Status Epilepticus and Coexisting Nonepileptic Atypical Abdominal Myoclonus in a Preterm Neonate with Hypoxic Ischemic Encephalopathy: A Case Report

Marie Charmaine S. Lukban, MD, MBA, Gerald T. Pagaling, MD,
Marissa B. Lukban, MD and Benilda C. Sanchez-Gan, MD

Department of Neurosciences, College of Medicine and Philippine General Hospital, University of the Philippines Manila

ABSTRACT

We describe an unusual case of hypoxic ischemic encephalopathy in a preterm female of 36 weeks who presented with status epilepticus and atypical abdominal myoclonus. The seizures were confirmed electrographically using video electroencephalography (EEG), while the abdominal myoclonus was demonstrated to be nonepileptic, as it had no EEG correlate. Other possible causes of neonatal seizures were excluded. The infant then responded to a gamut of antiseizure medications but the myoclonus persisted. To the best of our knowledge, this is the first report of atypical myoclonus in a preterm baby caused by hypoxic ischemic encephalopathy.

Keywords: hypoxic ischemic encephalopathy, status epilepticus, myoclonus, neonate

INTRODUCTION

Hypoxic ischemic encephalopathy (HIE) is a significant birth complication due to disturbed neurologic function in an infant.[1] The usual pathomechanism is due to impaired cerebral blood flow and oxygen delivery to the brain with resulting primary and secondary energy failure. Infants may manifest with depressed sensorium, difficulty initiating and maintaining respiration, seizures and in some cases, movement disorders.[2] Accurate and early differentiation between epileptic seizures and nonepileptic movements is of utmost clinical importance, as management decisions, prognosis, and counseling for families hinge significantly upon this distinction. While epileptic seizures often require aggressive pharmacological treatment, nonepileptic events—such as myoclonus or tremors—do not benefit from antiseizure medications and may reflect different underlying neurophysiological mechanisms. Here, we report an illustrative case of a preterm neonate presenting with status epilepticus alongside atypical abdominal myoclonus, emphasizing the critical role of early EEG monitoring to accurately differentiate these phenomena and guide appropriate clinical management.

CASE

A female infant was born preterm at 36 weeks of gestation from twin pregnancy by spontaneous vaginal delivery to a 24-year-old gravida 2, para 1 mother. The mother was generally healthy but had poor prenatal care, no prenatal vitamin supplementation and had no history of previous miscarriages. The prenatal ultrasound at 30 weeks showed

Paper presentation – Department of Neurosciences Research Contest, October 2021, Philippine General Hospital.

eISSN 2094-9278 (Online)
Published: September 15, 2025
https://doi.org/10.47895/amp.v59i13.11194

Corresponding author: Marie Charmaine S. Lukban, MD, MBA
Department of Neurosciences
Philippine General Hospital
University of the Philippines Manila
Taft Avenue, Ermita, Manila 1000, Philippines
Email: charmysy@gmail.com
ORCiD: https://orcid.org/0000-0003-1135-6400

a breech-transverse presentation. There was no significant family history of any disease or maternal exposure to infection or radiation. Moreover, there was no family history of seizures, epilepsy, or any other neurologic disorder.

The infant's birth weight was 1.9 kg, head circumference of 31 cm, and length of 45 cm, all appropriate for gestational age. The infant was thickly meconium stained, cyanotic, limp and apneic. The Apgar scores were 3 at 1 minute, 5 at 5 minutes, and 5 at 10 minutes of life. Early cord clamping was done, followed by resuscitation and eventual intubation. The initial arterial blood gas after intubation showed partially compensated metabolic acidosis with moderate hypoxemia. With the suspicion of hypoxic ischemic encephalopathy (HIE), the patient was then immediately placed on therapeutic hypothermia.

On the 40th minute of life, the infant had continuous cycling movements of the bilateral upper and lower extremities with lip smacking accompanied by recurrent, brief, rhythmic to semi-rhythmic jerky movements of the abdomen. She had depressed tone, normal neonatal reflexes, and bilateral clonus. There were no dysmorphic features or neurocutaneous markers, and the rest of the examination was unremarkable. Full blood count, C-reactive protein, serum glucose and electrolytes levels were all normal. She was then given a loading dose of intravenous levetiracetam at 60 mg/kg/dose (MKD). There was still persistent jerking of the right upper extremity hence intravenous phenytoin 20 MKD and oral phenobarbital 10 MKD were given. Subsequently a bolus of dose of midazolam 0.1 MKD was given followed by initiation of Midazolam drip which reached a ceiling dose of 23 mcg/kg/min. The exam at the 1st hour of life was consistent with encephalopathy and status epilepticus.

A video electroencephalogram (EEG) was performed at the 3rd hour of life after giving the medications mentioned, which showed diffuse attenuation of the background activity indicative of a diffuse cerebral dysfunction and left centrooccipital sharp transients (Figure 1). The events consisting of jerking of the abdomen lasting for 0.5 to 1 seconds were captured but were not associated with ictal EEG pattern (Figure 2). No evolving electrographic seizure patterns were noted during the video EEG recording. The abdominal jerks observed clinically were not associated with any ictal activity, this considered as nonepileptic myoclonus. A computed tomography (CT) of the brain at 24th hour of life showed patchy, ill-defined hypodensities and loss of gray, white matter differentiation throughout the brain (Figure 3). A magnetic resonance imaging (MRI) MRI was not performed due to logistical and resource constraints.

After two days of tapering midazolam, she had no recurrence of cycling movements of extremities but had

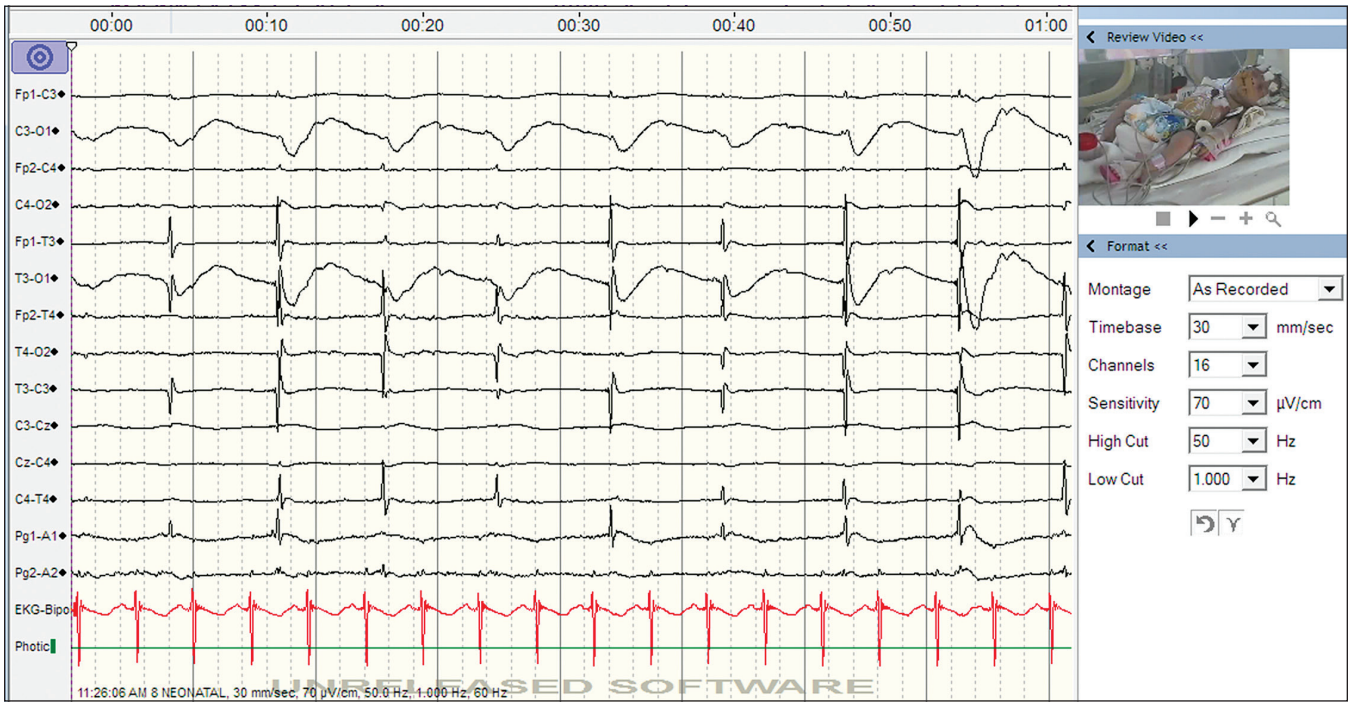

Figure 1. The background of this EEG was attenuated and consisted of almost frequent runs of rhythmic 1.5 Hz activity at the O1 region, consistent with a brief rhythmic discharge (BRD) based on American Clinical Neurophysiology Society criteria.[6] Trains of medium to high voltage 0.5-1 Hz delta waves were noted over the left occipital region with subsequent change in amplitude and morphology. Frequent medium to high voltage sharp transients were present at C3-O1. This is an abnormal interictal EEG due to the presence of diffuse attenuation of the background activity indicative of a diffuse cerebral dysfunction of nonspecific etiology, nonspecific left posterior focal slowing, and left centrooccipital sharp transients.

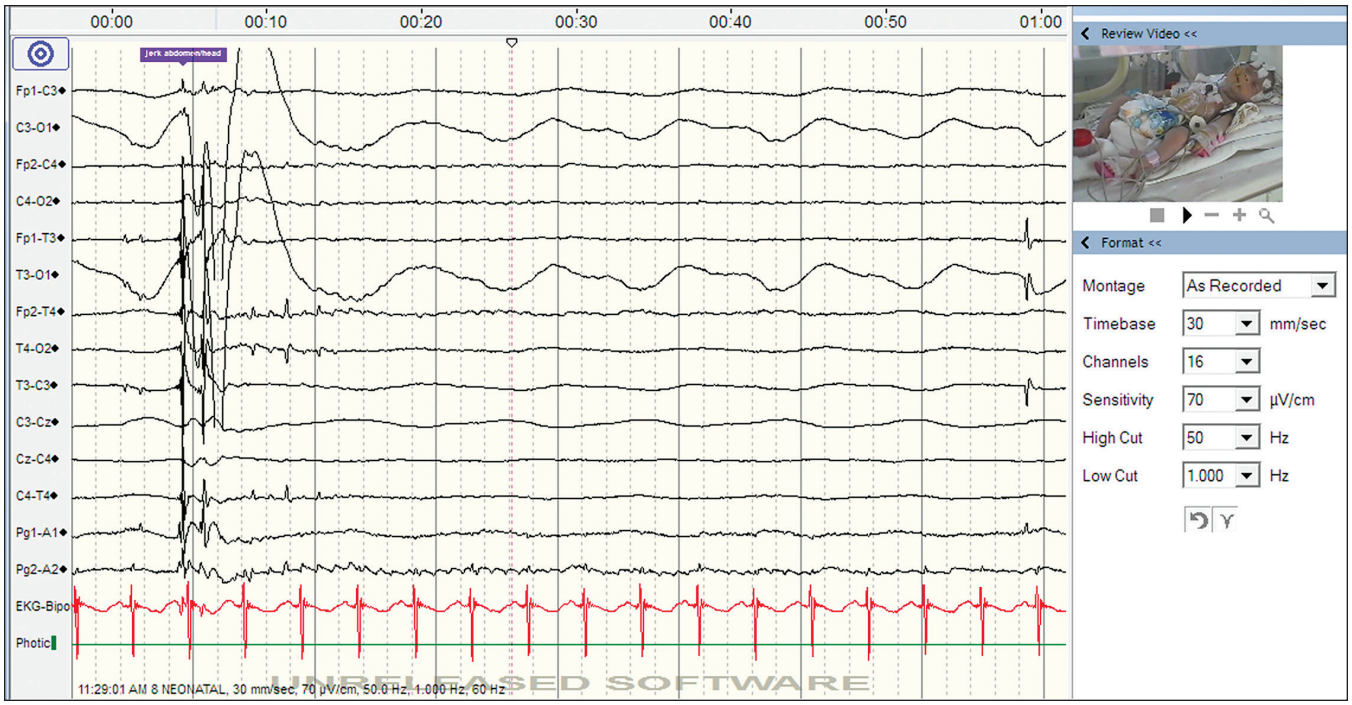

Figure 2. The events consisting of jerking of the abdomen lasting for 0.5 to 1 seconds were captured. These events were not associated with ictal EEG pattern.

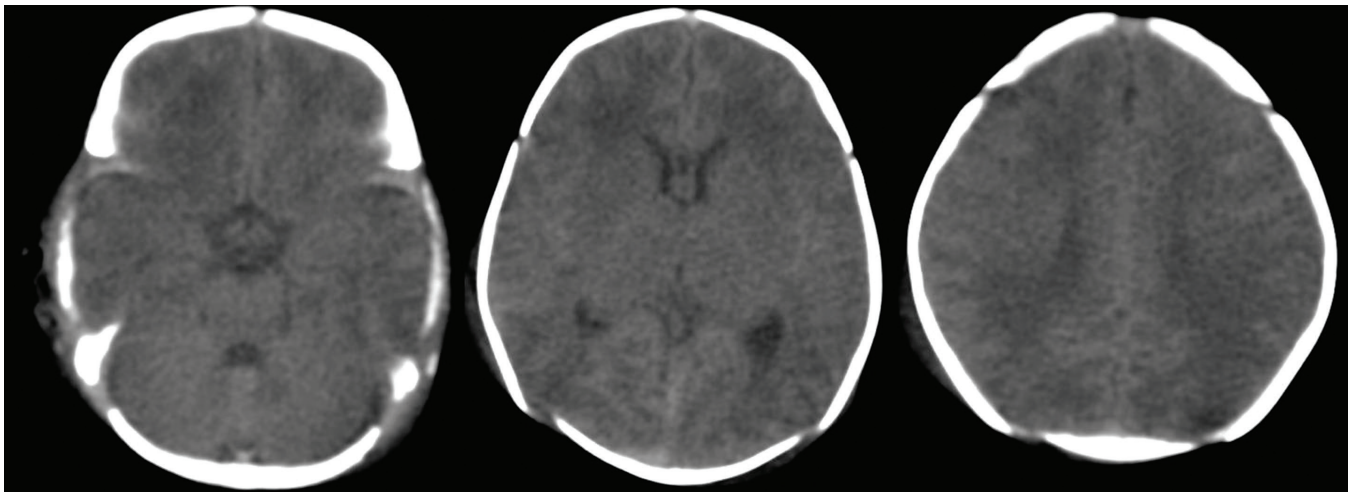

Figure 3. CT images reveal patchy, ill-defined hypodensities scattered throughout the supratentorial brain, with gray matter predominance. Patchy areas of loss of gray-white matter differentiation is also noted in the supratentorial brain. Decreased attenuation of the basal ganglia is seen. These findings are compatible with hypoxic-ischemic encephalopathy.

persistent jerky movements of the abdomen. A follow-up EEG showed a markedly abnormal background characterized by prolonged interburst intervals with low voltage activity, consistent with a burst suppression pattern. No ictal events were captured during this recording. The infant was maintained on levetiracetam, phenytoin and phenobarbital. These medications were **administered for seizure activity concerning for focal seizures,** particularly those initially confirmed on video EEG. These medications were **not intended to treat the atypical abdominal myoclonus**, which was determined to be nonepileptic in nature based on the absence of an ictal correlate. She was admitted to the neonatal intensive care unit (NICU) for two months on ventilatory support without recurrence of the seizures nor myoclonus. The patient did not experience any additional adverse or unanticipated events during the hospital admission. She was subsequently discharged on low-flow oxygen support but was unfortunately lost to follow-up in the outpatient clinic.

DISCUSSION

HIE refers to a central nervous system dysfunction or encephalopathy associated with perinatal asphyxia.[1] The etiology of HIE is multifactorial, which includes maternal factors, conditions during delivery and fetal factors with the level of neurological injury influenced by both the degree of ischemic insult and the infant's gestational age.[2]

In the neonatal period, the clinical manifestations of HIE vary between depression and excitation. Patients may have depressed level of consciousness, hypotonia, absent reflexes and apnea.[2] On the other hand, seizures occur in the majority of patients who experience moderate to severe HIE usually in the first 24 hours of life. Neonatal status epilepticus (SE) has been reported in 15 to 25% of newborns with seizures which is commonly caused by HIE.[3] Optimal anti-epileptic management of SE with anti-seizure medications must be administered immediately.

Another manifestation of HIE is nonepileptic myoclonus which has been reported by Walsh et al. in 2015. They discussed a case of a newborn who developed rhythmic myoclonus which was first assumed to be a seizure activity, but was unresponsive to anti-seizure medications.[4] Newborn infants may be prone to clinical motor phenomena such as tremors and myoclonus that are nonepileptic in nature due to a brainstem release phenomena.[4] As discussed in the case, our patient similarly presented with myoclonus described as recurrent, brief, rhythmic to semi-rhythmic jerky movements of the abdomen lasting for 0.1 to 0.5 seconds. The appearance of myoclonus after a hypoxic injury to the brain may be considered a poor neurologic marker. The pathophysiology of post-hypoxic myoclonus focuses on neuronal loss of the cortex and ischemic damage in the hippocampus, thalamus and brainstem.[5]

This case highlights several important strengths. First of all, it documents a novel clinical presentation of isolated abdominal myoclonus in a preterm infant with HIE, a phenomenon not previously reported in the literature. Moreover, early and continuous video EEG monitoring was pivotal in distinguishing epileptic from nonepileptic events, directly informing treatment decisions and preventing unnecessary escalation of antiseizure therapy. Lastly, the case emphasizes the importance of a multimodal diagnostic approach, including neuroimaging, laboratory workup, and EEG, in the acute evaluation of neonatal seizures and abnormal movements. However, there are also several limitations to consider. The patient was lost to follow-up after discharge, limiting our ability to provide long-term neurologic or developmental outcomes. Consequently, formal neurodevelopmental assessments and parent-reported outcomes were not obtained.

Ultimately, differentiating between seizures and nonepileptic myoclonus is important because it directly influences clinical management decisions, helps avoid unnecessary escalation of antiseizure medications, and may provide more accurate prognostic information. Timely diagnosis can prevent unnecessary diagnostics or treatment and further support the patient's prognosis.

CONCLUSION

HIE is a common presentation that can be linked to both maternal and fetal factors. Our case highlighted a rare incidence of status epilepticus with atypical abdominal myoclonus in a neonate with HIE. Identification of such phenomena can aid in appropriate management and more accurate prognostication.

Ethical Statement

The authors declare that all details, including laboratory data and test results, are entirely original. The authors ensure that a thorough informed consent process was carried out prior to the preparation of this case report. The authors have adequately explained the nature, risks, and benefits of this research to the patient's family, and have given them the opportunity to withdraw their consent prior to its submission.

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

All authors declared no conflicts of interest.

Funding Source

The study was funded by the authors.

REFERENCES

1. Allen KA, Brandon DH. Hypoxic Ischemic Encephalopathy: Pathophysiology and Experimental Treatments. Newborn Infant Nurs Rev. 2011 Sep 1;11(3):125–133. doi:10.1053/j.nainr.2011.07.0042.
2. Gillam-Krakauer M, Carter BS. Neonatal Hypoxia and Seizures. Pediatr Rev. 2012;33(9):387-397. doi:10.1542/pir.33-9-387
3. Farmania R, Garg D, Sharma S. Status Epilepticus in Neonates and Infants. Ann Indian Acad Neurol. 2020 Dec 18;23(6):747-754. doi:10.4103/aian.AIAN_189_20
4. Walsh BH, Baumer FM, Bernson-Leung ME, Lerou P, Peters JM. Teaching video neuroimages: nonepileptic myoclonus in a neonate following severe hypoxic-ischemic injury. Neurology. 2015 Mar 24; 84(12):e90. doi: 10.1212/WNL.0000000000001397. PMID: 25802277.
5. Gupta HV, Caviness JN. Post-hypoxic Myoclonus: Current Concepts, Neurophysiology, and Treatment. Tremor Other Hyperkinet Mov (N Y). 2016 Sep 17;6:409. Published 2016 Sep 17. doi:10.7916/D89C6XM4
6. Tsuchida TN, Wusthoff CJ, Shellhaas RA, Abend NS, Hahn CD, Sullivan JE, et al. American clinical neurophysiology society standardized EEG terminology and categorization for the description of continuous EEG monitoring in neonates: report of the American Clinical Neurophysiology Society critical care monitoring committee. J Clin Neurophysiol. 2013 Apr;30(2):161-73. doi: 10.1097/WNP.0b013e3182872b24