Medical Journal of the
Islamic Republic of Iran

Volume 12
Number 2
Summer 1377
August 1998

Case Reports

SUPRAVENTRICULAR TACHYCARDIA FOLLOWING SECOND INDUCTION OF ANESTHESIA IN A PATIENT DIAGNOSED AS MALIGNANT HYPERTHERMIA AFTER FIRST ANESTHETIC EXPOSURE: TERMINOLOGY REQUIRES A SECOND LOOK

ZAHID HUSSAIN KHAN, M.D.

From the Department of Anesthesiology, Tehran University of Medical Sciences, Tehran, Islamic Republic of Iran.

ABSTRACT

A 17 year old male who underwent surgery for thoracic outlet syndrome developed a postoperative complication which was labelled and diagnosed as malignant hyperthermia in Kerman Medical Center. The temperature recorded was 43°C, $PaCO_2$ 70 mmHg and serum potassium level was 7 mEq/L. The patient was hospitalized in the intensive care unit for 10 days. On discharge, the patient had aphasia and incoordination in gait. Gradually the symptoms improved. However dysarthria, a reduction in muscle force and a staggering gait persisted. The same patient underwent a second operation for a spinal cord tumor located in the cervical region at this center. Following induction of anesthesia, the pulse rate showed a stepladder rise reaching a peak of 198 per minute. Except for a rapid heart rate, the patient had an uneventful operative course and a speedy recovery following the operation without any sequelae. The case is being reviewed and the plethora of diverse symptomatology and clinical picture explored to highlight the controversy of malignant hyperthermia in this particular case.

Keywords: Malignant hyperthermia, Anesthesia, Supraventricular tachycardia.

MJIRI, Vol. 12, No. 2, 159-162, 1998.

INTRODUCTION

The syndrome of malignant hyperthermia (MH) consists of skeletal muscle rigidity, rhabdomyolysis, myoglobinuria, elevated creatine phosphokinase, hypercapnia, hyperkalemia, metabolic and respiratory acidosis, cardiac arrhythmia and heat production.[1] A significantly large incidence of ventricular arrhythmias (13.2%) has been reported in children whose airways were managed via a face mask compared with those managed by endotracheal

intubation (3.3%).[2] An estimate and correct evaluation of the patient's perioperative risk is therefore imperative before administration of drugs capable of initiating MH in predisposed or susceptible individuals.

Case report

A young male scheduled for elective cervical laminectomy and resection of an intramedullary tumor appeared normal except for dysarthria and a gait abnormality, both of which were reminiscent of a complicated anesthesia and a compulsory admission in an intensive care unit in the past.

The patient had a dark complexion and slim built. Neurological assessment was normal.

The parents had undergone anesthesia and had seemingly had an uneventful course. The patient's uncle had died in the operating room while being operated for a fractured femur.

Preoperative assessment revealed the following data: B.P. 120/80 mmHg, pulse rate 84/minute, temperature 36.5°C, Hb 13g/dL, Hct 43%, WBC 4900/mm^3, serum Ca^{++} 10.1 mEq/L, Na^+ 140 mEq/L and K^+ 4.1 mEq/L. ECG was normal and CK 370 I.U. Arterial blood gas (ABG) provided the following information: pH 7.438, $PaCO_2$ 37.6 mmHg, PaO_2 87.4 mmHg, BE 1.4 mEq/L, HCO_3^- 24.7 mEq/L and SaO_2 97%.

Before starting anesthesia, oxygen 6L/minute was alllowed to flow through the system for a period of 10 minutes to wash out any residual anesthetic in the circuit. Meanwhile 500 mL Ringer's lactate solution was started to hydrate the patient. Monitoring included EKG, pulse oximetry, capnograph and an arterial line to monitor blood pressure.

Thalamonal was slowly started for induction. After injecting 7 mL thalamonal, the respiration slowed down to 4 per minute and the eye lash reflex could not be elicited. Oxygenation by mask was initiated and 6 mg pancuronium given i.v. to facilitate tracheal intubation. The patient was mechanically ventilated with a tidal volume of 600 mL and a respiratory rate of 14/minute. Following intubation, the heart rate started rising and reached 198/min after the patient was placed in the prone position. The mean arterial blood pressure (MABP) was 106 mmHg. Propranolol was started in increments of 1 mg each time and the patient received a total of 5 mg propranolol within a period of 5 minutes. Surgery was allowed to proceed when the HR reached 130/minute. At the start of surgery, HR was 140/minute, MABP 110 mmHg, O_2 sat 98-99% and $ETCO_2$ 22. During the early part of the operation, MABP was 130-140 mmHg and the HR ranged between 130-140/min and did not show any decline after injection of 150 micrograms fentanil. Pancuronium (2 mg) was repeated after every 45 minutes.

One hour after surgery, the ABG showed a pH of 7.48, $PaCO_2$ 28, PaO_2 397, [BE] -9, [HCO_3] 20.3 and an O_2 sat of 99.9%. During tumor resection, bleeding was profuse and 2 units of whole blood was transfused. At the end of surgery, residual neuromuscular blockade was reversed with prostigmine (2.5 mg) and atropine (1.25 mg) and the patient extubated. In the recovery room, the HR declined to 100/min. The patient responded well to verbal commands and was sent to the ward after having obtained a John Steward Score of 9/10. ABG and vital signs were monitored 4 hourly for the next 24 hours. The patient had a blood loss of 900 mL and a urinary output of 550 mL during the 3-hour operation. The patient received 3000 mL Ringer's lactate, 2 units of whole blood and 300 micrograms of fentanil during the operation. He had an uneventful postoperative course and was discharged on the 6th postoperative day. A quadriceps muscle biopsy taken during the operation revealed non-specific degenerative changes.

DISCUSSION

MH continues to haunt the surgical team in general and the anesthesiologist-in-charge in particular. This particular case developed hyperpyrexia (43°C), a serum K^+ level of 7 mEq/L and a $PaCO_2$ of 70 mmHg postoperatively at another center. The triad was dubbed as MH. After a 10 day stay in the ICU, the patient was discharged with a staggering gait, stuttering speech and lack of orientation to time and space.

In retrospect if we look at the case, the patient was fortunate to escape an attack during the second anesthetic exposure–if he had actually been MH susceptible. However, the absence of masseter muscle spasm (MMS) during anesthesia, no skeletal muscle rigidity and other findings suggestive of MH practically rule out the probability that the patient had developed MH during the first anesthetic exposure. Clinching the final diagnosis is difficult even in well-equipped centers. At present the only method for establishing non-susceptibility to MH is to demonstrate a normal *in vitro* contracture test (IVCT).[3]

Among the agents highly incriminated to cause MH are succinylcholine and halothane. MMS during anesthesia, especially during induction, occurs in patients of all ages, but more commonly in children who receive succinylcholine in combination with a volatile agent.[4] MMS occurs in 0.3% to 1% of children receiving inhalational agents along with succinylcholine.[5] There was no evidence of MMS during the first anesthetic exposure, and we also failed to notice MMS although we avoided the use of triggering agents. Some however argue that anesthesia can be continued safely in cases of isolated MMS when careful monitoring accompanies diagnostic evaluation.[4] Numerous diagnostic tools are utilized in patients prone to develop MH. Laboratory diagnosis of MH rests on the measurement of contracture tension generated in electrically stimulated muscle strips in the presence of halothane and caffeine. The MH rests on the measurement of contracture tension

generated in electrically stimulated muscle strips in the presence of halothane and caffeine. The contracture test however is invasive, expensive and time consuming. Owing to the fears of permanent disfigurement, the test is contraindicated in children weighing less than 20 kg, a population at substantial risk for MH.[1] Due to non-availability of the means to carry out this test at this center and difficulties in interpretation of the test, the patient was not subjected to this invasive test before undergoing anesthesia for the second time at this center.

Some take MMS to be of serious prognostic value and urge clinicians to disregard the advice that triggering anesthetic agents for MH be continued after MMS even with end tidal CO_2 and arterial gas monitoring.[5]

An increased CO_2 production is reflected in a raised end tidal level of CO_2 ($PETCO_2$), which is the most sensitive and specific sign for the presence of hypermetabolism, and also the earliest.[6] With capnographic monitoring, we could not find a rise in $PETCO_2$ which strongly favors that the patient did not develop MH during the first anesthetic exposure, although reportedly the patient had a raised $PaCO_2$ after the end of surgery. A raised $PaCO_2$ reported in this case could be attributed to inadequate minute ventilation, which is a common finding after oxygen is withheld from the patient and residual effects of the anesthetic persist.

Although elevated serum creatinine kinase (CK) levels lacks specificity,[6] our levels before anesthetic challenge were within the normal range and did not warrant further testing. The likelihood of MH susceptibility in patients with persistently elevated serum levels of CK is great enough to warrant a thorough neurological examination together with muscle biopsy.[7] It should however be borne in mind that increased levels of CK have been reported during exercise, trauma, etc.

Fever is a late sign of MH. The syndrome may take several hours of anesthetic exposure to develop[5] and fever up to 46°C has been observed.[8] The temperature was normal during the second anesthetic exposure at this center. It is speculated that the presence of a high temperature following the first anesthetic exposure and the development of dysarthria and a staggering gait could be attributed to a hypoxic brain insult of the basal ganglia and the hypothalamic region.

During the second anesthetic exposure at our center, the patient developed supraventricular tachycardia. This abnormal H.R. of 198/min seemed intractable to conventional modes of treatment. The rate slowed down after repeated increments of propranolol but never reached an acceptable level. The rate varied from 130-140/min during the surgical procedure. Paroxysmal tachycardia is characterized by sudden increases in heart rate up to 250 beats/min.[9] Ventricular performance was not impaired as evident from an adequate blood pressure. There had been no evidence of ischemia, electrolyte imbalance or a drug effect which could possibly have triggered this arrhythmia. Naturally this finding alone cannot be attributed to MH. A variety of factors such as the effects of anesthetics, the response to surgical or anesthetic stimuli, respiratory acidosis, cardiac reflexes, medications, exogenous or endogenous epinephrine and pre-existing diseases may account for this arrhythmia,[2] but we could not find a tentative cause in our case. A strong relationship between hypercarbia and ventricular arrhythmias[2] has been forwarded in pediatric patients managed by mask ventilation. Hypercarbia as a cause of arrhythmia in our case can be safely excluded because the patient was intubated and under controlled ventilation and the capnograph displayed a normal $ETCO_2$ throughout the surgical procedure. The incidence of persistent perianesthetic cardiac arrhythmias in children without pre-existing factors is approximately 10%,[2] and perhaps our case fell into this category.

Inadequate depth of anesthesia can also cause arrhythmias but this speculation was also ruled out in our case.

A very rare complication of Innovar is the malignant neuroleptic syndrome characterized by hyperthermia, muscular rigidity and autonomic instability.[10]

Tracing back the events, we feel that the label of MH after the first anesthetic exposure had been speculative, and after the second anesthetic exposure no findings suggesting MH could be found. It is suggested that appropriate measures should be adopted even if there is the slightest suspicion of MH. Whether the presence of isolated MMS heralds malignant hyperthermia is debatable, but nevertheless a second dose of suxamethonium is certainly not advocated.

Littleford et al.[4] reported that MMS may not herald the onset of MH and suggested that the anesthetic be continued after observation of isolated masseter spasm with appropriate monitoring. Although generalized rigidity is more ominous than isolated rigidity, we feel that anesthesia for elective surgery be aborted and testing for MH be considered to avoid any catastrophic outcome. Propofol is safe to use in MH-susceptible patients and offers a valuable alternative in the anesthetic management of these patients.[11] Since its release in Canada in Nov. 1990, propofol has been used as the primary anesthetic agent in three biopsy-proven MH patients without any adverse effects.[12] Physicians are advised to reserve the term MH to where tentative findings are established and avoid this label on intraoperative complications not related at all to MH. Conditions such as light anesthesia, thyroid storm, pheochromocytoma, drug and pyrogen reactions, congenital or acquired succinylcholine resistance, and other muscle diseases such as myotonic muscular dystrophy and disuse myopathy can mimic MH and should be included in the differential diagnosis.[8]

Adenosine is a recent addition to the family of antiarrhythmic agents which is useful in the treatment of

supraventricular tachycardia[13] and should be considered.

Calcium channel blockers such as verapamil have been advocated in supraventricular tachycardia, but it was not available in injectable form in the O.R.

We may conclude that, just as the "Holy Roman Empire" was neither holy, nor Roman and actually not an empire, similarly all intraoperative complications are not necessarily MH or related to this disorder and a little reservation should be exercised in diagnosing this dreaded and unfortunate complication.

ACKNOWLEDGEMENT

The author is grateful to Dr. Masood Kazemi (resident-in-training) for his interest in the case.

REFERENCES

1. Hogan CF, Power PA, Gregg RG: A cysteine for arginine substitution in the human skeletal muscle calcium release channel consegregates with malignant hyperthermia. Anesth Analg 75: 441-8, 1992.
2. Rolf N, Cote CJ: Persistent cardiac arrhythmias in pediatric patients: effects of age, expired carbon dioxide values, depth of anesthesia, and airway management. Anesth Analg 73: 720-4, 1991.
3. Ellis FR, Hopkins PM, Hallsal PJ, Christiam AS: Masseter muscle spasm and the diagnosis of malignant hyperthermia susceptibility (Letter). Anesth Analg 75: 143, 1992.
4. Littleford JA, Patel AR, Bose D, Cameron CB, McKillop C: Masseter muscle spasm in children: implications of continuing the triggering anesthetic. Anesth Analg 72: 151-60, 1991.
5. Rosenberg H, Shutack JG: Masseter muscle spasm in children (Letter). Anesth Analg 73: 361-2, 1991.
6. Van der Spek AFL: Triggering agents continued after masseter spasm: there is proof in this pudding (Letter). Anesth Analg 73: 364, 1991.
7. Lingaraju N, Rosenberg H: Unexplained increases in creatine kinase levels: its relation to malignant hyperthermia susceptibility. Anesth Analg 72: 702-5, 1991.
8. Miller JD, Chingmuh L: Muscle diseases. In: Katz J, Benumof JL, Kadis LB, (eds.), Anesthesia and Uncommon Diseases. Philadelphia: W.B. Saunders Co., pp. 626-629, 1990.
9. Herman R, Vetterman J: Change of supraventricular tachycardia to sinus rhythm during administration of propofol. Anesth Analg 75: 1030-2, 1992.
10. Guze BH, Baxter LR Jr: Current concepts: neuroleptic malignant syndrome. N Eng J Med 313: 163, 1985.
11. Gallen JS: Propofol does not trigger malignant hyperthermia (Letter). Anesth Analg 72: 413-14, 1991.
12. Allen G: Propofol and malignant hyperthermia (Letter). Anesth Analg 73: 359, 1991.
13. Lawson NW: Update on new cardiovascular drugs. Anesth Analg 82 (Suppl): S 74-80, 1996.