A Cardiovascular Collapse during Cemented Total Hip Replacement in a Diabetic Patient

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Diabetes mellitus is often associated with cardiovascular abnormalities. Several case reports have suggested that the diabetic patient undergoing surgery may have a higher incidence of undesirable cardiovascular events than the general population[1].

Catastrophic cardiovascular collapse during cemented total hip replacement has been reported[2]. This has been attributed to vasodilating effects of the absorbed volatile monomer, to emboli forced into the circulation by the force of insertion of the prothesis[3].

In this report an intraoperative cardiac arrest recently experienced during the cemented total hip replacement in a patient with severe diabetes mellitus. A combined negative inotropic effect of bone cement and diabetic state has been proposed to be a major cause of sudden cardiac arrest shortly after placement of bone cement.

Report of a Case

A 56-yr-old woman with a subcapital fracture of the left femur, 141 cm and 38 kg, was admitted for left total hip replacement. The patient had mild hypertension and a 30-yr history of adult-onset type 2 diabetes mellitus and was in renal failure secondary to diabetic nephrosclerosis. The patient complained of postprandial fullness, occasional bouts of diarrhea, and orthostatic hypotension. Current medications included prazocin, furosemide, and NPH and regular insulin. Her blood pressure was 156/98 mmHg and heart rate was 88 beats·min^{-1}. Her chest roentgenogram showed slight cardiomegaly. Electrocardiogram showed previous inferior wall myocardial infarction and nonspecific ST-segment and T-wave abnormalities, but the patient denied remembering symptoms suggestive of angina pectoris. Hemoglobin was 9.2 g·dl^{-1}. Total protein was 6.7 g·dl^{-1}, with albumin 2.8 g·dl^{-1}. The serum creatinine and blood urea nitrogen were 2.7 mg·dl^{-1} and 48 mg·dl^{-1}. The serum potassium and glucose were 4.3 mEq·l^{-1} and 224 mg·dl^{-1}, respectively. An arterial blood sample obtained under room air showed pH, 7.38; Pa_{O_2}, 86 mmHg; and Pa_{CO_2}, 33 mmHg.

The patient received diazepam 10 mg and famotidine 20 mg p.o. approximately 2 h before the operation. Insulin was withheld. In the operating

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room, prior to induction of anesthesia, a 22-gauge cannula was inserted into the left radial artery for arterial blood pressure monitoring and blood sampling. An epidural catheter was inserted into the L2–3 interspace. Anesthesia was induced by thiamylal 200 mg IV and vecuronium bromide 5 mg IV was used to facilitate endotracheal intubation. Intermittent positive pressure ventilation was instituted. Anesthesia was maintained with nitrous oxide (60%) with oxygen and isoflurane (0.5–1%), and 1.5% lidocaine with 1:200,000 epinephrine was given through the epidural catheter. Continuous intraoperative monitoring included electrocardiogram, esophageal stethoscope, pulse oximetry (Spo_2), capnography ($P_{ET}CO_2$), arterial BP, and nasopharyngeal temperature.

After induction of anesthesia, the patient was hemodynamically stable and had no evidence of impaired gas exchange. No changes in $P_{ET}CO_2$, BP, or Spo_2 were noted with drilling of the intramedullary canals. Lactated Ringer's solution 750 ml and whole blood 400 ml, were given intravenously, when the estimated blood loss being 350 ml.

After a trial reduction with the prothesis of selected size, bone cement (Surgical Simplex Inc., USA) was mixed for about 2 min to the consistency of soft putty. This was then inserted into the shaft of the femur followed by the stem of the prothesis, which was tapped lightly into position. The head of the prothesis was reduced into the acetabulum after the cement had hardened.

A few minutes after the insertion of the bone cement, the blood pressure and pulse rate decreased rapidly from 160/100 to 78/52 mmHg and 72 to 30 beats·min^{-1}, respectively. The lungs were ventilated with 100% oxygen. Intravenous administration of ephedrine 15 mg and atropine 2.0 mg elicited no response. External cardiac massage was performed. However, the bradycardia progressed to asytole. Isoproterenol 0.01 mg and epinephrine 1 mg were given intravenously. Restroration of heart beat was achieved within 3 min. Arterial blood gas analysis showed Pa_{O_2} 548 mmHg, Pa_{CO_2} 43 mmHg, BE −9.4 mEq·l^{-1}, Hgb 7.9 g·dl^{-1}. ECG at this time showed sinus rhythm with occasional ventricular extrasystoles. Sodium bicarbonate 60 mEq was given intravenously. Systolic blood pressure increased to 100 mmHg in the immediate post-arrest period and remained at this level while the surgery was completed. The surgery was completed in 15 min after the cardiac arrest. Her chest X-ray obtained immediately after the operation showed no signs of pulmonary fat embolism. The pupils remained equal and reactive.

Spontaneous respiration was reestablished by intravenous atropine 0.5 mg and edrophonium 30 mg, but it was weak. Consciousness level was 8 (Glasgow coma scale: GCS).

In the recovery room, arterial blood gas values were within normal limits, and the electrocardiogram showed no evidence of myocardial ischemia.

She was mechanically ventilated for 2 days, and her consciousness level was 13 (GCS). During the 4th week she developed left ventricular failure and died on the 50th postoperative day.

Postmortem examinations were not performed.

Discussion

For nearly a decade after Charnley introduced the cemented total hip arthroplasty in 1961, episodes of transient hypotension[4], cardiac arrest[5], and sudden death[6] occurring minutes after insertion of the cement and prothesis were reported. Deburge and Guepar reported 14 patients with intraoperative "blood pressure collapse" resulting

in 5 cardiac arrests from their series of 292 patients[2]. Intraoperative mortality has ranged from 0.02 to 6.6 per cent[7].

Although the mechanism by which these hemodynamic changes are brought about is still not clear, methylmethacrylate monomer is a possible etiologic factor in such catastrophic cardiovascular reations during total hip arthroplasty[5]. Peebles et al. have shown that the monomer is a potent systemic vasodilator, and a direct negative cardiodepressant when injected intravenously[3]. In our case, her capacitance vessels were constricted preoperatively due to hypovolemic state, sudden vasodilation resulted in a significant decrease in arterial blood pressure after methylmethacrylate monomer was used.

The fact that chronic diabetes has a higher incidence and mortality from cardiac disease has been known for many years. During the perioperative period bradycardia, hypotension, and cardiopulmonary arrest have been reported among diabetics[1]. Cardiac disease in the diabetic is not due to atherosclerosis alone, but due to a combination of microangiopathy, macroangiopathy, autonomic neuropathy, and various other factors, which produce structural, functional, and biochemical alterations in the heart[8]. This disease also has been shown to be associated with a depression in myocardial function[9]. Therefore, hemodynamic instability is not well tolerated in this population[10].

A recent study demonstrated impaired cardiovascular homeostatic response to general anesthesia in diabetic patients with advanced autonomic neuropathy[11]. Page and Watkins postulated that death in diabetics may be due to abnormalities in the chemoreceptor pathways secondary to neuropathy[1]. The fact that this patient did not respond well to atropine and isoproterenol suggested that she had long-standing diabetes with autonomic neuropathy. The cardiovascular system, in the absence of sympathetic innervation, is less able to withstand hypoxia, blood loss, or toxic substances. Therefore, in this patient cardiovascular autonomic neuropathy may explain the cardiovascular collapse due to a toxic substance such as bone cement monomer.

In addition to the autonomic neuropathy, this patient had abnormal ST segments as well as cardiomegaly on admission. Diabetic cardiomyopathy may also be involved in the mechanism of her catastrophic cardiodepression.

In summary, an intraoperative cardiac arrest occurred during the cemented total hip replacement in the patient with severe diabetes mellitus. This episode may have been due to the depressant effects of bone cement monomer on myopathic heart in a patient of devoid of sympathetic compensation.

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