General Anesthesia

Cerebral fat embolism diagnosed by magnetic resonance imaging at one, eight, and 50 days after hip arthroplasty: a case report

[L'embolie graisseuse cérébrale diagnostiquée par imagerie par résonance magnétique aux jours 1, 8 et 50 après une arthroplastie de la hanche : une étude de cas]

Nobuko Sasano MD PhD,* Susumu Ishida MD,† Shinichiro Tetsu MD,† Hiroe Takasu MD PhD,†
Kiyoshi Ishikawa MD PhD,† Hiroshi Sasano MD PhD,* Hirotada Katsuya MD PhD*

Purpose: To describe cardiovascular collapse during a cemented hip hemiarthroplasty in a patient who, despite a successful cardiopulmonary resuscitation, remained in a persistent vegetative state due to cerebral fat embolism diagnosed by magnetic resonance imaging (MRI).

Clinical features: A 75-yr-old woman with no medical history underwent cemented hip hemiarthroplasty under spinal anesthesia for a right femoral neck fracture. Shortly after insertion of the prosthesis, a sudden oxygen desaturation, hypotension, bradycardia, and cardiac arrest occurred. The patient was successfully resuscitated, but did not regain consciousness. The patient developed high-grade fever, thrombocytopenia, anemia, and oliguria. MRI scans of the brain revealed multiple high intensity signals throughout the white matter, the basal ganglia, the cerebellum, and the brain stem. The diagnosis of fat embolism was made on the basis of clinical findings and MRI images. Although her cardiorespiratory status improved over the next week, the patient remained in a persistent vegetative state.

Conclusion: When fat embolism is suspected, serial MRI scans of the brain should be performed to diagnose the etiology of cerebral embolism as well as to evaluate the severity of brain damage.

Objectif : Décrire le collapsus cardiovasculaire survenu pendant une hémi-arthroplastie de la hanche chez une patiente qui, malgré une réanimation cardiopulmonaire réussie, est demeurée dans un état

végétatif persistant à cause d'une embolie graisseuse diagnostiquée grâce à l'imagerie par résonance magnétique.

Éléments cliniques : Une femme de 75 ans, sans antécédents médicaux, a subi une hémi-arthroplastie cimentée de la hanche sous rachianesthésie à la suite d'une fracture du col du fémur. Peu après l'insertion de la prothèse, il y a eu l'apparition soudaine de désaturation en oxygène, d'hypotension, de bradycardie et un arrêt cardiaque. La patiente, réanimée avec succès, n'a pourtant pas repris conscience. Une forte fièvre, une thrombocytopénie, de l'anémie et de l'oligurie se sont développées. L'IRM du cerveau a révélé de multiples signaux de haute intensité dans la matière blanche, les noyaux gris centraux, le cervelet et le tronc cérébral. Les constatations cliniques et les images d'IRM ont conduit au diagnostic d'embolie graisseuse. Même si l'état cardiorespiratoire s'est amélioré au cours de la semaine suivante, la patiente est demeurée dans un état végétatif persistant.

Conclusion : En cas d'embolie graisseuse présumée, une série d'examens d'IRM du cerveau devraient être faits pour découvrir la cause de l'embolie et évaluer la sévérité de la lésion.

J OINT arthroplasty and femoral nailing have been reported to cause perioperative fat embolism (PFE),[1–12] because high intramedullary pressures associated with insertion of a prosthesis or nailing induce the intravasation of bone marrow contents and microembolization.[13–15] Embolization to the lungs can cause

hypotension, hypoxemia, cardiac arrest and death.[1–8] Recently, it was shown that these fat emboli can traverse the pulmonary vasculature, and cause brain embolization,[16] which results in postoperative coma.[9–12]

We report a patient who suffered an intraoperative cardiovascular collapse due to severe fat embolism, and remained with severe neurological deficit despite successful cardiopulmonary resuscitation. Magnetic resonance imaging (MRI) contributed to the diagnosis of cerebral fat embolism.

Case report

A fit and active 75-yr-old woman with no medical history fell and sustained a right femoral neck fracture. She was admitted to our hospital and was doing well until the elective hemiarthroplasty of the hip performed three days later. In the operating room, the patient was alert, her heart rate (HR) was 90 beats·min^{-1} and her blood pressure was 170/80 mmHg. Oxygen saturation measured by pulse oximetry (SpO$_2$) was 96% while breathing room air. Intravenous access was obtained and a radial artery catheter was inserted to monitor blood pressure. Spinal anesthesia was administered with intrathecal injection of 4 mL isobaric 0.5% bupivacaine in the left lateral decubitus position. This resulted in an analgesic level of T7. After the block was placed, the patient's blood pressure gradually fell to 130/60 mmHg. Sedation was obtained by the *iv* injection of 2 mg midazolam and 2 L·min^{-1} nitrous oxide inhalation mixed with 1 L·min^{-1} oxygen through a nasal cannula. SpO$_2$ was maintained in the 95 to 97% range, and blood pressure was 120/60 to 140/70 mmHg. She was asleep for about 30 min, but complained of the reaming noise. One additional milligram of midazolam was injected intravenously. No detectable change in cardiorespiratory status was noted. The femoral medullary canal was reamed and pulsatile lavage of the femoral canal was performed. Fifty minutes after the incision, methylmethacrylate cement was injected into the femoral canal, immediately followed by the insertion of a stemmed femoral prosthesis. Three minutes later, oxygen desaturation (SpO$_2$ of 90%) was noted. Nitrous oxide was discontinued and the oxygen flow rate was increased to 3 L·min^{-1}, improving SpO$_2$ to 93%. The patient looked slightly uncomfortable but was still breathing normally. Ten minutes after injection of the cement, her blood pressure dropped abruptly from 140/70 mmHg to 70/50 mmHg. She opened her eyes in response to calling her name, but quickly lost consciousness. Despite the immediate *iv* injection of methoxamine (10 mg total) and norepinephrine (1 mg total), her blood pressure progressively fell to 50/40 mmHg and severe bradycardia (HR of 40 beats·min^{-1}) occurred. External cardiac massage was begun immediately, she was turned supine, the trachea intubated and the lungs ventilated with 100% oxygen. Epinephrine (3 mg total) was injected intravenously. Within ten minutes, heart beat (sinus tachycardia of 140 beats·min^{-1}) returned. Continuous dopamine (30 mg·hr^{-1}) and norepinephrine (1 mg·hr^{-1}) infusions were required to maintain blood pressure. Echocardiography performed at this point revealed right-ventricular dilatation and good left-ventricular wall motion. Although she moved her head and upper extremities, she was unresponsive and her breathing pattern was irregular. She was paralyzed with vecuronium, returned to the left lateral decubitus position and the operation was completed as quickly as possible.

To investigate the cause of the sudden cardiovascular collapse, an angiocardiography, coronary angiography (CAG) and pulmonary angiography (PAG) were performed immediately after the operation. The angiocardiography revealed good contractility of the left ventricle, mild right ventricular dilatation, and no septal defects or patent foramen ovale. The CAG demonstrated normal coronary perfusion, and the PAG revealed no remarkable embolism and mild pulmonary hypertension (30–40/25 mmHg). Electrocardiography showed sinus tachycardia (140–150 beats·min^{-1}) with right bundle branch block, which had not been present preoperatively.

She was transferred to the intensive care unit where she was paralyzed and ventilated to maintain oxygenation and to stabilize vital signs. Hence her level of consciousness could not be assessed. Her pupils showed isocoria with normal right reflex. The next day, she moved her legs in response to painful stimuli but did not regain consciousness. The patient Glasgow coma scale (GCS) score was 5 and anisocoria (right < left) with no light reflex was noted. Her breathing pattern was still irregular. The chest radiograph showed bilateral diffuse pulmonary infiltrates. She developed high-grade fever (38–39°C), thrombocytopenia (platelet count 55,000·mm^{-3}), anemia (hemoglobin 8.1 g·dL^{-1}), and oliguria. Opthalmoscopy revealed petechial hemorrhages. Electroencephalography showed diffuse slow wave activity. A MRI scan of the head performed 20 hr after the operation revealed multiple high intensity signals throughout the white matter, the basal ganglia, the cerebellum, and the brain stem (Figure 1). The patient developed petechial rash on the trunk on postoperative day three. Her cardiorespiratory status, thrombocytopenia, and renal function improved over the next week. She underwent a tracheostomy and was weaned from the ventilator. She occasionally opened her eyes
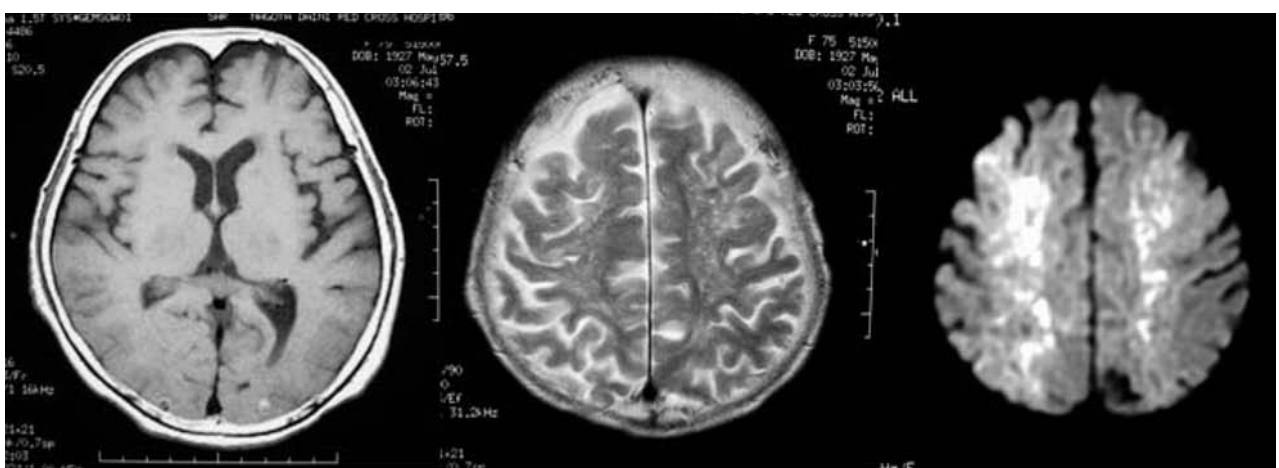
FIGURE 1 Magnetic resonance imaging (MRI) scans performed 20 hr after surgery. T2-weighted MRI (left) showing no abnormalities. T2-weighted MRI (middle) and diffusion-weighted MRI (right) showing spotted high-intensity lesions in the white matter.
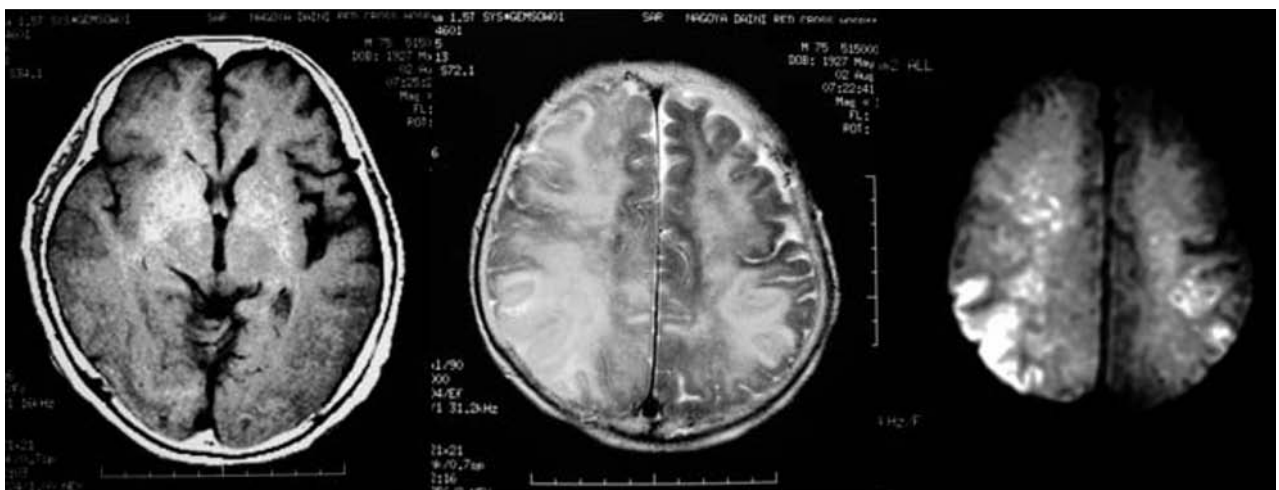

FIGURE 2 Magnetic resonance imaging (MRI) scans performed eight days after surgery. T1-weighted MRI (left) showing high-intensity lesions in the white matter conspicuously in the internal capsules, suggesting petechial hemorrhage. T2-weighted MRI (middle) showing fused and large macular high-intensity lesions in the white matter and right hemisphere edema. Diffusion-weighted MRI (right) showing high-intensity lesions in the white matter and the right parietooccipital cortex, suggesting further ischemic lesions.

spontaneously, but did not follow commands (GCS = 9). Her pupils showed isocoria and normal light reflex. A repeat MRI performed on postoperative day eight showed multiple hemorrhagic infarctions in both hemispheres and the brain stem, fused large macular high-intensity lesions in the white matter, and marked edema in the right hemisphere (Figure 2). A computed tomography scan performed on postoperative day 14 showed multiple small low density area in the white matter. The patient remained in a persistent vegetative state. A MRI scan performed 50 days after surgery showed persistent high-intensity lesions (Figure 3).

Discussion

PFE is a serious complication of both long bone fractures and orthopedic surgery with intramedullary manipulation. The latter, as it is associated with bone cement, is known as bone cement implantation syn-
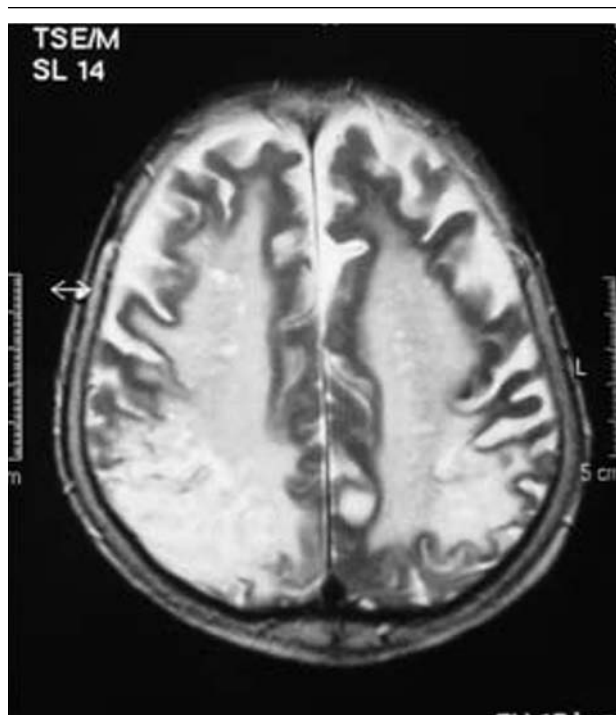
FIGURE 3 T2-weighted magnetic resonance imaging scan performed 50 days after surgery and showing persistent high-intensity lesions.

drome.[2,14,17] It is thought that high intramedullary pressures during insertion of a cemented prosthesis cause intravasation of bone marrow contents.[13–15] Emboli have been demonstrated in the femoral vein[18] and in the right heart[17] during insertion of the prosthesis, although most patients tolerate the embolic load and compensate for the increased right ventricular afterload. However, embolization of the lungs with bone marrow contents can cause intraoperative hypotension, hypoxemia, cardiac arrest and death.[1–8] These fat emboli can traverse the pulmonary vasculature and cause brain embolization,[16] which causes postoperative coma,[9–12] and may underlie postoperative cognitive dysfunction.[1,19] Brain embolization has been demonstrated in as much as 40 to 60% of patients during total joint arthroplasty by the use of transcranial Doppler ultrasonography.[19,20]

In the patient we describe, massive fat emboli seem to have occurred, resulting in acute right heart failure and cardiac arrest, based on the fact that it happened shortly after insertion of the cemented prosthesis. It seems likely that during and after resuscitation the fat globules passed through the pulmonary vasculature, entered the systemic circulation, and embolized to the brain, since angiocardiography showed neither an intracardiac shunt nor a patent foramen ovale. The PAG performed immediately after the operation failed to show any emboli in the major pulmonary arteries. This is probably because the fat globules occupied arterioles and capillaries and caused microembolization.[7]

In our patient the diagnosis of PFE was made on the basis of clinical findings and MRI appearance characteristic of PFE. Takahashi *et al.* characterized the lesions after cerebral fat embolism as high intensity signals in the deep white matter in T2-weighted images (T2WI), and graded them into four categories (grade 0: normal to grade 3: the most severe form) according to the severity of brain lesions. This case was first grade 1, deteriorated to grade 3 within a week and persisted for 50 days. This is consistent with the patient's sustained neurological deficit. Takahashi *et al.* showed that resolution of the high intensity MRI lesions parallelled neurologic recovery.[21] The spotted high intensity lesions in T2WI and diffuse-WI (DWI) in the acute phase in this case seem to represent acute focal perivascular edema. With time, petechial hemorrhage and hemorrhagic infarcts seem to have occurred, which is conspicuous in the internal capsules as shown by Kamenar[22] (Figure 2). At the same time, diffuse ischemic injury in the deep white matter seems to have occurred (Figure 2), as well as cortical damage in the right parietooccipital region, possibly associated with hypoperfusion due to the right hemisphere edema. The persisting diffuse white matter lesion in the chronic phase may represent diffuse white matter degeneration or demyelination (Figure 3).

The poor neurological outcome was more likely due to the severe cerebral fat embolism than to hypoxic encephalopathy associated with cardiac arrest, since cardiopulmonary resuscitation was started immediately and was rapidly effective. Fat embolism can lead to a persistent vegetative state without hemodynamic deterioration.[21] Massive brain fat emboli can occur as soon as four hours after surgery even in the absence of a patent foramen ovale.[1] In support of our hypothesis, the MRI showed numerous spotted lesions spreading throughout the white matter, the basal ganglia, the cerebellum and the brain stem, which were compatible with the presence of serious brain damage as shown by Takahashi *et al.*[21] In contrast, the characteristic MRI appearance of hypoxic encephalopathy includes early changes in cortex and basal ganglia, delayed changes in the white matter, and cortical laminar necrosis in the chronic phase.[23,24] This is thought to be consistent with the selective vulnerability principle; that is, gray matter is more vulnerable to global ischemia and anoxia than white matter. The delayed white matter degeneration in hypoxic encephalopathy

is considered Wallerian degeneration. Recently it was reported that white matter injury can occur in the acute phase in hypoxic encephalopathy,[25] which, however, showed diffuse white matter injury in DWI, different from the spotty lesions seen in this patient.

Most patients with coma secondary to PFE have been reported to recover fully, or almost, although all of them were relatively young (16–37 yr old).[9–12] The possible factors responsible for the poor outcome in this case are the concomitant cardiac arrest, the patient's advanced age and, possibly, the large amount of fat which embolized a large area of the brain.

We conclude that when PFE is suspected, serial MRI scans of the brain should be performed to make an early diagnosis of cerebral fat embolism as well as to evaluate the potential severity of brain damage.

References

1 *Colonna DM, Kilgus D, Brown W, Challa V, Stump DA, Moody DM.* Acute brain fat embolization occurring after total hip arthroplasty in the absence of a patent foramen ovale. Anesthesiology 2002; 96: 1027–9.

2 *Fallon KM, Fuller JG, Morley-Forster P.* Fat embolization and fatal cardiac arrest during hip arthroplasty with methylmethacrylate. Can J Anesth 2001; 48: 626–9.

3 *Pietak S, Holmes J, Matthews R, Petrasek A, Porter B.* Cardiovascular collapse after femoral prosthesis surgery for acute hip fracture. Can J Anaesth 1997; 44: 198–201.

4 *Sevitt S.* Fat embolism in patients with fractured hips. Br Med J 1972; 2: 257–62.

5 *Gresham GA, Kuczynski A, Rosborough D.* Fatal fat embolism following replacement arthroplasty for transcervical fractures of femur. Br Med J 1971; 2: 617–9.

6 *Dandy DJ.* Fat embolism following prosthetic replacement of the femoral head. Injury 1971; 3: 85–8.

7 *Orsini EC, Richards RR, Mulen JM.* Fatal fat embolism during cemented total knee arthroplasty: a case report. Can J Surg 1986; 29: 385–6.

8 *Patterson BM, Healey JH, Cornell CN, Sharrock NE.* Cardiac arrest during hip arthroplasty with a cemented long-stem component. J Bone Joint Surg 1991; 73: 271–7.

9 *Dive AM, Dubois PE, Ide C, Bulpa PA, Broka SM, Installe E.* Paradoxical cerebral fat embolism: an unusual cause of persistent unconsciousness after orthopedic surgery. Anesthesiology 2002; 96: 1029–31.

10 *Byrick RJ, Korley RE, McKee MD, Schemitsch EH.* Prolonged coma after unreamed, locked nailing of femoral shaft fracture. Anesthesiology 2001; 94: 163–5.

11 *Ozelsel TJ, Hein HA, Marcel RJ, Rathjen KW, Ramsay MA, Jackson RW.* Delayed neurological deficit after total hip arthroplasty. Anesth Analg 1998; 87: 1209–10.

12 *Satoh H, Kurisu K, Ohtani M, et al.* Cerebral fat embolism studied by magnetic resonance imaging, transcranial Doppler sonography, and single photon emission computed tomography: case report. J Trauma 1997; 43: 345–8.

13 *Schemitsch EH, Turchin DC, Anderson GI, Byrick RJ, Mullen JB, Richards RR.* Pulmonary and systemic fat embolization after medullary canal pressurization: a hemodynamic and histologic investigation in the dog. J Trauma 1998; 45: 738–42.

14 *Ereth MH, Weber JG, Abel MD, et al.* Cemented versus noncemented total hip arthroplasty: embolism – hemodynamics, and intrapulmonary shunting. Mayo Clin Proc 1992; 67: 1066–74.

15 *Orsini EC, Byrick RJ, Mullen JB, Kay JC, Waddell JP.* Cardiopulmonary function and pulmonary microemboli during arthroplasty using cemented or non-cemented components. J Bone Joint Surg 1987; 69: 822–32.

16 *Byrick RJ, Mullen JB, Mazer CD, Guest CB.* Transpulmonary systemic fat embolism. Studies in mongrel dogs after cemented arthroplasty. Am J Respir Crit Care Med 1994; 150: 1416–22.

17 *Urban MK, Sheppard R, Gordon MA, Urquhart BL.* Right ventricular function during revision total hip arthroplasty. Anesth Analg 1996; 82: 1225–9.

18 *Herndon JH, Bechtol CO, Crickenberger DP.* Fat embolism during total hip replacement. J Bone Joint Surg 1974; 56: 1350–62

19 *Edmonds CR, Barbut D, Hager D, Sharrock NE.* Intraoperative cerebral arterial embolization during total hip arthroplasty. Anesthesiology 2000; 93: 315–8.

20 *Sulek CA, Davies LK, Enneking FK, Gearen PA, Lobato EB.* Cerebral microembolism diagnosed by transcranial Doppler during total knee arthroplasty. Correlation with transesophageal echocardiography. Anesthesiology 1999; 91: 672–6.

21 *Takahashi M, Suzuki R, Osakabe Y, et al.* Magnetic resonance imaging findings in cerebral fat embolism: correlation with clinical manifestations. J Trauma 1999; 46: 324–7.

22 *Kamenar E, Burger PC.* Cerebral fat embolism: a neuropathological study of a microembolic state. Stroke 1980; 11: 477–84.

23 *Takahashi S, Higano S, Ishii K, et al.* Hypoxic brain damage: cortical laminar necrosis and delayed changes in white matter at sequential MR imaging. Radiology 1993; 189: 449–56.

24 *Arbelaez A, Castillo M, Mukherji SK.* Diffusion-weighted MR imaging of global cerebral anoxia. AJNR 1999; 20: 999–1007.

25 *Chalela JA, Wolf RL, Maldjian JA, Kasner SE.* MRI identification of early white matter injury in anoxic-ischemic encephalopathy. Neurology 2001; 56: 481–5.