Masseter Muscle Spasm following Succinylincholine administration

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INTRODUCTION

Masseter muscle rigidity is a rare (less than 1%) side effect of administration of succinylincholine and could be an early indicator of malignant hyperthermia.

The masseter muscle arises from the lower border of the zygomatic arch and attaches to the lateral aspect of the angle of the mandible. Its contraction elevates the mandible.

The masseter and lateral pterygoid muscles contain slow tonic fibers that can respond to depolarizing neuromuscular blockers with a contracture.

Masseter muscle is the strongest muscle based on its weight. With all muscles of the jaw working together it can close the teeth with a force as great as 55 pounds (25 kilograms) on the incisors or 200 pounds (90.7 kilograms) on the molars.

We present a case of masseter muscle rigidity following a rapid sequence induction of anesthesia using Succinylincholine in a patient with ruptured globe due to ocular trauma.

This case report highlights the possibility of MMR sequence variants that are likely to cause Malignant Hyperthermia (MH) or other Mendelian disorders related to these genes.

CASE DESCRIPTION

A 9 year, 45 Kg, healthy male with no history of anesthesia, a negative family history for anesthesia complications was brought to the OR for an emergent ruptured globe repair. The patient had nothing by mouth for more than 8 hours before surgery. After placement of peripheral IV and preoxygenation with 100% oxygen, anesthesia was induced with 90mg of Propofol and 90 mg of Succinylincholine to facilitate endotracheal intubation.

Following observing muscle fasciculation, initial attempt to open mouth was impossible due to masseter spasm. However patient was able to be ventilated without difficulty. Hemodynamic profile, temperature and ETCo2 remained stable. Physical exam revealed only masseter spasm. The masseter spasm lasted approximately 5 minutes and eventually the airway was successfully secured with an endotracheal tube.

Anesthesia was maintained with Sevoflurane and oxygen. The case proceeded uneventfully and vital signs remained stable. Skeletal muscle were examined intermittently throughout the procedure and remained relax. At the conclusion of Anesthesia, He became fully conscious, his muscle power was adequate with good tidal volume and stable hemodynamics. The patient was extubated without difficulty and transferred to the PACU.

The patient was admitted overnight for any signs or symptoms of possible MH as well as signs of rhabdomyolysis, CK level and electrolyte monitoring. The only abnormal lab value was a high CK (1245 and 1279 units/lit ; Reference range: 30-233 units/lit). After consultation with MHAUS, it was recommended the patient be referred to a genetic counselor and obtain a muscle biopsy.

Masseter spasm is defined as jaw muscle rigidity in association with limb muscle flaccidity and happens approximately in 1% of children receiving succinylincholine during induction for anesthesia1.

• If trismus occurs, proper monitoring should include Vital signs, EtCO2, examination for pigimenturia, serum CK, acid-base, and electrolyte level.

• If the jaw is slightly resistant to mouth opening, the anesthesiologist may continue anesthesia with proper monitoring.

• If the jaw is moderately tight, there are two choices: halt the procedure or continue with nontriggering agents.

• For patients with “jaws of steel,” if there is rigidity of other muscles in addition to masseter spasm, or any suggestion of MH, the procedure should be halted, and treatment of MH begun.

• Currently there is no indication to switch to a nontriggering anesthetic technique in instances of isolated masseter spasm2.

REFERENCES

1- Lymnae S, et al, Masseter Spasm with Anesthesia Incidence and Implications, Anesthesiology 1984, 775-776

DISCUSSION

The genotype test for Malignant Hyperthermia Susceptibility via RYR1, CACNA1S, and STAC3 Gene found no sequence variants that are likely to cause Malignant Hyperthermia (MH) or other Mendelian disorders related to these genes.