

# Adversive seizures associated with periodic lateralised epileptiform discharges (PLEDs) after left orbital contusion

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Received March 1, 2011; Accepted July 18, 2012

**ABSTRACT** – We report a patient who presented with adversive seizures associated with periodic lateralised epileptiform discharges (PLEDs), a month after head trauma. The PLEDs predominantly involving the left frontal contacts became more frequent at the onset of adversive seizures during EEG. Brain MRI demonstrated a contusion scar in the left orbital cortex with reduced diffusion, not only around this orbital lesion but also in the ipsilateral anteromedial thalamus. Single photon emission computed tomography revealed focal cerebral hyperperfusion in the left medial orbitofrontal region, basal ganglia, and thalamus. The abnormal metabolism involving the thalamus and striatum could be associated with the ipsilateral orbital contusion and might have been caused by cortical-subcortical, trans-synaptic hyperactivity. Further studies are warranted to determine the role of subcortical structures in the generation of PLEDs and adversive seizures. [*Published with video sequences*]

**Key words:** adversive seizure, periodic lateralised epileptiform discharge, thalamus, contusion



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## Case Report

A 60-year-old man sustained trauma to the head during an accidental fall after heavy drinking. He was admitted to the emergency room of our hospital, but was soon discharged after a neurological exam-

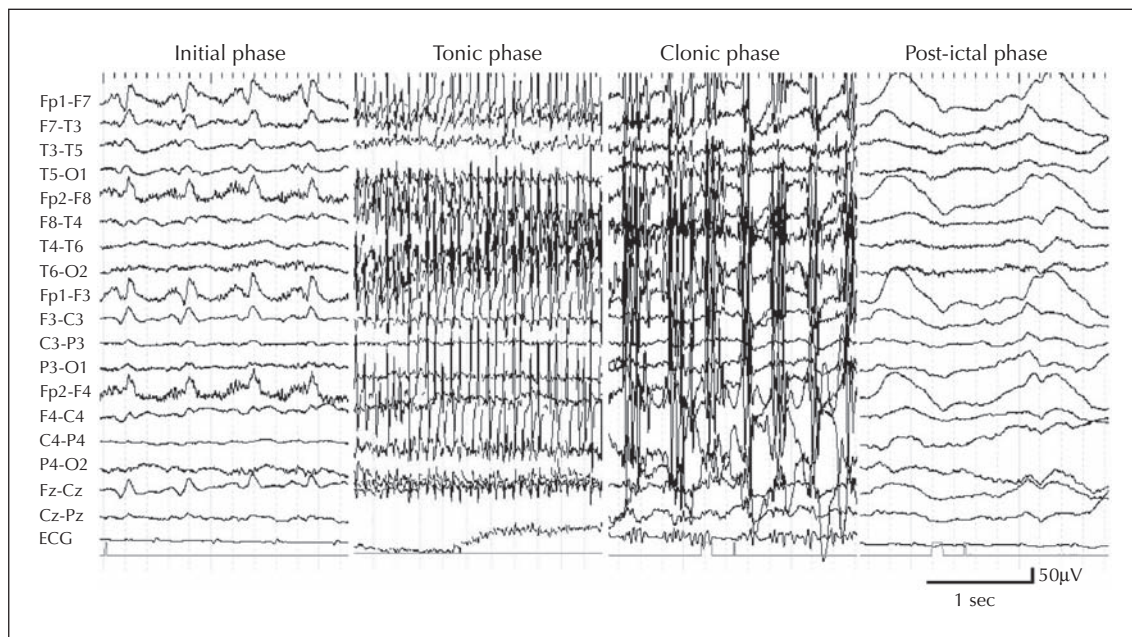
ination proved normal. A month after the head injury, he experienced seizures and an acute deterioration of consciousness. Upon arrival to the emergency room, he was unconscious and had repetitive seizures despite intravenous administration of diazepam. The seizures began

with tonic neck rotation and eye deviation to the right, accompanied by tonic flexion of the right arm (see *video sequence*). This was followed by a clonic seizure involving the right side of the mouth and right arm lasting 10 to 20 seconds, which then gradually subsided. Real-time video electroencephalography (EEG) showed that periodic lateralised epileptiform discharges (PLEDs), predominantly seen in the left frontal contacts (F3, F7), had started about 20 seconds before the seizures and were subsequently replaced by left frontal spiking. During the clonic phase of the clinical seizure, the EEG is marred by artefact. Postictally, the EEG showed (see *video sequence*) severe delta activities (Grand'Maison *et al.*, 1991). The waveform of the PLEDs tended to be relatively, but not completely, uniform and monomorphous. The continuous epileptic spike waves gradually became intermittent, while the tonic seizure progressed to a clonic phase (*figure 1*). Immediately after the disappearance of seizures, PLEDs were not apparent. Brain MRI revealed a traumatic contusion in the left orbital region (*figures 2A and B*). Diffusion-weighted imaging showed high signal intensity, not only in the left orbital gyrus but also in the left anteromedial thalamic regions (*figures 2C and D*). Single photon emission computed tomography demonstrated an increased uptake of  $^{99m}\text{Tc}$ -ethyl cysteinate dimer (ECD) in the left frontal cortices, left anterior striatum, and regions of the thalamus (*figures 2E and F*). The patient was treated

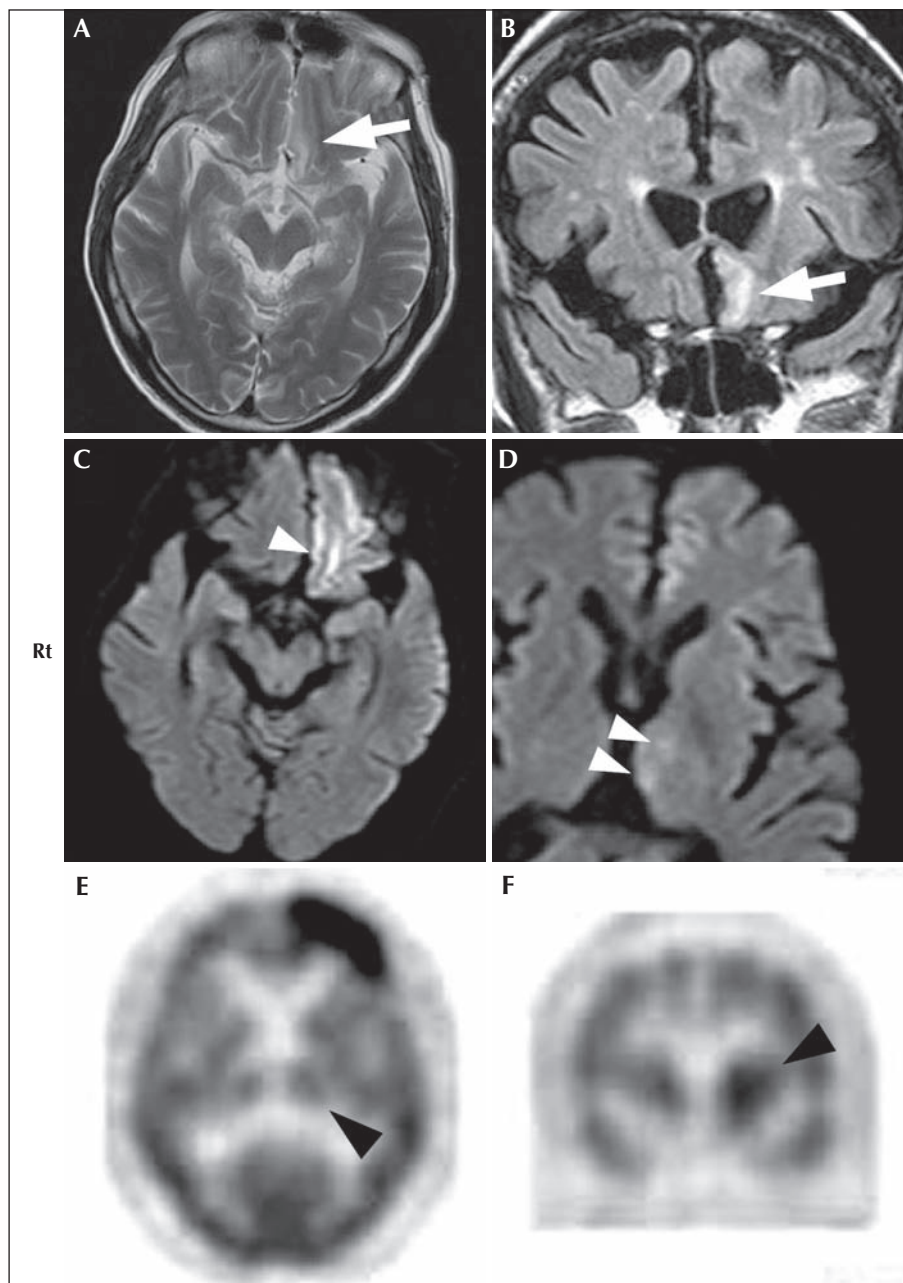
with continued intravenous administration of midazolam, which was successful in controlling the attacks. After drowsiness and right-sided Todd's hemiparesis lasting several days, the patient gradually recovered. An oral antiepileptic agent (carbamazepine) was prescribed and epileptic attacks did not recur thereafter.

## Discussion

An adversive seizure is a form of partial seizure characterised by neck rotation and conjugate gaze deviation in a direction contralateral to the responsible epileptic focus. A medial frontal lesion is regarded as the major focus (Ropper and Samuels, 2009). In the present patient, hypermetabolism in the striatum and thalamus was possibly a result of ipsilateral orbital contusion, which might be a consequence of trans-synaptic hyperactivity transmitted *via* the excitatory corticothalamic and corticostriatal projections (Afifi and Bergman, 1998). While the mechanisms remain to be fully elucidated, it is presumed that an irritable thalamic lesion has an important role in the generation of subcortical PLEDs (Katramados *et al.*, 2009). Subcortical lesions, including those of the thalamus, are known to be involved in monomorphous PLEDs, thus corroborating our notion that the PLEDs in the present patient were probably related to thalamic insufficiency



**Figure 1.** EEG changes seen over the left frontal region in the initial epileptic phase with the appearance of PLEDs. Polyspike waves were seen predominantly at left frontal contacts during the tonic seizure phase.



**Figure 2.** The contusion scar is apparent (arrows) in the left orbitofrontal region on T2WI (A) and FLAIR (B) images. Note high signal intensities (arrows) in the left medial frontal regions (C) and thalamus (D) on DWI, and increased uptake (arrows) in the left striatothalamic regions (E and F).

(Chatrian *et al.*, 1964; Kalamangalam *et al.*, 2007). This thalamic-medial frontal hypermetabolism could be a basis for the origin of the adverse seizure (neck rotation and eye deviation) and epileptic extension to the lateral frontal cortex, possibly linked to the tonic-clonic seizure of the right arm.

In conclusion, we highlight that an adverse seizure is a possible consequence of not only a medial frontal lesion but also concomitant subcortical excitation. These are mutually related and possibly linked to an ipsilateral epileptogenic site, as represented by the PLEDs. □

**Disclosures.**

None of the authors has any conflict of interest to disclose. This work was not supported by a grant or any other means and has not been presented anywhere before.

**Legend for video sequence**

Real-time video-EEG showing seizure features: eye deviation and neck rotation to the right, corresponding to the appearance of PLEDs, followed by a tonic-clonic seizure.

**Key words for video research  
on [www.epilepticdisorders.com](http://www.epilepticdisorders.com)**

*Syndrome:* focal non-idiopathic frontal lobe epilepsy

*Etiology:* head trauma

*Phenomenology:* versive seizure (ipsilateral)

*Localization:* orbitofrontal

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