Reversible Catastrophic Fat Embolism: A Case Report

Kethy Jules-Elysee, MD • Thomas P. Sculco, MD

Published online: 8 March 2006
© Hospital for Special Surgery 2006

A case of near-cardiac arrest preceded by acute pulmonary hypertension during cementing of the femoral component of total hip replacement is presented. Treatment consisted of both epinephrine and nitroglycerin with normalization of hemodynamics within 1 h of the event. This is an unusual case where very rapid reversal of near-fatal profound pulmonary hypertension is seen without sequelae postoperatively. We are unaware of such cases in the literature. Given the setting in which it occurred and its relationship to cementing of the prosthesis, we believe this was most likely due to fat embolism syndrome.

Case report

A 71-year-old woman with a history of hypertension, glaucoma, and hypercholesterolemia underwent revision of a total hip replacement. Her anesthesia consisted of a combined spinal epidural upon which she received 15 mg of 0.5% bupivacaine with the spinal. The epidural catheter was not dosed. She was initially sedated with 9 mg midazolam and 50 mcg fentanyl. Sedation was maintained with a propofol drip at $30 \text{ mcg kg}^{-1} \text{ h}^{-1}$ during surgery. Hypotensive anesthesia as previously described was achieved with a blood pressure (BP) of 80–85/55 mmHg and heart rate in the 60 s bpm [1]. Her pulmonary artery pressure (PAP) was 22–25/7–10 mmHg until cementing of the femoral prosthesis when it rapidly increased to 70/35 mmHg while her BP dropped to almost 0 (Fig. 1). She was

immediately given 1 mg epinephrine followed by 20 μg nitroglycerin and her systemic pressure responded rapidly to 170/80 mmHg. The length of the femoral prosthesis was 250 mm. A 16-mm cement restrictor was inserted prior to cementing. The femoral shaft was not vented with a catheter. A pressurizing injection syringe was used to inject the femoral canal with three packets of methylmethacrylate cement. Within 1 h of the event, her PAP decreased to 27/13 mmHg, with a BP of 120/50 mmHg, and heart rate of 90 bpm. Her oxygen saturation was 100%. All other laboratory parameters including platelet count were normal. There was no change in her mental status. She was observed overnight in the recovery room where she remained hemodynamically stable. Chest radiograph or spiral CT scan were not performed because the patient was asymptomatic with normal arterial blood gas. The patient was subsequently discharged after an uneventful hospital course.

Discussion

This case is very interesting because the patient had an uneventful and full recovery postoperatively in spite of evidence of a near-fatal functional electrical stimulation (FES) occurring intraoperatively. Patterson et al. [2] reported seven cases of cardiac arrest during hip arthroplasty. Hypotension was accompanied by an increase in pulmonary pressure occurring either during the cementing phase or with hip relocation. Four of the seven died either in the operating room, or within 1 h of the end of surgery. The other three required several days of intensive care to recover from the insult. Fallon et al. also reported a case of intraoperative cardiac arrest during hip arthroplasty where the patient subsequently died 18 h postoperatively [3].

The risk factors for FES include advanced age, osteoporosis, long-stem femoral component, several packets of methylmethacrylate (three or more), metastatic

K. Jules-Elysee, MD (✉)
Department of Anesthesiology,
Hospital for Special Surgery,
535 East 70th Street, New York, NY 10021, USA
e-mail: JulesElyseeK@hss.edu

T. P. Sculco, MD
Department of Orthopaedic Surgery,
Hospital for Special Surgery,
535 East 70th Street, New York, NY 10021, USA

A = Insertion of prosthesis into the femoral canal

Fig. 1. Intraoperative tracing of arterial and pulmonary artery pressure during surgery

disease, and collagen vascular disorders [2, 4]. The manifestations acutely include a rise in pulmonary arterial pressure, and pulmonary vascular resistance [5]. This may be accompanied by an acute drop in blood pressure along with hypoxemia and cardiac arrest [6]. If the patient survives the initial insult, FES usually evolves into a second phase characterized by pulmonary dysfunction. This dysfunction may range from mild hypoxemia to overt acute respiratory distress syndrome. Furthermore, mental status changes varying from mild confusion to coma, and petechiae may also occur [7–9]. Risk factors in our patient included a long stem femoral component (usual length used is 119 mm), and three packets of cement. On the average, two packets of cement are used in the majority of patients.

Although our patient had all the initial signs of FES including cardiovascular collapse in the setting of an acute rise of PAP during cementing of the femoral component, she had full recovery of normal hemodynamics within 1 h of the event. Her hospital course was also uneventful. What allowed such rapid resolution of her symptoms without clinical sequelae is unclear. In dogs, intravascular fat has been shown to move through the pulmonary vasculature over the first 3 h after bilateral cemented arthroplasty [10]. Consistent with this theory is that fat has been noted to occur in the cerebral circulation during cementing of total hip arthroplasty [11]. In our patient, acute mechanical obstruction of the pulmonary vasculature by fat decreased over time, probably as a result of compressible fat moving through the lung.

The rapid resolution of the pulmonary artery hypertension and uncomplicated postoperative course in this patient could be attributable to several factors. First, the immediate administration of intravenous epinephrine resulted in a rapid resuscitation without the need for external chest massage or controlled ventilation. Second, nitroglycerine was given in an attempt to relieve the acute pulmonary hypertension. Its use has been advocated for the relief of pulmonary hypertension [12]. Nitroglycerin is known to convert to nitric oxide (NO), leading to vasodilatation [13]. Byrick et al. [10] has suggested that the vasoconstriction and inflammatory changes occurring in the later phase of

FES may be suppressed by NO. Fahmy et al. [14] has shown improvement in elevated PAP occurring during orthopedic procedures with the use of inhaled nitric oxide. Whether nitroglycerin, which acts on vessels from its conversion to NO, helped in the resolution and eventual outcome of FES in this patient is of interest. In patients undergoing bilateral total knee replacement where the release of emboli intraoperatively is universal, it has been shown that pulmonary vasodilatation and recruitment of other capillary beds do occur at the time of tourniquet release [15, 16]. This may be the main mechanism by which patients are able to accommodate the burden of emboli. It is possible that nitroglycerin allowed our patient to vasodilate and recruit unused pulmonary capillaries, which led to the rapid resolution of the pulmonary hypertension. In an animal model, Byrick et al. [17] were unable to show any improvement from inhaled NO in the acute pulmonary hypertension or RV dilatation after cemented arthroplasty, although very large increases in pulmonary vascular resistance occurred. Further studies are needed to investigate the role of nitroglycerin or NO in the treatment of FES.

In conclusion, this is an unusual case of a patient who suffered near-cardiac arrest in the operating room at the time of prosthesis insertion during cementing, but had a rapid and full recovery immediately post insult without clinical sequelae. We were unable to find such cases in the literature with complete resolution of findings within an hour while using epinephrine and nitroglycerin as therapy. The possibility of FES should always be anticipated during operations such as revision arthroplasties and should be treated aggressively. The use of pulmonary artery catheter allowed for prompt diagnosis and treatment. Intravenous epinephrine and nitroglycerin appear to be an excellent treatment approach in these patients.

References

1. Sharrock NE, Salvati EA (1996) Hypotensive epidural anesthesia for total hip arthroplasty. *Acta Orthop Scand* 67:91–107
2. Patterson BM, Healy JH, Cornell CN, Sharrock NE (1991) Cardiac arrest during hip arthroplasty with a cemented long-stem component. *J Bone Jt Surg Am* 73-A:271–277
3. Fallon KM, Fuller JG, Morley-Forster P (2001) Fat embolization and fatal cardiac arrest during hip arthroplasty with methylmethacrylate. *Can J Anaesth* 48:626–629
4. Monto RR, Garcia J, Callaghan JJ (1990) Fatal fat embolism following total condylar knee arthroplasty. *J Arthroplast* 5:291–299
5. Orsini EC, Byrick RJ, Mullen JB, Kay JC, Waddell JP (1987) Cardiopulmonary function and pulmonary microemboli during arthroplasty using cemented or non-cemented components. The role of intramedullary pressure. *J Bone Jt Surg Am* 69-A:822–832
6. Duncan JA (1989) Intra-operative collapse or death related to the use acrylic cement in hip surgery. *Anaesthesia* 44:149–153
7. Carr JB, Hansen ST (1990) Fulminant fat embolism. *Orthopedics* 13:258–261
8. King MB, Harmon KR (1994) Unusual forms of pulmonary embolism. *Clin Chest Med* 15:561–580
9. Jacobson DM, Terrence CF, Reinmuth OM (1986) The neurologic manifestations of fat embolism. *Neurology* 36:847–851
10. Byrick RJ, Mullen JB, Murphy PM, Kay JC, Stewart TE, Edelist G (1999) Inhaled nitric oxide does not alter pulmonary

- or cardiac effects of fat embolism in dogs after cemented arthroplasty. *Can J Anaesth* 46:605–612
11. Edmonds CR, Barbut D, Hager D, Sharrock NE (2000) Intraoperative cerebral arterial embolization during total hip arthroplasty. *Anesthesiology* 93:315–318
 12. Jiang ZY, Costachescu T, Derouin M, Blaise G (2000) Treatment of pulmonary hypertension during surgery with nitric oxide and vasodilators. *Can J Anaesth* 47:552–555
 13. Frankish H (2002) Researchers solve the mystery of nitroglycerin's mechanism of action. *Lancet* 359:2006
 14. Fahmy NR, Head A, Nathan N, Zapol WM (1997) Inhaled nitric oxide reverses acute pulmonary hypertension and hypoxemia during orthopedic surgery. *Anesthesiology* 87:A1120
 15. Berman AT, Parmet JL, Harding SP, Israelite CL, Chandrasekaran K, Horrow JC, Singer R, Rosenberg H (1998) Emboli observed with use of transesophageal echocardiography immediately after tourniquet release during total knee arthroplasty with cement. *J Bone Jt Surg Am* 80-A:389–396
 16. Jules-Elysee K, Blanck TJ, Catravas JD, Chimento G, Miric A, Kahn R, Paroli L, Sculco T (2004) Angiotensin-converting enzyme activity: a novel way of assessing pulmonary changes during total knee arthroplasty. *Anesth Analg* 99:1018–1023
 17. Byrick RJ, Mullen JB, Mazer CD, Guest CB (1994) Transpulmonary systemic fat embolism. Studies in mongrel dogs after cemented arthroplasty. *Am J Respir Crit Care Med* 150:1416–1422