A 63-year-old woman was urgently transferred to our institution from a regional hospital with no facilities for cardiac catheterization after she presented with sudden-onset nausea and left arm pain associated with transient inferior ST-segment elevation and lateral ST-segment depression on ECG. Seven weeks earlier, she had undergone elective 3-vessel coronary artery bypass grafting when an abnormal dipyridamole 99mTc-sestamibi scan demonstrated reversible myocardial ischemia but normal left ventricular function. Surgical cryoablation (box lesion isolation of the pulmonary veins, with targeted ablation of the left atrium and left atrial appendage) was also successfully performed to treat persistent atrial fibrillation (AF). Postoperatively, she underwent anticoagulation therapy with warfarin; enoxaparin was also administered until her international normalized ratio was ≥2.0. Continuous telemetry confirmed that normal sinus rhythm was maintained during her entire postoperative stay. She continued taking warfarin on discharge, and her international normalized ratio was monitored regularly. Her comorbidities included type 2 diabetes mellitus, hypertension, Graves’ disease, and chronic kidney disease.

On examination, she was in normal sinus rhythm (heart rate 70/min) and hypertensive (blood pressure 155/90 mm Hg). Her cardiovascular and respiratory examination was normal. Her initial troponin level was 3954 ng/L (normal: <14 ng/L). Despite taking warfarin, her international normalized ratio was subtherapeutic (international normalized ratio 1.1). She received aspirin, clopidogrel, and intravenous heparin before transfer. Coronary angiography revealed multiple filling defects in a previously patent, large, ungrafted intermediate branch, consistent with coronary artery embolism (Figure 1 and online-only Data Supplement Movie I). Thrombus extraction was performed, and a drug-eluting stent was deployed after intravascular ultrasound showed moderate residual stenosis. Histopathological examination of the extracted material demonstrated fibrin thrombi with no evidence of organization or granulation tissue, consistent with recent embolism.

Because of her history of persistent AF and recent AF ablation, transeosophageal echocardiography was performed on the suspicion that the coronary artery embolism was from an intracardiac source. Although there was mild impairment in overall left ventricular contraction (ejection fraction 45%) secondary to inferior wall hypokinesia, no left ventricular mural thrombus was found. The atrial septum was intact. A 21×12-mm thrombus, however, was noted within the left atrial appendage (Figure 2 and online-only Data Supplement Movie II). Despite the presence of sinus rhythm, there was impaired left atrial mechanical function (peak left atrial appendage velocity 15 cm/s; Figure 3) associated with moderate spontaneous echocardiographic contrast (online-only Data Supplement Movie III). As a result, warfarin was continued in addition to antiplatelet therapy.

Coronary artery embolism is an uncommon but important cause of ST-segment elevation myocardial infarction. Its diagnosis is suspected during coronary angiography when multiple
filling defects are present in an otherwise apparently normal coronary artery. Sources of emboli include conditions that predispose to left atrial thrombus (e.g., AF), left ventricular mural thrombus (e.g., myocardial infarction, severe cardiomyopathy), valvular vegetations (e.g., infective endocarditis), and paradoxical thromboembolism through defects in the atrial septum. On diagnosis, transesophageal echocardiography should be performed to detect intracardiac sources of embolus.

Risk factors for left atrial thrombus in the presence of normal sinus rhythm include (1) poor atrial mechanical function caused by recent cardioversion, ablation, or AF-related atrial cardiomyopathy and (2) conditions associated with hypercoagulable state. Although surgical ablation significantly increases the chances of maintaining sinus rhythm in patients with AF, severe impairment in left atrial mechanical function is common even if sinus rhythm is achieved postoperatively, and this is associated with an increased risk of thromboembolic complications. Occlusion or excision of the left atrial appendage is often considered at the time of surgical ablation, although individual practice varies because of uncertainty regarding efficacy in reducing future thromboembolism. Antiarrhythmic agents and anticoagulation should be continued for at least 3 months after AF ablation, with cessation considered only if ECG and echocardiographic assessment demonstrate normal atrial function. Transesophageal echocardiography after AF ablation can help determine the duration of anticoagulation, because the presence of poor left atrial mechanical function is a strong predictor of future stroke.

Disclosures

None.

References


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