Case Report: Control of Epilepsia Partialis Continua with Medical Management

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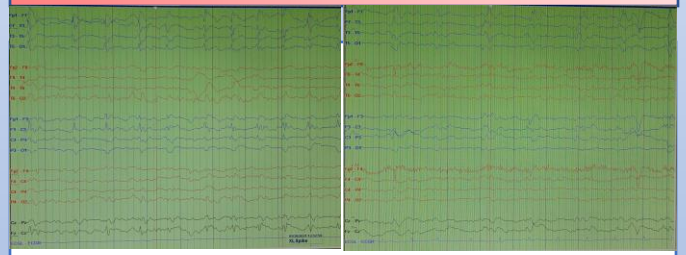
INTRODUCTION

Epilepsia Partialis Continua (EPC) is a rare and notoriously treatment-refractory form of focal motor status epilepticus, classically associated with focal involuntary movements and cognitive impairment that can persist for weeks, months, or even years despite appropriate therapy with antiepileptic drugs (AEDs)[1-4]. It is almost universally caused by an underlying cortical lesion such as an infarct, neoplasm, congenital malformation, encephalitis, or tuberculoma[1-4]. Metabolic causes such as hyperglycemia have also been reported[1,2,4].

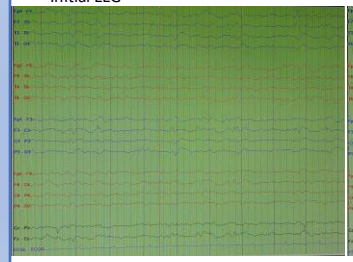
Although the jerking movements associated with EPC are slower than most other forms of status epilepticus[4,5], the disorder can be debilitating to affected patients, especially since they often require long courses of multiple AEDs to control. Given the rarity of this disorder (estimated less than one per million[2,4]), any case report documenting effective control of EPC is noteworthy.

Epilepsia Partialis Continua

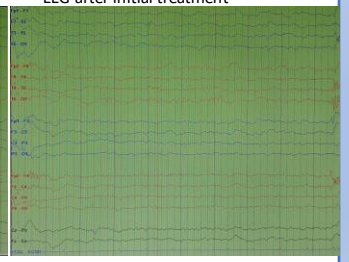


Initial EEG

EEG after initial treatment



EEG with clinical improvement



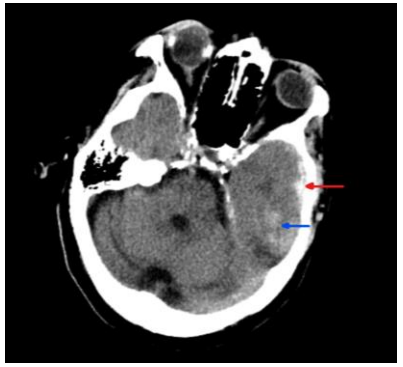
EEG with resolution of EPC

DISCLOSURES

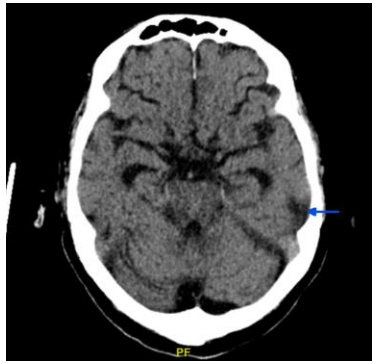
The authors do not have any financial interest, arrangement, or affiliation with any organizations listed. The authors have no conflict of interest to report.

CASE PRESENTATION

- **Patient**: 74-year-old right-handed Caucasian male.
- **Presentation**: altered mentation, mixed transcortical aphasia, right-sided hand and face twitching the day after a mechanical fall at home.
- **Imaging:** CT head left temporal intraparenchymal hematoma and small left frontotemporal subdural hematoma. CTA head/neck no underlying aneurysm or vascular malformation.
- **EEG**: epilepsia partialis continua, originating from left temporal leads. Routine 20-minute EEGs were performed daily after the initial EEG report.
- **Medical history**: hypertension, hyperlipidemia, depression, benign prostatic hypertrophy. Per family his baseline memory was declining but he could manage finances, drive alone, and converse normally.
- **Medications:** no antiplatelets, anticoagulants, or other home medications.
- **Physical exam**: repetitive, involuntary twitching of right hand, right eyelid, and right side of the mouth. Follows simple commands with hints and pantomiming, but often fixates on a single command. Speech consists of repeating a single word or syllable, although he attempts to respond to conversation.
- **Treatment**: started IV Levetiracetam and IV Lacosamide, titrated to maximum tolerated doses. Next started IV Fosphenytoin with a loading dose, and serum levels were monitored. Pulsed doses of IV methyl-prednisolone and IV magnesium sulfate were also given. Due to a shortage of IV formulation, a PEG tube was placed for PO Valproate, and serum levels were monitored.
- **Results**: EEGs showed diminishing EPC discharges coinciding with clinical improvement, and nine days after presentation an EEG found only moderate bihemispheric slowing. Daily EEGs failed to show any recurrence of EPC.
- **Modification**: Titration off of four AEDs began two days after resolution of EPC. First Foshenytoin was weaned, then Keppra, then Lacosamide. Lamotrigine was slowly started with plans to wean Valproic acid after discharge.
- **Follow up**: CT head three weeks after presentation negative for residual blood. Contrast MRI brain afterwards negative for underlying lesion.
- **Resolution**: Involuntary movements ceased with resolution of EPC on EEG. Other clinical improvement followed resolution of bleeding on imaging. Three to four weeks after presentation patient appeared alert and was able to follow commands reliably and converse appropriately, although his memory was not at baseline at time of his discharge six weeks after presentation.



CT head on presentation



Resolution of bleed on CT

DISCUSSION/CONCLUSION

The decision of which AEDs to use was based on clinical judgement in combination with evidence-based guidelines. A combination of medications with different mechanisms of action is found to be effective[6,10]. Primary mechanisms include that of a voltage-gated sodium channel modulator (Lacosamide), SV2A antagonist (Levetiracetam), voltage-gated sodium channel antagonist (Fosphenytoin), and multiple mechanisms (Valproic acid)[10]. AEDs chosen have been shown effective both in broad spectrum and focal-specific epilepsies[7]. Benzodiazepines have not been shown to be effective long-term treatment for EPC[2,4]. Pulsed doses of steroids and magnesium have also been shown to be useful in treatment-resistant status epilepticus, although the mechanism of action for this is uncertain[8,9]. Given the patient's cognitive impairment, preference was given to AEDs available in IV formulation.

After clinical and electrophysiologic control of EPC, the decision to wean AEDs was made to reduce sedation and limit adverse effects. Fosphenytoin was initially weaned due to its long-term side effect profile. Levetiracetam was then weaned due to new-onset irritable mood and combativeness. Lacosamide was weaned as paroxysmal atrial fibrillation was seen on EKG. Finally, the decision was made to wean off Valproate as serum levels were labile requiring frequent dose adjustments. Lamotrigine was chosen for long-term therapy due to additional benefits of mood stabilization; however, due to the requirement for slow titration, Valproate was kept until Lamotrigine could be titrated to a therapeutic dose.

Epilepsia Partialis Continua typically has a poor prognosis due to difficulty controlling it with AEDs[1,5]. However, with aggressive treatment and EEG monitoring this patient had a good clinical and electrophysiologic outcome, even with subsequent curtailing of therapy. It's possible that EPC has a better prognosis if the underlying cause is acute rather than chronic[5], however further study is required to elaborate on this.

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