Multiple Air Embolism During Coronary Angiography: How Do We Deal With It?

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ABSTRACT: Coronary air embolism remains a serious complication of cardiac catheterization despite careful prevention. The complications of coronary air embolism range from clinically insignificant events to acute coronary syndrome, cardiogenic shock, and death. We report here a case of multiple air emboli in both left coronary arteries, complicated by cardiogenic shock and ventricular fibrillation in a 49-year-old male patient undergoing elective percutaneous coronary intervention. The patient recovered after supportive measures, including oxygen, intravenous dopamine infusion, and cardiac compression, and repeated forceful injection of heparinized saline successfully resolved the air emboli. He then eventually underwent successful percutaneous coronary intervention in the left anterior descending artery without any residual stenosis.

KEYWORDS: coronary arteries, air embolism, cardiogenic shock, ventricular fibrillation, saline injection

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Introduction

Coronary air embolism is a rare complication of cardiac catheterization, ranging in incidence from 0.1% to 0.3%.[1] Although preventable, it is mostly iatrogenic and occurs when the catheters used have not been adequately aspirated and flushed, causing introduction of air into coronary vasculature.[2] The complications of air embolism range from a clinically insignificant event to an acute coronary syndrome and death.[3] The management consists of supportive measures, including administration of 100% oxygen (to minimize ischemia and to establish a diffusion gradient encouraging elimination of gas from the bubble) and pain relief. The use of mechanical interventions has been reported in an attempt of dispersal or aspiration of the air embolism.[1,2]

Case Report

A 49-year-old, hypertensive and nondiabetic male patient with a history of heavy cigarette smoking was planned for elective percutaneous coronary intervention (PCI) after previous diagnostic coronary angiography due to stable angina and anteroseptal old myocardial infarction in electrocardiogram (ECG). He was never admitted to the hospital for acute myocardial infarction. The diagnostic coronary angiography four months before revealed a 70% proximal stenosis of left anterior descending artery (LAD), 80% mid stenosis of left circumflex artery (LCX), and 60% proximal and 60% mid stenosis of right coronary artery.

Current angiography was performed by a new trainee undertaking cardiac intervention course. The right femoral artery was cannulated with Heartrail II 3.5 7F catheter, engaged at the left main coronary artery. Following cannulation, the patient was given a bolus of 7,000 IU unfractioned intravenous (IV) heparin. Engagement and initial injection of left coronary angiogram was uneventful, showing an 80% proximal lesion of LAD. However, while shooting RAO cranial view, the angiogram showed multiple air emboli were introduced to both LAD and LCX along with the contrast injection.

The patient rapidly became hemodynamically unstable with hypotension and bradycardia and also lost his consciousness. The patient was given 100% oxygen and IV atropine injections and put on IV dopamine. Immediately, an attempt was made to disperse the air emboli into distal coronary circulation by repeated forceful injection of heparinized saline through the catheter. After two minutes, angiography demonstrated no contrast flow beyond the proximal segment of LCX and the mid-segment of LAD, with ECG showing ventricular fibrillation.

Cardiopulmonary resuscitation (CPR) was initiated with vigorous external cardiac massage, while the forceful injection of heparinized saline was continued. DC shock was prepared but before we performed DC shock, ventricular fibrillation resolved spontaneously to sinus bradycardia. The patient gradually recovered his heart rate and blood pressure.

After ascertaining the patient was hemodynamically stable and had fully recovered consciousness, another angiography

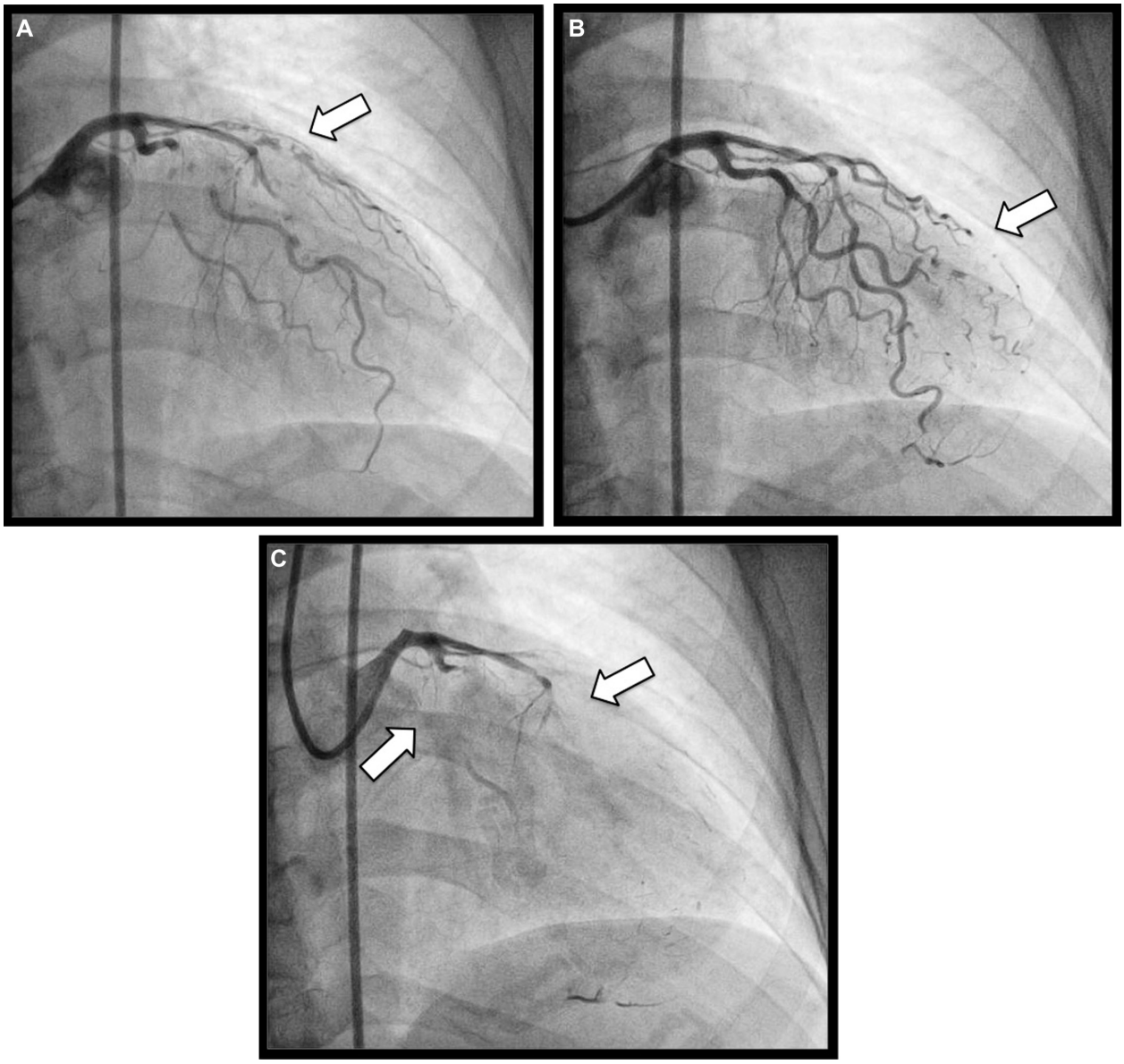

Figure 1. During contrast injection on the next angiogram, multiple air emboli were introduced to LAD and LCX (**A**) and went to distal circulation (**B**). The air bubbles blocked the flow beyond the mid LAD and proximal LCX. The patient then became hemodynamically unstable (**C**).

was taken that showed Thrombolysis in Myocardial Infarction (TIMI)-3 flow and no residual air emboli in both LAD and LCX. PCI procedure was continued, and a 0.014″ Run-through NS Hypercoat guidewire was used to pass the proximal lesion of LAD. After predilatation with Balloon Sapphire II 3.0 × 28 mm, successful stent implantations (Stent DES Combo 3.0 × 28 mm in mid LAD and Stent DES Combo 3.5 × 15 mm in proximal LAD) were made, without residual stenosis or any dissections. The patient was transferred to the intensive care unit; his further hospital stay was uneventful and he was discharged the following day in stable condition.

Discussion

Coronary air embolism is a rare yet preventable complication of coronary angiography and angioplasty. The incidence of this complication depends on the operator's experience and awareness. The estimated incidence of significant coronary air embolism might approach 0.19% in the hands of new trainees, and the total incidence is almost 0.27% when including the unnoticed or unreported asymptomatic air embolisms.[1] Air can be introduced into the coronary arteries inadvertently by inadequate aspiration of the angiographic or guiding catheters, balloon rupture, leakage of air through a defective manifold system, insinuation of air with balloon catheter introduction or withdrawal, structural failures of the equipment, and the constant negative suction of self-venting catheters that are left outside the body.[2]

The diagnosis of air embolism is made angiographically, when discrete bubbles are seen in the coronary artery. It may occlude the coronary artery, where the occluded site often appears vaguely defined rather than discrete vessel cutoff typically seen in artery occluded by thrombus. It may also result in

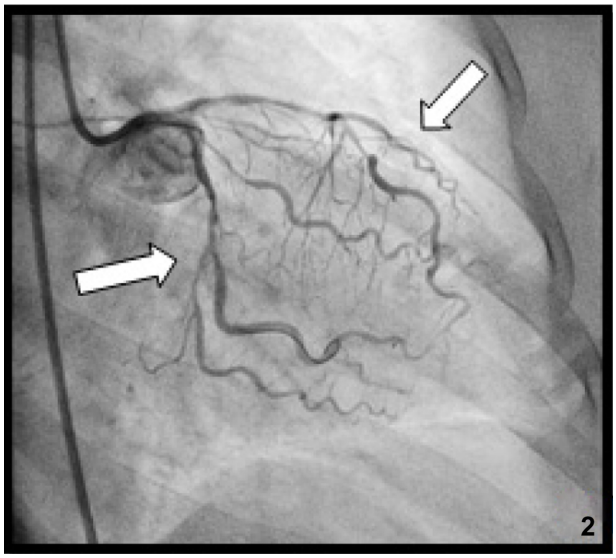

Figure 2. With supportive management and repeated forceful injection of heparinized saline, the patient later fully recovered. The angiogram showed TIMI-3 flow without any residual air emboli in both arteries.

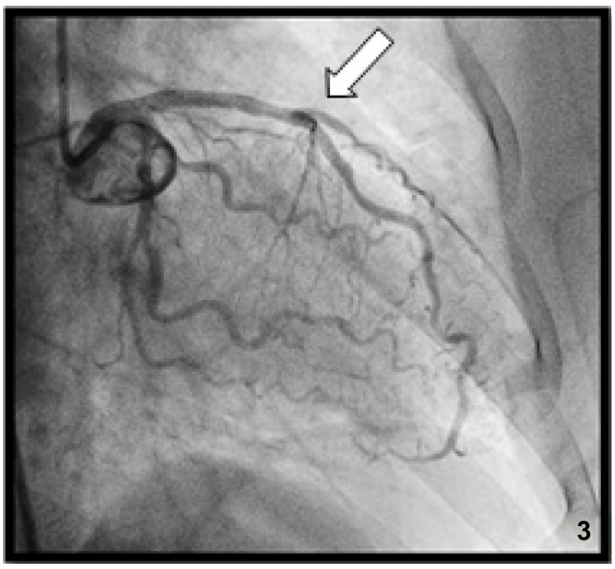

Figure 3. Successful stent implantations in proximal and mid LAD were made, without any complications.

the angiographic appearance of *no reflow* or *slow flow*.[4] In our patient, inadequate aspiration of diagnostic catheter seemed to permit the entrance of air, causing multiple air bubbles in both LAD and LCX that were seen during angiography.

The degree of consequences related to air embolism depends on the amount of air that enters the coronary arteries. Once air is injected, it might develop air lock that prevents perfusion of the distal coronary bed. Air embolism might be asymptomatic, or manifest as chest pain, hypotension, myocardial ischemia, arrhythmias, including bradycardia, heart block, ventricular tachycardia, and fibrillation, and cardiac arrest.[5] A study on dogs showed that intracoronary injection of 0.02 mL/kg of air caused death in 28% of the animals.[6] In another study on pigs, injection of micro air bubbles at a volume of 2 μL/kg to the LAD artery resulted in a significant reduction of segmental contractility.[7] The myocardial injury was determined by the sizes of these micro air bubbles. Multiple air bubbles in our patient caused the slow-flow phenomenon due to blockage of distal perfusion of both left coronary arteries. Initially, it caused hypotension and bradycardia, but later the patient was unconscious and developed ventricular fibrillation. There were no signs of ST elevation or ST depression during the event.

There is no established consensus on the adequate management of air embolism and its complications. The principal management is prevention.[8] Operators should prepare the systems very well before the procedure, aspirate the catheters adequately, and make sure that all the connections are tightened and that manifold is always held in upright position. Most cases include small amounts of air without any hemodynamic consequences, requiring no therapy. For mild to moderate symptoms and all cases, supportive management is given until the air bubbles dissolve spontaneously. This consists of immediate institution of 100% oxygen, analgesics for pain relief, and treatment of arrhythmias.[2] The oxygen helps to minimize ischemia and eliminates nitrogen by diffusing down its concentration gradient and out of the air embolism, so the size of the air bubbles is reduced.[9] In addition, inotropes, intracoronary vasodilators, and intra-aortic balloon pump may be used to maintain the coronary blood flow.[10,11] If massive air embolism occurs, we need to restore the coronary blood flow as quick and safe as possible in order to reduce injury to myocardium and recover from the hemodynamic crisis. Case reports have described mechanical methods including aspiration of the bubbles (*sucking method*),[12] disruption or dislodgement by the guidewire, and forceful injection of saline (*pushing method*) to fragment the air embolus and allow dispersal distally.[13,14] Aspiration has been attempted with diagnostic or guide catheters and export aspiration catheters.[15] Another case showed that intracoronary thrombus aspiration catheter systems can be used safely and harmlessly to resolve air embolism.[16] The aspiration procedure is actually preferable to other methods. Disruption of bubbles by guidewire or balloon may dissect the coronary artery. While the pushing method results in main vessel patency, it may damage distal circulation due to widespread small emboli, causing smaller infarct.[17,18] However, since there is no correct way to treat air embolism, operators should pick any method considered the best in each case.

Our patient had multiple air bubbles in distal segments of both LAD and LCX that blocked the flow beyond proximal segments to distal. When the patient became hypotensive and bradycardic, 100% oxygen was given along with infusion of dopamine and IV atropine injections. We attempted to restore the blood flow by injecting heparinized saline forcefully through the catheter. Later, when the patient developed ventricular fibrillation, we initiated CPR

and continued the forceful injection of saline. For a short time, the patient went back to sinus bradycardia and gained consciousness. These bubbles were introduced during contrast injection on initial angiogram, what we had in hands was only the diagnostic catheter that could not be pushed deep enough to reach the distal emboli. Pushing it in an attempt to break the bubbles might also damage the artery. Aspiration of the bubbles using the diagnostic catheter would take some time, and there was no guarantee that we could aspirate all the bubbles, especially if all were already stayed distally. While using a specific thrombus aspiration catheter would take more time for preparation. There was no guidewire and balloon inside the arteries; therefore, disrupting the bubbles with those was not an option for us. All these methods may be used if the air bubble is single or located in only one artery, or located in proximal segments of the coronary artery. In our case where there were multiple bubbles in the distal parts of two coronary arteries, forceful injection of saline to disperse the bubbles into distal circulation would probably be the best way to restore the flow quickly. It is also possible that continuous chest compression facilitated the dispersion of the air bubbles distally. These along with the supportive measurements worked well and saved the patient.

Summary

We report here a case of multiple air emboli in both LAD and LCX during angiography in a male patient who underwent elective PCI, complicated by cardiogenic shock and ventricular fibrillation. The management included supportive treatments (oxygen, IV dopamine infusion, injection of atropine, and CPR) and forceful injection of heparinized saline. After the patient fully recovered, he underwent successful PCI in LAD without any residual stenosis. Several methods had been advised in the management of massive air embolism to restore the flow, including aspiration of the bubbles, disruption by the guidewire, and forceful injection of saline; operators should pick any method considered the best in each case. The forceful injection of heparinized saline worked well in our case, which was multiple air bubbles in the distal segment, causing total occlusion of both left coronary arteries.

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Author Contributions

Wrote the first draft of manuscript: OS. Contributed to the writing of manuscript: OS, YHO. Agree with result and conclusion: OS, YHO. Jointly developed structure: OS, YHO. Made critical revision and final version: OS, YHO. Both authors reviewed and approved of the final manuscript.

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