Clinical Reports

Rhabdomyolysis in association with Duchenne's muscular dystrophy

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Purpose: To present a case of rhabdomyolysis which developed in a child with a known history of Duchenne's muscular dystrophy, following an anesthetic which included sevoflurane.

Clinical features: An 11 yr old boy with a known history of Duchenne's muscular dystrophy underwent anesthesia for strabismus repair. The anesthetic consisted of sevoflurane and nitrous oxide without the use of a muscle relaxant. His postoperative course was complicated by a complaint of heel pain and the development of myoglobinuria. He was treated with dantrolene sodium and discharged home after two days, without further complication.

Conclusion: Sevoflurane anesthesia has not been shown previously to be associated with the development of acute rhabdomyolysis in a child with a history of Duchenne's muscular dystrophy. As with halothane and isoflurane, the continued use of sevoflurane in the presence of Duchenne's muscular dystrophy should be questioned.

Objectif : Présenter le cas d'un enfant aux antécédents de myopathie de Duchenne, chez qui s'est développée une rhabdomyolyse à la suite d'une anesthésie comprenant du sévoflurane.

Éléments cliniques : Un garçon de 11 ans aux antécédents de myopathie de Duchenne a reçu une anesthésie pour la réparation d'un strabisme. Les anesthésiques comprenaient du sévoflurane et du protoxyde d'azote sans myorelaxant. Des complications postopératoires sont survenues: talalgie et myoglobininurie. L'enfant a été traité avec du dantrolène et il a quitté l'hôpital deux jours après sans avoir présenté d'autres complications.

Conclusion : L'anesthésie au sévoflurane n'a jamais été associée au développement d'une rhabdomyolyse aiguë chez un enfant souffrant de myopathie de Duchenne. Comme dans le cas de l'halothane et de l'isoflurane, l'utilisation continue de sévoflurane chez un patient atteint de myopathie de Duchenne devrait être remise en question.

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ALTHOUGH there are descriptions of complications arising from the use of halothane and isoflurane¹⁻² in the presence of Duchenne's muscular dystrophy, no cases of such complications have been described in association with the use of sevoflurane. We describe an 11 yr old boy who developed acute rhabdomyolysis following an anesthetic with sevoflurane during strabismus repair.

Case Report

An 11 yr old, 40 kg boy was admitted for the treatment of strabismus. At about three years of age, difficulty in walking became apparent and gradually progressed. At the age of eight years, Duchenne's muscular dystrophy was diagnosed. The boy had a history of bronchial asthma starting at two years of age, treated with 25 mg prednisolone *po* and sometimes with theophylline in acute episodes. For his progressive muscle atrophy, he had continued to undergo physiotherapy.

Preoperatively, he had atrophy of the proximal muscles, pseudohypertrophy of the calf muscles, and slight scoliosis. Although he used a wheelchair, he was able to operate independently in his daily life. Preoperative laboratory data were: AST 76 IU·L⁻¹ (normal: 11-30); ALT 136 IU·L⁻¹ (normal: 5-42); CPK 4,430 IU·L⁻¹ (normal: 40-182); BUN 6.6 mg·dL⁻¹ (normal: 8.6-21.6); creatinine 0.14 mg·dL⁻¹ (normal: 0.46-0.82). Left ventricular hypertrophy was observed on ECG.

Preanesthetic medication was not given. In the operating room, an *iv* catheter was placed in the left hand, and an inhalational induction was employed using sevoflurane 4%, nitrous oxide 66%, and oxygen. The trachea was intubated easily under deep sevoflurane anesthesia, and neither muscle relaxant nor atropine was required. After induction of anesthesia, 100 mg hydrocortisone were administered. During the operation, anesthesia was maintained with sevoflurane 1.5-3.0 %, nitrous oxide 64 %, and oxygen under assisted ventilation with spontaneous breathing. The surgical procedure lasted 51 min, and the anesthesia time was 98 min. A total of 250 ml acetated Ringer's solution was given during the operation. Rectal temperature varied 36.7-36.9°C; and the $P_{ET}CO_2$ 35-42 mmHg. Fever, muscle rigidity, and arrhythmia indicating the onset of malignant hyperthermia were not observed. After discontinuation of anesthesia, the patient promptly regained consciousness, and the trachea was extubated. The patient was given 25 mg diclofenac *pr* for postoperative pain. He complained of slight pain in his eye and in his feet in the recovery

FIGURE 1 Change of perioperative serum CPK. Serum CPK indicated maximum when myoglobinuria appeared three hours postoperatively, decreased gradually, and recovered to preoperative value on the tenth postoperative day.

FIGURE 2 Changes of perioperative serum AST, ALT, and LDH. The enzymes that leaked from muscles elevated when myoglobinuria appeared three hours postoperatively and decreased gradually.

room, but the pain was not severe enough to necessitate additional analgesics. The axillary temperature in the recovery room was 36.1°C.

On the ward, the patient did not complain of any symptoms except for heel pain during the first few hours. However, three hours postoperatively, 300 ml of dark red urine were passed. The urine contained 100 mg·dL⁻¹ protein; was strongly positive for occult blood, and had a pH of 6.0. The dark red urine contained a large volume of myoglobin, 20,573 ng·mL⁻¹ (normal value < 20). The axillary temperature at that time was 37.3°C. The laboratory results revealed an

increase in serum enzymes: AST 458 IU·L⁻¹, ALT 254 IU·L⁻¹, CPK 55,700 IU·L⁻¹, LDH 1,626 IU·L⁻¹ (normal: 101-193). Serum BUN, creatinine, K, and Ca were 10.4 mg·dL⁻¹, 0.07 mg·dL⁻¹, 4.2 mEq·L⁻¹, and 3.6 mg·dL⁻¹, respectively. Arterial blood gas analysis showed pH 7.40, PaO₂ 116 mmHg, PaCO₂ 33 mmHg, and BE -2.8 mEq·L⁻¹ while breathing 2 L·min⁻¹ oxygen via nasal cannula. After the episode, fluid was administered at 5 ml·kg⁻¹·hr⁻¹ along with 1 mg·kg⁻¹ dantrolene sodium. The pain in his feet that he had complained of in the recovery room disappeared eight hours postoperatively. The dark red urine gradually disappeared and became clear concurrently with the patient ceasing to complain of pain. The next morning, the occult blood test for the urine was negative, and he was discharged on the second postoperative day without any other complication. On the tenth postoperative day, serum enzymes of AST, ALT, and CPK returned to preoperative values (Figures 1, 2).

Discussion

We observed markedly elevated myoglobinuria after sevoflurane anesthesia in a case of Duchenne's muscular dystrophy. We believe that the rhabdomyolysis in this case could have been caused by the inhalation of sevoflurane because the other factors that induced rhabdomyolysis could not have been considered.

There have been cases of rhabdomyolysis following anesthesia with halothane,¹ enflurane,³ and isoflurane² without succinylcholine in muscular dystrophy. Two cases^{1,3} were similar to our case in that anesthesia were uneventful, but myoglobinuria was observed for the first time when dark red urine was passed postoperatively. In the isoflurane case² rhabdomyolysis occurred postoperatively after cardiac arrest in the recovery room without any symptoms during anesthesia. There is a report that inhalation anesthetics are contraindicated in patients with Duchenne's muscular dystrophy,⁴ while on the other hand a report that indicate the safety of the use of them.⁵ Although sevoflurane had not been shown to be associated with similar complications, we consider it is possible that rhabdomyolysis is caused by sevoflurane similar to the other agents.

Several other factors than inhalation agents may have been associated with development of rhabdomyolysis. Although there have been reports of rhabdomyolysis developing after the administration of diclofenac⁶ and acetaminophen⁷ used in this perioperative period, our case would be different from them in the amount of drugs and taking a alcohol together.

Rhabdomyolysis might have been caused by muscle compression due to an inadequate position during anesthesia.⁸ As this patient complained of heel pain in

the recovery room, muscle compression and injury was thought to be a cause of the rhabdomyolysis. But the duration of the surgery was not long enough to induce muscle injury. Moreover, as we were very careful with the patient's position, it appears that muscle compression should be eliminated as a possible cause of the rhabdomyolysis.

In conclusion, this patient with Duchenne's muscular dystrophy developed acute rhabdomyolysis secondary to the use of sevoflurane. Because of this complication, we believe that caution should be exercised with the continued use of sevoflurane in the presence of Duchenne's muscular dystrophy.

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