A massive Lung Hemorrhage after Successful Weaning from Cardiopulmonary Bypass

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Introduction

Massive lung hemorrhage is a potentially lethal condition that may ensues reduced alveolar gas exchange and the unstable hemodynamic condition. So, it should be investigated thoroughly and brought under control promptly. There are rare cases of massive lung hemorrhage in patients undergoing cardiopulmonary bypass (CPB) or weaning from CPB. We experienced a case of a 20-year-old woman who presented with a massive lung hemorrhage (about 4000 mL) in about one hour and 30 minutes after successful weaning from CPB while undergoing elective repair of atrial septal defect (ASD). Here, we report our case.

Case Report

A 20-year-old woman with a weight of 52.7 kg and a height of 167.5 cm visited the department of cardiovascular and thoracic surgery of our medical institution to undergo elective repair of ASD. The patient had a past history of dyspnea on exertion (DOE) of the New York Heart Association (NYHA) class III, but presented with no other notable symptoms. On preoperative transthoracic echocardiography, the patient had a left ventricular ejection fraction (LVEF) of 33.7%, an ASD of 2.89 cm in size, a mild pulmonary hypertension (PAP = 33.7 mmHg) and the right ventricular (RV) volume overload. On electrocardiography (ECG), the patient had a complete right bundle branch block (RBBB). On chest X-ray, the patient had findings that are suggestive of congenital ASD and RV dilatation. But there were no other abnormalities.

The patient did not receive premedications. On being transferred to an operation room, the patient was evaluated for vital signs. This showed blood pressure of 100/70 mmHg, heart rate of 70 beats/min and SpO2 of 98%. For the induction of anesthesia, etomidate 0.4 mg/kg, remifentanil 0.3 µg/kg/min and vecuronium 0.2 mg/kg were administered intravenously. Because the right lung collapse is essential for performing a minimally invasive cardiac surgery, we performed the endotracheal intubation using a 35-Fr left-sided double lumen tube (DLT) after the induction of anesthesia. We successfully performed the endotracheal intubation in the first attempt. Then, we confirmed the location of a 35-Fr left-sided DLT on fiberoptic bronchoscopy. Anesthesia was maintained with 50% O2, sevoflurane 0.8-2.0 vol% and remifentanil 0.3 µg/kg/min. Tidal volume of 450 mL and respiratory rate of 10 rates/min were set up. End-tidal carbon dioxide partial pressure was maintained at 35-40 mmHg. In addition, to monitor the invasive arterial pressure and central venous pressure, we performed the catheterization for the right radial artery
and the left internal jugular vein under the ultrasonographic guidance.

For minimally invasive cardiac surgery, we performed the superior vena cava cannulation through the right internal jugular vein under the ultrasonographic guidance and confirmed the location of cannula on transesophageal echocardiography (TEE). We also performed the cannulation of the right femoral artery and vein. The patient was intravenously given heparin 300 unit/kg. Then, we performed the CPB using hypothermic fibrillation of the heart.

After one hour and five minutes of pump time, the patient had a repair of ASD. This was followed by one lung ventilation for weaning from CPB, but we noted that the patient had a decrease in SpO₂ to 88%. We confirmed the position of DLT on fiberoptic bronchoscopy. It was normal, and there was a small amount of brown-colored blood around the carina. But we did not perform the suction because we perceived no problems with the ventilation due to a small amount of blood. After replacing the left one lung ventilation to two lung ventilation, SpO₂ was restored to normal (pH: 7.338, PCO₂: 58.9 mmHg, PO₂: 551.9 mmHg, hemoglobin (Hb): 8.8 g/dL, hematocrit (Hct): 26% on the arterial blood gas analysis (ABGA) (FiO₂ = 1.0)). Blood pressure and heart rate were 114/96 mmHg and 91 beats/min, respectively. Therefore, the patient continuously underwent surgical procedure and weaning from CPB.

Protamine 240 mg was slowly infused through peripheral vein to reverse the effects of heparin. After 10 minutes of weaning, the ABGA (FiO₂ = 1.0) results showed a pH of 7.477, a PaCO₂ of 33.5 mmHg, a PaO₂ of 438.7 mmHg, a Hb of 9.9 g/dL and a Hct of 30%. Blood pressure and heart rate were 92/59 mmHg and 119 beats/min, respectively. Activated clotting time was 135 sec. With the administration of fresh frozen plasma (FFP), we removed the cannula in the femoral artery, femoral vein and superior vena cava. During the skin closure, 40 minutes after protamine administration, SpO₂ and blood pressure were decreased to 88% and 72/35 mmHg, respectively. Heart rate was increased to 130 beats/min. We started the continuous infusion of dopamine, dobutamine and epinephrine. This was followed by the ABGA (FiO₂ = 1.0), showing a pH of 7.388, a PaCO₂ of 41.5 mmHg, a PaO₂ of 67.9 mmHg, a Hb of 8.5 g/dL and Hct of 26%.

On fiberoptic bronchoscopy, we confirmed the normal position of DLT and noted the presence of foamy bleeding in the right lung. And then there was a dramatic decrease in SpO₂. We could not therefore examine the left lung. Although we performed the suction and manual positive pressure ventilation, we noted that there was a gradual decrease in SpO₂ and blood pressure. In addition, we also noted that the bleeding side of the lung became fresh and the amount of bleeding was increased. According to the judgement of the surgeon, we prepared the CPB again and then inserted the cannula in the left femoral artery and vein. There was a decrease in SpO₂ and blood pressure to 62% and 44/28 mmHg, respectively, accompanied by an increase in the heart rate up to 150 beats/min. Meanwhile, the patient developed the ventricular tachycardia. We therefore performed the cannulation during the cardioversion and chest compression. During the cardiopulmonary resuscitation (CPR), the patient was intravenously given 4 mg of epinephrine, 1 mg of atropine, methylprednisolone 1 g and thiopental sodium 1 g. We converted from the CPR to the CPB for 15 minutes. After CPB restart, the ABGA results showed a pH of 7.305, a PaCO₂ of 26.2 mmHg, a PaO₂ of 493.7 mmHg, a Hb of 4.9 g/dL and a Hct of 15%. On noticing the stabilization of the hemodynamic profile, we examined the surgical sites and performed the TEE for the purposes of identifying the causes. But there were no abnormalities. After the CPB, the lung showed a bright red color. Based on these findings, the patient was suspected of having pulmonary edema. Bleeding were observed in both lungs on fiberoptic bronchoscopy (Figures 1,2). The patient was placed in a semi-left lateral position. We therefore assumed that the bleeding of right lung was shifted to the left, for which we isolated both lungs by ballooning the left bronchial cuff. But there was a gradual increase in the amount of bleeding; about 4,000 mL of bleeding was seen in both lungs for an hour. We attempted to remove the bleeding on fiberoptic bronchoscopy and to locate its origin. But we could not locate it because of a continuous massive bleeding. With the discontinued ventilation and the decreased pulmonary circulation after the CPB, the patient had a decrease in the amount of bleeding. On blood gas analysis from the DLT (FiO₂ = 1.0), the values were showed pH 7.235, PaCO₂, 58.0 mmHg, PaO₂ 38.5 mmHg, Hb 7.9 g/dL and Hct 24%. These findings are suggestive of the venous bleeding. In addition, on ABGA (FiO₂ = 1.0) from the
pump, the above values were 7.326, 33.5, 454.6, 6.9 and 21% in the corresponding order. With the extracorporeal life support (ECLS), the patient was transferred to an intensive care unit (ICU).

Flow rate of ECLS was started at 3.7 L/min (FiO2 = 1.0), and then was controlled adjusted to the patient condition. Ventilation mode was also adjusted to the patient condition. The patient was intraoperatively given a 5,000 mL of fluid, 18 units of packed RBC and two units of FFP. Just before goes to ICU, the ABGA (FiO2 = 1.0) results showed a pH of 7.392, a PaCO2 of 28.7 mmHg, a PO2 of 452.3 mmHg, a Hb of 8.7 g/dL and a Hct of 26%. Mean arterial blood pressures were maintained 50 - 60 mmHg.

The patient postoperatively underwent chest X-ray at an ICU, as shown in (Figure 3). On postoperative day 1, the patient underwent fiberoptic bronchoscopy at an ICU; this showed that the patient had a scratching wound of the right main bronchus just below the carina and still bleeding in both lungs. It was thought from repeated suction and bronchoscope previous day. But we could not locate the origin of bleeding. The patient was also given injection of diluted epinephrine in the bronchi on fiberoptic bronchoscopy, which was not effective. On noticing that there was a decrease in the amount of bleeding, we attempted to revise the ECLS and then to wean the patient from it. Due to the persistent presence of the bleeding in both lungs on fiberoptic bronchoscopy, the patient was intraoperatively evaluated for the surgical sites again. But there were no abnormalities at the surgical sites. With the resection of scratching wound in the right main bronchus, we attempted to wean the patient from the ECLS. But we found that the patient had an increase in the amount of bleeding again. Meanwhile, the patient had a bleeding of 500 mL in volume for an hour from the DLT tube. We could not therefore wean the patient from the ECLS. The patient had a poor circulation in the left leg, for which we inserted the cannula in the left femoral artery and vein. The patient was transferred to an ICU. The patient was intravenously given seven units of packed RBC, three units of FFP and ten units of platelets. With the postoperative maintenance of the ventilator, the patient was transferred to an ICU. The patient postoperatively underwent chest X-ray at an ICU, as shown in (Figure 4).

On postoperative day 2, the patient had no further bleeding. Then, we revised the ECLS and thereby successfully weaned the patient from it. After weaning from ECLS, the ABGA results showed a pH of 7.411, a PaCO2 of 44.4 mmHg, a PO2 of 124.1 mmHg, a Hb of 7.9 g/dL and a Hct of 24%. Blood pressure and heart rate were 100/50 mmHg and 118 beats/min, respectively. Thereafter, the patient had no further bleeding. Intraoperatively, the patient was given three units of packed RBC, eight units of platelets and eight units of cryoprecipitate. With the postoperative maintenance of the ventilator, the patient was transferred to an ICU. The patient postoperatively underwent chest X-ray at an ICU, as shown in (Figure 5).
On postoperative day 4, DLT was replaced by an ordinary endotracheal tube. On postoperative day 10, the patient was weaned from the ventilator and presented with no complications.

Discussion

Both the bronchial artery and pulmonary artery are responsible for the blood supply in the lung: 90% and approximately 5% of total circulation originate from the bronchial artery and the pulmonary artery, respectively [1].

A massive lung hemorrhage frequently arises from the bronchiectasis, active pulmonary tuberculosis and malignancy [1, 2]. In addition, it may also occur as a result of the infection, vasculitis, vascular injury, immunologic problem and coagulopathy [3]. Furthermore, its etiologies may also include the use of anticoagulants, poor hemodynamic profile, cardiac dysfunction, pulmonary artery catheterization, the misplacement of the left ventricular vent catheter and severe complications arising from pulmonary artery hypertension [4-9]. During open heart surgery, heparin is used to perform the CPB and thereby causes the anticoagulation. As compared with patients undergoing other surgeries, those doing open heart surgery are more vulnerable to bleeding during endotracheal intubation because of the airway suction and damages to the trachea or tracheal mucosa. Moreover, patients who are given protamine for the purposes of reversing the effects of heparin are at risks of developing interalveolar hemorrhage [10]. Also, it may be accompanied by pulmonary edema [11].

It is generally known that it would be mandatory to locate the origin of bleeding on fiberoptic bronchoscopy, which is essential for treating patients with massive lung hemorrhage. With the identification of the origin of bleeding, bleeding lungs should be isolated using a DLT or bronchial blockers. To do this, topical vasoconstrictors may be administered through an endotracheal tube. In patients with massive lung hemorrhage who are refractory to conservative management such as FFP, cryoprecipitate and PLT, recombinant activated factor VII may be used [12]. More than 90% of bleedings originate from the bronchial artery. Therefore, the bronchial artery embolization (BAE) has been widely used to treat patients with massive lung hemorrhage [13]. In patients who presented with massive lung hemorrhage while being weaned from the CPB, the airway management can be performed on rigid bronchoscopy. This is accompanied by the discontinuation of the CPB with the injection of protamine. In patients where the airway management was not successful after weaned from CPB, the CPB should be performed again [6]. In patients with massive hemorrhage arising from the rupture of the pulmonary artery, the rapid application of the CPB would be effective in avoiding the pulmonary resection and the overloading of pulmonary circulation and improving the systemic arterial perfusion and oxygenation [7].

In the current case, we attempted to identify the origin of bleeding on noticing its occurrence in the lung based on the following assumptions:

First, the patient underwent minimally invasive surgery. We therefore assumed that the patient was at increased risks of vascular injury in such a condition that operative field could not be secured. It is a matter of course, however, that bleeding would occur before the patient was weaned from the CPB when the patient was at risks of vascular injury during surgery. In the current case, however, the bleeding was developed during the skin closure after successfully weaned from the CPB. This is supported by a lack of notable gross findings at surgical sites when it was reopened. So, we ruled out this.

Second, the patient underwent placement of the DLT. We therefore assumed that the patient was vulnerable to tracheal or bronchial injury by the DLT in the first attempt. It is a matter of course that the patient would be vulnerable to tracheal wall injury as well as scratching during the placement of a 35-Fr left-sided DLT. But, the patient successfully underwent endotracheal intubation in the first attempt. On bronchoscopic view, the endotracheal tube was accurately positioned and there was no bleeding. If there was a severe vascular injury, the bleeding should be detected immediately after the injection of heparin. In addition, the patient developed the bleeding after the reversal of the effects of heparin with the administration of protamine. We therefore assumed that massive lung hemorrhage did not occur as a result of the injury during the endotracheal intubation.

Although the patient sustained a tracheal injury during the intubation, there is a remote possibility that the patient developed a 4,000 mL of bleeding within an hour. This is because no sufficient blood vessels are distributed in the trachea. Immediately before weaning from the CPB, the patient was evaluated for the decreased SpO2. There was a small amount of secretion with brown-colored blood around the carina on fiberoptic bronchoscopy. But there was no continuous fresh bleeding, for which no suction was required. We assumed not only that the tracheal minor injury occurred due to scratching during the insertion of DLT in the trachea but also that and then small amount of bleeding occurred at the sites of injury following the administration of heparin. We did not perform the suction, which may cause the tracheal injury, because we did not administer protamine to reverse the effects of heparin yet. Also, we ruled out this.

Third, the patient had a large amount of bleeding (4,000 mL) within an hour. The patient was therefore suspected to having a rupture of the bronchial artery. On blood gas analysis through the tube, the data showed PaCO2 58.0 mmHg and PaO2 38.5 mmHg. Because it was venous blood, we thought it was not the bronchial artery rupture.

Fourth, the patient had a chronic pulmonary artery hypertension with LVEF of 33.7%. Therefore, the patient was suspected of having a rupture of the pulmonary artery. Because the blood gas analysis from the tube was venous blood, we assumed that the patient had a bleeding due to a rupture of the pulmonary artery in the right lung. But the patient actually had a bleeding in both lungs. We could not confirm the origin of bleeding because the patient could not undergo angiography due to her poor systemic status. But, if there is a chance that the patient had a bleeding due to a rupture of the pulmonary artery, bleeding would be observed at the time weaned from CPB. But, still we cannot rule out this.

Fifth, we assumed that the patient had a bleeding due to immunoreactions from the heparin-protamine complex or allergic responses to the protamine. It has been reported that the severe adverse effects arising from protamine occur at an incidence of approximately 0.2 - 3% [10]. But, the incidence of noncardiogenic pulmonary edema after protamine administration in unknown.
Allergic or immune reactions to heparin-protamine complex may cause severe pulmonary artery hypertension or activated the complement system, eventually leading to the sequestration of neutrophils and the synthesis of thromboxane A2 [10-14]. They may trigger the occurrence of pulmonary edema and capillary leakage. Patients with pulmonary edema are vulnerable to diffuse lung hemorrhage [11]. So, we assumed that the patient had a sudden onset of pulmonary vasoconstriction and edema as well as diffuse alveolar hemorrhage arising from the microvascular injury. Although we did not examine whether there was an increase in the pulmonary artery pressure because we did not insert Swan Ganz catheter, we found that the patient had a foamy, bloody secretion on the bronchoscopic view when there was a sharp decrease in SpO2. If we examined RV failure with TEE when SpO2 was reduced and blood pressure dropped, it would be more certain. In a tense situation, we had no time to check it out until we re-started the CPB. Usually, these complications are revealed at several minutes after the protamine administration. In the current case, 40 minutes elapsed since the administration of protamine. We could not doubt the possibility of the immune or allergic responses to the heparin-protamine complex.

Sixth, we could not rule out the possibility that the patient developed anaphylaxis while receiving the FFP. The patient had a decrease in SpO2 while first receiving the FFP. It has been reported that the anaphylactic reaction to the FFP occurs at a prevalence of 1/(18,000-172,000) [15]. Despite a lack of changes in the airway pressure, the patient had a decrease in the blood pressure and an increase in the heart rate up to 150 beats/min. This leads to the speculation that the patient had underlying factors involved in the anaphylaxis in the blood. But we failed to consider this possibility because pulmonary edema or bleeding is rarely associated with anaphylaxis. So, we did not discontinue the transfusion or re-analyzed the blood. The patient had a large amount of bleeding but did no changes in the airway pressure, which is suggestive of bronchospasm. In addition, the patient presented with no symptoms, such as urticaria or angioedema, following the removal of surgical drape. We did not consider the possibility of anaphylaxis arising from the FFP. Still we could not fully suspect it in 100 % because pulmonary edema is rarely associated with anaphylaxis.

We assumed that the patient had a bleeding as a result of intraoperative damages to the blood vessels or trachea. We would consider that allergic or immune responses are involved in a massive lung hemorrhage if the patient undergoes other surgeries than open heart surgery. Based on the findings that the patient had a large amount of bleeding, underwent minimally invasive open heart surgery and presented with a collapse of unilateral lung, however, we initially assumed that the vascular or tracheal injury was the most probable cause. The patient had no past history of diabetes and protamine exposure. In addition, the patient had no past history of food allergy. We did not perform the intradermal skin test to protamine. Also we didn't perform other laboratory examinations to assess the activity of complement system. We missed retest the FFP with the patient's blood. If we did, it might be very helpful to diagnosis.

In summary, we could not identify exact causes of massive lung hemorrhage (4,000 mL) in our patient. And we could not find any case report of massive lung hemorrhage like our case after successfully weaned from CPB. In these cases, regardless of cause, it would be mandatory to maintain oxygenation, to reduce the pulmonary circulation, to promote the hemostasis and to increase the systemic arterial perfusion with the immediate use of the ECLS, which is essential for improving the prognosis of patients. Thus, we report this case.

References