Postablation asymptomatic cerebral lesions: Long-term follow-up using magnetic resonance imaging

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BACKGROUND Catheter ablation of atrial fibrillation (AF) is complicated by cerebral emboli resulting in acute ischemia. Recently, cerebral ischemic microlesions have been identified with diffusion-weighted magnet resonance imaging (MRI).

OBJECTIVE The clinical course and longer-term characteristics of these lesions are not known and were investigated in this study.

METHODS Of 86 patients, 33 (38%) had new asymptomatic cerebral lesions documented on MRI after catheter ablation for AF; 14 of these 33 (42%) underwent repeat MRI at different time intervals (2 weeks to 1 year) during follow-up, and clinical symptoms as well as size and number of residual lesions were documented.

RESULTS In postablation cerebral MRI, 50 new lesions were identified (3.6 lesions/patient) in 14 patients. No patient presented any neurological symptoms. Distribution of the lesions was predominantly in the left hemisphere (60%) and the cerebellum (26%); 52% of the lesions were small (≤3 mm maximum diameter), 42% were medium (4 to 10 mm) and 3 lesions (6%) had a maximum diameter >10 mm. Follow-up MRI after a median of 3 months revealed 3 residual lesions in 3 of 14 patients corresponding to the large acute postablation lesions (>10 mm). The remaining 47 of 50 (94%) of the small or medium-sized lesions were not detectable at follow-up evaluation.

CONCLUSIONS Most asymptomatic cerebral lesions observed acutely after AF ablation procedures were ≤10 mm in diameter. 94% of all lesions healed without scarring at follow-up >2 weeks after ablation. The larger acute lesions produced chronic glial scars. Neither chronic nor acute lesions were associated with neurological symptoms.

KEYWORDS Cerebral microlesions; Atrial fibrillation ablation; Magnet resonance imaging; Radiofrequency ablation; Silent cerebral ischemia

ABBREVIATIONS **ACT** = activated clotting time; **AF** = atrial fibrillation; **CFAE** = complex fractionated atrial electrogram; **CV** = cardioversion; **DWI** = diffusion-weighted echo-planar imaging; **FLAIR** = fluid-attenuated inversion recovery; **INR** = international ratio; **LMWH** = low-molecular-weight heparin; **MRI** = magnetic resonance imaging; **PVI** = pulmonary vein isolation; **RF** = radiofrequency; **SCL** = silent cerebral lesions

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Introduction

Catheter ablation of atrial fibrillation (AF) has evolved as a second-line therapeutic option in patients with symptomatic AF after antiarrhythmic drug therapy.[1–3] Systematic magnetic resonance imaging (MRI) conducted 24 to 48 hours after ablation revealed a 14% to 40% incidence of clinically asymptomatic cerebral lesions.[4–7] The primary objective of this study was to evaluate the clinical consequences and longer-term characteristics of the lesions in patients with acutely detected ischemic embolic lesions after catheter ablation of AF.

Methods

Consecutive patients with silent cerebral embolism documented in cerebral MRI scans conducted 1 to 2 days after left atrial AF ablation procedures were asked to participate and included if willing to undergo a follow-up cerebral MRI. Patients were excluded if additional left atrial ablation procedures or cardioversions (CV) were performed between the postinterventional and the follow-up MRI. A complete neurological examination was performed in all patients after ablation at the time of acute and follow-up MRI by an experienced neurologist aware of the MRI results.

All patients underwent cerebral MRI scanning 1 day before and 1 to 2 days after the ablation procedure, and once during follow-up (0.5 to 12 months after ablation procedure). The study was approved by the local hospital ethics committee. All patients gave informed consent.

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Cerebral MRI

All cerebral MRI scans were analyzed independently by 2 radiologists experienced in the field of cerebral MRI imaging. No differences in evaluation, classification, and outcome results were noted between the 2 experts.

All patients underwent cerebral MRI scanning before the ablation procedure to identify any cerebral pathologic findings relevant for this study. Cerebral MRI was performed using a 1.5-T device (Siemens, Erlangen, Germany). The imaging protocol for all images consisted of a T2-weighted axial fluid–attenuated inversion recovery (FLAIR) sequence (TI = 2,500 ms, TR = 9000 ms, TE = 125 ms, slice thickness 4.0 mm, in-plane resolution: $0.9 \times 0.9 \times 4.0$ mm, flip angle: 160°) and a diffusion-weighted echo-planar imaging (DWI) sequence (TR = 3,700 ms, TE = 105 ms, in-plane resolution: $1.2 \times 1.2 \times 6.0$ mm). For each DWI sequence, the apparent diffusion coefficient map was obtained. DW-MRI imaging has been shown to be extremely sensitive to acute cerebral ischemia.[8] Additionally, after ablation and during follow-up, a T2- and PD-weighted imaging sequence (TR: 3,190 ms, TE: 118 (T2) 15 (PD)ms, in plane resolution: $0.8 \times 0.7 \times 6.0$ mm, flip angle: 30°) were also performed. PD scans were performed additionally in postablation follow-up MRI to increased sensitivity with regard to possible acute postischemic lesions and bleedings. Especially, Virchow Robin spaces can be differentiated is a more sophisticated manner compared to regular T2 measurements.

Ablation procedure

All patients gave informed consent for catheter ablation of AF. Before the procedure, left atrial thrombus was ruled out by transesophageal echocardiography on the day of the procedure. Oral anticoagulation was stopped before the ablation procedure, and international ratio (INR) values were checked to be ≤ 2.0 on the day of ablation. If INR was ≤ 2.0, low-molecular-weight heparin (LMWH) was administered in body-weight-adjusted dosage to bring it to the therapeutic range of >2.0. In patients without oral anticoagulation, LMWH was administered after admission to the hospital. After the procedure, all patients continued to receive LMWH in addition to oral anticoagulation until INR of >2.0 was documented.

Ablation procedures were carried out under intensive anticoagulation using an intravenous heparin bolus injection after transseptal access to the left atrium. Pulmonary vein isolation (PVI) was performed either using multielectrode ablation catheters and duty-cycled phased radiofrequency (RF) technology or electroanatomical mapping and unipolar cooled-tip RF ablation as previously presented.[9–12] Phased duty-cycled RF ablations were performed using multielectrode catheters (PVAC, Medtronic, Carlsbad, California) in 2:1 bipolar-to-unipolar ratio with a target temperature of 60°C and a maximum power of 10 W. Multiple overlapping ablations per vein were performed to produce bidirectional PV block.[9,10] In cases with electroanatomical mapping (CARTO3, Biosense Webster, Diamond Bar, California), PVI was performed after left atrial anatomic mapping using a decapolar circular mapping catheter (LassoNav, Biosense Webster). Pulmonary vein encircling ablation was performed in a point-by-point ablation pattern using cooled-tip ablation catheters (EZ-SteerNAV, Biosense Webster) (target power 25 to 30 W, target temperature 48°C, irrigation during ablation 30 ml/min).[11,12]

Wide circumferential PVI was performed in all patients, and PVI was validated using differential pacing from the coronary sinus, left atrial appendage, and superior caval vein using a circumferential mapping catheter in the PV and additional stimulation catheter directed toward the different locations.

In patients with persistent AF, additional ablation of complex fractionated atrial electrograms (CFAE) was performed at the discretion of the operator. Using multielectrode catheters and duty-cycled RF energy, CFAE ablation was performed at the left atrial septum (MASC, Medtronic) and left atrial body (MAAC, Medtronic) (1:1 bipolar-to-unipolar ratio, target temperature 60°C, maximum power 10 W). Using cooled-tip RF ablation (specifications see earlier) was performed with a staged protocol (including linear left atrial ablations at the roof and the mitral isthmus and ablation of CFAEs in the left and right atrium) as previously reported.[12,13]

Activated clotting time (ACT) was checked every 20 minutes, and ablation was interrupted if ACT <300 seconds was documented. In such cases, additional heparin was given and ACT rechecked to be >300 seconds before resuming ablation. Minimum ACT was registered for each patient.

If patients were in AF at the beginning of the procedure but no persistent AF (>6 months) was documented, patients were cardioverted after transseptal access to the left atrium and ablations were performed in sinus rhythm. Patients with documented AF >6 months were not cardioverted before ablation. The number of direct current-shock CVs was documented. CV was only performed after documenting an ACT >300 seconds at least once but no ACT monitoring directly before CV was undertaken. In addition, the number of patients with AF during the ablation procedure (either transient with conversion to sinus rhythm without CV or persistent and undergoing CV), duration of RF energy delivery, and procedure duration were recorded. All patients were restarted on oral anticoagulation in the evening postablation and were bridged until effective using LMWH in adjusted dosage.

Preablation MRI

Patients underwent cerebral MRI scanning the day before the ablation procedure. Any abnormalities were documented and compared to postinterventional MRIs.

Postinterventional MRI

All patients underwent cerebral MRI scanning 1 to 2 days after the ablation procedure to document any new cerebral lesions. Acute embolic lesions were defined as a focal hy-