Maternal Masseter Muscle Rigidity and Neonatal Fasciculations after Induction for Emergency Cesarean Section

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TRISMSUS, or masseter muscle rigidity (MMR), reportedly occurs in 1% of children following administration of halothane and subsequent intravenous succinylcholine.1 When MMR occurs in this clinical setting, it is reported to be associated with a 40–60% incidence of malignant hyperthermia (MH).2 In addition, children undergoing strabismus surgery have an even greater incidence of MMR than other children.3 This latter observation has led to an assumption that patients with underlying muscle disease are at higher risk for MMR and, consequently, MH.

The incidence of MMR after administration of succinylcholine in adults is unknown but most likely lower than in children in light of a reported incidence of 1 in 12,000 in a combined pediatric and adult population.4 The occurrence of MMR may be an indication of underlying muscle disease other than MH and should be investigated. The association of MMR with MH in the adult is likely similar to that of children and requires the same precautions and treatment.5 We report a case of MMR that occurred in a healthy parturient after intravenous succinylcholine for a rapid-sequence induction during emergency cesarean section and the associated neonatal fasciculations observed at delivery. Subsequent muscle biopsy studies resulted in a diagnosis of central core myopathy in mother and child.

**Case Report**

A 35-yr-old, gravida 2 para 1 woman at 34 weeks gestation had been doing well prenatally until admitted for leakage of fluid vaginally. An ultrasound revealed an estimated fetal weight of 2,285 g with a foaling breech presentation. Continuous fetal monitoring was begun, and 4 days into her admission, it was noted that the cervix was 3–4 cm dilated and a single foot and leg were presenting. The patient was informed of the need for an emergency cesarean section and taken to the operating room. She requested general anesthesia.

Evaluation at that time revealed an anxious, otherwise healthy gravid woman. She reported no prior surgeries or medical problems. She did not recall any anesthetic problems within her family. Physical examination revealed good temporomandibular joint mobility, good neck mobility, and normal laryngeal position.

While breathing 100% O2, she underwent a rapid-sequence induction with 3 mg intravenous curare, 250 mg intravenous thiopental, and 100 mg intravenous succinylcholine. Extreme difficulty opening her mouth was treated initially with an additional 100 mg intravenous thiopental and 50 mg intravenous succinylcholine. The masseter spasm persisted, but the trachea subsequently was intubated orally with great difficulty. Anesthesia was maintained until delivery with 50% N2O/50% O2 and atracurium for relaxation. Midazolam and fentanyl were added after delivery. Emergence and recovery were uneventful.

Postoperative review of her history revealed that the patient, her father, and her brother experienced debilitating calf pain on initiation of running. Creatine phosphokinase levels obtained immediately postoperatively and on postoperative days 1 and 2 were 10,550 IU/L, 9,798 IU/L, and 4,664 IU/L, respectively. She was discharged on day 7 with a creatinine phosphokinase level of 1,551 IU/L. Urine tested positive for myoglobin on day 1. A neurology consultation noted prolonged relaxation times when testing deep tendon reflexes, as can be seen in hyperthyroidism. Sensory and motor nerve conduction study results were normal, while an electromyogram demonstrated increased insertional and percussion activity consistent with membrane instability. A routine muscle biopsy performed for non-MH muscular disorders had a normal result, but electron microscopic examination of the muscle specimen resulted in a diagnosis of central core myopathy. The patient's clinical course worsened over the ensuing months with regard to her myopathy.

A premature male infant was delivered with Apgar scores of 2 and 4 at 1 and 5 min, respectively. He required tracheal intubation, and his lungs were mechanically ventilated for 12 h. Hypotonia, as well as facial and upper torso fasciculations, were noted at delivery. He was unable to flex his hips against gravity. At 1 h of age, train-of-four appeared normal. Creatine phosphokinase at 1 day was 745 IU/L. At 24 h, he still had no cough or gag with suctioning and a poor Moro reflex. An electromyogram result was mildly abnormal with asymmetry. A diagnosis of pseudobulbar palsy was considered. Over his 6-week admission, his neurologic status slowly improved, and he was sent home to be fed by nasogastric tube. At 5 months, he underwent Nissen fundoplication and gastrostomy tube placement without incident. The infant is being followed for a presumed di-
agnosis of central core myopathy, which is an autosomal dominant disorder. Muscle biopsy studies are planned in the future.

Discussion

This case is presented to demonstrate that a patient’s response to anesthetic drugs can be diagnostic for undiagnosed disease states. In our patient, the MMR following intravenous thiopental and succinylcholine was an early manifestation of central core myopathy. Central core myopathy is an autosomal dominant muscle disease characterized by a core of altered myofibrils in each muscle cell, resulting in a clinical picture of hypotonia and proximal muscle weakness.6

The association of MMR and central core myopathy with MH is of great importance in the clinical presentation we described. Although we elected not to treat this patient with dantrolene, we avoided any further triggering agents and monitored her for MH. The effects of dantrolene in patients with central core myopathy have not been reported. Masseter muscle rigidity after halothane/succinylcholine combinations is associated with a high incidence of positive biopsies for MH in children, but less is known about MMR after thiopental/succinylcholine combinations, particularly in adults.7,8

Regardless, when a clinician is presented with MMR, the patient must be monitored closely for the acute onset of fulminant MH because of the coincidence of MMR and MH susceptibility. Although our patient refused to travel for MH muscle biopsy studies, there is a strong association between central core myopathy and MH susceptibility.8,9 Our patient was informed of MH susceptibility in herself and in her son.

While it has been suggested that thiopental exerts a protective effect with regard to reducing the incidence of MMR after succinylcholine compared to halothane/succinylcholine alone, we believe that, when postsuccinylcholine MMR occurs after thiopental administration in adults, it is of such significance that a search for an underlying myopathy should be considered.10

During emergency surgery, canceling the procedure after an MMR response is usually not an option. Altering anesthetic plans by designing a trigger-free anesthetic is appropriate. With the utilization of continuous capnography, the need for immediate dantrolene therapy after MMR may be reduced. With fulminant MH, an early onset of hypercarbia should be detected by capnography in patients with a healthy cardiopulmonary system and dantrolene therapy instituted immediately if it occurs.11

We believe this infant’s brief fasciculations at delivery were a manifestation of the placental transfer of succinylcholine and its interaction with his myopathy. Historically, it was thought that succinylcholine could not pass transplacentally.12 However, Drabkova et al.13 have documented the transplacental transfer of succinylcholine in the monkey, whose hemochorial placenta is similar to humans. In humans, Cheraia et al.14 described neonatal respiratory depression secondary to the placental transfer of succinylcholine. Since this infant also was diagnosed with central core myopathy, succinylcholine served as the diagnostic tool in both patients. Fasciculations from succinylcholine in a normal neonate are unusual unless a underlying myopathy exists.15 Normal neonates can hydrolyze the small amount of transplacental succinylcholine quickly if the mother does not have an atypical pseudocholinesterase, which would result in a high maternal succinylcholine level and, therefore, transplacental passage of a sufficient amount of succinylcholine to result in paralysis and apnea in the neonate.14

In summary, we report a case of maternal MMR and neonatal fasciculations after administration of thiopental and succinylcholine for emergency cesarean section. Both mother and newborn were diagnosed with central core myopathy and MH susceptibility.

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References


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Paradoxical Vocal Cord Motion: An Unusual Cause of Stridor in the Recovery Room

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ACUTE inspiratory stridor in a patient recovering from the effects of general endotracheal anesthesia is a serious concern, because it may represent a life-threatening airway obstruction requiring immediate medical or surgical intervention. In a previously healthy preoperative patient, functional and organic anomalies of the upper airway can manifest postoperatively as acute inspiratory stridor. The differential diagnosis usually includes laryngeal stridor (from residual muscle relaxant effect), laryngeal spasm, vocal cord paralysis, intubation injury, hypocalcemic and alkalotic tetany, foreign body inhalation, angioedema, and undiagnosed tumors involving the upper airway. Abnormal vocal cord motion, although a well documented cause of stridor in other medical specialties, is usually not included in the differential diagnosis, because it is poorly documented in the anesthesia literature as a cause of recovery room stridor.1–5 This condition, therefore, may pose a significant diagnostic dilemma resulting in an inappropriate airway intervention. We present the case history of one such patient in whom timely diagnosis of this benign entity prevented an invasive airway intervention in the recovery room.

Case Report

A 49-yr-old healthy woman with a history of partial nasal obstruction presented to the ambulatory surgery center for nasal turbinectomy and excision of a nasal polyp. She had no history of recent respiratory tract infections, allergies, asthma, or angioedema, and had not been taking any medications at the time of surgery or immediately before it. The only past medical history was that of anemia during pregnancy. The patient was 165 cm tall and weighed 85 kg. The systemic examination was unremarkable except for the airway, which was graded to be Mallampati class 2, with adequate thyromental distance.4 No abnormality was detected on a routine chemistry, hemogram, chest x-ray, and electrocardiogram.

The patient received 2 mg midazolam intravenously in the preoperative holding area. Anesthesia was induced with 100 µg fentanyl,