Lance-Adams Syndrome Following Cardiac Arrest Secondary to Cocaine Overdose
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INTRODUCTION: Lance-Adams Syndrome (LAS) is a rare neurological condition following anoxic brain injury. We present a case of a 24 year-old woman who developed LAS following cardiac arrest from cocaine overdose.

CASE PRESENTATION: A 24 year-old female with prior history of anxiety/depression and daily 3 gram intravenous cocaine use was found unresponsive at home. CPR was started and emergency medical services noted cardiac arrest with an asystolic rhythm. Return of spontaneous circulation was achieved in 15 minutes. She was admitted to the Cardiac Intensive Care Unit and targeted temperature management (TTM) was initiated. Her course was complicated by rhabdomyolysis and compartment syndrome of the calf requiring fasciotomy. Post-TTM she was sent to the medical intensive care unit (MICU). On hospital day (HD) 11 she was noted to have intention rhythmic myoclonus, but was responsive to voice and followed simple commands. Myoclonic status epilepticus (MSE) was ruled out. Her mental status improved despite persistent intention myoclonus. Her myoclonic movements slowly improved with complete cessation of myoclonus without directed pharmacotherapy on HD 27. She was transferred out of the MICU on HD day 29 and discharged home on HD 35.

DISCUSSION: LAS is a rare condition and may be underrecognized in the MICU. It is important to recognize this condition early as it has clear prognostic and treatment implications. The pathology of LAS is not completely understood. The current body of evidence suggests that disinhibition of GABAergic systems is responsible for myoclonus seen in anoxic brain injury. The diagnosis of LAS is based on a constellation of symptoms: action myoclonus associated with postural imbalance and ataxia with mild to absent cognitive defect in a patient with hypoxic brain injury. Imaging and EEG are not helpful in diagnosis. The difference between LAS and posthypoxic myoclonus (PHM) is that LAS patients have intact consciousness, whereas PHM patients remain comatose. The appearance of MSE with 24 hours is considered a marker of poor outcome, while LAS is associated with survival of preserved intellect. It is important for clinicians to delineate these two conditions when prognosing patients after anoxic brain injury. Treatment for LAS is antiepileptic medications and benzodiazepines [1]. Symptoms can resolve or remain chronic. Our patient’s symptoms resolved without directed pharmacotherapy.

CONCLUSIONS: LAS is a rare condition presenting after anoxic brain injury. Early clinical delineation between LAS and PHM must be made due to markedly different prognosis. Despite being found in an asystolic rhythm, our patient had a full neurological recovery after TTM post-cardiac arrest. This treatment, in combination with early recognition of LAS, led to proper prognostication and full recovery.


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