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Repeated total intravenous anesthesia for a patient with a history of enflurane-induced rhabdomyolysis

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Introduction

Becker-type progressive muscular dystrophy is an X-linked muscular dystrophy that is distinguished clinically from Duchenne-type dystrophy by late onset (mean age of 12 years old) and a more benign course [1]. Recently, immunologic dystrophin analysis has been indicated for diagnosis of the disease [2].

Because patients with Becker-type muscular dystrophy have abnormal reactions to some general anesthetics, we often encounter difficulties with anesthetic management [1,3–6]. The following is a case report of our experience with repeated total intravenous anesthesia (TIVA) for a patient with Becker-type muscular dystrophy who had developed rhabdomyolysis after enflurane anesthesia.

Case report

In 1990, an 11-year-old boy, weighing 38 kg, was admitted to our hospital to undergo a primary urethroplasty for treatment of hypospadia. A diagnosis of Becker-type progressive muscular dystrophy had been made when he was 6 years old. On preoperative examination, his serum creatine phosphokinase (CPK), respiratory function, and cardiovascular function were within normal limits. Anesthesia was induced and maintained with inhalation of 2.0%–4.0% enflurane in nitrous oxide (70%) and oxygen (30%). The anesthetic course was uneventful, and he recovered smoothly from the anesthesia.

However, about 2 h after the end of the operation, myoglobinuria and mild hyperthermia (38.1°C, oral) were observed. Also, we found a marked increase in his serum CPK, muscle–brain CPK (CPK-MB), and myoglobin. The maximum CPK level was 137 000 $U \cdot l^{-1}$ (normal range, 30–90 $U \cdot l^{-1}$), CPK-MB, 2000 $U \cdot l^{-1}$ (normal range, 0–8 $U \cdot l^{-1}$), and myoglobin, 2000 $U \cdot l^{-1}$ (normal range, 0–70 $U \cdot l^{-1}$). With a diagnosis of rhabdomyolysis, diuretic therapy with volume loading and intravenous furosemide, 2–4 $mg \cdot h^{-1}$, was started in our intensive care unit. The patient improved over the next 48 h and was discharged 10 days after the operation without any sequelae.

In the next year, a secondary urethroplasty was scheduled. His preoperative serum CPK was 1619 $U \cdot l^{-1}$, but his cardiorespiratory function was normal. Because his parents and surgeon refused any regional anesthesia and because the previous general anesthesia with enflurane-nitrous oxide had induced rhabdomyolysis, we decided to give total intravenous anesthesia (TIVA) with droperidol, fentanyl, and ketamine (DFK). For maintenance of anesthesia, we gave this patient total doses of droperidol, 11 mg (0.25 $mg \cdot kg^{-1}$), fentanyl, 600 μg (13 $\mu g \cdot kg^{-1}$), and ketamine, 180 mg (2 $mg \cdot kg^{-1} \cdot h^{-1}$). Although his arterial blood pressure was at the highest normal level, ranging from 140/80 mmHg to 160/90 mmHg during the entire course of the operation, no treatment for control of blood pressure was applied. The anesthetic course was uneventful, and he recovered from the anesthesia 40 min after the end of the operation. The serum CPK, CPK-MB, and myoglobin did not increase postoperatively.

Since then, he has undergone three plastic surgery procedures under TIVA (DFK) from the ages of 13 through 16 years. Although transient hypertensive

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Table 1. Serum creatine phosphokinase (CPK), muscle–brain CPK (CPK-MB), and myoglobin levels during six anesthesias

Parameter (normal range)		Pre-Op.	Pre-Induc.	Intra-Op.	Post-Op.	POD-1	POD-2	POD-3~10
CPK	Enfl.	185	—	—	35046	137000	49600	250
($U \cdot L^{-1}$)	DFK-1	1619	464	371	331	327	—	—
(30–190)	DFK-2	822	700	544	462	338	—	—
	DFK-3	569	725	712	609	442	414	497
	DFK-4	772	674	620	512	395	—	632
	PFK	708	542	449	515	566	451	—
CPK-MB	Enfl.	—	—	—	282	2000	770	19
($U \cdot L^{-1}$)	DFK-1	—	7	10	2	8	—	—
(0–8)	DFK-2	—	13	6	5	5	5	—
	DFK-3	—	7	6	3	1	—	0
	DFK-4	—	7	3	4	3	—	7
	PFK	—	7	4	5	5	6	—
Myoglobin	Enfl.	—	—	—	—	100	2000	—
($ng \cdot ml^{-1}$)	DFK-1	113	160	50	60	62	—	—
(0–70)	DFK-2	—	37	43	—	—	—	—
	DFK-3	—	97	53	87	52	40	60
	DFK-4	—	68	42	29	45	—	58
	PFK	—	—	—	—	—	—	—

Enfl., enflurane (2.0–4.0%) and nitrous oxide (70%); DFK, total intravenous anesthesia with droperidol, fentanyl, and ketamine; PFK, total intravenous anesthesia with propofol, fentanyl, and ketamine.

episodes were observed during anesthesia, he did not have any serious anesthetic complications.

At the age of 17, the patient was again admitted to our hospital for closure of the urethral fistula. His preoperative serum CPK was slightly elevated ($708\,U \cdot l^{-1}$), but all other laboratory data were within normal limits. To avoid the hypertension observed in previous DFK anesthesias, we chose propofol instead of droperidol. For maintenance of anesthesia, propofol, 5–$10\,mg \cdot kg^{-1} \cdot h^{-1}$, fentanyl, a total $100\,\mu g$ ($1.6\,\mu g \cdot kg^{-1}$), and ketamine, 0.8–$1.0\,mg \cdot kg^{-1} \cdot h^{-1}$ (PFK) were given. The intraoperative arterial blood pressure ranged from 100/55 mmHg to 110/60 mmHg. His recovery time from anesthesia was 14 min, much shorter than that with DFK. No anesthetic complications were observed. Serum CPK and CPK-MB did not change during or after anesthesia.

The perioperative serum CPK, CPK-MB, and myoglobin levels during these six anesthesias are listed in Table 1.

Discussion

Our patient developed rhabdomyolysis following enflurane–nitrous oxide anesthesia. Our result is comparable to that in a case of Umino et al. [7]. They observed myoglobinuria and an increase in the muscle-related enzymes such as CPK, lactic dehydrogenase, glutamic oxaloacetic transaminase, and glutamic pyruvate transaminase in a 22-year-old man after enflurane anesthesia. Patients with muscular dystrophy have an inherent membrane defect that may make the muscle susceptible to injury [1]. Also, Shinohara et al. [8] reported that enflurane itself increases serum levels of CPK and myoglobin in pediatric patients. Both Umino et al. [7] and Shinohara et al. [8] speculated that an increase in the permeability of the muscle cell membrane by enflurane is the most important factor contributing to myoglobinuria and/or increase in the muscle-related enzymes.

The body temperature of our patient increased slightly after the first operation. This finding, together with the increase in muscle-related enzymes, suggests that the rhabdomyolysis may have some relationship to malignant hyperthermia. In fact, Ohkoshi et al. [9] reported a case of clinically defined malignant hyperthermia in a patient with Becker-type muscular dystrophy. According to Heiman-Patterson et al. [10], a patient with Becker-type dystrophy had positive halothane contracture tests on muscle fiber bundles. These findings indicate that adverse reactions similar to malignant hyperthermia may occur from exposure to inhaled anesthetics in patients with Becker-type progressive muscular dystrophy.

When we were asked to care for the patient during the second urethroplasty, we confronted the problem of his management during anesthesia. We decided that general anesthesia under TIVA with droperidol, fentanyl, and ketamine would be possible for two reasons. First, the cytotoxic effect on muscle structures observed in enflurane–nitrous oxide anesthesia can

be minimized by TIVA. Second, we considered that TIVA has the least likelihood of inducing malignant hyperthermia.

The use of regional anesthetic techniques was also considered. However, we did not use these because urethroplasty is psychologically invasive for an adolescent male and because the patient's parents refused regional anesthesia.

To avoid the increase in arterial blood pressure associated with DFK, we selected PFK for the final operation. As compared to DFK, the patient's arterial blood pressure was maintained well without a hypertensive reaction. Propofol might have canceled the hypertensive reaction caused by ketamine. However, whether propofol is the anesthetic of choice in such patients is not clear from this case report. Myocardial involvement is often complicated in patients suffering from progressive muscular dystrophy [1,3]. This impaired cardiac function can be depressed further by the use of propofol, leading to life-threatening complications. For a successful outcome, anesthesia should be managed with great care with evaluation of the patient's general condition.

In summary, we report on repeated anesthesia in a patient suffering from Becker-type progressive muscular dystrophy. Although the patient developed rhabdomyolysis after enflurane–nitrous oxide anesthesia, he was safely managed during the following anesthesia by use of TIVA. We conclude that TIVA can be a promising choice for general anesthesia in patients with Becker-type progressive muscular dystrophy.

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