General Anesthesia

Fat embolization and fatal cardiac arrest during hip arthroplasty with methylmethacrylate

[Embolie graisseuse et arrêt cardiaque fatal pendant l'arthroplastie de la hanche réalisée avec du méthyle méthacrylate]

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Purpose: This case report describes a cardiac arrest during a cemented hip arthroplasty procedure. Hemodynamic instability during methylmethacrylate use in arthroplasty surgery can be explained by fat embolization rather than the inherent toxicity of the monomer.

Clinical features: A 78-yr-old woman required a cemented hemiarthroplasty for a pathologic left subcapital fracture. The patient's past medical history included stable angina, diet-controlled type II diabetes and metastatic breast cancer. During the cementing of the canal and insertion of the femoral prosthesis, desaturation, hypotension and cardiac arrest occurred. The patient underwent a successful intraoperative resuscitation and was transferred to the intensive care unit where she subsequently developed disseminated intravascular coagulopathy. The patient died 24 hr later and autopsy confirmed the cause of death as fat embolization.

Conclusion: The deleterious cardiovascular effects of methylmethacrylate have been discussed in the literature. However, clinical evidence supports fat embolization during arthroplasty surgery as a greater determinant of hemodynamic compromise. Surgical precautions are paramount in minimizing the sequelae of Bone Implantation Syndrome and anesthetic treatment consists of supportive care.

Objectif : *Rapporter un arrêt cardiaque survenu pendant l'arthroplastie de la hanche cimentée. L'instabilité hémodynamique notée pendant l'utilisation de méthyle méthacrylate pour une arthroplastie peut être secondaire à une embolie graisseuse plutôt qu'à la toxicité inhérente au monomère.*

Éléments cliniques : *Une femme de 78 ans devait subir une hémiarthroplastie cimentée pour une fracture sous-capitale gauche. L'histoire médicale de la patiente indiquait une angine stable, un diabète de type II sous contrôle diététique et un cancer du sein métastatique. Pendant la cimentation de la fracture et l'insertion de la prothèse fémorale, une désaturation, une hypotension et un arrêt cardiaque sont survenus. La patiente a été réanimée avec succès et dirigée à l'unité des soins intensifs. Une coagulopathie intravasculaire disséminée s'est ensuite développée et la patiente est décédée 24 h plus tard. L'autopsie a confirmé que l'embolie graisseuse avait causé le décès.*

Conclusion : *Les effets cardio-vasculaires nocifs du méthyle méthacrylate sont connus. Toutefois, des preuves cliniques démontrent que l'embolie graisseuse pendant l'arthroplastie constitue un facteur déterminant, plus important, des altérations hémodynamiques. Il est essentiel de prendre toutes les précautions chirurgicales pour réduire les séquelles du syndrome de l'implant intra-osseux. Le traitement anesthésique est essentiellement un traitement de soutien.*

HIP fractures in the elderly are associated with a high rate of morbidity and mortality. The factors which significantly influence mortality are advanced age, pathological fractures, postoperative chest infections, dementia and wound infection.[1,2] Hip fracture repair may include the use of methylmethacrylate monomer as a cementing agent. It has been associated with hypotension and hypoxia when employed intraoperatively for arthroplasty surgery.[3,4,5] The toxic effect of the monomer on the cardiovascular system is unproven and more recently the importance of intraoperative fat embolization during cement insertion has been

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shown.[4,6,7] The following case report describes a cardiac arrest in a patient undergoing a bipolar-cemented hemiarthroplasty. This report provides further clinical evidence of the risks and consequences of fat embolization during hip arthroplasty surgery.

Case report

A 78-yr-old female presented to the operating room with a three-day history of a pathologic left hip fracture. Her medical history consisted of breast cancer with metastatic spread to the spine and femur, stable angina, hypertension and type II diabetes. Current medications were nifedipine and acetaminophen with codeine and there were no known drug allergies. Previous anesthetics for mastectomy and cholecystectomy were uneventful. The patient's physical examination revealed a blood pressure of 140/90 mmHg, heart rate 90 and regular, axillary temperature of 36C, respiratory rate of 12 and oxyhemoglobin saturation of 99% on room air. The patient's mental status was alert and coherent. Respiratory examination revealed clear bilateral breath sounds and the cardiac examination was normal with no extra heart sounds or murmurs. The remainder of the physical examination was unremarkable. Preliminary blood analyses included a normal arterial blood gas. The chest radiograph, complete blood count, serum electrolytes, urea and creatinine were also within normal limits.

After three minutes of pre-oxygenation with standard monitors consisting of electrocardiogram, oxygen saturation and non-invasive blood pressure cuff applied, the patient received a rapid sequence induction with 15 μg sufentanil, 140 mg propofol, and 100 mg succinylcholine. Anesthesia was maintained by 70% nitrous oxide and 0.8% isoflurane in oxygen. After ascertaining that the depolarizing blockade had worn off, 50 mg of rocuronium were administered. She was then placed in the right lateral decubitus position for surgery.

During the reaming and rasping of the femoral canal the patient's oxyhemoglobin saturation decreased to 89%, blood pressure declined to 86/60 mmHg and an axillary temperature of 38.1°C was noted. This was successfully treated with administration of 100% oxygen, a 1000 mL *iv* bolus of Ringer's Lactate and two doses of phenylephrine 100 μg. Once the patient was stabilized with a blood pressure of 120/80 mmHg, sinus heart rate of 80 and oxyhemoglobin saturation of 96%, anesthesia was continued with isoflurane and 100% oxygen to maintain oxygenation saturation of 95%. The surgeons proceeded to cement the canal. The femur was not vented. Upon insertion of the prosthesis, the patient's blood pressure decreased to 40/20 mmHg and the heart rate decreased progressively resulting in a brief period of asystole. The patient was turned supine, 100% oxygen was administered, cardiopulmonary resuscitation (CPR) commenced and a total 3 mg of epinephrine was injected *iv*. Within five minutes sinus tachycardia at 120 beats·min^{-1} and a blood pressure of 103/60 mmHg was obtained. A 20 gauge right radial arterial line was placed, and the patient was returned to the right lateral decubitus position to facilitate expeditious surgical closure. Within six minutes pulseless electrical activity occurred. CPR and *iv* epinephrine 2 mg in total were administered. A blood pressure of 177/128 mmHg with sinus tachycardia at 105 beats·min^{-1} was achieved within seven minutes. A 9-French percutaneous sheath was inserted into the right internal jugular vein as the surgeons closed the surgical site.

At this time the patient developed pink frothy sputum and bilateral chest crackles. This was treated with furosemide 80 mg, calcium 1 g and milrinone 5 mg. As the signs of pulmonary edema resolved, a pulmonary artery catheter was inserted which yielded the following measurements: central venous pressure 9 mmHg, pulmonary catheter wedge pressure 12 mmHg and cardiac index 2.4 L·min^{-1}·m^2. The CVP was lower than expected in a situation of massive embolization, possibly due to the administration of furosemide and milrinone prior to insertion of the pulmonary artery catheter. The CVP remained relatively low, so it was decided not to use a milrinone infusion. The urinary catheter measured less than 10 mL of urine output throughout the entire case. Subsequently, persistent hypotension developed, requiring a continuous infusion of norepinephrine to maintain a mean arterial pressure of 60 mmHg. The patient was transferred to the intensive care unit with oxyhemoglobin saturations of 95–100% (F_1O_2=1.0), heart rate of 150 beats·min^{-1} and blood pressure of 90/50 mmHg. Within one hour, extreme swelling of the surgical area and oozing from *iv* catheter sites was observed. Laboratory measurements confirmed the presence of disseminated intravascular coagulopathy (DIC; international normalized ratio 4.3, partial thromboplastin time >150, fibrinogen 1.09: normal ranges 0.9–1.1, 20–38, >2.0 respectively). The hemoglobin concentration declined from 98 g·L^{-1} (postoperatively) to 33 g·L^{-1} and measured lactate concentration was 10.9 (normal range 0.5–2.2).

Eight units of packed red blood cells, eight units of fresh frozen plasma, ten units of platelets and ten units of cryoprecipitate were transfused in an effort to treat the severe coagulopathy. The patient continued to have persistent bleeding with continued laboratory evidence of DIC, worsening pulmonary edema and renal failure.

Fatal cardiac arrest with irreversible pulseless electrical activity occurred 18 hr postoperatively. The autopsy findings revealed multiple fat vacuoles and hemopoeitic elements in the pulmonary vasculature and confirmed the cause of death as fat embolization. The autopsy report indicated massive amounts of pulmonary fat embolic material, characterized as "the largest amount the pathologist had ever seen". There was no evidence of tumour embolus.

Conclusion

Bone Implantation Syndrome is a risk whenever a femoral prosthesis is inserted. This case serves to highlight the presentation and potential severity of the syndrome. The authors wish to raise the possibility that, in the case of a pathologic fracture with potential alteration of femoral vascular architecture, the risk of marrow embolization may be increased.

This patient exhibited many of the classical features of Bone Implantation Syndrome, which consists of bronchoconstriction, hypoxemia, hypotension and pulmonary hypertension. Rarely, it can progress to cardiac arrest.[6] The two predominant theories that have been advanced to explain this syndrome are methylmethacrylate toxicity and pulmonary fat embolization.[7] The mechanism of methylmethacrylate toxicity is unproven. Although the toxic effect of methylmethacrylate was at one time widely considered to be the main contributor to hemodynamic instability during arthroplasty surgery, this has not been supported by animal studies. Greater than 30 times the level of methylmethacrylate typically employed in human arthroplasty is required to produce changes in cardiopulmonary parameters.[5,8,9]

Orsini *et al.* compared the effects of inert bone wax, cemented and non-cemented arthroplasty using a dog model. The use of either cement or inert bone wax produced significantly more cardiopulmonary dysfunction, and the amount of pulmonary fat emboli was ten times greater at autopsy.[3] Assay determinations of methylmethacrylate levels were undetectable. They concluded that the pressurizing effect of bone cement or inert bone wax produces high intramedullary pressures and, as a result, bone marrow is forced into the circulation at the time of cement and prosthesis insertion, causing the observed cardiopulmonary changes.[3]

In human studies, venous embolization has been detected by transesophageal echocardiography (TEE) during cemented total hip arthroplasty.[6,9,10] Animal and human studies have demonstrated embolism of intramedullary debris as a consistent finding during total hip arthroplasty surgery. Several factors determine the severity of cardiopulmonary effects. The degree of embolization visualized by TEE, the compliance of the pulmonary vasculature, cardiovascular status before embolism and humoral reflexes are major determinants of fat embolism severity.[6]

This case report illustrates a fulminant form of fat embolization syndrome. The presence of hypoxemia, pulmonary edema, tachycardia, hyperthermia and rapidly developing anemia and thrombocytopenia are diagnostic for this condition. In this case, the presence of the secondary tumour in the femur may have altered the medullary vasculature of the femur, increasing the risk of escape of medullary contents. The patient's age, severe osteoporosis, undisturbed femoral canal and the use of a long-stem femoral component were additional risk factors for this event.[4] TEE might have been useful in the management, but was not readily available to the orthopedic operating rooms at the time this case occurred.

Surgical precautions are paramount in avoiding or minimizing Bone Implantation Syndrome. The avoidance of excessive cement pressurization, low viscosity cement, meticulous high-pressurized canal lavage and the use of a venting hole are effective techniques to minimize intramedullary canal pressures.[4,12] Anesthetic management of the Bone Implantation Syndrome necessitates supporting the cardiovascular system and treating a state of acute right heart failure. The first step includes administration of 100% inspired oxygen and aggressive volume support. Invasive hemodynamic monitoring should be instituted early, in light of the potential for severe pulmonary hypertension, impaired cardiac output and to guide inotropic support. Early placement of a pulmonary artery catheter may be needed in order to utilize selective pulmonary vasodilators and assess the effects of high PEEP levels in extreme circumstances.[a]

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