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A patient with cardiac amyloidosis successfully managed with propofol anesthesia

Etsuko Miyamoto[1], Kumiko Fujise[2], Sanae Matsumoto[1], and Koh Shingu[1]

[1] Department of Anesthesiology, Kansai Medical University 10-15 Fumizono-cho, Moriguchi, Osaka 570-8507, Japan
[2] Department of Anesthesia, Ishikiri Seiki Hospital, Osaka, Japan

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Introduction

Amyloidosis is a progressive connective-tissue disorder with amyloid deposition in multiple sites, including the heart, liver, kidney, skin, gut, autonomic nervous system, tongue, and carpal tunnel. This disease is classified according to the type of amyloid protein as AL, AA, ATTR, $A\beta_2M$, or $A\beta$. AL-type primary amyloidosis has clinical manifestations that include cardiomyopathy, hepatosplenomegaly, nephrotic syndrome, macroglossia, and carpal tunnel syndrome [1]. Cardiac amyloidosis may cause restrictive cardiomyopathy, heart failure, conduction disorder, and ischemic heart disease [2], all of which should be borne in mind when planning the anesthetic management of patients with this condition. We describe anesthesia using propofol for a patient with an AL-type primary amyloidosis.

Case report

A 68-year-old man (body weight, 58 kg) was scheduled for bilateral decompression of carpal tunnel syndrome. There was no personal or family history of cardiac disease, except as mentioned below. Two years previously, he had undergone vitrectomy under local anesthesia because of opacity in the vitreous, and histological examination of the vitreous revealed AL-type primary amyloidosis. Additionally, cardiac amyloidosis was diagnosed by a myocardial biopsy to investigate the cause of first degree AV block and thickness of interventricular septum.

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The preoperative electrocardiogram (ECG) showed first degree AV block alone; the heart rate was 70–80 min^{-1}. Ultrasound assessment (UCG) showed a hypertrophied interventricular septum and a normal left ventricular ejection fraction of 68%, but left ventricular diastolic function was decreased on UCG. Pulse Doppler methods of UCG showed that the atrial peak velocity/early peak velocity (A/E) ratio was <1 (0.7) and the deceleration time (DT) was 190 ms. These data suggested a pseudonormalization state. Although these data seemed to be normal, the diastolic function of the left ventricle had been progressively damaged. His cardiac symptoms were evaluated as New York Heart Association (NYHA) degree 2. Coronary angiography showed no abnormal findings. Blood chemistry showed slight elevation of blood urea nitrogen (BUN) (25 mg dl^{-1}) and creatinine (1.2 mg dl^{-1}), which reflected renal amyloidosis.

After premedication with 5 mg of diazepam and 150 mg of ranitidine orally 1 h before arrival at the operating room, anesthesia was induced with 70 mg of propofol, 100 μg of fentanyl, and 8 mg of vecuronium. Anesthesia was maintained with 4–5 $mg \cdot kg^{-1} \cdot h^{-1}$ of propofol and intermittent injections of fentanyl and vecuronium. His respiration was supported by ventilation with air and oxygen. Total doses of propofol and fentanyl were 863 mg and 250 μg, respectively. In addition to basic monitoring, a cannula was inserted into the right dorsalis pedis artery, and a pulmonary artery (PA) catheter (Edwards Swan-Ganz CCO/ $S\bar{v}O_2$/VIP Thermodilution Catheter 8F, Baxter) was inserted in order to evaluate hemodynamic changes during the procedure.

Intraoperative changes in hemodynamic variables are shown in Fig. 1. Because the cardiac index (CI), blood pressure, and pulmonary capillary wedge pressure (PCWP) decreased to 1.7 $l \cdot min^{-1} \cdot m^{-2}$, 78/46 mmHg, and 8 mmHg, respectively, although the oxygen saturation in mixed venous blood ($S\bar{v}O_2$) was within normal range

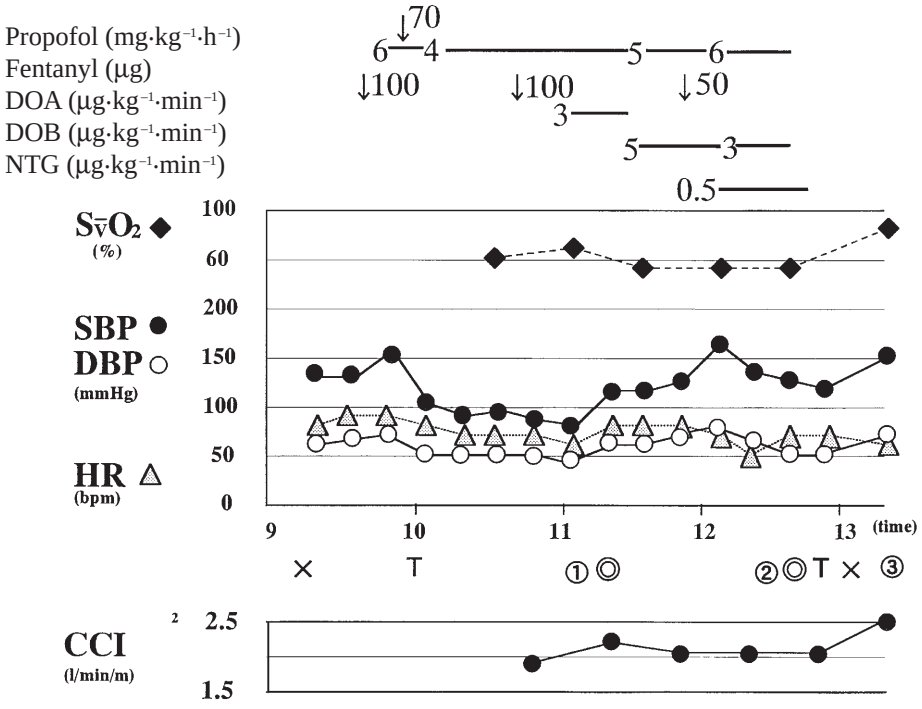

Fig. 1. Hemodynamic changes during the procedure. *DOA*, Dopamine, *DOB*, dobutamine; *NTG*, nitroglycerin; (◆) *$S\bar{v}O_2$*, hemoglobin saturation of mixed venous blood; (●) *SBP*, systolic blood pressure; (○) *DBP*, diastolic blood pressure; (▲) *HR*, heart rate; (●) *CCI*, continuous cardiac index; (×) start or end of anesthesia; (T) intubation or extubation; (◎) start or end of surgery; (①) attachment of tourniquet; (②) removal of tourniquet; (③) arrival in the recovery room

(78%), we started an infusion of 3 μg·kg^{-1}·min^{-1} of dopamine. Following the application of tourniquet to both arms and skin incision, mean arterial blood pressure (MAP) and systemic vascular resistance (SVR) increased, and then the infusion of dopamine was changed to dobutamine and nitroglycerin. The infusion of dobutamine, 5 μg·kg^{-1}·min^{-1}, and nitroglycerin, 0.5 μg·kg^{-1}·min^{-1}, was continued until release of the tourniquet at the end of surgery.

No dysrhythmia was observed during the procedure. Invasive hemodynamic monitoring was terminated in the recovery room. The postoperative course was uneventful, and the patient was discharged from the hospital on the third postoperative day.

Discussion

Patients with amyloidosis require close cardiovascular monitoring in the perioperative period because of the risk of cardiac complications [3–8]. These problems include heart failure due to restrictive cardiomyopathy, conduction disorders, and ischemic heart disease [1,9]. Although our patient had no clinical signs of heart failure before surgery, ventricular diastolic function was impaired according to the UCG. Under these circumstances, poorly controlled anesthetic management could result in acute left ventricular failure resistant to digitalis and potentially causing sudden death [3–8]. To avoid the risk, we performed careful monitoring using invasive hemodynamic monitoring.

In general, cardiac amyloidosis is characterized by restrictive cardiomyopathy, due to a diastolic dysfunction that requires careful attention to contractility, heart rate, preload, and afterload [10]. We initially selected dopamine and subsequently selected dobutamine and nitroglycerin (after tourniquet application) to control the cardiac function utilizing continuous monitoring of cardiac output (CO) $S\bar{v}O_2$, central venous pressure (CVP), pulmonary arterial pressure, and systemic arterial blood pressure. By using this monitoring, we were able to identify and treat the hemodynamic changes associated with tourniquet application, namely increase in MAP, CVP, PCWP, and SVR and decrease in stroke volume, CO, and $S\bar{v}O_2$. These changes are consistent with those in a previous report [11].

We changed from dopamine to dobutamine because we considered that dobutamine had more potent β effects than dopamine and would therefore be suitable for decreasing afterload and increasing cardiac contractility [12]. We infused nitroglycerin simultaneously to improve the compliance of large arteries without causing excessive afterload reduction [13].

Surgery in the upper extremities can be performed by using local or regional anesthesia, but we were persuaded to select general anesthesia in this case because of the patient's preference. Isoflurane is recommended for patients with cardiac amyloidosis because of its vasodilating effects and lesser arrhythmogenicity [3,14]. However, we selected propofol because it optimizes the relationship between the left ventricle and the arterial vascular bed [15], by preserving aortic pressure, increas-

ing aortic compliance, and improving energy transmission from the left ventricular to the arterial system. A possible disadvantage of propofol anesthesia for a patient with cardiac amyloidosis is its potential to cause bradycardia [16], although it has no direct effect on the cardiac conduction system in the isolated heart [17]. Bradycardia induced by propofol is commonly associated with surgery such as laparoscopy [18] and strabismus surgery [19], when vagal tone might be potentiated. Under these circumstances, atropine will readily overcome the problem. However, treatment with isoproterenol or a pacemaker is recommended, because amyloid often accumulates in the sinus node of patients with cardiac amyloidosis [20], and bradycardia is reported to be resistant to atropine [6]. We were prepared for insertion of a temporary pacemaker catheter via the introducer sheath for a PA catheter, if necessary.

In conclusion, we anesthetized a patient with cardiac amyloidosis without problems by using propofol. The cardiac function of the patient was not so deteriorated, and arrhythmias did not develop.

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