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Propofol in a patient at risk for malignant hyperthermia: report of a case

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Introduction

Malignant hyperthermia (MH) is a syndrome triggered by volatile inhalation anesthetics and/or succinylcholine. The symptoms are the result of hypermetabolism in skeletal muscle. Although the mortality rate has now dropped to less than 5%, MH is still one of the most serious complications of general anesthesia [1]. Although several reports have suggested that propofol is safe to use in patients who have a history of MH [2,3] or who are considered to be susceptible to MH [4–6], there have been no studies of changes in serum creatine kinase (CK), serum myoglobin (Mb), and urine Mb during and after propofol anesthesia. We report a case of the successful use of propofol in a patient with a history of MH.

Case report

A 25-year-old white man was scheduled for hemorrhoidectomy at our University Hospital in 1996. He had had an episode of MH during anesthesia for fracture of the humerus when he lived in the United States in 1980. On that occasion, halothane was used as the anesthetic agent. Unfortunately, his MH status, including clinical findings and laboratory data, was unknown. Neither he nor his family could remember the episode in detail. After he had successfully recovered from MH, he underwent skeletal muscle biopsy for the halothane/caffeine contracture test at the Uniformed Services University (Bethesda, MD, USA) in 1982, in which exposure to 2% halothane increased the basal tension induced by caffeine by 0.5 g. His responses to caffeine and to caffeine/ 1% halothane are shown in Table 1. These results showed that the patient was susceptible to MH.

Preoperative physical examination showed a height of 185 cm, weight of 67 kg, blood pressure (BP) of 98/59 mmHg, heart rate (HR) of 60 beats per minute (bpm), and axial temperature of 36.6°C. He had been known to have mitral valve prolapse since he was 20 years old, but the preoperative chest X-ray, 12-lead electrocardiogram (ECG), and echocardiogram were all normal. No abnormalities were seen on hematological and biochemical examination; his serum CK level was 147 $IU \cdot l^{-1}$ (normal level 57–183 $IU \cdot l^{-1}$). General anesthesia was scheduled because of the patient's wishes and with his complete consent after being informed of the advantages and disadvantages of the various anesthetic options.

He was premedicated with oral diazepam 5 mg, but dantrolene was not given. After the ECG monitor (leads II and V_5), noninvasive blood pressure monitor, pulse oximeter (SpO_2), and capnometer had been established, anesthesia was induced with propofol 140 mg, fentanyl 150 μg, vecuronium 8 mg, and oxygen. At the time of induction, BP was 110/65 mmHg and HR 78 bpm. A laryngeal mask was inserted into the hypopharynx without difficulty. Controlled mechanical ventilation with a vapor-free machine was initiated. A Foley catheter was inserted and the bladder temperature was found to be 36.7°C. Deep body temperatures at the forehead and palm were also monitored using the core thermometer (CTM-205, Terumo, Tokyo, Japan).

Anesthesia was maintained by means of a propofol infusion, initially at 10 $mg \cdot kg^{-1} \cdot h^{-1}$ and then at 6 $mg \cdot kg^{-1} \cdot h^{-1}$, with intermittent bolus doses of fentanyl and vecuronium. No abnormalities in BP or HR were

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Table 1. Results of the caffeine halothane contracture test

Caffeine response		Caffeine/1% halothane response	
Caffeine (mM)	Tension (g)	Caffeine (mM)	Tension (g)
0.25	ND	0.25	0.50
0.50	0.20	0.50	1.15
1.00	0.40	1.00	1.35
2.00	1.45	2.00	1.59
4.00	4.05	4.00	2.43

These tests were done at the Uniformed Services University in 1982. A positive caffeine contracture test is defined as the observation of the development of ≥0.2 g tension at 2 mM caffeine. A positive halothane/caffeine contracture assay is defined as the development of contracture more than 1 g after exposure to 1 mM caffeine or less in the presence of 1% halothane [13]. ND, Not determined.

seen during the operation. SpO_2 and end-tidal CO_2 ($EtCO_2$) during the operation were 99%–100% and 34–35 mmHg, respectively. The highest bladder temperature during the period of anesthesia was 36.8°C, and no elevation of the deep body temperatures at the forehead or the palm were observed. The urine remained clear. The total doses of drugs used during the 1-h operation were 661 mg of propofol, 175 μg of fentanyl, and 9 mg of vecuronium. In the intraoperative period, serum CK was 73 $IU \cdot l^{-1}$, serum Mb 60 $ng \cdot l^{-1}$ (normal level is up to 60 $ng \cdot l^{-1}$), and urine Mb 16 $ng \cdot l^{-1}$ (normal detectable level is up to 10 $ng \cdot l^{-1}$). Serum CK, serum Mb, and urine Mb were all measured using standard methods.

Reversal of neuromuscular blockade was achieved with 1.0 mg of atropine and 2.5 mg of neostigmine, and the laryngeal mask was removed 10 min after the discontinuation of propofol. Emergence was smooth and uncomplicated. The vital signs were BP 117/60 mmHg, HR 47 bpm, oxygen saturation (F_{IO_2} 0.21) 98%, and bladder temperature 36.5°C. He had no signs or symptoms of MH throughout the operation and the postoperative period. Serum CK was 62 $IU \cdot l^{-1}$, serum Mb 64 $ng \cdot l^{-1}$, and urine Mb 10 $ng \cdot l^{-1}$ 24 h later. The patient was discharged from the hospital on the second postoperative day. On the fifth postoperative day, serum CK was 100 $IU \cdot l^{-1}$, and serum Mb was 52 $ng \cdot l^{-1}$.

Discussion

Anesthetic-induced MH is a life-threatening event. Consequently, the management of anesthesia in MH-susceptible patients demands serious consideration. This patient had a known history of MH and the results of his muscle biopsy were positive. After we had discussed the anesthetic options and their risks with the patient, he refused regional anesthesia and chose general anesthesia. All volatile inhalation anesthetics and succinylcholine are known to trigger MH [1], whereas propofol and nondepolarizing relaxants are considered not to trigger MH [1] . Propofol has been tested in an MH-susceptible pig model and shown to be safe for use [7,8]. Moreover, it does not appear to induce contractures in vitro in skeletal muscle taken from either MH-susceptible humans or pig models [9]. Further, several reports [2–6] have suggested that propofol is safe to use in patients who have a history of MH or who are known to be susceptible to MH. Therefore, we decided to use propofol in this patient for both the induction and maintenance of general anesthesia, and vecuronium for muscle relaxation. The value of pretreatment with dantrolene is still debatable and is undergoing a reevaluation [10,11]. In this case, dantrolene was not given as a premedication, but an adequate supply was readily available for administration if MH syndrome did develop.

There were no clinical signs of MH such as increase in $EtCO_2$, tachycardia, myoglobulinuria, or temperature elevation during the course of the anesthesia in this case. The levels of muscle-related enzymes have not previously been studied during and after propofol anesthesia in patients at risk for MH. In this patient, serum CK remained within the normal range during and after anesthesia. Serum Mb and urine Mb were slightly above the normal level at several points during or after the anesthesia, but they were always far below the levels said to be associated with MH (>170000 $ng \cdot l^{-1}$ and >60000 $ng \cdot l^{-1}$, respectively) [12]. The slight elevations of serum Mb and urine Mb in our case were probably of no significance in terms of the development of MH. As we did not measure the preanesthetic levels of serum Mb and urine Mb, we could not establish whether changes had occurred in those variables since the preanesthetic period.

In conclusion, we report a case of the successful use of propofol in a patient with a history of MH. Our results support the idea that propofol will be safe to use in MH patients and MH-susceptible patients.

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