Lance-Adams Syndrome: A Rare Neurological Condition Following Suicidal Hanging: A Case Report

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Abstract

Background

Lance-Adams syndrome, also known as chronic hypoxic myoclonus, is a rare neurological condition following hypoxic brain injury.

Case Presentation

A 21-year-old female patient presented to the Emergency Room with a Glasgow Coma Scale (GCS) of 6/15 and was intubated immediately. On the 4th day after regaining consciousness, she started to develop action-induced myoclonic jerks. Necessary examinations and investigations were done, after which the diagnosis of Lance-Adams syndrome was made. She was started on sodium valproate, and her symptoms improved on the 7th day of initiation of treatment.

Conclusion

In survivors of hanging with hypoxic brain injury, the development of sudden myoclonus after regaining consciousness for a few days should raise suspicion of Lance-Adams syndrome. Early recognition and treatment with antimyoclonic agents should be initiated.

Keywords

Hanging, Lance-Adams syndrome, Myoclonus, Post-hypoxic myoclonus, Valproate

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INTRODUCTION

Lance Adams Syndrome (LAS), first described by James Lance and Raymond Adams in 1963, is a rare neurological condition that occurs in survivors of hypoxic brain damage. It may occur following respiratory arrest, airway obstruction, or cardiac arrest.[1,2]

Myoclonus can be defined as a sudden, brief muscle contraction or muscle tone lapses that is brief, involuntary, and shock-like.[3] LAS, also known as chronic post-hypoxic myoclonus, presents as action or intention myoclonus that occurs days to months after a hypoxic insult, typically involving the trunk, face, and extremities.[4] In contrast, acute hypoxic myoclonus occurs in patients while still in a coma within 12-48 hours.[5,6]

In LAS, no definitive findings are observed in the Magnetic Resonance Imaging (MRI) or Computed Tomography (CT) scan; however, these tests are performed to rule out other potential causes. However, some roles of Electroencephalography (EEG) have been identified, with one-third showing cortical spikes or polyspike time-locked with myoclonic jerks.[3,5] The diagnosis is based on medical history and typical symptoms. Still, due to the rarity of the case (<300 reported cases) and lack of definitive evidence in neuroimaging, LAS is frequently underrecognized or misdiagnosed as epileptic, functional, or drug-induced myoclonus, particularly in psychiatric and general medical settings.[7] Several articles have mentioned better response with valproate, clonazepam, levetiracetam, and zonisamide, often in combination.[3,6]

Here, we present a case of LAS following suicidal hanging.

CASE

Patient's information:

A 21-year-old female was brought to the emergency department unconscious approximately 2 hours following a hanging attempt. She had a Glasgow Coma Scale (GCS) of 6/15 (E2V1M3), heart rate 70 beats per minute, blood pressure of 80/50 mmHg, and a ligature mark around her neck. She was intubated immediately by the anesthesia team and shifted to the intensive care unit (ICU). Routine investigations, including CBC, RBS, TFT, RFT, and X-ray cervical spine, were within normal limits.

She was extubated after 40 hours upon improvement in her GCS. On the fourth day of admission, she developed irrelevant talk, disorientation, agitation, and hallucination.

Psychiatric evaluation confirmed delirium, for which she was started on olanzapine (5mg/day) in divided doses. Delirium subsided on the sixth day, and she was shifted to the ward for further evaluation of her underlying psychiatric illness.

Figure 1: Showing ligature marks

Clinical course:

Psychiatric evaluation revealed underlying depression (Hamilton Depression Rating Scale–17 score: 29) then Fluoxetine 20mg/day was started, and olanzapine was tapered to 2.5mg/day. On the seventh day, she developed sudden jerky movements when attempting to sit, stand, or perform any voluntary action. The jerks were absent at rest or sleep, aggravated by stress and worsened progressively over the next 3-4 days, rendering her unable to walk or feed herself. The movements affected four limbs, with greater severity in the lower limbs. However, she could speak properly but had difficulty writing (ataxic dysgraphia).

Neurological examination:

A detailed neurological examination on the 3rd day of developing myoclonus (9th day of admission) revealed that she was well oriented to time, place, and person, memory was intact except for peri-event amnesia, and power was 5/5 on all limbs. Reflexes were normal in all four limbs. Knee clonus and ankle clonus were observed in the bilateral lower limbs. The plantar on the right was downgoing, and the left leg was normal. The finger-nose test and heel-shin test revealed dysmetria, and she had difficulty performing dysdiadochokinesia.

Investigations:

EEG revealed lots of artifacts due to jerks but lacked epileptiform discharges or other significant findings. MRI-Cervical spine was normal. MRI brain revealed suspected subarachnoid hemorrhages in the bilateral occipital region, which did not correlate with clinical presentation or neurological findings and were considered incidental or hypoxia-related vascular changes.

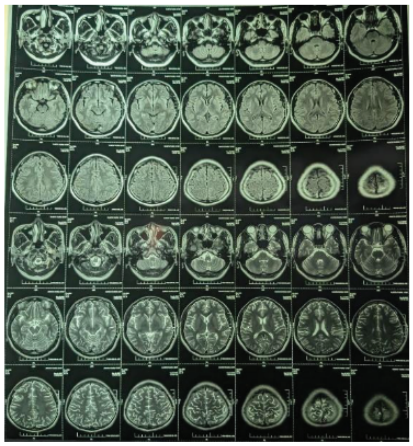

Figure 2: MRI Brain

Figure 3: MRI cervical spine

Diagnosis:

Multiple differential diagnoses were considered. Epileptic myoclonus was excluded by the absence of epileptiform activity on EEG, while drug-induced causes and serotonin syndrome were ruled out due to the lack of temporal correlation. Acute post-hypoxic myoclonus was excluded based on the delayed onset after recovery of consciousness. Functional neurological symptom disorder was considered unlikely given the presence of reproducible action-induced myoclonus with objective cerebellar signs, symptom consistency, and absence of distractibility. Taking together with the history of hypoxic brain injury, these findings supported the diagnosis of Lance-Adams syndrome.

Management and outcome:

A referral for neurological consultation was advised due to the unavailability of neurology services at our hospital, but they refused due to their financial status. A thorough literature review suggested valproate and clonazepam were the first-line drugs.[3,8] As the patient had a recent

history of delirium, we did not use clonazepam. Considering evidence, availability, and affordability, we started her on sodium valproate 500 mg at bedtime, increased to 800 mg/day in divided doses over the next 2 days. Significant improvement was observed starting from the seventh day of initiation, and by the ninth day, she was able to walk and eat on her own. On the ninth day, she was able to perform cerebellar examination properly, her clonus had subsided, and plantar reflexes were normal. She was discharged on the twentieth day of admission (10th day of starting valproate).

On follow-up in the OPD after 2 weeks of discharge, her depression had improved (HAMD-17 =12), and her myoclonus had further subsided, occurring only rarely during stress. Family members mentioned that she was able to perform all the household activities independently.

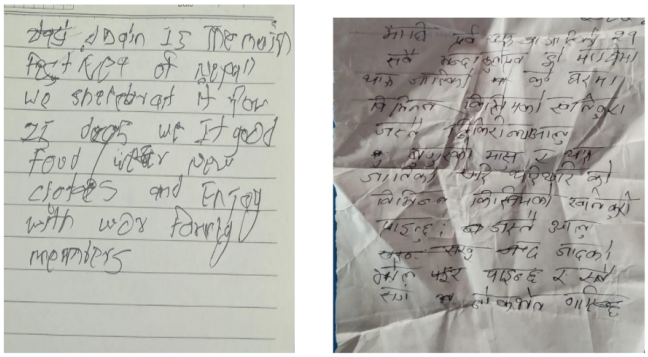

Figure 4: Handwriting before and after initiation of treatment with Valproate.

DISCUSSION

LAS is an uncommon but important differential diagnosis in patients developing myoclonus after recovery from hypoxic brain injury. Cases occurring after suicidal hanging are relatively rare compared to other etiologies, with only a limited number described in the literature.[2,5]

The present case illustrates the diagnostic challenge of LAS in the presence of comorbid psychiatric illness, delirium, and non-diagnostic MRI and EEG findings. Similar findings have been reported previously, where neuroimaging is often normal or shows incidental changes, and the EEG lacks consistent epileptiform activity.[3,7] Myoclonus in LAS is thought to arise from dysfunction of cerebellar-thalamo-cortical and subcortical networks, which may not be reliably detected on surface EEG, making the diagnosis largely clinical.[3,7] The frequent absence of significant MRI findings further supports the involvement of functional networks rather than structural pathology

The rarity, combined with nonspecific investigations, often leads to a delayed/missed diagnosis or mislabeling it as functional.

The cerebellar features observed in our patient are consistent with prior reports, where cerebellar signs such as gait disturbance and hypermetric movements have been described in approximately 55% of LAS cases.[7] The clinical course in our patients was comparable to earlier reports, with delayed onset of action myoclonus after regaining consciousness and preserved cognition.[2,6] A previously reported post-hanging case also demonstrated similar features and response to treatment.[9]

Pharmacologic management includes Valproate, Clonazepam, Levetiracetam, Piracetam, or Zonisamide, often in combination. The recovery varies with some patients improving within weeks, while others may take months to years and remain wheelchair-bound.[5] In this case, early recognition and prompt initiation of valproate therapy led to rapid clinical recovery, starting on the seventh day of treatment.

CONCLUSION

LAS should be considered in any patient who develops stimulus or action-induced myoclonus days after recovery from a hypoxic insult. This case emphasizes the need for psychiatrists, who frequently encounter and manage survivors of hanging, to be alert to neurological complications such as LAS, as timely diagnosis and treatment with antimyoclonic agents such as sodium valproate can lead to significant clinical improvement and functional recovery.

ETHICAL CONSIDERATION

Written informed consent was taken from the patient for the publication of details.

CONFLICT OF INTEREST

None

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