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Clinical reports

Myocardial ischemia due to paradoxical air embolism during living-related donor liver transplantation in a child

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Introduction

Paradoxical air embolism is life-threatening, as it can cause cerebral or myocardial ischemia [1]. In orthotopic liver transplantation, paradoxical air embolism has been reported to occur at the time of donor liver perfusion [2] or during the dissection of the recipient liver [3]. A fatal case has recently been reported [4]. We present two episodes of sudden elevation of the ST segment on the electrocardiography (ECG) during liver dissection from the inferior vena cava (IVC) in a child who was the recipient of living-related donor liver transplantation (LRDLT). Paradoxical air embolism was strongly suspected as the cause of the myocardial ischemia, because the two episodes occurred following clinical manifestations of pulmonary air embolism.

Case report

A 4-year-old girl (height 85 cm, weight 11.2 kg) was scheduled for LRDLT. Biliary atresia was diagnosed 38 days after her birth, and she underwent hepatoportojejunostomy (the Kasai procedure) at the age of 47 days, but jaundice persisted after surgery (total bilirubin $>20\,mg\cdot dl^{-1}$). She developed bone fractures in both legs, the left clavicle, and the left upper arm at 3 years of age. No other complications besides growth retardation were observed, nor were any cardiac abnormalities found by echocardiography.

On the day of surgery, diazepam (2 mg) was administered IV as premedication. Anesthesia was induced by propofol (22 mg) and fentanyl (33 μg) IV. The trachea was intubated following 2.2 mg of vecuronium IV and the lungs were artificially ventilated with an inspired oxygen concentration of 30% with 2 cm H_2O of positive end-expiratory pressure (PEEP). Anesthesia was maintained by continuous infusion of propofol ($10\,mg\cdot kg^{-1}\cdot h^{-1}$) and fentanyl ($5\,\mu g\cdot kg^{-1}\cdot h^{-1}$). No inhaled anesthetics were used. Muscle relaxation was obtained with continuous infusion of vecuronium ($0.1\,mg\cdot kg^{-1}\cdot h^{-1}$). Monitoring included electrocardiography (ECG) (lead II) and direct measurement of radial artery pressure (BP), central venous pressure (CVP), pulse oximetric oxygen saturation (SpO_2), and end-tidal pressure of CO_2 ($PETCO_2$).

Two hours and 35 minutes after skin incision, we noticed a 0.3 mV elevation of the ST segment on the ECG (Fig. 1A). $PETCO_2$ decreased from 29 to 17 mmHg, SpO_2 from 100% to 94%, and BP from 108/68 to 74/48 mmHg, while the heart rate increased from 69 to 106 beats per minute (bpm) and CVP from 1 to 7 mmHg (Fig. 2A). We suspected pulmonary air embolism and increased the inspired oxygen concentration to 100%. The surgeon suggested the possibility of air aspiration from the left hepatic vein. Five minutes after the elevation of the ST segment, all measurements returned to the basal level.

About 50 min after the first episode of ST elevation, $PETCO_2$ suddenly decreased again from 26 to 16 mmHg, SpO_2 from 100% to 98%, BP from 95/60 to 58/38 mmHg, and heart rate from 109 to 56 bpm, while the ST segment was elevated by 1.2 mV and CVP increased from 2 to 11 mmHg during dissection of the short hepatic vein following division of the portal vein. (Figs. 1B and 2B). We increased the inspired oxygen concentration to 100% and manually ventilated the lungs. A few minutes later, the surgeon noticed air aspiration from the IVC and completely clamped it for repair. Fifteen

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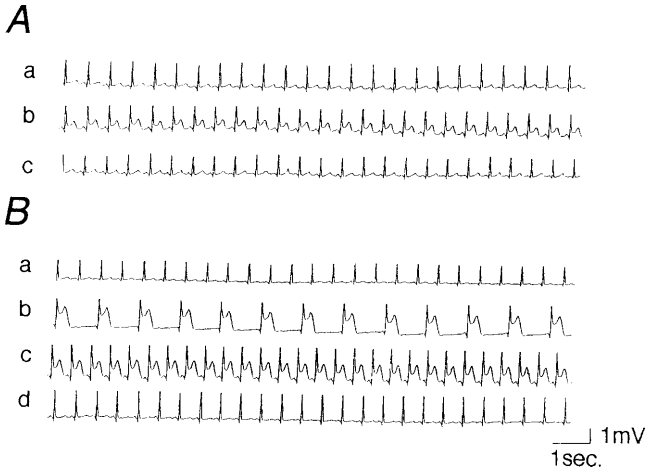

Fig. 1. Electrocardiogram (lead II) during operation. **A** First episode of air embolism. **a** 2 h 36 min, **b** 2 h 39 min, **c** 2 h 45 min after skin incision. **B** Second episode of air embolism. **a** 3 h 24 min, **b** 3 h 30 min, **c** 3 h 35 min, **d** 3 h 46 min after skin incision

minutes later, BP, the ST segment of the ECG, and SpO_2 recovered. The IVC was then declamped. P_{ETCO_2} remained low and recovered to 27 mmHg 40 min after the reperfusion of the portal vein. The changes in respiratory condition and arterial gas tension are shown in Table 1. The total anesthesia time was 12 h, the time of surgery was 11 h, and the total blood loss was 1870 g. The postoperative course was uneventful, and no neurological dysfunction was observed. The patient was discharged from our hospital 42 days after surgery with normal liver function.

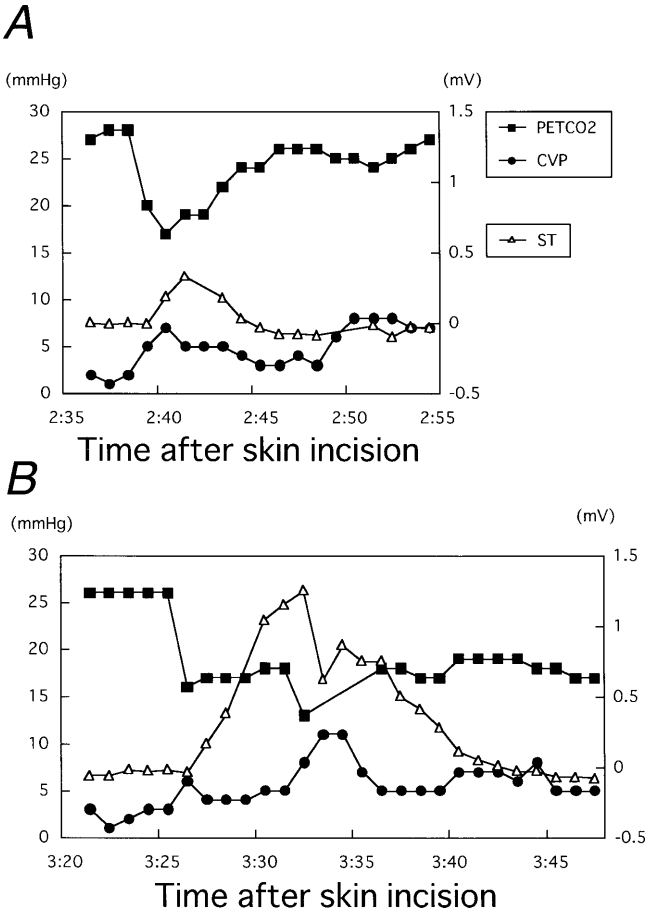

Fig. 2. Changes in end-tidal CO_2 (P_{ETCO_2}), central venous pressure (CVP), and ST segment on the electrocardiogram at the time of first (**A**) and second (**B**) episodes of air embolism. Abscissa shows elapsed time after skin incision

Discussion

There are some differences between the surgical procedures for cadaveric total liver transplantation (CTLT) and those for LRDLT. For CTLT, the recipient liver is usually removed together with the IVC, whereas in LRDLT the recipient IVC is preserved [5]. Therefore, there might be a greater chance of air entry into the venous system in the dissection phase of LRDLT than in that of CTLT.

In our case, venous air embolism is suspected to have occurred at the time of a sudden decrease in P_{ETCO_2}. After this episode, the difference between P_{ETCO_2} and arterial carbon dioxide tension ($Pa - ETCO_2$) increased to about 10 mmHg (Table 1), suggesting a decrease in pulmonary blood flow. When P_{ETCO_2} decreased, CVP increased by 6–9 mmHg before ST elevation (Fig. 2). This increase in CVP may have been caused by pulmonary artery air emboli and thus may have induced right-to-left shunt of air (paradoxical air embolism). We suspect that myocardial ischemia caused by paradoxical air embolism in the coronary artery resulted in ST elevation on the ECG. Patent foramen ovale is the most likely route of the right-to-left shunt, because the incidence of patent foramen ovale is reported to be about 34% for the age of our patient [6]. The possible existence of a paradoxical air embolism via a noncardiac shunt has also been suggested [7]. Echocardiography of this patient before surgery showed no right-to-left shunt, but it has been reported that a technique to cause right atrial pressure elevation, such as the Valsalva maneuver, is necessary to detect right-to-left shunt via the foramen ovale [8].

Coronary vasospasm should be also considered as the cause of the sudden ST elevation during surgery. However, we considered that coronary vasospasm was unlikely in our patient, because ST elevation spontaneously disappeared 5 min after the first and 15 min after the second episode without any specific treatment, such as nitrate administration. Such a reversible ST elevation is compatible with coronary air embolism reported during cardiac surgery [9], cardiac catheteriza-

Table 1. Changes in respiratory condition and arterial gas tension

Time after skin incision	Preanhepatic 1 h 27 min	Anhepatic 2 h 55 min	Anhepatic 3 h 42 min	Anhepatic 4 h 37 min	Postanhepatic 5 h 49 min	Postanhepatic 6 h 37 min	Postanhepatic 7 h 35 min
TV (ml)	110	110	100	100	100	100	100
RR	20	20	20	20	20	20	20
FiO_2	0.3	0.5	1	0.4	0.4	0.3	0.3
PaO_2 (mmHg)	164.9	274.4	625	235.5	243.5	105.9	221.3
$PaCO_2$ (mmHg)	34.5	37.6	28.9	20.8	27.7	37	35.7
P_{ETCO_2} (mmHg)	30	27	17	16	21	27	27
$Pa - ETCO_2$ (mmHg)	4.5	10.6	11.9	4.8	6.7	10	8.7

TV, Tidal volume; RR, respiratory rate.

tion [10], or central venous cannulation in a patient with a right-to-left shunt [11]. Furthermore, our patient was very young and had no history of ischemic heart disease.

After the second episode of ST elevation, P_{ETCO_2} did not immediately return to its basal level. It recovered 40 min after the reperfusion of the graft liver through the portal vein. Such a decrease in P_{ETCO_2} might be caused not only by the increase in $Pa - ETCO_2$ due to pulmonary air embolism but also by low $PaCO_2$ during the anhepatic period (Table 1). After reperfusion, both $PaCO_2$ and P_{ETCO_2} gradually increased (Table 1), as reported previously [12].

Serious events, including cardiac arrest and severe neurological deficit following paradoxical air embolism, have been reported in patients in the sitting position during neurosurgical operations [1]. In contrast, our patient fortunately developed no neurological or other complications after surgery. Compared with neurosurgical operations, the ease of clamping or repair of the injured vein in hepatic surgery seemed to be beneficial. In addition, the absence of the use of nitrous oxide should have prevented the increase in the volume of air emboli. For the treatment of venous air embolism, PEEP is advocated in an effort to increase CVP; however, PEEP promotes paradoxical embolism. In this case, we maintained 2 cm H_2O of PEEP as a routine procedure. It is not certain whether this small PEEP promoted paradoxical air embolism.

In summary, two episodes of sudden marked ST elevation occurred during LRDLT in a 4-year-old girl. Myocardial ischemia due to paradoxical air embolism in the coronary artery was strongly suspected as the cause. During LRDLT surgery, air embolism should be carefully monitored by measurement of P_{ETCO_2}, especially during the IVC dissection from the liver.

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