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Synconvulsion in a Preeclamptic Patient

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Title: Convulsive Syncope vs Eclampsia

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Case:

A 35 year old, 65 kg, G2P0 female patient presented at 34 weeks gestation for a primary cesarean section (CS) due to worsening hypertension and headache. The patient had no significant medical history other than anxiety and was recently diagnosed with preeclampsia 12 hours prior to presentation. The patient’s medications included a magnesium sulfate (MgSO4) infusion at 2 gm/hr and intermittent intravenous administrations of 10 mg labetalol and 10 mg hydralazine doses. The patient’s preoperative blood pressure ranged from 162/85 to 196/97 with heart rate of 78-92 bpm. Her platelet count and liver function tests were within normal range. The anesthetic plan for CS was spinal anesthesia.

Upon arrival to the OR, the patient’s MgSO4 infusion was discontinued in compliance with standard intraoperative MgSO4 management at our institution. The patient was quite anxious with an elevated BP at 186/102 and a HR of 101. A 0.5 mg IV Midazolam dose was administered with good response. A spinal anesthetic was then placed on first attempt by the anesthesia resident at the L3-4 interspace with an intrathecal injection of 1.6 ml of 0.75% hyperbaric bupivacaine, 0.2 mg of duramorph and 10 ug of fentanyl. The patient was placed in left uterine displacement position, and a bilateral T4 sensory block was obtained using a pin-prick sensation measure. The patient had no complaints after recumbency. Her BP gradually decreased over 8 minutes and settled at 119/63 and with a HR of 70. Ephedrine 5 mg IV was administered at this time to maintain a relative baseline BP. Immediately after ephedrine administration, the patient began to have upper and lower body myoclonic jerks consistent with an eclamptic seizure. She was unresponsive to verbal and tactile stimuli. Mask ventilation with 100% oxygen was started immediately. The patient’s BP rose to 187/85 and the EKG showed sinus bradycardia at 56 bpm. The patient’s myoclonic movements lasted for approximately 5 seconds and ended spontaneously before a midazolam 0.5 mg IV dose was administered. She returned to baseline mental status within 45 seconds and had no recollection of the myoclonic activity. Over the subsequent 3 minutes, her BPs remained steady between 156/76 and 187/85 with a HR 61-68. The patient’s immediate return to baseline mental status and bradycardia was inconsistent with seizure activity, and therefore eclampsia remained as the presumed cause of her myoclonic activity.

MgSO4 was restarted at 2 gm/hr. The patient had no complaints and was prepped for surgery; The CS began approximately 10 minutes after the seizure. A healthy infant was delivered within 8
minutes of skin incision with APGAR scores of 7 and 9, at 1 and 5 minutes respectively. The patient had no further myoclonic activity and BP’s ranged from 136/74 - 160/82 and HR of 80-102 during the remainder of the case. She remained on MgSO4 for 24 hours after delivery and started on labetalol 400mg q 8 hours and Procardia XL 30 mg BID. Her postoperative course was uneventful with a BP range of 120s/70s - 130s/80s and was discharged home on postoperative day 5. A multidisciplinary team-review of the case concluded that the patient’s myoclonic movements were inconsistent with the clinical features of a seizure, given the short duration and sudden onset bradycardia. After a further case review, the patient’s seizure activity was determined to be due to convulsive syncope instead of eclampsia.

Discussion:

Myoclonus or myotonic jerks can often lead to misdiagnosis when differentiating between syncopal convulsions (SC), epilepsy and eclamptic seizures (ES). Confusion lies in their overlapping clinical features. All may involve a transient loss of consciousness caused by their different pathophysiology. Syncope is often attributed to reversible cerebral hypoperfusion that is sudden, resolves spontaneously and related to many non epileptiform causes such as vasovagal, situational syncope, carotid sinus hypersensitivity, cardiac issues, autonomic dysfunction, medications and cerebrovascular events. Eclamptic seizures are thought to have similar etiology via hypoperfusion but from cerebral vasospasm and cerebral edema. ES tends to occur in the setting of hypertension and proteinuria with the seizures being of short duration and varying post episode states. Epileptiform seizures are quite distinct with etiology from aberrant electrical brain activity and occur in the setting of auras and postictal states.

The overlap of visual similarities between epilepsy, CS and ES induced movements equivocates a proper diagnosis, which can have significant therapeutic consequences by delaying the appropriate intervention. Often times, advanced diagnostic tools such as videotelemetry with EEG and EKG recording may be required for proper distinction—a method that is quite impractical for patient receiving intraoperative anesthesia care. Such a diagnostic challenge was seen in our preeclamptic patient presenting with severe hypertension and headache. Her myoclonic activity was presumed to be eclampsia, and she was treated with a benzodiazepine and MagSO4. Upon review of the inconsistent clinical features of the patient’s seizure, such as seizure duration of 5 seconds, sudden onset bradycardia and immediate return to baseline mental status within 1 minute, the diagnosis was determined to be convulsive syncope, not eclampsia.

Diagnostic errors are frequent when loss of consciousness is followed by involuntary movements. The predominant cardiovascular mechanisms causing cerebral hypoperfusion and convulsive syncope are vasovagal reactions and bradyarrhythmias. In our presentation, we will present a detailed comparison of the clinical features of convulsive syncope versus other seizure types and discuss the physiological relationship between syncope and seizures.

References:


5. Bromfield E, Cavazos J, Sirven J, MD. An Introduction to Epilepsy [Internet]. West Hartford (CT): American Epilepsy Society; 2006.