A 60-year-old man underwent coronary angiography because of exertional angina and was found to have a 90% mid–left anterior descending artery lesion (Figure 1A). A 2.5×20-mm paclitaxel-eluting stent was positioned in the lesion. When contrast was injected to verify the stent position, air was introduced into the guide catheter through a loose manifold connection, resulting in a massive coronary air embolism (Movie and Figure 1B). Because the undeployed stent was occlusive, there was no bubble embolization in the distal left anterior descending artery. After 20 s, the patient developed ST elevation and hypotension (Figure 2A), followed by a short period of pulseless electrical activity (Figure 2B). The guide was vigorously aspirated and the stent was deployed. The patient received chest compressions, oxygen and intracoronary atropine, and epinephrine, resulting in complete hemodynamic recovery (5 minutes after initial air embolism) and TIMI (Thrombolysis In Myocardial Infarction) 3 flow in the left coronary artery. The patient had an uneventful recovery, and his troponin-T level increased from 0.01 ng/mL before the procedure to 0.23 ng/mL the following day. Left ventricular wall motion was normal as assessed by transthoracic echocardiography.

There are few case reports of coronary artery air embolization in the literature, likely because of the underreporting of this complication. Massive air embolism seems to be a rare event. Coronary air embolism has been reported to cause chest pain, ST segment elevation, bradycardia, hypotension, and ventricular fibrillation. Our case demonstrates that hemodynamic compromise can occur within seconds and may resolve within a few minutes after prompt treatment, which includes aspiration of the air if possible, 100% oxygen administration and (often intracoronary) atropine, and epinephrine injections.

Careful preparation of the manifold angioplasty balloons and guiding catheters is crucial to prevent this potentially life-threatening complication.
Figure 1. Diagnostic coronary angiography of the left anterior descending artery in the right anterior oblique cranial projection, showing (A) a severe mid–left anterior descending artery lesion (black arrow) and (B) several coronary intraluminal filling defects attributable to massive air embolism.
Figure 2. Electrocardiography and coronary guide used to obtain blood pressure immediately after air embolism (A) showing rapid development of hypotension, followed by the onset of pulseless electrical activity (B).
Hemodynamic Consequences of Massive Coronary Air Embolism
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