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Cardiac arrest and rhabdomyolysis after succinylcholine in a healthy child

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Introduction

Intractable, unexpected cardiac arrest following use of succinylcholine has been reported in children who are apparently in good health preoperatively. Most of these patients were boys who were subsequently found to have occult myopathies, primarily Duchenne's muscular dystrophy [1–7]. Some, if not most, of these patients developed clinical signs compatible with malignant hyperthermia (MH). We report a case of sudden cardiac arrest after administration of succinylcholine, followed by severe rhabdomyolysis without hyperthermia, in a healthy girl. Possible association with MH is discussed.

Case report

A 9-year-old girl weighing 32 kg presented for adenoidectomy and myringotomy. The patient had had an inguinal hernia repair under halothane/N_2O/O_2 anesthesia without problems 2 years previously. The patient had no signs or symptoms of muscle diseases. There was no family history of muscle diseases or of adverse reactions to anesthesia. The results of preoperative laboratory examinations were all within normal limits: specifically, creatine kinase (CK) was 159 IU·l^{-1} (normal range, 0–180 IU·l^{-1}).

Atropine (0.5 mg) was given intramuscularly 60 min prior to induction. Monitoring of noninvasive blood pressure, electrocardiogram (ECG) and oxygen saturation was started. Anesthesia was induced with intravenous midazolam (3 mg) and inhalation of 3% sevoflurane in oxygen for 3 min. Succinylcholine (30 mg) was given intravenously (i.v.) to facilitate tracheal intubation. Masseter as well as generalized muscle rigidity occurred and the mouth was opened with considerable difficulty. A tracheal tube was put in position. Two minutes after administration of succinylcholine, ECG showed a peaked T-wave immediately followed by ventricular fibrillation. External cardiac massage was started, and an i.v. bolus of 60 mg lidocaine and 50 μg adrenaline were given. Cardiac activity was restored in less than 10 min. Stat arterial blood gas and electrolytes results taken approximately 5 min after cardiac arrest were as follows: pH, 7.14; $PaCO_2$, 47 mmHg; PaO_2, 342 mmHg (FIO_2 = 1.0); base excess, −8.5 mM; potassium, 7.16 mEq·l^{-1}. Suspecting MH, 20 mg dantrolene was also given i.v. during cardiac resuscitation. Twenty milliliters of 50% dextrose with 4 units of insulin, as well as 5 ml of 2% calcium chloride, were given to treat hyperkalemia. Cola-colored urine was noted, which was later found to be myoglobinuria. Urine output (>1 ml·kg^{-1}·h^{-1}) was maintained with furosemide and mannitol. During this acute episode, creatine kinase was 5210 IU·l^{-1}. Throughout the episode, rectal temperature was between 36.2 and 36.4°C.

The patient was transferred to the intensive care unit (ICU) in a stable cardiorespiratory condition. She was awake and neurologically intact upon admission to ICU, and was extubated. To prevent a return of symptoms, four doses of 20 mg dantrolene were given every 6 h. Her maximal temperature while in ICU was 37.9°C within 3 h from induction. She did not develop cardiac or renal failure, or coagulopathy. The patient also did not show metabolic or respiratory acidosis, or cardiac arrhythmia in ICU. CK values increased to a maximum of 57 000 IU·l^{-1} within 24 h and 98 000 IU·l^{-1} within 48 h, and then decreased over 5 days. She made an uneventful recovery and was discharged to the pediatric ward on the fourth postoperative day. Two weeks later, she

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had myringotomy under local anesthesia with lidocaine. Muscle biopsy under general anesthesia with non-triggering agents was performed 3 months later. No problems were encountered during these two anesthesias. Histological examination, as well as the result of a calcium-induced calcium release (CICR) test, was normal. Other members of her family were not tested for MH susceptibility.

Discussion

Although the clinical manifestations of fulminant MH are quite dramatic, most of the signs are not unique to MH and are of variable intensity and time course, and therefore early diagnosis during anesthesia is often difficult [8]. Another cause of diagnostic difficulty might be the early use of dantrolene, which can halt the hypermetabolic process in the muscles before full-blown MH develops. Lack of internationally accepted clinical diagnostic criteria also leaves clinicians puzzled when attempting to diagnose equivocal cases.

According to the recently published clinical grading scale of MH [9], the initial score of the patient in this case was 33 (15 points for generalized muscular rigidity, 15 points for elevated CK ($>20000\,IU\cdot l^{-1}$), and 3 points for ventricular fibrillation), and her MH rank was 4, i.e., the likelihood of MH was somewhat greater than normal.

The caffeine halothane contracture test (CHCT) has previously been the "gold standard" for diagnosing MH susceptibility in North America and Europe. However, four false-negative results of CHCT have been documented in patients who were almost certain to be MH-susceptible [10]. In Japan, however, CICR from sarcoplasmic reticulum using skinned muscle fiber has been studied as a diagnostic test of MH-susceptibility [11].

The major clinical difference between fulminant and abortive MH is that the maximal temperature is above 40°C or the rate of temperature increase is more than $2°C\cdot h^{-1}$ in the former, while the latter does not fulfill this criteria of hyperthermia. Results of CICR also differ significantly between fulminant and abortive MH; 87% of fulminant MH cases had accelerated CICR, while 82% of abortive MH had normal CICR [12]. This is a striking difference, and it is not yet clear if these two types of MH should be considered clinically different entities, or whether both types just lie in the wide spectrum of a single clinical entity. Increased recognition of this potentially fatal disorder and early initiation of specific treatment, i.e., the use of dantrolene, may have led to an increase in the number of abortive cases of MH being reported rather than unequivocal cases.

Dantrolene is effective for MH because it inhibits CICR. It is not known whether dantrolene is also effective for MH cases in which the rate of CICR is not accelerated. Although it is difficult to know if dantrolene had any effect on this patient's favorable outcome, it would be both reasonable and practical to administer dantrolene during an acute MH-like episode even when the patient is subsequently found to have normal CICR. Dantrolene is not acutely toxic, and might be helpful after sudden cardiac arrest following succinylcholine, as the clinical differentiation from MH is often difficult. However, the effectiveness of dantrolene under life-threatening circumstances does not necessarily imply that the situation is due to MH. Dantrolene has been used effectively to treat neuroleptic malignant syndrome and thyrotoxic crisis [13].

Most cases of cardiac arrest after succinylcholine in pediatric patients have occurred in boys affected with Duchenne's muscular dystrophy. However, there is a report of a patient who developed masseter muscle rigidity and increased CK after succinylcholine without muscle disease or MH-susceptibility as evidenced by a normal muscle biopsy specimen and a normal CHCT [14]. The case in our report is another patient who had a cardiac arrest following sevoflurane and succinylcholine, but who did not show any abnormality with muscle biopsy and CICR. As the prevalence of MH in Japan is unknown, the predictive value of negative CICR cannot be calculated. Although the present patient did not fulfill the clinical criteria of fulminant MH, we believe that when the clinical (cardiac arrest, rhabdomyolysis) and biochemical (hyperkalemia, increase in CK) findings occur in reaction to anesthetic drugs, patients should be managed clinically as MH-susceptible in future anesthesia.

The case reports available to the MH Hotline in the United States suggest that sudden unexpected cardiac arrest in reaction to succinylcholine occurs about five to six times per year across the U.S. In view of the lack of an effective way to recognize patients at risk of a hyperkalemic response to succinylcholine, the Food and Drug Administration in the U.S. determined to contraindicate succinylcholine except in cases for which it is clearly indicated [15]. At the moment, the situation regarding the use of succinylcholine in pediatric as well as adult patients in Japan is quite different from that in the U.S. The authors are of the opinion that the decision of indication or contraindication should be left to clinicians and not to pharmaceutical companies. However, the report of this case may prompt reconsider ation of the use of succinylcholine in elective pediatric anesthesia [16].

References

1. Rosenberg H, Gronert GA (1992) Intractable cardiac arrest in children given succinylcholine. Anesthesiology 77:1054
2. Miller ED, Sanders DB, Rowlingson JC, Miller ED, Sanders DB, Rowlingson JC, Berry FA, Sussman MD, Epstein RM (1978) Anesthesia-induced rhabdomyolysis in a patient with Duchenne's muscular dystrophy. Anesthesiology 48:146–148
3. Wang JM, Stanley TH (1986) Duchenne muscular dystrophy and malignant hyperthermia—two case reports. Can Anaesth Soc J 33:492–497
4. Henderson WAV (1984) Succinylcholine-induced cardiac arrest in suspected Duchenne muscular dystrophy. Can Anaesth Soc J 31:444–446
5. Mehler J, Bachour H, Simons F, Mehler J, Bachour H, Simons F, Wolpers K (1991) Cardiac arrest during induction of anesthesia with halothane and succinylcholine in an infant. Anaesthetist 40:497–501
6. Stelzner J, Kretz FJ, Rieger A, Stelzner J, Kretz FJ, Rieger A, Reinhart K (1993) Anaesthesia-induced cardiac arrest in two infants with unsuspected muscular dystrophy. Anaesthetist 42:44–46
7. Solares G, Herranz JL, Sanz MD (1986) Suxamethonium-induced cardiac arrest as an initial manifestation of Duchenne muscular dystrophy. Br J Anaesth 58:576
8. Rosenberg H (1988) Clinical presentation of malignant hyperthermia. Br J Anaesth 60:268–273
9. Larach MG, Localio AR, Alloen GC, Larach MG, Localio AR, Allen GC, Denborough MA, Ellis FR, Gronert GA, Kaplan RF, Muldoon SM, Nelson TE, Ording H, Rosenberg H, Waud BE, Wedel DJ (1994) A clinical grading scale to predict malignant hyperthermia susceptibility. Anesthesiology 80:771–779
10. Isaacs H, Badenhorst M (1993) False-negative results with muscle caffeine halothane contracture testing for malignant hyperthermia. Anesthesiology 79:5–9
11. Endo M, Yagi S, Ishizuka T, Endo M, Yagi S, Ishizuka T, Horiuti K, Koga Y, Amaha K (1983) Mechanism in the sarcoplasmic reticulum of the muscle from a patient with malignant hyperthermia. Biochem Res 4:83–92
12. Matsui K, Kikuchi H (1994) Diagnosis of malignant hyperthermia by muscle biopsy—Ca-induced Ca release. J Jpn Soc Clin Anesth 14:402–404
13. Bennett MH, Wainwright AP (1989) Acute thyroid crisis on induction of anaesthesia. Anaesthesia 44:28–30
14. Kaplan RF, Rushing E (1992) Isolated masseter spasm and increased creatine kinase without malignant hyperthermia susceptibility or other myopathies. Anesthesiology 77:820–822
15. Katz L, Wright C, Harter J, Katz L, Wright C, Harter J, Zung M, Scally D, Spyker D (1994) Revised label regarding use of succinylcholine in children and adolescents. Anesthesiology 80:243–244
16. Delphin E, Jackson D, Rothstein P (1987) Use of succinylcholine during elective pediatric anesthesia should be reevaluated. Anesth Analg 66:1190–1192