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CLINICAL REPORT

A patient with possible TRALI who developed pulmonary hypertensive crisis and acute pulmonary edema during cardiac surgery

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Abstract There are very few case reports of transfusion-related acute lung injury (TRALI) under close hemodynamic monitoring. We encountered a case of possible TRALI during on-pump coronary artery bypass grafting (CABG). A 66-year-old man who had undergone on-pump CABG was administered fresh frozen plasma (FFP). One hour after FFP transfusion, pulmonary hypertensive crisis and subsequent hypoxic decompensation occurred. A second cardiopulmonary bypass (CPB) was needed for circulatory and respiratory deterioration. Extracorporeal life support (ECLS), intraaortic balloon pumping (IABP), and nitric oxide therapy were required after the surgery. Despite the severity of the initial state, his recovery was comparatively smooth. ECLS and IABP were removed on postoperative day (POD)1; the patient was extubated and discharged from the ICU on POD7 and POD12, respectively. The diagnosis of TRALI was confirmed by human leukocyte antigen antibody detection in the administered FFP. In addition, lymphocytic immunofluorescence test showed that a cross-match of the plasma from the pooled FFP against the recipient leukocytes was positive. The clinical course of the pulmonary artery hypertension was followed by a decrease in dynamic lung compliance. The mechanism of this phenomenon is unclear. However, it might suggest the possibility of vasoconstriction or obstruction of the peripheral pulmonary artery preceding lung damage, as in the case in animal models reported previously.

Keywords TRALI · Pulmonary hypertensive crisis · Pulmonary edema · Cardiac surgery

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Introduction

Transfusion-related acute lung injury (TRALI) was first described by Popovsky et al. [1] in 1983. The Canadian Consensus Conference proposed the criteria for TRALI in 2004 [2], which defined TRALI in the clinical context. We encountered a case of possible TRALI that occurred during a cardiac surgery under close hemodynamic monitoring. Some reports have shown that pulmonary arterial pressure (PAP) increases during the development of TRALI. However, none of these reports mention the time gap between the beginning of the increase in PAP and the decrease in dynamic lung compliance (DLC). Here, we describe the time course of a potential case of TRALI, with emphasis on PAP and DLC during TRALI development.

Case report

A 66-year-old man (height 171 cm; weight 68 kg) with known coronary artery disease [coronary angiography (CAG) revealed right coronary artery (RCA) and left anterior descending artery (LAD) stenosis: #3, 75%; #5, 90%; #6, 90%; #9, 90%] was transferred to our institute for coronary artery bypass grafting (CABG); right internal thoracic artery bypass to the LAD, left internal thoracic artery bypass to the posterolateral branch, saphenous graft bypass to the LAD, and posterior descending branch were

planned. He had been treated for asthma with prednisolone (10 mg/day) and theophylline by oral and steroid inhalation. Electrocardiography (ECG) showed no ischemic signs, and transthoracic echocardiography showed mild hypokinesis in the basal and inferior walls. However, constriction and diastolic function were maintained [ejection fraction, 66%; E/e′ (ratio of early transmitral velocity to tissue Doppler mitral annular early diastolic velocity), 5.6], and heart size and valve function were almost normal. Laboratory examination gave the following results: hemoglobin level, 94 g/l; platelet count, 182×10^9/l; international normalized ratio of prothrombin time, 1.17; activated partial thromboplastin time, 39.1; and fibrinogen level, 4.48 g/l. Chest radiographs showed a clear lung field and no cardiomegaly. General anesthesia was induced with midazolam, fentanyl, and rocuronium and maintained with air, oxygen, and sevoflurane. Mechanical ventilation was pressure controlled. Although his vital signs were stable after induction, when the heart was turned over and a stabilizer was placed, an ST depression emerged on the ECG monitor, and the procedure was then altered to on-pump CABG. Weaning of the cardiopulmonary bypass (CPB) was smooth, and all the vital signs were stable until surgical closure. However, 1 h after fresh frozen plasma (FFP) administration, the PAP suddenly increased from 24/13 to 57/13 mmHg. After 10 min, the radial arterial pressure (RAP) dropped from 130/60 to 48/33 mmHg (Fig. 1). During this period, the central venous pressure (CVP) remained unchanged at 8 mmHg; however, it gradually increased to 15 mmHg after PAP increased. During circulatory devastation, the end-tidal CO_2 remained at 38 mmHg. In addition, the end-tidal CO_2 waveform did not exhibit an obstructive pattern. Transesophageal echocardiography (TEE) performed at this point showed a severely enlarged and hypokinetic right ventricle, a collapsed and hyperkinetic left ventricle, and septal flattening. There were no new findings of valvular dysfunction or wall motion abnormalities. Twenty minutes after PAP increased dramatically, DLC decreased from 60 to 24 ml/cmH_2O and tidal volume decreased from 597 to 292 ml (peak inspiratory pressure, 15 cmH_2O; Fig. 1). There was no evidence of rash or bronchospasm, which would imply an anaphylactic reaction. The total in–out balance at this point was +2,000. The CVP was stable at 8 mmHg; no rapid infusion was performed after CPB weaning. The TEE did not suggest any transfusion-associated circulatory overload (TACO). At first, we thought that this was a severe asthma attack or pulmonary embolism. We changed the sedative agent from propofol to sevoflurane, ventilated with 100% oxygen, and initiated 20-ppm nitric oxide therapy, salbutamol inhalation, continuous intravenous (c.i.v.) magnesium sulfate infusion, and 125-mg IV methylprednisolone. Hypotension was treated with aggressive fluid administration and infusion with norepinephrine, dopamine, and ephedrine. However, soon after the tidal volume decreased, a frothy exudate from the endotracheal tube was noted, and his circulatory and respiratory states were devastated. CPB was subsequently restarted. During these events, the PaO_2:FiO_2 ratio (P/F) decreased from 445 (during surgical closure) to 157 [from the right radial artery on extracorporeal life support (ECLS)]. At this point, TRALI was suspected. We administered 100-mg IV hydrocortisone.

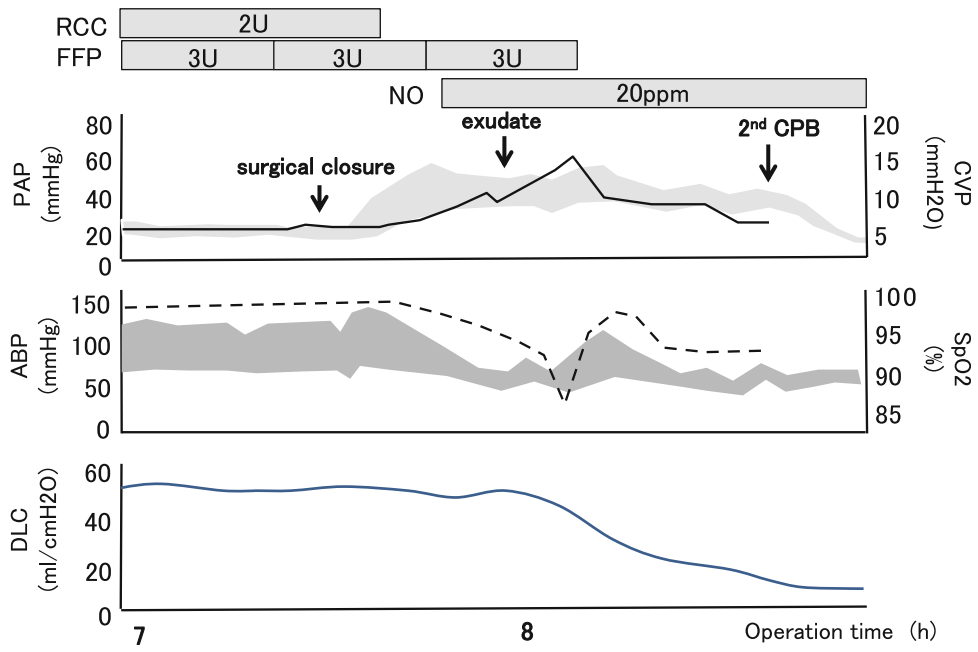

Fig. 1 Clinical course of the patient. *PAP* pulmonary arterial pressure, *ABP* arterial blood pressure, *CVP* central venous pressure, *DLC* dynamic lung compliance, *RCC* red cell concentrate, *U* units, *FFP* fresh frozen plasma. *2nd CPB*, cardiopulmonary bypass was restarted

We asked the Japanese Red Cross Society to perform the leukocyte antibody test. After the patient was weaned from CPB, he was admitted to the ICU with ECLS (cannulations of the femoral artery and femoral vein were performed for V-A ECMO), intraaortic balloon pumping (IABP), and nitric oxide inhalation. The CPB time was 226 min, with a cross-clamp time of 120 min; 2 units of RCC and 9 units of FFP were transfused. Chest computed tomography (CT) and the radiograph after the operation are shown in Fig. 2. Despite the severity of the patient's initial state, his recovery was comparatively smooth. Twelve hours after the event, the circulatory and respiratory states improved (DLC, 46.0 ml/cmH_2O; P/F, 246; RAP, 109/62 mmHg; PAP, 22/16 mmHg). The ECLS and IABP were removed after 12 and 18 h, respectively. The patients was extubated on postoperative day 7 (POD7), and discharged from the ICU on POD12. The diagnosis of TRALI was confirmed by HLA class 1 antibody detection in the administered FFP. The lymphocytic immunofluorescence test showed that the cross-match between the plasma from the pooled FFP against the recipient leukocytes was positive. We were then informed that the FFP that was suspected to have caused TRALI was obtained from a multiparous female donor from the Japanese Red Cross Society.

Discussion

TRALI is a fatal complication associated with transfusion. In 2004, common clinical criteria were proposed in Canada [2]. In Japan, case reports on TRALI, possible TRALI, and TRALI developing in the perioperative period numbered 118, 39, and 12, respectively [3]. Although TRALI usually develops in the perioperative period [4–6], rare cases develop under close hemodynamic monitoring during cardiac surgery. In our case, the diagnosis of TRALI was confirmed by leukocyte antibody–antigen cross-matching. The pathophysiological mechanism of TRALI follows the antibody and neutrophil priming hypotheses [7], both of which occur with leukocyte priming and activation, peripheral pulmonary endothelial cell damage, and capillary leakage. In contrast, the pathophysiological mechanism of vasomotor derangement that is followed by pulmonary hypertension before the development of acute pulmonary edema is not much emphasized in the clinical literature and animal models [8]. Lin et al. [9] reported a case of TRALI after on-pump CABG with vasomotor derangement and pulmonary hypertension, similar to our case. They also mentioned that vasomotor derangement might be related to peripheral pulmonary artery vasospasms or occlusive platelet thrombi. In our case, a sudden

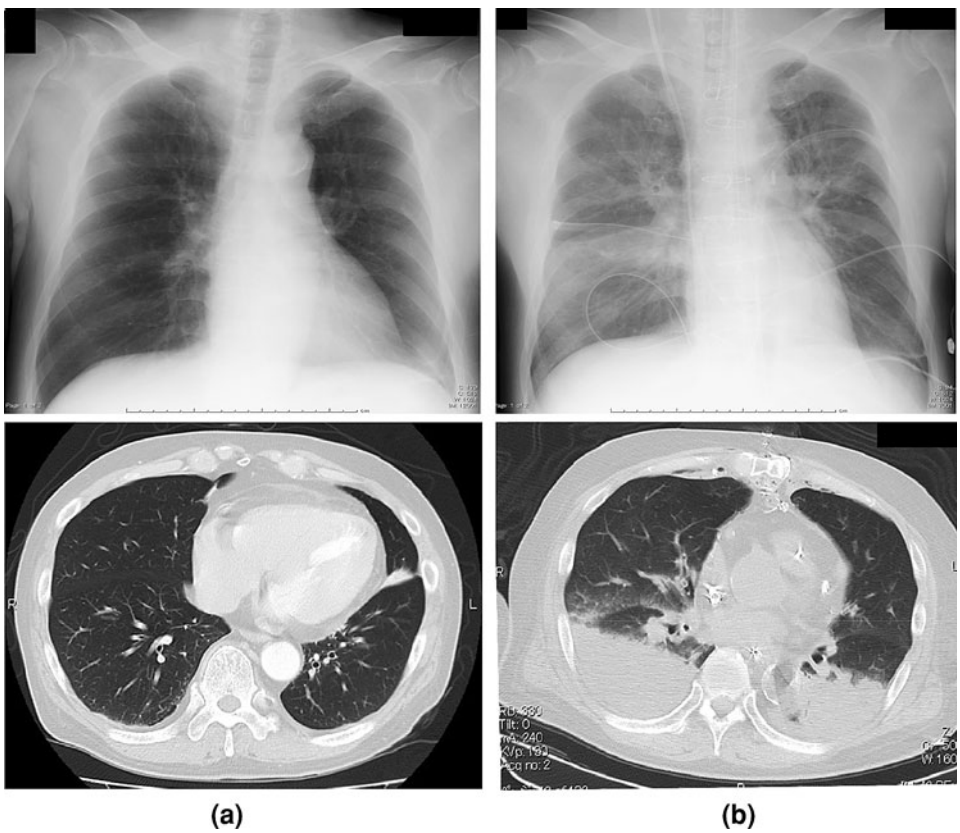

Fig. 2 Chest radiograph and computed tomography (CT): before operation (**a**); 1 h after operation (**b**)

PAP increase occurred initially. At that time, the DLC and end-tidal CO_2 waveform were unchanged. Twenty minutes later, copious pulmonary exudate was observed, and the DLC and tidal volume decreased dramatically, which implies that a peripheral vascular event occurred before capillary leakage. In clinical settings, an increase in PAP before capillary leakage should be detected because, in some cases, this is followed by pulmonary hypertensive crisis and circulatory devastation. TACO is an important differential diagnosis of TRALI; both TEE and pulmonary artery catheter would be needed to precisely differentiate them. Clinicians can evaluate wall motion abnormalities and ventricular size by using TEE and wedge pressure by using a pulmonary artery catheter. According to a review, CVP is normal during TRALI [10]. In our case, the increase in PAP immediately resulted in right heart failure. In such settings, clinicians cannot evaluate volume status based on CVP, which increases independent of cardiac volume overload. When we rule out TACO, we should be cautious whether CVP is used as an index of volume status. Whether brain natriuretic peptide (BNP) can differentiate TACO from TRALI during cardiac surgery is controversial. In our patient, we measured BNP when TRALI symptoms developed (297.6 pg/ml). During cardiac surgery, many factors are expected to cause volume overload, which possibly affects BNP. Therefore, BNP may not be a good marker of TACO during cardiac surgery. As a preventive strategy, the UK National Blood Service has adopted the use of predominantly male blood products to minimize the risk of exposure to leukocyte antibodies from multiparous female donors. The UK Serious Hazards of Transfusion data show that TRALI cases decreased from 16 in 2003 to 3 in 2005 [10], proving that avoiding FFP from female donors and checking the HLA antibody before transfusion is helpful for preventing TRALI.

Conclusion

We encountered a case of possible TRALI during cardiac surgery and PAP monitoring showed that initial pulmonary hypertension occurred simultaneously after a hypoxic event. Both TEE and pulmonary artery catheter are needed to precisely differentiate TACO and TRALI. Although the mechanism of pulmonary hypertensive crisis is unclear, it might indicate the possibility of initial vasoconstriction or obstruction of the peripheral pulmonary arteries when TRALI is triggered, as mentioned in the existing literature on animal models.

References

1. Popovsky MA, Abel MD, Moore SB. Transfusion-related acute lung injury associated with passive transfer of antileukocyte antibodies. Am Rev Respir Dis. 1983;128:185–9.
2. Kleinman S, Caulfield T, Chan P, Davenport R, McFarland J, McPhedran S, Meade M, Morrison D, Pinsent T, Robillard P, Slinger P. Toward an understanding of transfusion-related acute lung injury: statement of a consensus panel. Transfusion. 2004;44:1774–89.
3. Iijima T, Okazaki H. Current concept of TRALI (transfusion-related acute lung injury). Masui (Jpn J Anesthesiol). 2007;56:1319–28. (in Japanese with English abstract).
4. Kanemura S, Ohta N, Yamanaka H, Takahashi R, Hirao O, Uchiyama A, Fujino Y, Mashimo T. Two cases of transfusion-related acute lung injury following massive transfusion during the perioperative period. Masui (Jpn J Anesthesiol). 2009;58:480–3. (in Japanese with English abstract).
5. Ito T, Kusunoki S, Kawamoto M. A case of transfusion-related acute lung injury associated with severe intraoperative hypoxemia. Masui (Jpn J Anesthesiol). 2008;57:1265–8. (in Japanese with English abstract).
6. Sakata U, Wada H, Oshima T, Aramaki Y, Kikuta Y, Iwasaki Y. Anesthetic management of a patient with transfusion-related acute lung injury (TRALI). Maui (Jpn J Anesthesiol). 2008;57:1017–20. (in Japanese with English abstract).
7. Benson AB, Moss C, Silliman CC. Transfusion-related acute lung injury (TRALI): a clinical review with emphasis on the critically ill. Br J Haematol. 2009;147:431–43.
8. Bux J, Sachs JHU. The pathogenesis of transfusion-related acute lung injury (TRALI). Br J Haematol. 2007;136:788–99.
9. Lin U, Kanani N, Naughton F, Pendergrast J, Karkouti K. Case report: transfusion-related acute lung injury (TRALI)—a clear and present danger. Can J Anesth. 2007;54:1011–6.
10. Triulzi DJ. Transfusion-related acute lung injury: current concepts for the clinician. Anesth Analg. 2009;108:770–6.