

Coronary Air Embolization in the Left Coronary Artery

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Coronary air embolism is a recognized complication of cardiac catheterization occurring in 0.1–0.3% of cases.¹ Sequelae range from various clinically-insignificant events to an acute coronary syndrome and death.² The possible mechanisms of the introduction of air into the coronary artery include incomplete aspiration of angiographic or guiding catheters, balloon rupture, introduction of air with the balloon catheter introduction or withdrawal, and mechanical or structural failures of the equipment. Symptomatic episodes include a rapid onset of chest pain and electrocardiographic (ECG) evidence of myocardial ischemia. This is likely due to the trapping of air within the microcirculation and its eventual spontaneous passage or absorption, allowing restoration of flow. Air emboli are composed of 80% nitrogen and will rapidly decrease in size if the amount of nitrogen is lower. Therefore, inhalation of 100% oxygen facilitates the absorption of the air embolus throughout the microcirculation.

The routine management of mildly-to-moderately symptomatic patients consists of treating chest pain with analgesics, oxygen inhalation, and supporting them as necessary for the few minutes it takes for the air to clear spontaneously by either absorption or diffusion within the microcirculation. The air embolism in the coronary circulation may have more serious consequences such as bradycardia, hypotension, asystole, and ventricular fibrillation may result and require immediate resuscitation, temporary pacing, intra-aortic balloon pump counterpulsation, electrical defibrillation or respiratory support. When there is a large amount of air involved, an attempt to aspirate the air is suggested. Also, by breaking up the larger air mass into a greater number of smaller bubbles can increase the surface area. This will lead to a dispersion of the bubbles and an increase in the absorption of the air and resolution of the bubbles within the coronary circulation.

Another treatment strategy is forceful injection of saline or contrast medium to propel the air bubbles to the terminal portion of the coronary artery to restore adequate coronary blood flow. Disruption or dislodgement by the guidewire or over-the-wire balloons or the angiography catheter itself and the forceful injection of saline are aimed at fragmenting the air embolus to allow dispersal or to force it distally.^{3,4} Various attempts at mechanical interventions to cause dispersal or use of nondedicated equipment for aspiration have been described.

Such interventions may result in main-vessel patency and have the potential to cause damage in the main vessel, *e.g.*, the left main coronary artery, during aggressive maneuvers and to damage the distal microvasculature due to widespread, smaller embolizations. The use of an angiographic catheter as an aspiration tool is limited by the ability to manipulate it deeply enough into the coronary tree to affect the embolus. This also predisposes the coronary artery to mechanical damage of the artery, increasing the risk of dissection, perforation, thrombus formation and complete occlusion. In contrast, aspiration aims at resolving the blockage by removing the air that is trapped in the visible epicardial vessel. However, the treatment has consisted mainly of supportive measures, the use of 100% oxygen (to minimize ischemia and to establish a diffusion gradient encouraging elimination of gas from the bubble), and pain relief.^{3,4} Also, the placement of the patient in a left lateral decubitus position has been proposed as a mechanism to facilitate the breakup and movement of the air embolus down the coronary circulation.³

Case presentation. *A 62-year-old female with a past medical history of hypertension, diabetes, hypercholesterolemia and a positive family history of coronary artery disease was admitted for elective coronary catheterization as part of the investigation of a positive stress test showing hypokinesia of the anterior and anterolateral wall. Angiography was performed by the general cardiology fellow who was in training for basic cardiac catheterization techniques. The patient's right femoral artery was accessed. According to routine hospital procedure, the patient received an intravenous benadryl bolus of 25 mg for sedation. Standard JRA and JLA diagnostic catheters (Cordis Corp., Miami, Florida) were used. The cardiology fellow flushed the catheter and the manifold. On engagement and initial injection of the left coronary artery (LCA), angiography showed a stump in the position of the proximal left anterior descending artery (LAD) and proximal left circumflex artery with contrast slowly eddying in these proximal vessels. It appeared that the manifold may have contained air bubbles that were inadvertently injected into the LCA. The contrast column terminated at an equal distance from the left main artery in all of the LCA branches. The patient immediately began to experience severe chest pain and her ST segments in leads V3–V6 began to rise, subsequently peaking at 3 mm.*

The interventional cardiology attending physician immediately attempted to aspirate the air from the left coronary artery. However, this did not alleviate the patient's symptoms nor the air embolism in the coronary circulation. The patient was immediately given 100% oxygen, intravenous neosynephrine (100 µg), and a right femoral venous access sheath was placed for a transvenous pacemaker. The patient's blood pressure was not palpable, nor was the registration of the blood pressure by the

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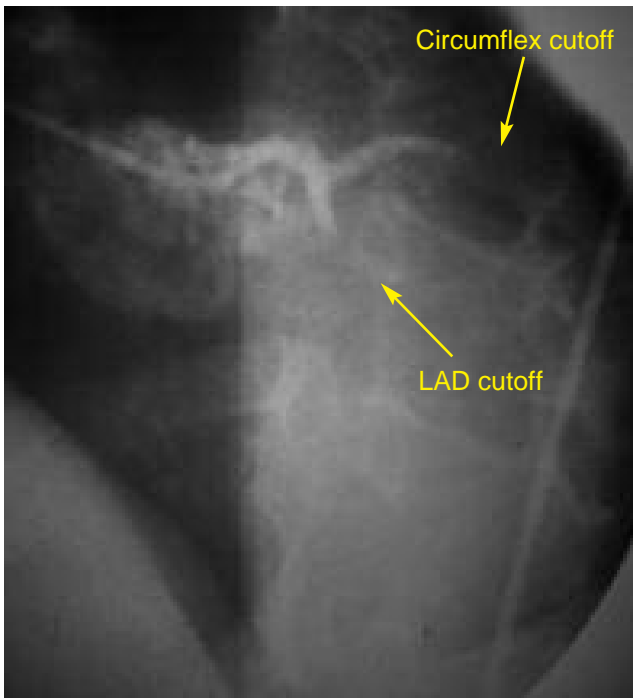


Figure 1

catheterization manometer appreciated. A code was called and the patient underwent an initiation of advanced cardiac life support protocol (ACLS) with intubation and vigorous CPR performed by staff. The patient received intravenous neosynephrine and epinephrine to try to support her blood pressure during the ACLS. After two minutes of ACLS protocol, the patient experienced tachycardia, and her blood pressure increased immediately after resuscitative attempts were instituted. The patient was able to be immediately extubated after her blood pressure stabilized and the ACLS was terminated. The patient remained tachycardic at 134 beats per minute, with a blood pressure of 247/119 after ACLS protocol. The patient then underwent intubation of the left coronary artery with a JLA which revealed resolution of the air embolism that was seen during initial angiography in which the entire left coronary artery system was filled with contrast. The patient's symptoms rapidly disappeared and her electrocardiogram (ECG) normalized. Angiography showed TIMI 3 flow into a branching distal territory with some spasm where the occlusion was visualized. Catheterization of the right coronary artery was uneventful, showing a 70% right posterior descending artery lesion. The lesion in the right coronary artery was immediately stented with excellent angiographic results showing TIMI 3 flow.

The patient had an uneventful overnight stay and was discharged the following day with a normal ECG. The cardiac

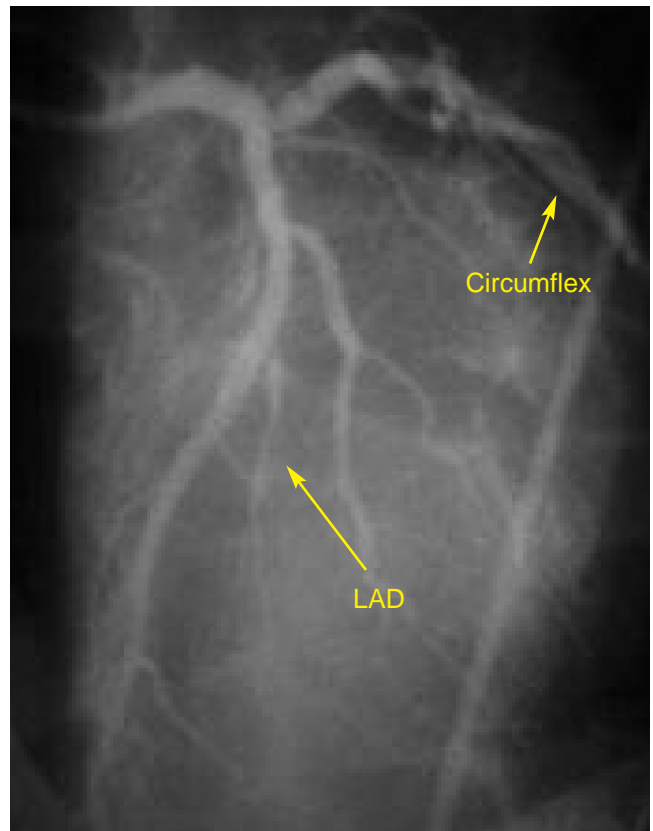


Figure 2

enzymes increased slightly to a peak of troponin of 1.5. The patient was seen by the interventional attending physician's clinic later that week without any complaints.

Conclusion

Air embolization during a cardiac catheterization or a percutaneous coronary interventional procedure can and must be avoided to prevent morbidity and mortality. With continued training of general cardiology fellows in the proper technique and the careful management of equipment and attention to flushing and injection technique, these potential catastrophic events can be avoided.

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