Pulmonary fat embolism is a life-threatening complication for patients with long-bone fractures undergoing surgery.\textsuperscript{1–4} The incidence of fat embolism syndrome ranges between 0.9 and 2.2\%, and the mortality rate has been reported to be 13–87\%.\textsuperscript{5–8} We report a case of severe haemodynamic instability following fat embolism, which was supported successfully using percutaneous cardiopulmonary support (PCPS).

**Case report**

A woman of 61 yr of age, hit by a car, was admitted for repair of left femur fracture. An intramedullary nail was planned to stabilize the fracture. Standard monitoring (ECG, non-invasive blood pressure, and oxygen saturation) was started. Lumbar puncture was performed and spinal anaesthesia given using isobaric bupivacaine 0.5\% (2.5 ml). The patient was sedated using intermittent midazolam 2 mg, and the breathing air was enriched with oxygen using a nasal cannula. After subarachnoid block her blood pressure was 100/60 mm Hg, heart rate 80 beats min\textsuperscript{-1}, and $S_{\text{PO}_2}$ 100\%. Approximately 10 min after the insertion of intramedullary nail, the patient suddenly became excited and this excitement progressed to grand mal seizures. The oxygen saturation decreased from 100 to 87\%. The patient became tachypnoeic, and ECG showed bigeminy and paroxysmal ventricular contractions. Within seconds, she developed profound hypotension and shock-like state; the oxygen saturation could not be monitored. The trachea was immediately intubated and the lungs were mechanically ventilated. At the onset of ventilation, the end-tidal carbon dioxide concentration was noted to be between 0 and 0.27 kPa. Radial artery cannulation was performed and her arterial blood gas analysis showed pH 7.019, $P_{\text{aO}_2}$ 2.7 kPa, and $P_{\text{acO}_2}$ 14.4 kPa with a base deficit of 3.6 mEq litre\textsuperscript{-1} while breathing 100\% oxygen. We hurried the surgeon to finish suturing and the surgery was over. While the surgeon was suturing we placed a pulmonary artery (PA) catheter. The systolic pulmonary arterial pressure was 50 mm Hg and diastolic pressure was 30 mm Hg, and pulmonary hypertension (PH) was diagnosed. Transthoracic ultrasoniccardiogram (UGG) revealed massive dilation of the right ventricle, the diameter of the inferior vena cava being 27 mm, and diaphoric change accompanying inspiration was not observed. The wall motion of the left ventricle was preserved. We diagnosed the cause for circulatory insufficiency as right heart failure and acute cor pulmonale. The ECG changes showed pulseless ventricular tachycardia and fibrillation, and atrial fibrillation. Defibrillation had been attempted and epinephrine administered, but her cardiopulmonary condition had been unstable, and resuscitation (CPR) was continued in the operation theatre. Blood gas analysis during CPR showed pH 7.075, $P_{\text{aO}_2}$ 45.6 kPa, and $P_{\text{acO}_2}$ 5.6 kPa with a base deficit of 16.9 mEq litre\textsuperscript{-1} while breathing 100\% oxygen. The end-tidal carbon dioxide was between 0 and 1.6 kPa, and oxygen saturation could not be monitored.

After successful resuscitation (i.e. CPR, epinephrine administration, and defibrillation), continuous i.v. infusions of norepinephrine 1.5 $\mu$g kg\textsuperscript{-1} min\textsuperscript{-1} and dopamine...
20 μg kg⁻¹ min⁻¹ were started and the rates of infusions were adjusted to maintain the arterial systolic blood pressure >80 mm Hg. Systolic PA pressure was 51 mm Hg and pulmonary capillary wedge pressure (PCWP) was 33 mm Hg. Dobutamine 20 μg kg⁻¹ min⁻¹ was added for lowering the PA pressure. Ultrasonic ECG showed that her right ventricle had been severely dilated but left ventricular wall motion was maintained and there was no asynergy of left ventricle. About an hour after the resuscitation, despite optimal maximal dose of pharmacological support, we could not maintain the systolic blood pressure >80 mm Hg; at this stage, we decided to place a portable PCPS.

The PCPS is composed of heparin-coated circuits (Carmeda™ Closed Chest Support System), a biopump, a heat exchange unit, and a Maxima™ membrane oxygenator (Medtronic Cardiopulmonary CO, Anaheim, CA, USA). We inserted 19 Fr of drainage cannula into the femoral vein and 17 Fr of re-infusion cannula into the femoral artery percutaneously. The initial blood flow was 2.5 litre min⁻¹ and rotation rate of biopump was 2500 rpm. The oxygen fraction was 1.0 at 4.0 litre min⁻¹. About 1 h after the PCPS had been started, the ECG returned to normal sinus rhythm and her cardiopulmonary collapse began to recover; her systolic blood pressure was 92 mm Hg and heart rate was between 90 and 100 beats min⁻¹. The PA pressure gradually decreased from 50 to 34 mm Hg and her general condition became stable and she obeyed our commands. The patient’s blood gas analysis showed pH 7.34, Pao₂ 63.9 kPa, and Paco₂ 3.7 kPa with 100% oxygen. The mixed venous oxygen saturation increased from 48 to 75%. The blood flow was maintained at 2.5 litre min⁻¹ and her core temperature had been lowered to less than 34°C for the protection of cerebral function. After the initiation of PCPS and having stabilized her cardiovascular condition, we moved the patient from operation theatre to a radioscintigraphic examination as by this time we had strong suspicion of fat embolism. The radioscintigram showed diffuse multiple defects of blood flow in both lungs. After scintigraphic examination, we brought the patient to the intensive care unit (ICU).

We diagnosed the patient as having pulmonary fat embolism from the presence of lipid granules, sampled from the tip of the PA catheter and stained with oil red O.⁹ On the first ICU day, the patient’s conditions were stable under the PCPS. On the second ICU day, we lowered PCPS flow to 1.0 litre min⁻¹ and then to 0.5 litre min⁻¹. Throughout this time, the systolic blood pressure was maintained above 120 mm Hg and heart rate was between 80 and 90 beats min⁻¹. The cardiac output (CO) was more than 3.5 litre min⁻¹. The PA pressure had been between 25 and 30 mm Hg. We therefore decided to stop and remove the PCPS from the patient. Immediately after disconnecting the drainage cannula from the femoral vein, the systolic blood pressure decreased to 10 mm Hg. The ECG showed wide QRS complex and the patient was in severe shock state—PCPS was resumed immediately. The ECG showed dilatation of the right ventricle, septal akinesia, and hypokinesis of the left ventricle. Ejection fraction was lowered to <10% and PCPS was continued. We had to wait for the recovery of her cardiac function. On the third ICU day, CO was 4.0 litre min⁻¹ and PCWP 20 mm Hg. On the fourth ICU day, systolic blood pressure was 140 mm Hg, heart rate between 80 and 90 beats min⁻¹, systolic PA pressure between 25 and 30 mm Hg, PCWP 20 mm Hg, and cardiac index 3.6 litre m⁻² min⁻¹ using the PCPS flow of 1.0 litre min⁻¹. Adequate blood supply to both the lungs, except for one apical part of the upper left lung, and good wall motion of the heart were observed by i.v. digital subtraction angiography. The PCPS was stopped for 30 min and haemodynamic conditions remained stable. We decided to wean the patient from PCPS. When the PCPS drainage cannula was withdrawn from the femoral vein, systolic PA pressure suddenly increased from 30 to 65 mm Hg. ECG showed paroxysmal supra-ventricular tachycardia with the heart rate between 180 and 190 beats min⁻¹. Cardioversion was attempted, and this resulted in sinus rhythm with the heart rate between 90 and 100 beats min⁻¹. The systolic arterial blood pressure was between 100 and 120 mm Hg. However, PA pressure remained as high as 65 mm Hg and we administered prostaglandin E1 (PGE₁) 0.5 μg kg⁻¹ min⁻¹. After PGE₁ infusion, PA pressure gradually decreased from 65 to 40 mm Hg. One hour after weaning from PCPS, the patient showed blood pressure of 120/90 mm Hg, heart rate between 90 and 100 beats min⁻¹, cardiac index of 3.0 litre min⁻¹ m⁻², and mixed venous oxygen saturation of 73%. However, pulmonary hypertensive state continued. PGE₁ 0.2 μg kg⁻¹ min⁻¹ gradually relieved the PH and PA pressure decreased to 25 mm Hg (mean) and PCWP was 9 mm Hg on the fifth postoperative day.

On the eighth postoperative day, PA pressure was 33/15 mm Hg (mean 19 mm Hg) and PCWP was 10 mm Hg and the infusion of PGE₁ was stopped. Sedation was stopped, and the patient opened her eyes and obeyed our commands. She was extubated, transferred to a general ward, and discharged without neurological complications.

Discussion

In our case, soon after the cardiovascular collapse, right heart failure and right ventricular dysfunction were detected by ECG. It is acknowledged that the increases in pulmonary arterial pressure and resistance induce right ventricular failure that may persist for several hours.¹⁰¹¹ Subsequent right ventricular dilatation may result in septal shift resulting in a decrease in left ventricular filling volume, leading to low CO. High right ventricular end-diastolic pressure in addition to low systemic blood pressure results in ischaemia of the right ventricle, potentiating the vicious circle of right ventricular depression, dysfunction, and death. We kept cardiac massage going for an hour. Conventional treatment for right ventricular dysfunction includes the use of pulmonary vasodilator, volume loading, inotropic vasopressors, and adequate
perfusion pressure. Mechanical circulatory support is indicated for patients with right heart failure who show no improvement in response to conventional therapy.10–12

PCPS is a powerful resuscitative tool that may stabilize the condition of patients with cardiac arrest and improve their survival.12–14 PCPS is an extracorporeal life support that involves the continuous drainage of venous blood to a pump and membrane oxygenator and re-infusion to a major vein or artery. Venovenous support is the primary mode for respiratory failure, known as extracorporeal membrane oxygenation; this is primarily used in respiratory failure when oxygenation is preferred to cardiac support. Venoarterial bypass provides full support for both respiratory and cardiovascular failure and is called as PCPS in Japan. Venoarterial bypass involves accessing the right atrium or inferior vena cava for venous drainage and infusion into femoral artery; this type of support is used for patients who require cardiovascular support. PCPS can be used for patients with circulatory collapse and patients with damaged pulmonary circulation.

PCPS drains blood from the right atrium, shunting right ventricle and pulmonary circulation, and oxygenated blood returns to systemic circulation. Therefore, we think the use of PCPS for patients with pulmonary embolism is also a suitable method for resuscitation from cor pulmonale, catastrophic cardiopulmonary collapse, damaged pulmonary circulation, and PH.

The advantage of PCPS is cannulation into the femoral vessels can be performed relatively quickly percutaneously. PCPS can be started within 15 min. The heparin-coated PCPS circuit can be used for the patients soon after surgery. The main complications of PCPS are haemorrhage, ischaemia to lower legs, infection, and haemolysis. Arterial blood flow is mechanically returned into the femoral artery in the antegrade direction to spontaneous heart beat that increases afterload of the left ventricle and might cause left ventricular dysfunction. Considering the limitation and complications during longer PCPS, we had tried rapid initial weaning from PCPS, but we should have continued the PCPS until we confirmed the recovery of cardiac function and blood supply to PA as acknowledged on the fourth postoperative day. Administration of a large dose of PGE1 reduces afterload of the right ventricle and it improves refractory right heart failure. PGE1 is reported to be more pulmonary specific than nitroglycerin, sodium nitroprusside, and hydralazine, and resulted in the largest decrease in PA pressure.15 PGE1 induced the largest decrease in PA pressure compared with the other pulmonary vasodilators isoproterenol, prostacyclin, and nifedipine.16 The PCPS maintained our patient’s condition well so that we did not use PGE1 under the PCPS support. However, we should have administered PGE1 from the start of the PCPS, and the cardiopulmonary condition of the patient might have been more stable. If conventional pharmacological measures cannot restore the patient from the vicious circle from pulmonary fat embolism, PCPS can be considered. This case showed that PCPS could provide effective support for the patients with fatal pulmonary fat embolism syndrome. Administration of PGE1 seemed effective for PH after weaning from the PCPS support.

References
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