FAT embolism syndrome is a fatal complication after total hip arthroplasty. But its pathophysiology is not exactly clear.1 We report a case of hemodynamic collapse after fulminant fat embolism, which was supported effectively with percutaneous cardiopulmonary support (PCPS). In addition, we report histopathologic analysis of fatty embolisms that were captured by the artificial lung of the PCPS.

**Case Report**

A 76-yr-old man with a right femoral neck fracture was scheduled to undergo bipolar hip arthroplasty during general anesthesia. The patient has been under appropriate medication for hepatitis C, hypertension, and diabetes mellitus. We prepared the patient for general anesthesia with minimum monitoring devices for noninvasive blood pressure, blood oxygen saturation, end-tidal carbon dioxide, electrocardiogram, and body temperature.

The patient went through induction with 200 mg thiopental, 0.1 mg fentanyl, 6 mg vecuronium, and 5% sevoflurane uneventfully. After induction, we changed the patient to the right-side up position and added a sacral block (20 mg lidocaine [1%] and 3 mg morphine) for the postoperative pain. The operation started with this position, and anesthesia was maintained with sevoflurane. Soon after compressive insertion of a metal stem device with bone cement into the femoral bone canal, the end-tidal carbon dioxide decreased suddenly from 35 mmHg to 15 mmHg, and a shock state followed. The surgery was stopped, and we quickly returned the patient to the supine position. While central venous blood access was being placed, the patient’s heart rate decreased to less than 40 beats/min. His blood pressure decreased unmeasurably lower; a state of pulseless electrical activity was observed.

Cardiopulmonary resuscitation in conformity with Advanced Cardiac Life Support 20052 was started. Five minutes after the initial pulseless state, blood pressure was 70/30 mmHg, and heart rate increased to 120 beats/min. An arterial catheter was placed at this point to the brachial artery for arterial blood pressure monitoring. We executed transesophageal echocardiography. A huge thrombus in the right atrium and massive dilation of the right atrium and ventricle were revealed. Despite an almost maximal dose of pharmacologic support (0.5 μg·kg⁻¹·min⁻¹ epinephrine and 10 μg·kg⁻¹·min⁻¹ dobutamine), the cardiopulmonary condition remained unstable, and the blood pressure continued to decrease. We decided to place a portable PCPS (Capiox EBS; Terumo, Tokyo, Japan) with an 18-French drainage cannula into the femoral vein and a 16-French infusion cannula into the femoral artery. The PCPS started with the initial rotation rate at 1,500 rpm, but we could not achieve sufficient blood flow output for poor venous drainage. After increasing the rotation rate to 3,000 rpm, the blood drainage improved to obtain enough blood flow (3.5 l/min). We observed compact clusters trapped in the artificial lung of the PCPS device (fig. 1). Approximately 10 min after PCPS was started, the vital signs became stable. We tried a second observation of the heart with transesophageal echocardiography and found normalization of the right chambers. Approximately 1 h after PCPS was started, we began decreasing PCPS blood flow gradually to 0.5 l/min. The cardiopulmonary condition remained stable. So we decided to remove the PCPS from the patient. Even after the removal of the PCPS, the patient’s hemodynamic condition remained unchanged long enough; the patient was transferred from the operating room to the intensive care unit. He was discharged from the hospital 2 months after the surgery.

The compact clusters in the artificial lung were analyzed histopathologically in the following procedure: (1) Make a hole in the artificial lung and remove the compact clusters as specimens. (2) Fix them with 10% formalin. (3) Make some of them into paraffin sections and stain the sections with hematoxylin and eosin. (4) Make some of them into frozen sections and stain the sections with oil red O. (5) Examine the stained sections under the light microscope. Figures 2–4 show that the compact clusters in the artificial lung were mixed thrombus with large quantities of lipid granules.

**Discussion**

This is the first observation of the histopathologic features of a fatty embolism in a living body. Although fat embolism syndrome has been recognized for approximately 140 yr,1 its pathophysiology is not exactly clear.

This syndrome can be categorized clinically into three classes: subclinical, nonfulminant subacute type, and fulminant type.3 The fulminant fat embolism syndrome is distinguished by its rapid progress and high mortality, occasionally reported after cemented endoprosthesis total hip arthroplasty.3 In an orthopedic procedure or in a bone fracture, when the intramedullary pressure becomes higher than the venous pressure of the feeding vessels of the bones, fat globules can be pushed into the vein, and they travel back to the heart and reach the lung. Then, fat globules embolize the lung vessels, which is called “the mechanical theory,”4 and it is considered the main mechanism of the fulminant fat embolism syndrome.

It has been unclear whether the agent that embolizes the lung in fulminant fat embolism syndrome is a fat globule itself. Kao et al.5 reported that there were fat droplets and fibrin thrombi in the lungs at autopsies performed in patients with fulminant fat embolism syn-

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drome. Meanwhile, Timothy et al. 6 reported that an autopsy performed in a patient with fulminant fat embolism syndrome revealed no evidence of thromboembolic clot in the lung. However, in our case, the agent in the living body was mixed thrombus with large quantities of lipid granules. This fact suggests that, after entrance into venous circulation, fat globules form a thrombus in the inferior vena cava and embolize in the lung. There is some possibility that the thrombus was formed in the artificial lung.

However, when PCPS started, heparin was already given, and the activated coagulation time was more than 300 s. So it is unlikely that the thrombus was formed in the artificial lung. From these findings, we can speculate that thrombolytic therapy may be a therapeutic option for fulminant fat embolism syndrome. Intraoperative use of a thrombolytic agent is contraindicated because of its common complication, adverse bleeding. However, Jackson et al. 7 reported a case of successful use of recombinant tissue plasminogen activator intraopera-

tively during orthotopic liver transplantation. In this report, they suggested that monitoring of fibrinogen levels may reduce the incidence of adverse bleeding associated with continuous infusion of recombinant tissue plasminogen activator. In addition, they mentioned that cryoprecipitate could be administered if adverse bleeding is problematic. It may be worth investigating this therapy for fulminant fat embolism syndrome.

Recently, several case reports showed that PCPS was a strong therapeutic tool for patients with fulminant fat embolism syndrome.8–10 In this case, PCPS was also effective for stabilization of the condition of the patient.

In conclusion, in this case, we could observe the histopathologic features of a fatty embolism in a living body complicated by fulminant fat embolism syndrome, and there was mixed thrombus with large quantities of lipid
granules. In addition, this case showed the efficacy of PCPS for patients with fulminant fat embolism syndrome.

References