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The Intraoperative Hazard of Acrylic Bone Cement: Report of a Case

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A fatal cardiac arrest occurred following the experimental use of acrylic bone cement. At least nine cases of operative cardiac arrest (four successfully resuscitated) following the use of intramedullary methyl methacrylate have been reported in the British literature,[1–4] but a review of the American literature failed to reveal any such cases. The following is a case report of a patient who died following insertion of a Tronzo femoral prosthesis and bone cement and whose lungs, at autopsy, contained fat and bone marrow emboli and a yet-unidentified foreign material which may be acrylic emboli.

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REPORT OF A CASE

The patient, a 64-year-old obese woman in good general health, came to the hospital with a dislocated Austin-Moore prosthesis. Preoperatively, cardiorespiratory history and physical examination, and ECG, were normal; blood pressure was in the high-normal range, and roentgenogram of the chest revealed tortuosity of the aorta with calcification of the aortic arch.

After premedication with 2 ml Innovar, im, we injected a hypobaric solution of tetracaine via a subarachnoid catheter at the L3–4 interspace with the patient in the right lateral decubitus position with the head slightly down. The operation began at 9:25 AM and proceeded uneventfully for about 90 minutes. At 11:00 AM the acetabular prosthesis was cemented into the pelvis without incident. At 11:15 AM the surgeon forced the femoral prosthesis and bone cement into the femur. At this time the patient was alert and moved herself into a more comfortable position because of discomfort in the right shoulder. Two to three minutes later she groaned loudly for several seconds, became unresponsive and pulseless, but continued breathing. The ECG showed sinus bradycardia and QRS complexes of diminished amplitude. Ephedrine (20 mg, iv) was given

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immediately, and the following therapeutic maneuvers were tried: iv atropine, tracheal intubation and 100 per cent O_2 by assisted and then controlled ventilation, sodium bicarbonate and isoproterenol infusions. Electrical and complete mechanical cardiac arrest occurred about 20 minutes later. Other treatment consisted of external cardiac massage, defibrillation, external cardiac pacing and lidocaine, iv. The patient was pronounced dead at 12:25 PM.

The last injection of 0.15 per cent tetracaine had been at 10:50 AM, and the blood pressure and pulse were stable from 11:00 until the profound hypotension developed as described. Blood loss of 2,250 ml had been replaced with 1,000 ml of dextran 75, 1,000 ml of whole blood, and 1,000 ml of 5 per cent dextrose in half-strength saline solution. The patient had received a total of 20 mg of diazepam (Valium) intravenously, the last dose (5 mg) at 10:10 AM, and a total dose of 0.1 mg of fentanyl (Sublimaze)—the last 0.05 mg at 10:55 AM. During the entire operation she received O_2 (3 l/min) via nasal prongs. Clearly, neither anesthetic drugs nor sedatives can be incriminated.

At autopsy, we saw severe acute congestion and edema of the lungs, which weighed 1,550 g (about twice normal weight). Sections showed diffuse embolization of fat and other marrow elements in the pulmonary capillaries (demonstrated by oil red O stain). The lungs also contained dark brown, slightly birefringent foreign material in the small arterioles and capillaries. There were no fat emboli or hemorrhages in the brain and there was no evidence of acute myocardial infarction.

Discussion

Methyl methacrylate polymer is currently under evaluation in the United States for use in the semisolid state as a cement with orthopedic prosthetic replacements; it has many accepted uses, such as in dental prostheses and filling skull defects, when it is cured outside the body. The self-curing cement consists of a powder of polymerized methyl methacrylate and a liquid monomer which, when mixed together, begin polymerization of the liquid, binding together granules of polymer. When this "dough" becomes viscous, it is forced down into the canal of the bone along with the prosthesis. A transient fall in blood pressure within a minute or two of inserting acrylic cement into the medullary canal of the femur is often observed; usually, blood pressure returns to normal in 3–6 minutes without specific therapy.[5,6] In our experience with implantation of acrylic hip arthroplasties under hypobaric spinal anesthesia, hypotension has occurred in nine of 12 cases, with blood pressure decreases ranging from 10 to 30 torr (excluding the present case), with an average of less than 15 torr. The early peak in blood level and rapid clearance found in dogs showed that only surface monomer is available for absorption and, therefore, dosage depends on vascularity, surface area of acrylic paste in contact with tissue, and length of time between ingredient mixing and implantation.[7] Because of this, Charnley recommends both aeration of the mix during the two minutes of mixing and leaving the cement bolus undisturbed until its insertion in order to minimize the amount of free monomer. During installation of acrylic arthroplasties in six patients, Homsy and Tullos[7] found that monomer vapor due to monomer migration into the circulation was easily detected by smell in expired anesthesia gas. Its presence was confirmed by scintillation analysis. Maximum blood levels reached were about 1 mg/100 ml, which is similar to those found in dogs.

No canine morbidity was observed with levels below approximately 50 mg/100 ml, which occurred only after large iv doses. Respiratory arrest resulted when the monomer level rose to 125 mg/100 ml. A dose of this size given to a rabbit in our laboratory caused an initial abrupt fall in expired CO_2 and, shortly thereafter, apnea. Homsy and Tullos also found that the lungs of dogs given large iv doses of monomer and sacrificed at the completion of the experiment showed hemorrhage and edema at autopsy. Those animals allowed to survive for 30 days showed multiple small areas of pleural scarring, thought to represent a healing phase of pulmonary hemorrhage.

Fat and bone marrow elements, air, and methyl methacrylate embolism could explain the decrease in blood pressure and also the rapid onset after prosthesis insertion. Autopsies of patients dying immediately after impaction of the prosthesis into the femur have shown fat emboli in the lungs[8,9] and air and fat in pelvic veins, right heart, and coronary vessels.[4] Methyl methacrylate has not been demonstrated in the lungs of patients or dogs dying immediately after its use. Perhaps the dark brown, birefringent material found in

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this patient's lungs was methyl methacrylate polymer that was extruded out of the femur under the high pressure generated when the prosthesis and cement were forced into the medullary canal. The possibility remains that the death in this case was due to the effect of the methyl methacrylate polymer in addition to the effects of the monomer and the fat and marrow emboli.

An alpha-adrenergic pressor may be suitable to treat the hypotension, because a study in animals showed a pronounced vasodilatory action of the monomer, without myocardial depression.[10] Probably such a drug should have been used to treat this patient, although because of the presence of diffuse pulmonary embolization in addition to the monomer-induced hypotension, it seems unlikely that she had reversible hypotension. Since the reported incidence of severe hypotension appears to be rather small, it is questionable whether prophylactic pressors and/or volume expanders are warranted because of the complications that they may cause in the elderly patient. Although the anesthesiologist should be prepared to treat the hypotension and possible cardiac arrest that may follow the placement of the prosthesis with acrylic bone cement into bone, the orthopedic surgeon seems better able to avoid complications by minimizing both the amount of free monomer present as the cement is placed into the bone and the amount of pressure developed in the medullary canal.

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