

## Case Report

# Improvement of Post Anoxic Myoclonus with Valproic Acid and Add on Levetiracetam Therapy- A Case Report

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## Keywords

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- Myoclonus
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## Abstract

Myoclonus may be defined as sudden muscle contractions that are brief, involuntary, and shock-like. There are many conditions that can lead to myoclonus including spinal cord injury, stroke and as a side effect of certain drugs. Post hypoxic myoclonus is a recognized entity that develops post successful resuscitation of cardiac arrest and this is known in the literature as Lance Adams Syndrome. Often myoclonus is confused with an underlying seizure disorder. Myoclonic status epilepticus at any time after return of spontaneous circulation (ROSC) is even considered to reliably indicate a poor prognosis. Different treatments have been attempted to manage post hypoxic myoclonus including valproic acid and clonazepam and even Bilateral Pallidal Deep Brain Stimulation. The authors would like to present a case report of a 59-year-old male with chronic post hypoxic myoclonus who was successfully treated with a combination medication regime of valproic acid and levetiracetam.

## INTRODUCTION

Post hypoxic myoclonus complicates a significant number of presentations with out of site cardiac arrest [1]. The exact pathophysiology and etiology of the neuronal damage resulting in myoclonus has not been clearly defined. However, histopathological studies point towards ischemic damage to the hippocampus, Purkinje cell layer of the cerebellum and the reticular thalamic nucleus [2]. The major causes of hypoxia include respiratory arrest, followed by anesthesia and surgical accidents [3]. Drug intoxication and asthma attacks have also been implicated [3]. Usually the myoclonus manifests days or weeks after the event when consciousness is regained and is exacerbated during stimulation [3]. Different pharmaceutical treatments have been attempting in managing this condition including lamotrigine, riluzole and clonazepam in dosages as high as 18mg per day [4]. However, results have been mixed and a firm treatment protocol has not been established. The authors of this case report hope to shed some light on the usefulness of combination treatment with valproic acid and levetiracetam especially in a patient with chronic renal failure on hemodialysis.

## CASE PRESENTATION

A 58-year-old male with recently diagnosed end stage renal disease on hemodialysis developed pulseless electrical activity (PEA) lasting about 18 minutes. Patient required emergency intubation and 3 cycles of cardiopulmonary resuscitation prior to

return of spontaneous circulation. Soon after event he developed generalized myoclonus involving both upper and lower extremities and facial musculature. These were sudden, brief and involuntary. Neurology was consulted and they proceeded to do an EEG which showed mild diffuse slowing of background and no clear cut epileptiform activity or consistent focal abnormalities. Patient was commenced on levetiracetam 500mg oral once daily with an additional 500mg after every cycle of hemodialysis. However, he continued to have intermittent generalized myoclonus precipitated and worsened by stimulation. Neurology planned to add Clonazepam 0.5mg oral twice daily for better control. This addition reduced frequency and intensity of the episodes but they continued. Clonazepam was increased to three times a daily however the episodes stabilized in frequency without remission. Patient's myoclonus was suspected to be due to uremia at one point however even with regular scheduled hemodialysis treatments there was minimal improvement in same. Eventually valproic acid was added to his regime at a dose of 500mg oral three times daily with which patient and family noted significant improvement in involuntary movements and these were localized to minimal semi purposeful movements in the peri oral and left facial musculature.

## DISCUSSION

The first step in managing myoclonus in a patient is appropriate and timely diagnosis. This may be challenging in patients with prior history of seizure disorder or patients who

have been on antipsychotic therapy in the past in which case myoclonic movements may be confused with tardive dyskinesia. Psychogenic movement disorders may also pose a diagnostic challenge and they are usually characterized by sudden onset; spontaneous remissions; variable frequency and distractibility (5). Dystonia which is a sustained twisting movement progressing to prolonged abnormal postures also is a reasonable differential diagnosis in a patient with history of antipsychotic use. In patients with previous history of cerebrovascular accidents, clonus which is a rhythmic, involuntary muscular contraction and relaxation should be ruled out. Entities as simple as shivering which is a thermoregulatory mechanism to protect the body against hypothermia is often confused with myoclonus. Toxic metabolic injury and infectious etiologies all need to be thoroughly investigated to reach an appropriate diagnosis. Once diagnosed, the task of instituting adequate evidence based treatment arises. In our index patient, the level of social and occupational dysfunction was significant to the point of disabling him physically and mentally. Although various classes of medications have shown evidence when used individually or in combination,

no protocol or guideline exists for adequate treatment. This may be due to the fact that the etiology of this disorder is still not well known. In our index patient, a combination approach with valproic acid and levetiracetam was successful in reducing symptoms.

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