**Anesthetic management of a patient with large atrial septal defect undergoing laparoscopic cholecystectomy: A case report**

**ABSTRACT**

A 51-year-old woman presented with symptomatic GB stone was planned for elective laparoscopic cholecystectomy. She had known osteum secondum type atrial septal defect, moderate pulmonary hypertension, and atrial fibrillation. We report the case of a patient with a large atrial septal defect (65 mm) and hemodynamic instability who underwent laparoscopic cholecystectomy under total intravenous anesthesia with careful hemodynamic monitoring. Thorough surveillance and effort could help to make the surgery successful.

**Key words:** Laparoscopic surgery; large ASD (6.5 mm); pulmonary hypertension; TIVA

**Introduction**

Atrial septal defect (ASD) accounts for about one-third of cases of congenital heart disease detected in adults. ASDs are classified into four types: Osteum secondum (85%), osteum primum (10%), sinus venosus (5%), and coronary sinus defect (rare).

Younger patients are usually asymptomatic, but individuals in whom the ASD remains uncorrected may exhibit exertional dyspnea by their third or fourth decade. Persistence of the left-to-right shunt resulting from uncorrected defects can lead to complications such as pulmonary artery hypertension, right heart failure, atrial fibrillation, stroke, and Eisenmenger’s syndrome.

We report the case of a patient with a large ASD (65 mm) who underwent laparoscopic cholecystectomy under total intravenous anesthesia with careful hemodynamic monitoring.

**Case**

A 51-year-old woman (height: 152 cm; weight: 35 kg) presenting with a symptomatic gallbladder stone was scheduled to undergo elective laparoscopic cholecystectomy. The patient’s medical history was significant for congenital ASD, congestive heart failure, and atrial fibrillation. She also had a history of an upper respiratory infection lasting two weeks and exertional dyspnea NYHA class II with general weakness.

Blood investigations showed PT-INR 1.4 and platelets 124,000, while other tests were within normal limits. Chest radiography revealed marked cardiomegaly and pulmonary vascular engorgement. ECG showed atrial fibrillation with rapid ventricular response, right bundle branch block, and minimal voltage criteria for left ventricular hypertrophy. Transthoracic echocardiography showed a large osteum secundum ASD (65 mm in diameter) with a left-to-right shunt,

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right ventricular hypertrophy with moderate pulmonary hypertension with an estimated right ventricle systolic pressure of 62 mmHg, moderate tricuspid regurgitation, atrial fibrillation with bilateral atrial enlargement, and a left ventricular ejection fraction of 68% [Figure 2]. Pulmonary function tests revealed FVC 36%, FEV1 24%, and FVC/FEV1 66% with severe restrictive and obstructive disease.

The patient was premedicated with midazolam 2 mg in the preoperative room. In the operating room, initial blood pressure was 123/79 mmHg, heart rate was 130–160 beats/min, and SpO₂ was 92%. Radial artery catheterization was performed prior to induction, and invasive arterial blood pressure was monitored. Esmolol 5 mg was administered intravenously owing to the patient’s atrial fibrillation (heart rate 130–160 beats/min, non-invasive blood pressure 119/75 mmHg).

To maintain baseline mean arterial blood pressure and decrease heart rate, continuous infusions of norepinephrine (0.05 µg/hr) and diltiazem (10 mg/hr) were administered prior to endotracheal intubation. The patient’s heart rate subsequently decreased to 100–108 beats/min. She was preoxygenated with 100% oxygen. General anesthesia was induced and maintained with total intravenous anesthesia of 2% propofol and remifentanil. Rocuronium 30 mg was injected, immediately followed by endotracheal intubation, and pressure-controlled ventilation with volume guaranteed was applied. To monitor the patient’s intravascular volume status and ventricular function, central line catheterization of the right internal jugular vein and transesophageal echocardiography (TEE) monitoring were performed [Figure 3]. Reverse Trendelenburg and right side-up position were applied during surgery.

During the intraoperative period, persistent hypotension (BP 72/49–84/53 mmHg) and atrial fibrillation (120–160 beats/min) were observed. Phenylephrine, ephedrine, norepinephrine, and diltiazem were used to maintain blood pressure and heart rate. Body temperature measured with an esophageal thermometer was 37.3°C before the operation and 37.0°C at the end of the operation. Peak inspiratory pressure and CO₂ insufflation pressure were 20 mmHg and 10–12 mmHg, respectively.

The duration of surgery was 45 min. The total fluid administered to the patient intraoperatively was 450 ml, and the measured blood loss was 20 ml. Following the operation, the patient was transferred to the surgical intensive care unit without extubation. After 6 hr, ABGA (7.40‑39‑79‑24.2‑96%) was measured and extubation was performed. Norepinephrine infusion was tapered and stopped 2 hr after surgery when the patient’s vital signs were stable. The following day, the patient was transferred to the general ward, and she was discharged four days later.

**Discussion**

ASD is one of the most common congenital heart diseases, and the progression of the disorder may result in the
reversal of the shunt. ASDs can be classified based on their anatomy, but they can also be classified according to the size of the defect. Most defects less than 3 mm in size close spontaneously, while approximately 80% of defects 3–8 mm in size close on their own, and defects 9 mm or more almost always remain unclosed. The patient described here had a defect 65 mm in diameter, much larger than the 9 mm value in the ASD classification.

The magnitude of the left-to-right shunt depends on the size of the ASD. In this patient, the large ASD likely resulted in a clinically remarkable left-to-right shunt (Qp/Qs: 4.14:1). Right heart failure and pulmonary venous hypertension resulting from irreversible vascular changes and increasing pulmonary vascular resistance (PVR) developed as the shunt progressed, so the patient had already been experiencing exertional dyspnea, pulmonary hypertension, and right heart failure at the time of her presentation.

Pulmonary hypertension that occurs with ASD progression can be classified as mild (36–49 mmHg), moderate (50–59 mmHg), or severe (>60 mmHg) with right ventricular systolic pressure values measured using echocardiography. The right ventricular systolic pressure of the patient described here was 62 mmHg. In cases in which pulmonary artery pressure increases further during surgery due to increased PVR or other factors, pulmonary hypertensive crisis can result, which may lead to decreased cardiac output and hypoxemia. Therefore, the goal of anesthetic management in patients with pulmonary hypertension is to lower PVR and maintain systemic vascular resistance (SVR). To prevent an increase in PVR, acidosis should be corrected, hyperventilation should be avoided, sympathetic nervous system stimulation should be avoided, normothermia should be maintained, and intrathoracic pressure should be minimized.

The operation, in this case, was performed by laparoscopy. In laparoscopic surgery, carbon dioxide insufflation may produce significant hemodynamic and pulmonary changes due to increased intra-abdominal pressure and hypercarbia. Studies conducted with healthy individuals have demonstrated that SVR and MAP rise abruptly within the first 5 min of abdominal insufflation, owing to abdominal aortic compression and neuroendocrine effects. In anesthetic consideration of patients with ASDs, the elevation of SVR should be avoided because this could increase the amount of shunt by producing hemodynamic instability during the surgery. In the current patient, the CO2 insufflation pressure did not rise above 12 mmHg, at our request. No abrupt hemodynamic instability occurred during the first 5 min after carbon dioxide insufflation.

Insufflation of CO2 gas can also cause hypercarbia. This condition can both increase PVR by causing pulmonary artery constriction and exacerbate pulmonary hypertension. During the operation in this case, the patient’s PCO2 was estimated using the EtCO2 value, and ABGA was performed. In addition, the minute ventilation was adjusted so that the EtCO2 values were within 32–35 mmHg.

Perioperative hypothermia may also increase PVR, which exacerbates pulmonary hypertension. To lower the PVR, we performed continuous monitoring to maintain normothermia. The patient’s body temperature remained within the normal range until the end of the operation.

Because sympathetic stimulation may affect SVR, the patient was sedated earlier with midazolam as a premedication. We attempted to minimize the infusion of propofol within the Bispectral Index (BIS) range of the patient to minimize changes in SVR. During the operation, norepinephrine and diltiazem were used to maintain baseline blood pressure and heart rate.

ASD patients usually present with a left-to-right shunt, but occasionally a right-to-left shunt may cause air to enter the systemic circulation and result in a paradoxical cerebral air embolism. The possibility of this complication could be augmented by positive end-expiratory pressure (PEEP) in mechanical ventilation. Therefore, we did not apply PEEP in the current case.

Also, the reverse Trendelenburg and right side-up position of the patient during surgery could increase the risk of cerebral embolism. Therefore, the risk of introduction of air along with medication into the patient’s peripheral or central IV lines was carefully monitored.

In summary, we have demonstrated here that a patient with a large ASD (65 mm) and hemodynamic instability can undergo non-cardiac laparoscopic surgery with the use of TIVA and careful monitoring. We monitored EtCO2, TEE, PIP, and body temperature to minimize PVR increases and SVR changes. In addition, we carefully monitored the risk of introduction of air into the patient’s IV lines to prevent paradoxical air embolism.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will
be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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