

Successful resuscitation of cardiac arrest caused by CO₂ embolism with intra-aortic injection of epinephrine during off-pump coronary bypass surgery

-a case report-

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Although compressed gas (CO₂) blowers have been used safely to aid accurate grafting during off-pump coronary bypass surgery, hemodynamic collapse due to gas embolism into the right coronary artery may occur. Supportive measures to facilitate gas clearance by increasing the coronary perfusion pressure have been reported to be successful in restoring hemodynamic stability. However, right ventricular dysfunction and atrioventricular nodal ischemia may hinder effective systemic delivery of the vasoactive medications, even when performing resuscitative measures such as direct cardiac massage. We herein report a case of cardiac arrest that was caused by a right coronary gas embolism and that could not be restored by cardiac resuscitation. When supportive measures fail, direct aortic injection of epinephrine to increase the coronary perfusion pressure can be attempted before initiating cardiopulmonary bypass, and this approach may be life-saving in situations that limit systemic drug delivery from the venous side despite the performance of direct cardiac massage. (Korean J Anesthesiol 2013; 65: 562-564)

Key Words: Ascending aorta, Cardiac arrest, Epinephrine, Gas embolism, Off-pump coronary artery bypass.

During off-pump coronary bypass surgery (OPCAB), bleeding from the coronary arteriotomy site presents a challenge to accurate anastomotic suturing. In order to optimize the surgical view, insertion of an intra-coronary shunt or coronary snaring with a compressed CO₂ blower have been used safely to overcome this impediment. However, applying a gas blower to the

coronary arteriotomy site may provoke a rare but devastating gas embolism [1]. We herein report a case of cardiac arrest caused by a gas embolism into the right coronary artery territory during OPCAB and successful resuscitation by direct injection of epinephrine to the ascending aorta.

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Case Report

A 72-year-old male patient with acute coronary syndrome underwent OPCAB. He had a left ventricular ejection fraction of 42% and regional wall motion abnormalities compatible with multi-vessel territories. The patient was in normal sinus rhythm. Coronary angiography findings revealed total occlusion at the proximal left anterior descending artery (LAD), 50–75% stenosis at the mid to distal left circumflex artery, and 90% stenosis at the mid right coronary artery (RCA).

Cardiac surgery was performed through a median sternotomy, and the heart was displaced using a deep pericardial retraction suture, gauze swab, and tissue stabilizer. After confirming the lack of atheroma burden in the ascending aorta by epi-aortic ultrasound examination, the left radial artery was connected to the ascending aorta. After completing the LAD grafting using the left internal thoracic artery, grafting was performed on the obtuse marginalis (OM) branch. The patient was in the Trendelenburg position, and norepinephrine was infused at a dose of 0.15 $\mu\text{g}/\text{kg}/\text{min}$. After stabilizer application, there was a temporary deterioration in the patient's hemodynamic status, which was stabilized without the aid of incremental doses of norepinephrine dosage.

After opening the anastomotic site at the OM branch, the surgeon informed us the patient was bleeding from the arteriotomy site despite placement of a proximal snare. The CO_2 blower was more aggressively used to aid grafting at a flow rate of 3–5 L/min. Bleeding was replaced by colloid solution, and all hemodynamic variables were stable: systemic blood pressure 121/58 mmHg, heart rate 59 beats/min, pulmonary arterial pressure 24/11 mmHg, cardiac index 2.6 L/min/m² and mixed venous oxygen saturation 82%. Approximately 5 minutes after the commencement of grafting, the surgeon decided to insert an intracoronary shunt for better visualization. Suddenly, the patient's systemic blood pressure decreased to 64/56 mmHg despite increasing the norepinephrine dose from 0.05 $\mu\text{g}/\text{kg}/\text{min}$ to 0.30 $\mu\text{g}/\text{kg}/\text{min}$. The pulmonary arterial pressure increased to 34/20 mmHg, and the mixed venous oxygen saturation decreased to 66%. Vasopressin 0.6 IU was quickly administered intravenously, and the transesophageal echocardiography (TEE) revealed a massive gas floating at the orifice of the RCA (Fig. 1). On the TEE, no gas was visible in the right heart without any patent foramen ovale. Although the patient remained in normal sinus rhythm, complete heart block occurred within minutes, and ventricular pacing was commenced. Over the next several minutes, ventricular fibrillation developed; despite aggressive treatment, including defibrillation, epinephrine, and direct manual cardiac massage, adequate circulation could not be restored, and cardiac massage was continued. Upon preparing for emergent conversion to cardiopulmonary bypass (CPB), epinephrine

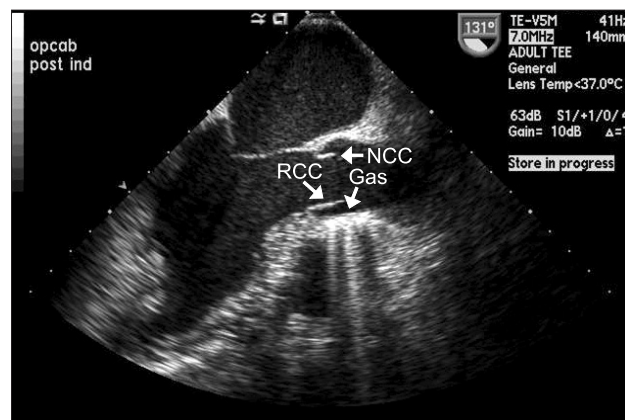


Fig. 1. Midesophageal aortic valve long-axis view showing a significant amount of gas confined in the sinus of Valsalva at the right coronary cusp. In contrast to the sinus of Valsalva at the noncoronary cusp, the sinus of Valsalva at the right coronary cusp shows obliteration of the natural curvature by the highly echogenic mass of gas; RCC: right coronary cusp, NCC: noncoronary cusp.

300 μg was injected directly to the aortic root by the surgeon using a 26-gauge needle. Subsequently, manual cardiac massage was continued for several cycles, and spontaneous recovery of unspecified cardiac rhythm was observed. Then the patient's systemic mean arterial pressure gradually rose from 38 to 103 mmHg, and recovery of normal sinus rhythm and contractile performance followed immediately. Other hemodynamic parameters, including pulmonary arterial pressure, also returned to normal levels. The remaining grafts were performed successfully without conversion to CPB, and no sign of bleeding was observed at the puncture site in the aorta. The patient had an unremarkable postoperative course without neurologic deficits and was discharged 10 days after the surgery.

Discussion

Despite using a proximal coronary snare, this method of hemostasis can be incomplete in the presence of severe atheroma or calcification, causing bleeding through the arteriotomy site. Cardiac displacement invariably causes hemodynamic deterioration, and when the diastolic systemic blood pressure decreases below the pressure generated by the CO_2 gas blower, gas may migrate in a retrograde fashion to the aortic root. Even in the presence of a proximal snare, the proximal coronary lumen may not be completely obstructed due to the presence of calcified atheroma, as evidenced by continued bleeding from the arteriotomy site after application of the snare. As the RCA ostium is in the highest position of the sinus of Valsalva, gaseous materials would be prone to migrate into the RCA, causing embolic infarction [1].

A minor gas embolism may pass unnoticed or be overcome

with supportive measures to aid emboli clearance. However, in the case of a massive embolism with the possible introduction of room air due to Venturi effect, hemodynamic collapse can occur and may require aggressive treatment, such as cardiopulmonary resuscitation (CPR) with aspiration of the air by direct puncture of the aortic root [2]. It may even require emergent conversion to CPB, which is known to be associated with increased morbidity and mortality [3].

Therefore, when bleeding from the arteriotomy site persists despite the use of a proximal snare or intracoronary shunt, anesthesiologists should be aware of the possibility of a gas embolism and advise the surgical team to stop or limit the use of the CO₂ blower. When a gas embolism results in hemodynamic deterioration, supportive measures should be immediately undertaken to increase the coronary perfusion pressure and facilitate emboli clearance [1]. Gas embolism should also be confirmed with TEE, and a pacemaker should be at hand given that complete heart block due to atrioventricular nodal ischemia may occur in the case of an embolism involving the RCA. Finally, in the case of failure of supportive measures such as drug administration to the venous side, direct aortic injection of the drugs can be attempted before initiating CPB. In coronary artery bypass graft surgery, direct cardiac massage may not be aggressively and effectively performed due to the fear of disrupting the previously

grafted conduits, limiting the drug delivery from the venous side to the systemic circulation. Prior to the 1960s, intracardiac injection of medications was used blindly or directly, but this approach was abandoned due to the devastating complication of lacerating the epicardial coronary arteries and cardiac tamponade [4,5]. However, direct injection to the aortic root following a sternotomy appears to be feasible and may be life-saving, as severe right ventricular dysfunction may limit systemic drug delivery injected through the venous side despite the performance of direct cardiac massage [6]. In the current case, intra-aortic injection of epinephrine may have resulted in a more effective increase in the systemic vascular resistance, thereby increasing the coronary perfusion pressure and thus facilitating emboli clearance.

In conclusion, in the rare case of a gas embolism occurring during OPCAB occurs, the potential for catastrophic results necessitates immediate and appropriate managements including supportive measures, needle aspiration of gas, and CPR with direct cardiac massage. In refractory cases, while considering emergent conversion to CPB, direct injection of drugs to the aortic root can be attempted before initiating CPB. This may be a simple, relatively safe, and life-saving approach for the management of a gas embolism during OPCAB because a sternotomy has already been conducted.

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