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CASE REPORT
Electrophysiology

Multivessel coronary spasm triggered by ganglionated plexi stimulation during atrial fibrillation radiofrequency catheter ablation: a case report

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Background Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in adults, and it is associated with a high burden of mortality and morbidity worldwide. AF can be managed with rate-control or rhythm-control strategies. The latter is increasingly used to improve symptoms and prognosis in selected patients, especially after the development of catheter ablation. Although this technique is generally considered safe, it is not free from rare but life-threatening procedure-related adverse events. Among these, coronary artery spasm (CAS) is an uncommon but potentially fatal complication that requires immediate diagnosis and treatment.

Case summary We report a case of severe multivessel CAS triggered by ganglionated plexi stimulation during pulmonary vein isolation with radiofrequency catheter ablation in a patient with persistent AF, promptly resolved after intracoronary nitrate administration.

Discussion Although rare, CAS is a serious complication of AF catheter ablation. Immediate invasive coronary angiography is key for both diagnosis confirmation and treatment of such dangerous condition. As the number of invasive procedures increases, it is important that both interventional and general cardiologists are aware of possible procedure-related adverse events.

Keywords Atrial fibrillation • Catheter ablation • Ganglionated plexi • Coronary artery spasm • Case report

ESC Curriculum 5.3 Atrial fibrillation • 3.4 Coronary angiography • 5.3 Atrial fibrillation • 3.4 Coronary angiography

Learning points

- Coronary artery spasm (CAS) is a rare but potentially fatal complication of atrial fibrillation (AF) catheter ablation;
- Prompt recognition and immediate treatment of ablation-related coronary spasm are essential in the management of patients experiencing this life-threatening condition;
- Intracoronary nitrate administration is the treatment of choice for CAS in the setting of AF catheter ablation.

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Introduction

Atrial fibrillation (AF) is the most frequent sustained cardiac arrhythmia in adults. First described more than 20 years ago,[1] catheter ablation currently represents an established strategy for the rhythm-control therapy of AF for both symptoms relief and improvement in prognosis in selected patients.[2,3] Due to technical improvements and increased safety with a low rate of major complications, catheter ablation is generally considered a safe procedure. However, some uncommon but potentially fatal procedure-related complications may occur. Among these, coronary artery spasm (CAS) is a rare life-threatening event which requires prompt recognition and management. Here, we report a case of severe multivessel CAS occurred after ganglionated plexi (GP) stimulation in the setting of pulmonary vein isolation (PVI) during radiofrequency (RF) catheter ablation of persistent AF.

Timeline

Date	Event
6 months prior to admission	First diagnosis of persistent/paroxysmal atrial fibrillation (AF) and heart failure with preserved ejection fraction.
Day 1 admission	AF radiofrequency transcatheter ablation (endocardial) under general anaesthesia. Ganglionated Plexi ablation with subsequent diffuse and prominent ST-segment elevation with transient complete atrioventricular block. Transient reduction in left ventricular ejection fraction Invasive coronary angiography showing diffuse coronary artery vasospasm. Spasm resolution after intracoronary nitrate administration.
Day 4 admission	Hospital discharge.
30 days after discharge	No symptoms. Normal Left Ventricular Systolic Function. Sinus Rhythm.

Case presentation

A 58-year-old Caucasian non-smoker male with a history of symptomatic paroxysmal and persistent AF from 6 months, arterial hypertension, and heart failure with preserved ejection fraction (HFpEF, NYHA Class 2), without previous ablation procedures, was admitted to our hospital for AF catheter ablation. At the time of admission, his vital signs were in the range of normality and he was in sinus rhythm after spontaneous conversion of AF. He was receiving a rate-control treatment with a beta-blocker (bisoprolol 2.5 mg qd) and digoxin (0.125 mg qd), both in therapeutic range, a direct oral anticoagulant for stroke prevention (CHA_2DS_2-Vasc = 2), and an antihypertensive combination therapy with an angiotensin converting enzyme inhibitor and diuretic. He was also unable to tolerate antiarrhythmic drugs in the past. His physical examination was unremarkable. A chest X-Ray obtained before the procedure was completely normal. Routine blood tests were normal at the time of admission except for mildly increased N-terminal prohormone B-type natriuretic peptide levels (630 pg/mL; reference range <125 pg/mL). Transthoracic echocardiography (TTE) showed normal left ventricular and right ventricular systolic function and the absence of significant valvular heart disease with an only mildly enlarged left atrium (LA) and left ventricular concentric remodelling. A transoesophageal echocardiography was performed 24 h before the procedure in order to rule out the presence of LA thrombosis. Catheter ablation was performed under general anaesthesia with orotracheal intubation. Induction of anaesthesia was obtained with propofol 2 mg/Kg, fentanyl 100 mcg, and rocuronium 1 mg/Kg, and it was completely regular. General anaesthesia was maintained with sevoflurane 1.2–1.4% in a 1:1 oxygen:air combination. Analgesia was maintained with remifentanil infusion 0.03–0.08 mcg/Kg/min. A five Fr pig-tail catheter was placed at the aortic valvular plane through the right common femoral artery and a decapolar steerable diagnostic catheter was placed in the coronary sinus through the right common femoral vein. Then a fluoroscopy-guided transseptal puncture was performed without complications (SL0™ Swartz sheath, Abbott; BRK™ transseptal needle, Abbott). An electroanatomical three-dimensional map (EnSite NavX, Abbott) of the LA was obtained using a high-density mapping catheter (Advisor™ HD Grid Mapping Catheter, Sensor Enabled™ (SE), Abbott). PVI with RF energy (standard energy, 30–35 W) was started with the use of a 4 mm tip bidirectional irrigated catheter (FlexAbility Ablation Catheter, Abbott). The activating clotting time was maintained between 250 and 350 s through unfractionated heparin iv infusion at standard dose. At the time of RF initiation, the patient vital signs were completely normal. The procedure was initiated targeting the left superior pulmonary vein (LSPV). After a few seconds, during the encircling of the LSPV and RF delivery at the site of posterior left pulmonary venous carina, the patient developed a reproducible asystolic reflex associated with hypotension, suggesting GP irritation with subsequent vagal activation (*Figure 1*). RF delivery was stopped for a few seconds and then resumed after recovery of electrical activity. About 2 min after the first RF delivery, a prominent and diffuse ST-segment elevation was noted (*Figure 2*) and the patient developed a transient complete atrioventricular block with a junctional escape rhythm requiring temporary right ventricular pacing and i.v. administration of atropine 0.5 mg. Bedside TTE was performed in order to rule out pericardial involvement or mechanical complications and showed a mildly reduced left ventricular ejection fraction (LVEF) without clear regional wall motion abnormalities in the absence of pericardial effusion. An invasive coronary angiography (ICA) was immediately performed. Severe multivessel CAS in the absence of significant atherosclerotic disease or air embolism was detected (Supplementary material online). CAS resolution and subsequent flow normalization were obtained after intracoronary administration of nitroglycerin (*Figure 3*). The ST-segment promptly returned to isoelectric line (*Figure 4*). Repeated bedside TTE performed after spasm regression showed recovered LVEF and confirmed the absence of pericardial effusion. After complete resolution and stabilization of the patient clinical parameters, the procedure was resumed and PVI with encircling and adjunctive LA lesion lines (mitral isthmus and roof line) were completed without further complications (*Figure 4*). After the procedure, myocardial injury markers (high-sensitivity cardiac troponin I) resulted slightly elevated with a 'rise and fall' pattern (peak 1060 ng/L, normal range <14 ng/L). After 48 h of clinical monitoring, the patient was discharged with a non-dihydropyridine calcium channel blocker (verapamil, 40 mg tid) therapy. At 30 days, 3 and 6 months follow-up the patient was in sinus rhythm without left ventricular wall motion abnormalities.

Discussion

Catheter ablation is increasingly used for the treatment of AF.[4] Despite technical improvements and increased safety, some potential life-threatening complications may occur. CAS during RF ablation of AF

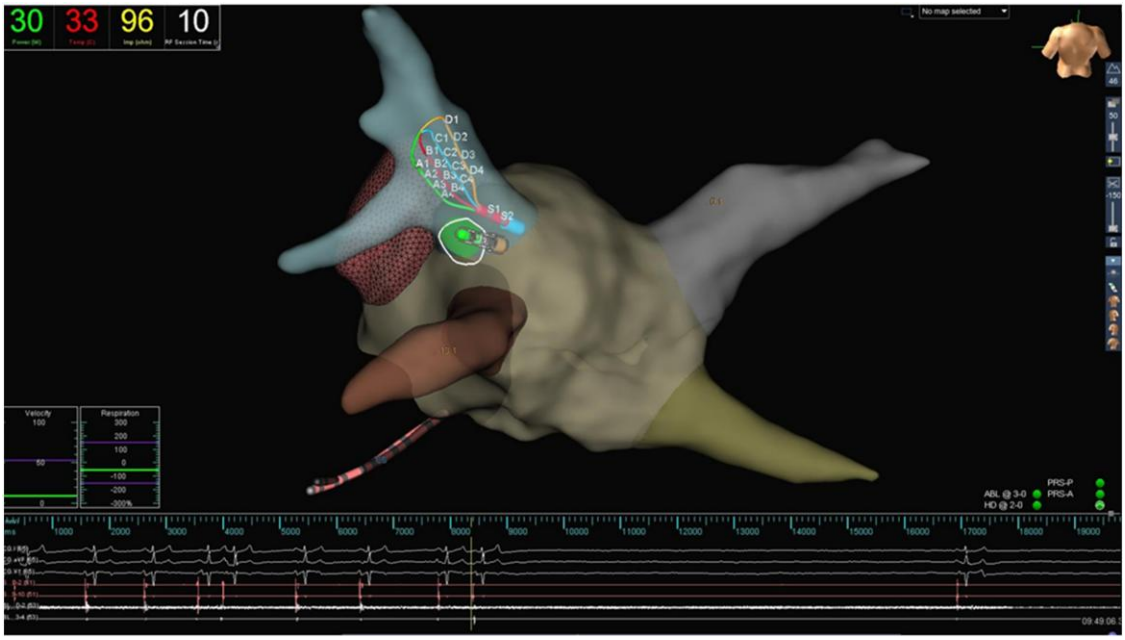

Figure 1 Electroanatomical map (EnSite NavX, Abbott) of the LA in PA view and ECG showing sinus rhythm with a marked asystolic reflex at the time of radiofrequency ablation (30 W) in the posterior aspect of the left superior pulmonary vein (LSPV). This response was associated with concomitant transient hypotension. ECG, electrocardiogram; LA, left atrium; PA, posteroanterior.

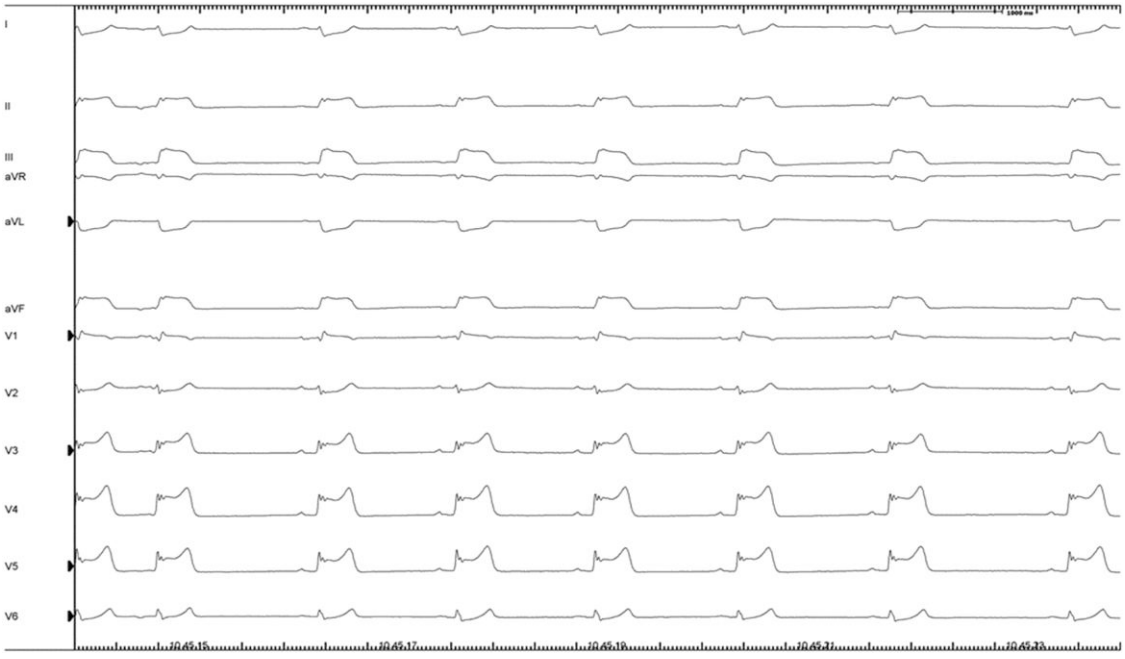

Figure 2 ECG showing sinus bradycardia and prominent and diffuse ST-segment elevation and ST-segment depression limited to leads I and aVL after GP ablation. The patient also developed transient complete atrioventricular block with an escape junctional rhythm requiring temporary right ventricular pacing (not shown). GP, ganglionated plexi.

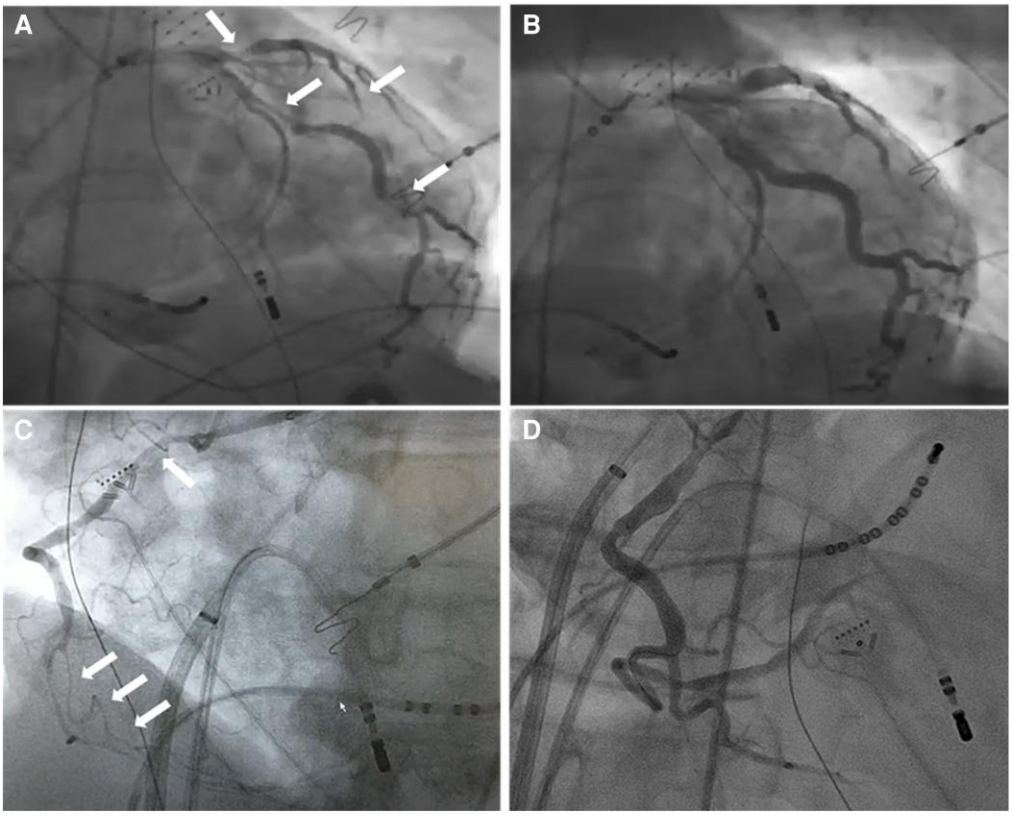

Figure 3 ICA showing severe multivessel coronary spasm (arrows) in both LCA (*A*) and RCA (*C*). Coronary spasm disappeared after i.c. nitrate administration (*B* and *D*). ICA, invasive coronary angiography; LCA, left coronary artery; RCA, right coronary artery; i.c., intra-coronary.

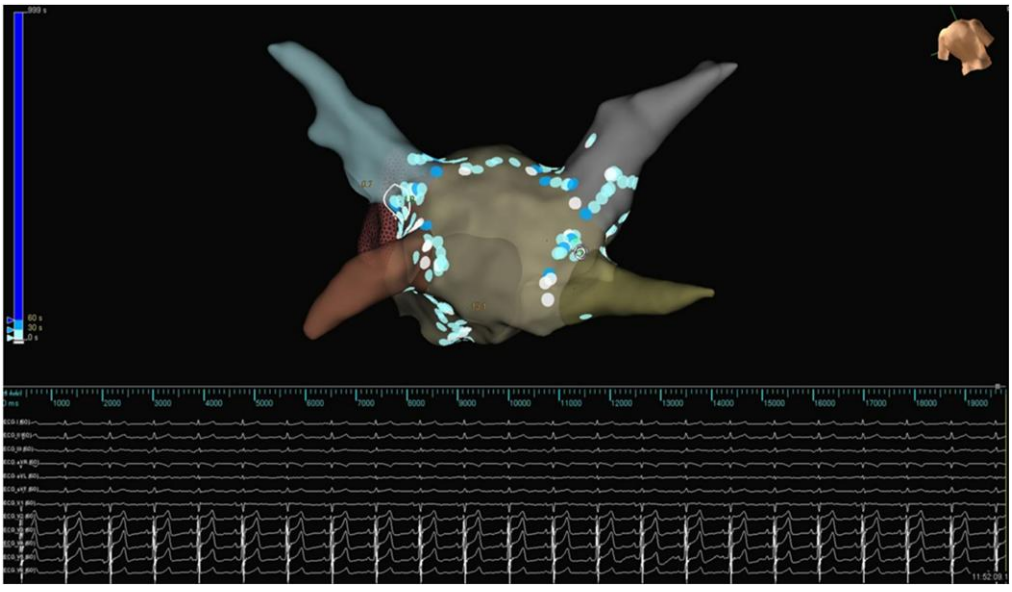

Figure 4 PVI with encircling and adjunctive left atrial lesion lines (mitral isthmus and roof line). Complete regression of ST-segment elevation at 12-lead ECG.

has been previously described in other reports[5] with an estimated incidence of about 0.3% in a large Japanese cohort of patients.[6] CAS was also reported with the use of cryoballoon[7] and after vein of Marshall ethanol infusion[8] for AF ablation. Proposed mechanisms of CAS related to the ablation procedure include direct thermal effects on the coronary artery, indirect effects of cryoenergy-induced blood cooling, and autonomic nervous system imbalance. Our case highlights the possibility of severe multivessel CAS after GP stimulation in the setting of PVI during RF catheter ablation of AF. GP are autonomic structures located in different points of the epicardial surface of the heart and strictly associated to the pulmonary veins.[9,10] GP stimulation is a well-recognized event during both endocardial and epicardial AF catheter ablation. Furthermore, GP are involved in AF initiation and maintenance,[11] and their ablation was associated with better procedural outcomes in some reports.[12–14] However, GP irritation during catheter ablation may be responsible for sympathetic imbalance with consequent coronary vessel motility impairment in predisposed individuals. At the best of our knowledge, there are no recognized risk factors, with the possible exception of smoking habit, or preventing measures for AF ablation-related CAS. Therefore, immediate diagnosis and treatment are crucial to avoid poor outcomes in patients experiencing such complication.

In our patient, a clear temporal relation between GP ablation, with the development of bradycardia and hypotension, and the appearance of CAS was noted. For such reason, we excluded a possible relationship with anaesthetics or other administered drugs. We hypothesized that the transient sympathetic imbalance produced by GP modulation determined the diffuse CAS in our patient. ICA ruled out other possible causes of diffuse ST-segment elevation such as thrombotic or air embolism, spontaneous coronary artery dissection, and pre-existing coronary artery disease. Multi-parameter monitoring along with immediate ICA was key for the optimal management. Early diagnosis and treatment with intracoronary nitroglycerin administration with complete recovery allowed the completion of the procedure without further adverse consequences for the patient.

Conclusions

The present case highlights the importance of early recognition of ablation-related CAS and the on-site availability of ICA in the setting of AF catheter ablation. Intracoronary nitrate administration is a safe and effective treatment of such complication.

Lead author biography

Carlo De Innocentiis graduated from "G. D'Annunzio" University, Chieti-Pescara, Italy, in 2014. He completed his Cardiology Fellowship in the same University, and he is currently working as Cardiac Electrophysiologist at the Gemelli Molise Hospital, Italy. He has interest in cardiac electrophysiology, interventional cardiology, and cardiovascular imaging. Carlo De Innocentiis graduated from "G. D'Annunzio" University, Chieti-Pescara, Italy, in 2014. He completed his Cardiology Fellowship in the same University and he is currently working as Cardiac Electrophysiologist at the Gemelli Molise Hospital, Italy. He has interest in cardiac electrophysiology, interventional cardiology, and cardiovascular imaging.

Supplementary material

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

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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 written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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