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RESEARCH ARTICLE

MALIGNANT HYPERTHERMIA - CASE REPORT

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ABSTRACT

Malignant hyperthermia (MH) is a severe and sudden increase in metabolism, the common clinical manifestations are: High central temperature, increase in ETCO2, hyperkalemia, acidosis, muscle rigidity and cardiovascular instability, after exposure to certain anesthetic drugs. Malignant hyperthermia occurs in persons who have a genetic alteration in the sarcoplasmic reticulum of striated.

INTRODUCTION

We describe a case of an acute onset of an MH episode in a female patient who underwent intubated general anesthesia for plastic surgery, without family history to anesthetic alterations and otherwise healthy. This case report describes the identification and management of this life-threatening condition in the ABC Medical Center in Mexico with a further discussion of the pathology.

Case Report

A 33-year-old female was scheduled for augmentation mammoplasty and dermolipectomy. Without pathological history or allergies, nonsmoker and airway without prediction of difficulty. The patient had previously undergone neuraxial anesthesia for C-section without complication. No family history of problems with anesthesia was elicited, only her sister received previous general anesthesia without any complications and neither the parents nor siblings had undergone any surgical procedures. The patient arrived at the OR having been NPO for solids and liquids for over eight hours, premedication with palonosetron 0.075mg and midazolam 1.5 mg was given. Oriented and conscious, the monitoring included; noninvasive blood pressure (NIBP), pulse oximeter (SpO_2), continuous electrocardiogram, capnography, end-tidal CO2 (ETCO2) ,esophageal temperature probe (placed immediately after induction), train of four, bispectral index and urinary catheter. Mask preoxygenation FiO2 100% for 3 minutes, induction with fentanyl 150 mcg, propofol 100mg, and rocuronium 50 mg, with easy mask ventilation (HAN scale 1).

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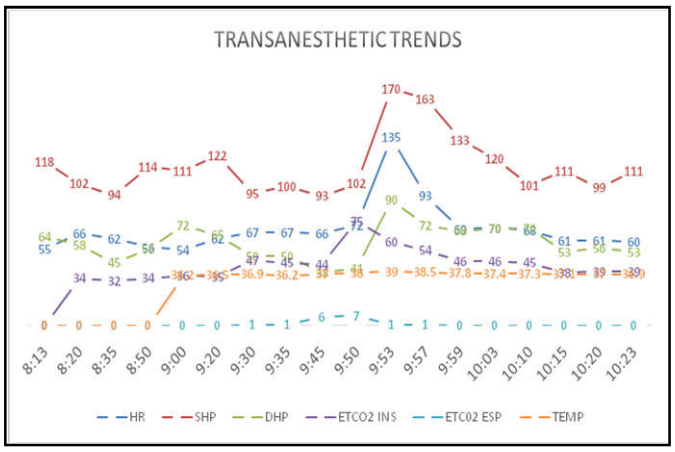
Following onset of muscle relaxant (TOF =0) the patient was intubated without difficulty, 7.5 oral tube. Bilateral breath sounds without wheezing and equal chest rise were noted, and end-tidal CO_2 ($EtCO_2$) was detected by capnography. The tube was secured, the eyes were taped, a thermic blanket and hot line for fluids was placed. After intubation, anesthesia was maintained with controlled mechanical ventilation, 300ml tidal volume, FiO2 65%, desflurane 6 vol%, dexmedetomidine at 1mcg/kg/hr, fentanyl 1mcg/kg/hr, and rocuronium 12mcg/kg/min continuous infusion. Baseline measurements at onset of surgery were SpO_2 at 99%, NIBP at 102/58, heartrate at 66 beats per minute, $EtCO_2$ at 32 mmHg, and esophageal temperature at 36.3°C. After induction, the $EtCO_2$ began to rise 1 or 2 mmHg, no suspicious was given at the moment and the ventilatory parameters were modified, nonetheless this few mmHg of ETCO2 raised again, at the auscultation bilateral breath sounds without wheezing were maintained, but flow-volume loop was observed with signs of a mild obstruction and hydrocortisone 100mg intravenous + salbutamol in spray through endotracheal tube was given to prevent a bronchospasm without any improvement. The following changes from baseline were noted in one-hour course: ETCO2 44 mmHg, heart rate 80, esophageal temperature was still 36.2°C. NIBP also was unchanged. Upon patient survey, no masseter rigidity was detected, and the upper and lower extremities remained lithe (the fentanyl, rocuronium and dexmedetomidine were on at the time). In the next 30 minutes ETCO2 raise to 55 and T° 36.9, so the thermic blanket and hot line for fluids was suspended and the temperature returned to 36.2 but few minutes later raised to 37°. Without any explication for these changes a presumptive diagnosis of malignant hyperthermia was made, the surgery was suspended; Stanford protocol was started and the emergency cart containing a malignant hyperthermia kit was obtained 9

minutes after. While waiting for help and cart, another intravenous line was obtained and arterial blood gas sample with metabolic acidosis and serum potassium of 4.77mEq/L was noted; the $EtCO_2$ had risen into 75 mmHg, T° 39°C, HR was now in 135 and NIBP 160/80 mmHg. Desflurane and rocuronium was discontinued and 100% O_2 was given. We continued manual hyperventilation and maintained anesthesia with continuous infusion of propofol. When other physicians arrived at the OR we divided ourselves in 2 groups and 1 leader:

- Group leader in charge of cognitive support with Stanford Manual
- In charge to prepare IV-line, cold fluids
- Reconstitution of Dantrolene with sterile water

Following the administration of the first 20 mg of dantrolene the HR were 70 bpm and NIBP 120/70 mmHg, a total of 112 mg dantrolene was given over the next 20 minutes. The patient's vital signs continued to improve, immediately prior to transport to the Critical Care Unit were: SpO_2 at 99%, NIBP at 111/53, HR at 60 bpm, and $EtCO_2$ at mmHg range in the 40s with mechanical ventilation and propofol infusion. She was admitted to the intensive care unit and was extubated 36 hrs later without complication, and kept in the intensive care for two days. During her stay she presented with elevation of potassium levels to 6.8 mEq/L, slight increase of creatine phosphokinase (CPK) 202 mcg/L but did not require further dosage of dantrolene. She was translated to a surgery roomdue to the lack of rabdomyolisis, no need for amines and stable hemodynamics. At discharge, the patient was advised to go to the MH Association of the United States for muscle biopsy and definitive diagnosis.



DISCUSSION

Malignant hyperthermia (MH) is a pharmacogenetic disorder that manifests as a hypermetabolic response to potent inhalation agents (such as halothane, isoflurane, sevoflurane, desflurane), the depolarizing muscle relaxant succinylcholine (Mullins, 2018). The incidence of malignant hyperthermia episodes during anesthesia is thought to be between one in 5000 and one in 50,000 to 100,000 anesthetic encounters, but because of accurate diagnosis, timely recognition, and appropriate treatment, mortality rates have fallen from 70 percent when the first cases came to light to less than 5 percent over 30 years later (Gurunluoglu, 2009). Unfortunately, in Mexico we don't have data about the incidence due of the lack of reports regarding this matter. Even though an MH crisis may develop at first exposure to anesthesia with those agents known to trigger an MH episode, on average, patients require three anesthetics before triggering (Mullins, 2018). MH is an autosomal dominant myopathy that most commonly involves mutations in the type 1 ryanodine receptor (RYR1) gene. This gene encodes the ryanodine receptor protein which is found mostly on skeletal muscle (Butala, 2018). During muscle contraction, the action potential arriving at the neuromuscular junction initiates a rapid increase in the intracytoplasmic concentration of Ca2+, which is released from the sarcoplasmic reticulum via induction of conformational changes in the dihydropyridine calcium channel receptor (DHPR) and RYR1. The relaxation phase is also an energy-dependent process as adenosine triphosphate is consumed by the intracellular Ca2+ pumps to sequester intracytoplasmic Ca2+ into the sarcoplasmic reticulum lumen. In malignant hyperthermia, there is a persistent increase in the intracytoplasmic Ca2+ [4] Which leads to increased binding of calcium to the myofilament, causing dysregulated skeletal muscle contraction. This contractile state results in rigidity and, eventually, skeletal myocyte breakdown. Rigidity in this setting results in a hypermetabolic state, causing increased body temperature over time.[3] This rise in body temperature can increase dramatically by 1º C to 2º C [1.8º F to 3.6º F] every five minutes. Sophisticated algorithms have been added to some anesthesia information systems to help perioperative personnel recognize a pattern of temperature change that can herald MH (Seifert, 2014). The physiologic effects of an increase in muscle activity are an increase in oxygen consumption and carbon dioxide production. This leads to an increase in minute ventilation which depends on the rate of carbon dioxide production and coexisting metabolic acidosis (Halsall, 2005). The early clinical signs are: increased body temperature, increased ETCO2, masseter muscle spasm, and tachycardia. [5]. A variety of unusual conditions that include sepsis, thyroid storm, pheochromocytoma, and iatrogenic overheating may resemble malignant hyperthermia during anesthesia.[2] Its important to recognize this signs and rule out the differential diagnostics to start the treatment as soon as possible to minimize the complications occurred by the elevation of myoglobin in serum by rhabdomyolysis.

Conclusion

In the presented case, the unexplained increase in the $EtCO_2$ level was the first sign of our concern, as is reported in the literature, so we should be aware of the importance of this monitoring, nonetheless the remain vital signs cannot be put aside. It is vital to perform a complete clinical history and emphasized about anesthetic complications in relatives especially in patients who reported no previous exposure to general anesthesia. In our country we do not have a center for definitive diagnosis of MH so patients have to be advised to travel to a country where a definitive diagnosis can be made. Unfortunately the cost of the biopsy and travel to a center in the United Stated made it impossible for our patient to get a definitive diagnosis but is still considering doing a DNA testing ad genetic counseling. In the ABC Medical Center the training residents have a program in simulation where once a week they rehearse clinical cases of anesthetic emergencies and its open to the staff, this can be a useful tool to perform Crisis Resource Management and we are sure that this program was a keystone for the success of the organization and prompt delivery of adequate treatment in this patient.

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