ACTA SCIENTIFIC CLINICAL CASE REPORTS

Volume 5 Issue 4 April 2024

Case Report

A Successful Cardiopulmonary Resuscitation followed by Post Hypoxic Action Myoclonus (PHAM) Presentation in a 67-year-old Female

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DOI: 10.31080/ASCR.2024.05.0528

Received: February 12, 2024

Published: March 07, 2024

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Abstract

Myoclonus may be a prominent feature in patients who have survived severe Cerebral anoxia following cardiopulmonary arrest. Several post hypoxic sequlae have been described, Including myoclonus, dementia, Parkinson's disease, Cerebral palsy in children etc. Post hypoxic myoclonus presents in two forms. Either immediately after hypoxia while the patient remains in coma myoclonus develops and usually carries poor prognosis. A second type of delayed post hypoxic action myoclonus described by Lance and Adams in 1960s following cardiac or respiratory arrest. Although some patients are severely affected by action myoclonus, postural lapses and other deficits, those with pure myoclonus can improve gradually.

Keywords: Myoclonus; Hypoxia; Encephalopathy

Introduction

A rare case of Post hypoxic stimulus sensitive action myoclonus (PHAM) described by two physicians named LANCE AND ADAMS. This syndrome is presented as a sequela of cardiopulmonary arrest or after a successful cardiopulmonary resuscitation. This case report is of a 67-year-old lady who had an intraoperative cardiopulmonary arrest which was resuscitated successfully. However, she later presented with myoclonic jerk of right upper limb during her follow up visit [1-13].

Clinical Presentation

A 65-year-old lady who presented with bleeding mass per vagina and underwent Total vaginal hysterectomy for grade 3 uterine prolapse and had intraoperative cardiac arrest. Cardiopulmonary resucitation was initiated as per ACLS regimen. Three cycles of CPCR given for about 30 minutes. CPCR was successful and patient was connected to ventilator machine for assisted breathing and transferred to intensive care unit. The very next day she was extubated, sensorium was adequate, Neurological examination was unremarkable, no evidence of cranial nerve palsy seen. Lab investigations were unremarkabke except Fasting blood glucose 220mg/dl, Cardiac troponin T was elevated after 6 hours of successful cardiopulmonary resucitation. Echocardiography showed mild left ventricular dysfunction. Further coarse in the hospital was uneventful. She got discharged on the 7th post operative day . How ever she presented with abnormal jerky movement of right hand predominantly while attempting voluntary movements. T2 weighted MRI brain revealed hyperintensity of bilateral capsulo ganglionic region, bilateral tempero-occipital lobes and peri rolandic cortex. Considering the bilateral hyper intensities and diffuse cerebral involvement and the onset of clinical symptoms with respect to recent resuscitated Cardiopulmonary arrest this paculiar myoclonic jerk was labelled as sequlelae of Hypoxic ischemic insult. She was put on Sodium valproate and levitrecetam, followed up for a period of 30 days and gradual improvement clinically was observed. There was no other sequlae like memory impairment, spasticity, gait ataxia etc throughout the follow up period. There was a good prognosis observed interms of absence of myoclonus and Normal EEG on last followup visit. However considering the possibility of recurrnce and long term neurologic sequelae she was advised to continue antiepileptics further and adher to regular followup.

Discussion and Conclusion

Post hypoxic a myoclonus exists in acute and delayed forms and often with other deficits. Global cerebral hypoxia of any etiology can result in myoclonus. Delayed form of stimulus sensitive action myoclonus was described by Lance and Adams in 1960s. Less than 150 case reports available from literature. This delayed form usually develop within one to four weeks of cardio pulmonary arrest or after a successful cardiopulmonary resucitation. Pathophysiological basis of PHAM is a matter of conjecture. Only a few

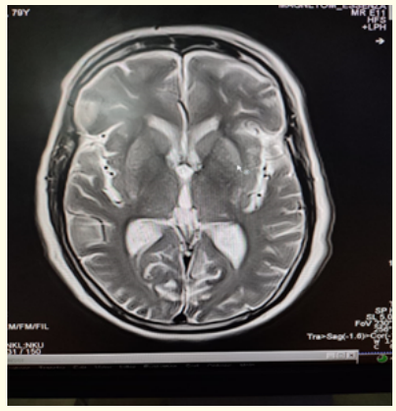

Figure 1: T2 FLAIR- arrow showing infarct in ganglio capsular and rolandic region.

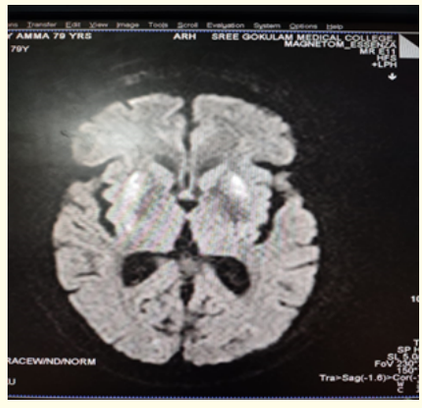

Figure 2: DWI showing infarct in ganglio capsular, temporal, occipital and rolandic region.

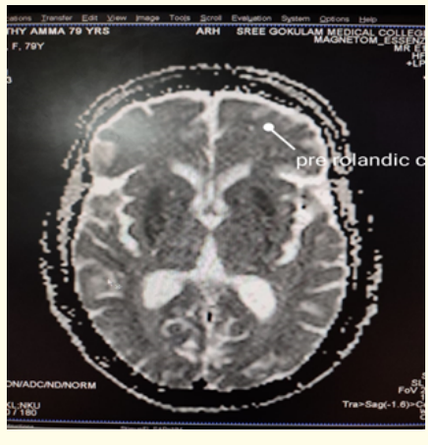

Figure 3: DWI showing pre Rolandic cortex hyperintensities.

pathophysiologic investigations exist and are not significantly helpful in understanding the pathophysiology of PHAM. Role of brain serotonin have been described. Hence 5 hydroxy tryptophan(5HT) agonists alone or with carbidopa has been found effective in reducing the amplitude of myoclonic jerks. Autopsy findings shows basal ganglia infarct, infarct of cerebral cortex, purkenje cells, striatum and the Globus pallidus Myoclonus predominantly occure during movements that require precision. Prolonged partial ischemia especially in young can cause damage to basal ganglia and presented with movement disorder or parkinsonism. Prognosis depends on the duration and depth of coma, Brain stem reflexes (pupillary reflex, corneal reflex and vestibular ocular reflex), motor responses to stimuli, eye movements and spontaneous reflexes like coughing, sneezing, swallowing, gagging etc. Even though acute forms of Post hypoxic myoclonus which develop while the patient remains in coma usually carry poor prognosis. On the other hand those with pure action myoclonus usually carry a better outcome.

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