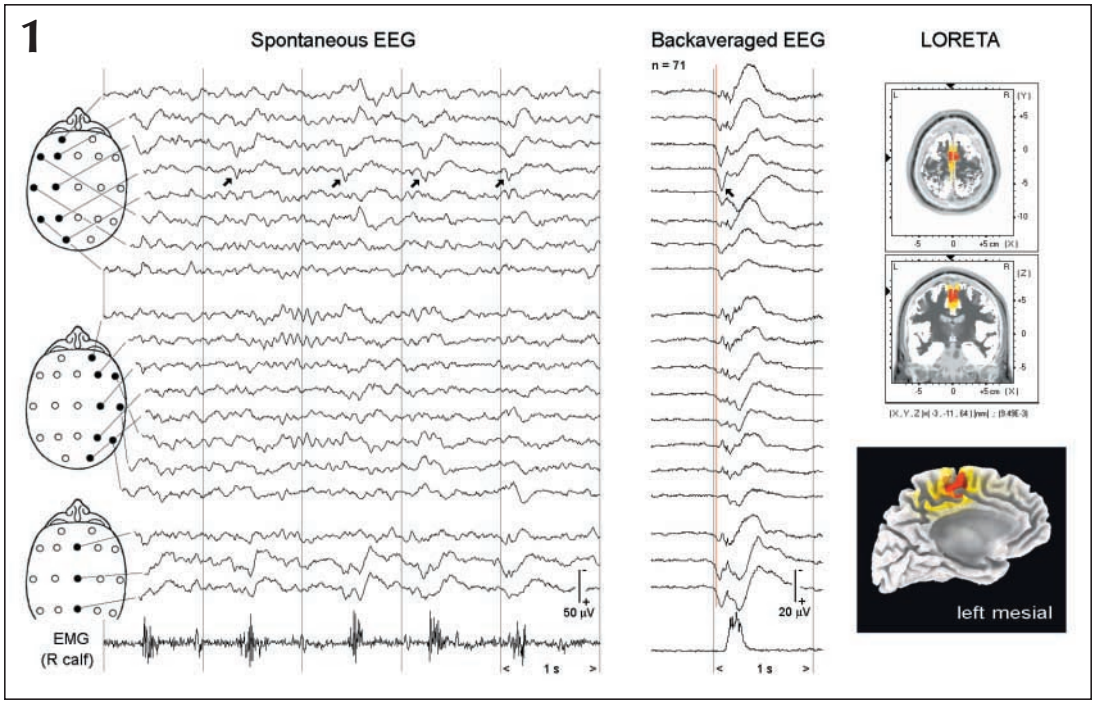
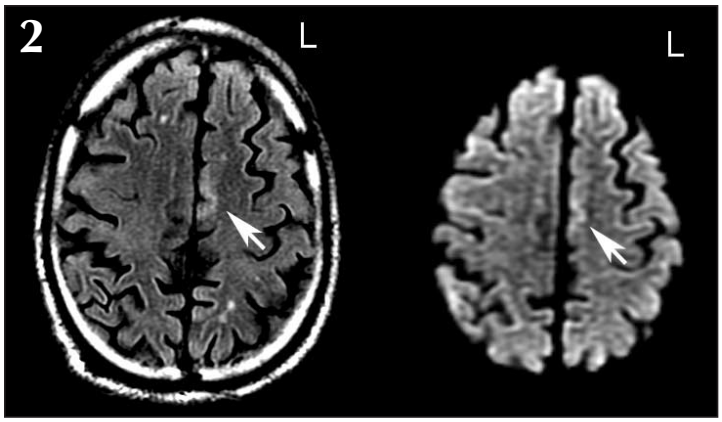
CLINICAL VISTAS

A 61-year-old man with continuous clonic jerks of his right leg





A 61-year-old man with type 2 diabetes mellitus presented with a 3-day history of partial status epilepticus exhibited as continuous rhythmic clonic jerks of his right leg. Head CT scans appeared normal, and an MRI scan with diffusion-weighted imaging (DWI) was reported as showing no acute abnormality, with evidence of mild bilateral microangiopathic disease. Results of laboratory tests, including cerebrospinal fluid analysis, were normal apart from an elevated serum glucose level of 14.7 (normal 3.8–7.0) mmol/L and a serum osmolality of 303 (normal 275–295) mmol/kg. Administration of lorazepam and phenytoin on admission to the medical ward had no effect. Substitution of sodium divalproex for phenytoin 3 days later was similarly without benefit.

Electroencephalography 4 days after admission showed rhythmic sharp waves over the left central region, synchronously preceeding the clonic limb jerks by about 35 ms (Fig. 1, left). Low-resolution electromagnetic tomography (LORETA) localized the seizure focus to the motor leg area (Fig. 1, right; yellow = raw cortical activity, red = area with highest level of significance). After another review of the original MRI scan, a focal area of high signal intensity (Fig. 2, arrows) was noticed in the same area of the left medial motor cortex on a slice of both the fluid-attenuated inversion recovery (FLAIR) and DWI sequences (Fig. 2, left and right respectively), which was in keeping with the recognized MRI changes of partial status epilepticus.

Because of continued seizures, the patient's hyperglycemia was aggressively managed with insulin and fluid replacement; the clonic jerks resolved about 48 hours after the glucose level returned to normal. The patient remained free of seizures and was not taking antiepileptic medication 4 months later.

Epilepsia partialis continua (EPC) is rare, observed in association with cortical lesions of various origin and in some metabolic

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disorders (Box 1). As the name implies, the presentation usually consists of continuous partial seizures; however, occasionally, generalized tonic-clonic seizures may also occur. EPC as a manifestation of nonketotic hyperglycemia (NKH) was first described in 1965,[1] and cases have been intermittently reported since then.[2,3] How systemic NKH causes a focal status epilepticus remains unknown. Hypotheses have implicated decreased levels of the inhibitory neurotransmitter gamma aminobutyric acid (owing to inhibition of the Krebs cycle in NKH) or direct effects of hyperglycemia, dehydration or hyperosmolarity on the brain, possibly acting on a previously silent cortical lesion to render it epileptogenic.[2,3] However, none of these hypotheses works in isolation: seizures may stop before glucose levels completely return to normal, hyperosmolarity is not invariably present, and previous cases reported no evidence of an associated structural lesion in the brain.[2,3]

In elderly people, NKH is a typical cause of EPC, and the EPC may be the first presentation of diabetes in many of these patients. The response to antiepileptic drugs is poor. Instead, the hypergylcemia should be corrected, with seizure activity improving depending on the degree of stabilization of the blood glucose level; fluctuation of seizures is possible during the initial therapy.

However, because the causative hyperglycemia is often no more than moderate in degree, as in our patient, EPC may not be recognized as a manifestation of NKH and treated in a timely fashion. Left untreated, increasing hyperosmolarity and dehydration may result in more severe neurologic deterioration. With correct diagnosis and appropriate treatment, early resolution of the EPC and a good prognosis for complete neurologic recovery can be expected.

Box 1: Various causes of epilepsia partialis continua in adults

Infectious
- Encephalitis
- Cerebral abscess

Vascular
- Embolic or thrombotic ischemic stroke
- Intraparenchymal cerebral hemorrhage
- Subdural hematoma

Neoplastic
- Primary or metastatic cerebral tumour

Metabolic
- Diabetic ketoacidosis
- Nonketotic hyperglycemia

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