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Case Report

Epilepsia Partialis Continua without Radiological Abnormalities in an HIV-infected Patient: A Case Report

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Abstract

Epilepsia partialis continua (EPC) without intracranial abnormalities is a rare presentation in patients with human immunodeficiency virus (HIV) infection. In our case report, the patient presented with left-sided clonic-myoclonic seizures that were resistant to initial anti-seizure treatment. This condition is classified as EPC. Lesions that could be linked to episodes of EPC were not evident on magnetic resonance imaging. Electroencephalography showed right-sided centroparietal epileptiform activity. After blood testing, the patient was diagnosed with HIV. Dual antiseizure medications were administered, leading to seizure cessation after three days.

Keywords: Epilepsia, epilepsia partialis continua, human immunodeficiency virus infection

INTRODUCTION

A retrovirus called human immunodeficiency virus (HIV) infects immune cells and destroys their function. In people with HIV, disorders of the central nervous system (CNS) arise either as a direct outcome of HIV itself or due to different opportunistic infections. Additionally, conditions like lymphoma, cerebral infarction, and immune reconstitution inflammatory syndrome are also observed in these patients. In imaging, there are various radiological findings, such as intracranial lesions, hydrocephalus, edema, infarcts, signal changes within the parenchyma, and contrast enhancement, etc. depending on the etiology.[1,2]

There are several potential causes for epilepsia partialis continua (EPC), which can be either localized or systemic. From a pathophysiological standpoint, EPC appears to involve a feedback loop characterized by alternating excitation and inhibition, though the precise mechanisms behind this process are not yet well understood.[3]

We report a case of a patient who presented with EPC, was diagnosed with HIV, and had no intracranial pathology.

CASE PRESENTATION

A 42-year-old woman from a foreign country presented to the emergency room (ER) with bilateral tonic-clonic seizures. She was admitted to the neurology inpatient clinic after receiving initial anti-seizure treatment in the ER. Although the patient's examination was suboptimal because of the language barrier, her neurological examination revealed decreased consciousness with no meningeal irritation or paresis. Blood tests indicated pancytopenia, elevated C-reactive protein, and positive anti-HIV staining. Serum antibodies for Borrelia, Toxoplasma, syphilis, and hepatitis were negative. No significant pathology was observed on cranial imaging (Figure 1). After reaching out to a relative of the patient, we learned that a lumbar puncture had been performed 5 days earlier at an external clinic for a differential diagnosis of encephalitis. Cerebrospinal fluid (CSF) investigations showed that biochemical indicators were within normal ranges, with no erythrocytes or leukocytes and a mildly elevated micro-total protein level. Follow-up observations indicated prolonged reaction time and ongoing clonic-myoclonic seizures with no response to diazepam infusion; therefore, levetiracetam treatment at a dose of 3000 mg/day was initiated for the patient. The patient was cooperative and oriented; however, his left-sided focal motor seizures persisted. Since the patient still had no response to treatment, the case was classified as EPC, and valproate treatment at a dose of 800 mg/day was added. As a result of candida esophagitis, additional antibiotic treatment was given after consulting with the infectious disease department. The electroencephalography (EEG) results revealed bioelectrical disorganization in both hemispheres and epileptic activity in the right centroparietal region (Figure 2).

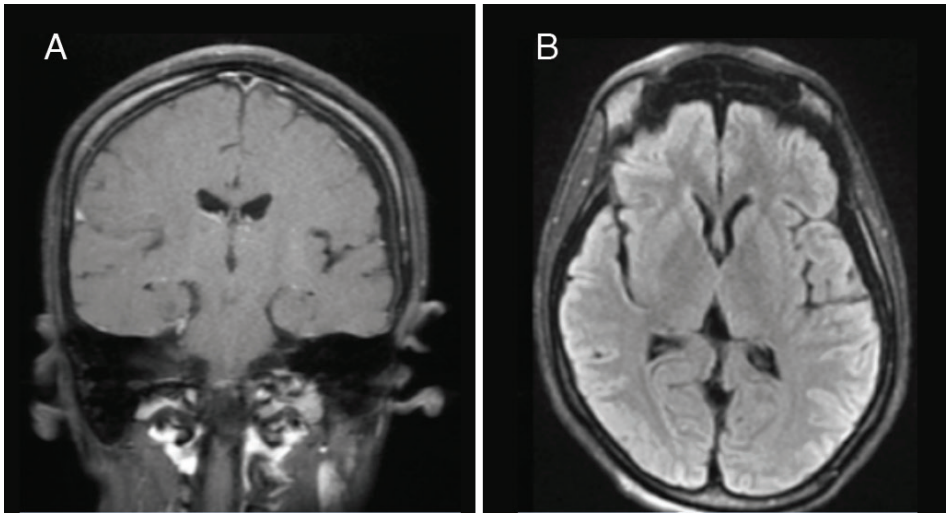

Figure 1. No parenchymal lesion or contrast enhancement is observed in (A) C+T1 coronal and (B) FLAIR axial sequences

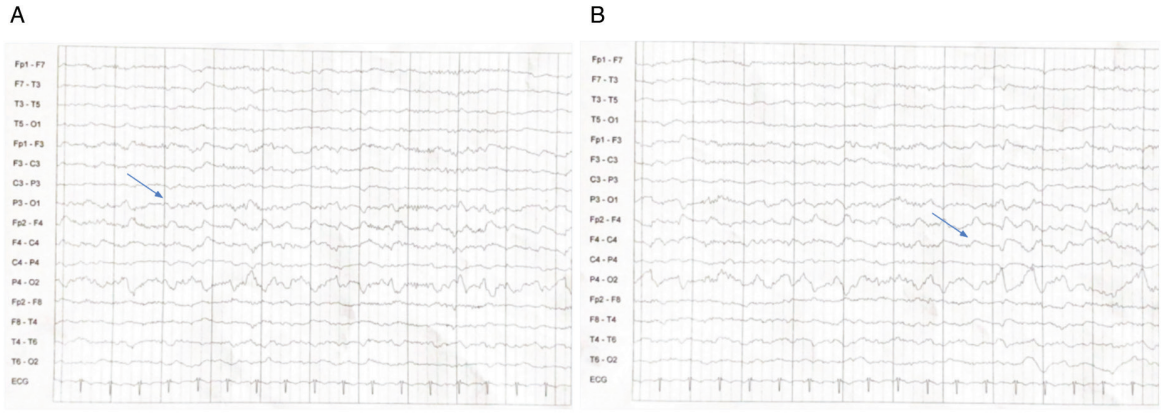

Figure 2. The EEG investigations (A) and (B) revealed bioelectrical disorganization in both hemispheres and epileptic activity in the right centroparietal region
EEG: Electroencephalography

During the follow-up, valproate was discontinued and replaced with low-dose lamotrigine due to worsening pancytopenia with valproate. The patient's seizures finally ceased and remained under total control on the third day after the initiation of anti-seizure treatment, and there was no pathology on the final neurological examination.

MAIN POINTS

- This case demonstrates a rare instance of epilepsia partialis continua (EPC) in an HIV-infected patient without central nervous system (CNS) pathology, highlighting the diverse neurological manifestations of human immunodeficiency virus (HIV).
- The pathophysiology of EPC in HIV-infected patients is thought to involve neurotoxic interactions among activated macrophages, astrocytes, and neurons, leading to neuronal death through increased glutamate activity and calcium influx, which disrupts the balance of excitatory and inhibitory neurotransmitters, thereby increasing seizure susceptibility.[4]
- This case emphasizes the need to consider HIV-related neurotoxicity as a potential cause of EPC in immunocompromised patients, even in the absence of radiological findings.

DISCUSSION

Garg[3] mentioned that new-onset generalized-type seizures are common manifestations in people infected with HIV. These seizures are mostly caused by CNS disorders.[4] Rarely EPC can be seen without any CNS pathology in people infected with HIV.

Mameniškienė and Wolf[4] describe that EPC represents a form of simple focal motor status epilepticus, characterized by frequent and repetitive muscle jerks. These muscle jerks usually lack a regular pattern. EPC can persist from one hour to years. Typically, it doesn't respond well to medical treatment. Benzodiazepines, levetiracetam and topiramate are some treatment options.[3]

The archetypical presentation of EPC is a continuous focal myoclonus involving the distal extremity. In our case, however, it had presented in both the upper and lower left extremities. The patient presented with EPC and mild loss of consciousness as the first manifestations of HIV-related encephalopathy without any radiological findings. Our CSF findings were inconclusive, with a lack of leukocytes and only a slight increase in CSF protein (49 mg/dL) and normal glucose levels, thereby ruling out any other

opportunistic infections. Epileptiform discharges in the right centroparietal region helped us localize the epileptic zone of EPC.

After ruling out HIV-associated opportunistic infections and space-occupying lesions we had come to the conclusion that newly onset EPC was the sole neurological manifestation in this patient. There have been many theories as to why seizures might occur in the absence of the previously mentioned causes. Garg[3] suggest that HIV or toxins related to immune deficiency cause macrophages, astrocytes and the neurons producing the neurotoxic substances to interact in a manner that indirectly results in neuron death.[4] The neurotoxic substances include eicosanoids, platelet-activating factor, quinolinate, cysteine, cytokines and free radicals. The macrophages that get activated by HIV-1 envelope protein gp120 also release similar toxins.

The final pathway, as described by Garg,[3] is through increased glutamate activity, which results in an influx of calcium into cells that leads to untimely death. This cascade of cell death results in an imbalance of excitatory and inhibitory neurotransmitters, which is coupled with neurotoxicity, resulting in a predisposition to seizures. It was our theory that these were the underlying mechanisms that caused the seizures in our patient.

Dawson's[5] research dating back to 1947 suggests that the waveforms commonly associated with EPC had a positive wave, followed by negative shifts with a train of superimposed spikes.

Inherent lateral inhibition in the neocortex is understood to keep specific pathway-associated responses precise. It has been suggested that this phenomenon might explain why seizures observed in EPC are localized and self-limiting compared with seizures that arise from the allocortex of the limbic pathways, which usually spread.[6] This phenomenon was observed in our patient as well, whose EEG revealed self-limiting sharp wave activities in the right centrotemporal region, which led us to believe that this region was affected at a neuronal level that could not be observed in magnetic resonance imaging (MRI).

While seizures are common manifestations of CNS involvement in HIV-infected patients, generalized seizures are the most common type.[4] Convulsive status epilepticus is also a common manifestation due to serum electrolyte abnormalities. While our patient had also presented with status epilepticus, after the serum abnormalities were quickly dealt with the only manifestation left was continuous clonic-myoclonic jerks with preserved consciousness.

HIV presenting with EPC is rare as is, with the first case being reported in 1999,[4] and the fact that our patient had no cerebral mass lesion to speak of made this a case worth documenting and presenting. While most patients who present with EPC have parenchymal lesions that can be viewed in an MRI, those who do not have any have also been reported. That is why while all common causes must be investigated when a patient presents with EPC, an immunocompromised state like HIV seropositivity, even without imaging findings, might be the underlying cause.

CONCLUSION

In summary, this case highlights the occurrence of epilepsia partialis in a patient with HIV infection. While generalized seizures, contrast enhancement, or parenchymal occupation on imaging are common among patients with HIV infection, this case stands out by presenting as focal seizures without detectable pathological brain findings on imaging.

Ethics

Informed Consent: Informal consent was obtained from the patient.

Footnotes

Authorship Contributions

Surgical and Medical Practices: H.,O., Concept: F.,E., Design: F.,E., Data Collection or Processing: B.,A., Analysis or Interpretation: U.K., Literature Search: A.E.O., Writing: E.N.T.

Conflict of Interest: None of the authors has any conflicts of interest to disclose.

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