Weathering the Sympathetic Storm: Cesarean Delivery as a Therapeutic Strategy for Refractory Paroxysmal Sympathetic Hyperactivity in a Parturient

Emilie Cohn MD, Matthew Sikora DO, Bushra Abdul Aleem MD

Department of Anesthesiology, Case Western Reserve University / MetroHealth
Cleveland, OH

PSH (a.k.a. "neurostorming") is a complication of severe neurologic injury characterized by episodic hyperthermia, hypertension, tachycardia, and dystonic posturing in response to stimuli

Pathophysiology: Increased excitotoxicity and sympathetic outflow in the brainstem, hypothalamus, and spinal cord with loss of inhibitory cortical control resulting in exaggerated response to external stimuli and uncontrolled sympathetic output

- Increased association with diffuse axonal injury, TBI, and young age
- Pregnant patients with TBI are at increased risk, though guidance in management is limited due to scarce case reports and unknown impact on maternal-fetal outcomes

PSH during pregnancy is especially rare, with only few case reports, none of which resulted in a live birth

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Case Summary

34-year-old G4P1 at 27w gestation presented following an MVA with diffuse axonal injury and ischemic stroke

Hospital Course

- Admitted to SICU intubated and sedated with continuous fetal monitoring; initial FHR monitoring was reassuring
- Day #4: Developed persistent hyperthermia and recurrent episodes of tachycardia and hypertension associated with fetal tachycardia and decelerations that improved with maternal stabilization
- Day #19: PSH episodes refractory despite increasing sedation regimen: propofol, dexmedetomidine, & fentanyl infusions, scheduled propranolol, diazepam, clonidine, meperidine
- Multidisciplinary discussion with anesthesiology, MFM, obstetrics neuro and neonatal ICU held to plan a scheduled C-section 30w5d under general anesthesia
- Prophylactic magnesium infusion was initiated for fetal neuroprotection one day prior to surgery with no overnight PSH events
- Cabergoline was started postoperatively for lactation suppression to further reduce metabolic demand

Anesthetic Plan:

IV Access: CVC and arterial line in place from ICU

TIVA: propofol, remifentanil, and dexmedetomidine infusions

Rocuronium and midazolam as adjuncts

Surgical uterine manipulation (potential PSH trigger) was minimized by in-situ repair

No intraoperative complications or PSH events occurred and a healthy neonate was delivered

Postpartum, PSH episodes decreased, allowing discontinuation of sedation by POD#10. Patient returned home with her infant within 2 months, ***achieving a full neurologic recovery*** ☺

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Case Discussion: Key Points and Takeaways

Therapeutic Strategy: Reducing Maternal Metabolic Demand

Cesarean delivery removes potential physiologic triggers (fetal movement) and delivery of fetus leads to a rapid decrease in maternal metabolic rate

Promotes maternal neurologic improvement, reduces frequency of PSH episodes, and mitigates ongoing risk of fetal acidosis with recurrent PSH episodes

Cabergoline can also be used postpartum to suppress lactation; further ↓ metabolic demand

Anesthetic Priorities

Proactive approach focusing on maintaining deep plane of anesthesia

Avoiding sympathetic stimulation and intra-op PSH triggers (minimizing uterine manipulation)

Tight hemodynamic control

Multidisciplinary coordination essential for timing of delivery and perioperative planning

Role of Magnesium Therapy

Vasodilation → improved perfusion & mitigation of hypertension with PSH episodes

NMDA receptor antagonism → ↓ excitotoxicity

Prior success in autonomic dysreflexia, with treatment leading to almost immediate resolution of episodes

Use in management of PSH warrants further investigation

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