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Inicio Home

Indice del volumen
Volume indexComité Editorial
Editorial BoardComité Científico
Scientific CommitteeNormas para los
autores Instruction to
AuthorsDerechos de autor
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Contacto/Contact:

Letters to the Editor / Cartas al Editor

LANCE ADAMS SYNDROME WITH SECOND CARDIAC ARREST AND DISAPPEARANCE OF MYOCLONUS

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Dear editor:

Cardiac arrest is the common cause of anoxic brain injury. Other reasons to have brain anoxia include severe hypotension, hypotension with limited CNS blood flow like high ICP, carbon monoxide poisoning, and strangulation. Lance et.al¹ published in 1963 the first presentation of post anoxic recovery myoclonus. The presence of myoclonus in post-cardiac arrest patients is frequently seen, though the syndrome is named after the original paper; Lance-Adams syndrome is the residual presence of myoclonus after the patient survives. The literature review was done and there is no mention of second cardiac arrest in Lance- Adams syndrome patients with the disappearance of myoclonus.

45 year old male with a preadmission history significant for hypertension, diabetes, diabetic neuropathy, necrotizing fasciitis secondary to a boating accident, and bipolar disorder admitted to the hospital a year before his present admission with shortness of breath. He was admitted with bilateral pneumonia. During this first hospitalization, he underwent surgery for left below-the-knee amputations. The day after his surgery, he was found to have difficulty breathing after which he suffered asystole cardiopulmonary arrest with ROSC being achieved after 12 minutes of resuscitation. He underwent a targeted temperature management protocol. Post hypothermia, he woke up and started following commands. He stayed on the ventilator for 12 days followed by successful extubation. In subsequent days, he suffered myoclonic jerking of his upper and lower extremities. Electroencephalogram did not show any seizures. CT head was negative for findings consistent with anoxic brain injury. MRI brain was not possible at that stage due to concern for hardware for a history of cervical spine surgery. He was able to follow commands, and move his extremities independently with intermittent myoclonic jerking, track movement, and garbled, incoherent speech. He was discharged to rehab and eventually home but required total care from his mother. A clinical diagnosis of Lance-Adams syndrome was made and symptomatic treatment was initiated.

He was brought to the emergency department after the second asystole cardiac arrest followed by pulseless electrical activity at home. ROSC was achieved on arrival to ED after 5 rounds of CPR following ACLS protocol. Per his mother, his baseline mental status prior to arrest was intermittently following commands and ability to express himself. He was intubated and subsequently underwent targeted temperature management protocol in the intensive care unit.

After completion of hypothermia, he stayed in a coma without any sedation. The myoclonus due to Lance-Adams syndrome was not present. His eyes opened up and was able to track but not able to follow commands or move extremities. CT head did not show any active process. Brain MRI was able to be done showing minimal small vessels with subtle ischemic changes. EEG was negative for any seizures with marked encephalopathic changes. Over the next many days, there was no change and the family proceeded to terminal extubation and terminal care.

Since first report of 4 cases in 1963¹, over 150 cases are reported in the literature. Review of the literature showed that there are 80 published papers specific for this condition. Most dealing with the case reports and therapies used to manage myoclonus and rehabilitation. The key feature of this diagnosis is recovery from an anoxic event. It is usually seen after cardiac arrest²⁻⁷, but other causes mentioned include snakebite envenomation⁸, COVID-19 induced hypoxemia⁹, infective endocarditis¹⁰, and strangulation¹¹. There is no one clear decided therapy to control myoclonus in these patient. Our patient was on valproic acid and it was stopped post 2nd arrest due to absence of myoclonus. Therapies tried and mentioned in the literature include Perampanel¹²⁻¹⁷, deep brain stimulation¹⁸⁻²², sodium oxybate^{23,24}, cannabidiol²⁵, volatile anesthetic agent²⁶, Levatiracetam^{27,28}, Gamma-hydroxybutyrate²⁹, L-5-hydroxytryptophan³⁰, piracetam³¹, levodopa³², intrathecal baclofen³³, lacosamide³⁴, and valproic acid³⁵⁻³⁷. The treatment includes therapy as our patient was getting at home after first cardiac arrest. The disappearance of myoclonus was thought to be due to second insult and injury to deep gray matter. Although MRI did not show extensive anoxic changes, patient continued to require intubation to protect his airway and not following command. This case is the first reported case where a second cardiac arrest happened in a patient with established diagnosis of Lance-Adams syndrome and his myoclonus was absent after ROSC was achieved.

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