Masseter Muscle Rigidity (MMR): Definition, Relationship to Malignant Hyperthermia (MH), and Management

Developed in 2018

Background:

MMR can be generally defined as a marked difficulty in manual mouth opening that interferes with and impedes direct laryngoscopy and tracheal intubation without the presence of temporomandibular joint dysfunction. When MMR occurs in response to administration of succinylcholine in the absence of an underlying temporomandibular joint disorder or myotonia, it may be an initial sign of MH[1-4]. As early as 1969, Gibson et al reported two cases where they described the severe nature of the rigidity of masseter muscles when MMR occurs[5, 6]

Discussion:

Confusion often arises when diagnosing MMR due to its similarity with the normal but variable degree of increase in masseter muscle tension that may occur after succinylcholine administration in healthy patients [6-8]. This is an inherent characteristic of succinvlcholine administration and has also been linked to administration of subclinical doses in children[9, 10]. To differentiate between the normal rises in masseter tension versus a case of true MMR, assessing masseter rigidity is helpful. The term 'jaws of steel' (coined by Kaplan et al[11]) aptly emphasizes the severe nature of the rigidity. When MMR occurs, it may be both a harbinger of acute MH and/or associated with clinically significant rhabdomyolysis[1-4, 12, 13]. Therefore, clinicians should seek other concomitant signs of the presence of acute MH, such as tachycardia or hypercarbia that are inappropriate for the clinical situation, generalized trunk or limb rigidity, hyperthermia, cola-colored urine indicative of myoglobinuria, and/or peaked T waves or other arrhythmias consistent with hyperkalemia. However, in some patients who have subsequently progressed to MH, those signs did not appear immediately after MMR appearance. Sufficient evidence exists in the literature of cases in which MH ensued following MMR that it is prudent to cancel elective surgery when MMR occurs[1-4]. If the surgical procedure is emergent, then a non-MH triggering anesthetic should be instituted. Whether or not the case is cancelled, several hours of careful observation for additional signs of MH are warranted. This approach of employing non-triggering anesthetic in emergency cases, was first reported in a definitive fashion by Donlon et al in 1978[14] and later by others[15, 16]. The anesthesia provider should obtain a blood sample to screen for metabolic acidosis, hyperkalemia, and elevated creatine kinase levels. A urine sample should also be obtained to check for heme, which if positive without microscopic red blood cells may represent either myoglobinuria or hemoglobinuria. Serum creatine kinase measurements (CK) should follow immediately after and at 6-8 hours. CK may not be elevated immediately following MMR with peak levels not achieved until 12 to 24 hours following succinvlcholine administration[17]. If CK is greater than 5 times the upper limit for normal value, then appropriate treatment for rhabdomyolysis, including measures to prevent acute damage to the kidneys from myoglobinuria should be instituted [18]. While cola colored urine and elevated

creatine kinase may occur following MMR, development of any other additional signs of MH should prompt immediate dantrolene administration and other adjunctive therapies[1]. In patients who have myotonia, administration of succinylcholine may result in MMR and rigidity of the total body[19, 20]. Prior history of myotonia is the most helpful factor in differentiating between MMR and myotonic contractures

Conclusions:

MMR may be the first sign of an acute MH event. However, no conclusive data exist for clinicians to determine the likelihood of developing MH after an episode of MMR. If no other signs of MH are observed, the patient may still be at risk for developing clinically significant rhabdomyolysis and should be observed and treated as necessary. Patients who develop rhabdomyolysis without other signs of MH should be referred to a neurologist to rule out underlying myopathies. If no myopathies are found, evaluation for MH susceptibility may be indicated (<u>https://www.mhaus.org/testing/introduction-to-mh-testing/</u>). When an anesthetic is necessary in patients who experienced MMR during a previous anesthetic but have not had a full evaluation for MHS or myopathy, such patients should receive a non-triggering anesthetic for their procedure. Caveat: No MH or neurologic workup is indicated if no postoperative rhabdomyolysis or signs of MH occur and the patient informs the anesthesiologist that he/she has a history of temporomandibular joint disorder and/or his/her post-anesthetic examination reveals an inability to open the mouth well.

References

- 1. Rosenberg, H. and J.E. Fletcher, Masseter muscle rigidity and malignant hyperthermia susceptibility. Anesth Analg, 1986. **65**(2): p. 161-4.
- 2. O'Flynn, R.P., et al., Masseter muscle rigidity and malignant hyperthermia susceptibility in pediatric patients. An update on management and diagnosis. Anesthesiology, 1994. **80**(6): p. 1228-33.
- 3. Allen, G.C. and H. Rosenberg, Malignant hyperthermia susceptibility in adult patients with masseter muscle rigidity. Can J Anaesth, 1990. **37**(1): p. 31-5.
- 4. Larach, M.G., et al., Clinical presentation, treatment, and complications of malignant hyperthermia in North America from 1987 to 2006. Anesth Analg, 2010. **110**(2): p. 498-507.
- Gibson, J.A. and D.M. Gardiner, Malignant hypertonic hyperpyrexia syndrome. Can Anaesth Soc J, 1969.
 16(2): p. 106-12.
- Van der Spek, A.F., et al., The effects of succinylcholine on mouth opening. Anesthesiology, 1987. 67(4): p. 459-65.
- 7. Smith, C.E., F. Donati, and D.R. Bevan, Effects of succinylcholine at the masseter and adductor pollicis muscles in adults. Anesth Analg, 1989. **69**(2): p. 158-62.

- 8. Plumley, M.H., et al., Dose-related effects of succinylcholine on the adductor pollicis and masseter muscles
- in children. Can J Anaesth, 1990. **37**(1): p. 15-20.
- 9. Meakin, G., Underdosage with succinylcholine may lead to incorrect diagnosis of masseter spasm in children. Anesthesiology, 1988. **69**(6): p. 1025-7.
- 10. Meakin, G., R.W. Walker, and O.R. Dearlove, Myotonic and neuromuscular blocking effects of increased doses of suxamethonium in infants and children. Br J Anaesth, 1990. **65**(6): p. 816-8.
- 11. Rosenberg, H., et al., Malignant hyperthermia. Orphanet J Rare Dis, 2007. 2: p. 21.
- 12. Larach, M.G., et al., Prediction of malignant hyperthermia susceptibility by clinical signs. Anesthesiology, 1987. **66**(4): p. 547-50.
- 13. Rosenberg, H., Trismus is not trivial. Anesthesiology, 1987. 67(4): p. 453-5.
- Donlon, J.V., et al., Implications of masseter spasm after succinylcholine. Anesthesiology, 1978. 49(4): p. 298-301.
- 15. Christian, A.S., F.R. Ellis, and P.J. Halsall, Is there a relationship between masseteric muscle spasm and malignant hyperpyrexia? Br J Anaesth, 1989. **62**(5): p. 540-4.
- 16. Littleford, J.A., et al., Masseter muscle spasm in children: implications of continuing the triggering anesthetic. Anesth Analg, 1991. **72**(2): p. 151-60.
- 17. Antognini, J.F., Creatine kinase alterations after acute malignant hyperthermia episodes and common surgical procedures. Anesth Analg, 1995. **81**(5): p. 1039-42.
- 18. Petejova, N. and A. Martinek, Acute kidney injury due to rhabdomyolysis and renal replacement therapy: a critical review. Crit Care, 2014. **18**(3): p. 224.
- 19. Farbu, E., E. Softeland, and L.A. Bindoff, Anaesthetic complications associated with myotonia congenita:
- case study and comparison with other myotonic disorders. Acta Anaesthesiol Scand, 2003. 47(5): p. 630-4.
- 20. Parness, J., O. Bandschapp, and T. Girard, The myotonias and susceptibility to malignant hyperthermia. Anesth Analg, 2009. **109**(4): p. 1054-64.