Anesthetic Management for a Patient with Restrictive Cardiomyopathy

Koji SUMIKAWA, Takanori KATO and Sonoko NAKANO

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Restrictive cardiomyopathy is a form of myocardial disease associated with pathologic evidence of inflammation, infiltration and fibrosis[1]. The prognosis is usually poor[1]. The clinical and hemodynamic features are similar to those of constrictive pericarditis and cardiac tamponade[1,2]. However, there seem to be some differences among these from an anesthetic viewpoint. We describe anesthetic management for a patient with idiopathic restrictive cardiomyopathy who had markedly elevated left ventricular end-diastolic pressure and revealed the clinical symptom of severe heart failure.

Case Report

A 46-year-old man, weighing 53 kg, was diagnosed as having restrictive cardiomyopathy. Heart murmur and cardiomeglay had been pointed out when he was 19, and he began to feel dyspnea and palpitation on exertion at the age of 28. When he was 44, he was admitted to a hospital because of his progressive symptoms and additional pretibial edema. Restrictive cardiomyopathy, liver cirrhosis and iron deficiency anemia were diagnosed. Subsequently, he was admitted to the hospital several times for congestive heart failure. His medications were digoxin, 0.125 mg qd, and furosemide, 80 mg qd. Several kinds of Ca channel blocker, such as diltiazem and verapamil, had been attempted to improve his hemodynamic condition because of their action to increase ventricular compliance, but they exerted no obvious efficacy.

He was hospitalized for the fourth time because of melena and easy fatigability, which had been developing for two weeks. Stomach varices with hemorrhagic erosion were diagnosed, and surgery was anticipated. Significant physical findings included a grade 2/6 diastolic rumbling murmur at the apex and a grade 2/6 systolic murmur at the left sternal border, hepatomegaly, arrythmia, pretibial edema, and conjunctival anemia. EKG revealed atrial fibrillation. Echocardiography showed marked thickening of the ventricular septum, left ventricular wall thickening, and left and right ventricular enlargement, but no significant thickening or calcification of the pericardium. Chest x-ray indicated cardiomegaly (cardiothoracic ratio = 0.72). Cardiac catheterization revealed the following findings: cardiac index (CI), 3.2 l/min/m^2; systemic arterial pressure (SAP), 124/60 mmHg; left ventricular end-diastolic pressure (LVEDP), 43 mmHg; right ventricular end-diastolic pressure (RVEDP), 36 mmHg; right atrial pressure (RAP), 26 mmHg; ejection fraction, 0.72; left ventricular dp/dt, 1540 mmHg/s. Endomyocardial biopsy showed thickening and fibrosis of the endomyocardium. Blood analysis revealed marked anemia (Ht 17%, Hgb 4.5 g/dl), and blood chemistry indicated moderate hepatic and renal dysfunction (GOT 70, GPT 71, BUN 37, creatinine clearance 31–45 ml/min).

As surgical premedication, scopolamine, 0.5 mg, and hydroxydine, 50 mg, were given im

Department of Anesthesiology, Toyonaka Municipal Hospital, Toyonaka, Japan

Address reprint requests to Dr. Sumikawa: Department of Anesthesiology, Toyonaka Municipal Hospital, 2-1-1 Okakamino-cho, Toyonaka, 560 Japan

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30 min before anesthesia. Anesthesia was induced with pancuronium, 1 mg, diazepam, 10 mg, thiamylal, 50 mg, and succinylcholine, 100 mg, followed by endotracheal intubation. For maintenance of anesthesia, he received fentanyl, 0.2 mg, pancuronium, 4 mg, and N_2O, 66%. When the operation was started, SAP was 90/50 mmHg, central venous pressure (CVP) was 31 mmHg, and pulmonary capillary wedge pressure (PCWP) was 38 mmHg. To lower preload, furosemide, 20 mg, was given iv. Thereafter, dopamine was additonally given at a rate of 3 μg/kg/min, which effectively lowered the PCWP to 30 mmHg with maintaining the CI at the preoperative value. Electrolyte and glucose solution was infused at a rate of 200 ml/hr throughout the operation. Partial gastrectomy and splenectomy were performed as the surgical operation. Total blood loss was 1180 ml and 1200 ml of whole blood was transfused. Urine output during the five-hour operation was 630 ml. SAP was stable during anesthesia (90/55–130/60 mmHg). The hemodynamic data at the end of anesthesia were pulmonary artery pressure (PAP), 50/32 mmHg; PCWP, 28 mmHg; CVP, 13 mmHg; CI, 2.7 l/min/m^2; Ht, 31%. Arterial blood gas analysis revealed slightly increased A-aDo_2 probably due to pulmonary congestion. The patient was admitted to the ICU. The endotracheal tube was extubated on the next day. Continuous infusion of dopamine was continued until the fourth postoperative day at the rate of 3 μg/kg/min. He made an uneventful recovery and was discharged from ICU on the fifth postoperative day. The hemodynamic data at this time was SAP, 110/60 mmHg; CVP, 7 mmHg; PCWP, 12 mmHg; CI, 2.7 l/min/m^2.

Discussion

The differential diagnosis of cardiac conditions in which diastolic pressure is elevated in all four cardiac chambers includes constrictive pericarditis, cardiac tamponade, and restrictive cardiomyopathy[3]. The present patient was diagnosed mainly by the findings of echocardiography, cardiac catheterization and myocardial biopsy. Similarly to constrictive pericarditis and cardiac tamponade[4], intraoperative management of restrictive cardiomyopathy requires strict hemodynamic control to optimize preload, afterload, and myocardial contractility. In spite of similarities among these compressive diseases, there seems to be an important difference in the hemodynamic features of restrictive cardiomyopathy and the others. In constrictive pericarditis, rapid volume loading similarly elevates LVEDP and RVEDP. A restrictive cardiomyopathic process usually involves the left ventricle more extensively. Here, any stressfull hemodynamic maneuver effects a disproportionate elevation in LVEDP[3,5]. The present patient also showed marked differences between the two pressures, i.e., there was an inordinate elevation of LVEDP and PCWP. The consequence was pulmonary congestion, reduced lung distensibility, an increase in the work of breathing, and dyspnea. This implies that, in addition to CVP, monitoring of PCWP is indispensible during anesthetic management of a patient with restrictive cardiomyopathy.

There is no definitive treatment for this disease[6]. Diuretics and venous vasodilators are considered to be useful in alleviating congestive symptoms, although they may provoke hypotension consequent to a precipitous decline in cardiac preload[4]. It has been suggested that the patient with severely impaired diastolic compliance deserves a therapeutic trial of Ca antagonist because of its action to enhance ventricular relaxation[3]. However, in the present patient, the hemodynamic efficacy of this medication was not confirmed. The reason for this might be preexisting severe left ventricular failure.

For perioperative management of this patient, a low dose of dopamine and diuretics was very effective for improving the hemodynamic state, i.e., PCWP declined from 38 mmHg to 12 mmHg while normal cardiac output was maintained. It is considered reasonable that a restrictive condition with extremely high LVEDP deserves a therapeautic trial of dopamine for perioperative management.

In summary, anesthetic management for a patient with restrictive cardiomyopathy is described. The hemodynamic features of this disease are similar to those of constrictive pericarditis and cardiac tamponade. However, a cardiomyopathic process usually involves

the left ventricle more extensively than right ventricle, resulting in susceptibility of left ventricle to a failure under any stressful hemodynamic maneuver. Accordingly, strict hemodynamic control is required with assessing the left ventricular performance.

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