POST-HYPOXIC MYOCLONUS AFTER POSTPARTUM HEMORRHAGE: A CASE REPORT

Njagi J.N[1]

Affiliation

1. Department of Obstetrics and Gynecology, PCEA Tumutumu Hospital, Karatina, Kenya.

Correspondence: njagijr@gmail.com

ABSTRACT

Background: Post-Hypoxic Myoclonus (PHM) is the occurrence of involuntary muscle jerks following hypoxia. Myoclonus Status Epilepticus (MSE) and the Lance-Adams Syndrome (LAS) are the primary PHM types. The Lance-Adams syndrome has better clinical outcomes compared to myoclonus status epilepticus.

Case presentation: A 24-year-old para 1 gravida 2 at term gestation presented at the Mbarara Regional Referral Hospital with obstructed labor as a referral. She was scheduled for emergency cesarean delivery, during which she suffered two cardiac arrests. She was diagnosed with anoxic post-hypoxic myoclonus on the fourth postoperative day. She was managed with haloperidol, antibiotics, clonazepam and rehabilitated on physiotherapy.

Conclusion: Post-hypoxic myoclonus was previously associated with a poor prognosis. However, recent findings have demonstrated positive clinical outcomes with pharmacotherapy and physiotherapy. Early diagnosis is critical for effective management.

Keywords: myoclonus, post-hypoxic myoclonus, physiotherapy, clonazepam

INTRODUCTION

Myoclonus is the sudden, jerky, and brief involuntary muscle movements. These sudden spasms are classified according to their etiology, body parts involved, temporal pattern, and relationship to action (1). Of the several types of myoclonus, Post-Hypoxic Myoclonus (PHM) is the least understood on account of challenging diagnosis and management. The deficiency of oxygen in the blood due to excessive bleeding results in hypoxemia, leading to hypoxia. There is a direct relationship between the anoxic event of excessive blood loss and myoclonus. Thus, the occurrence of myoclonus due to hypoxia is termed as post-hypoxic myoclonus. Post-hypoxic myoclonus is often associated with poor prognosis; however, positive outcomes have been reported in the literature (2). Myoclonus Status Epilepticus (MSE) and the Lance-Adams Syndrome (LAS) are the primary PHM types. The Lance-Adams syndrome has better clinical outcomes compared to myoclonus status epilepticus (2). Severe muscle jerks in PHM intensify with short movement. However, the myoclonic jerks are generally mild and non-existent when the patient is at rest or asleep, respectively. PHM patients may show signs of dysarthria and ataxia, which suggest cerebellar dysfunction. A combination of physiotherapy and pharmacologic management is recommended to rehabilitate PHM patients (3). This is a case report of a 24-year-old at term gestation who presented with obstructed labor. On cesarean section, she suffered two cardiac attacks. She was diagnosed with post-hypoxic myoclonus on the fourth postoperative day. She was managed with clonazepam and physiotherapy, and regained ambulation.

CASE PRESENTATION

A 24-year-old para 1 gravida 2 at term gestation presented at the Mbarara Regional Referral Hospital with obstructed labor. She was referred from a Class III healthcare facility, where she had been in labor

for 22 hours. Amniorrhexis had occurred nine hours before admission. On clinical examination, her fundal height was 40 cm, the fetal heart rate was 157 Beats Per Minute (BPM), and the presentation was cephalic. The cervix was 7 cm dilated and edematous, and the infant's scalp registered a severe caput succedaneum. Cephalopelvic Disproportion (CPD) was the cause of obstructed labor. The patient was scheduled for an emergency cesarean section. The outcome was a live infant weighing 4 800g. Intraoperatively, a uterine rupture with laceration extending to the left lateral uterine wall was discovered. The patient had lost approximately 2 500 mL of blood. A sub-total hysterectomy was done, during which the patient suffered a cardiac arrest that lasted three minutes. Cardio-Pulmonary Resuscitation (CPR) and 1 mg adrenaline were admnistered. Spinal anesthesia was converted to general anesthesia. The surgery lasted one and a half hours. The patient was transfused with two units of blood. A drain was left in-situ to monitor bleeding.

The patient was reversed with difficulty and sustained a second cardiac arrest that lasted 2 minutes. Cardiopulmonary resuscitation and adrenaline were administered successfully. Her oxygen saturation level was 96%. She was transferred to the Intensive Care Unit (ICU) and maintained on intravenous (IV) adrenaline. The drain was noted to be active, and the bleeding was discovered from the right uterine artery. The artery was ligated to achieve homeostasis, and the patient transfused a third pint of blood. The patient was maintained on intravenous fluids, phenytoin, adrenaline, and antibiotics. Fifteen hours postoperatively, her drain was inactive, and her vitals revealed normotension, tachycardia (116 BPM), and 100% oxygen saturation.

On the first postoperative day, the patient was extubated and had spontaneous breathing. Her vital signs revealed a blood pressure of 103/76 mmHg, 124 bpm, and 100% oxygen saturation. Two days postoperatively, the patient was fully conscious with a Glasgow Coma Scale (GCS) score of 14/15. However, she had developed involuntary twitching and jerky movements of the lower limbs. The lower limb tone was increased, power registering 4/5 with brisk reflexes. The upper limb tone was reduced, with power 3/5 and normal reflexes. The patient's electrolytes at the time were 143, 3.49, and 8.5 mmol/L of Na^+, K^+, and Ca^{++}, respectively. She was started on haloperidol. On the fourth postoperative day, a post-hypoxic myoclonus diagnosis was made by a neurologist. She was managed with antibiotics and other supportive treatment. On the twentieth postoperative day, the patient was discharged on 0.5mg clonazepam twice a day for six months. At the time of discharge, the patient was not ambulant and was discharged through physiotherapy. Six months into the physiotherapy, the patient could stand and walk with support and presented with a minimal and controlled jerky movement. Currently, she can ambulate without support, albeit with an abnormal gait.

DISCUSSION

Acute post-hypoxic myoclonus occurs in 19-37% of patients who undergo successful resuscitation. Besides, acute PHM following successful cardiopulmonary resuscitation is associated with poor prognosis (4). A positive outcome for myoclonus patients is considerable recovery and moderate disability six months into physiotherapy. Within six months, the patient, in this case, could walk with support. She also presented with a minimal, controlled jerky movement. The pathophysiology of post-hypoxic myoclonus is poorly understood. However, it is thought that PHM originates from either or both subcortical and cortical structures (4, 5). Distinguishing the origin of the involuntary muscle jerks is critical for optimal treatment. There are no published guidelines for treating acute PHM, and caution is suggested while providing treatment (6). The primary treatment for cortical myoclonus is piracetam or levetiracetam, while clonazepam is used for subcortical myoclonus (5). A combination of pharmacology with physiotherapy is associated with favorable clinical outcomes (3). Improved outcomes with positive neurological function in patients whose PHM manifests early has been reported in the literature (7). In the early 2000s, it was common practice for the withdrawal of life support upon PHM's early onset. However, recent studies have negated that the condition almost always deteriorates to death (7), as it was in this case. Rehabilitation significantly improves life quality, although the patient may not be fully cured (8).

CONCLUSION

Post-hypoxic myoclonus was previously associated with a poor prognosis. However, recent findings have demonstrated positive clinical outcomes with pharmacotherapy and physiotherapy. Early diagnosis is critical for effective management.

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