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Epilepsia Partialis Continua that Improved in a Pediatric Patient with Sub-dural Empyema

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Abstract

In epilepsia partialis continua (EPC), the EEG tracings may fail to show epileptiform activity because the electrical activity is too subtle or too deep to be picked up by surface electrodes. EPC can occur at any age and may have many etiologies, including genetic, metabolic, structural, infectious, and idiopathic. Typical EEG in EPC is characterized by discharges of cortical origin that commonly consist of sharp waves, spikes or periodic lateralized epileptiform discharge; however, EEG findings at large are variable and often not even identified. Here we present a pediatric case of EPC in the setting of subdural empyema with atypical EEG seizure associated with focal clonic activity who made rapid improvements.

Keywords

pediatric, epilepsy, EEG, adolescents, seizures

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Case

A 14-year-old female with Type 1 diabetes mellitus, presented with sudden onset altered mental status and right hemiplegia to the emergency department and was found to have right frontal and ethmoid sinusitis along with right mesial frontal necrotizing subdural empyema with 6 mm midline shift and early uncal herniation. She underwent emergent craniotomy and drainage of subdural abscess. She was started on IV antibiotics and transferred to inpatient rehabilitation service once stable. Follow up magnetic resonance imaging (MRI) on day 18 revealed slight residual subdural empyema with residual cerebritis of anterior and frontal lobe. During her post-operative recovery on day 24, the patient began to develop left foot twitching that progressed to involve her entire left leg with preserved consciousness and mental status. She developed difficulty walking from frequent jerking and weakness of left leg with foot drop. She reported reported no vision changes, headaches, or changes in mentation. On physical exam, she was noted to have rhythmic, 1 Hertz, low amplitude clonic movement of the left lower extremity originating from hip flexor and quadriceps muscle. She had hip flexor weakness of 4/5 and left foot drop. The remainder of her neurological examination was unremarkable. Given the history of right subdural empyema with new onset left leg clonic activity that persisted for more than 12 h, which was not distractible and continued in sleep, focal epileptic activity was suspected and continuous video EEG was started.[1,2]

EEG showed slow rhythmic activity in the centro-parietal zones lacking a typical spike-wave discharge morphology correlating with left leg clonic activity (Figure 1). This activity persisted while asleep, and she did demonstrate clonic movements in sleep. There was no spread of this activity on the EEG to other regions. The clinical picture was consistent with Epilepsia Partialis Continua (EPC) although EEG did not show typical features of electrographic seizures. The clonic jerks did not abort with lorazepam or diazepam, so the patient was started on maintenance lacosamide after a loading dose. She was also continued on the levetiracetam that she was already on for seizure prophylaxis. MRI brain was repeated that showed slight enlargement of the right posterior falcine subdural fluid and interval improvement of the frontal subdural empyema. MRI images showed restricted diffusion in the right mesial frontal motor area (Figure 2). She was managed conservatively with anti-seizure medications and continuation of her

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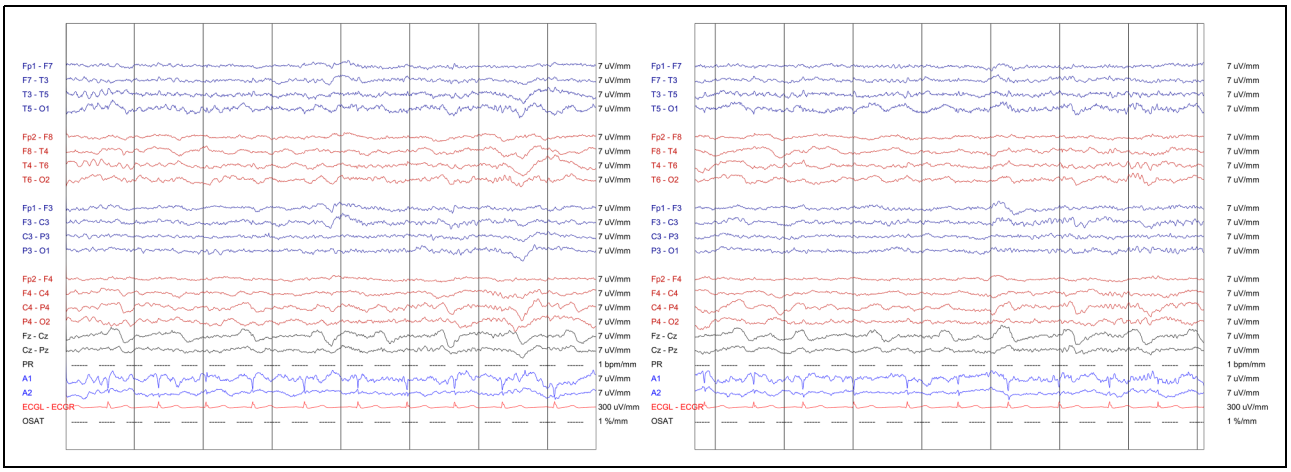

Figure 1. EEG demonstrating monomorphic slow rhythmic activity noted in the central region while asleep interspersed with sleep spindles.

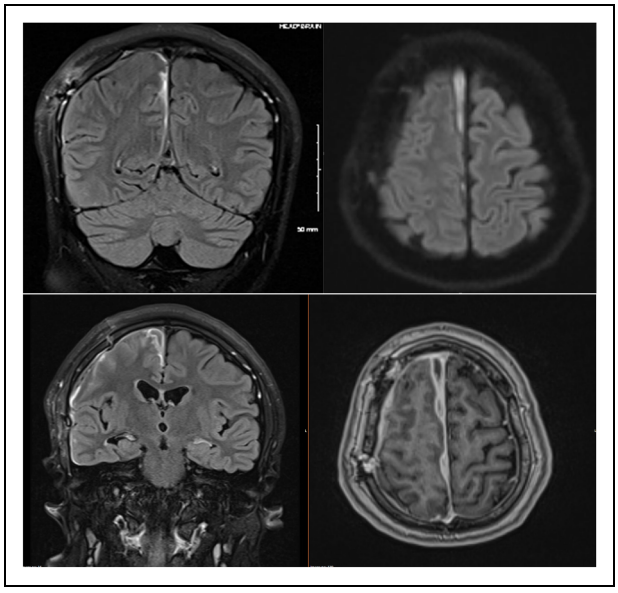

Figure 2. MRI T2 flair and DWI demonstrating hyperintensity in the right mesial frontal motor area.

IV antibiotics. Her left hip and knee clonic movements improved after three days of lacosamide, but clonic movement of the left calf area was still noted for which short term clonazepam was added. Left calf muscle clonic activity as well as her ambulation, improved after the addition of short-term clonazepam. Repeat MRI brain on day 33 showed improvement in the posterior falcine abscess. By the day of discharge, she had returned to her baseline ambulation abilities with cessation of focal seizures.

She recovered without residual neurologic deficits and with improvement in EPC. She was discharged home on day 34 with levetiracetam, lacosamide, and the remaining clonazepam wean, and at one-month post-discharge follow up, she had continued seizure freedom.

Discussion

EPC is defined as a condition of recurrent epileptic seizures with preserved consciousness lasting more than one hour secondary to a localized epileptic activity.[3]

It is a rare epilepsy that impacts both the pediatric and adult populations. There is a paucity in evidence of the prevalence, but previous regional studies have suggested a prevalence of one per million.[4] EPC has been associated with mitochondrial disorders, inflammatory changes, tuberulomas, and vascular compromise such as ischemic stroke.[5,6] There are few case reports citing changes in fluid collections as associated with EPC. Clinical manifestations are highly variable in severity, duration and seizure semiology, ranging from second-long jerks to abnormal generalized motor activity for days. It is understood that cortical hyperexcitability or slow rhythmic activity can play a prominent role in EPC, but EEG findings are poorly characterized and may not be identified if abnormalities are too deep to be identified with the common scalp electrode diagnostics.[3,7] Slow rhythmic activity that correlated with prolonged abnormal left leg movements and MRI brain pathologic changes led to the suspicion of EPC in this patient.[4,8] The more typical spike wave discharges of a seizure were not seen because the epileptic focus was midline in the cortical leg area that is deep and cannot be picked up by scalp electrodes. Once EPC is identified, prognosis is more favorable if the underlying etiology can be addressed, though most pediatric cases report residual psychomotor deficits.[9,10] For our patient, her blood glucoses were well-controlled throughout the hospital stay, and her antibiotic regimen had already been optimized.

She did not show any signs otherwise of new infectious processes or neurological deficits, so we optimized her anti-epileptic regimen in parallel to the improvements of her empyema that were evidenced by resolution of empyema and swelling on repeat neuroimaging. Very little literature captures such short-term correlation between maintained clinical improvement after polypharmacotherapy began weaning, and imaging.

In summary, this patient had very subtle EEG findings and evolving clinical changes with better and more rapid outcome than is often seen in other reported cases.

Our collective findings demonstrate the importance of maintaining a wide differential when evaluating new onset focal neurological deficits. This can can also help in further developing characterization of EPC in the pediatric population. It is prudent to maintain a high index of suspicion of even rare pathologies.

Further case studies may be beneficial in raising awareness of this subtle and ominous, but potentially treatable malady.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical Approval

Our institution does not require ethical approval for reporting individual cases or case series.

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