Fatal Cardiovascular Collapse during Insertion of Bone Cement and a Prosthesis: Two Case Reports

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Abstract: Over the last two decades, hip surgery with methylmethacrylic cement has become a popular and successful procedure. Improvements in the techniques of surgery and anesthesia have led to increased numbers of operations in the elderly. However, cardiovascular collapse is an occasional but well documented complication of the use of acrylic bone cement during hip surgery. Although the etiology has not yet been completely resolved, fat emboli and hypersensitivity to the methylmethacrylate monomer have been implicated. We have now documented two cases of fatal cardiovascular collapse during orthopedic surgery, presumably resulting from intraoperative fat embolism.

Key words: Bone cement, Cardiovascular collapse, Fat embolism

Case Reports

Case 1: An 86-year-old woman sustained a fracture of the left femoral neck. The patient was approximately 145 cm tall and weighed 40 kg. There was a history of severe dementia and left hemiplegia due to multiple cerebral infarction. Her family reported no history of angina pectoris, pulmonary disorder, or thromboembolic disease. However, preoperative arterial blood gas analysis showed an arterial PaO₂ of 63 mmHg, an arterial PaCO₂ of 29 mmHg, and pH of 7.515. Although total bilirubin was increased to 2.46 mg/dl, other laboratory data were within normal limits. Four days after admission to hospital she was scheduled for prosthetic replacement of the femoral head.

As the patient was excitable and exhibited severe dementia, we selected general anesthesia. After the application of routine monitors (blood pressure cuff, electrocardiogram, pulse oximeter), general anesthesia was induced with thiamylal and vecuronium given intravenously, and maintained with sevoflurane and nitrous oxide in oxygen. The patient was carefully turned and placed in the right decubitus position. Oxygen saturation (SpO₂) was maintained at 98%, blood pressure fluctuated between 150/86 and 96/54 mmHg, and pulse was 58–68 beats/min until the placement of the acrylic cement. Following the manual placement of the acrylic cement in the femoral shaft, the heart rate decreased from 58 to 38 beats/min, and blood pressure and SpO₂ could not be obtained. Although pure oxygen supplementation was given immediately, and ephedrine 20 mg and atropine 1 mg were given intravenously, blood pressure remained unobtainable. Isoproterenol, epinephrine, and norepinephrine were also administered. However, the patient did not respond to these drugs. The surgeons were urged to close the operative site as rapidly as possible, and cardio-
pulmonary resuscitation (CPR) was begun after the patient was placed in the supine position. Arterial blood gas analysis revealed \( \text{PaO}_2 \) 15.7 mmHg, \( \text{PaCO}_2 \) 41.8 mmHg, \( \text{pH} \) 7.234. Attempts at resuscitation were abandoned 70 min after the beginning of CPR. Intraoperative blood loss was estimated at 90 mL.

**Case 2:** An 88-year-old woman sustained a subcapital fracture of the right femoral neck. The patient was 140 cm tall and weighed 35 kg. There was a history of hypertension. Preoperative blood pressure fluctuated between 160/90 and 140/70 mmHg, and pulse was 60–75 beats/min. She had no history of angina pectoris, pulmonary disorder, or thromboembolic disease. However, preoperative arterial blood gas analysis showed an arterial \( \text{PaO}_2 \) of 52 mmHg, an arterial \( \text{PaCO}_2 \) of 30 mmHg and a \( \text{pH} \) of 7.515. She had anemia (hemoglobin 8.9 g/dl), but this was not corrected preoperatively with a blood transfusion. Other laboratory data were within normal limits. Fifteen days after admission to hospital she was scheduled for prosthetic replacement of the femoral head.

After application of the routine monitors, the patient was carefully turned and placed in the left decubitus position. A 23-gauge spinal needle was inserted into the subarachnoid space at the lumbar 2/3 interspace and a sensory level to Th-8 was obtained with 8.2 ml of 0.5% bupivacaine. Prior to spinal anesthesia, blood pressure was 145/75 mmHg, heart rate was 80 beats/min, and \( \text{SpO}_2 \) was 92%. After the patient was placed in the left decubitus position, \( \text{SpO}_2 \) decreased from 92% to 84% and blood pressure was 114/64 mmHg. Although the patient did not complain of dyspnea, oxygen supplementation was given immediately, using a face mask. Care was taken to replace blood loss with packed red cell and balanced electrolyte solution. The patient's condition was satisfactory until the beginning of medullary reaming. Approximately 5 minutes after the acrylic cement was placed in the femoral shaft with a cement gun, the patient's heart rate decreased from 75 to 40 beats/min. Blood pressure could not be obtained and she became severely disoriented. Ephedrine 10 mg was given intravenously. Bradycardia persisted, and blood pressure remained unobtainable. The surgeons were urged to close the operative site as rapidly as possible. The patient was intubated and manual ventilation of the lungs with pure oxygen was performed. Epinephrine 0.5 mg was given intravenously and epinephrine 1 mg was given by intratracheal tube. However, the electrocardiogram still indicated bradycardia (30 beats/min), which did not respond to epinephrine. Blood pressure was also unobtainable. Approximately 5 minutes after the bradycardia had begun, the patient was placed in the supine position, and external cardiac massage was commenced. Arterial and central vein catheters were inserted into the left femoral artery and vein, respectively, and blood gas analysis revealed \( \text{PaO}_2 \) 75 mmHg, \( \text{PaCO}_2 \) 39 mmHg, \( \text{pH} \) 7.16, and serum potassium 4.2 mEq/L. Although epinephrine 1 mg was given into the central vein catheter, the bradycardia became severer. Attempts at resuscitation were abandoned 60 min after the beginning of CPR. Intraoperative blood loss was estimated at 150 mL.

**DISCUSSION**

Cement toxicity was originally thought to be caused by myocardial depression and vasodilation, brought about by absorption of the liquid acrylic monomer.\(^{2,7,10}\) Certainly, the substance can cause these effects when injected into animals,\(^{12}\) but the blood levels measured during operation in humans are far too low to cause a systemic reaction.\(^{13}\)

It is now widely accepted that most complications that occur during hip surgeries are caused by fat embolism.\(^{14,15}\) The methylmethacrylic cement exacerbates embolism by sealing the femoral shaft, and this creates a very high intramedullary pressure (pressures
greater than 600 mmHg have been recorded\(^6\). This increased pressure forces more fat emboli into the circulation.

Clinically unsuspected hypoxemia is commonly seen after fractures\(^7\) and it has been postulated that this may be the result of fat embolus\(^8\). In healthy young males, it was reported that prolonged recumbency (10 days in bed) may have caused an increase in the alveolar-arterial oxygen partial pressure differences\(^9\). These factors may explain the circulatory collapse in the second patient.

However, there is another possible explanation for the circulatory collapse. Open venous sinuses in the femoral canal, as well as the patient being in the decubitus position (with hip above heart), favor air entrapment during this operation. Various methods have been utilized to reduce the likelihood of venous air embolism during hip surgery. Harvey and Smith\(^10\) reported a decrease from 16% to 0% when the femoral shaft was insufflated with CO\(_2\). Evans \textit{et al.}\(^20\) reduced the incidence from 100% to 33% when the femoral cement was inserted with a cement gun, rather than by manual instillation. Heinrich \textit{et al.}\(^21\) reduced the incidence from 92% to 30% with the placement of a venting hole in the femoral shaft. In our second patient, we did not vent the medullary cavity of the femur with a catheter or with drillholes in the femoral shaft, and the femoral cement was inserted with a cement gun, so that the volume of air within the femoral medullary cavity was limited. Therefore, the possibility of air embolism as a main cause of cardiac collapse in this patient seems to be low.

Barron\(^22\) and Sherman \textit{et al.}\(^23\) suggested that the most important surgical considerations in hip replacement were removal of the products of medullary reaming by lavage or aspiration, and venting of the shaft to lower intramedullary pressure. In our two patients surgeons took great care with medullary reaming and did not insert the acrylic cement into the femoral medullary cavity until it was firm enough to be conventionally handled, so as to diminish absorption of free acrylic monomers into the general circulation. Moreover, in the second patient, the femoral cement was inserted with a cement gun. However, we were unable to prevent cardiovascular collapse in these two patients.

In summary, we had the experience of fatal cardiovascular collapse during orthopedic surgery, presumably resulting from intraoperative fat embolism. We believe that the orthopedic surgeon and the anesthetist should always be aware of this potentially life-threatening complication when using press-fit femoral components.

**References**

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