Community Memorial Health System

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Bilateral Hemiballismus After Cardiac Arrest in a 23-year-old Male

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Introduction

- Post-hypoxic movement disorders (PMD) and chronic post-hypoxic myoclonus (CPM) are rare complications of cardiac arrest.
- Studies investigating the spectrum of these diseases in patients with hypoxic-ischemic encephalopathy after cardiac arrest showed that the true incidence of PMD/CPM is complicated by a poor survival rate.
- Paucity of this phenomenon poses challenges in evaluation and management.
- Here we present a patient who suffered a perioperative cardiac arrest during a thoracotomy after a gunshot wound and subsequently developed PMD and CPM.

Case Presentation

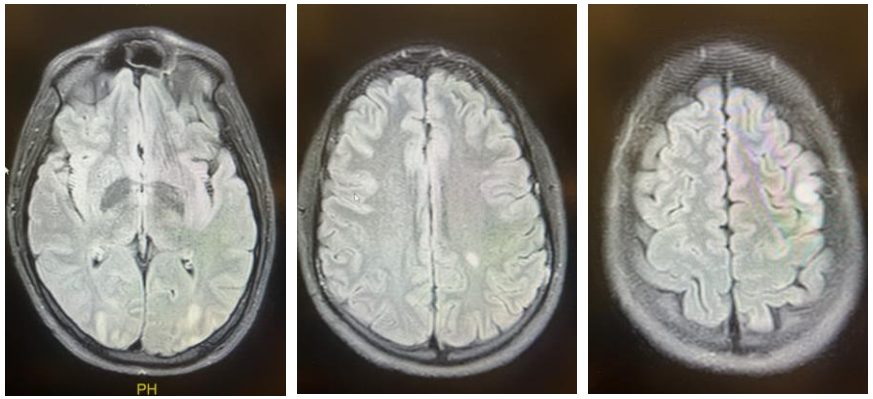
A 23-year-old male with past medical history of methamphetamine use suffered a gunshot wound to the right upper chest requiring a thoracotomy that was complicated by cardiac arrest with resuscitation and return of spontaneous circulation.

Four days during his post-operative course, he experienced involuntary thrashing movements in his upper and lower extremities which did not improve after initiation of levetiracetam on initial discharge, prompting subsequent admission to our emergency department four days later. The patient was also briefly given droperidol for nausea and vomiting. These involuntary movements began with akathisia in his feet spreading proximally until all extremities were affected, with the right upper extremity being the most severely affected. The patient reported aching right arm pain triggered by each attempt to consciously move his arm to reach for a target. The patient has no history of seizure or tic disorders, though reportedly had ADHD as a child and a history of methamphetamine and marijuana use.

On examination, the patient displayed rapid jolting twitches consistent with chorea and large amplitude ballistic limb movements, more prominent in the right arm. Neurological examination revealed intact mental status, cognition, and cranial nerve functions. Muscle strength, sensation, and deep tendon reflexes were present and symmetric bilaterally. Upper extremity dysmetria was more severe on the right.

Hospital Course

The patient was admitted for involuntary hyperkinetic movements affecting both sides of his body asymmetrically (Right>Left). Neurology was consulted and characterized the PMD as bilateral hemiballismus/hemichorea. Laboratory analyses were remarkable for anemia, secondary to recent blood loss, and elevated C-reactive protein and creatine kinase. The urine drug screen was only positive for THC.



Brain MRI confirmed bilateral multifocal hypodensities consistent with bihemispheric watershed infarctions.

An electroencephalogram was only remarkable for excessive beta waves in the background, suspected to be caused by the GABAergic effects of lorazepam given to the patient.

We initially treated the patient with clonazepam however this was ineffective in quelling his PMD; the patient had the best response to haloperidol 5 mg PO every 6 hours and benztropine 1 mg PO BID. QTc interval was monitored daily and without significant fluctuations. The patient improved significantly after treatment and was safely discharged with recommendations for rehabilitation and further outpatient follow up with neurology.

Video demonstration of the patient's involuntary hyperkinetic movements on admission and after treatment with haloperidol. Expressed permission given by the patient for use for academic purposes

Discussion

Initially the patient's movement disorders represented a broad differential including tardive dyskinesia, acute dystonais after receiving droperidol, or cerebrovascular insults.

- The largest studies investigating PMD/CPM involved 72 patients with hypoxic-ischemic encephalopathy after cardiac arrest.
- Only 26.4% (n=19) survived to develop PMD or CPM; the incidence of PMD/CPM is complicated by a poor survival rate in 36% (n=26).
- Only 6.9% (n=5) from this study sample developed ballismus.

These movement disorders are associated with hypoxic lesions in the basal ganglia with a clinical presentation including dystonia, ballismus, choreoathetosis or akinetic-rigidity syndrome that can exacerbate therapy-resistant hyperkinetic storms. The involuntary hyperkinetic movements in this patient were attributable to infarctions in bihemispheric watershed areas.

The broad pathophysiology underlying PMD/CPM is related to vascular supply differences, oxidative metabolism, disinhibition, altered sensory gating and impaired plasticity of basal ganglia-thalamo-cortical circuits, which can lead to hyperexcitatory neuronal transmission. Absence of clear guidance from the literature given the rarity of this presentation, prompted our exploration of different therapeutic approaches including anti-cholinergic, antipsychotic, and GABA-agonistic agents. Haloperidol proved to be the most successful therapy for the patient's hyperexcitatory movements evidenced by the significant improvement of this patient's bilateral hemiballismus.

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