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Case Report Open Access

HIV Encephalitis Presenting as Epilepsia Partialis Continua

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Abstract

Seizures in HIV positive patients can be a consequence of opportunistic infections, neoplasms, or HIV encephalopathy. Most commonly, these are acute symptomstic seizures secondary to focal cortical lesions, however onset with Epilepsia Partialis Continua is extremly rare. We report case of a 21 year old boy, with nopre-morbid illnesses, presenting with Epilepsia Partialis Continua, MRI Brain showed bihemispherical cortical lesions, and was detected to be HIV positives. He showed good clinical improvement with IV Immunoglobulins and multiple Anti-epileptic drugs.

Keywords: Heavy metal; Cadmium; Sensitivity; Nitrification

Introduction

Acute Symptomatic Seizures can occur in 2% to 20% of all Human Immunodeficiency Virus (HIV) infected individuals [1]. The most common underlying cause is focal cortical lesion, which etiologically can be opportunistic infections (Toxoplasmosis, Tuberculosis, Cryptococcosis), neoplastic lesion (CNS Lymphoma), and HIV encephalopathy [2]. Seizure at onset in HIV patients with Epilepsia Partialis Continua (EPC) in is a rare entity [3]. We discuss a case of 21 year old male who presented with Epilepsia Partialis Continua, and was detected to be HIV positive with significantly high HIV RNA load [4].

Case study

21 years male, resident of Tamil Nadu, India, right handed individual, educated upto Class 10th, unmarried and no past co-morbidities [5]. He presented with history of witnessed seizure three months back [6]. Semiology of event was characterized by no preceding aura, Ictus with jerking movement of left angle of mouth and left upper limb associated with uprolling of eyeballs and tongue bite (duration of episode nearly 2 minutes), followed by post ictal phase drowsiness, post-ictal amnesia for the episode [7]. He was initially managed in peripheral hospital, wherein he continued to have repetitive, involuntary clonic-myoclonic movements of left upper limb associated with deviation of angle of mouth towards right, without impairment of sensorium [8]. He was managed at peripheral hospital with sequential Anti-epileptic drugs (Phenytoin, Levitracetam and Oxcarbazepine), with no significant reduction in frequency and severity of seizures [9]. He was thereafter transferred to Command Hospital, Bangalore for further management [10]. Initial evaluation revealed normal higher mental functions, speech characterized by hypotonic, scanning dysarthria, bilateral lower motor neuron 7th Nerve palsy left>right) and focal myoclonic jerks involving left angle of mouth and distal left upper limb with phenomenology suggestive of Epilepsia Partialis Continua (EPC) [11]. In view of the characteristic clinical profile, possibilities of Rasmussen's Encephalitis, Autoimmune Encephalitis and Focal cortical lesions involving Right Motor Cortex were considered (Dysplasia, Space occupying lesions) [12]. He was managed with IV Immunoglobulin at 2 g/kg over five days, along with Anti-epileptic drugs (Tab Oxcarbazepine 750 mg BD, Tab Levitracetam 1.5 g BD, Tab Perampanel 6 mg OD) [13]. Initial evaluation revealed normal hemogram and biochemical parameters. MRI brain showed focal area of cortical T2/FLAIR hyperintensity in right pre-central gyrus (hand knob area) with dedifferentiation of grey-white interface and focal diffusion hyperintensity, PET-CT showed focal increased FDG uptake in same region-right Pre-central gyrus. CSF Study including Cytology, biochemistry, ADA levels, CSF for Auto-immune Encephalitis panel (Anti-NMDA, VGKC, AMPA antibodies) and CSF Biopanel (PCR for HSV, CMV, EBV, *pneumococus*) was Negative. He was detected to have HIV ELISA Positive, with HIV RNA load of 163771 IU/ml, CD4 T cell count of 95 cells/cmm. EEG showed Right Fronto-central spike and wave discharges, occurring periodically at frequency of one Hz with phase reversal at C4 (Figures 1-4).

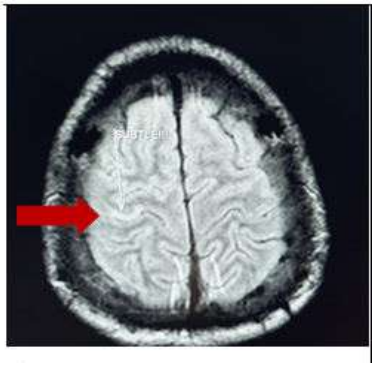

Figure 1: Axial flair MRI image shows hyperintensity along precentral gyrus. MRI brain (Day 2 of onset).

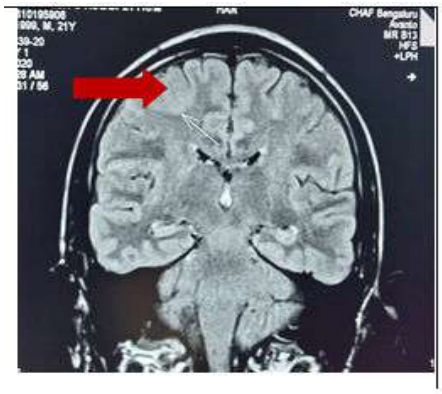

Figure 2: Coronal flair showing subtle dedifferentiation of grey-white interface and bulky precentral gyrus.

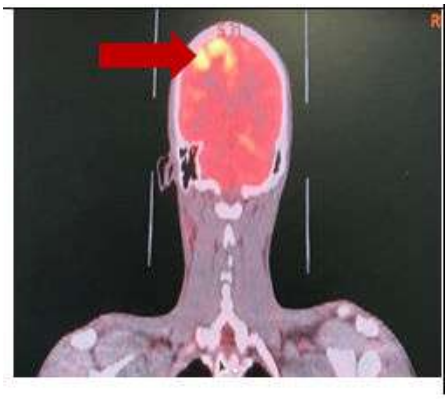

Figure 3: PET -CT coronal and flair showing focal increased FDG uptake in entire right pre-central gyrus.

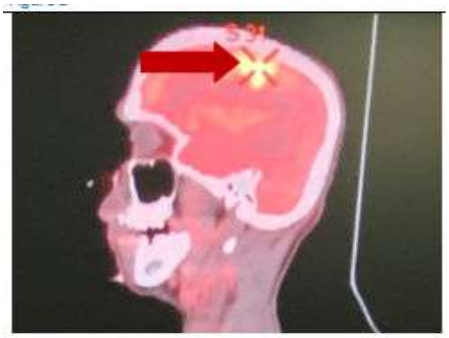

Figure 4: PET-CT coronal and flair showing focal increased FDG uptake in entire right pre-central gyrus.

At Day 7 days, he had worsening in severity of seizures, with progression to status epilepticus (bilateral upper and lower limb myoclonic jerks and deterioration of sensorium). This necessitated endotracheal intubation, mechanical ventilation, intravenous midazolam infusion along with anti-epileptic drugs. Repeat MRI brain (Day 8) showed increase in cortical signal changes, with in the involvement of entire right motor cortex and subtle FLAIR hyperintensity in left Globus pallidus. In consideration of clinical and radiological findings, he was managed with additional IV Immunoglobulin at 1 g/kg over 02 days. During the course of hospitalizarion, he developed Aspiration Pneumonitis while on Mechanical ventilator. He was managed with culture sensitive antibiotics, AEDs and supportive management (Figures 5-8).

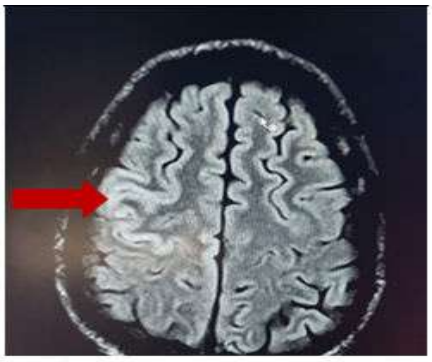

Figure 5: Axial flair sequence showing in increase in cortical signal changes with involvement of entire right motor cortex MRI brain day 7 after onset.

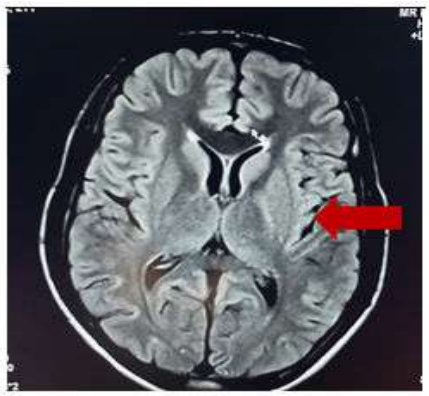

Figure 6: Axial flair sequence showing focal hyperintensity involving left globus pallidus.

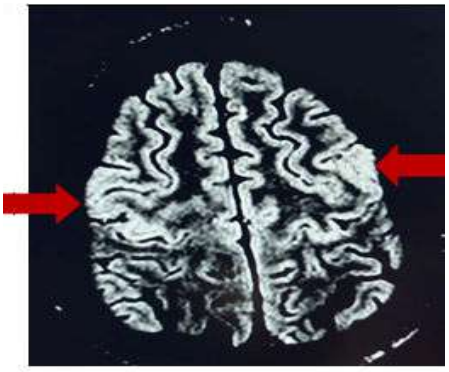

Figure 7: Axial flair sequence showing new areas of signal hyperintensity involving left motor cortex, with pre-existing changes in right pre-motor cortex MRI brain day 14.

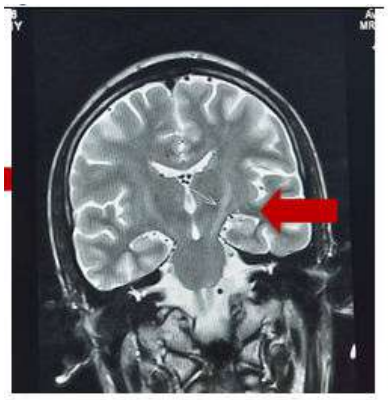

Figure 8: Coronal T2 sequence showing hyperinyensity along entire visulaised course of left cortico-spinal tract.

MRI brain (Day 15) showed new areas of signal abnormalities involving left motor cortex, along with T2 prolongation (with areas of diffusion restriction) along the entire course of left cortico-spinal tract. There were new discrete focal signal changes in left cerebellar hemisphere and right occipital lobe, with previously noted lesions in right motor cortex and left globus pallidus. He showed gradual clinical improvement over next one week with remission of seizures and improvement in sensorium. He was finally discharged on day 30, with advice to continue Anti-epileptic drugs (Tab Perampanel 6 mg once daily, Tab Lacosamide 200 mg twice daily, tab leviteracetam 1 gm twice daily, Tab Oxcarbazepine 750 mg twice daily), along with Anti-Retroviral treatment (Tab Tenofovir 300 mg once daily, tab lamivudine 300 mg once daily, tab efavirenz 600 mg OD), along with cap fluconazole 150 mg once daily and tab septran DS once daily. Follow-up MRI brain done at 6 weeks showed complete resolution of previously noted cortical and sub-cortical lesions in the (Figures 9-13).

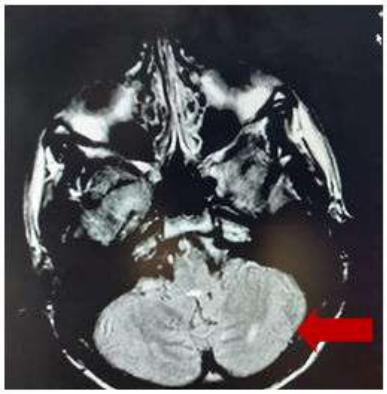

Figure 9: Axial flair shows focal hyperintensity in left cerebellarhemisphere.

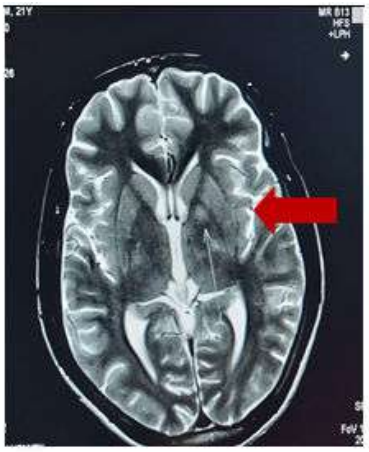

Figure 10: Axial T2 equence showing persistence of hyperintensity left globus pallidus.

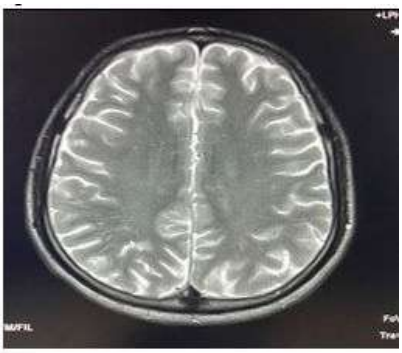

Figure 11: Axial and coronal T2 sequences showing significant resolution of cortical and sub-cortical hyperintensities MRI brain at day 28.

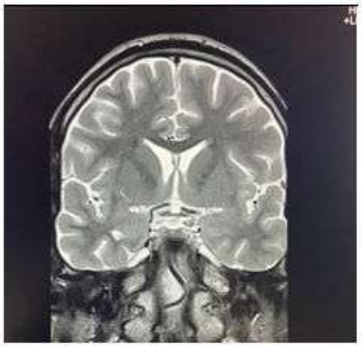

Figure 12: Axial and coronal T2 sequences showing significant resolution of cortical and sub-cortical hyperintensities.

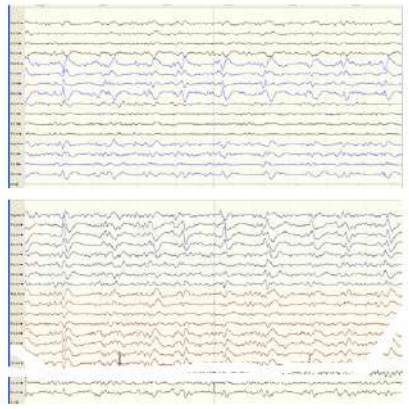

Figure 13: longitudinal montage (above) and average montage (below), with standard filter settings (high frequency filter setting 60 hz, low frequency filter setting 1 hz.

classification: Dyes iii right fronto-central region. **Interpretation:** Awake record showing background activity of 8-10 hz (better seen over left hemisphere). Longitudinal montage shows right fronto-central spike and wave discharges, occurring periodically at frequency of one hz with phase reversal at c4. Average montage shows epileptiform discharges with maximum amplitude at c4.

Discussion

Acute symptomatic seizures in HIV Infection has been described in 4% of HIV patients as initial presentation, and in 20.1% of HIV patients with previous neurological involvement in 8.2% among HIV patients with seizure as presentation. In the study 93.9% of patients had opportunistic infections responsible for seizures including tuberculosis, cryptococcosis and toxoplasmosis affecting the central nervous system. Epilepsia partialis continue is defined as a variant of simple focal motor status epilepticus, characterized by stereotyped, arrhythmic repetitive muscle jerks, affecting single muscles, muscle groups, an entire limb or larger parts of one hemibody continuing over prolonged periods of time. EPC has been included in subclass of focal motor status, in the ILAE task force report on status epilepticus. There are very few case studies reporting epilepsia partialis continua as the presenting symptom of HIV infection. The present case has several peculiarities, firstly this case presented epilepsia partialis continua with secondary generalization. There was worsening of sensorium with refractory status epilepticus, necessitating endotracheal intubation, IV Benzodiazepine Infusion, IV Immunoglobulins and four anti-epileptic drugs. He made uneventful clinical recovery over 3 weeks period. It have reported a case with Isolated focal mycoclonic jerks involving left upper limb with rare secondary generalization. The case was managed with prednisolone, carabamazepine and ART, and showed gradual protracted recovery over six month period, with radiological resolution 4. The other two reported case series reported no significant change in outcomes with treatment. Treatment with IV Immunoglobulins in this setting has not been previously described. Initiation of immunomodulation along with aggressive ART plays an important role in clinical profile suggestive of epilepsia partialis continua and HIV infection (Table 1).

Secondly, sequential MRI Imaging of Brain showed progressive increase in distribution of lesions, initial imaging showing right Precentral gyral thickening, which were non-enhancing followed by signal changes in left cortico -spinal tract as well as changes in sub-cortical region (globus pallidus) and Cerebellar hemisphere. CSF PCR

Author	Ferrari et al.[4]		Bartolomei et al.[5]	Ramanujan et al.[6]	Present case
Year	1998		1999	2016	2021
Number	Two		One	One	One
Age, years	39	36	54	14	21
Gender	Male	Male	Male	Male	Male
Semiology of seizure	EPC-Right UL	EPC-Left UL	EPC-distal right upper limb	Generalized tonic clonic seizure followed by EPC involving Left half of body	Complex partial seizure followed by EPC involving distal left upper limb
MRI brain	Bilateral frontal cortical and subcortical	Right frontal cortical	Left pre-central sulcus	Right parieto-occipital cortex and thalamus	Right precentral sulcus, followed by bilateral pre-central sulcus
EEG	Spike wave discharges-left frontal	Normal	Irregular theta rhythm over left fronto-central region	PLEDS-right fronto-central region.	PLEDs-right Fronto-central region
CSF Study Cytology (per cmm, pred. cells) Protein Glucose PCR	No cells	Normal	No cells	400, Neutrophils	Normal
	Normal	68	Normal	120	Normal
	Normal	Normal	Normal	60	Normal
	JCV +	JCV +	Negative	CMV Positive	Negative
CD4 Tcell count	60	270	-	51	95
HIV RNA	-	-	-	-	163771
Management ART	Didanosine	Details N/A	Dideoxycyti-dine, Indinavir,	Acyclovir, Meropenem	Tenofovir, Lamivudine, Effavirenz Perampanel,
AED	Clonazepam		Lamivudine Carbamazepine		Oxcarbazepine, Levitracetam, Lacosamide
Outcome	Death	Death	Improved, resolution of MRI changes	Death	Improved, resolution of MRI changes

Table 1: Previous case reports: EPC on presentation in HIV Patients.

negative for HSV, CMV, JCV, *pneumococus* and *Cryptococcus*. These lesions were Non-contrast enhancing, hyperintense on T_2 / FLAIR, no significant change on T_1 sequence (unlike lesions in progressive multifocal leucoencephalopathy). These signal changes have not been previously reported in HIV-related Encephalitis. HIV related cortical lesions can be due to CMV, toxoplasma, lymphoma, and PML. Previously reported case from AIIMS, EPCs were secondary to CMV-related Encephalitis in HIV (MRI brain showing signal changes in the parieto-occipital cortex and thalamus). It have reported two cases with EPC secondary to PML (proved on CSF positive for JCV PCR, and other case brain biopsy positive for JCV PCR), with MRI brain showing features of PML involving frontal cortical and sub-cortical region.

MRI imaging in CMV encephalitis is characterized by ventricular prominence, enhancement in the periventricular subependymal lining along the lateral ventricles, septum pellucidum, corpus callosum and fornices due to ventriculitis, or diffuse or patchy increased signal intensities in white matter owing to demyelination. Toxoplasma encephalitis is radiologically characterized by usually multiple ring enhancing lesions with surrounding edema, and predilection for basal ganglia. Primary CNS Lymphomas usually have solitary and multiple mass lesions with approximately equal frequency, and majority of lesions display irregular or patchy contrast enhancement. Besides, lesions larger than 4 cm in size, and with involvement of corpus callosum or peri-ventricular/periependymal areas are more likely to be lymphoma. PML usually occurs as non-contrast enhancing periventricular and subcortical, bilateral asymmetrical lesions with no surrounding edema. However, PML lesions in setting of Immune Reconstitution Inflammatory Syndrome (IRIS) associated with increase in CD4 T cell counts and drop of HIV plasma viral load on ART. In this context, PML/IRIS can present with contrast enhancement on MRI, as wellas focal edema and mass effect.

Periodic Lateralized Epileptiform Discharges on EEG in HIV patients have been described by (PLEDS over the right fronto-polar region, MRI revealing meningeal thickening along with nonspecific findings), (Aphasic status epilepticus and PLEDS on EEG, as the first presentation of AIDS-toxoplasma complex), (EPCs secondary to AIDS-associated CMV encephalitis) and (periodic lateralized epileptiform discharges in neurosyphilis and HIV Infection).

Conclusion

Cases presenting with acute onset EPC should undergo extensive evaluation, including HIV seropositivity. The present case has shown that early initiation of immunomodulation along with anti-epileptic drugs was helpful in bringing excellent clinical outcomes.

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