ESC European Society of Cardiology

European Heart Journal - Case Reports (2023) **7**, 1–6
https://doi.org/10.1093/ehjcr/ytad521

CASE REPORT
Structural intervention

Coronary air embolism during transcatheter patent foramen ovale closure for platypnea-orthodeoxia syndrome in a patient with severe respiratory disorder: a case report

Kosuke Tanimura, Hiroyuki Yamamoto, Ken-Ichi Hirata, and Hiromasa Otake *

Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, 7-5-1 Kusunoki-cho, Chuo-ku, Kobe 650-0017, Japan

Received 29 May 2023; revised 3 October 2023; accepted 16 October 2023; online publish-ahead-of-print 24 October 2023

Background Coronary air embolism (CAE) is a rare and life-threatening complication of endovascular procedures, mostly due to procedure-related causes.

Case summary A 70-year-old man with severe respiratory disorder presented with patent foramen ovale (PFO)-related platypnea-orthodeoxia syndrome (POS). Transcatheter PFO closure was performed under local anaesthesia and intracardiac echocardiographic guidance. After a 5-Fr catheter was passed through the PFO via a 7-Fr femoral vein sheath, the patient suddenly coughed and breathed deeply. Thereafter, intracardiac echocardiography showed massive microbubbles in all cardiac chambers and the ascending aorta, and an electrocardiogram showed ST-segment elevations in the anterior and inferior leads. Emergency coronary angiography confirmed occlusion of the mid-left anterior descending artery, suggesting CAE. As the intracoronary infusion of saline, nitroglycerine, and nicorandil was ineffective, we performed air aspiration using a thrombectomy device, achieving coronary blood flow improvement and ST-segment resolution. Thereafter, positive pressure support using manual bag-valve-mask ventilation under intravenous sedation supported successful transcatheter PFO closure without further air embolization.

Discussion In this case with severe respiratory dysfunction, spontaneous deep breathing (spontaneous Valsalva manoeuvre) caused negative intrathoracic pressure and large drops in intravascular pressure. This phenomenon might have induced air contamination during device advancement, either by entrapping or leaving residual air in the gaps between the catheter and the sheath. Additionally, PFO with right-to-left shunts is more likely to cause paradoxical air embolization. Thus, the spontaneous Valsalva manoeuvre should be avoided with appropriate respiratory management to prevent paradoxical air embolization, including CAE, during transcatheter PFO closure under local anaesthesia in severe respiratory dysfunction patients.

Keywords Coronary air embolism • Patent foramen ovale • Platypnea-orthodeoxia syndrome • Respiratory disorder • Transcatheter patent foramen ovale closure • Case report

ESC curriculum 3.2 Acute coronary syndrome • 9.7 Adult congenital heart disease

* Corresponding author. Tel: +81 78 382 5846, Fax: +81 78 382 5859, Email: hotake@med.kobe-u.ac.jp
Handling Editor: Giulio Russo
Peer-reviewers: Mathias Lange; Felipe Hernandez; Giulio Russo
Compliance Editor: Emmanouil Mantzouranis

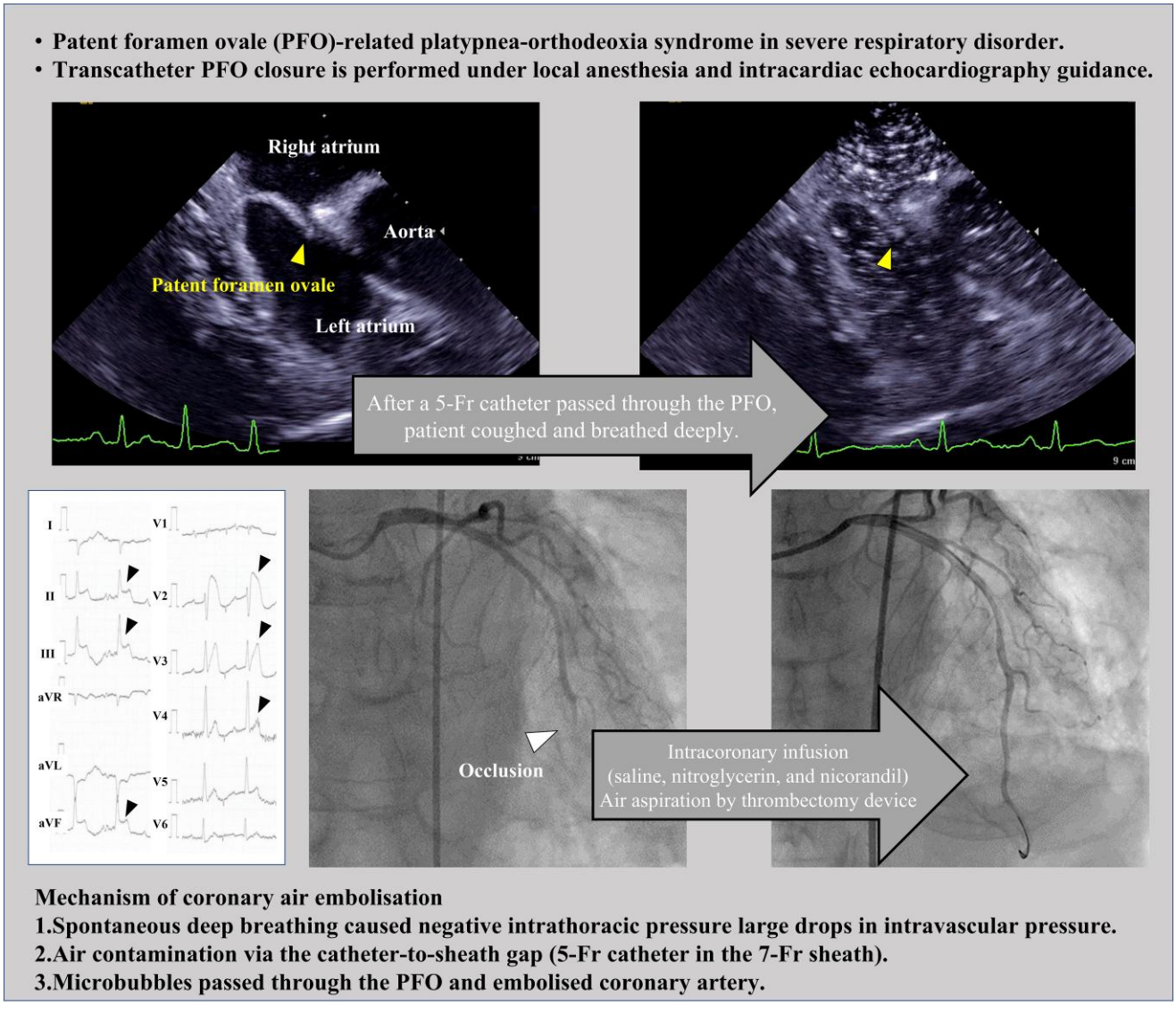
Learning points

- During transcatheter patent foramen ovale (PFO) closure under local anaesthesia, appropriate respiratory management is crucial to prevent 'spontaneous Valsalva manoeuvres' in patients with severe respiratory dysfunction.
- Echocardiography during transcatheter PFO closure is useful for the immediate detection of coronary air embolization and can guide appropriate interventions.

Introduction

Platypnea-orthodeoxia syndrome (POS) is characterized by dyspnoea and hypoxia exacerbated in the sitting position due to increased right-to-left shunt flow. Patent foramen ovale (PFO)-related POS is a rare but notable entity because of its close association with paradoxical embolization. Transcatheter PFO closure, a safe and effective interventional treatment to prevent recurrent paradoxical cerebral infarction, can also improve PFO-related POS effectively.[1–3]

Summary figure



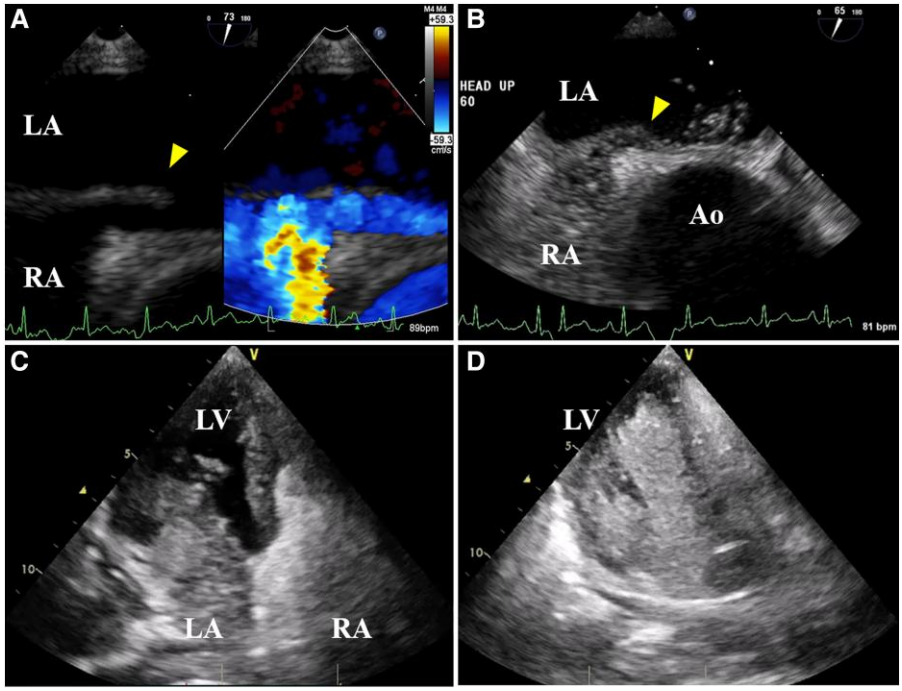


Figure 1 Patent foramen ovale with right-to-left shunts. (*A–B*) Transoesophageal echocardiogram with bubble test reveals a patent foramen ovale (arrowhead) with right-to-left shunt flow in head-up position. (*C–D*) A bubble test on transthoracic echocardiography in the sitting position reveals massive right-to-left shunts. Ao, aorta; LA, left atrium; LV, left ventricle; RA, right atrium.

An iatrogenic air embolism is a common catheter-related complication.[4] Among them, coronary air embolism (CAE) is a life-threatening complication that mainly occurrs during percutaneous coronary intervention.[5] Theoretically, paradoxical air embolism derived from massive microbubbles migrating through intracardiac right-to-left shunts can lead to fatal conditions.[6–8]

Herein, we describe a rare case in which a CAE leading to ST-segment elevations on an electrocardiogram occurred during transcatheter PFO closure for POS under local anaesthesia in an older patient with a severe respiratory disorder.

Case presentation

A 70-year-old man receiving home oxygen therapy (2 L/min) for severe respiratory disorder due to interstitial pneumonia presented with progressive exertional dyspnoea. The patient had no other medical history, including lifestyle diseases. On examination, his body temperature, blood pressure, pulse, and oxygen saturation in the supine position were 36.5°C, 100/56 mmHg, 56 beats per minute, and 97%, respectively. The dyspnoea worsened in the sitting position (desaturation to 80%) rather than in the supine position. Physical examination revealed that fine crackles were noted bilaterally at the base of the lung field, and the heart sounds were of a regular rhythm, without gallop. Transoesophageal echocardiography (TEE) revealed right-to-left shunts through the PFO with the patient in the head-up position (60°). Subsequent transthoracic echocardiography with a bubble test revealed massive right-to-left shunt flow in the sitting position, which was diagnosed as PFO-related POS (*Figure 1A–D*). Right heart catheterization revealed no finding of heart failure and pulmonary hypertension (mean right atrial pressure, 0 mmHg; pulmonary artery pressure, 16 mmHg; pulmonary capillary wedge pressure, 13 mmHg; cardiac index, 2.3 L/min/m^2; and pulmonary vascular resistance, 101 dynes/sec/cm^5), and no significant step-up in oxygen saturation. Chest computed tomography showed emphysematous changes in the lungs bilaterally with ground-glass opacities in the right upper lobe, suggesting interstitial pneumonia, and no anatomical abnormalities such as kyphoscoliosis, tortuous or elongation of the ascending aorta, or hemidiaphragmatic paralysis. Because general anaesthesia was challenging due to severe respiratory dysfunction [vital capacity (%vital capacity), 0.86 L (25.5%)], transcatheter PFO closure was performed under local anaesthesia and intracardiac echocardiography (ICE) guidance (8-Fr ACUNAV, Siemens Medical Solutions, USA). First, a 7-Fr sheath for PFO closure and a 9-Fr sheath for ICE was inserted via the right and left femoral veins (FV), respectively. The patient was on dual antiplatelet therapy (aspirin, 100 mg/day; clopidogrel, 75 mg/day) for 5 days before the procedure and received intravenous heparin (5000 U) after sheath insertion to maintain an activated clotting time of ≥250 s during the procedure. A 5-Fr multipurpose catheter, connected to a 2.5-cc syringe to check the blood backflow and to prevent air contamination, was inserted into the 7-Fr sheath and passed through the PFO without a guidewire. During this procedure, the patient suddenly coughed and breathed deeply. Thereafter, ICE showed massive microbubbles in all cardiac chambers and the ascending aorta, and visualization of the echographic images subsequently became difficult (*Figure 2A* and *B*). Electrocardiography revealed significant ST-segment elevations in the anterior and inferior leads (*Figure 2C*). Emergency coronary angiography revealed complete occlusion of the mid-left anterior descending (LAD) artery, suggestive of CAE (*Figure 3A*). While the intravenous administration of catecholamines supported his haemodynamic, an intracoronary infusion of saline, nicorandil, and nitrates into the LAD was performed. However, the coronary blood flow did not improve completely. We then performed air aspiration using a 6-Fr Thrombuster® III GR (Kaneka, Osaka, Japan), achieving coronary blood

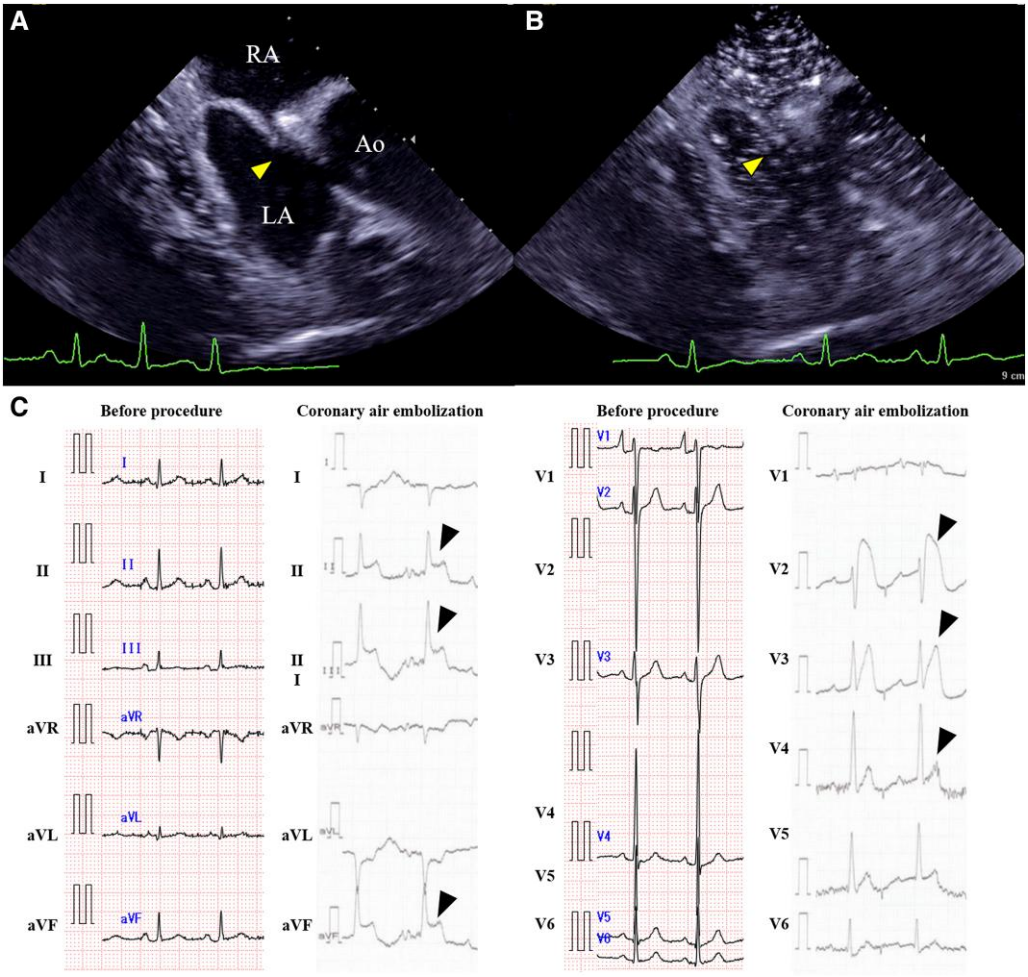


Figure 2 Intracardiac echocardiography and electrocardiography during transcatheter patent foramen ovale closure. Intracardiac echocardiogram shows (*A*) a patent foramen ovale (arrowhead) and (*B*) massive microbubble migration to the aorta via the patent foramen ovale. (*C*) Electrocardiogram showing ST-segment elevations in the anterior and inferior leads (arrowheads) after microbubble migration. Ao, aorta; LA, left atrium; RA, right atrium.

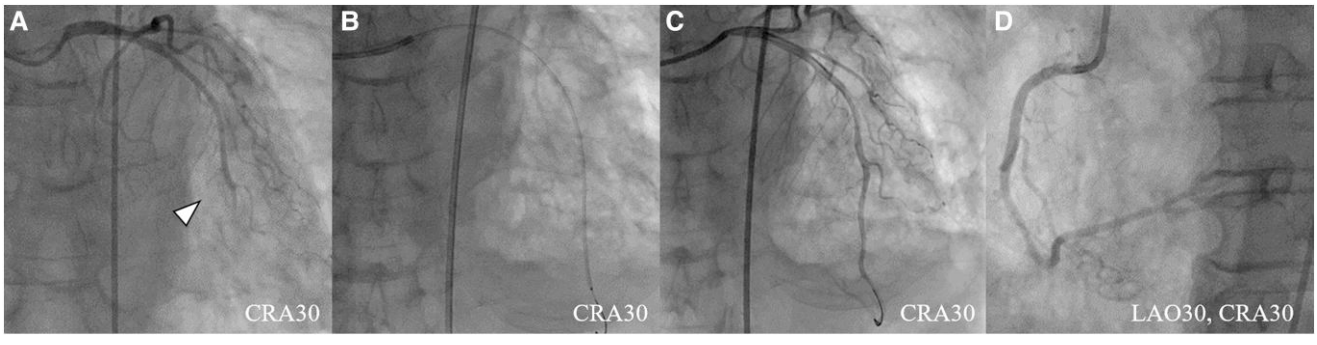


Figure 3 Coronary angiography. Coronary angiography shows (*A*) an obstruction in the mid-left anterior descending artery, (*B*) air aspiration, (*C*) coronary blood flow improvement after aspiration, and (*D*) no obstruction in the right coronary artery.

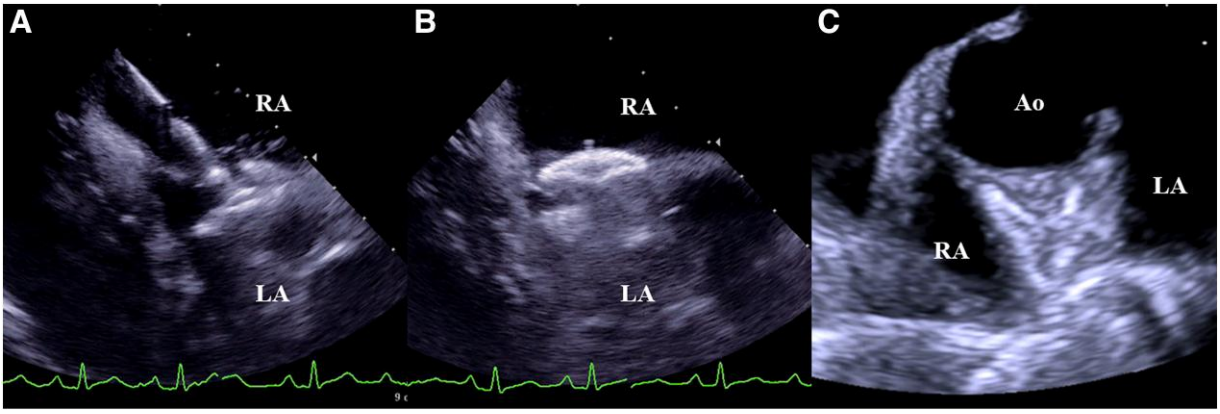


Figure 4 Transcatheter patent foramen ovale closure. (*A–C*) No further microbubble was observed on the intracardiac echocardiogram during transcatheter patent foramen ovale closure. Ao, aorta; LA, left atrium; RA, right atrium.

flow improvement and ST-segment resolutions in leads V2–4 (*Figure 3B and C*). Thereafter, intracoronary infusions of saline and nicorandil into the RCA resolved the ST-segment elevations in the inferior leads (*Figure 3D*). We controlled his respiratory condition under intravenous sedation and positive pressure support using manual bag-valve-mask ventilation to avoid coughing and subsequent deep breathing. The right femoral sheath was down-sized from a 7-Fr to a 5-Fr catheter to prevent air contamination via the catheter-to-sheath gap. A 25-mm nitinol-occluded device (Amplatzer Cribriform, Abbott, St. Paul, MN, USA) was implanted using a 9-Fr long sheath without further air contamination (*Figure 4A–C*). The postoperative course was uneventful, and the patient was discharged five days later with a drastic improvement in exertional dyspnoea and no neurological disorders.

Discussion

Air embolism is a cautionary complication of endovascular procedures. Recently, transvenous endovascular treatment, such as catheter ablation and interventions for structural heart disease, has increased. Paradoxical embolization leading to systemic air embolism should be prevented, especially in patients with congenital or acquired intracardiac right-to-left shunts.[9,10] This case demonstrated massive intracoronary air embolism due to negative intrathoracic pressure, potentially caused by deep inspiration and coughing during transcatheter PFO closure for POS under local anaesthesia in an older patient with a severe respiratory disorder, which was promptly diagnosed and successfully treated using ICE and TEE.

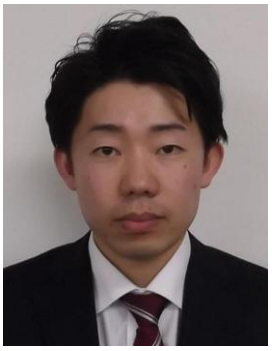
In this case, a potential mechanism of CAE was speculated that spontaneous deep breathings inducing negative intrathoracic pressure and strong drops in intravascular pressure led to air attraction through a catheter-to-sheath gap (5-Fr catheter in 7-Fr sheath) due to air contamination via the advancement of the device entrapped or left residual air in the catheter-to-sheath gaps. First, as the patient had severe respiratory disorders, the intravascular pressure drastically changed according to the influence of respiratory variations. Deep inspiratory conditions lead to a significant decrease in the intravenous pressure, whereas strong expiration, such as coughing, leads to an increase in the intravenous pressure.[11] In the present case, it was not possible to observe changes in intracardiac pressure during deep breathing or coughing, because the pressure line was not connected to the catheter as it passed through the PFO. Large negative intravenous pressure might have induced air contamination via a catheter-to-sheath gap, and increased intravascular pressure by subsequent cough promoted massive right-to-left shunts, leading to systemic air embolisms; these series of breathing patterns were thought to be equivalent to the 'spontaneous Valsalva manoeuvre.' Since stable respiratory control is crucial in cases of POS, respiratory management using a ventilator under general anaesthesia may prevent rapid changes in intravenous pressure. However, in the present case, local anaesthesia was preferable because of severe respiratory dysfunction. In such settings, respiratory management avoiding the 'spontaneous Valsalva manoeuvre' is mandatory to prevent large intracardiac pressure variation.

Second, regarding the source of air contamination, we speculated the association of catheter-to-sheath gaps (5-Fr catheter in a 7-Fr sheath). The technique of covering the sheath port with water or connecting a syringe is useful to prevent air contamination; these techniques were performed in the present case. Potential risks of air contamination include the loosening of haemostatic sheath valves and the advancement of devices with entrapped or residual air in the delivery sheaths.[12] After the procedure, we confirmed that the 7-Fr sheath valve showed no obvious deficits. Indeed, after the sheath was down-sized from 7-Fr to 5-Fr and manual compression of the femoral vein was performed, air contamination was not detected on the ICE images. Thus, the potential risk of air contamination via the advancement of the device entrapped or left residual air in the catheter-to-sheath gaps should be noted in cases with a potential risk of large intracardiac negative pressure.

In this case, early detection of paradoxical air embolization was achieved using ICE images, which immediately guided the diagnosis of CAE and its optimal management, including intracoronary infusions of several medications and subsequent air aspiration. Although this patient maintained stable haemodynamics during the CAE event without any mechanical circulatory support, elevation of blood pressure by inotropic agent administration with/without mechanical circulatory support is required to stabilize haemodynamics and to improve coronary blood flow. Saline injection into an occluded vessel can improve coronary blood flow by washout of microbubbles. The administration of vasodilators, such as nicorandil and nitrates, may also be useful.[13] In this case, direct air aspiration was required because massive microbubbles had migrated and completely occluded the coronary arteries. Air aspiration using a thrombectomy device may be effective in eliminating massive air embolisms.

Thus, CAE is a rare and cautionary complication of transcatheter PFO closure under local anaesthesia in patients with severe respiratory disorders. Appropriate respiratory management and careful attention to air contamination via catheter-to-sheath gaps are mandatory to prevent such critical situations.

Lead author biography



Dr Kosuke Tanimura received his licence to practice medicine at Tokushima University in 2012 and completed a PhD at the Kobe University Graduate School of Medicine in 2021. His main areas of research are intravascular coronary imaging and interventional cardiology, including coronary artery disease and structural heart disease.

Acknowledgements

All authors thank medical staffs related to this case in Kobe University Hospital for their excellent assistance in the patient treatment.

Consent: Informed consent was obtained from the patient for the publication of this case report in line with the COPE guidelines.

Conflict of interest: None declared.

Funding: None declared.

Data availability

The data underlying this article are available in the article and in its online supplementary material.

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