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CLINICAL REPORT

Pulmonary embolism caused by a carbon dioxide blower during off-pump coronary artery bypass grafting

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Abstract We report a rare case of pulmonary embolism (PE) caused by a carbon dioxide (CO_2) blower during off-pump coronary artery bypass grafting (OPCAB). When the anastomosis of the right internal thoracic artery to left anterior descending artery was performed, the operator tore the right ventricle outflow track (RVOT) that was adjacent to the left anterior descending artery. Immediately after the anastomosis and repair of the torn RVOT with CO_2 blower, the systolic pulmonary artery pressure (PAP) increased from 28 to 64 mmHg, and end-tidal CO_2 decreased from 32 to 12 mmHg. Because transesophageal echocardiograph (TEE) showed numerous gas bubbles in the main pulmonary artery, we diagnosed PE caused by invasion of CO_2 gas bubbles via the torn RVOT. Although a CO_2 blower is useful to enhance visualization of the anastomosis during OPCAB, it should not be used for the venous system because it may cause CO_2 embolism.

Keywords Pulmonary embolism · Coronary artery bypass grafting · Transesophageal echocardiogram

Introduction

A carbon dioxide (CO_2) blower has been routinely used in coronary artery bypass grafting (CABG) to facilitate visualization of the anastomotic site. Although it is a very useful device, gas embolism is a possible complication caused by high flow and pressure. Several cases of gas embolism by a CO_2 blower during endoscopic saphenectomy for CABG have been reported [1–3], and there has been only one report that gas embolism by CO_2 flow into injured an coronary vein leads to critical condition during CABG in the English literature [4]. In this report, we describe a rare case of pulmonary embolism (PE) caused by CO_2 flow into a torn right ventricular outflow track (RVOT) during off-pump CABG.

Case report

A 62-year-old man with unstable angina pectoris was scheduled for OPCAB surgery. His preoperative coronary angiography showed 90% stenosis of the distal left anterior descending artery (LAD) and 99% stenosis of the proximal circumflex artery. Systolic ventricular function was normal (ejection fraction 60%) on echocardiography, and no serious abnormalities were found on routine laboratory studies, electrocardiogram, or chest X-ray. He was taking nitrate and carvedilol for hypertension. On the day of surgery, he received premedication of intramuscular midazolam (5 mg) and atropine (0.5 mg). On arrival in the operating room, his systolic blood pressure was 130/63 mmHg and heart rate was 70 bpm. General anesthesia was induced with intravenous midazolam (4 mg), fentanyl (500 μg), and vecuronium (10 mg). Anesthesia was maintained by continuous infusions of propofol, fentanyl, and vecuronium. Monitoring followed a routine protocol for CABG that included the use of transesophageal echocardiography (TEE), a central venous catheter, and a pulmonary artery catheter (PAC) placed through the right

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internal jugular vein. Systolic and diastolic pulmonary arterial pressures (PAP 25/9 mmHg), cardiac output (5.2 L min^{-1}), and cardiac index (2.5 L min^{-1} m^{-2}) were normal. After median sternotomy, both internal thoracic arteries were harvested without causing any hemodynamic changes. The patient was placed in the Trendelenburg position, and the heart was lifted with a pericardial aspirator to expose the anastomotic site. Anastomosis of the right internal thoracic artery to LAD was started using a CO_2 blower at a flow of 3 L min^{-1}. Soon after the start of the anastomosis, the operators informed us that they had torn the RVOT that was adjacent to the LAD. At that time, hemodynamics, oxygen saturation, and end-tidal CO_2 were normal. After 20 min, the anastomosis and repair of the torn RVOT were finished, and the heart was freed from the pericardial aspirator. Immediately after the heart was returned to its original position, the systolic PAP increased from 28 to 64 mmHg, end-tidal CO_2 decreased from 32 to 12 mmHg, and oxygen saturation decreased from 100% to 90%, although the systemic arterial pressure and heart rate did not change (Fig. 1). TEE examination showed a massive amount of gas bubbles in the main pulmonary artery and no gas bubbles in the left side of the heart (Fig. 2). Invasion of CO_2 gas bubbles via the torn RVOT seemed to be the most likely cause of the PE. The lungs were ventilated with 100% oxygen and aspiration of gas bubbles via the PAC was attempted. Operators started preparation for a cardiopulmonary bypass, and the excessive Trendelenburg position was maintained. Although aspirated gas from the PAC was only 1–2 ml, gas bubbles gradually disappeared and PAP, end-tidal CO_2, and oxygen saturation normalized within 10 min after PE. Because the hemodynamics were stable 20 min after the occurrence of PE, anastomosis of the left internal thoracic artery to the proximal circumflex artery was performed without cardiopulmonary bypass. The postoperative course of the patient was unremarkable. Tracheal extubation was performed 4 h after surgery, and no neurological abnormalities were noted. He was discharged from the surgical intensive care unit on postoperative day 2 and from the hospital on postoperative day 10.

Discussion

During CABG, bleeding from a coronary artery may interrupt the anastomotic procedure. To facilitate visualization of the anastomotic site, several techniques have been used, including a coronary shunt or occluders, intermittent irrigation with normal saline, or a gas blower [5]. In particular, a CO_2 blower is widely used and effective in maintaining a bloodless field [6]. The underlying cause of PE in this case was that the operators used a CO_2 blower during repair of the RVOT, and blood pressure in the RV was much less than that in the arterial system. There are few reports of a CO_2 gas blower causing PE. Lee et al. [4] reported massive CO_2 embolism via a torn coronary vein during OPCAB that was caused by a CO_2 blower. In our case, there is a possibility that the CO_2 gas bubbles flew into a torn coronary vein. However, we could not detect gas bubbles in the coronary sinus and the RV with TEE examination soon after PE. Based on TEE and information from the operators, we believe that the CO_2 directly entered the venous circulation via the torn RVOT. Previous studies reported venous air embolism that was more common when the patient was in the sitting position during neurosurgical cases [7, 8]. Because our patient was in the Trendelenburg position and central venous pressure was 8–12 mmHg during the anastomosis, it seems unlikely that air was aspirated into the venous system.

The pattern of CO_2 embolism is that dissolved CO_2 increases end-tidal CO_2, and then undissolved CO_2 forms bubbles that increase physiologic dead space, leading to

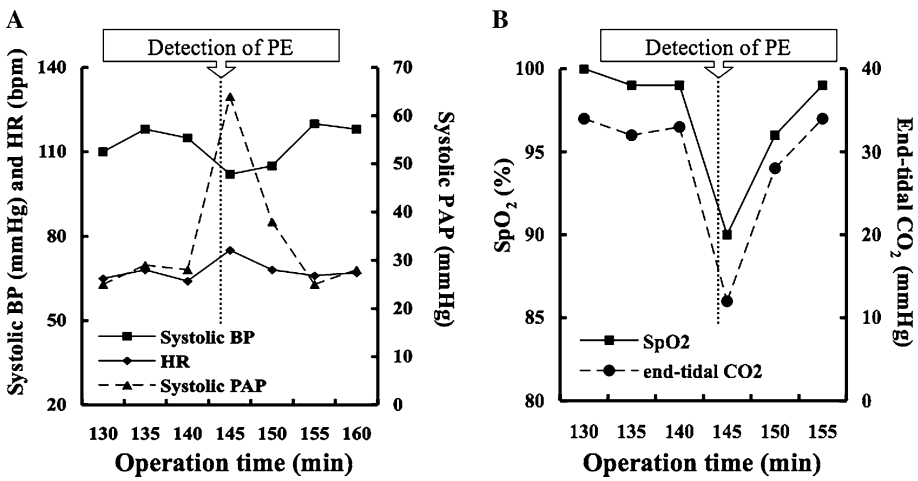

Fig. 1 **a** Changes in systolic blood pressure (*SBP*), pulmonary artery pressure (*PAP*), and heart rate (*HR*) during pulmonary embolism (*PE*). **b** Changes in oxygen saturation and end-tidal carbon dioxide (CO_2)

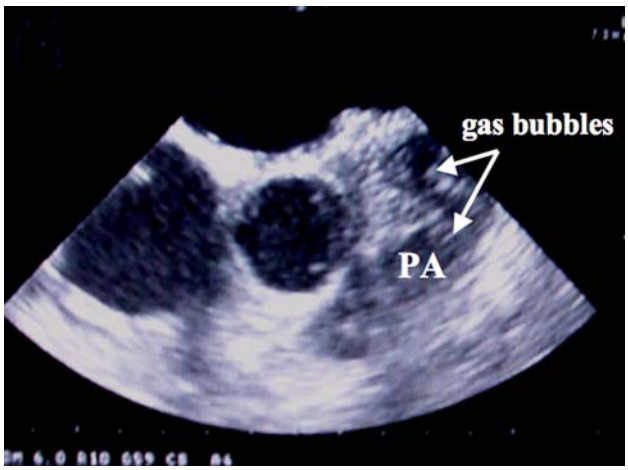

Fig. 2 Transesophageal echocardiographic (TEE) examination showed gas bubbles in the main pulmonary artery (*PA*)

pulmonary hypertension, oxygen desaturation, and reduced end-tidal CO_2 [3, 4]. Sustained PE may lead to cardiac failure, systemic hypotension, and cardiovascular collapse [4]. In this case, there were no findings of elevated end-tidal CO_2, oxygen desaturation, and/or pulmonary hypertension just before the PE occurred. Therefore, it seemed that there was rapid accumulation of excess CO_2. Lee et al. [4] also reported that there was no evidence of hypercapnia before the occurrence of PE. It would be difficult to predict PE from only end-tidal CO_2, oxygen saturation, and PAP when there is rapid accumulation of CO_2 gas bubbles in the pulmonary circulation.

In our case, it is worth noting that PE occurred just after the heart was returned to a normal position from retraction for anastomosis. During anastomosis, the patient was placed in the Trendelenburg position, and the heart was lifted with a pericardial aspirator. These facts suggest that many of the CO_2 bubbles were trapped at the top of the right ventricle during anastomosis because of the position of the patient and the heart during the procedure. When the heart was returned to its original position, the trapped CO_2 bubbles rapidly entered the pulmonary artery. After diagnosing PE, we tried to aspirate the CO_2 bubbles via a PAC, but the effectiveness of this procedure was not clear. Fortunately, CO_2 bubbles disappeared naturally in this case without hemodynamic deterioration. If continued PE caused hemodynamic instability, pharmacological treatment or cardiopulmonary bypass might be required.

In conclusion, a CO_2 blower should not be used for repair or plasty of venous systems in order to avoid CO_2 embolism. Careful observation with TEE may be useful for early detection of a gas embolism so that anesthesiologists and surgeons could prepare for events and prevent development of hemodynamic deterioration.

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