

# Chapter 13

## Head Trauma and Posttraumatic Seizures

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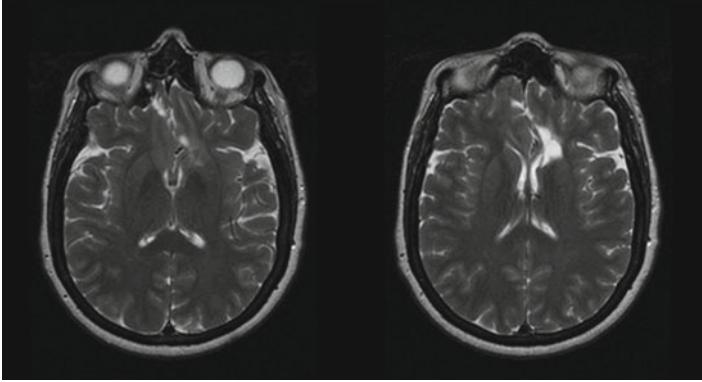
### Case Presentation

A 44-year-old, right-handed, white male presents for an evaluation and opinion regarding the underlying etiology of his seizures. The patient had his first seizure in the summer of 2009. This occurred out of sleep. He noted significant work-related stress and alcohol usage the night before. His girlfriend awoke in the early morning hours when the patient experienced a generalized tonic-clonic seizure. He bit the left side of his tongue and had post-event confusion. He returned to sleep but had a second generalized tonic-clonic seizure 30 min later. EEG was normal awake and asleep. He was prescribed levetiracetam but did not take on a long term basis. He did well until October 2010, when he had a third nocturnal generalized tonic-clonic seizure. This event was preceded by sleep deprivation for over 24 h, significant alcohol use, and the use of cocaine. His girlfriend awoke to vocalizations associated with a convulsion. He was re-started on levetiracetam 500 mg twice daily. Review of risk factors for epilepsy disclosed that he had been involved in a motor vehicle accident in 1985 where he sustained a skull fracture that was associated with a loss of consciousness for approximately 5 days. He did not require neurosurgical intervention and recovered. He was later involved in an altercation in 1996 and was stabbed near his right eye, from which he also recovered. No family history of seizures, meningitis or encephalitis, stroke, brain tumor, brain surgery, migraines, collagen vascular disease, or febrile convulsions was evident. A brain MRI was obtained (Fig. 13.1).

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**Fig. 13.1** T2-weighted brain MRI with bilateral frontal lobe encephalomalacia

## Clinical Questions

1. How does the MRI help answer the patient's question?
2. What is his diagnosis?
3. What factors may affect his seizure control?
4. What is the best course of treatment?
5. What is the prognosis for developing epilepsy after a traumatic brain injury?

## Diagnostic Discussion

1. The axial T2-weighted images show cystic encephalomalacia within the right orbitofrontal and left medial frontal region. Surrounding gliosis and ex-vacuo dilatation of the left frontal horn are also present. This is oriented along a linear trajectory extending from the medial right orbital roof. In this patient's case, the location likely represents the sequelae of a prior transorbital penetrating injury.
2. The patient has a clinical diagnosis of localization-related epilepsy, anticipated to be of frontal lobe origin. This diagnosis is supported by the MRI findings and by the generalized semiology of the seizures. The patient does not note an aura, though this is probably because his seizures arise out of sleep and are unable to be recalled. Frontal lobe seizures often occur during sleep, while temporal lobe seizures tend to occur while the patient is awake.
3. Posttraumatic epilepsy is the leading cause of new-onset seizures in young adulthood with acquired epilepsy. A number of factors may lower the seizure threshold in patients with epilepsy. Stress, sleep deprivation, intercurrent infections, medication noncompliance, and hypoglycemia have all been described as potential exacerbating factors. Drugs of abuse such as cocaine and amphetamines may also lower the seizure threshold.

4. The best course of treatment is based upon proper classification. His advanced age of onset and the abnormal brain MRI would be unlikely for someone with genetic generalized epilepsy. Aggressive management with antiseizure drugs directed at treatment for focal seizures should be undertaken. Accenting nighttime dosing and extended-release preparations (i.e., levetiracetam, lamotrigine, carbamazepine) may be useful choices. Given the MRI-defined structural abnormality that is often predictive of the epileptogenic zone, an epilepsy surgery evaluation is indicated if the patient continues to have debilitating seizures despite appropriate treatment with at least two first-line ASDs. Trauma acts as a negative predictor in patients that undergo temporal lobe resection.
5. The risk of epilepsy after traumatic brain injury (TBI) is influenced by the severity of head trauma. Military injuries are likely to carry a higher risk than civilian head injuries with only a small number of patients that develop posttraumatic epilepsy following mild, closed head injury. The risk of epilepsy is highest within the first year after the injury, but head injury remains a causal link to epilepsy for over 10 years. Seizures occurring “early” after a TBI within the first week are distinguished from “late” posttraumatic epilepsy because they differ in respect to mortality and prognosis of persistent seizures. Many early seizures do not recur as late posttraumatic epilepsy. Posttraumatic epilepsy is defined as seizures occurring at least 1 week after the acute injury. Although phenytoin may decrease early seizures within the first week after head trauma, no AED has demonstrated efficacy in stopping the development of posttraumatic epilepsy. Seizure recurrence may be higher than in those without a history of traumatic injury following control.

## Pearls of Wisdom

1. The pattern of TBI that is present on brain MRI may help to explain the cause in patients with posttraumatic epilepsy.
2. “Early” seizures occurring within 24 h after injury do not predict the development of “late” seizures that occur beyond the first week post trauma in patients with posttraumatic epilepsy.
3. The risk of developing posttraumatic epilepsy remains high for many years after the acute injury. Additionally an underlying injury or an abnormal brain MRI carries a high risk for a second seizure.
4. Treatment with ASDs that render a patient with posttraumatic epilepsy seizure free have a greater risk of relapse following discontinuation. Trauma acts as a negative predictor when epilepsy surgery is considered.

## **Bibliography**

1. Christensen J. Traumatic brain injury: risks of epilepsy and implications for medicolegal assessment. *Epilepsia*. 2012;53 Suppl 4:43–7.
2. Lowenstein DH. Epilepsy after head injury: an overview. *Epilepsia*. 2009;50 Suppl 2:4–9.
3. Temkin NR. Antiepileptogenesis and seizure prevention trials with antiepileptic drugs: meta-analysis of controlled trials. *Epilepsia*. 2001;42(4):515–24.