CASE REPORT

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Beware of Bubbles: Coronary Air Embolism During Transcatheter Patent Foramen Ovale Closure

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Abstract

Coronary air embolism is a rare and life-threatening side effect of endovascular interventions, mostly due to procedure-related causes. A 51-year-old man presented with a history of multiple transient ischemic attacks. Patent foramen ovale was found, and transcatheter closure was considered necessary. During the intervention, ST-segment elevation in leads II, III, and aVF was observed. Emergency coronary angiography revealed a typical air bubble that occluded the distal part of the right coronary artery. After nitrate intracoronary injection, the air bubble was dissolved, and ST-segment elevations were resolved. The electrocardiogram rhythm had turned to atrial fibrillation, and intravenous amiodarone was administered. The patient was closely monitored until sinus rhythm was restored. In this study, the distal RCA occlusion did not cause important hemodynamic instability but provoked an arrhythmia that was later restored. Thorough preparation of the equipment and proper intra-procedural techniques must be followed to prevent this serious complication.

Keywords: Coronary air embolism, patent foraman ovale, transcatheter patent foramen ovale closure, case report

INTRODUCTION

Coronary air embolism (CAE) is an uncommon side effect of transcatheter cardiac interventions with an incidence ranging from 0.1% to 0.3%. It arises when air is introduced into the coronary circulation due to inadequate flushing of the guiding and diagnostic catheters. Literature provides various consequences related to CAE, ranging from clinically insignificant occurrences to myocardial infarction, cardiogenic shock, and even death.[1] The aim of this paper is to present a rare complication during a patent foramen ovale (PFO) closure intervention.

CASE REPORT

A 51-year-old male presented to our outpatient cardiology clinic for further investigation because of multiple transient ischemic attacks in the preceding year, resulting in transient loss of sight. The patient had no other medical history, except for mild hyperlipidemia. Thrombophilia tests were negative, and carotid ultrasound did not reveal a plaque or clot.

On examination, his body temperature, blood pressure, pulse, and oxygen saturation in the supine position were 36.1 °C, 128/74 mmHg, 75 beats per minute, and 97%, respectively. Physical examination of the lungs was normal, and the heart sounds were of a regular rhythm, without gallop. Electrocardiogram (ECG) was sinus rhythm, without any irregularity.

Transoesophageal echocardiography (TEE) with a bubble test was positive (showed air bubbles into the left atrium) for PFO, with no signs of atrial septal defect and no findings of heart failure or pulmonary hypertension. Cardiac magnetic

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resonance imaging had normal findings, and the pulmonary vs systemic blood flow ratio (Qp/Qs) was approximately one.

According to the current guidelines for the management of adult congenital heart disease,[2] it was decided to proceed to transcatheter PFO closure. The intervention was performed under general anesthesia and under TEE guidance.

An 8-Fr sheath for PFO closure was inserted via the right femoral vein in the left atrium to deploy a CoCoon 30 mm septal occluder device (Vascular Innovations Co., Ltd.). The patient was on dual antiplatelet therapy (aspirin, 100 mg/day; clopidogrel, 75 mg/day) for 30 days before the procedure and received intravenous heparin (6000 U) after sheath insertion.

After the deployment of the device across the atrial septum (Figure 1), TEE showed that the closure device was in the right place, but the cardiac monitor suddenly showed significant ST-segment elevation in the inferior leads and sinus tachycardia. Emergency coronary angiography revealed an occluded distal part of the right coronary artery (RCA) with a moving air column distal to the occlusion, suggestive of a CAE (Figure 2A).

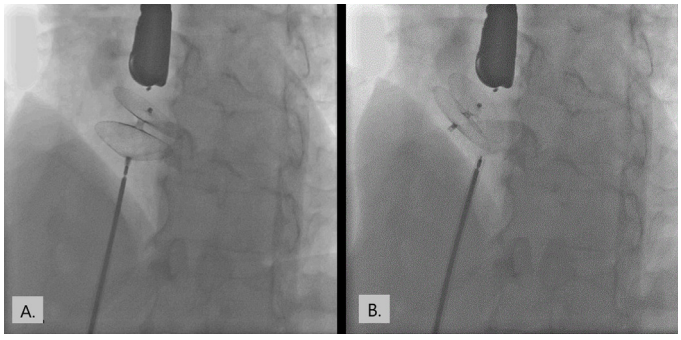

Figure 1: Fluoroscopy. Deployment of a 30 mm CoCoon septal occluder device for transcatheter treatment of patent foramen ovale. The correct placement of the PFO occluder is ascertained by the "Pacman sign" in Figure 1A

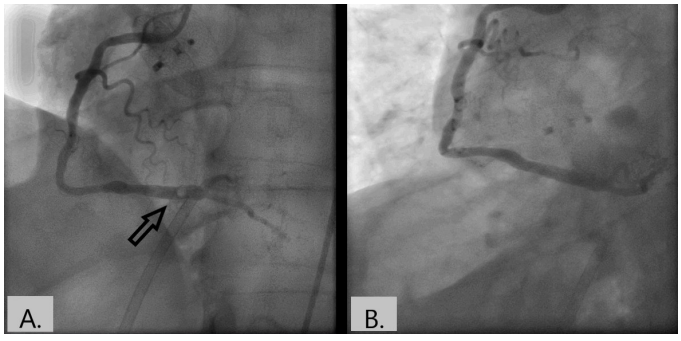

Figure 2: Coronary angiography. Coronary angiography showing an air bubble (arrowhead) obstructing the distal part of the right coronary artery (A) and no obstruction in the right coronary artery after injecting 150 mcg intracoronary nitrate (B)

An intracoronary 150 µcg nitrate injection was administered in the RCA to dissipate the air column. The air column was dispelled, and a TIMI-3 flow was achieved in the RCA with simultaneous resolution of the ST-segment elevations (Figure 2B). Afterwards, the cardiac monitor showed that the patient had atrial fibrillation with a rate of approximately 130 bpm; therefore, he was given IV amiodarone and transferred to the coronary care unit. Four hours later, his rhythm was restored with an ECG with sinus rhythm, no ST-segment elevations, and a rate of 80 beats/min. The rest of the postoperative course was event-free, and the patient was discharged one day later with medical instruction to receive clopidogrel 75 mg once daily and apixaban 5 mg twice daily for a month. Informed consent was obtained from the patient for the publication of this case report in accordance with the ICMJE guidelines.

DISCUSSION

CAE appears to be an uncommon and avoidable side effect of transcatheter cardiac interventions. Inadvertent air introduction into the coronary arteries can occur from a variety of sources, including inadequate cleaning of diagnostic or guiding catheters, air leakage from a malfunctioning manifold system, balloon rupture, air infiltration during balloon catheter insertion or removal, and structural equipment failures.[3,4] In particular, in PFO closure interventions, once the device advances through the delivery sheath, residual or entrapped air is forced into the left atrium, which typically causes an air embolism. This is believed to be the potential mechanism of coronary artery embolism in this study. It is important to note that the operator's experience and skill level are major predictors of the prevalence of this issue.[5]

The volume of air and the place of air that enters the coronary arteries determine the severity of the repercussions associated with air embolism. The clinical symptoms of air embolism can range from asymptomatic air embolism to chest discomfort, hypotension, myocardial infarction, arrhythmias (bradycardia, ventricular tachycardia, ventricular fibrillation, heart block), and cardiac arrest.[6] In this study, we were not able to assess any clinical manifestations because our patient was under general anesthesia.

Angiography is used to diagnose air embolism, but distinct bubbles are rarely observed in the coronary arteries. In some patients, closure of the affected artery might be complete, while the occluded site usually looks vague rather than possessing the distinct arterial cut-off that can be characteristic of an artery blocked by a thrombus. Additionally, it might lead to angiographic appearances of no-reflow or delayed flow. In this case, a typical distinct bubble was observed in the RCA. Given that the right-sided sinus of Valsalva is positioned anteriorly, the RCA is the epicardial coronary artery most frequently impacted by air emboli.[3] This clarifies the appearance of ST-segment

elevation in the inferior leads. Further electrocardiographic findings could include a complete atrioventricular (AV) block due to supplementation of the AV node by the RCA.[7] Apparently, in this case, the bubble was placed distally to the AV node infusion artery, and we did not notice any AV node electric disturbances.

Regarding the treatment of air embolism and its consequences, there is a lack of defined guidelines. Operators should thoroughly prepare the catheterization systems, ensuring that all connections are firm. In most cases, there are few air leaks that have no hemodynamic effects and do not require treatment. Supportive care, including rapid administration of analgesics for alleviating pain, 100% oxygen supplementation, hemodynamic support in case it is needed, and arrhythmia counseling, is provided for all instances of mild to moderately symptomatic patients until the air bubbles naturally dissolve.[1,8] Apart from supportive care, administration of arterial vasodilators like adenosine or nitrates, as in our case, and mechanical techniques such as bubble aspiration with certain catheters and fragmentation and displacement of air bubbles more distally are usually proposed.[9] Preventive care is a fundamental management principle.

Ethics

Informed Consent: Informed consent was obtained from the patient.

Authorship Contributions

Surgical and Medical Practices: I.B., F.I.E., Concept: A.P., F.I.E., Design: A.P., Data Collection or Processing: A.P., E.B., I.B., Analysis or Interpretation: I.B., F.I.E., Literature Search: A.P., E.B., Writing: A.P., E.B.

Conflict of Interest: No conflict of interest was declared by the authors.

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