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Vivid recollection of a past traumatic event: a rare manifestation of temporal lobe seizures

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ABSTRACT – Vivid recollection of a particular past memory is a rare ictal phenomenon in temporal lobe epilepsy. When the memory is traumatic, the presentation may mimic post-traumatic stress disorder (PTSD). We report a woman with temporal lobe epilepsy who was misdiagnosed with post-traumatic stress disorder for two years before she had a suspected generalised seizure; video-EEG monitoring confirmed that her episodes were epileptic. Adequate treatment with an antiepileptic medication resulted in total resolution of the episodes.

Key words: temporal lobe epilepsy, temporal lobe seizures, déjà vu, déjà vécu, post-traumatic stress disorder

Hughlings Jackson (Jackson and Colman, 1898), as well as later authors (Bancaud *et al*., 1994; Penfield, 1955; Vignal *et al*., 2007), reported that temporal lobe epilepsy can present as a "*dreamy state*", consisting of vivid recollections of past events and *déjà vu* (or *déjà vécu*), the sense of having previously experienced the same situation. Different investigators observed that such phenomena could be elicited by electrically stimulating the lateral temporal neocortex (Penfield, 1955), the mesial temporal structures (Vignal *et al*., 2007), or the fornix and hypothalamus (Hamani *et al*., 2008). This paper reports a woman with a two-year history of episodes of vivid recollection of a past traumatic memory associated with panic; the localisation of this rare ictal phenomenon is discussed.

Case report

A 34-year-old, right-handed woman presented with a two-year history of recurrent paroxysmal episodes, consisting of a vivid recollection of a traumatic childhood memory related to sexual abuse by a male adult relative. All of her episodes occurred in bed as she started to get drowsy, and she had difficulty falling asleep. The episodes were associated with panic and occasional visual hallucination of the traumatic

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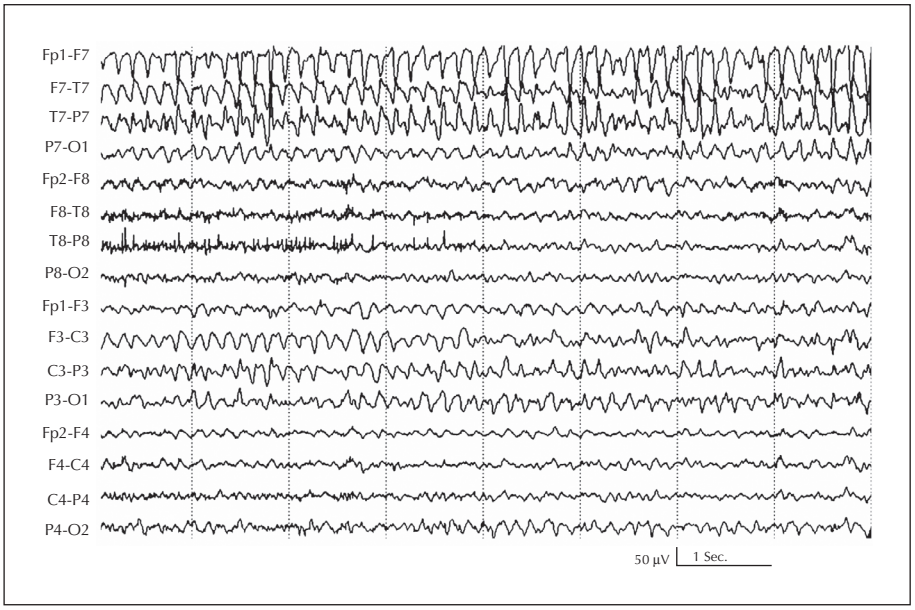

Figure 1. This left temporal ictal discharge was seen during the period when the patient appeared to have panic and confusion before secondary generalisation. It consists of a theta-range, rhythmic, spike activity, typical of mesial temporal lobe seizures.

event in the same setting in which it actually occurred. On one occasion, she experienced a strange smell that she described as "*as if it is going to rain*". The episodes would ultimately be followed by sleep.

The frequency of her episodes increased from once a month to one or two per week after she was laid off from work. Her primary care physician suspected delayed-onset post-traumatic stress disorder (PTSD), and referred her to a psychiatrist, but she declined the psychiatric evaluation. The patient also reported severe anxiety that she attributed to her being a single mother with financial difficulties.

Two years after the onset of her episodes, she awakened with sore muscles and her 10-year-old son told her that she was shaking violently in the preceding night. At that stage, epilepsy was suspected and she had an EEG and brain MRI, which were normal. Levetiracetam was started at the dose of 500 mg twice a day, but she had variable compliance with this medication which she justified based on the diagnostic uncertainty of her condition. Her episodes of recollection of the past event persisted at a frequency of at least once a week, and were extremely distressing as she thought she was "*going crazy*". Two additional EEGs failed to reveal any abnormalities.

The patient was then referred to the Neurology Clinic, where she was found to have a completely normal physical and neurological examination with a Folstein Mini-mental Status Examination score of 30/30. She was referred to the Epilepsy Monitoring Unit (EMU) for characterisation of her episodes. In the EMU, she had left temporal spikes with phase reversals over F7, or spikes that were equipotential over F7 and T7. These were occasional, not exceeding 10-15 per 24 hours. Two episodes were captured that started with arousal from sleep followed by panic and apparent confusion. By the time the EMU staff came to assess her, she was unresponsive with right hand posturing. Her eyes then deviated to the right before secondary generalisation. Her ictal EEG consisted of medium-voltage, left temporal, semirhythmic activity at 5-6 Hz, which soon evolved into a left temporal, 6-7 Hz, high-voltage, organised sharp wave discharge (*figure 1*), and then generalised. Postictally, the EEG showed diffuse slowing. The patient later reported that these episodes were associated with her habitual aura of vividly recollecting her past traumatic memory. She was educated about her seizures and levetiracetam dosage was increased to 2,000 mg/day in two divided doses. Her compliance improved and she has been seizure-free for six years with no psychiatric complaints, ruling out concomitant psychiatric disorders.

Discussion

We describe a patient with episodes resembling PTSD that proved to be due to seizures of presumed temporal lobe origin. While certain features of this patient's epilepsy were consistent with mesial temporal localisation, others were not. For example, whereas in mesial TLE interictal epileptiform discharges are often commonly seen, our patient had three normal 20-minute

EEGs, and only occasional spikes during her EMU stay. In addition, frequent secondary generalisation is more common in neocortical than in mesial temporal seizures. However, unlike neocortical seizures, her secondary generalisation was not rapid, but followed a prolonged partial seizure. This prolonged duration of her partial seizure probably explains the patient's recollection of the aura despite seizure generalisation. Moreover, her aura and interictal and ictal EEG discharges were consistent with mesial TLE.

The earliest descriptions of experiential phenomena as manifestations of epilepsy date back to Abu al-Qasim Al-Zahrawi (936-1013), an Arab physician who lived in Córdoba and is considered the father of surgery. In his famous book, Kitab al-Tasrif, a thirty-volume encyclopedia of medicine, Abu al-Qasim describes two individuals with epilepsy whose seizures started with experiential hallucinations of a person approaching them (Penfield, 1955; Spink and Lewis, 1973). J Hughlings Jackson was the first to describe such seizures in great detail, and correctly localised them to the mesial temporal lobe (Jackson and Colman, 1898). Jackson termed such phenomena as "*dreamy states*", which encompassed vivid hallucinations of scenes, reliving true past experiences, or feeling the ability to predict what is going to happen (see also Editorial Comment from Jean Aicardi, 2001). He also associated these phenomena with oral or olfactory hallucinations and with oral automatisms.

For Wilder Penfield, *déjà vu* represented a sense of familiarity for current events as well as recalled events. He used the term "*experiential hallucinations*" to describe memory flashbacks and found that they could be elicited by electrical stimulation of the lateral temporal neocortex (Penfield, 1955). However, Bancaud *et al*. found that lateral temporal stimulation was less likely to elicit a "*dreamy state*" than medial temporal stimulation, and concluded that the "*dreamy state*" resulted from activation of a neuronal network that included both medial and lateral temporal regions (Bancaud *et al*., 1994). A later study reported *déjà vécu* and visual hallucinations of a scene resulting from spontaneous seizures, as well as from electrical stimulation of the amygdala, hippocampus, and parahippocampal gyrus, without temporal neocortical involvement (Vignal *et al*., 2007). Indeed, neocortical propagation of the ictal discharge appeared to prevent the "*dreamy state*". Vignal's report included patients with seizures manifesting as reliving a personal memory that could be recent or remote, with such memories often being consistent across seizures in the same patient. Hamani *et al*. applied deep brain stimulation to bilateral hypothalamic and descending fornices, which evoked detailed autobiographical memories, but without increasing familiarity-based recognition (Hamani *et al*., 2008). The authors concluded that the stimulation activated the hippocampus as suggested by electroencephalographic source localisation that showed activity in mesial temporal lobe structures.

Although trauma is a risk factor for both epilepsy and PTSD, our case is unique in that the seizures themselves had manifestations that resembled those of PTSD. As mentioned above, other authors reported patients with recollection of particular past events occurring during seizures or electrical stimulation of the brain, but the memories were not traumatic. Features of our patient's seizure symptomatology that are consistent with PTSD include flashbacks of reliving the original trauma and increased arousal, among other symptoms. PTSD has three sub-forms: acute, chronic, and delayed-onset. Delayed-onset PTSD has been described since PTSD became a diagnostic entity in Diagnostic and Statistical Manual of Mental Disorders (DSM)-III. Later, delayed-onset PTSD was defined in DSM-IV-TR as occurring at least six months after the traumatic event, although PTSD onsets delayed by as long as 30 years have been described by Korean War veterans (Andrews *et al*., 2007).

Indeed, the similarities between PTSD and some temporal lobe seizures are not only semiological, but also at the level of the neural substrates of these conditions. The amygdala and hippocampus have altered function in PTSD. For example, a number of studies have shown bilaterally smaller hippocampal volumes in individuals with PTSD compared with controls (Woon and Hedges, 2011; Gurvits *et al*., 1996). Moreover, neuroimaging research has found hyperactivation in emotion-related regions, including the amygdala, in individuals with PTSD (Sripada *et al*., 2012). At the molecular level, some authors have found that hippocampal insulin-growth factor 2 (IGF2) facilitates fear extinction and insulin growth factor binding protein 7 (IGFBP7) impairs fear extinction, suggesting that therapies that enhance IGF2 signalling can probably help disorders like PTSD (Agis-Balboa *et al*., 2011).

In summary, our patient had a rare presentation of temporal lobe epilepsy that was misdiagnosed as PTSD for years. Although different pathophysiologically, epilepsy and PTSD share neuroanatomical substrates that are centred around the mesial temporal structures. In this reported case, antiepileptic medication treatment resulted in complete resolution of the episodes, ruling out concomitant psychiatric conditions. □

Disclosures.
The author has no disclosures related to the current work.

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