

Profound Hypotension immediately following Insertion of Methyl Methacrylate during Bipolar Endoprosthesis in a Patient with Long-term Levodopa-Treated Paralysis Agitans

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Insertion of methyl methacrylate polymer into newly reamed bony cavities has sometimes resulted in profound hypotension, cardiac arrest, or sudden death which are more common in patients with hemodynamic instability or hypovolemia.

In paralysis agitans (Parkinson's disease), dramatic worsening of the disease often occurs when another illness or trauma accompanies it. And it is possible that chronic medication with levodopa can cause the loss of ability to support blood pressure. So, it involves some risk to use methyl methacrylate in chronic levodopa-treated paralysis agitans.

We present a case of paralysis agitans who demonstrated profound hypotension immediately following insertion of methyl methacrylate polymer in spite of normovolemia and proper anesthetic management.

Key Words : *Hypotension, Methyl methacrylate, Paralysis agitans.*

INTRODUCTION

Paralysis agitans (Parkinson's disease) is an adult onset degenerative disease of the extrapyramidal system, characterized by the loss of dopaminergic fibers in the basal ganglia of the brain. As a result of depletion of dopamine in the basal ganglia, diminished inhibition of the extrapyramidal motor system and unopposed action of acetylcholine occur (Ngai, 1972; Roizen, 1990; Beal et al., 1991; Stoelting and Dierdorf, 1993).

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Exogenous administration of levodopa, the immediate precursor of dopamine, will increase the total body concentration of dopamine. In cases of anesthesia in paralysis agitans who have been treated with levodopa, it should be administered during the perioperative period because such treatment seems to decrease drooling, the possibility of difficult intubation and ventilation due to skeletal muscle rigidity, and the potential for aspiration (Calne, 1971; Pallis, 1971; Ngai, 1972; Hetherington and Rosenblatt, 1980; Stoelting and Dierdorf, 1993). Careful monitoring is required during the perioperative period because of the possibility of profound hypotension or cardiac arrest in such cases (Roizen, 1990; Stoelting and Dierdorf, 1993) and dramatic worsening of the disease coincided with another disease or trauma such as femoral

neck fracture(Diamond et al., 1987).

There have been several reports that profound hypotension immediately following insertion of methyl methacrylate has resulted in cardiac arrest or death(Michelinakis et al., 1971 ;Cohen and Smith, 1971 ;Herndon et al., 1974 ;Coventry et al., 1974 ;Sharrock and Savarese, 1990). Moreover, these complications are more common in patients with hemodynamic instability and hypovolemia(Sharrock and Savarese, 1990). So, the application of methyl methacrylate in patients with paralysis agitans who are continuously treated with levodopa can cause cardiac arrest or death.

We report a case of paralysis agitans who demonstrated profound hypotension immediately following insertion of methyl methacrylate polymer in spite of normovolemia and proper anesthetic management.

CASE REPORT

A 71-year-old, 62 kg, 168 cm male patient with a 10-year history of paralysis agitans and neurogenic bladder was scheduled for bipolar endoprosthesis of the right hip due to femoral neck fracture caused by a fall.

He had been receiving levodopa, lisuride, and amantadine for the treatment of paralysis agitans with gradual improvement except freezing. He had had several operations for hemorrhoids under spinal anesthesia without special complications.

His preoperative vital signs were stable. Laboratory studies were significant only for a blood urea nitrogen of 29.3 mg%. Chest X-ray showed no abnormalities. Electrocardiogram showed left anterior hemiblock. Three hundred milligrams levodopa and 0.3 mg lisuride three times a day were given orally until the night before the surgery.

In the operating room, the radial artery cannulation and a central venous catheterization were done in addition to a pulse oximeter and a capnogram(Nellcor® multi-function monitor N-1000, Nellcor Inc., California, USA). Because the patient was very irritable, we decided to perform general anesthesia. Anesthesia was induced with 5 mg diazepam and 125 mg thiopental intravenously followed by oxygen, nitrous oxide, enflurane, and 0.08 mg intravenous vecuronium for priming. Tracheal intubation was facilitated with 3 mg vecuronium. From the induction of anesthesia to the application of the methyl methacrylate, 400 ml of 10% pentastarch and 3,100

ml of lactated Ringer's solution were administered for maintaining central venous pressure, 7-11 cmH₂O and a low concentration of enflurane, 0-0.8 volume percent, were delivered.

About 2 min after application of the methyl methacrylate bone cement(BONELOC® vacuum pac, Biomet Ltd., Bridgend, UK), systolic blood pressure fell suddenly below 60 mmHg in spite of normal central venous pressure(Fig. 1). But, there were no significant changes in pulse rate, central venous pressure, oxygen saturation, hematocrit level, serum electrolytes, and arterial blood gas values except for a transient fall of the end-tidal carbon dioxide values. Blood pressure was gradually recovered within 10 minutes by the delivery of 100% oxygen with discontinuance of volatile anesthetic, rapid hydration, and 5 mg intravenous ephedrine.

The remainder of the anesthetic course was unremarkable. The surgery lasted about 4 hours with an estimated blood loss of 1,000 ml and a urine output of 700 ml. Fluid replacement consisted of 3,900 ml of lactated Ringer's solution, 500 ml of 10% pentastarch, and 1 unit of packed red blood cells(Fig. 1). At the completion of anesthesia, his recovery was uneventful and the trachea was extubated in the operating room.

During the postoperative course, 100-300 mg levodopa and 0.1-0.3 mg lisuride were medicated daily via oral route.

Oliguria, difficult voiding, paralytic ileus, and chronic ulcer bleeding from the rectum were transiently noted during postoperative course, but improved with the appropriate treatments such as diuretics, Foley catheterization, wheelchair ambulation, and transfusion.

The patient was discharged on postoperative day 47 without any complications and is under follow-up treatment for paralysis agitans at the outpatient department of internal medicine.

DISCUSSION

Profound hypotension immediately following application of cemented femoral prostheses sometimes has resulted in cardiac arrest or sudden death(Michelinakis et al., 1971 ;Cohen and Smith, 1971 ;Herndon et al., 1974 ;Coventry et al., 1974 ;Sharrock and Savarese, 1990). These complications are related to the use of bone cement because they are not seen with noncemented pros-

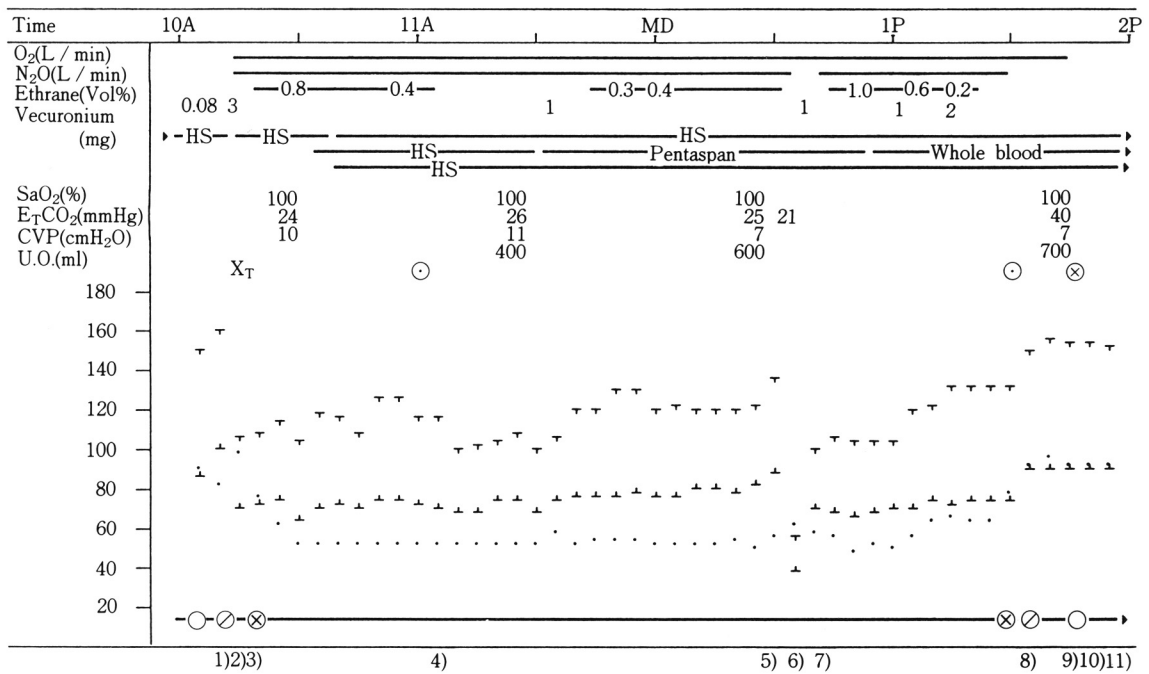


Fig. 1. The Anesthesia Record

X_T: Anesthesia start ⊙; Surgery start/end ⊗; Anesthesia end ⊖; Spontaneous respiration ⊗; Assisted respiration ⊕; Controlled respiration ⊗; Systolic blood pressure ⊕; Diastolic blood pressure ⊖; Pulse rate HS; Hartman's solution

- 1) Radial artery cannulation
- 2) diazepam 5mg, thiopental 125mg, vecuronium 0.08(priming)+3mg
- 3) Subclavian catheterization
- 4) CBC ; 11.5/32.9/225,000, ABGA ; 7.446-30-178-20(99.6%), Elect. ; 141-4.2-110
- 5) Bone cement application
- 6) ephedrine 5mg
- 7) CBC ; 10.1/29.1/212,000, ABGA ; 7.445-31-143-21(99.2%), Elect ; 145-4.3-107
- 8) pyridostigmine 4 Amp., glycopyrrolate 2 Amp.
- 9) Extubation
- 10) Eye opening
- 11) Transfer to ICU

theses(Freeman et al., 1983; Morscher and Dick, 1983). Its definitive mechanism is still unclear, but two possible explanations are (1) direct vasodilation and/or myocardial depression from methyl methacrylate due to absorption of the still liquid or vaporized monomer via the raw bony surface(Pahuja et al., 1974; Convery et al., 1975; Sharrock and Savarese, 1990) and (2) forced entry of air, fat, or bone marrow into the venous system with resultant pulmonary emboli as a result of cemented impregnation(Gresham and Kuczynski, 1970; Brooker et al., 1973; Breed, 1974; Sharrock and Savarese,

1990). Sharrock and Savarese(1990) reported that these complications are more common in cases of hemodynamic instability and concurrent hypovolemia, and the incidences of it are related to depth of anesthesia at the time of cement insertion and the relative size of the exposed raw bony surface to which the cement is applied.

Hypoxemia secondary to the embolic effects of femoral shaft cement or fat embolism occurs immediately following insertion of methyl methacrylate bone cement(Orsini et al., 1987) and lasts up to 5 days into the postoperative period(Sharrock and

Savarese, 1990). A way to avoid this complication is the use of a plug in the femoral shaft to limit the distal spread of cement in the femur, venting and lavage of the intramedullary canal, and waiting for the cement to become more viscous before its insertion (Breed, 1974; Sherman et al., 1983; Engesaeter et al., 1984; Sharrock and Savarese, 1990).

Levodopa therapy should be continued in paralysis agitans during the perioperative period to avoid chest muscle rigidity that interferes with the maintenance of adequate ventilation and to avoid salivation from difficulty in swallowing (Paulson and Tafrate, 1970; Ngai, 1972; Stoelting and Dierdorf, 1993). Another reason is that sudden withdrawal of levodopa therapy may leave patients with a diminished capacity to synthesize catecholamines in peripheral adrenergic tissues (Dairman and Udenfriend, 1971).

Levodopa, a naturally occurring aromatic amino acid, is the most commonly used drug for the treatment of paralysis agitans to increase the concentration of dopamine in the basal ganglia. But, chronic treatment with levodopa causes several side effects (Ngai, 1972). Among these, inability to maintain blood pressure must be kept in mind during anesthesia. There are several suggested mechanisms. Levodopa causes accumulation of dopamine with concurrent loss of norepinephrine stores in the peripheral adrenergic nerve terminals and reduced production of norepinephrine due to negative feedback inhibition of dopamine. Consequently, the ability to maintain blood pressure is reduced because dopamine, a relatively weak pressor drug, replaces norepinephrine (Liu et al., 1971; Stoelting and Dierdorf, 1993). In animal studies, baroreflex is obtunded during levodopa infusion (Whitsett et al., 1970). Stoelting and Dierdorf (1993) showed that renin release is reduced during chronic levodopa therapy because dopamine increases renal blood flow. So, hypotension is caused by reduction of intravascular fluid volume and decreased activity of the renin-angiotensin-aldosterone system. Another side effect of levodopa is psychic disturbances and abnormal movements (Langrell and Joseph, 1971), which may disturb performance of surgery under regional anesthesia except in minor operations. In our case, the patient had received several hemorrhoidectomies under spinal anesthesia without special complications, but we performed general anesthesia for this major operation.

In our case, there was no evidence of hypoxemia or pulmonary embolism immediately following application of the bone cement (Fig. 1) or during the postoperative course according to the results of blood oxygen saturation, arterial blood gas value, and follow up chest X-ray. And we avoided hypovolemia and an excessively deep level of anesthesia during the anesthetic course (Fig. 1). The possible causes of the profound hypotension in our case regardless of normovolemia and proper anesthetic managements were (1) direct vasodilation and myocardial depression due to absorption of the still liquid or vaporized monomer via the relatively large size of the exposed area of raw bony surface to which the cement was applied, (2) further aggravation of the paralysis agitans due to major trauma, and (3) age-related diminution of autonomic tone. So, we think that the careful monitoring and the preventive management against the presumed hypotension seemed to prevent cardiac arrest or sudden death caused by profound hypotension.

To avoid this risk, we make the following recommendations;

1. Use noncemented prostheses, if possible (Petty, 1991).
2. Introduce proper techniques to apply the methyl methacrylate, if used (Sherman et al., 1983; Sharrock and Savarese, 1990).
3. Monitor the patient carefully.
4. Aggressive fluid infusion, high inspired fraction of oxygen (100 percent), and low concentration of anesthetic are required before application of the bone cement (Kallos, 1975; Stoelting and Dierdorf, 1993).
5. Avoid drugs which interact with the effects of dopamine; droperidol, fentanyl, alfentanil, ketamine, and antihypertensive drugs, etc (Ngai, 1972; Ngai and Wirklund, 1972; Mets, 1991).
6. Avoid halothane (Stoelting and Dierdorf, 1993).

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