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Epilepsy & Behavior 7 (2005) 311–315
Epilepsy & Behavior
www.elsevier.com/locate/yebeh

Case Report

Episodic depersonalization in focal epilepsy

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Received 5 April 2005; revised 25 May 2005; accepted 27 May 2005
Available online 25 July 2005

Abstract

In this report a patient with episodic depersonalization is described. As the depersonalization episodes had been attributed to partial seizures, this patient was treated with antiepileptic medication. However, clinical evaluation with long-term video/EEG revealed no evidence of seizure activity during the depersonalization episodes. On the other hand, further evaluation revealed findings that are frequently associated with focal epilepsy. In addition to episodic depersonalization, this patient had secondary generalized seizures. The relationship between episodic depersonalization, temporal lobe pathology, and epilepsy is discussed against the background of this case.

Keywords: Depersonalization; Temporal lobe; Epilepsy; Limbic system

1. Introduction

Depersonalization is a peculiar alteration in the perception of the self; that is, the person feels detached from her- or himself, feels like an outside observer of her or his own body or mental processes, feels like an automaton, or feels she or he is in a dream. Closely related to depersonalization is derealization—an alteration in the perception or experience of the outer world as unreal. Depersonalization occurs as a primary condition in its own right—the so-called depersonalization disorder [1–4]—and as secondary depersonalization in other psychiatric disorders, for example, anxiety disorders and major depression. Depersonalization is also encountered in neurological diseases such as epilepsy, migraine, and traumatic brain injury [5]. To account for the similar narratives of depersonalization experiences in a variety of different psychiatric disorders and neurological diseases, Mayer-Gross proposed that depersonalization results from activation of a specific biologically preformed response pattern by rather nonspecific factors [6]. A particular neurological etiology of depersonalization, however, has been linked with depersonalization from the early days of its scientific study: the resemblance of the phenomenology of depersonalization to experiential symptoms of temporal lobe epilepsy (TLE). Starting from early accounts of the depersonalization syndrome, a relationship with (temporal lobe) epilepsy has been proposed [7,8]. In TLE, alteration of consciousness occurs as part of the epileptic seizure: Closely related to depersonalization is *jamais vu* (the opposite of *déjá vu*), an ictal disturbance of the feeling of familiarity. The ''dreamy state'' is a mental condition in temporal lobe seizures characterized by an altered sense of reality that may affect the experience and perception of the internal and external environment. ''Dreamy states'' may comprise recollections in the form of déjà vu, unfamiliarity–unreality (jamais vu), forced thinking, and hallucination-like recollection of the past (panoramic vision) [9]. Seizures with these psychic

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1525-5050/$ - see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.yebeh.2005.05.023

symptoms most often arise from the temporomesial limbic structures and, more rarely, temporolateral neocortical areas; an extratemporal origin is uncommon [10–12]. TLE is therefore frequently considered a neurological "model disorder" for the study of the neurobiology of depersonalization [13]. The phenomenological resemblance of the ictal semiology of temporal lobe seizures to depersonalization of other etiology led to the suggestion that temporal lobe mechanisms are involved in the pathogenesis of depersonalization [14,15]. The main lines of argument favoring the importance of the temporolimbic system for the pathogenesis of the depersonalization syndrome are the depersonalization-like semiology of temporomesial seizures as described above and the results of electric stimulation of the temporal lobe in the context of epilepsy surgery: Depersonalization-like unreality feelings have been reported after electrical stimulation of the cortex, mainly the temporal neocortex of the subdominant hemisphere, in the pioneering work of Penfield et al. [16,17]. More recently, electrostimulation studies have stressed the contribution of temporolimbic structures to the generation of complex experiential phenomena [18]. (However, remarkably, in Gloor's study [19] on the contribution of temporolimbic structures to the complex experiential phenomena of TLE, no depersonalization-like experience was noted.) The phenomenological resemblance of depersonalization to ictal temporal lobe semiology led Harper and Roth [15] to compare the signs and symptoms of patients with TLE and depersonalization as part of the so-called "phobic anxiety depersonalization syndrome." A variety of symptoms like déjà vu, metamorphosis, and anxiety attacks were common in both diagnostic groups. Because of this phenomenological overlap, these authors postulated a contribution by temporal lobe dysfunction to the pathogenesis of episodic depersonalization.

The DSM-IV [1] specifically mentions TLE as a medical factor causing depersonalization and, therefore, as a differential diagnosis of (primary) depersonalization disorder. The intricate relationship between temporal lobe dysfunction, focal epilepsy, and depersonalization is illustrated in the following case history.

2. Case history

2.1. History of the clinical syndrome

Miss K, a 39-year-old clerk with episodic depersonalization since the age of 30, complained of altered states of consciousness lasting several days during which she felt "strangely altered" and "unreal." These states occurred about once a week. She experienced her environment as "far away" or "covered by an invisible veil." During such states she felt her body was "weightless, like floating in the air." Sometimes she felt completely "out of her body". Her thoughts and movements were "the thoughts and movements of a robot." During these episodes of depersonalization she also experienced strange tastes and smells (e.g., like the smell of a cigarette) and nausea. Objects appeared closer or more remote than in reality. She reported difficulties in effortful mental activities requiring concentration during these depersonalization states. She claimed full awareness and memory for all events during a depersonalization episode. The episodes started at the time of a move that separated her from close friends and relatives. The depersonalization episodes could be triggered by stress and relaxation and, sometimes, by strange smells. The patient believed that she had similar symptoms for about a week when she was 6 years old, but was now unable to recall the details. Her depersonalization experiences were explored with the Cambridge Depersonalization Scale [ad hoc translation in German language, the patient yielded a score of 90 (cutoff 70)], which is designed to capture the frequency and duration of depersonalization symptoms over the last 6 months. It is a self-rated questionnaire based on items derived from the literature on the descriptive psychopathology of depersonalization. A high score indicates that the patient has a rich phenomenology of depersonalization experiences [20].

In addition to episodic depersonalization, the patient experienced panic attacks lasting about half an hour with mainly vegetative symptoms such as palpitation and dyspnea. The patient could clearly discriminate the panic attacks and the episodes of depersonalization. About 5 years earlier, she had suffered from agoraphobia, which made it impossible for her to leave her home for about 8 months. From the beginning of the depersonalization episodes, this patient was treated with antiepileptic drugs because it was assumed that these episodes of altered state of consciousness were due to (simple) focal seizures. However, treatment with valproic acid, lamotrigine, and carbamazepine did not alleviate the depersonalization. About 6 years into the disorder, antiepileptic medication was rapidly reduced, resulting in two generalized tonic–clonic seizures, and antiepileptic medication was reintroduced. The patient denied other signs and symptoms of possible epileptic seizures beyond the episodic depersonalization and the two grand mal seizures 6 years after onset of the depersonalization episodes.

At the time of admission, psychopathological examination of Miss K. was otherwise unremarkable. Antiepileptic medication was continued and a selective serotonin reuptake inhibitor (citalopram) was introduced. The patient started outpatient psychotherapy for depersonalization and panic attacks. Secondary generalized epileptic seizures were controlled by antiepilep-