Malignant Hyperthermia Susceptibility and the Trauma Patient

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Assorted casualties are expected from combat. Triage of the wounded may result in some going directly to surgery. Although every minute is essential, anesthetic care of these trauma patients must adhere to all established standards of care. A timely preoperative assessment must include identifying the patient's risk for malignant hyperthermia (MH). If a patient is found to be malignant hyperthermia susceptible, all appropriate measures must be taken to provide the patient with a safe anesthetic. In the forward, austere military environment, anesthesia providers may experience logistical and manpower constraints when administering anesthesia. In this setting, it may be more even more crucial for preoperative recognition of MH and when this is not possible, focus must shift to perioperative detection and early treatment. The following case report emphasizes the importance of preoperative recognition and having an established MH protocol and access to dantrolene.

Introduction

A 26-year-old male presented to the Emergency Medical Treatment (EMT) section of a level III Combat Support Hospital in Northern Iraq following a crush injury to the abdomen. The patient was conscious and described a history per interview of malignant hyperthermia (MH) diagnosed during a tonsillectomy as a child.

In the acutely injured trauma patient, circumstances may not allow for the preoperative identification of malignant hyperthermia susceptibility (MHS). Focus must then shift to preoperative recognition and treatment of the disorder. In this instance, MHS was immediately discovered preoperatively and the case proceeded with the avoidance of triggering agents. No changes indicative of MH, such as elevated central body temperature or end tidal carbon dioxide ($ETCO_2$), were detected. This report focuses on the importance of preoperative recognition and administering a trigger-free anesthetic for the trauma patient in the remote field anesthesia setting.

Case Report

A 26-year-old patient initially presented to a Battalion Aid Station (level I) in Northern Iraq after being pinned between two military vehicles, a Bradley and M88. Initial assessment at this level of care noted the patient was alert and oriented to person, place, and time, with no loss of consciousness; the patient's vital signs were stable with a slightly decreased blood pressure (BP), and physical examination revealed contusions on the abdomen. An intravenous line and fluids were started and the patient was air-evacuated to a higher echelon of care for further evaluation.

Upon arrival to the EMT section of a Combat Support Hospital (level III), on primary survey the patient had stable vital signs: BP, 130/80 mm Hg; pulse, 73 beats/minute; respiration, 28/minute; and oxygen saturation of 98%. The FAST ultrasound was positive for blood on the right side. Past medical history was unremarkable and past surgical history included a tonsillectomy and adenoidectomy as a child. The patient stated he was allergic to "succinylcholine" and had a "bad reaction" to anesthesia as a child. He was unsure if he had ever been tested for MH, but other family members "died" from anesthesia. The patient was sent immediately to the operating room for an exploratory laparotomy.

Initial vital signs on arrival to the operating room were BP, 143/83 mm Hg; pulse, 81 beats/minute; saturation, 100% on 100% oxygen via non-rebreather mask; and relative risk, 24. The patient was premedicated with 2 mg of midazolam. General endotracheal anesthesia was initiated using a rapid sequence induction consisting of 100 μg of fentanyl, 14 mg of etomidate, and 50 mg of rocuronium. The airway was secured without difficulty. General anesthesia was maintained with total intravenous anesthesia consisting of propofol, fentanyl, midazolam, and rocuronium. $ETCO_2$ and core temperature were monitored throughout the case. The patient was hand-ventilated with a clean circuit on the anesthesia machine and 100% oxygen.

The case proceeded with total intravenous anesthesia. Initial hemoglobin and hematocrit were 47.3 and 15.4, respectively. Approximately 2 hours after incision, there was a decrease in blood pressure to 80/40 and increase in heart rate to 110. Estimated blood loss was 1000 mL. The patient was transfused with 2 units of packed red blood cells (PRBCs). The propofol infusion was discontinued and anesthesia was maintained with rocuronium and intermittent boluses of midazolam. Multiple attempts to place a radial arterial line were made without success. Vital signs stabilized following the transfusion of blood for the remainder of the case. Total intraoperative fluids included 2 L of normal saline, 4 L of lactated Ringer's, and 2 units of PRBCs. Urinary output was 300 mL. The following surgical procedures were performed: repair of a transected right rectus muscle and fascia; an excision of devitalized jejunum with side-to-side jejunostomy; a resection of jagged mesenteric laceration; and the closure of a mesenteric defect. Approximate surgical time was 3 hours.

The patient was extubated at the end of the case and transferred to the intensive care unit. Postoperatively, the patient had an uncomplicated course in the intensive care unit for 3 days, after which the patient was air-evacuated to Germany (level IV) for further care. The patient did not receive any oral or intravenous dantrolene over his course of stay.

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Discussion

As sited in Barash,[1] MH was first described in *Lancet* [2] and, subsequently, in the *British Journal of Anesthesia*.[3] Since the initial description of MH in 1960, much has been learned about this rare life-threatening disorder. A cornerstone to the treatment of the potentially fatal syndrome is prevention by avoiding the agents that trigger the metabolic derangement associated with MH. In the acutely injured trauma patient, circumstances may not allow for preoperative recognition of MHS and focus must then shift to preoperative recognition and prompt treatment. In this instance, MHS was immediately discovered perioperatively and the case proceeded with avoidance of the triggering agents and monitoring of central body temperature and $ETCO_2$.

The pathophysiology of MH is well known. MH reaction involves an autosomal-dominant inherited sensitivity to triggering agents which, when used on MHS patients, can cause rapid accumulation of calcium in striated muscle myoplasm, resulting in muscle contracture followed by rhabdomyolysis and an intense heat-producing reaction.[4] The clinical picture is often dramatic with intense tachycardia, increased CO_2 production, muscle rigidity, respiratory and metabolic acidosis, hyperkalemia, and terminal hemodynamic collapse.[4]

The exact incidence of MH is unknown. The rate of occurrence has been estimated to be as frequent as 1 in 5,000 or as rare as 1 in 65,000 administrations of general anesthesia with triggering agents.[5] Those at risk for developing MH during anesthesia are survivors of a MH reaction or patients with a positive caffeine halothane contracture test; first-degree relatives of such patients or members of known MHS families with neuromuscular disorders; patients who suffer from Duchenne muscular dystrophy, King-Denboroug syndrome, or central core myopathy; patients who have exhibited masseter muscle spasm during anesthesia with halothane and succinylocholine; and patients with a history of neuroleptic malignant syndrome or heat stroke.[4] Per history, the patient in this case was a survivor of a MH reaction and was believed to have first-degree relatives with a history of MH.

MH had a mortality rate of nearly 80% at the time it was identified in 1960.[5] Improved awareness and understanding of the MH syndrome, better preanesthetic identification of MHS patients, along with much better intra- and postoperative monitoring and early use of dantrolene, has decreased mortality of acute fulminant MH episodes to 10%. Dantrolene has become the gold treatment for managing an acute MH crisis. Although the mechanism of action is still unclear, dantrolene appears to inhibit release of calcium from the sarcoplasmic reticulum to the myoplasm. Prophylactic use of dantrolene in MHS patients is still debatable. There is a very low incidence (0–0.63%) of MH reactions in MHS patients who receive a trigger-free anesthetic, thus making the practice of using dantrolene perioperatively not routine.[4] Consequently, the use of prophylactic dantrolene was not used in this case although it was available in the event of an emergency.

There have been no deaths reported from MH in previously diagnosed MHS patients when the anesthesia team is aware of the history.[1] Consequently, it is especially important for all members of the team to elicit the history from the patient at the earliest point in the health care system. In this case, the prehospital team noted the allergy to succinylcholine at the Battalion Aid Station. This information was relayed to the EMT department at the Combat Support Hospital and then further received by the anesthesia team. Early detection of the potential problem was key in this situation since the anesthesia team had little time to prepare for the emergency, thus a potentially life-threatening complication was avoided.

Recommendations for the management of the MHS patient include: use of nontrigger anesthetic agents to include nondepolarizing muscle relaxants; continuous $ETCO_2$ and central temperature monitoring; and a MH cart in the operating room stocked with an adequate supply of dantrolene. It is standard procedure to prepare the anesthesia machine by flushing the anesthesia machine with 10 L/minute oxygen for 10 minutes (reducing the anesthetic concentration to one part per million), replacing the fresh gas outlet hose, and using a new disposable anesthesia unit. The other option is to have a dedicated anesthetic machine for use only with MHS patents.[6] A dedicated anesthesia machine was not available and the patient's unstable status did not allow a delay of 20 minutes to prepare the available machine. Monitoring of the heart rate and temperature in the recovery room for at least 4 hours is also recommended. MH reactions have been reported to occur in the postoperative period even if nontriggering anesthetics were used. The patient was admitted to the intensive care unit for heart rate and temperature monitoring.

To avoid triggering agents (i.e., inhalational agents and succinylcholine), this patient received midazolom, fentanyl, propofol, and rocuronium. It has been noted that use of propofol/opiate combinations in the military field anesthesia setting for the shocked, hypovolemic or reduced cardiac reserve patient may put the patient at greater risk for hypoperfusion.[7] During this case, the propofol infusion was stopped until adequate volume was achieved with intravenous fluid and PRBCs to compensate for blood loss.

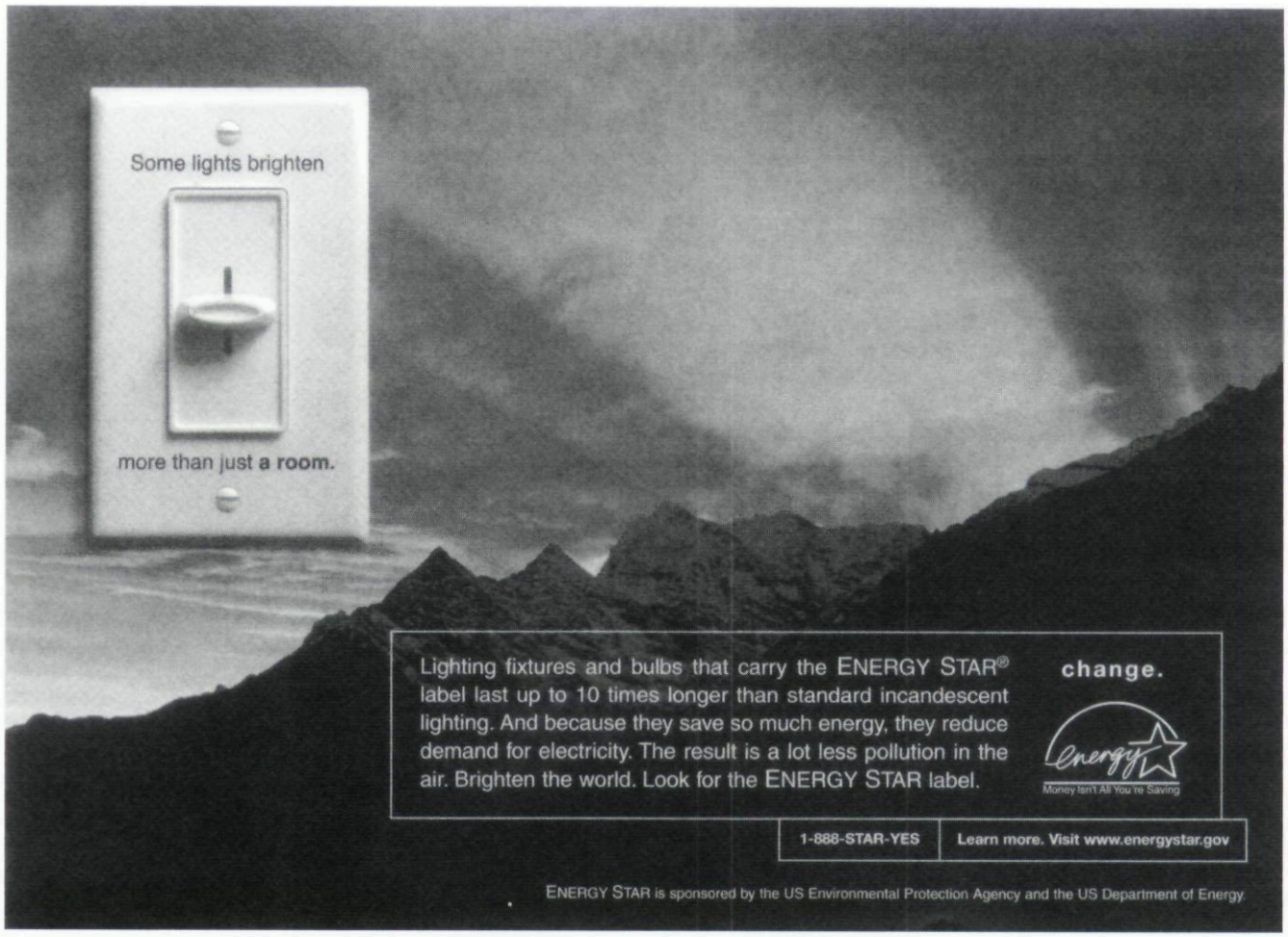
Continuous $ETCO_2$ and temperature monitoring via the Propaq monitor were employed throughout the case. $ETCO_2$ readings were consistently between 45 and 55 mm Hg and the nasal temperature was 37°C. Placement of a radial arterial line was unsuccessfully attempted numerous times; consequently, monitoring $PaCO_2$ with arterial blood gas sampling was not available. An arterial blood gas was obtained from a femoral stick at the end of the case. The data were pH 7.132; pCO_2, 79; pO_2, 461; HCO_3, 26; base excess, −3; and saturation, 100%. Although, the CO_2 was elevated, the patient had no other signs or symptoms consistent with a MH crisis. The increased CO_2 was assessed secondary to inadequate rate and depth of assisted hand ventilation during the procedure although the $ETCO_2$ readings were as noted above. One hundred percent O_2 was used throughout the procedure and oxygen saturation readings were consistently 100%. At the end of the case, muscle relaxation was reversed and the patient resumed spontaneous ventilation. When adequate tidal volume and rate were achieved as confirmed through spirometry and a repeat arterial blood gas revealed a normal pCO_2, the patient was successfully extubated and transferred to the intensive care unit. Continued monitoring in the intensive care unit revealed no signs of MH. On postoperative day 3, the patient was transferred for further definitive care out of the theater of military operations.

Conclusion

Especially in an austere environment, anesthesia providers need to be vigilant in assessing the patient for previous anesthesia history. Although every minute counts, a thorough assessment can make the difference in a successful patient outcome. In this case, the appropriate measures were taken, including the availability of dantrolene. To maintain this standard of care, military anesthesia providers must be advocates to ensure all soldiers that are MHS be identified through either predeployment screening or by carrying identification material. Military units performing major surgery in the combat setting must continue to maintain a sufficient supply of dantrolene and establish a protocol for handling a crisis.[8-10] If early recognition and efforts to avoid a MH crisis fail, treatment with dantrolene still remains the standard of care. Our soldiers deserve and should expect this standard in the combat setting.

References

1. Barash BF, Cullens C, Stoelting RK: Clinical Anesthesia, p 521. Philadelphia, Lippincott, 2001.
2. Denborough MA, Lovell RRH: Anaesthetic deaths in a family. Lancet 1960; 2: 45.
3. Denborough MA, Forester JFA, Lavell RRH, et al: l. Anesthetic death in a family. Br J Anaesth 1962; 34: 395.
4. Abraham B, Cahana A, Krivosic-Horber R, Perel A: Malignant hyperthermia susceptibility: anaesthetic implications and risk stratification. Q J Med 1997; 90: 13–18.
5. Schonell LH, Sims C, Bulsara M: Preparing a new generation anesthetic machine for patients susceptible to malignant hyperthermia. Anaesthsiol Intensive Care 2003; 31: 58–62.
5. Elster E, Harrison J, Stasiewicz S, Wang D, Golocovsky M: Malignant hyperthermia in the adult trauma patient. Am Surg 2002; 68: 883–5.
7. Lilly K: Considerations on propofol administration in the field anesthesia setting (echelons II and III). Milit Med, 1991; 156: 129–31.
8. http://www.mhaus.org/index.cfm/fuseaction/On-lineBrochures; accessed May 1, 2004.
9. http://www.aana.com/crna/prof/hyperthermia.asp; accessed May 7, 2004.
10. http://www.usamma.army.mil/publinsh-old/SB1/CH2.htm; accessed June 15, 2004.



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