

Clinical report

Simple deep hypothermia for repair of congenital tracheal stenosis: a case report

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Introduction

Congenital tracheal stenosis (CTS) is a rare disease that is frequently accompanied by cardiovascular malformations such as pulmonary artery sling (PA sling). It is difficult to make the diagnosis soon after birth, and death may occur before surgery. Anesthetic management for surgical repair is technically difficult, and the prognosis is particularly poor when the stenotic lesion is extensive or close to the tracheal carina [1–4]. The present report describes tracheal repair of CTS using a costal cartilage graft under simple deep hypothermia.

Case report

A 6-month-old boy, 58 cm in height and 4.1 kg in weight, was admitted to our hospital because of stridor and dyspnea. He was born in the 37th gestational week with a birth weight of 2088 g, and was diagnosed postnatally as having anorectal stenosis, idiopathic intestinal perforation, and trisomy 13. Partial intestinal resection and proctostomy were performed immediately after birth. He suffered from tachypnea, stridor, and tracheal tug after the surgery. At 5 months of age, these tracheal symptoms deteriorated, and emergency cardioangiography confirmed the diagnosis of PA sling, for which left pulmonary artery grafting was performed. Artificial

ventilation was continued for one month after the surgery, with no symptomatic improvement. At 6 months of age, CTS was diagnosed, based on computed tomography, bronchoscopy and esophagography (Fig. 1). Surgery for tracheal repair under simple deep hypothermia was scheduled.

The patient was premedicated with 0.05 mg of atropine sulfate, 2.5 mg of hydroxyzine hydrochloride, and 5 mg of pethidine intramuscularly 30 min before the induction of anesthesia.

Two hundred micrograms of fentanyl were administered i.v. at the beginning of the surgery, and then a piece of costal cartilage was extracted for tracheal repair. After a further 300 µg of fentanyl (for a total of 700 µg) and 150 mg of methyl prednisolone had been administered i.v., the patient was covered with a vinyl sheet and immersed in iced water for surface cooling. During the cooling period, 55 ml of 10% low-molecular-weight-dextran in lactated Ringer's solution (LMWD-L), 55 ml of 5% dextrose, and lidocaine at the high dose of 40 mg·h⁻¹ (10 mg·kg⁻¹·h⁻¹) were infused continuously. Dobutamine hydrochloride (DOB) at 5 to 15 µg·kg⁻¹·min⁻¹ was also given continuously to maintain an optimal arterial blood pressure during anesthesia. When the esophageal temperature had fallen to 30°C, 500 units of heparin were administered i.v. When severe hypotension was observed, 1 ml of cardiotoxic cocktail (10 ml of 20% dextrose, 10 ml of 2% CaCl₂, and 1 ml of 0.1% norepinephrine in 21 ml solution) was administered i.v. When the target esophageal temperature of 20°C had been achieved, iced water was removed and surgery was started. The esophageal temperature fell naturally to 19.6°C.

After median sternotomy, the venae cavae, the pulmonary artery, and the aorta were clamped. Ten milliliters of Young's solution (5 g of potassium citrate, 12.3 g of magnesium sulfate, and 5 mg of neostigmine bromide in 500 ml solution) was injected into the coronary artery through the aorta to induce cardiac standstill. Tracheal

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repair was then completed using the costal cartilage as a graft.

After the tracheal repair, the patient underwent intubation with a specially designed long nasotracheal tube (26 cm length, 4 mm ID; Portex, Kent, England) just above the carina (13 cm from the nares), beyond the stenotic region. To confirm the position of the endotracheal tube in postoperative management, a titanium marker was attached to the distal end of the costal cartilage patch. Cardiac resuscitation was then performed at 19.9°C esophageal temperature, as follows. Artificial ventilation was started and the clamps were removed from all large vessels, except the aorta. Ten milliliters of the cardiotoxic cocktail and autologous blood were pumped with a syringe several times into the

coronary artery through the aorta. Because of bradycardia and hypotension, cardiac massage was then performed for about 10 min. When the heart started to beat, the aortic clamp was removed. The duration of aortic clamping was 73 min. The patient was surface-warmed to 36°C esophageal temperature with warm water at 42°C.

The acid-base balance was corrected with 20 ml of 7% sodium bicarbonate solution after cardiac resuscitation. During rewarming, lactated Ringer's solution at 20 ml·h⁻¹ and stored whole blood were infused to maintain the level of CVP at 10 mm Hg. DOB was administered at 5 to 15 µg·kg⁻¹·min⁻¹ to maintain an optimal arterial blood pressure for the body temperature. At an esophageal temperature of 30°C, 5 mg of protamine sulfate was administered to reverse the effect of heparin, and 15 mg of furosemide was administered to induce urination at the end of rewarming. After reaching an esophageal temperature of 36°C, the patient was transferred to the intensive care unit, where mechanical ventilation was performed. Although an artificial heart-lung machine had been on stand-by in case of urgent events such as difficulty in cardiac resuscitation or circulatory arrest for over 75 min, it was not necessary to use it.

The anesthesia record and laboratory data during surgery are shown in Fig. 2 and Table 1. Fentanyl decreased heart rate but slightly increased arterial blood pressure at induction of anesthesia. Both arterial blood pressure and heart rate decreased gradually with decreased body temperature and recovered to baseline levels after rewarming to 36°C (Table 1). ECG revealed no risky changes such as ventricular arrhythmia or abnormal ST segment during the entire course of anesthesia.

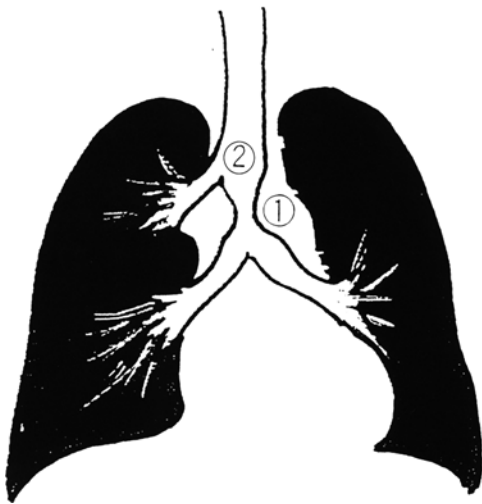


Fig. 1. Congenital tracheal stenosis. ① Stenotic lesion; ② tracheal bronchus to right upper lobe

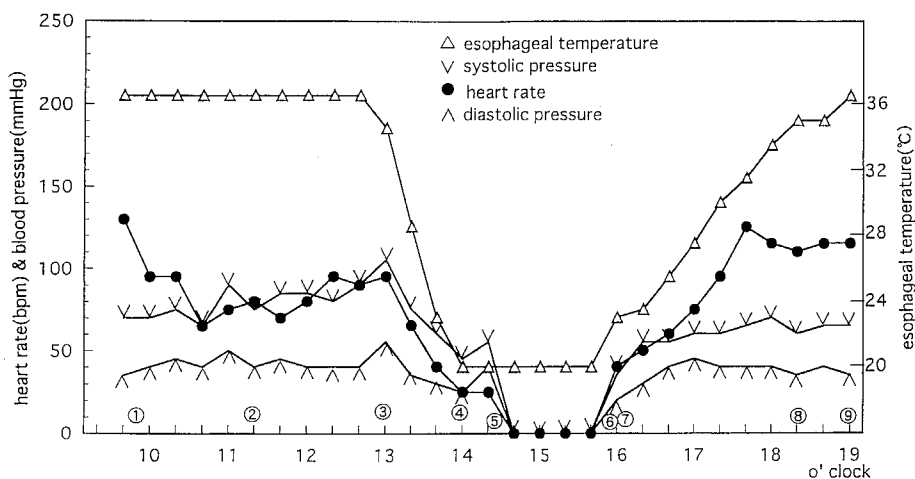


Fig. 2. Anesthesia record. ① Induction of anesthesia; ② beginning of surgery; ③ beginning of cooling; ④ end of cooling; ⑤ induced cardiac arrest; ⑥ release of aortic clamp; ⑦ beginning of rewarming; ⑧ end of rewarming; ⑨ end of surgery

Table 1. Laboratory data during anesthesia

Time	BT (°C)	Ph	PaCO ₂ (mmHg)	PaO ₂	BE (mEq·l ⁻¹)	Na ⁺	K ⁺	Ca ⁺⁺ (mmol·l ⁻¹)	BS (mg·dl ⁻¹)
9:35	36.5								
10:20	36.5	7.22	61	215	-3.1	129	4.8	1.00	80
12:30	37.0	7.37	39	353	-1.9	127	4.9	0.84	
13:15	29.0	7.47	30	544	-0.8	128	3.5	0.88	89
14:05	19.6	7.59	20	690	-0.4	125	3.5	0.78	93
16:12	24.4	7.15	30	455	-17.2	119	6.1	2.49	530
17:25	30.0	7.38	29	406	-6.8	130	3.7	1.26	253
19:00	36.0	7.44	36	458	1.4	132	4.8	1.01	113

BE, Base excess; BT, esophageal temperature; BS, blood sugar.

Blood gas was analyzed at 37°C (alpha-stat).

The heart was arrested for 73 min (14:35 to 15:48).

The fluid balance during the 10h of anesthesia was as follows. The measured blood loss and urine volume were 31g and 120ml (2.9ml·kg⁻¹·h⁻¹), respectively. Fifty-five milliliters of LMWD-L, 10ml of lactated Ringer's solution, 55ml of 5% dextrose, and 130ml of stored whole blood were infused. The platelet count was 290 × 10³μl⁻¹ before surgery and 250 × 10³μl⁻¹ after surgery. The patient was conscious on the following morning and reacted well to sound and light. No neurological abnormalities, such as convulsions, were observed.

The postoperative management plan was to maintain mechanical ventilation until the cartilage graft was assimilated in the trachea and smooth respiration had become possible. Although there was no ventilation problem during the early postoperative period, the tip of the endotracheal tube drifted easily with body movement, and it was difficult to stabilize at the marked position. Obstruction of the airway due to proliferation of granulation tissue in the trachea was noticed several days after surgery. Tracheal dilatation was performed with a balloon on the 24th postoperative day, without any satisfactory result. Artificial ventilation remained difficult afterwards and multiple organ failure supervened. The patient died of hemorrhage from the granulation tissue in the trachea on the 61st postoperative day.

Discussion

CTS is a life-threatening disease, particularly if it involves a long segment of the trachea. If the lesion involves or is close to the carina, as in this case, perioperative management is technically more difficult. Although many institutions use extracorporeal support, such as cardio-pulmonary bypass (CPB) [5,6], or extra-

corporeal membrane oxygenation (ECMO) [7] for tracheal repair, these procedures give rise to many problems. CPB canulae may crowd the operative field in such a small child as our patient. V-A ECMO can easily be accomplished through a cervical incision, and this approach for intraoperative support is attractive. Another benefit of ECMO is the option to provide postoperative extracorporeal support, which will spare the repaired portion from the stress of mechanical ventilation and suction during the early postoperative period. Although ECMO requires less heparin than CPB, the risk of bleeding is present in both types of extracorporeal support. Normal or high-frequency jet ventilation is one method of respiratory management during the surgery. However, this application is unusual, because it is lacking in reliability in ventilatory function, and blood is dispersed in different directions by the jet stream. The operating field can be narrowed by the endotracheal tube, and there is a risk of barotrauma to the lung.

We used simple deep hypothermia to reduce these difficulties, as described above. The platelet count was almost unchanged during the surgery, and platelet transfusion was unnecessary. Although cardiac massage was necessary, cardiac resuscitation was easily accomplished. The period of cardiac arrest slightly exceeded the time limit (60 min at 20°C [8]), but emergence from the anesthesia was smooth and no signs of cerebral damage were observed. Respiratory and hemodynamic functions were stable during the surgery. These findings suggest that simple deep hypothermia similar to CPB or ECMO can be recommended as management for repair of CTS.

A large dose of lidocaine (10mg·kg⁻¹·h⁻¹) was infused continuously during the cooling phase to prevent serious ventricular arrhythmia due to hypothermia. Although we have infused a similar large dose of lidocaine during the cooling phase in all cases of simple deep

hypothermia under a large dose of fentanyl anesthesia, we have never experienced any problems due to lidocaine intoxication such as convulsions, circulatory dysfunction, or delayed awakening [9]. The total dose of lidocaine administered reached $15\text{ mg}\cdot\text{kg}^{-1}$ for 90 min. We were not concerned about lidocaine intoxication, because a large dose of lidocaine (approximately $5\text{--}10\text{ mg}\cdot\text{kg}^{-1}$ or more i.v. by rapid infusion) is recommended for ventricular fibrillation in the pediatric patient [10]. Lidocaine was also expected to have a protective effect for the ischemic brain during circulatory arrest [11].

Postoperative respiratory management required special attention for this case. The right upper lobe was not ventilated during the perioperative period, because the trachea was intubated over the tracheal bronchus. Although ventilation of the lobe might be possible by using a special endotracheal tube sideholed for the abnormal bronchus, it was not carried out in the operating room. The main reason was the difficulty of adjusting the sidehole of the tube to the position of the tracheal bronchus, as well as of placing the tip of the tube just above the carina. Moreover, even if such a sideholed tube could be obtained, it might be very difficult to put it in the proper position. We decided not to ventilate the lobe, expecting that it would recover even after prolonged atelectasis [12,13].

In this case, stabilization of the endotracheal tube at a suitable position and maintenance of complete rest were very difficult because of tracheal narrowing by granulation formation and bleeding after the operation. Though there is a report in which the operation was performed without oxygenation support such as ECMO, and respiration could be managed by an endotracheal tube alone [14], ECMO may be indicated when the endotracheal tube cannot provide a proper airway because of a long stenotic lesion close to the carina, as in this case. In such a situation, simple deep hypothermia may facilitate intraoperative management by preventing hemorrhage due to thrombocytopenia, and ECMO may be useful for postoperative respiratory management.

In conclusion, simple deep hypothermia can be used for the surgical repair of CTS in babies with difficulties in anesthesia.

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