

Massive Air Embolism during off-pump CABG: A Case Report

Abstract

Off-pump coronary artery bypass grafting (OPCAB), although devoid of the morbidity associated with cardiopulmonary bypass (CPB), has its own technical difficulties. Achieving optimum tissue stabilization on a beating heart along with hemodynamic fragility due to extreme positioning also complicates the anesthetic management. In addition, it is difficult to obtain a clear surgical field in the presence of arteriotomy. The use of catheter-directed high-flow gas blower (mister blower) helps achieve a clear surgical field to a great extent. However, there have been reported cases of arterial and pulmonary embolism caused by these high-flow gas blowers. The present case reports a case of massive venous air embolism caused by the use of mister blower.

Keywords: Air embolism, mister-blower, Off-pump coronary artery bypass

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Introduction

Although off-pump coronary artery bypass grafting (OPCAB) is known to be devoid of cardiopulmonary bypass (CPB)-induced morbidity, it is technically more challenging.^[1] Till date there is no “ideal” surgical technique reported in the literature for OPCAB. The use of octopus stabilizer with suction arm-tips has been the cornerstone to facilitate stabilization during distal anastomosis. Still arteriotomy continues to be a challenging step that directly affects the visualization of the anastomotic site.^[2] Intracoronary shunts, snuggers, bull-dog clamp, saline injection, and high-flow gas blowers are routinely used to facilitate exposure during distal anastomosis.^[3] The use of high-flow compressed gas alongside hemodynamic fragility that the beating heart is subjected to during OPCAB may be considered conducive for occurrence of accidental embolism. Although CABG is associated with 5–25% risk of air embolism, the incidence is unknown in OPCAB.^[4]

Case History

A 54-year-old male diagnosed with coronary artery disease with triple vessel involvement on angiogram [proximal left anterior descending (LAD) 90% stenosis, obtuse marginal (OM) 80% stenosis and

proximal right coronary artery (RCA) 100% stenosis] was referred for coronary artery bypass grafting (CABG). His preoperative echocardiogram showed moderate LV dysfunction with ejection fraction (EF) of 40%.

Following a standard cardiac induction with etomidate, fentanyl and rocuronium, right femoral artery and right internal jugular vein (IJV) were cannulated followed by insertion of a pulmonary artery catheter (PAC) through IJV. The plan was to harvest left internal mammary artery (LIMA), left radial artery (LRA), and left saphenous vein (LSV) as the required conduits for grafting. After completing LIMA to LAD and OM to LRA anastomosis, the patient was positioned in the Trendelenburg position with a slight right tilt for RCA to LSV grafting. The octopus stabilizer was positioned at the proximal RCA segment that was slinged and clamped with a bulldog. RCA endarterectomy was planned due to full thickness calcified plaque. It was a routine practice to use a mister blower with high-flow CO₂ (6 l/min at 30 mmHg) to enable a bloodless field for distal anastomosis. Within 10 min of starting RCA grafting patient developed sudden bradycardia (heart rate 35 beats per minute) and hypotension (BP 58/32 mmHg, mean 43 mmHg). End tidal carbon -dioxide (ETCO₂) suddenly decreased from 32 mmHg to 14 mmHg. Within the next one minute patient went

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into asystole and the surgeon started immediate cardiac massage. Adrenaline bolus 1 mg IV was immediately administered and cardiac massage was continued. RV distention was noticed by the surgeon. Immediate TEE examination showed distended air-filled right atrium and right ventricle [Figure 1]. Thirty milliliter of air was aspirated from PAC. On no signs of return of cardiac activity after repeated boluses of adrenaline and cardiac massage, decision on immediate institution of CPB was made and subsequently aorta and RA were cannulated in the next 10 min. The RCA grafting was completed with CPB. After rewarming, all cardiac chambers were vigorously de-aired. With the return of sinus rhythm, patient was gradually weaned off CPB with inotropic support over the next 30 min.

After the surgery patient was shifted to the intensive care unit (ICU) and electively ventilated till the next day. Once awake, his sensorium and motor power was examined. Because there was no evidence of neurological deficit, he was carefully extubated. His brain tomography was normal and he was discharged on day 10 without any deficits.

Discussion

In the present case, the use of high-flow CO₂ blower directly over the arteriotomy site that was clamped proximally with a bulldog clamp may have caused a high pressure jet of CO₂ to enter coronary capillaries in an anterograde fashion, and the subsequent absorption in the myocardial tissue. This resulted in RV distention that was observed clinically, and entrainment of air in the coronary veins leading to detection of air in the RA. Thirty milliliter of air could be aspirated from the proximal lumen of the PAC. Although the connection between the PAC sheath and the catheter is a known source of air entrainment,^[5] it was ruled out in this case as the connection was securely fastened and the embolism occurred much later during grafting. Also the preoperative TEE did not show presence of air in any cardiac chamber. There have been

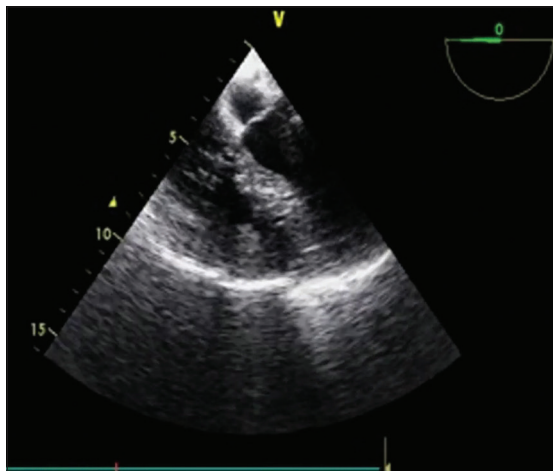


Figure 1: Air in RA and RV

case reports about pulmonary embolism and arterial embolism due to CO₂ blower during OPCAB.^[6,7] The proposed mechanisms were inadvertent injury to right ventricular outflow tract during right internal mammary dissection and embolization from OM into ascending aorta. Other published causes in literature include patent foramen ovale, coronary arteriovenous fistula, endoscopic saphenous vein harvesting, and increased intrathoracic pressure during sternal closure.^[6-8] In the present case, massive air embolism occurred secondary to RV distention due to air entrainment into venous circulation from RCA arteriotomy site.

The use of CO₂ is considered comparatively safer than oxygen as it is 34 times more soluble in blood than oxygen.^[9] Therefore, it does not lead to microbubble formation. However, fast inadvertent injection of large volume of CO₂ can cause air-lock leading to profound hypotension culminating into asystole and cardiac arrest.^[8] Venous air embolism is clinically suspected in cases of unexplained sudden profound hypotension with a decreasing ETCO₂ trend. Although TEE is the most sensitive in detecting air embolism,^[10] in this case, the diagnosis was not suspected till RV distention was grossly apparent. Moreover, the authors considered it prudent to pursue hemodynamic resuscitation as the top priority. First line of management includes administering 100% oxygen, hemodynamic support (adrenaline, dobutamine etc.), prompt aspiration of air from PAC, in cases of cardiac arrest, cardiac massage, and eventually CPB institution.

Therefore, a strong index of suspicion is warranted in cases of intraoperative air embolism. Sudden hypotension and decreasing ETCO₂ should alert the anesthetist of an impending cardiovascular collapse due to air embolism. Authors believe that presence of TEE probe *in situ* in this case enabled a prompt decision for institution of CPB. Moreover, the presence of PAC aided in aspirating the air from the right side of the heart, thus helping to prevent a more catastrophic outcome.

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Conflicts of interest

There are no conflicts of interest.

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