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Carpal spasm observed during and after sevoflurane anesthesia

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Introduction

Carpal spasm was observed in a patient with normal plasma calcium levels during and after sevoflurane anesthesia. We suspected that the level of calcium was decreased somehow during and after sevoflurane anesthesia.

Case report

A 63-year-old, 150-cm, 48-kg female patient was scheduled for skin plasty in the right neck. Her past medical and surgical history was unremarkable. She is now diagnosed as having slight mitral regurgitation and dilating cardiomyopathy, but is free of symptoms.

Three years ago she underwent resection of a malignant skin tumor of the right cheek and radical neck dissection of the same side. Thereafter, she underwent related facial plastic surgeries six times. Five previous surgeries were performed under enflurane anesthesia supplemented by intravenous fentanyl. On two occasions, the patient complained of stiffness and pain in the left index finger postoperatively. Before those surgeries, her serum Ca (4.2–5.4 mEq/L)/P (2.7–4.5 mg/dL) ratio were 4.5/3.2 and 4.6/4.6, respectively. Following the last surgery performed under sevoflurane anesthesia 10 months ago, she had stiffness and pain in all the fingers of both hands for a few days postoperatively, but no further complaints were known.

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Preoperative data were as follows: An ECG recording showed a left bundle-branch block (LBBB) and a significant ST-T segment change that were diagnosed as being an intraventricular disturbance secondary to cardiomyopathy. Her cardiothoracic ratio was more than 70%. Laboratory findings revealed Na 142 mEq/L, Cl 113 mEq/L, K 3.2 mEq/L, ionized Ca 4.3 mEq/L (4.2–5.4 mEq/L), total protein 6.9 g/dl, RBC 505×10^4, hematocrit 44.7%, cholinesterase activity 250 IU, blood urea nitrogen (BUN) 14.5 mg/dl, and all the other variables were within normal limits. Her blood pressure was 142/94 mmHg and heart rate was 72 bpm. The patient was taking digoxin 0.2 mg p.o./day and furosemide 5 mg p.o./day; she took food by mouth and there was no cardiorespiratory distress. There were no signs suggesting tumor metastasis to the brain, the spinal cord, or to local lymph nodes. A skull X-ray film was negative for abnormal shadows including calcification of the basal ganglia, as seen in some hypoparathyroid patients [1]. Neither Trousseau's sign nor Chvostek's sign had been noticed prior to surgery. No signs of common cold or diarrhea were known.

As a premedicant drug, she received 50 mg hydroxydione p.o. 1 h before entering the operating room. Anesthesia was induced with thiopental 4 mg/kg i.v. and her trachea was intubated following administration of succinylcholine chloride (SCC) 1.5 mg/kg i.v. The response to SCC appeared normal. Surgical anesthesia was maintained with N_2O (66%), O_2, and sevoflurane (0.8%–1.2%). About 40 min after the commencement of anesthesia, we noticed all the fingers of her hands were stretched out and rigid, a condition described as obstetrician's hand (Fig. 1). A blood pressure cuff was applied on the left upper arm. Each finger had continuously fine movements just like SCC-induced fasciculation. Rigidity was observed only distal to the wrists. The fingertips were normally warm with a full peripheral oxygen saturation (Spo_2) value. Ringer's lactated solution (500 ml) was administered intravenously. We re-

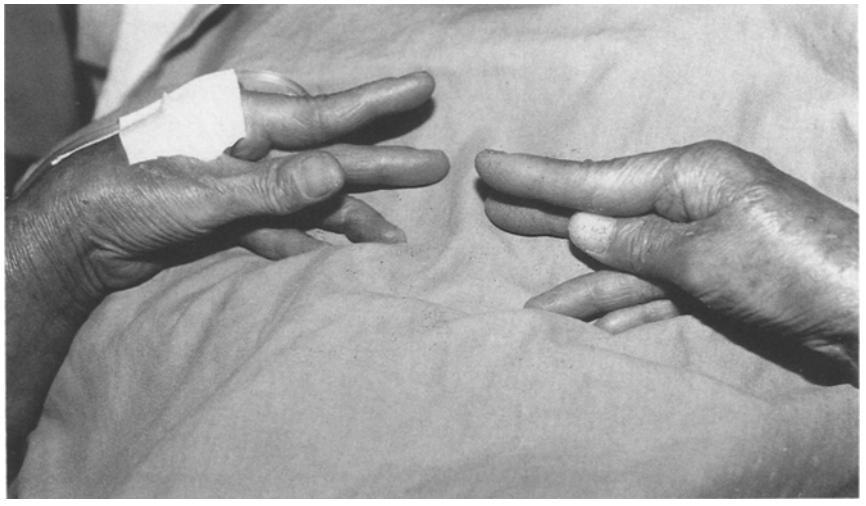

Fig. 1. Obstetrician's hand (carpal spasm). The spasm was induced by the blood pressure cuff application before the most recent sevolfurane anesthesia. The same type of spasm with continuous involuntary fine movement was observed during and after the anesthesia described in the text

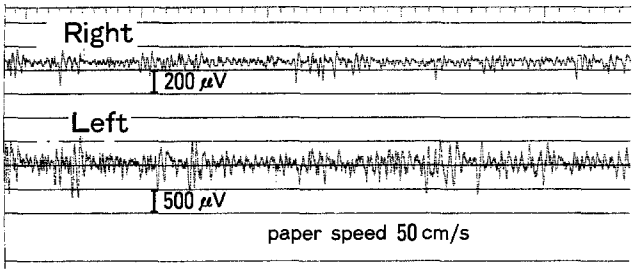

Fig. 2. Electromyographic recordings of fine involuntary movements. Electrodes were placed on the right and left index fingers and the brachioradialis muscles in each hand, but myograms were recorded with different amplitudes because of sensitivity

corded the electrical activity of the fine movements 2 h after the onset (Fig. 2). Laboratory data at this time were as follows: Na 142 mEq/L, K 3.0 mEq/L, Cl 106 mEq/L, ionized Ca 4.4 mEq/L, and blood sugar concentration 96 mg/dl. Blood gas status: pH 7.43, $Paco_2$ 41 mmHg, Pao_2 168 mmHg (Fio_2 0.33), BE 3.3 MEq/L. After 2.5 h of anesthesia, she woke up, opened her eyes spontaneously, and was reminded of her hand's stiffness in a characteristic figure with the continuous fine movements. She felt that all her fingers were stiff. The second and third fingers of the left hand in particular were too stiff to flex. She attempted to clench a fist but could not fully accomplish it because of the involuntary fine movements and rigidity. Tests for either Chvostek's sign or Trousseau's sign were not done. Both hands as a whole were too painful, 90/100 on a visual analog pain scale (VAS). She described these as largely the same as previously experienced. The pain was eased by indomethacin 25 mg p.o. to 10/100 on VAS and she fell asleep. The next morning, the pain had improved (VAS 35/100) but she could not yet fully flex her fingers. Her consciousness was totally clear and she was free from any distress. In the afternoon of the 2nd postoperative day, the pain of the fingers and hands were much less (VAS 20/100) and full finger flexion at the distal interphalangeal joints was possible although each finger persistently had the involuntary fine movements. About 56 h after the surgery, the pain rigidity and fine movements had resolved completely.

We suspected the presence of hypoparathyroidism in this patient based on the finding of a positive Trousseau's sign observed separately by our internist, but the endocrinological examinations revealed normal parathyroid function as follows: intact parathyroid hormone (PTH) 53 pg/ml, C-PTH 0.6 ng/ml, Ca^{2+} 2.50 mEq/L, total protein 7.2 g/dl, albumin 61.9%, and a urinary P : 17-OHCS mg/day : 17KS mg/day ratio on two separate occasions of 32.3/3.3/4.9, 43.4/2.7/4.1, respectively. Subtraction scintigram revealed a blurred nodule in the left upper parathyroid gland but the remainder in the hot nodule [1]. Recently, about 2 years after the last surgery reported above, she underwent a short surgical revision of the face under sevoflurane anesthesia. A positive Trousseau's signs was induced by a blood pressure cuff but disappeared soon in both hands, before and after anesthesia administration, but a persistent Trousseau's sign was not observed during anesthesia. The Ca ion concentration was 4.1 mEq/L, the lower limit of normal, before and during anesthesia.

Discussion

The carpal spasms observed in this patient appeared to be related to factors common among the status newly developed either by blood pressure cuff application or by sevoflurane or enflurane anesthesia. Hypocalcemia is most strongly suspected. Hypocalcemia is sometimes observed in fluoride poisoning in which the fluoride ions absorbed combine with the serum calcium ions [2,3].

Her Ca ion concentration might have been slightly decreased by combining with fluoride ions, a sevoflurane metabolite. The involuntary fine movements were considered to be myoclonus, which is one of the symptoms associated with hypocalcemia. At the sevoflurane concentration used in this patient, the fluoride level was not higher than during enflurane and certainly persistence of the fluoride plasma level is not longer than after enflurane [4–6]. This documented evidence supports hypocalcemia as the etiology. However, methoxyflurane, which produced much higher fluoride levels than sevoflurane, was never suspected of such an event [7]. This rare phenomenon has not been observed during sevoflurane anesthesia before.

The fluoride ion determination during and after sevoflurane anesthesia revealed that inorganic fluoride ion remained at a significant concentration over a few days postoperatively [4]. The distribution volume of fluoride is 0.5–0.7 L/kg, and its plasma half-life is 2–9 h [5,8]. Computations using 9 h as the maximum half-life indicates that fluorides are excreted almost totally from the blood after 54 h (1/64 left) after ingestion or absorption. This time course explains why the carpal spasms lasted for 56 h in the present case. The fluoride ion is usually thought to be free in the plasma and the bound fraction is thought to be negligible [8]. Fluoride ions combine also with the bone cells over a few hours after absorption and are gradually released into the blood [9]. This phenomenon seems to be observed by 50% fraction in infants and 10% in adults [10]. This may partly explain why the symptoms of hypocalcemia continued for a few days after enflurane and sevoflurane anesthesia in this patient [11].

Normal serum fluoride concentration is 0.052–10.53 μM/L [12], the serum level of a patient who did not need any toxicological treatment and survived was 178.9 μM/L [13] and that of a surviving patient who needed an intensive treatment was 773.6 μM/L [14]. Serum Ca levels in these situations were not known, but clinical symptoms at these fluoride levels were an increase in skeletal muscle excitability, hyperactive reflexes, painful spasms, weakness, and tetanic contractures [13,14]. Paresis of the affected muscle groups, especially those of the eyes, face, hands and lower extremities, dysphagia, and uncoordinated eye movements may also occur [14].

We cannot rule out the effect of sevoflurane on the central nervous system although the postoperative time course appeared too long for such an effect. Also, we cannot rule out the effect of hypocapnea probably caused by painful stress-induced hyperventilation on the serum calcium level and cerebral blood flow. Abnormal movements have often been observed during anesthesia [15,16]. This case may suggest that sevoflurane inhibits the inhibitory pathway of the central nervous system. Radical neck surgery that causes nerve damage remains to be elucidated.

In conclusion, the stiffness and pain of the fingers following sevoflurane anesthesia in this patient may be explained by sevoflurane metabolite, inorganic fluoride itself, and/or the effect on serum calcium concentration.

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