'

DISCUSSION

This patient had both dissociative fugues and unique hallucinatory fugues. The dissociative fugues began several years after a TBI, a well-known risk factor.[1 2] Limited functional imaging data have associated dissociative fugue with limbic as well as right frontal and temporal hypometabolism.[3 4] Our patient did not have functional imaging, but interictal EEG indicated bitemporal dysfunction, right worse than left, and his short-term memory impairment suggested limbic dysfunction. These findings thus independently support those of the functional imaging studies.

The fugue hallucinations were remarkable in several respects. The hallucination itself was stereotyped, much like a recurring dream, and always involved travel to a destination where he socialised with his 'imaginary friends.' Also like a dream, he vividly remembered the 'experiences' as if they were real. However, he was actually awake and engaging in behaviour distinct from the hallucination, for which he had no memory.

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These episodes are obviously not classic dissociative fugues. They were not dreams, as EEG demonstrated waking rhythms during the ictus and several had began while he was walking outside. However, they often began at home during drowsiness, as occurred during the video-EEG procedure. Similarly, the episodes do not meet diagnostic criteria for any sleep disorder, including somnambulism.[5 6] Seizure and postictal psychosis are excluded on the basis of both semiology and EEG. Although he had some signs of Parkinsonism, these signs developed after the initiation of antipsychotic treatment and did not respond to levodopa, suggesting they were drug induced. In addition, visual hallucinations as a symptom of idiopathic Parkinson's disease occur along with multiple motor signs, and rarely if ever early in the disease course. An adverse drug reaction is excluded because drugs do not cause internally coherent and stereotyped hallucinations. Somatoform disorder is excluded because his symptoms were not physical and did not suggest a general medical condition. Factitious disorder and malingering seem highly unlikely because, among other things, the patient was curious to know his diagnosis, cooperated fully with the evaluation, and was satisfied with both the final diagnosis of hallucinatory fugue and the absence of any specific treatment.

Fugue hallucination shares features of dissociative fugue, somnambulism, dream and thought disorder, yet clearly is none of these in isolation. Many of these qualities are shared by one prior case report, although substantive differences remain. In that case, a 31-year-old woman would begin to somnambulate 60–90 min after falling asleep. She would often leave her house, drive her car several miles, exit the car and resume walking. While engaged in these activities she had a stereotypical 'nightmare' that re-enacted a brutal and sadistic rape she had suffered at age 10.[7]

We imagine that our patient's prior TBI is causally related to both the dissociative and hallucinatory fugues. In particular, the axonal damage that can occur with such injuries may have disrupted the anatomical and functional connectivity between centres of arousal, memory, and motor control, which we propose as a risk factor for this phenomenon. After the accident he did develop mild short-term memory dysfunction and Parkinsonism, suggesting injury to limbic and motor control circuitry. Such alterations in normal brain function may have been magnified by the patient's psychoactive medications, although we do not believe his symptomatology can be explained by medications alone. Indeed, TBI is the patient's only known risk for the focal bitemporal slowing seen on the EEG. While the incidence of cases like ours and that of the woman described above must be very low, it is important not to dismiss such symptomatology as factitious, and to provide these patients an appropriate diagnostic evaluation. Whether 'fugue hallucination' will enter the diagnostic armamentarium will depend on the elucidation of additional cases.

Learning points

- Hallucinatory or subjective fugue is an authentic diagnosis that shares features of but is distinct from dissociative fugue, somnambulism, dream and thought disorder.
- TBI may be a risk factor for hallucinatory fugue, as it is for dissociative fugue.
- The value of the patient history should never be underestimated, even when it does not fit into a typical or 'textbook' scenario.

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Competing interests None.

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