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Malignant Hyperthermia during Desflurane–Succinylcholine Anesthesia for Orthopedic Surgery

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POTENT inhalational anesthetics can trigger malignant hyperthermia (MH). Several studies[1,2] have shown that desflurane triggers MH in susceptible swine. Recently a case of tachycardia and hyperthermia associated with desflurane in humans was reported.[3] We describe an episode of MH in a patient undergoing anesthesia with desflurane.

Case Report

A 19-yr-old, 29-kg, 13and 0-cm woman with severe scoliosis and other congenital malformations (pectus carinatum, retrognatia, and so on) was scheduled for surgical correction of her spinal deviation. She described previous surgery as a child for tendon reinsertion in her right hip without complication (no reports available). No family history of previous problems involving anesthesia or surgery was known.

Anesthetic induction involved fentanyl, 50 μg, and thiopentone, 150 mg. Tracheal intubation was facilitated with succinylcholine (SCh), 45 mg. Anesthesia was maintained with 65% nitrous oxide and desflurane 6% in oxygen, along with additional fentanyl (2 μg/kg); neuromuscular blockade was achieved with atracurium in continuous infusion (6 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Monitoring consisted of electrocardiograph (ECG), invasive arterial pressure, expired CO_2 and anesthetic gases, nerve stimulator, pharyngeal temperature, and urine output.

Thirty minutes after induction, expired CO_2 increased to 40 mmHg, although hemodynamic parameters (blood pressure, 110/70 mmHg; heart rate, 80 beats/min), pharyngeal temperature (38°C), and Sp_{O_2} (100%) values remained unchanged. However, despite increased ventilatory minute volume, expired CO_2 kept increasing. An arterial blood gas, drawn 45 min after induction, showed: *p*H, 7.16; P_{CO_2}, 66 mmHg; P_{O_2} 118 mmHg; bicarbonate, 23 mEq/l; and potassium, 5 mEq/l. Approximately 90 min after induction, tachycardia (160 beats/min) and hypotension (80/45 mmHg) appeared; muscle rigidity was also observed, and expired CO_2 reached a high of 78 mmHg. MH was now suspected; pharyngeal temperature was 40.5°C. Surgery was interrupted and therapy instituted, including discontinuation of desflurane and nitrous oxide, 100% oxygen, cold intravenous fluids, cooling of skin, administration of mannitol and 60 mg of dantrolene sodium divided in two equal doses given within 40 min.

After starting treatment, all parameters progressively improved. After 75 min of treatment the patient was stable: heart rate, 90 beats/min; blood pressure, 112/75 mmHg; and temperature around 38°C. Laboratory values included *p*H, 7.33; P_{CO_2}, 37 mmHg; P_{O_2}, 443 mmHg; bicarbonate, 19.2 mEq/l; potassium, 4.1 mEq/l; and CPK, 2661. Urine output was 1250 cc in 2 h. An intercostal muscle biopsy was obtained before surgical closure; the result was congenital myopathy and central core disease. She was discharged from hospital on the 48th postoperative day.

Discussion

In two experimental studies, Wedel *et al.*[1,2] have shown the role of desflurane as a trigger of MH in susceptible swine. They conclude that desflurane is a trigger for MH and contraindicated in susceptible patients, sharing that characteristic with other inhalational anesthetics. In their second study they examined the relative ability of halothane, isoflurane, and desflurane to trigger MH, concluding that the exposure to halothane resulted in a more rapid onset of MH than either of the others. Most recently Fu *et al.*[3] reported a case of MH involving anesthesia with desflurane. SCh—another well-known trigger agent—was also administered. In their case, tachycardia was the first sign but was initially attributed to a desflurane-induced sympathetic hyperactivity.

In our patient, elevated expired CO_2 was the first evidence of difficulties. Because temperature, oxygenation, and hemodynamic values were normal, we did not immediately suspect MH and tried to improve the patient's condition by increasing ventilation, checking the

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anesthesia machine, and maintaining desflurane 1 MAC. When hemodynamic instability began and temperature increased, the diagnosis was confirmed.

This case of MH involved desflurane and SCh. The relatively slower onset may relate to the delaying effect of nondepolarizing relaxants and depressants in slowing the onset of MH.[4] Further, increased ventilation for management of increased expired CO_2 may in part mask and delay the diagnosis of MH.[5] Comprehensive monitoring and a high degree of suspicion, *i.e.*, vigilance, remain fundamental factors for a timely diagnosis.

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References

1. Wedel DJ, Iaizzo PA, Milde JH: Desflurane is a trigger of malignant hyperthermia in susceptible swine. ANESTHESIOLOGY 1991; 74:508-12
2. Wedel DJ, Gammel SA, Milde JH, Iaizzo PA: Delayed onset of malignant hyperthermia induced by isoflurane and desflurane compared with halothane in susceptible swine. ANESTHESIOLOGY 1993; 78:1138-44
3. Fu ES, Scharf JE, Mangar D, Miller WD: Malignant hyperthermia involving the administration of desflurane. Can J Anaesth 1996; 43:687-90
4. Gronert GA, Milde JH: Variations in onset of porcine malignant hyperthermia. Anesth Analg 1981; 60:499-503
5. Karan SM, et al.: Malignant hyperthermia masked by capnographic monitoring. Anesth Analg 1994; 78:590-2

Negative-pressure Pulmonary Edema Associated with Saber-sheath Trachea

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NEGATIVE pressure pulmonary edema (NPE) has been associated with the relief of upper airway obstruction in otherwise healthy patients.[1-5] The suggested pathophysiologic mechanisms have included increased venous return, reduced ventricular compliance, interventricular septal shift, and increased afterload.[2] "Saber-sheath" is a morphologic description applied to a trachea that is narrowed in the coronal plane and elongated in the sagittal plane.[6,7] It has been associated with obstructive lung disease and hypothesized to develop as a result of a type of tracheomalacia (Todd T, Winton T: Personal communication, April 1998).[7,8] This case report describes an episode of NPE associated with a saber-sheath trachea.

Case Report

A 77-yr-old man with a fractured proximal humerus presented for shoulder arthroplasty. The patient described symptoms of dyspnea on minimal exertion and was clinically diagnosed with obstructive lung disease. He had a history of congestive heart failure, a remote myocardial infarction, gastroesophageal reflux disease (GERD), and a 50 pack-yr smoking history. He had undergone laparoscopic cholecystectomy 2 yr previously, reportedly uneventfully. Reduced breath sounds in all lung fields were noted. Electrocardiogram (ECG) showed a left bundle branch block (LBBB). Chest radiograph revealed flattened hemidiaphragms.

The patient was premedicated with ranitidine and metoclopramide and taken to the operating room where the usual monitors were applied, including ECG, pulse oximeter, and noninvasive blood pressure cuff. An arterial line was placed in the nonoperative extremity. The patient was preoxygenated and induced in a rapid sequence fashion (fentanyl, 100 μg; propofol, 100 mg; and rocuronium, 50 mg), with cricoid pressure applied. Mild resistance to passage of the endotracheal tube (ETT) was encountered at 22 cm, which was overcome using slight rotation of the ETT, completing insertion to 24 cm.

The surgery was completed uneventfully, with minimal blood loss and infusion of 1.0 l of crystalloid. After return of consciousness and reversal of muscle relaxation (glycopyrrolate, 0.4 mg; pyridostigmine, 10 mg), the patient's respirations were rapid and shallow. Assessment of neuromuscular function indicated probable complete reversal (sustained tetanus for 5 s at 50 Hz), and positive pressure ventilation was resumed. Auscultation revealed coarse bilateral crackles. Chest radiograph showed bilateral infiltrates and a saber-sheath trachea (fig. 1), the latter having been overlooked on preoperative films. ECG showed LBBB in the same configuration as preoperatively. Diuresis was initiated with intravenous furosemide, 40 mg, (30 min postoperatively), and the patient was transferred to the intensive care unit. The differential diagnosis included high-pressure pulmonary edema caused by heart failure and low-pressure pulmonary edema caused by aspiration or NPE. NPE was suspected because of the sudden onset of symptoms on emergence. A pulmonary artery catheter was placed (90 min postoperatively), which showed a pulmonary artery occlusion pressure (PAOP) of 12 mmHg and pulmonary artery pressure

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