Anesthesia for a Patient with Total Lipodystrophy

— A Clinical Report —

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Total lipodystrophy is a rare disease characterized by complete loss of subcutaneous fat tissue, hyperlipemia and hepatomegaly with fatty infiltration[1, 2]. Although changes in uptake and elimination of anesthetic agents are suspected to occur because of diminished fatty mass, no report is available concerning this problem. We experienced a case anesthetized with enflurane for tonsillectomy, showing a slower elimination of the anesthetics.

Report of a Case

A 3 year- 3 months- old girl weighing 21 kg showed hyperlipemia and skin pigmentation (Fig. 1). She was referred to our institution for further examinations in February, 1985. She showed marked hepatomegaly, hyperlipemia (total cholesterol 254 mg/dl, triglycerides 415 mg/dl, phospholipid 258 mg/dl) and moderate liver damage (GOT 77 IU, GPT 140 IU). The Hb, WBC count, blood urea nitrogen, creatinine, serum electrolytes and urinalysis were within normal ranges. The ECG and chest X-ray were also within normal limits. Abdominal CT-scanning revealed diffuse fatty liver and hepatomegaly. Accordingly she was diagnosed as total lipodystrophy. She was scheduled for bilateral tonsillectomy under general anesthesia because of recurrrent tonsilitis.

The patient was premedicated with atropine (0.2 mg i.m.) 1 hour before anesthesia. Anesthesia was induced using a face mask with a slow induction technique. Orotracheal intubation was performed without neuromuscular blocking agents. Anesthesia was maintained with 2.0% enflurane and 66% nitrous oxide in oxygen. During anesthesia adequate pulmonary ventilation was manually controlled. A significant fall of systolic blood pressure appeared immediately after induction of anesthesia. Although the systolic blood pressure decreased from 130 to 60 mmHg and the heart rate increased from 90 to 120 beats/min, anesthesia by 2% enflurane was continued because of good pulse tension and no signs of either poor peripheral circulation nor acid-base imbalance. Remarkable subcutaneous vaso-dilation was observed in extremitas of the patient. During surgery her systolic blood pressure ranged from 50 to 70 mmHg and heart rate was from 120 to 140 beats/min. ECG displayed no signs of arrhythmia throughout the anesthetic course. Blood gas analysis showed Pao_2; 133.5 mmHg, $Paco_2$; 41.4 mmHg, pH; 7.358 and BE; -2.2 mEq/l at 20 min after the starting of enflurane anesthesia. Upon completion of the operation, enflurane was discontinued and systolic blood pressure spontaneously recovered to the preanesthetic level. Twenty min after discontinuing the anesthetic agents, she was fully awake and was breathing sufficient enough for extubation. Under room air breathing, blood gas values 40 min after anesthesia were 80 mmHg

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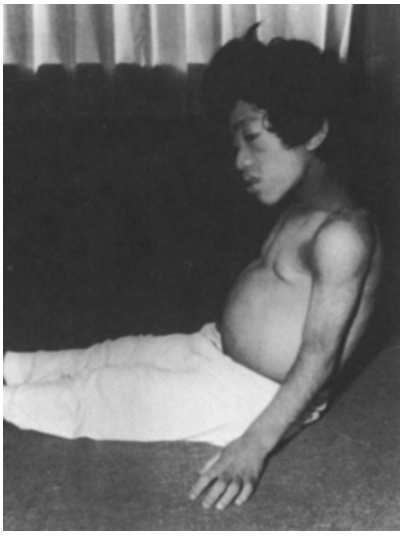

Fig. 1. A lipodystrophy patient

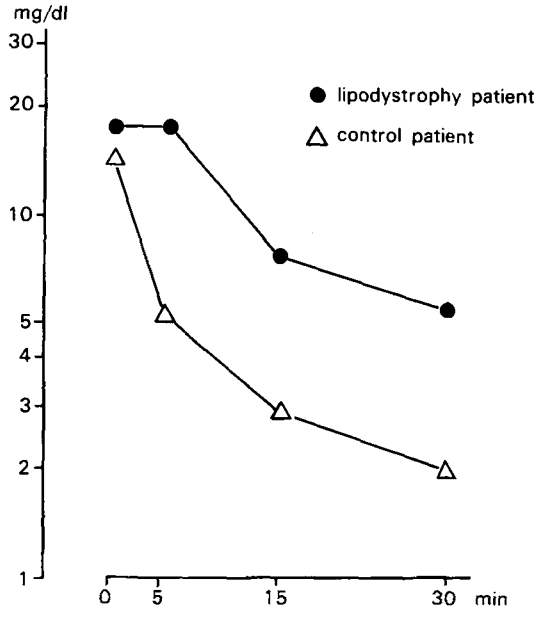

Fig. 2. Comparison of blood concentration of enflurane between lipodystrophy patient and the control patient
Horizontal axis represents time (min) after discontinuing anesthetics. Vertical axis shows blood concentration of enflurane in a logarithmic scale.

in Pao_2, 48 mmHg in $Paco_2$, 7.306 in pH and -3.0 mEq/l in BE. Post-operative complications did not occur and 10 days after surgery, GOT and GPT took 68 and 115 IU, respectively. We also checked 1.5 ml of arterial blood samples for blood concentrations of enflurane by the gas chromatography technique. Blood concentrations were measured four times: just after, 5, 15 and 30 min after discontinuing enflurane anesthesia (fig. 2). A two year-old boy weighing 15 .kg without any systemic disorder who underwent plastic surgery on the same day was selected as the control case. He was subjected to the same anesthesia method and his blood samples were also taken at corresponding intervals. There was no change in induction time between lipodystrophy patient and the control patient, suggesting lipodystrophy may not affect the distribution of enflurane. But recovery from anesthesia was sooner and smoother in the control case than that of the lipodystrophy patient. Although the blood concentrations of enflurane just after anesthesia were similar in the two patients, the elimination curve for the lipodystrophy patient shifted to the right compared with that of the control patient (fig. 2). Even 30 min after discontinuance of enflurane, blood concentration of the lipodystrophy patient (5.6 mg/dl) was still higher than that of the control patient (2.0 mg/dl). These data suggest that lipodsystrophy can significantly influence the elimination stage of enflurane anesthesia.

Discussion

Considering that the patient had moderate liver damage with marked fatty infiltration and the use of epinephrine during surgery, enflurane was selected for this case. Enflurane has a lower potential for hepatotoxicity[3] and is less sensitizing of myocardium to the epinephrine than halothane[4]. The accumulation of lipid in the liver might lead to the accrual of supranormal hepatic concentrations of lipid-soluble anesthetics and may cause hepatocellular injury. The liver function did not deteriorate after surgery. This may be attributable to lower lipid solubility and shorter elimination half life of enflurane compared with halothane. In this case, blood concentration level more than 5 min after discontinuance of enflurane was always higher than that of the control case inspite of shorter

anesthesia duration (85 min) than that of the control case (150 min). This is considered to be due to accelerated enflurane uptake and prolonged elimination caused by both increased blood/gas and fat/blood partition coefficients by hyperlipemia. At 30 min after discontinuing enflurane, $Paco_2$ was 48 mmHg in this patient and 31 mmHg in the control case, which might indicate decreased alveolar ventilation by a prolongation of enflurane elimination.

Sustained hypotension (70–50 mmHg) during anesthesia did not imply peripheral hypoxia since blood gas values were in the normal ranges and pulse tension was maintained well. The relatively developed peripheral vessels may have been dilated by anesthetic agents. Therefore, severe hypotension may be induced by both anesthesia and dehydration in total lipodystrophy patient. Thus, generarized loss of subcutaneous fat tissue may affect not only peripheral vessel tone but also the elimination of soluble anesthetics. In this respect the use of enflurane can be recommended to avoid prolongation of recovery from anesthesia at the present time.

We performed enflurane anesthesia for a patient with total lipodystrophy. Right shift of enflurane elimination curve and slight prolongation of awakening time was present. There was no clinical problem during and after anesthesia except sustained hypotension, which may challenge an anesthesiologist unless he is experienced in maintaining the peripheral circulation of the patient.

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