Atrial tachycardia related to atrial infarction treated with catheter ablation: a case report

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Background	Atrial infarction, usually concurrent with ventricular infarction, is under-recognized. Although most patients with atrial infarction have complicated supraventricular tachyarrhythmias, its mechanism is still unknown. We report a case of atrial tachycardia (AT) related to atrial infarction treated with catheter ablation.
Case summary	A 51-year-old man was referred for acute chest pain. Electrocardiography showed a junctional rhythm with ST depression in the precordial leads. Emergency coronary angiography revealed an occluded dominant left circumflex coronary artery (LCX). A drug-eluting stent was deployed; however, the atrial branch from the distal side of the LCX was jailed by the stent and became occluded. On the 7th day, the premature atrial contractions (PACs) became frequent and changed to AT. Owing to its resistance to medication, we performed catheter ablation. The electro-anatomical map revealed counter-clockwise macro-reentrant tachycardia at the tricuspid valve annulus, with low-voltage and fragmented potential (FP) areas at the posterior wall of the right atrium (RA). After terminating the AT through linear ablation for the cavotricuspid isthmus, multiple-focus PACs originating from the FP area in the RA posterior wall were documented. Coronary angiography revealed that these damaged areas were perfused by the atrial branch of the LCX. Defragmentation in the FP area could eliminate PACs. The patient was discharged with sinus rhythm and without any complications.
Discussion	We can perform electro-anatomical mapping to identify tachycardia circuit and PACs arising from the FP area in the posterior RA, where the atrial branch was perfusing. Multiple PACs from infarcted myocardium result in tachycardia.
Keywords	Acute myocardial infarction • Atrial infarction • Atrial tachycardia • Catheter ablation • Case report
ESC Curriculum	5.5 Supraventricular tachycardia • 5.4 Atrial flutter • 3.1 Coronary artery disease • 3.2 Acute coronary syndrome

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Learning points

- Atrial infarction is poorly recognized and understudied. More than half of the patients with atrial infarction are reported to have complicated supraventricular tachyarrhythmias. We should highlight the significance of atrial infarction.
- This is the first description of electro-anatomical mapping for the identification of supraventricular tachycardia associated with atrial infarction. Infarcted areas served as a zone of electrical slow conduction for tachycardia and an origin of frequent premature beats. We successfully treated the tachycardia by a linear ablation and the elimination of premature beats.

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Introduction

Atrial infarction, which is usually concurrent with ventricular infarction, is poorly recognized and understudied. Cushing *et al.*[1] reported that atrial infarction occurred in 17% of autopsy-proven myocardial infarction cases. Atrial infarction remains a clinical challenge with unknown incidence and consequences, mainly due to the lack of reliable diagnostic markers. It should be suspected in any patient who presents with typical chest pain, elevated cardiac biomarkers, and electrocardiogram changes consistent with atrial infarction, including the presence of abnormal *P*-wave morphologies, PR segment deviations, or supraventricular tachyarrhythmia.[2] More than half of the patients with atrial infarction are reported to have complicated supraventricular tachyarrhythmias.[3] Clinically, atrial infarction may be of clinical significance, independent of ventricular infarction.

Timeline

Date	Significant event
17 June 2020	Patient presented to the emergency room with chest pain. We performed emergency percutaneous coronary intervention.
24 June 2020	Atrial premature beats became frequent, and turned into atrial tachycardia (AT).
1 July 2020	Cardioversion
5 July 2020	Discharge
17 August 2020	AT recurred with palpitation. Anti-arrhythmia drugs were started.
15 February 2021	Catheter ablation

We report the case of a male patient with atrial infarction complicating atrial tachycardia (AT) that was treated with catheter ablation.

Case presentation

A 51-year-old man was referred to our hospital with persistent chest pain for the last 2 h. He was a chronic smoker and had dyslipidaemia and a family history of acute myocardial infarction. He had never experienced any chest pain or palpitations. Electrocardiography (ECG) showed a junctional rhythm at 30 bpm with ST depression on the precordial leads (*Figure 1A*). Bedside echocardiography revealed severe hypokinesis at the posterior left ventricular wall was also noted, and the left ventricular ejection fraction was 51%. In addition, the serum level of troponin was elevated to 1.44 ng/mL, confirming the diagnosis of acute myocardial infarction.

Emergency coronary angiography (CAG) revealed an occluded dominant left circumflex coronary artery (LCX) with atherosclerosis (*Figure 1B*). A drug-eluting stent was deployed at the occluded site, resulting in sufficient blood flow to the distal side of the lesions, as evidenced by the Thrombolysis in Myocardial Infarction score. The atrial branch from the distal side of the LCX was jailed by the stent and occluded (*Figure 1C*). During the remedy, a temporary pacemaker was inserted because of junctional bradycardia. After the remedy, his chest pain had resolved, and ECG still showed a junctional rhythm, so we finished the intervention. On the next day, bedside echocardiography

revealed that a hypokinesis at the posterior left ventricular wall was improved. The serum creatinine kinase was elevated to 6831 mg/dL at its peak.

The day after the coronary intervention, sinus rhythm recovered to 70 bpm, and premature atrial contraction (PAC) was documented frequently. AT occurred on the 7th day after the admission (*Figure 2*). Anticoagulant drug was started. After exclusion of atrial thrombus by transesophageal echocardiography, the patient underwent cardioversion and returned to sinus rhythm. He discharged on 14th day.

After discharge, the AT recurred with palpitations and exertional dyspnoea after one month. The anti-arrhythmia drugs including bisoprolol, verapamil and bepridil were ineffective, and his palpitations worsened.

We decided to perform a catheter ablation 6 months after discharge. All anti-arrhythmia drugs were stopped before the catheter ablation. An electro-anatomical map revealed typical atrial flutter. Low-voltage areas and many fragmented potential (FP) areas were observed in the posterior wall of the right atrium (RA) (*Figure 3*). During linear ablation between the inferior vena cava and tricuspid valve, the AT terminated and returned to sinus rhythm. Subsequently, multiple-focus PACs originating from the FP area in the RA posterior wall were documented (*Figure 4*). We performed ablation in the area, and the PACs almost disappeared. CAG revealed that the atrial branch jailed by the stent was naturally reperfused, and the artery was perfused to the FP area (*Figure 5* and see Supplementary material online, *Videos S1 and S2*). We completed this session with non-inducibility of any AT by rapid stimulation. The patient was discharged with sinus rhythm and without any complications. He had an uneventful course for 6 months.

Discussion

Several studies have reported that atrial infarction can cause supraventricular tachycardia.[2,3] To our knowledge, this is the first description of supraventricular tachycardia complicated by atrial infarction treated via catheter ablation with an excellent outcome. Electro-anatomical mapping was used to identify the tachycardia circuit and PACs arising from the FP area in the posterior RA, which was perfused by the atrial branch. This demonstrated that infarcted atrial myocardium resulted in multiple PACs. These areas might serve as a zone of electrical slow conduction for perpetuation of atrial flutter. Moreover, the temporal connection of the myocardial infarction and the atrial arrhythmias was another hint for the causal relationship.

The mechanisms underlying spontaneous supraventricular tachyarrhythmias associated with atrial infarction have not yet been completely elucidated. Avula *et al.* hypothesized that nitroso-redox imbalance in the ischaemic zone is associated with reactive oxygen species production and altered ryanodine receptor 2 responses to calmodulin in a sheep left atrial myocardial infarction study. Ischaemia-related atrial arrhythmias can occur.[4] In an autopsy report of a case of cardiac rupture caused by atrial infarction, a histopathological study revealed severe atrial scarring and fibrosis due to the replacement of extensive areas of cardiomyocyte loss. The coexistence of scarring and normal cardiomyocytes, such as mosaic tissue, is thought to be a cause of arrhythmias.[5,6] In our case, electro-anatomical mapping demonstrated FP in the infarcted area, which may be consistent with the mosaic tissue.

Most atrial infarctions are caused by arteriosclerotic heart disease. The largest atrial artery supplies the sinoatrial node. It arises from the left coronary artery in 39% and the right coronary artery in 61% of hearts. Its general course is from the anterior interatrial septum and then to the encircling termination at the base of the superior vena cava.[7] Some studies have noted the relatively high incidence of RA as compared with left or bi-atrial infarction, with the percentage of RA infarctions ranging from 81 to 98%.[3] The considerably higher
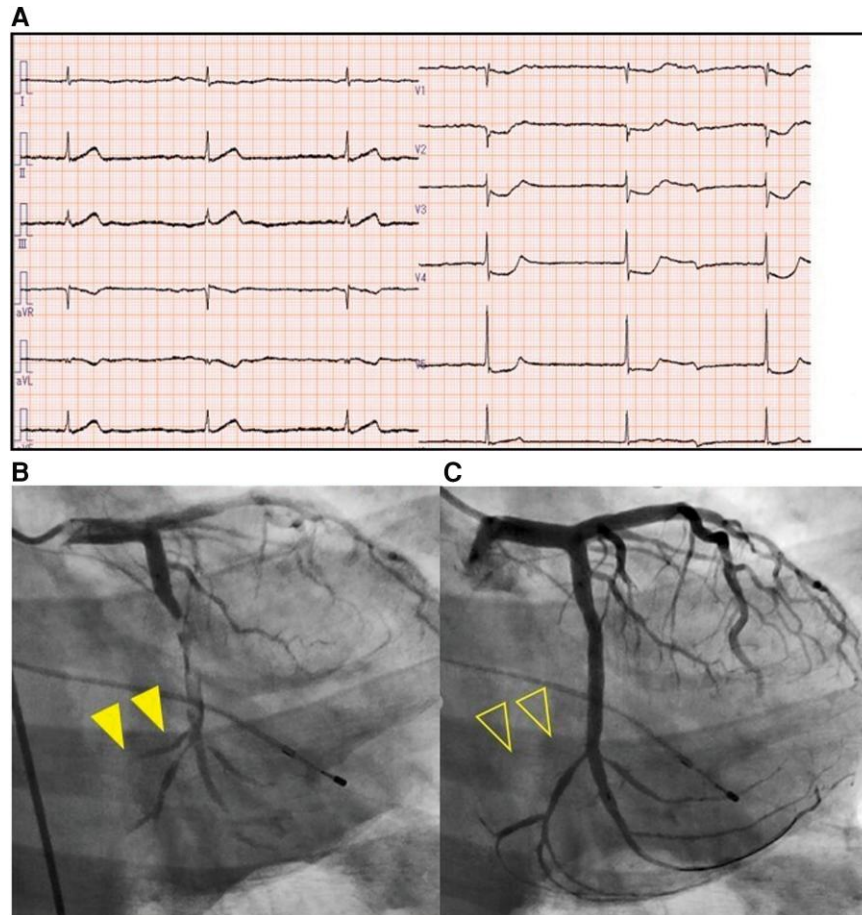
Figure 1 Electrocardiogram shows a junctional rhythm at 30 bpm with ST depression on the precordial leads (*A*). Coronary angiography revealed severe stenosis with a flow delay at the middle site of the left circumflex artery (right anterior oblique). A temporary pacemaker was inserted because of junctional bradycardia. The arrow shows the atrial branch (*B*). After reperfusion, the atrial branch was jailed by the stent and occluded (*C*; blank arrow).
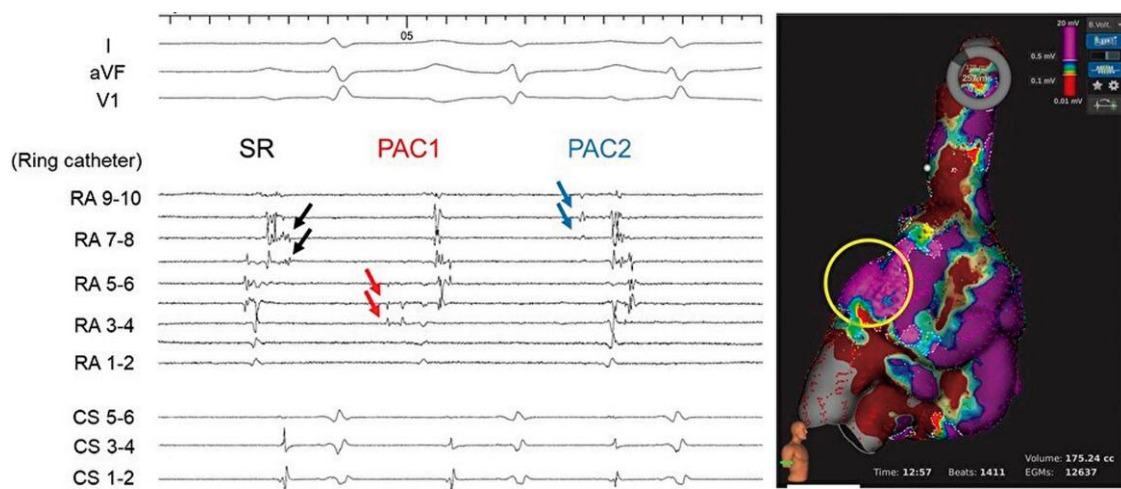

Figure 2 Atrial tachycardia frequently occurred after the coronary intervention. Cycle length of atrial tachycardia was 260 ms.
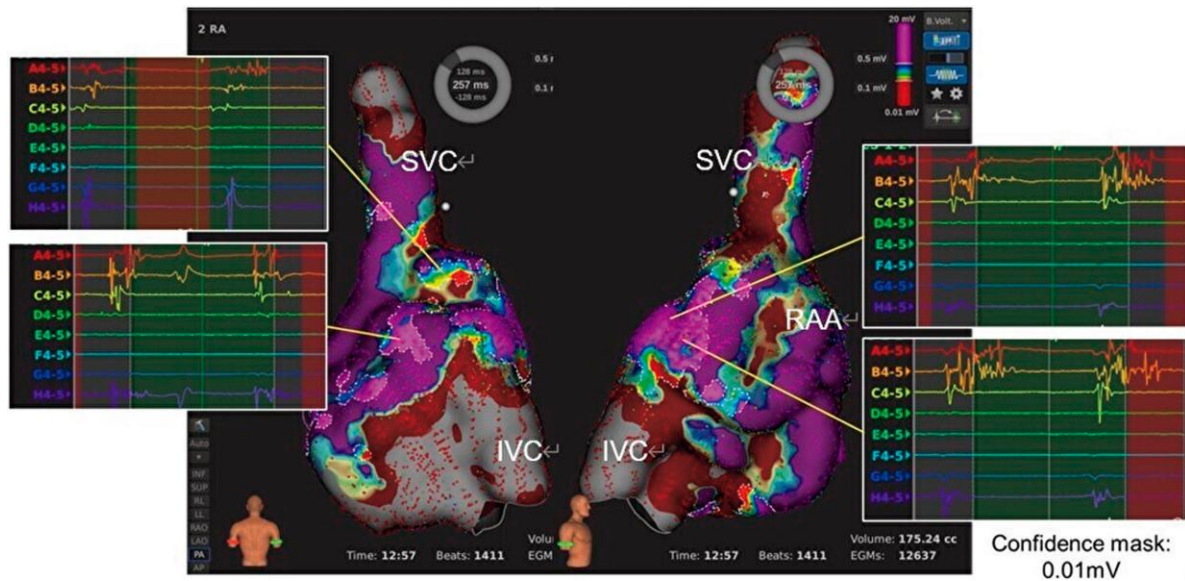
Figure 3 Electro-anatomical mapping revealed that there were low-voltage areas and many fragmented potentials at the posterior wall of the right atrium. The highlighted area shows the fragmented potentials.


Figure 4 The intracardiac electrocardiogram shows multiple PACs. The ring catheter is located at the fragmented potential area in the posterior wall of the right atrium (circle at the right panel). Fragmented late potentials are recorded by the ring catheter. Each PAC had the preceding potentials with different atrial activation sequences.

oxygen content of left atrial blood may explain the difference in incidence between right and left atrial infarction. Electrocardiogram criteria for localization of the site of infarction also reported that atrial infarction is more frequently found in the atrial appendages, rather than the lateral or posterior walls of the atria. There is no report about accurate anatomical location of infarcted area.

There are no specific recommendations for the management of atrial infarction. Blanton Jr *et al.*[8] reported a case of inferior myocardial infarction complicated by atrial fibrillation that was converted to sinus rhythm after balloon dilatation and stenting of the large left atrial branch of the LCX. The role of anti-arrhythmic drugs in atrial infarction remains to be explored. One large randomized controlled study revealed that the use of metoprolol in patients with acute myocardial infarction significantly reduced the incidence of supraventricular tachyarrhythmia.[9] We could not control AT using anti-arrhythmia drugs and cardioversion; therefore, we performed catheter ablation. Catheter ablation for supraventricular arrhythmias caused by atrial infarction can be beneficial as a treatment option.
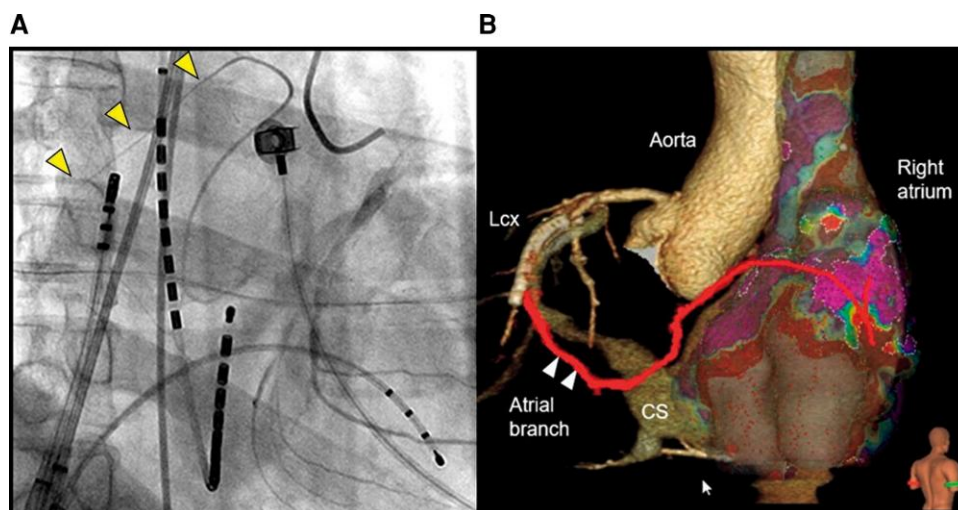
Figure 5 (*A*) Coronary angiography (right anterior oblique) revealed that jailed atrial branch was naturally reperfused. The branch perfused the abnormal electrocardiogram area at the posterior right atrium (arrows). The ablation catheter is located in the FP area. (*B*) Three-dimensional reconstructed computed tomography (3D CT) image shows the atrial branch from the left circumflex artery perfusing the fragmented potential area. Electro-anatomical map is manually overlayed on the 3D CT image. The atrial branch was manually drawn.

Lead author biography

I was graduated from Kobe University of medicine and now working as a resident for Hyogo Prefectural Awaji Medical Centre and studying diagnosis and treatment in cardiology. I am widely interested in clinical medicine of cardiology. The subject of this report impressed me, showing epoch-making figure about atrial infarction and electro-anatomical map. It is very interesting that there is closed relation between ischaemic heart disease and arrythmia. I will be happy this case report may help your practice.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that a consent for submission and publication of this report has been obtained from the patient in line with COPE guidance.

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