Accelerated idioventricular rhythm after left atrial tachycardia ablation as a marker of acute coronary ischemia

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Case report

A 43-year-old man with multiple previous left atrial ablation procedures for persistent atrial fibrillation, including pulmonary vein isolation and mitral and roof lines, presented for a repeat ablation of persistent atrial tachycardia (Figure 1A). He had no other medical history, and the procedure was performed on uninterrupted warfarin, as a routine practice at our institution. Echocardiography before the procedure confirmed a structurally normal heart with no regional wall motion abnormalities. He underwent transesophageal echocardiogram after induction of anesthesia to exclude left atrial appendage thrombus and guide 2 transseptal punctures with fluoroscopy. CARTO 3 (Biosense Webster, Inc, Diamond Bar, CA) was used to generate an electroanatomic map of the left atrium (Figure 1B). Atrial tachycardia was demonstrated to be a perimitral flutter with floor to roof activation of the left atrium on the anterior and posterior walls. The tachycardia was entrained from the distal coronary sinus (CS) with a postpacing interval of 0 ms.

Endocardial radiofrequency (RF) energy was delivered using an irrigated SmartTouch (Biosense Webster) F curve catheter to the mitral isthmus, which had recovered conduction since his previous ablation procedure. Despite this, the mitral isthmus was not blocked, so epicardial RF energy was delivered within the distal CS (Figure 1B) with settings to deliver a maximum of 20 W of power and a maximum temperature of 43°C with high-flow irrigation at 25 mL/min. In total, 103 seconds of RF energy was delivered epicardially. There was no unusual catheter rotation required in the CS during ablation, and throughout the procedure all anatomical areas were accessed easily and without incident. The average temperature achieved in the CS was 39°C with a mean power of 16 W. This resulted in the termination of the perimitral tachycardia and initiation of a focal atrial tachycardia indicated by a change in the atrial activation pattern. This focal atrial tachycardia was terminated to sinus rhythm with ablation anterior to the right inferior pulmonary vein. Subsequent burst pacing induced cavotricuspid isthmus–dependent atrial flutter, which was treated with a right-sided cavotricuspid isthmus line. During the whole procedure, the total duration of RF ablation was 47 minutes 7 seconds, with the average temperature reached being 39°C and an average power of 23 W. The activated clotting time was maintained above 300 seconds throughout the procedure using intravenous heparin.

There was no hemodynamic disturbance during the procedure; an echocardiogram showed no effusion, and the patient was extubated. After extubation in the recovery suite, the patient complained of chest discomfort, which was felt to be pericardial in nature. His electrocardiogram (ECG) showed an accelerated idioventricular rhythm (Figure 2A). He was given analgesia with morphine and atropine (600 μg) in view of the rhythm, which reverted to sinus rhythm, and the ECG after this showed no ischemic changes (Figure 2B). He continued to experience chest pain, now associated with up to 0.1 mV of ST-segment depression in leads V_4-V_6 at 40 minutes after reversion to sinus rhythm (Figure 3A).

Coronary angiography was performed immediately. A severe focal stenosis of a large dominant circumflex vessel after the origin of a large obtuse marginal was identified with TIMI grade 2 flow (Figure 2C). There was no evidence of significant coronary atheroma elsewhere. There was no response to intracoronary nitrates; therefore, we proceeded to balloon angioplasty followed by drug-eluting stent deployment (Abbott's XIENCE 3.5 mm × 23 mm; Figure 3C). The patient recovered from pain, and his ECG returned to baseline (Figure 3B). Subsequent echocardiography did not demonstrate any significant regional wall motion abnormality, and the patient was discharged 48 hours later on warfarin and dual antiplatelet therapy.

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KEY TEACHING POINTS

- Catheter ablation, in particular epicardial radiofrequency ablation, is occasionally associated with coronary artery injury, which may result in clinically significant coronary ischemia. When managing patients undergoing left atrial ablation procedures, vigilance should be maintained for clinical indicators of ischemia. Patients are often under general anesthesia, and so patient reporting of symptoms may not occur until a later time.

- Accelerated idioventricular rhythms are a marker of possible coronary reperfusion. When seen in patients after left atrial ablation, they should prompt careful consideration of the possibility of coronary artery injury and a low threshold for emergency coronary angiography should be maintained.

- Cardiac injury after radiofrequency ablation can be mediated by multiple possible mechanisms including coronary artery injury, direct myocardial tissue damage, and coronary embolization. Careful titration of radiofrequency energy, particularly on epicardial surfaces, and tight monitoring of intraprocedural anticoagulation may minimize the risk of these, but vigilance for such complications must be maintained even when there are no clear procedural factors likely to predispose to these processes.

Discussion

Circumflex artery damage is a well-recognized complication of RF energy delivery during ablation procedures.[1,2] It is more common in the setting of complex left atrial ablation.[3,4] It has been reported to result in acute sinus node dysfunction requiring permanent pacing as well as ventricular fibrillation[5] and the proximity of the artery to anatomical structures was relevant to the clinical outcome when assessed in pathological specimens.[6] Wong et al[4] report acute arterial injury in 28% of patients undergoing left atrial ablation procedures. Arterial changes seen on angiography were associated with epicardial CS ablation, where the circumflex artery is in closest anatomical proximity to the site of ablation. Risk of circumflex injury was associated with longer RF duration in the CS and total RF delivery. However, all injuries were subclinical and resolved with intracoronary nitrates, suggesting that the predominant mechanism behind angiographic abnormalities was coronary spasm. In our patient, the location of the focal severe circumflex lesion correlated with the location of the epicardial RF energy application. The failure to resolve with nitrates in our patient suggested coronary spasm was not solely responsible for the electrocardiographic, angiographic, and clinical features present. Porcine studies have demonstrated acute and chronic histopathological changes in coronary arteries subject to applied RF energy which suggest that additional mechanisms to coronary artery spasm may be involved.[7] There are published reports on coronary occlusion requiring treatment with percutaneous coronary intervention, with encouraging results at 2-year follow-up.[8] Following RF ablation in the left atrium, there are multiple possible mechanisms that could mediate coronary ischemia. These should be considered among the differential diagnoses for the underlying cause when there are any features indicating ischemia. Other possible mechanisms include coronary embolization[9] and direct myocardial injury.[10] It has been demonstrated that during ablation procedures, significant energy is delivered to the atrial blood pool and this can result in the formation of coagulum,[10] which may embolize. This process is recognized to occur in the cerebral circulation where it has been more systematically
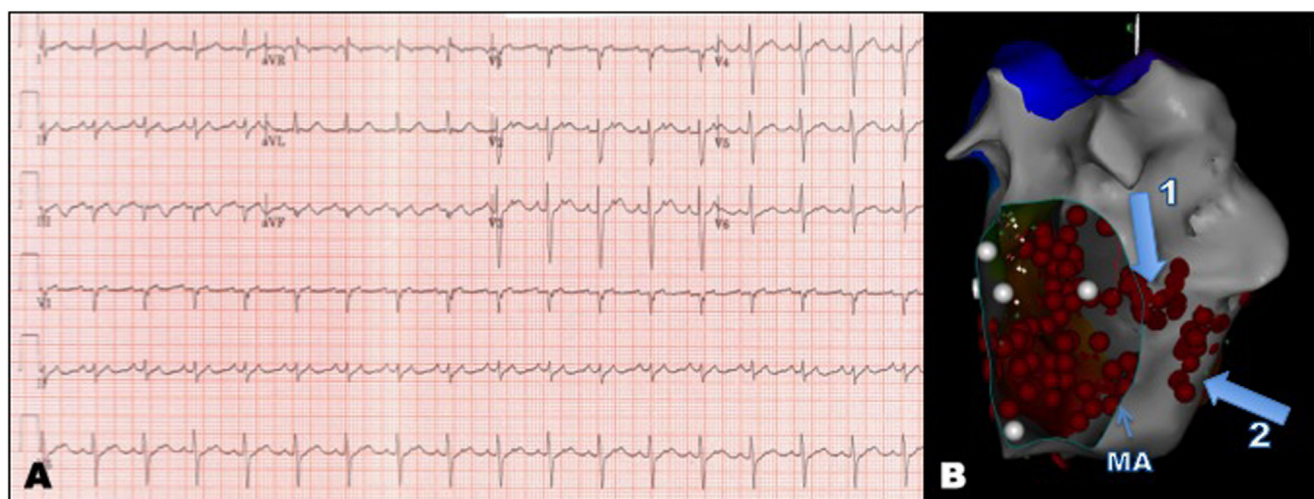

Figure 1   **A:** Electrocardiogram demonstrating atrial tachycardia causing symptoms before the ablation procedure. **B:** CARTO 3 anatomy (electro-anatomic mapping system used to generate visual representation of left atrial anatomy) in the left lateral view and location of radiofrequency lesions. Arrows indicate endocardial mitral isthmus ablation (1) and epicardial radiofrequency ablation within the coronary sinus (2). The mitral annulus (MA) is also indicated. Epicardial placement of lesions is likely to have increased the risk of direct circumflex artery injury such as that experienced by our patient.
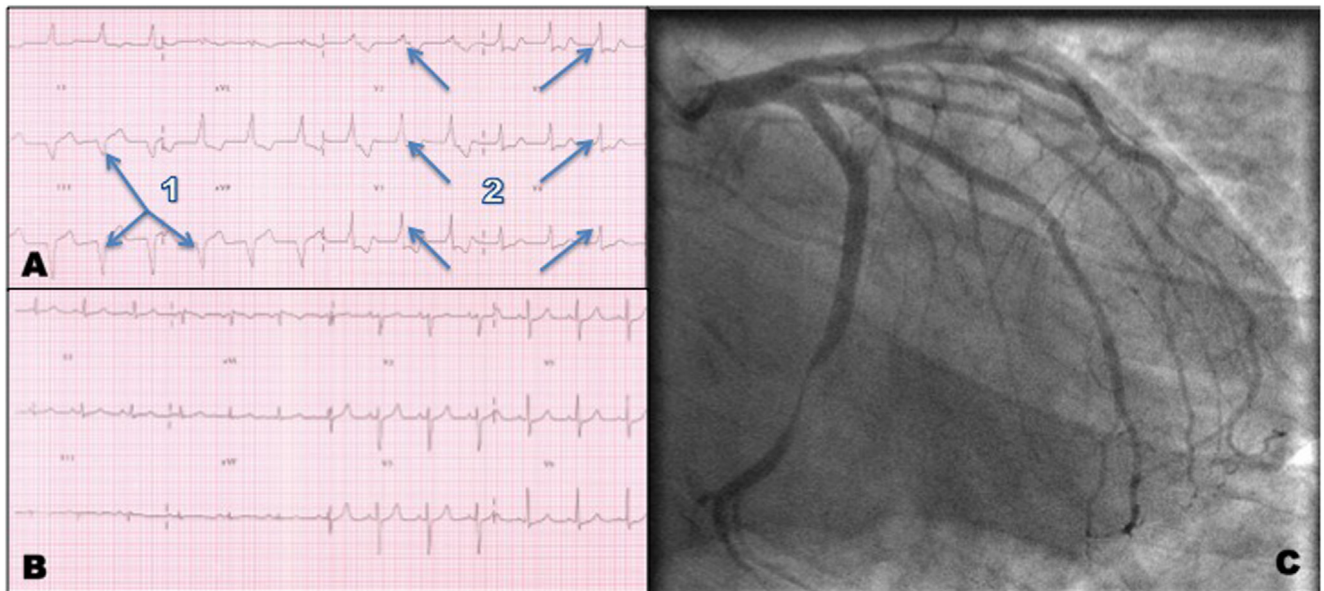
Figure 2   **A:** Accelerated idioventricular rhythm after radiofrequency ablation procedure: the first indication of significant coronary ischemia. The morphology of the QRS complex suggests an inferoposterior focus for the rhythm because of the superior axis (arrows 1) and the precordial positive concordance (arrows 2), which would localize it to the site of the identified circumflex stenosis and the site of epicardial radiofrequency ablation in the coronary sinus. **B:** Electrocardiogram in sinus rhythm after the administration of atropine and morphine (12 minutes after electrocardiogram shown in panel A). **C:** Coronary angiography in left anterior oblique 30°/caudal view, demonstrating severe distal circumflex stenosis at the site correlating with the application of epicardial RF application.

investigated[11] and where identification of ischemic tissue is not confounded by the possibility of direct arterial injury. In our case, we felt that the coronary appearance most favored direct coronary artery injury. In addition, the location of the discrete circumflex artery lesion correlated closely with the site of epicardial RF energy application.

The notable aspect of this case is the accelerated idioventricular rhythm after the procedure, which resolved with the appearance of ischemic changes in the ECG obtained after this. Accelerated idioventricular rhythms have been

recognized as markers of reperfusion after restoration of coronary flow since the thrombolytic era[12] when they were observed frequently, and in combination with other ECG and clinical factors, they can be a useful indicator of successful treatment of ST-segment elevation myocardial infarction.[13] The presence of an accelerated idioventricular rhythm was the first indication of a clinically significant event in our patient. Given that the artery had TIMI grade 2 flow at the time of angiography, it is possible that the artery was initially occluded and the accelerated idioventricular rhythm we
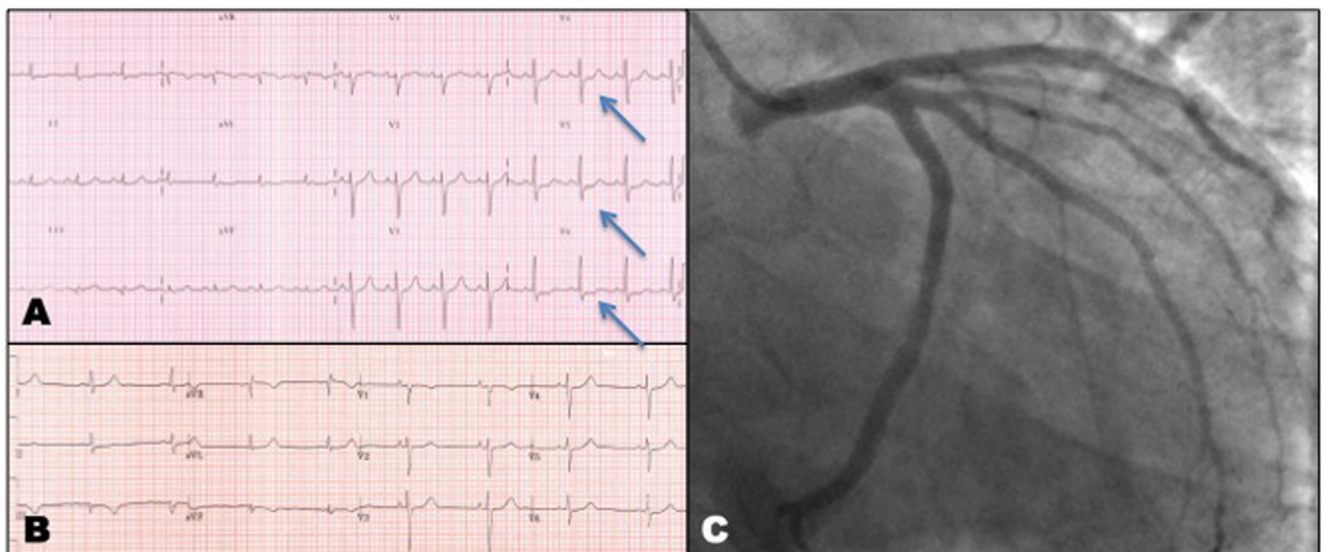

Figure 3   **A:** Electrocardiogram (ECG) 40 minutes after ECG shown in panel B, demonstrating mild ST-segment depression in leads V_4-V_6 (arrows). **B:** Resolution of ECG changes after percutaneous coronary intervention to left circumflex artery. **C:** Coronary angiography in left anterior oblique 30°/caudal view after treatment of circumflex stenosis, demonstrating an unobstructed circumflex artery after successful percutaneous coronary intervention.

observed occurred with reperfusion. The morphology of the idioventricular rhythm would suggest an origin from the inferoposterior wall in a territory supplied by the circumflex artery. When the rhythm reverted to sinus rhythm, the ECG changes were subtle and may not necessarily have precipitated emergency coronary angiography. The recognition of such rhythms as possible indicators of significant coronary artery damage with resultant ischemia after a left atrial ablation procedure is important and may increase awareness of this rare but important complication of RF ablation procedures.

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