# Anesthesia for Acute Pulmonary Embolism

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Administration of prostaglandin $E_1$ ($PGE_1$) for pulmonary hypertension shows inconsistent and unpredictable results. This report describes an emergency operation to remove large right atrial thrombi accompanied by pulmonary hypertension, which had caused right ventricular failure. The continuous administration of $PGE_1$ together with dopamine resulted in improved arterial blood gas data and cardiac output during and after operation. Intraoperative esophageal echocardiogram was found useful for detection of the clot moving out from the right atrium.

## Case report

A 46-year-old woman (weight: 60 kg; height: 161 cm) who had undergone resection of a brain tumor (glioblastoma III) at another hospital suddenly complained of dyspnea and coughing after an uneventful postoperative 2-week period. A chest X-ray demonstrated cardiomegaly (CTR 62%). Although dyspnea disappeared two days later, several pieces of an abnormal mass (myxoma, metastatic tumor or thrombosis suspected), pressure- and volume-loading in the right atrium and ventricle, and pulmonary hypertension were implied on the echocardiogram. Therefore, the patient was transferred to Sakurabashi-Watanabe Hospital for emergency removal of the intracardiac abnormal mass.

On admission, she was conscious and slightly dyspneic, and showed tachycardia (118 bpm) with normal arterial pressure (120/80 torr). Arterial blood gas analysis revealed pH 7.335, $Pa_{O_2}$ 41.0 torr, $Pa_{CO_2}$ 48.9 torr and a base excess of (BE) −0.5 $mEq \cdot l^{-1}$ at $F_{I_{O_2}}$ 0.21. Echo- and doppler-cardiography showed two or three pieces of an abnormal mass (2–3 cm diameter) in the right atrium. Sometimes these fell into the tricuspid valve, causing tricuspid regurgitation and severe pulmonary hypertension (systolic: 70–80 torr).

After induction with 0.3 mg fentanyl and 10 mg diazepam, the trachea was intubated. An initial 6 mg dose of pancuronium was used for muscle relaxation. Arterial blood gas analysis 15 min after induction showed: pH 7.387, $Pa_{O_2}$ 150.2 torr, $Pa_{CO_2}$ 44.5 torr and BE +1.1 $mEq \cdot l^{-1}$ ($F_{I_{O_2}}$ = 1.0). Cardiac output simultaneously measured by dye dilution was 1.4 $l \cdot min^{-1}$. Thermodilution with a Swan-Ganz catheter was avoided because of the risk of tearing off the intra-atrial mass. The transesophageal probe for the echocardiogram (Toshiba Model ESB50FS) was inserted and showed several pieces of a big mass in the right atrium. Ventilation and $F_{I_{O_2}}$ remained unchanged throughout anesthesia. Thirty min after the start of intravenous infusion of 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ dopamine and 50 $ng \cdot kg^{-1} \cdot min^{-1}$ $PGE_1$, arterial blood gas data improved to pH 7.431,

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$Pa_{O_2}$ 269.7, $Pa_{CO_2}$ 41.7, BE +2.9, while cardiac output was 2.3 $l \cdot min^{-1}$.

After median sternotomy, pericardiotomy showed a yellowish effusion, enlargement of the right atrium and ventricle with reduced wall motion, but normal wall motion of the left ventricle. During taping of the superior vena cava, systolic arterial pressure suddenly dropped to 30–50 torr, although no foreign body in the right atrium was detected on the transesophageal echocardiogram. Since cardiac massage was not effective, the aorta and the appendage of the right atrium were immediately cannulated, followed by a cardiopulmonary bypass into which methyl-predonisolone (30 $mg \cdot kg^{-1}$) was administered. After the superior and inferior vena cava had been cannulated instead of the right atrium, the right atrium was opened and several thrombi were found on the wall, while the large foreign bodies previously detected on the echocardiogram had disappeared. The stem of the pulmonary artery was longitudinally incised and massive thromboemboli weighing approximately 40 grams were removed. Peripheral thrombi were also removed through the incised portion of the right main pulmonary artery. After weaning from cardiopulmonary bypass with the aid of 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ dopamine and a 25–50 $ng \cdot kg^{-1} \cdot min^{-1}$ infusion of $PGE_1$, arterial blood gas analysis results were: pH 7.430, $Pa_{O_2}$ 336.0, $Pa_{CO_2}$ 40.9, BE +2.4 $mEq \cdot l^{-1}$ ($FI_{O_2}$ = 1.0), while cardiac output was 3.6 $l \cdot min^{-1}$. Mean pulmonary arterial pressure had decreased to 15 torr. Dopamine infusion was gradually reduced and then discontinued in the operating room.

Twelve hours after operation, the trachea was extubated. $PGE_1$ infusion of 50 $ng \cdot kg^{-1} \cdot min^{-1}$ was continued for 48 hr postoperatively. Anticoagulant therapy with urokinase, heparin, indomethacine or Warfarin was started postoperatively. On the third postoperative day, the patient started to walk and remained free of cardiopulmonary and neurological symptoms during her 2-month follow-up.

## Discussion

Since fat- or thromboembolism in the lung is rare in Japan, the abnormal mass in the right atrium of our patient was preoperatively assumed to be myxoma, metastatic cancer or coronary thrombosis. Now it is clear that general risk factors acompanying pulmonary thromboembolism, such as postoperative immobility, dehydration and obesity, were involved and that the pulmonary hypertension and right heart failure were caused by thrombosis in the lung and right atrium. Low cardiac output accompanying pulmonary embolism reduced mixed venous saturation ($Sv_{O_2}$) and $Pa_{O_2}$ as a result of ventilation/perfusion (V/Q) imbalance. In this condition, normal pulmonary arterioles are usually fully dilated while the blood flow in the disturbed region is reduced or stopped altogether. However, the use of a pulmonary vasodilator with an inotropic agent may increase the blood flow in the disturbed region and improve V/Q imbalance. Therefore, improved blood gas data, namely increased $Pa_{O_2}$ and decreased $Pa_{CO_2}$, were obtained after the infusion of $PGE_1$ and dopamine. Although $PGE_1$ is known to increase pulmonary shunt through the inhibition of pulmonary hypoxic vasoconstriction in normal man[1], it will dilate the downstream vessels in the stenotic pulmonary arteries and increase the blood flow, resulting in a reduction in dead-space ventilation caused by V/Q imbalance. In addition, $PGE_1$, as well as prostacyclin ($PGI_2$), also inhibits platelet adherence and aggregation in damaged vessels[2,3], where complement-arachidonic acid cascade, especially thromboxane $A_2$, promotes thrombus formation.

Common intraoperative monitoring procedures for pulmonary embolism are: arterial catheterization for arterial pressure and sampling, central venous cannulation for central venous pressure, capnograph, and pulse oximetry with routine monitoring. In addition, a transesophageal echocardiogram was found convenient for observing wall motion, valve function and existence of thrombi in the heart.

Although most vasodilator therapies are associated with significant systemic hypotension, $PGE_1$ may offer an advantage over other vasodilators in that it shows a high clearance rate for each passage through the lung[4]. It is, therefore, capable of reducing pulmonary vascular resistance without producing systemic hypotension[5]. The systemic effect of $PGE_1$ seems to become marked only when the infusion rate exceeds the rate of pulmonary clearance[6]. Even with a moderate dose as used in this caes, however, mean arterial pressure could be decreased without affecting the urine output, thus suggesting the direct diuretic effect of $PGE_1$[7,8].

In summary, a 46-year-old woman with low cardiac output and dyspnea due to pulmonary hypertension associated with acute pulmonary embolism was perioperatively treated with prostaglandin $E_1$ ($PGE_1$) and dopamine. Although $PGE_1$ is known to inhibit pulmonary hypoxic vasoconstriction and increase the chance of pulmonary shunt, the administration of $PGE_1$ in combination with dopamine remarkably improved arterial blood gas data. $PGE_1$ may thus be helpful by causing pulmonary vasodilation and inhibiting platelet aggregation in acute pulmonary embolism. However, many other factors may influence gas exchange. Animal study and/or prospective randomized studies will be necessary to confirm the effectiveness of $PGE_1$ for acute pulmonary embolism. Finally, transesophageal echocardiogram was found to be useful for observation of cardiac function and motion of abnormal masses.

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