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Unilateral thalamic lesion mimicking genetic generalized epilepsy

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A 26-year-old woman presented with absence seizures since age eight, diagnosed as juvenile absence epilepsy. Absence and bilateral tonic-clonic seizures were well-controlled on valproic acid. Examination was normal. EEG showed 3-Hz generalized spike-and-wave discharges (*figure 1*). MRI revealed an infiltrative lesion in the left thalamus and dorsal midbrain (*figure 2*). Advanced imaging suggested a low-grade glial neoplasm. Follow-up imaging showed no progression.

The thalamus plays an important role in regulating generalized epileptic networks (Avoli, 2012), including the initiation and propagation of generalized spike-and-wave discharges (Zhang *et al*., 2015). Prior case studies support the ability of a unilateral thalamic lesion to mimic genetic generalized epilepsy, including myoclonic, absence, and bilateral tonic-clonic seizure semiologies, and 3-Hz generalized spike/polyspike-and-wave discharges (Kelemen *et al*., 2006; Nguyen *et al*., 2006). In each case, it was not until an MRI, years later (often for medication-refractory seizures), that an underlying lesion was identified. In our patient's case, because neuroimaging was first performed at age 26, it is possible that the thalamic lesion was coincidental to her epilepsy, but we consider this less likely given the nature of the lesion and known role of the thalamus in generalized epileptic networks. This case further demonstrates that thalamic lesions can mimic the typical features of genetic generalized epilepsy, including pharmacoresponsive generalized seizure types and generalized spike-and-wave discharges in a patient with normal intellect. □

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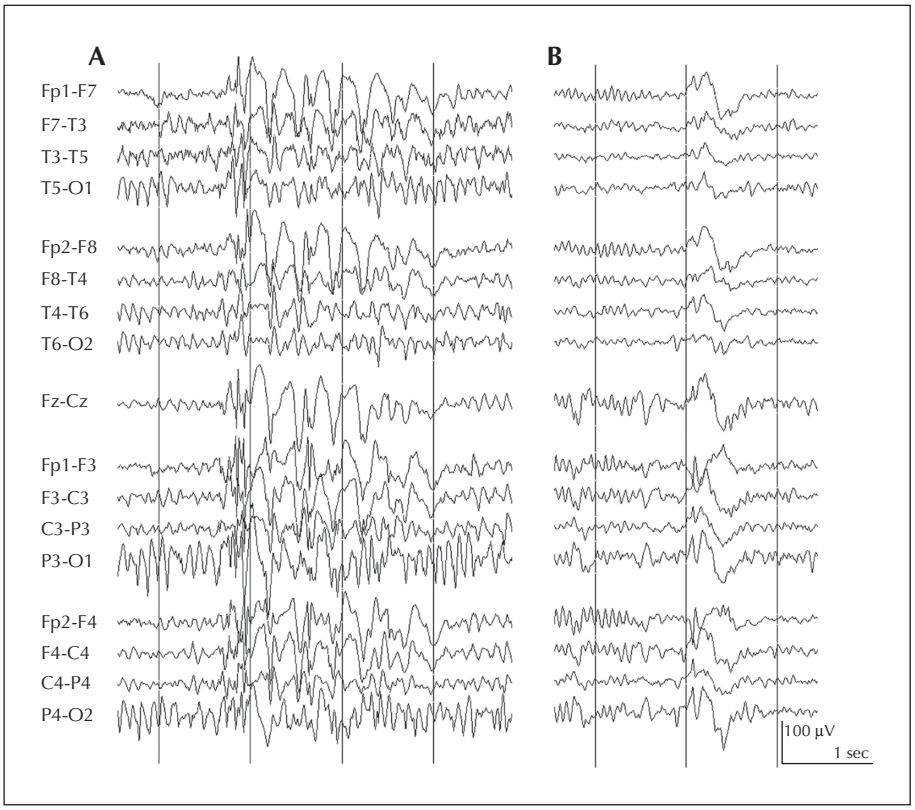

Figure 1. EEG showing 3-Hz generalized spike-and-wave discharges in wakefulness (A) and generalized spike-and-wave discharges embedded in sleep architecture (B).

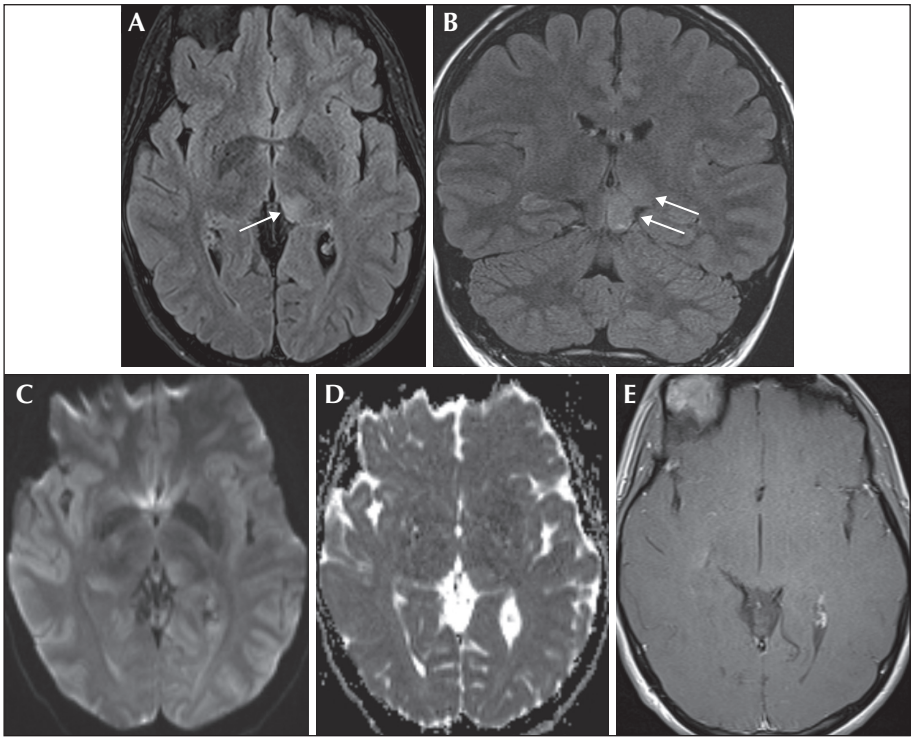

Figure 2. MRI images of the thalamic lesion. Axial (A) and coronal (B) T2-weighted fluid attenuated inversion recovery images show an infiltrative hyperintense lesion of the left thalamus and dorsal midbrain. Axial diffusion weighted (C) and apparent diffusion coefficient (D) sequences show facilitated diffusion. Axial T1-weighted post-gadolinium (E) shows no enhancement.

Supplementary data.
Summary didactic slides are available on the www.epilepticdisorders.com website.

Disclosures.
None of the authors have any conflict of interest to declare.

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TEST YOURSELF

(1) What is the role of the thalamus in generalized epileptic networks?

(2) What are the typical features of genetic generalized epilepsy?

(3) Does genetic generalized epilepsy require imaging for diagnosis?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".