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Case report

Lance-Adams Syndrome: Case series and literature review

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ABSTRACT

Background: Lance-Adams Syndrome (LAS) is a rare complication of successful cardiopulmonary resuscitation (CPR). It is a form of posthypoxic myoclonus characterized by action or intention myoclonus developing days to months after an hypoxic insult to the brain. LAS, especially early in a patient's clinical course, can be challenging to diagnose. Electroencephalogram (EEG) pattern of midline spike-wave discharge associated with favorable prognosis. There is no consensus in treatment of LAS but use of various anti-epileptic medications has been documented in literature.

Case Presentation: In this case series, all of the patients presented after achieving return of spontaneous circulation (ROSC) and subsequently developed myoclonus. EEG findings beyond the initial hospitalization continued to show later showed changes consistent with LAS in three of the four patients. Different combinations of AEDs were used in the management of LAS with variable success and adverse effects.

Conclusion: Our cases highlight that the characteristic EEG pattern can be useful in the diagnosis of LAS and allow for better prognostication and management in patients with posthypoxic myoclonus. We reviewed the available literature to better understand the prevalence, mechanism, clinical presentation, diagnosis, and management of LAS.

1. Introduction

Posthypoxic myoclonus is a term used to describe myoclonus after a hypoxic injury to the brain. They are of two types, acute, also known as posthypoxic myoclonus status epilepticus (MSE) and chronic, Lance-Adams Syndrome (LAS) (Lee and Lee, 2011). MSE starts hours after the injury and is associated with a poor prognosis (Lee and Lee, 2011). LAS typically starts days to weeks after the initial hypoxic injury and is associated with a more favorable outcome (Malhotra and Mohinder, 2012; Lee and Lee, 2011). Early in the course of the disorder and under the effects of sedation it can be difficult to distinguish between the two diagnoses (Malhotra and Mohinder, 2012). In face of the differences in management and prognosis, it is vital to differentiate between MSE and LAS. EEG is an essential, but often overlooked, diagnostic tool in the evaluation of LAS; showing characteristic polyspike waves that are highest in amplitude at the vertex (Elmer et al., 2016). EEG can provide clinicians with the ability to provide accurate prognoses and management in patients who have hypoxic brain injuries.

In this case series, we describe four cases of patients who suffered cardiac arrest and were successfully resuscitated. Over their clinical course, each patient exhibited myoclonus and showed characteristic EEG findings consistent with LAS. Table 1 summarizes the clinical characteristics, imaging profile, EEG characteristics, treatment and patient's outcomes in this case series.

2. Case 1

Patient is a 45-year-old male who had overdosed on fentanyl, became apneic, and then went into cardiac arrest. He was resuscitated in a few minutes, intubated, then extubated 2 days later. After extubation he was awake, alert, oriented to person, place, and time. He had lactic acidosis that eventually resolved, pneumonia that was treated, and underwent cardiac catheterization which did not reveal signs of coronary artery disease. He was discharged home 5 days later. EEG was not done during the initial hospitalization at the outside hospital for cardiac arrest. Approximately 7 days after arrest, he started experiencing negative myoclonus and hand

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Table 1
Summary of the demographics, clinical characteristic, imaging profile, EEG characteristics, treatment and patient's outcomes.

Case	Age and Sex	Clinical Characteristics	Imaging	EEG Findings	Timing of EEG finding	Treatment	Outcome
1	45 M	Generalized, worse in upper extremities, intention myoclonus and spontaneous myoclonus extremities	MRI: Mild restricted diffusion and enhancement in bilateral basal ganglia	Generalized central maximum bursts of pol- spikes/spike-slow wave discharges and brief subtle myoclonic seizures	First 7 days post cardiac arrest	Valproate was ineffective Levetiracetam 750 mg twice daily.	Improvement in frequency of myoclonus after start of levetiracetam, but myoclonus persisted
2	46 M	Intention myoclonus, spontaneous myoclonus and stimulus induced myoclonus involving all extremities that began 4 days after being resuscitated	Nonspecific findings on MRI	Days after cardiac arrest, left sided PLEDs and GPDs. After days to weeks, generalized midline maximum polyspike and spike with slow wave complexes, associated with myoclonic jerks.	Days - 5 months post-cardiac arrest	Clonazepam 2 mg tid, Lacosamide 200 mg bid, Clobazam 10 mg bid, Levetiracetam 1500 mg bid, Valproate 750 mg DQ/ 1000 mg HS. Zonisamide recently started	Continued to have myoclonus but frequency improved after increase in AEDs. Effects of zonisamide unknown.
3	34 M	Stimulus induced myoclonus involving the upper extremities and eyelids	CT: Poor gray/white matter differentiation with mild effacement in the sulci and cisterns.MRI: Normal	Generalized poly-spike and wave discharges with central maximum associated with bilateral upper extremity myoclonic jerks, consistent with electroclinical seizure	Within 24 h post-cardiac arrest	Levetiracetam 2000 mg BID Valproate 500 mg TID, Phenobarbital 50 mg BID, Clobazam 20 mg bid	Myoclonus was well controlled with medication although needed significant assistance with activities of daily living
4	56 M	Generalized action myoclonus aggravated by stress more prominent in the lower extremities.	CT: UnremarkableMRI: Mild to moderate cerebral atrophy and several small nonspecific white matter foci.	Irregular bursts of generalized spike, polyspike, and slow wave complexes frequently associated with myoclonus of limb or body.	2 years post-cardiac arrest	Oxcarbazepine, valproate, and lorazepam were discontinued due to adverse effects, Levetiracetam 1000 mg BID was continued	Myoclonus was well controlled on levetiracetam alone.
5	63F	Generalized tonic-clonic seizure began days after cardiac arrest, later developed generalized myoclonus involving the trunk and bilateral limbs and rare generalized tonic-clonic seizures	CT: None MRI: Several nonspecific FLAIR white matter intensities but otherwise unremarkable	Intermittent bifrontal and generalized spike and wave discharge. At times, the myoclonus was associated with a brief 0.5–1 s burst of generalized spike and wave activity.	7 years postcardiac arrest	Levetiracetam 1500 mg BID Clonazepam 0.5 mg TID Lacosamide 150 mg BID Valproate 250 mg BID	Infrequent myoclonus, had seizure when medications were decreased

tremors, for which he had presented to our hospital for evaluation and management. Brain MRI showed subtle signal abnormalities and mild restricted diffusion and contrast enhancement in the basal ganglia near the genu of the internal capsule bilaterally. Electroencephalogram (EEG) showed generalized, frontal/midline maximum, bursts of polyspikes and spike-slow wave discharges and brief subtle myoclonic seizures involving perioral region and eyelids (Fig. 1). Valproate 500 mg three times daily was ineffective in controlling the myoclonus and was later switched to levetiracetam 750 mg twice daily which provided marked improvement in myoclonus. At his 6 month follow-up visit, neurological examination continued to show persistent appendicular myoclonic jerks (present only when awake) and tremors in the right more than the left arm. He was recently started on zonisamide for myoclonus and has not yet presented for follow up.

3. Case 2

Patient is a 46-year-old male who had cardiac arrest from overdose of multiple drugs including benzodiazepine, cocaine and opioids. He was hospitalized for almost one month, underwent targeted temperature management. He had multiple seizures which were eventually controlled on anesthetic agents and antiepileptic medications. He later developed both stimulus induced and spontaneous myoclonus often involving the entire body which had a significant impact on performing activities of daily living. CT brain and MRI brain several days after arrest were unremarkable. His seizures were controlled on lacosamide 200 mg twice daily, levetiracetam 1500 mg twice daily, clobazam 10 mg in the morning and 5 mg in the evening, clonazepam 10 mg three times daily. He was eventually discharged to a long-term care facility after PEG and tracheostomy placement. Valproate and phenobarbital were tried but discontinued due to hyperammonemia. He had both generalized and multifocal myoclonus (including extremities and eyelids) which were often stimulus induced. On EEG, he had generalized midline-maximum polyspike and spike-slow wave discharges (Fig. 2) and abundant generalized epileptic myoclonic jerks which were spontaneous and stimulus induced. These myoclonic jerks and associated spike and slow wave complexes were significantly reduced during the periods of sleep. He was eventually put on clonazepam 2 mg three times daily, lacosamide 200 mg twice daily, clobazam 10 mg twice daily, levetiracetam 1500 mg twice daily, and valproate 750 mg in the morning and 1000 mg in the evening which provided significant improvement of the myoclonus. He continues to undergo seizure medication adjustments due to adverse effects and better clinical symptoms

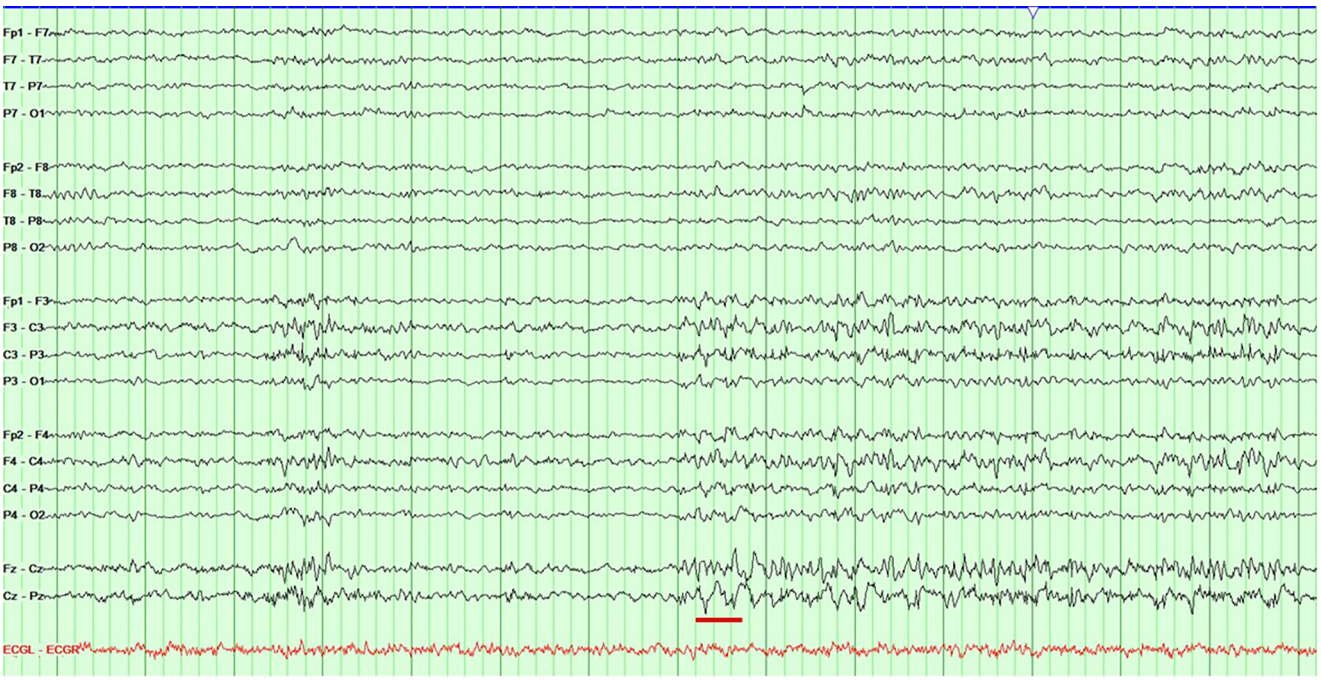

Fig. 1. Interictal electroencephalogram 7 days post-cardiac arrest, Longitudinal Bipolar Montage, Time base = 30 mm/sec, sensitivity = 7 μV/mm, low frequency filter = 1 Hz, high frequency filter = 70 Hz, Notch = 60 Hz showing poly-spike with central maximum (underlined in red), central maximum associated with brief subtle myoclonic seizures involving perioral region and eyelids. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

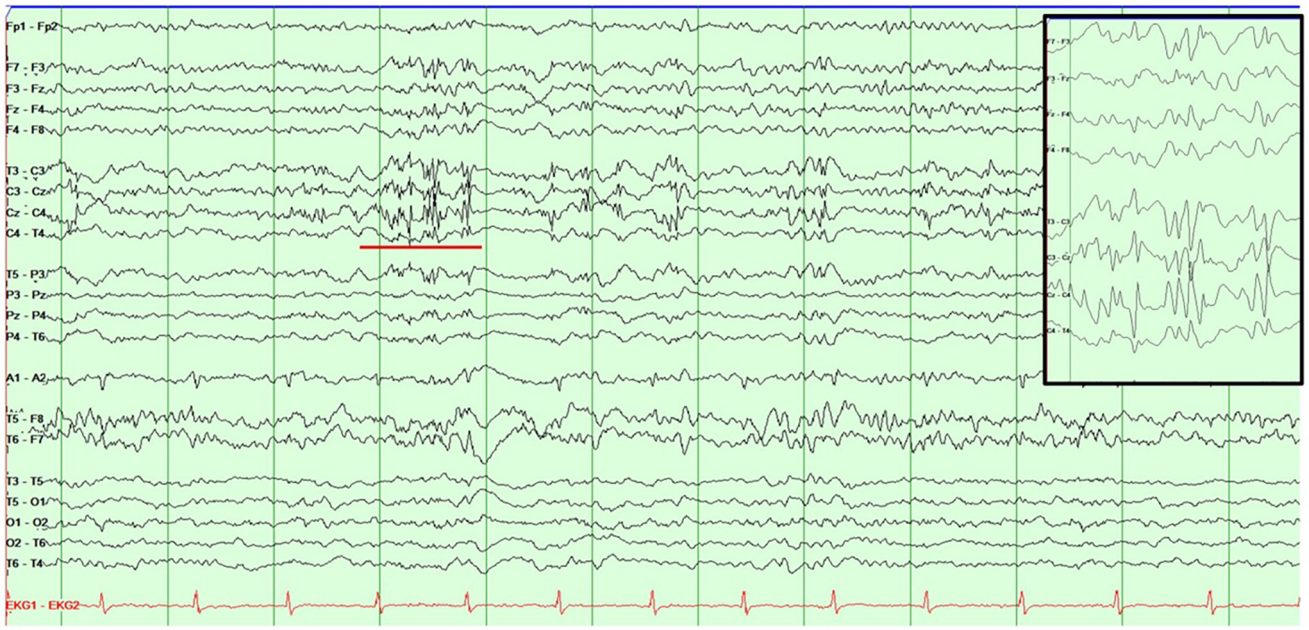

Fig. 2. Interictal and ictal EEG, Longitudinal Bipolar Montage, Time base = 30 mm/sec, sensitivity = 10 μV/mm, low frequency filter = 1 Hz, high frequency filter = 70 Hz, Notch = 60 Hz, showing poly-spike and slow-wave discharges with central maximum (underlined in red) associated with generalized myoclonic jerks. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

control. He started on zonisamide during the most recent clinic visit, and effects are not yet known.

4. Case 3

Patient is a 34 year-old male who had cardiac arrest of unclear etiology and unknown amount of downtime when he was found unresponsive behind a dumpster. After resuscitation, he was hemodynamically unstable, had multiple lab abnormalities including hyperammonemia, lactic acidosis, transaminitis, leukocytosis. He exhibited myoclonic jerks within hours of resuscitation. On the initial neurological exam, he was unresponsive to pain, sluggish pupillary reflex but lacked other brainstem reflexes, and had stimulus induced myoclonus of the upper extremities and eyelids.

He had myoclonus and generalized tonic-clonic seizures. CT brain and MRI brain several days later were normal. After seizures were controlled on anti-seizure medications and weaned off of sedation, the patient had spontaneous eye opening and started following simple commands. EEG showed external stimulation induced intermittent, low amplitude spike/polyspike-slow discharges predominantly in the midline (Fig. 3), with spread to the bilateral parasagittal chains with normal background during periods of restfulness. He was discharged on Levetiracetam 2000 mg twice daily, Valproate 500 mg three time daily, Phenobarbital 50 mg twice daily, Clobazam 20 mg twice daily which provided marked improvement in his myoclonus and seizure control. After an extended hospital stay with minimal improvement in neurological exam, the patient was eventually discharged to a long term acute care facility.

5. Case 4

This patient had a cardiac arrest at the age of 56 after he had sustained severe burns over 35% of his body. He had cardiac arrest with 25 min of downtime during surgery for skin graft. Within days of cardiac arrest, he had gaze deviation, and myoclonus, intact brainstem reflexes while intubated and sedated. EEG reported severe background suppression and lateralizing periodic epileptiform discharges suggestive of acute cortical injury but events of eye deviation and myoclonus did not have electrographic correlate. CT brain was unremarkable. Following the cardiac arrest, he had myoclonus, cognitive impairment, dysarthria, gait disturbance, mood disturbances, mild ataxia, and infrequent generalized tonic-clonic seizures. His myoclonus was well controlled on levetiracetam, lacosamide, and clonazepam at the time of initial hospital discharge. He had tried other seizure medications including topiramate, oxcarbazepine, valproic acid, and lorazepam which were all discontinued due to adverse effects.

Video EEG monitoring showed various EEG patterns including irregular bursts of generalized spike, polyspike, and slow wave complexes frequently associated with myoclonus of limb or body but sometimes without associated clinical changes. At times, EEG showed poly-spikes with central maximum associated with generalized myoclonic jerk (Fig. 4). His anti-seizure medications were changed to levetiracetam 1500 mg twice daily, clonazepam 0.25 mg nightly, and Lacosamide 50 mg twice daily. Over the next 10 years, his medications were adjusted multiple times due to adverse effects and was only on levetiracetam 1000 mg twice daily eventually. He was able to complete most activities of daily living and do simple chores around the farm although he had cognitive impairment and mood disturbances since the cardiac arrest. The myoclonus was well controlled with medications and was only occurring occasionally until his death in January 2022.

6. Case 5

A 63 year old female with a history of cardiac arrest in 2013, and during the same hospitalization had generalized tonic clonic seizure but later developed myoclonus. Myoclonus involved her trunk and was associated with vocalization but without changes in mentation. She had also later developed cognitive impairment, Capgras syndrome. She was maintained on levetiracetam, Lacosamide, Clonazepam for seizure and myoclonus. She had multiple dose adjustments to the AEDs over the years in an attempt to minimize cognitive impairment and somnolence. She was also concurrently on narcotics for chronic back pain and gabapentin for neuropathic pain, which likely contributed cognitive impairment and somnolence. She had an infrequent myoclonus and even less frequently, seizures. In 2020, her myoclonus became more persistent than before and was admitted for video EEG monitoring to characterize the myoclonus. In October 2020, her seizure medications were Levetiracetam 1500 mg twice daily, Clonazepam 0.5 mg three times daily, Lacosamide 150 mg twice daily. Her baseline EEG showed intermittent bifrontal and generalized spike and wave. Her seizure medications were reduced during the EEG monitoring, and she subsequently had an increase of myoclonus, increased frequency of spike and wave complex, and had an generalized tonic and tonic-clonic seizure associated with generalized

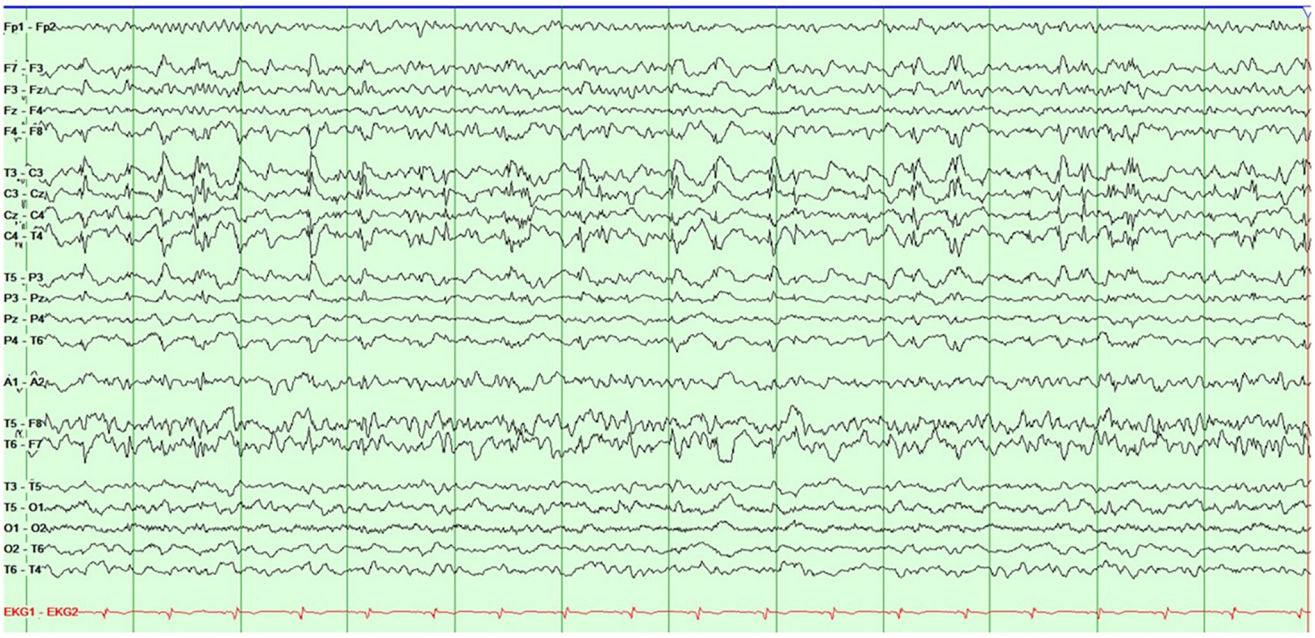

Fig. 3. Ictal and interictal EEG within 24 h post-cardiac arrest, Longitudinal Bipolar Montage, Time base = 30 mm/sec, sensitivity = 7 μV/mm, low frequency filter = 1 Hz, high frequency filter = 70 Hz, Notch = 60 Hz, showing generalized poly-spike and wave discharges with central maximum associated with bilateral upper extremity myoclonic jerks, consistent with electroclinical seizure.

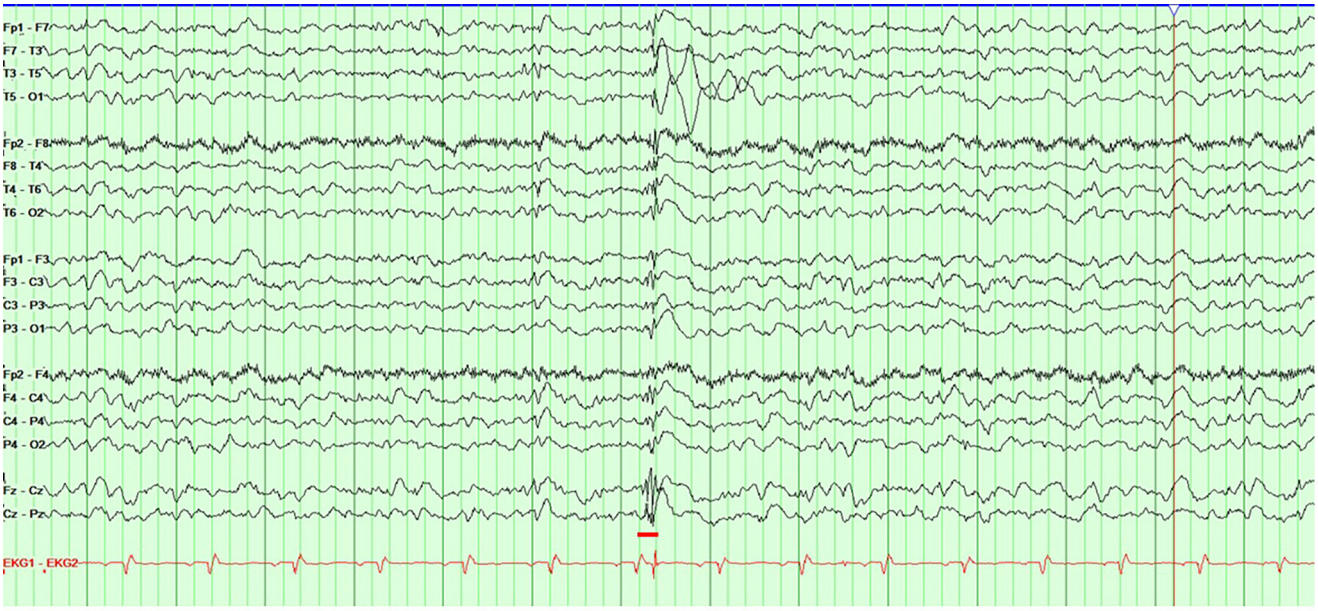

Fig. 4. Interictal EEG 2 years post-cardiac arrest, Longitudinal Bipolar Montage, Time base = 30 mm/sec, sensitivity = 7 μV/mm, low frequency filter = 1 Hz, high frequency filter = 70 Hz, Notch = 60 Hz showing poly-spike with central maximum (underlined in red) associated with generalized myoclonic jerk, artifact in the left temporal chain. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

polyspike and wave complexes. At times, EEG showed poly-spikes with central maximum associated with generalized myoclonus (Fig. 5). Generalized myoclonus was captured on video EEG monitoring with EEG correlates demonstrating polyspike and wave complexes (Video 1). She was resumed on her home medications and low dose of Valproate 250 mg twice daily was added with plan to up titrated in the outpatient setting.

7. Discussion

LAS was first described by James W. Lance and Raymond D. Adams in 1963 as a form of chronic posthypoxic myoclonus after cardiac arrest and successful resuscitation (Lee and Lee, 2011). LAS is characterized by intention/action myoclonus typically involving the trunk, face, and limbs that begins days to weeks after a hypoxic injury to the brain. LAS differs from MSE in onset, prognosis and outcome. Posthypoxic MSE is characterized by generalized myoclonic jerks occurring hours after the hypoxic insult (Malhotra and Mohinder, 2012). An important differentiation between the two is that patients with MSE are typically comatose, whereas patients with LAS are conscious although not always (Malhotra and Mohinder, 2012). It is important to distinguish between the two as the prognoses and management are vastly different, with LAS being a much better prognosis.

EEG can often serve as a study to differentiate LAS from MSE while the patient is comatose as early presentation of LAS can be masked by use of sedative medications (Elmer et al., 2016). In a cohort study by Elmer et al., several distinctive patterns of EEG were identified with pattern of suppression-burst background with high-amplitude polyspikes in lockstep with myoclonic jerks had poor prognosis, whereas pattern of continuous background with marrow, vertex spike-wave discharge with myoclonic jerks survived with favorable outcome which was defined by being discharged home or to acute rehabilitation (Elmer et al., 2016). This can be very important for prognostication and survivability as patients falsely diagnosed with hypoxic MSE may be taken off the ventilatory support too early due to its poor prognosis. In all four cases of cardiac arrest survivors, their EEGs had shown vertex spike-wave discharges often associated with myoclonus with a continuous background. Only case 3 had presented to our hospital for the initial cardiac arrest, and the EEG pattern had helped with prognostication. EEGs from the initial hospitalization after cardiac arrest are either incomplete or unavailable for the rest of the cases. However, this pattern EEG continues to persist weeks to years, which was long beyond the hypoxic insult.

The mechanism of MSE and LAS differs in the extent of damage to the cortex. Burst of polyspike with background suppression in MSE is thought to be due to diffuse cortical injury resulting from loss of disinhibition of various subcortical generators of discharges (Ching et al., 2012; Hofmeijer et al., 2014). Whereas in LAS with continuous background and a single midline discharge indicates mostly intact cortex with a different generator of the discharge possibly through the thalamus. One particular explanation for this EEG pattern is that loss of purkinje cell in the cerebellum results in disinhibition of GABAingeric signal to the ventrolateral thalamic nucleus. In the animal model where GABA A channel blocking agent was introduced via microinjection into the ventrolateral thalamic nucleus results in attenuation of myoclonus (Matsumoto et al., 2000). Purkinje cells are particularly susceptible to death after global brain ischemia due to aldolase C depletion and impaired glutamate clearance mediated by excitatory amino-acid transporter 4 (EAAT4) (Welsh et al., 2002). This would explain that LAS has a much better prognosis than MES because the majority of the cortex remained intact allowing for preservation of cognition and wakefulness.

The diagnosis of LAS is mostly clinical. However, various studies can be used to support the diagnosis. Imaging is not usually helpful in the diagnosis of LAS as head CT and magnetic resonance imaging is often unremarkable, especially later in the course of the disease (Lee and Lee, 2011). However, a case study presented by Ferlazzo et al. found bright lesions on diffusion weighted-imaging (DWI) MRI in the thalami and cerebellum during the early days of the syndrome (Ferlazzo et al., 2013). Fluid attenuated inversion recovery (FLAIR) imaging was normal in this case though (Ferlazzo et al., 2013). This finding is supported by the rat studies by Welsh et al.

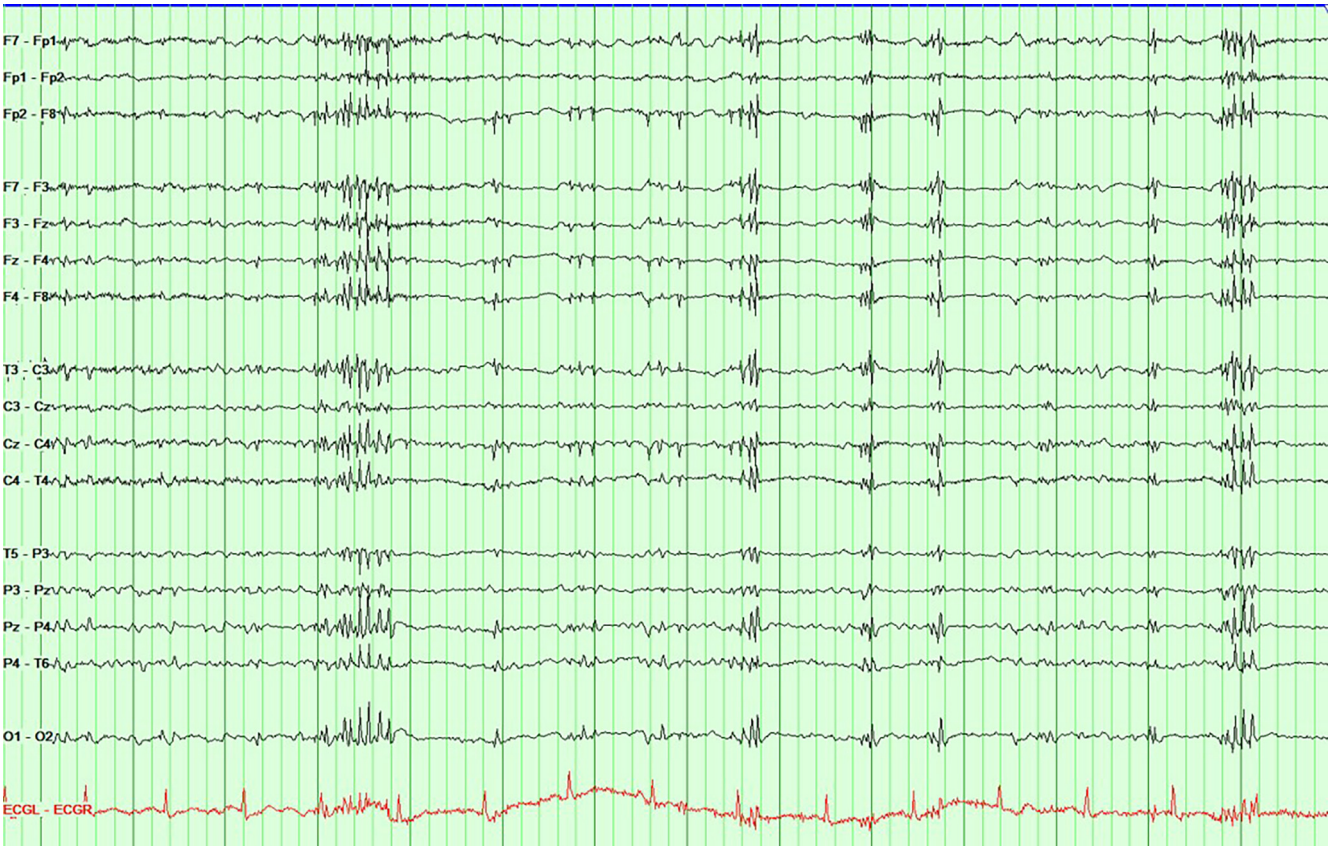

Fig. 5. Transverse Bipolar Montage, Time base = 30 mm/sec, sensitivity = 30 μV/mm, low frequency filter = 1 Hz, high frequency filter = 70 Hz, Notch = 60 Hz, Poly-spike with central maximum (underlined in red) associated with generalized myoclonus. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

and Ton et al. that demonstrated early damage to the cerebellum and thalamus respectively (Welsh et al., 2002; Ton et al., 2021). Single-photon emission CT (SPECT) and positron emission tomography (PET) scans have also shown potential. Frucht et al. demonstrated that patients with posthypoxic myoclonus showed increased glucose metabolism in the bilateral ventrolateral thalamus and pontine tegmentum on PET scans (Frucht et al., 2004), and separate case reports by Lee et al. and Zhang et al. found hypoperfusion to the left temporal lobe on SPECT (Lee and Lee, 2011; Zhang et al., 2007). Amongst the 4 patients, one patient had FLAIR change and DWI change on MRI in the basal ganglia with ADC reversal and contrast enhancement. The rest had nonspecific subcortical T2/FLAIR intensities on MRI, or had imaging abnormalities that predate the cardiac arrest. Unfortunately, SPECT or PET are not done at our facility.

There are no clear guidelines for management of LAS but case studies have shown various AEDs to be effective. In a review of case studies, Frucht and Fahn found that piracetam, clonazepam, and valproate were effective in managing about half of the cases (Frucht et al., 2004). Case studies by Lee et al. and Ilik et al. found success with levetiracetam as well (Lee and Lee, 2011; Ilik et al., 2014). Polesin and Stern also outline zonisamide as a potential treatment (Polesin and Stern, 2006) In our cases, patients responded well to levetiracetam (used in all 5 cases) but often had tried other medications including valproate being the second most commonly used. Other AEDs, including lacosamide, clonazepam, zonisamide, clobazam for both treatment of seizure and myoclonus, but usually used in conjunction with levetiracetam and never used as monotherapy in the 5 cases here. While there has been little published research on the matter, a case report by Shin et al. demonstrated that physical therapy could be effective in managing the symptoms of LAS (Shin et al., 2012). All in all, there is no one single therapy for the management of LAS. A combination of AEDs and, potentially, physical therapy would be most effective. However, more research needs to be done in the role that physical therapy plays in the management of LAS. Levetiracetam was used in all 5 cases either monotherapy or in conjunction with other AEDs. Valproate was tried in all 5 cases but often discontinued due to ineffectiveness or adverse effects. Other AEDs are tried but were used to treat seizures or refractory cases of myoclonus and effects are still not known. Zonisamide was added for 2 of the patients recently as it has different mechanisms of action, sparing of use of more sedating medications, and reported use for management of LAS.

8. Conclusion

Lance-Adams syndrome is a rare but important complication of hypoxic brain injuries and can often be confused with post-hypoxic myoclonus status epilepticus. LAS is associated with the EEG pattern of vertex spike-wave discharge with continuous background. EEG should supplement clinical findings to provide the most accurate diagnosis and thus provide the most appropriate management and prognosis. For symptomatic management, Levetiracetam was often used either as monotherapy or in conjunction with other AEDs.

Declaration of Generative AI and AI-assisted technologies in the writing process

AI was not used to generate or assist in writing this manuscript.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary material

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