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Case Series

Paediatrics Section

Diagnostic and Management Complexities in Epilepsia Partialis Continua: A Case Series

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ABSTRACT

Epilepsia Partialis Continua (EPC) is a variant of focal motor status epilepticus wherein frequent repetitive, usually rhythmic muscle jerks continue over prolonged periods of time. EPC can occur because of any space-occupying lesion, vascular aetiology, mitochondrial disease, and Rasmussen encephalitis. There is a paucity of evidence regarding the prevalence; however, regional studies have suggested a prevalence of one per million population. This case series presents three cases wherein the aetiology has been known to have a highly rare correlation with the clinical presentation of EPC. Index case one, already a known case of generalised epilepsy, presented with clinical features suggestive of acute viral hepatitis, later found to be hepatitis A positive, had EPC that lasted around four days. In index case two, the child presented with features suggesting focal status epilepticus, which also persisted despite use of multiple anti-epileptic agents, and was later found to be positive for parvovirus antibodies in serum as well as Cerebrospinal Fluid (CSF). In index case three, the child presented with focal seizures, which on imaging had structural changes and an abnormal Electroencephalography (EEG). These children were given multiple antiepileptic agents; however, the repetitive movement subsided on its own and all of them were discharged on haemodynamically stable conditions. The present case series highlights the clinical heterogeneity of the disease process, as well as the challenges and complexities involved in diagnosis and management.

Keywords: Antiepileptic agents, Focal seizures, Parvovirus, Tonic-clonic movements

CASE REPORT

The Epilepsia Partialis Continua (EPC) is a variant of focal motor status epilepticus wherein frequent repetitive, usually rhythmic muscle jerks continue over prolonged periods of time, which can occur because of any space-occupying lesion, vascular etiology, mitochondrial disease, and Rasmussen encephalitis [1]. There is a paucity of evidence regarding the prevalence; however, regional studies have suggested a prevalence of one per million population [2]. Herein, we present three highly interesting cases wherein the aetiology of EPC is known to have a rare correlation with the clinical presentation. Index case one, already a known case of generalised epilepsy, presented with clinical features suggestive of acute viral hepatitis, later found to be hepatitis A positive, had EPC that lasted around four days. In index case 2, the child presented with features suggesting focal status epilepticus, later found to be positive for parvovirus antibodies in serum as well as CSF. In index case 3, the child presented with focal seizures, which on imaging had structural changes and an abnormal EEG. These children were given multiple antiepileptic agents; however, the repetitive movement subsided on its own and both were discharged on oral anti epileptics and advised regular Outpatient Department (OPD) follow-up.

CASE SERIES

Case 1

A 12-year-old male child presented to the paediatric OPD with complaints of yellowish discolouration of the whole body, high-grade fever, decreased appetite and abdominal pain for five days. The child was a known case of generalised epilepsy since the age of four years. He was on valproate therapy at 40 mg/kg/day and was seizure-free for the past three years. Prior neurological investigations were normal. He was first in birth order, born out of a non-consanguineous marriage and had an uneventful birth history. He had been immunised till five years of age. There was no family history of any seizure disorder or intake of anti-epileptic drugs in the family.

On general physical examination, the child was conscious and well oriented to time, place and person. Vitals were stable with heart rate 92 beats per minute, respiratory rate 26 per minute, blood pressure was 102/64 mmHg measured in the right arm (<90th percentile), oxygen saturation at room air 98%, and temperature 100.2 degrees Fahrenheit. Icterus was present with no pallor, clubbing, cyanosis, lymphadenopathy and oedema. Abdominal examination revealed right hypochondrium tenderness, with the liver palpable 2 cm below the right subcostal margin and spanning 16 cm. The central nervous system examination was remarkable with a Glasgow Coma Scale (GCS) score of 15/15, no cranial nerve involvement, power in all four limbs as per Medical Research Council (MRC) grade 5/5, no hypo/hypertonia, no sensory deficits and absence of cerebellar signs. Cardiovascular system and respiratory system examination was unremarkable. Biochemical investigations done were as follows [Table/Fig-1].

TLC (cells/cumm)	18000
AST/ALT (U/L)	887/798
Albumin (g/dL)	3.6
Serum bilirubin(total/direct/indirect) (mg/dL)	5/2.5/2.5
INR	1.3
Blood urea (mg/dL)	33
Serum creatinine (mg/dL)	0.6
IgM Hepatitis A	Positive
Hep B/C	Non-reactive
IgM for Leptospira/Scrub typhus/Brucella	Negative
Anti-nuclear antibody	Negative
Serum ammonia	52
Cerebrospinal Fluid (CSF) (sugar/protein)	70 mg/dL, 22 g/dL
Cerebrospinal Fluid (CSF)- Polymerase chain reaction	Negative

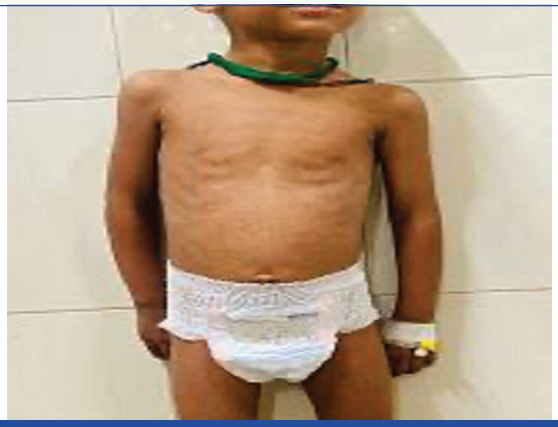
[Table/Fig-1]: Summary of biochemical investigations done in index case 1.

The child was admitted and a provisional diagnosis of hepatitis was made. Intravenous fluids and antibiotics (ceftriaxone@50 mg/kg/dose in two doses) were started. On day 3 of admission, the child developed jerky tonic clonic movements of the left lower limb with no loss of consciousness, which lasted for around 72 hours, despite the use of multiple antiepileptic drugs (levetiracetam, carbamazepine and clobazam). Child had mild intellectual disability (Intelligence quotient: 80). Child was continued on levetiracetam at 60 mg/kg/day, clobazam at 5 mg OD, and ox carbamazepine at 10 mg/kg/day. The focal motor tonic-clonic movements started to subside by day 6 of admission; hence, the antiepileptics were gradually tapered off. A diagnosis of EPC was made based on the clinical findings. Child was shifted to oral antiepileptics (oxcarbamezapine@8 mg/kg/day, levetiracetam@40 mg/kg/day) and clobazam@5 mg OD) and discharged on haemodynamically stable conditions. He was followed up and is doing well at the three-month follow-up.

Case 2

An eight-year-old male presented to the paediatric OPD with complaints of high-grade fever and continuous focal tonic clonic movements of the left upper and lower limbs and twitching of the left cheek for four days. He was first in birth order, born out of a non-consanguineous marriage and had an uneventful birth history. He had been immunised till five years of age. There was no family history of any seizure disorder or intake of any anti-epileptic drugs in the family. There was no prior history of hospital admissions.

On examination, child was sick appearing, with no pallor, icterus, clubbing, cyanosis, lymphadenopathy or oedema; however, vitals were stable with heart rate 92 beats per minute, respiratory rate 26 per minute, blood pressure was 100/60 mmHg measured in right arm (<90th percentile), oxygen saturation at room air 96%. The child had the classic 'slapped cheek appearance' of parvovirus infection and a macular rash over the trunk [Table/Fig-2]. Central nervous system examination was remarkable with GCS score 15/15, no cranial nerve involvement, power in all four limbs as per MRC grade 5/5, no hypo/hypertonia, no sensory deficits and absence of cerebellar signs. Cardiovascular, abdominal and respiratory system examinations were unremarkable.



[Table/Fig-2]: Rash (maculopapular) over the body of index case 2.

Child was admitted, investigated and managed conservatively with intravenous fluids, intravenous antibiotics (ceftriaxone) and anti-epileptic drugs including valproate (@40 mg/kg/day), phenytoin (10 mg/kg/day), levetiracetam (@60 mg/kg/day), despite which the movements did not subside and hence continuous infusion of midazolam at 2 microgram/kg/min was started. The tonic-clonic movements of the left half of the body lasted for around six hours and persisted despite the use of anti-epileptic drugs, including valproate, phenytoin, levetiracetam and continuous infusion of midazolam. After about eight hours of seizure-free activity, the left focal tonic-clonic movements restarted and further continued for about 96 hours (The focal motor activity persisted; however, the child was conscious) despite the use of other anti-epileptic drugs like topiramate, clonotril and carbamazepine. Biochemical investigations done were as follows [Table/Fig-3]:

Hb (g/dL)	10.2
TLC (cells/cumm)	7600
Platelets	3.3
AST/ALT (U/L)	42/34
Albumin (g/dL)	3.8
Serum bilirubin (total/direct/indirect) (mg/dL)	0.9/0/3/0.6
INR	1.2
Blood urea (mg/dL)	22
Serum creatinine (mg/dL)	0.5
IgM Hepatitis A/E	Negative
Hep B/C	Non-reactive
Widal	Negative
IgM for Leptospira/Scrub typhus/Brucella	negative
IgM parvovirus	Positive
Cerebrospinal Fluid (CSF) (sugar/protein)	74 mg/dL, 25 g/dL
Cerebrospinal Fluid (CSF)- Polymerase chain reaction	Negative
CSF- Parvovirus	Positive

[Table/Fig-3]: Summary of biochemical investigations done in index case 2.

The child was managed conservatively with intravenous steroids (methyl prednisolone @30 mg/kg/day) for five days and Intravenous Immunoglobulin (IVIG), at 2 gm/kg for two days. By day 5 of admission, the fever spiked and tonic-clonic movements started to settle and anti-epileptics were gradually tapered and shifted to oral therapy. The child was discharged on haemodynamically stable conditions on oral prednisolone at 2 mg/kg/day and topiramate. The child is currently doing well on a three-month follow-up and the steroids were tapered and eventually stopped.

Case 3

A three-year-old male, second in birth order, born out of a non-consanguineous marriage, presented to the paediatric OPD with complaints of fever for two days and five episodes of abnormal body movements in the form of tonic-clonic movements of the right upper and lower limbs along with deviation of the eyeballs to the right side that lasted for five minutes, with no loss of consciousness. He was developmentally normal, fully immunised for his age and had an uneventful birth history. There was no family history of seizure disorders.

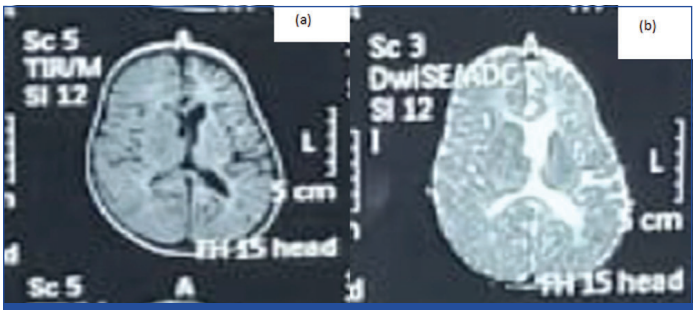
On general physical examination, the child was conscious and well oriented to time, place and person. Vitals were stable with heart rate 86 beats per minute, respiratory rate 24 per minute, blood pressure 98/74 mmHg measured in the right arm (<90th percentile) and oxygen saturation at room air 97%. There was no pallor, icterus, clubbing, cyanosis, lymphadenopathy and oedema. The central nervous system examination revealed right-sided hemiparesis, with power in both right upper and lower limbs, as per MRC grade 2/5 and 5/5 in left upper and lower limbs. There was no cranial nerve involvement, no sensory deficit, no involvement of the cerebellum and absence of meningeal signs. Respiratory system, cardiovascular system and abdominal examination revealed no abnormalities. Biochemical investigations are as follows [Table/Fig-4].

Hb (g/dL)	10.2
TLC (cells/cumm)	5200
Platelets	3
AST/ALT (U/L)	22/24
Albumin (g/dL)	3.6
Serum bilirubin (total/direct/indirect) (mg/dL)	0.4/0.2/0.2
Blood urea (mg/dL)	33

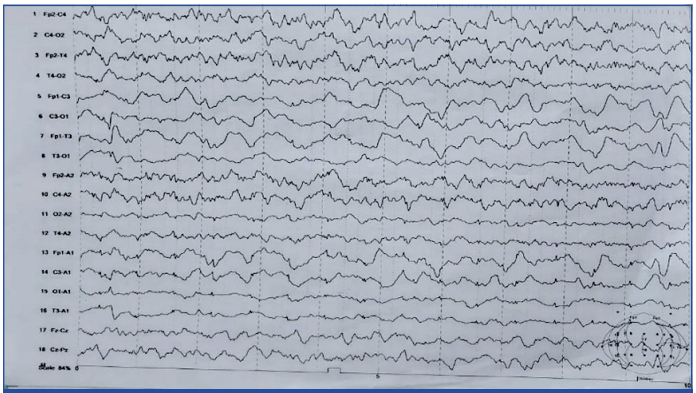
Serum creatinine (mg/dL)	0.6
Serum sodium/potassium (mEq/L)	137/3.8
Cerebrospinal Fluid (CSF) (sugar/protein)	88 mg/dL, 26 g/dL
Cerebrospinal Fluid (CSF)- Polymerase chain reaction	Negative

[Table/Fig-4]: Summary of biochemical investigations done in index case 3.

On neuroimaging, Magnetic Resonance Imaging (MRI) was suggestive of hemi-atrophy of left cerebral hemisphere along with atrophy of the left caudate nucleus with subtle diffusion restriction and mildly increased signal of left cerebral cortex in the parietal occipital region on diffusion and dilatation of the left lateral ventricle with mildly prominent sulcal spaces and a left Sylvian fissure with increased diffusion in left caudate and left posterior cortex [Table/Fig-5]. Electroencephalography (EEG) revealed an abnormal awake EEG record, suggesting a left hemispheric epileptic focus with a possibility of Rasmussen encephalitis [Table/Fig-6].



[Table/Fig-5]: MRI Brain of index case 3. (a) T1 weighted image showing hemiatrophy of the left cerebral hemisphere with atrophy of the left caudate nucleus and dilatation of the left lateral ventricle with mildly prominent sulcal spaces and a left Sylvian fissure. (b) T2 weighted image showing atrophy of the left caudate nucleus with subtle diffusion restriction within mildly increased signal of the left cerebral cortex in the parietal occipital region on diffusion.



[Table/Fig-6]: EEG of index case 3 shows stage II NREM sleep in the right hemisphere with delta slowing in the left hemisphere. There are abundant spike-wave discharges (100-150 uV, 2-2.5 Hz) seen arising from all left hemispheric leads.

Child was started on anti-epileptic drugs, levetiracetam @30 mg/kg/day and clobazam @5 mg OD along with initiation of systemic steroid, methylprednisolone at a dose of 30 mg/kg/day for five days, after which he improved and was shifted to oral steroids (prednisolone at 2 mg/kg/day) with tapering over a period of one month and oral anti epileptics. The child was discharged on haemodynamically stable conditions and is doing well on a four-month follow-up.

A summary of all the cases is presented in [Table/Fig-7].

DISCUSSION

Focal motor clonic and/or myoclonic seizures that persist for days, months, or even longer are termed EPC, which may be chronic progressive or non-progressive. They are an unusual manifestation of epilepsy wherein more typical paroxysmal events are replaced by sustained repetition of seizure fragments in rapid succession. It is a rare form of epilepsy that impacts both the paediatric and adult populations [3]. The causes of EPC are somewhat similar to that of any other form of epilepsy as the basic reason for seizures is abnormal electrical activity in the brain brought on by a myriad of pathologies including architectural damage, cortical dysplasia (such as Tuberous Sclerosis, Sturge-Weber syndrome, and linear sebaceous nevus syndrome), drugs, metabolic abnormalities, neoplastic process (oligodendroglioma, meningioma, high-grade glioma), autoimmune processes (autoimmune encephalitides), or infections. Neuronal cell loss, astrogliosis, and damage to the blood-brain barrier are the contributing factors for the hyperexcitability of the cerebral cortex and are usually associated with postictal confusion and weakness [4].

Clinical manifestations vary highly in severity, duration and seizure semiology, ranging from second-long jerks to abnormal generalised motor activity lasting days. It is primarily a clinical diagnosis and the prognosis is more favourable if the underlying aetiology is addressed. Although most paediatric cases have been reported to have residual psychomotor deficits [5]. It is understood that cortical hyperexcitability or slow rhythmic activity can play a prominent role in EPC; however, EEG findings are poorly characterised and may not be identified if the abnormalities are too deep to be identified with the common scalp electrode diagnostics. In an EEG-Functional MRI (fMRI) study of a patient with a perirolandic Focal Cortical Dysplasia (FCD) and EPC, Vaudano AE et al., demonstrated event-related BOLD signal increase, indicating a network maintaining epileptic activity and including the ipsilateral anterior cingulate and occipital cortex, the bilateral prefrontal cortex and the contralateral cerebellum but not the thalamus [6]. Furthermore, there are four previously-reported cases of paediatric patients ranging from 2 to 16 years of age with EPC or focal Non-Convulsive Status Epilepticus (NCSE) initially concerning for Rasmussen encephalitis and found to have definitive NMDAR receptor positivity [Table/Fig-8] [7-10].

In the index case one, although hepatitis A is not an established cause of EPC, it precipitated the continuous focal tonic-clonic movement. In the second case, the parvovirus B19 was supposedly the cause of EPC. These findings emphasise the importance of maintaining a wide differential when evaluating focal jerky movements. In index case three, a structural anomaly was the cause of hemiparesis and tonic-clonic movements; therefore, it is essential to have a high index of suspicion even for rarer pathologies. [Table/Fig-7] summarises

Index case	Age/ Sex	Symptoms	Etiology	Biochemical findings	EEG	Neuroimaging	Treatment	Recovery
1	12 year/ male	Jaundice, fever, focal seizure	Hepatitis A virus	IgM hepatitis A positive	Normal	Normal	Oxcarbamezapine @8 mg/kg/day, levertiracetam @40 mg/kg/day and clobazam @5 mg OD	Yes
2	8 year/ male	Fever, focal seizure	Parvovirus	IgM parvovirus positive in serum and Cerebrospinal Fluid (CSF)	Normal	Normal	Intravenous steroids methyl prednisolone @30mg/kg/day for 5 days and Intravenous Immunoglobulin (IVIG), at 2 gm/kg for 2 days.	Yes

3	3 year/ male	Fever, focal seizure	Structural	Normal biochemical findings	left hemispheric epileptic focus	Hemiatrophy of the left cerebral hemisphere, along with atrophy of the left caudate nucleus	Levetiracetam (@30 mg/kg/day) and Clobazam @5 mg OD along with initiation of systemic steroid, methyl prednisolone at a dose of 30 mg/kg/day for 5 days, later shifted to oral steroids (prednisolone at 2 mg/kg/day)	Yes

[Table/Fig-7]: Summary of all three cases in the current study.

Author	Place/Year	Age/gender	Chief complaint	Findings
Chheda A et al., [7]	Mumbai, India; 2020	16 years/female	A girl with epilepsy (onset at five years) presented with an acute increase in seizure frequency and behavioural changes.	Interictal EEG was suggestive of an intermittent right frontocentral and centroparietal delta rhythm with generalised background slowing right hemispheric extreme delta brush pattern. Her serum and CSF anti-NMDA antibody levels were strongly positive. LGI-1, AMPA, and GABA-A/B antibodies in serum and CSF were negative
Gurucharran K and Karkare S [8]	New York, USA; 2017	2 years/female	Patient presented with progressive right weakness and Epilepsia Partialis Continua (EPC)	She tested positive for anti-NMDA receptor antibodies, later experienced complete clinical recovery after immunotherapy. Anti-NMDA receptor antibodies were absent at three weeks and again at one year after the first treatment of Intravenous Immunoglobulin (IVIG).
Goldberg EM et al., [9]	Philadelphia, PA, USA; 2011	99 years/female	TThe patient presented to an Emergency Department (ED) with headache, dizziness, blurry vision, and abnormal visual perceptions	Over the course of days, became progressively somnolent and was found to have a right inferior quadrantanopsia and sixth nerve palsy. Magnetic Resonance Imaging (MRI) of the brain showed gyral swelling of the left parieto-occipital lobe and Continuous Electroencephalogram (EEG) monitoring revealed focal Non-Convulsive Status Epilepticus (NCSE) in the left occipital region. Cerebrospinal Fluid (CSF) was positive for antibodies directed against the N-Methyl-D-Aspartate Receptor (NMDAR). This case is the first report of anti-NMDAR encephalitis presenting with focal Non-Convulsive Status Epilepticus (NCSE).
Kim EH et al., [10]	Seoul, Korea; 2016	93 years/female-	A young child presented with focal status epilepticus, followed shortly by other characteristic manifestations	The patient showed gradual improvement of motor and cognitive function. This case serves as an example that a diagnosis of anti-NMDAR encephalitis should be considered when children with uncontrolled seizures develop dyskinesias without evidence of a malignant tumour. In these cases, aggressive immunotherapies are needed to improve the outcome of anti-NMDAR encephalitis.
Present index case A	India	12 year/male 8 years/male 3 years/male	Index case one, already a known case of generalised epilepsy, presented with clinical features suggestive of acute viral hepatitis. In index case two, the child presented with features suggesting focal status epilepticus, later found to be positive for parvovirus antibodies in serum as well as CSF. In index case three, the child presented with focal seizures.	Our case series of three patients highlights the clinical heterogeneity of EPC, as well as the challenges and complexities of diagnosis and management. Prompt recognition and diagnosis of EPC are critical for optimising treatment and preventing long-term cognitive and motor sequelae.

[Table/Fig-8]: Summary of previously-reported cases of paediatric patients with EPC or focal Non-Convulsive Status Epilepticus (NCSE) initially concerning for Rasmussen encephalitis and found to have definitive NMDAR receptor positivity [7-10].

the clinical data and outcome of the index cases. The treatment of EPC is decided upon primarily by the underlying aetiology. Benzodiazepines can interrupt EPC and serve as a diagnostic aid in aura continua; however, they are not a good long-term treatment. In the European survey, the relatively best results for continuous treatment were obtained with topiramate and levetiracetam [11]. The management involves treatment of the primary pathology supposedly causing the focal motor activity; however, newer techniques such as repetitive transcranial stimulation and botulinum toxin have reported some degree of success in specific cases [12].

CONCLUSION(S)

The EPC is a rare epilepsy syndrome characterised by continuous focal motor activity. Our case series of three patients highlights the clinical heterogeneity of EPC, as well as the challenges and complexities of diagnosis and management. Prompt recognition and diagnosis of EPC are critical for optimising treatment and preventing long-term cognitive and motor sequelae.

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