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Post-traumatic epilepsy with isolated memory flashbacks

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ABSTRACT – Some traumatic events can cause both post-traumatic stress disorder and epileptic seizures. We report the case of a woman who experienced a severe head injury which subsequently led to the development of paroxysmal episodes of isolated memory flashbacks related to the injury. Detailed analysis of her symptoms along with video-EEG telemetry recordings was helpful to distinguish between these two conditions. [*Published with video sequences*]

Key words: memory flashback, post-traumatic stress disorder

Reminiscence of past traumatic events in a "flashback" manner (*i.e.* intrusive memories) has been commonly recognised as a symptom of post-traumatic stress disorder (PTSD) (American Psychiatric Association, 2000). However, other potential causes, such as epileptic seizures, may be overlooked. Some epileptic seizures may manifest with experiential hallucinations of past experience (Gloor, 1990; Vignal *et al*., 2007).

We report a patient with post-traumatic epilepsy whose seizures consisted of isolated memory flashbacks. This case illustrates that a detailed clinical assessment and video-EEG telemetry may be helpful to differentiate between epileptic seizures and PTSD.

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Case study

A 43-year-old, right-handed woman was involved in a motor vehicle collision (MVC) one and a half years ago. At the time, she presented with an initial Glasgow Coma Scale (GCS) of 11/15 (E3V3M5). She was perseverative and confused upon her arrival to the hospital. She was unable to move her left arm and had a left-sided, upper motor neuron-type facial weakness. She was found to have an acute traumatic subarachnoid haemorrhage, diffuse axonal injury, minimal right hemothorax, and multiple fractures including the transverse process of C7 vertebra and left acetabular bone. She was amnestic to the events surrounding the MVC, one week before and one week afterwards. Approximately within a month after her MVC, she developed stereotyped paroxysmal episodes of perceiving "*awful feelings*" that were, at times, associated with flashback memories related to her MVC. These episodes were consistently associated with migraine-like headaches. During such episodes, she would

feel hot, flushed, sweaty, and "*awful*". The latter was poorly defined but was characterised by the perception of bad experiences from her MVC, such as intubation or insertion of a chest tube while she was in the intensive care unit or during her hospitalisation at a rehabilitation centre. She would recognise these episodes as unreal thoughts without seeing or hearing anything related to the events. The episodes each lasted for a few minutes.

Initially, she was treated with valproic acid but this did not help relieve her episodes or headache. Subsequently, valproic acid was discontinued and topiramate, at 150 mg daily, was started; this provided relief for her headaches and reduced the number of episodes.

Her neurological examination at the time of her admission for prolonged scalp video-EEG telemetry was remarkable for generalised hyperreflexia, which was more pronounced on the left side. Her cranial MRI showed diffuse axonal injury with scattered tiny spots of increased signal intensity on T2-weighted and Fluid Attenuated Inversion Recovery (FLAIR) sequences, which were most prominent in the right subcortical cerebral white matter and haemosiderin deposits in the left superior frontal region and splenium of the corpus callosum (*figure 1*). No abnormality was seen in the temporal lobes, particularly the hippocampi.

Routine outpatient EEGs revealed a few interictal epileptiform discharges (IEDs) in the right temporal regions and bilateral intermittent delta slow in the temporal areas. Scalp video-EEG telemetry revealed infrequent sequential IEDs in the right hemisphere, maximum at F8-T4-M2, with minimal spread to F4-C4. Interestingly, no focal slow wave was seen with topiramate, but was evident in the bilateral temporal regions after a reduction in topiramate, which resulted in a cluster of 19 clinical seizures.

The seizures were semiologically and electrophysiologically similar, but varied in duration. During these seizures, she reported feeling hot, flushed, and lightheaded, with headache and "*awful feelings*", but without motor manifestations. On two occasions, she stated that "*I am having bad thoughts*" referring to flashback memories of being in the rehabilitation hospital, which she was normally amnestic to (*see video sequence*). She could not describe these awful feelings in further detail, other than being "*unpleasant*". Her recorded seizures varied in duration from 40 to 220 seconds. She was unresponsive during the longer episodes.

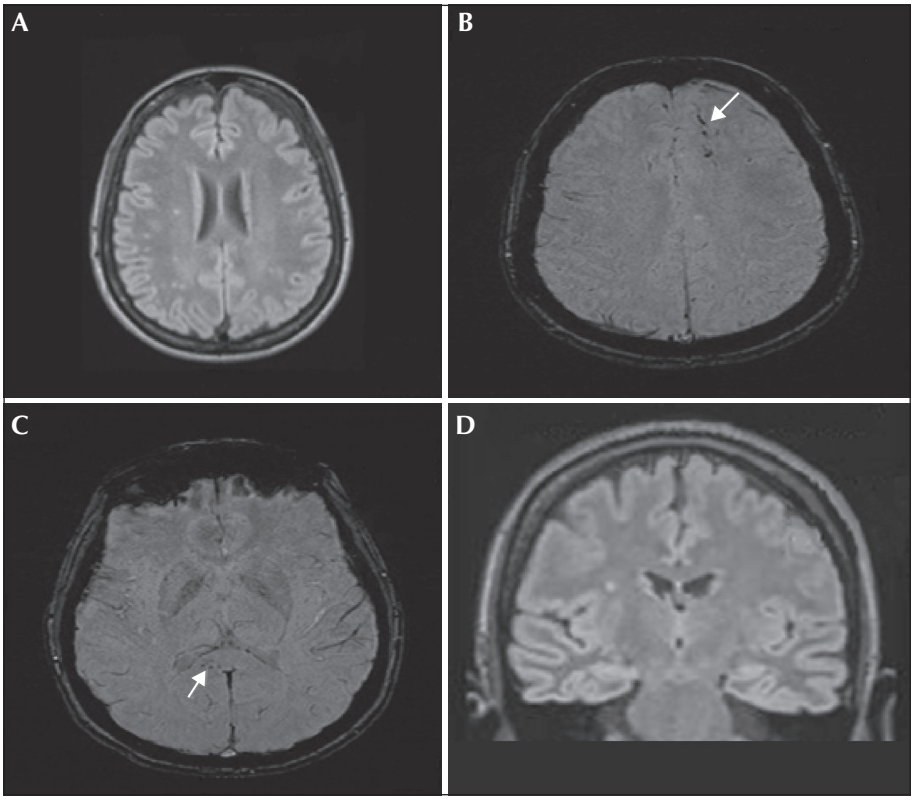

Figure 1. (A) Fluid Attenuated Inversion Recovery (FLAIR) sequence showing scattered tiny spots of increased signal intensity, observed mainly in right subcortical white matter. (B and C) Gradient echo sequence revealed evidence of diffuse axonal injury, showing haemosiderin deposits noted in the left superior frontal region and right-sided splenium, respectively. (D) No abnormalities were detected in bilateral temporal regions including bilateral hippocampi.

Electrographically, her seizures started with a build-up of subtle repetitive small-amplitude, 2-Hz spikes at F8-M2 (clearly discernible in our montage containing A1-A2 and F7-F8 derivations in which A1 referred to M1 [mandibular electrode] and A2 referred to M2). This was followed by diffuse, semi-rhythmic, 5-6-Hz waves with right hemispheric predominance for 30 seconds. For the longer seizures, they would be followed by 0.5-0.8-Hz, higher amplitude, repetitive spikes at M2-F8, with minimal spread to T4. These were intermingled with right hemispheric, semi-rhythmic, 4-5-Hz waves and generalised, low-amplitude, fast activity with right-sided predominance for another 90-150 seconds (*figure 2*).

Discussion

We herein report a case with temporal lobe seizures, as evidenced by video-EEG, manifesting with memory flashbacks. The immediate occurrence of these episodes after the patient's severe head injury and lack of typical characteristics of temporal lobe seizures led, for several months, to the misinterpretation of PTSD.

The DSM-IV-TR (American Psychiatric Association, 2000) includes the term "*dissociative flashback episodes*" as one of the diagnostic features of PTSD. The criteria emphasize a response which includes avoidance behaviour associated with symptoms of increased arousal, *e.g.* difficultly falling asleep, exaggerated startle response, and functional disturbance. The re-experienced event may include recurrent and intrusive distressing recollections of the event, such as images, thoughts, perceptions, or recurrent distressing dreams of the event. Among the clusters of symptoms required for the diagnosis of PTSD, avoidance or numbing is reported to be a strong determinant or marker (Ehlers *et al*., 1998; North *et al*., 2005). The present case did not present with any symptoms applicable to this cluster. During these episodes, the patient did not express fear nor did she exhibit avoidance behaviour during or in between her episodes.

"*Flashbacks*" have been reported with epileptic seizures (Gloor *et al*., 1982; Bancaud *et al*., 1994; Barbeau *et al*., 2005; Vignal *et al*., 2007). However, these previous cases were more consistent with "*dreamy states*" as they described recollection of visual scenes or fragments of scenes. In these cases, the patients described the visual scenes as if they were spectators (Vignal *et al*., 2007). Our patient, on the other hand, did not see or hear anything related to her MVC and her episodes were based only on her feelings. Vignal *et al*. (2007) proposed the semiological continuity between the phenomenon of *déjà vu* and such complex visual hallucinations, given the reciprocal connections between the amygdala/hippocampus and other cortical areas, including the primary and association visual and auditory cortices.

The International League Against Epilepsy (ILAE) Task Force on Classification and Terminology classified "*experiential aura*" as affective, mnemonic, hallucinatory, and illusive perceptions (Blume *et al*., 2001). Penfield and Perot distinguished "*dreamy state*" as either interpretive response or seizure and experiential hallucination (Penfield and Perot, 1963). The former referred to perceptual illusion (visual and auditory), *déjà vu*, and emotion, in the sense of false interpretation of the present. The latter was memory flashback, noted as a sudden re-experience of the past. Most of these were elicited from the temporal neocortex. Gloor (1990) used the term "*experiential*" in the broader sense, to encompass perceptual, mnemonic, and affective features. The mnemonic phenomena referred to the actual recall of a past event or situation (flashback) and a feeling of recognition, familiarity, or reminiscence. Experiential responses were elicited by stimulation of the limbic structures of the temporal lobe, for which the amygdala showed the highest incidence of positive responses, compared to the temporal neocortex (Gloor, 1990). This is in line with a stereoelectroencephalographic (SEEG) study which showed a role for the amygdala and hippocampus in recall of recent and distant memories (Vignal *et al*., 2007). Bancaud *et al*. (1994) demonstrated that both mesial and lateral temporal neocortices are involved in this phenomenon. Bartolomei *et al*. (2004) studied the role of the anterior subhippocampal structures (*i.e*. rhinal cortices) in eliciting *déjà vu* sensation. Based on their study, they suggested that *déjà vu* phenomenon is likely triggered in the anterior subhippocampal structures and requires transient functional coupling with other mesial temporal structures, which eventually results in recollection (Bartolomei *et al*., 2004; Guedj *et al*., 2010; Bartolomei *et al*., 2012). This phenomenon reflects positive expression of the function of temporal lobe structures. Once stimulated or elicited seizures occur, the epileptic discharges may strengthen interconnectivity of the neurons among these structures (Gloor, 1990; Vignal *et al*., 2007).

With regards to the underlying pathophysiology of PTSD, aside from psychological factors and personality characteristics that contribute to the development of PTSD, functional imaging and neurochemical studies have implicated a role of the amygdala, mesial prefrontal cortex (PFC), hippocampus, epinephrine, and cortisol. Acute stress can cause an increase in epinephrine secretion, which facilitates the encoding of emotionally arousing information in the amygdala, resulting in some form of implicit memory. A high concentration of epinephrine impairs PFC function

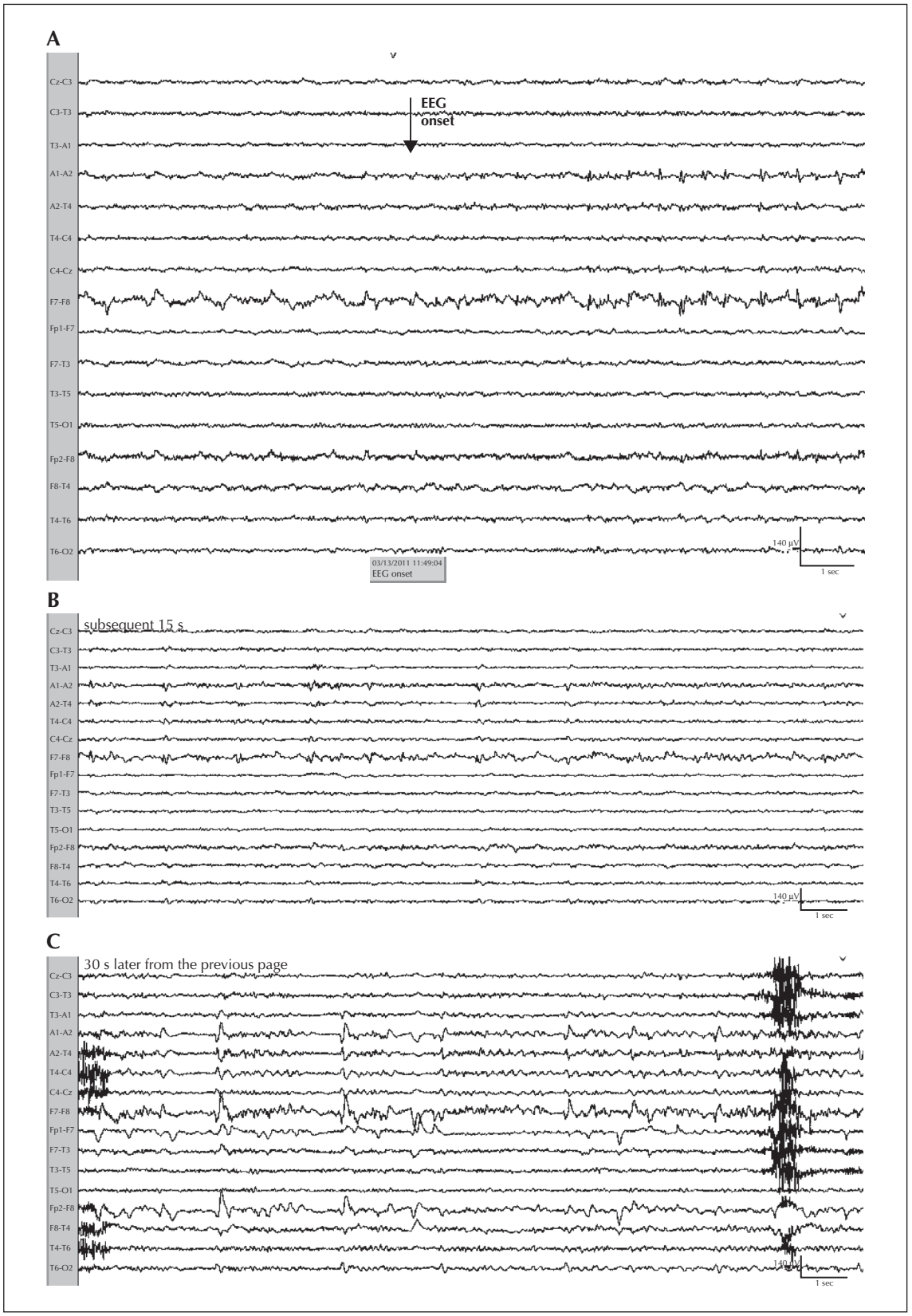

Figure 2. (A) Initial EEG onset, starting with a build-up of subtle, repetitive, low-amplitude spikes at 2 Hz, observed at F8-M2 (clearly discernible on special montage containing A1-A2 and F7-F8 derivations; A1 referred to M1 [mandibular electrode] and A2 referred to M2). (B and C) Subsequent ictal EEG changes.

through α-1 adrenergic receptor activation. Given the inhibitory effects of PFC on the amygdala *via* its reciprocal connections, PFC dysfunction could be related to the inability of PTSD patients to inhibit intrusive memories (Elzinga and Bremner, 2002). This would thus implicate disinhibition of the mesial temporal structures, particularly the amygdala, in PTSD, in contrast to their direct stimulation during epileptic seizures or electrical stimulation.

Conclusion

In summary, prolonged scalp video-EEG recordings confirmed that our patient's episodes of isolated memory flashbacks were epileptic seizures, arising from the right temporal region. An absence of typical features of temporal lobe seizures (altered awareness, automatisms, or typical auras) made it difficult to differentiate between epileptic seizures and PTSD on clinical grounds. A shorter duration of attacks, associated headache or autonomic symptoms, and absence of the avoidance/numbing symptom cluster may serve as possible clues for epileptic seizures in patients with isolated memory flashbacks, which can be confirmed by video-EEG telemetry. □

Disclosures.
None of the authors has any conflict of interest or financial support to disclose.

Legend for video sequence

Video recording of a seizure in which the patient described "head pounding" and "awful thought" at approximately two and three minutes after EEG offset. Of note, the initial part of ictal EEG was obscured by muscle and movement artefacts. The first clearly observed ictal EEG change was semi-rhythmic discharge of 6-7 Hz observed at F8-M2 (A2 referred to M2), which was noted at approximately 30 seconds after the beginning of this recording.

Key words for video research on www.epilepticdisorders.com
Syndrome: focal non-idiopathic temporal (tle)
Etiology: head trauma
Phenomenology: hallucinations (visual); nonepileptic paroxysmal event
Localization: temporal lobe (right)

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