Device-Based Left Atrial Appendage Closure

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A 56-year-old man with symptomatic paroxysmal atrial fibrillation refractory to both conventional antiarrhythmic drug therapy and catheter ablation was referred for transcatheter left atrial appendage (LAA) closure. The presence of hypertrophic cardiomyopathy with enlarged left atrial diameter (50 mm), heart failure, recurrent episodes of refractory atrial fibrillation, previous transient ischemic attacks, and hypertension led to a high annual predicted risk of stroke based on CHADS2 (congestive heart failure, hypertension, and diabetes mellitus, prior stroke or transient ischemic attack) criteria (score 4) and HAS-BLED (hypertension, abnormal renal/liver function, stroke, bleeding history or predisposition, labile international normalized ratio, elderly, drugs/alcohol concomitantly) (score 4). The patient also experienced gastrointestinal bleeding. Therefore, a transcatheter LA occlusion was attempted with the AMPLATZER Cardiac Plug (ACP) system, and throughout the procedure, the activated clotting time was constantly maintained ≥350 seconds. During implantation, 2 dramatic complications not related to each other developed, which included a massive coronary air embolism and an LAA dissection followed by fast-growing thrombus formation.

Massive Coronary Artery Embolism

Immediately after the deployment of the distal lobe of the ACP, the patient developed a marked hypotension (online-only Data Supplement Movie 1). The ST segment began to rise first in leads II, III, and aVF and then a few seconds later in leads V3 to V6, peaking to 7 mm and suggesting a global myocardial ischemia (Figure A). Because no proximal embolization was detected at coronary arteriography, a massive distal coronary air embolization was suspected. The patient received 100% oxygen administration and intravenous atropine, after which his blood pressure increased and stabilized. Two rapid, forceful injections of contrast medium administered with great caution into the coronary arteries resulted in immediate resolution of the ECG abnormalities and transient atrial fibrillation (Figure B), which began from the anterior leads because the left coronary artery was injected first.

Atrial Dissection

After resolution of the coronary embolism and 3 attempts of ACP positioning in the LAA, an endocardial dissection followed by an acute, fast-growing thrombus formation occupying most of the LAA space developed at the impact of the guiding catheter against the LAA wall (online-only Data Supplement Movie 2). The thrombus was completely entrapped by immediate and correct ACP implantation without residual shunt in the LAA, which was confirmed by the absence of a paradevice leak, an appropriate separation between the lobe and the disc, with a concave disc apposition. Pericardial effusion was excluded by serial echocardiograms at the time of and after the procedure. The patient had an uneventful overnight stay and was discharged the following day with a normal ECG without neurological defects as documented by CT scan. There were no adverse events at 5-month follow-up.

Discussion

Of a total of 27 ACP implantations performed in our laboratory, 2 life-threatening, uncommon complications developed in 1 patient (as reported here), but common transient complications occurred in 4 other patients. These findings, although confirming that LAA closure is a complex procedure, clearly extend the initial European experience that occurrence of serious complications with the ACP system are not uncommon. To our knowledge, global myocardial ischemia as a complication of ACP implantation has not been reported previously. This unusual complication was due to massive air emboli at the time of device advancement despite an accurate preparation to ensure that all air was purged. Because ST elevation was diffuse, simultaneous left- and right-side coronary air embolism was considered the cause. Air formation may inadvertently occur during the ACP system preparation and advancement, and the possible mechanisms include incomplete aspiration of guiding catheters; larger dead space in 13-F sheets with potential risk of air trapping; and ex vacuo air formation while advancing the large, fully deflated plug catheter into a cavity at low pressure, such as the left atrium. After this potentially lethal
complication rapidly develops, our experience suggests that to prevent the occurrence of potentially malignant arrhythmias, myocardial infarction, and death, an immediate restoration of coronary flow is required by supportive management of 100% inspired oxygen associated with rapid injections of saline solution or contrast medium, which should be used with great caution to avoid coronary dissection. In this patient, ECG abnormalities completely disappeared beginning from the first injected coronary artery, which forced emboli more distally with rapid resolution of coronary flow. LAA dissection with acute thrombus formation was another potentially lethal complication, but the prompt and correct device deployment at the beginning of thrombus formation was able to completely entrap the thrombus within the LAA. LA dissection was probably due to several attempts of device positioning, and this complication suggests limiting the number of attempts. The patient was discharged the day after the procedure, and there were no late complications at 5-month follow-up. Our experience suggests that patient selection is very important and should exclude patients at low risk of stroke.

**Disclosures**

None.

**References**


**Key Words:** arrhythmia | atrial fibrillation | stroke

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**Figure.** A, A marked ST-segment elevation is evident in all ECG leads, particularly in leads I, II, and aVF. B, Immediate normalization of ECG abnormalities after forceful injections of intracoronary contrast medium.
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