

Case report

Carbon dioxide embolism induced right coronary artery ischaemia during off-pump obtuse marginalis artery grafting

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Received 2 April 2009; accepted 29 April 2009; Available online 7 July 2009

Abstract

Although use of carbon dioxide (CO₂) blower has been regarded safe during off-pump coronary bypass surgery (OPCAB), we experienced a case of right coronary artery ischaemia induced by retrograde CO₂ embolism originating from the opened obtuse marginalis artery during OPCAB. The spray pressure can exceed the diastolic pressure, especially during grafting at the lateral or posterior wall when haemodynamic compromise due to mechanical heart displacement is most severe. In this situation, CO₂ blowing at an incompletely slinged coronary arteriotomy site can result in retrograde migration of CO₂ into the ascending aorta causing coronary embolism of the right coronary artery. When signs of ischaemia on the right coronary artery are encountered during grafting of other coronary artery, although CO₂ blower has been used, gas embolism should also be considered as the cause and identified at the mid-oesophageal aortic valve long-axis view. When confirmed, the use of gas blower should immediately be discontinued and coronary perfusion pressure increased while allowing time for the CO₂ to be absorbed. In case of massive embolism, needle aspiration of the gas should also be considered.

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Keywords: Gas embolism; Off-pump surgery; Coronary bypass

1. Introduction

To aid accurate grafting performance, compressed gas blower is often used to facilitate optimal visualisation of the coronary arteriotomy site during off-pump coronary bypass surgery (OPCAB) [1]. In contrast to compressed air, gas blower using CO₂ is more advantageous with regard to solubility in blood, and has been safely used without any reported coronary as well as systemic embolism, leading to adverse events, yet. We herein report a case of right coronary artery ischaemia induced by retrograde CO₂ embolism originating from the opened obtuse marginalis artery during OPCAB.

2. Case report

A 70-year-old woman with a stable angina was presented for elective OPCAB. Preoperative echocardiography revealed normal chamber sizes and valvular functions with a left ventricular ejection fraction of 64% and no regional wall

motion abnormalities. Coronary angiography revealed 70% stenosis at the mid left anterior descending artery, 80% stenosis at the proximal left circumflex artery and 90% stenosis at the proximal right coronary artery.

Surgery was performed under median sternotomy and both internal thoracic arteries were skeletonised and the left radial artery was also harvested. The left radial artery was anastomosed to the left internal thoracic artery in Y-graft configuration. The heart was displaced using posterior pericardial stitch, large (12 cm × 70 cm) gauze swabs and tissue stabiliser (Octopus Tissue Stabilization System[®], Medtronic Inc., USA) and humidified CO₂ blower was used at a flow rate of 3–7 l min⁻¹ with an average spray pressure of 50 mmHg. First, the left internal thoracic artery was anastomosed to the left anterior descending artery. Then, a soft silicone snare was placed under proximal obtuse marginalis artery and the radial artery anastomosis was commenced. The patient was in the Trendelenburg position and norepinephrine 0.1 µg kg⁻¹ min⁻¹ was infused. Due to calcification, proximal soft snare did not provide complete haemostasis and the flow rate of the CO₂ blower was increased to 7 l min⁻¹. The patient's haemodynamic status was stable with a heart rate of 73 beats per minute, blood pressure of 100/52 mmHg, central venous pressure of 9 mmHg, mean pulmonary arterial pressure (MPAP) of 22 mmHg, cardiac index of 2.3 l min⁻¹ m⁻² and mixed venous oxygen saturation of 68%. After approximately 5 min,

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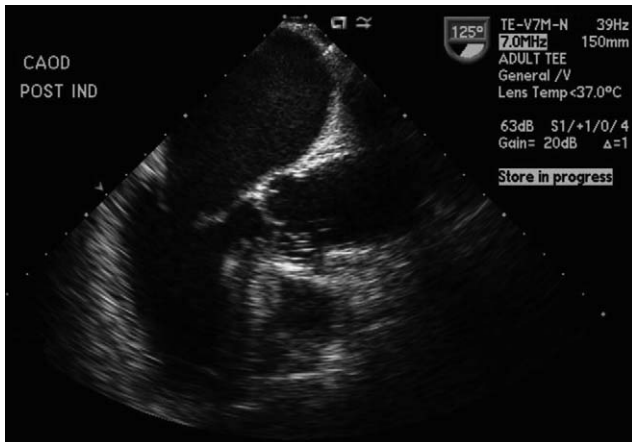


Fig. 1. Mid-esophageal aortic valve long-axis view showing gas confined in the sinus of Valsalva, mostly at the right coronary cusp portion.

when three-fourths of the anastomosis was complete, sudden increase in the lead II ST segment from 0.1 to 2.4 mV was noted, with concomitant increase in MPAP to 37 mmHg and decrease in blood pressure to 66/46 mmHg, despite an increase in the rate of norepinephrine infusion. Grafting was continued and transoesophageal echocardiography examination was immediately performed and revealed severe hypokinesia of the right coronary artery territory and severe mitral regurgitation. At the mid-oesophageal aortic valve long-axis view, gas bubbles could be identified at the sinus of Valsalva (Fig. 1), and retrograde CO₂ embolism of the right coronary artery originating from the opened obtuse marginalis artery was suspected. The use of CO₂ blower was immediately stopped and while norepinephrine was ineffective in restoring the mean arterial pressure (MAP), vasopressin 0.1 U IV (intravenous) was given, and the MAP could be increased to 65 mmHg; however the MPAP was still 39 mmHg with a lead II ST elevation of 2.3 mV. Within a minute, before completion of grafting, the ST segment of the lead II, MPAP, mitral regurgitation, cardiac function was normalised and norepinephrine infusion rate was decreased to maintain the MAP between 70 and 80 mmHg during grafting. Surgery could be performed without any further complications and complete re-vascularisation was performed. The post-operative course of the patient was uneventful without any signs of systemic embolic events. Postoperative electrocardiographs (ECGs) also showed no significant changes, and the highest level of creatine kinase-MB during the postoperative period was 7.51 ng ml⁻¹.

3. Discussion

In addition to coronary snaring, use of compressed gas blower facilitates bloodless surgical field and high-quality distal anastomosis. Spray pressure of the gas blower can exceed the diastolic pressure, especially during grafting on the lateral or posterior wall when haemodynamic compromise due to mechanical displacement is most severe [2]. During this period, blowing compressed gas at an incompletely snared coronary artery can result in retrograde migration of gas into the ascending aorta, resulting in hazardous

embolic complications. In a similar situation using compressed air, massive systemic air embolism resulting in hypoxic encephalopathy has been reported [3]. Coronary air embolism induced by compressed air blower during OPCAB requiring intra-aortic balloon pump has also been reported, recommending the use of CO₂ blower [4].

CO₂ is 34 times more soluble than air in water and not likely to cause embolic complications [4]. Indeed, use of deep pericardial CO₂ insufflation in valvular heart surgeries is associated with marked decrease in the incidence of micro-emboli [5,6]. However, massive CO₂ pulmonary embolism while repairing an injured coronary vein by aid of CO₂ blower leading to haemodynamic deterioration has been reported [7]. This suggests that gross embolisation of CO₂ overwhelming the vasculature can result in devastating results, which may require aspiration of the gas in addition to supportive measures.

The current case demonstrates that although CO₂ was used, it can be an iatrogenic cause of myocardial ischaemia of a different coronary artery territory from the one in which grafting is performed. Intra-operative myocardial ischaemia during OPCAB requires accurate diagnosis to treat accordingly. When sudden ST segment elevation and/or cardiac dysfunction are present while using the CO₂ blower, gas embolism should also be considered and identified as the cause at the mid-oesophageal aortic valve long-axis view to treat the patient appropriately. Retrograde gas embolism originating from opened coronary artery with incomplete proximal coronary sling should mostly be manifested as signs of ischaemia of the right coronary artery, especially when the patient is placed in the Trendelenburg position. When diagnosed, the use of CO₂ blower should be immediately stopped and supportive measures to increase coronary perfusion pressure should be considered while allowing the CO₂ to be absorbed. The patient should also remain in the Trendelenburg position to confine the gas in the sinus of Valsalva to prevent systemic embolisation and also to be able to perform needle aspiration in case of massive embolism and/or the patient's haemodynamic status cannot be restored after supportive measures.

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