JAFIB
Case Report
Journal of Atrial Fibrillation

www. jafib.com

Transient Inferior Lead ST Elevation During Radiofrequency Ablation of Atrial Fibrillation

Michael P. Soos [1], Mohan C. Madala [1], Khalil Kanjwal [1]

[1]Cardiology Section of McLaren Greater Lansing.

Abstract

Radiofrequency ablation (RFA) is a commonly performed procedure for symptomatic atrial fibrillation (AF). Herein, we describe a case of transient ST elevation during the isolation of right-sided pulmonary veins. The patient was hemodynamically stable and due to the transient nature of ST-elevation, the procedure was completed successfully. Subsequently, the cardiac catheterization was performed which did not reveal any significant obstructive coronary lesion or a thrombus. In this report, we attempt to explain possible mechanisms for ST-elevation during RFA of AF.

Introduction

Radiofrequency ablation (RFA) has emerged as the most effective rhythm control strategy for atrial fibrillation (AF) [(1)]. These procedures are routinely performed in most electrophysiology laboratories. Over years these procedures have become safe with emerging new technologies. The risk of complication during atrial fibrillation ablation is less than one percent [(1)]. We describe a very rare phenomenon of transient ST elevation in inferior limb leads during pulmonary vein isolation of right sides veins.

Case Study

A 70-year-old Caucasian female, with a history of hypertension, prior stroke, chronic kidney disease and hypothyroidism who presented to our arrhythmia clinic with symptomatic atrial fibrillation and flutter refractory to medical therapy including Cardizem, Digoxin, and Flecainide. She underwent multiple synchronized cardioversions in the past. Her CHADS-VASC score was 4. Her symptoms included shortness of breath, fatigue, and dizziness. Due to persistent symptomatic atrial fibrillation refractory to medical therapy, she was offered RFA. Risks and benefits were explained, and the patient decided to proceed with RFA for AF.

The patient was brought to the electrophysiology in the fasting state. Venous access was obtained using four vascular sheaths (SL-0 x2, SR-0 X 1) and short 11 Fr ultrasound guidance.

A Carto sound mapping catheter was advanced, and a three-dimensional electro-anatomical map of the right atrium, Cavo-tricuspid isthmus, left atrium and pulmonary veins was created. Pulmonary vein anatomy was normal. The electrophysiology catheters were advanced using electroanatomic mapping guidance to the His bundle and coronary sinus. Heparin boluses were administered throughout the procedure and the ACT was maintained between 350 - 400 sec. Isolation of the veins was performed through the creation of circumferential linear ablation lesions using an irrigated ablation catheter. The Pentarray catheter was used to guide and confirm the complete isolation of all four pulmonary veins through the identification of early activation within the ostia. The isolation of each pulmonary vein was confirmed by the presence of an entrance block to each vein. During the RFA of the right superior vein anterosuperiorly, the patient developed ST elevations in the inferior leads (Figure 1-3). Ablation was stopped and a hemodynamic assessment was performed. Her blood pressure and heart rate were stable. ST segments started coming down to baseline within 3 minutes and ablation was resumed with close attention to the surface EKG throughout the procedure (Figure 3). ACT at the time of ST elevation was noted to be 360 secs. Cardioversion was performed and all veins were checked for isolation (Figure 4 and 5). Cavo tricuspid isthmus ablation for atrial flutter was performed with the demonstration of a bidirectional block. There were no further ST elevations noted and after isolation of all veins and ablation of Cavo tricuspid isthmus a coronary angiography was performed.

Coronary angiography was performed and revealed a normal left main, left anterior descending, left circumflex and right coronary

Key Words

Atrial fibrillation, Radiofrequency ablation , Cardizem

Corresponding Author
Khalil Kanjwal, MD, FHRS, FACC, CCDS, CEPS(P).
Clinical Associate Professor of Medicine Michigan State University
Director of Electrophysiology and Electrophysiology Laboratory
McLaren Greater Lansing Hospital, Lansing, MI

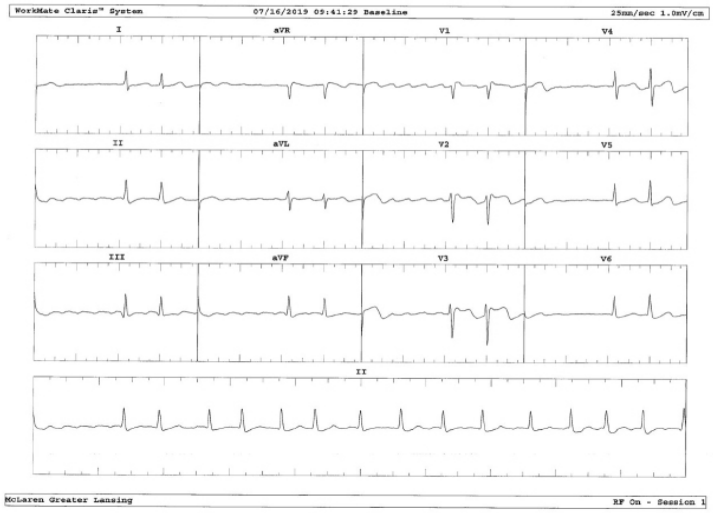

Figure 1: Baseline EKG showing atrial fibrillation without any ST deviations.

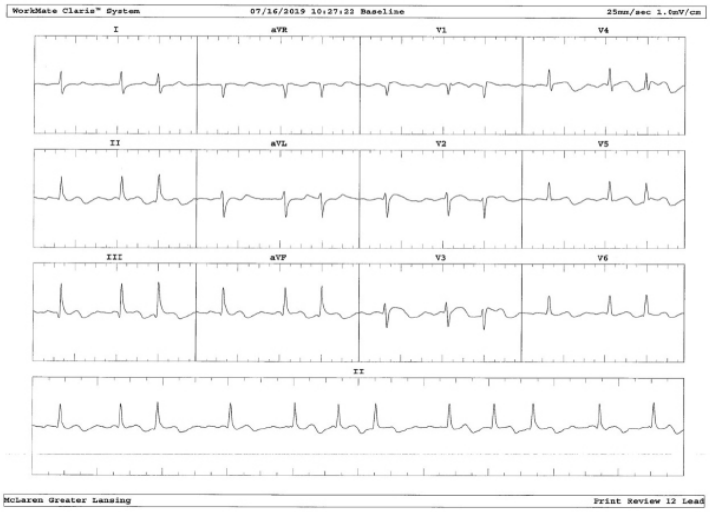

Figure 2: EKG showing ST elevation especially in inferior leads during ablation.

arteries (Figure 6 and 7) . The first obtuse marginal was noted to have a mid 50% focal lesion that was not determined to be hemodynamically significant. Anticoagulation was continued with Rivaroxaban in the post-operative period. The patient was monitored in the hospital for 24 hours without developing additional ischemic changes on telemetry or electrocardiogram. The patient was seen subsequently in follow-up in our arrhythmia clinic and has been doing well.

Discussion

AF is a common arrhythmia affecting up to 2% of the general population (1). AF commonly affects elderly patients and is associated with a lifetime risk of 26% in males and 23% in females by age 80 (1,2). RFA has emerged as the most effective rhythm control strategy for the management of AF.

Over years RFA as a rhythm-control strategy for AF has evolved as a very safe procedure with a low risk of complication rates. The potential complications of RFA include femoral vascular injury, myocardial perforation, stroke, atrio-esophageal fistula, and death. Myocardial ischemia or infarction has rarely been reported as a complication of RFA of AF ablation (1,2).

Due to the proximity of the right coronary artery to the Cavo tricuspid isthmus injury to the right coronary artery has been reported previously during ablation of Cavo tricuspid isthmus (3-7). Acute ST-elevation has been reported during transeptal puncture (8) and during slow pathway ablation for Atrioventricular reentrant tachycardia as well (9). Recently, ST-elevation has been reported during vagally mediated atrial fibrillation and a mechanism similar to the Bazold-Jarish reflex has been implicated (10).

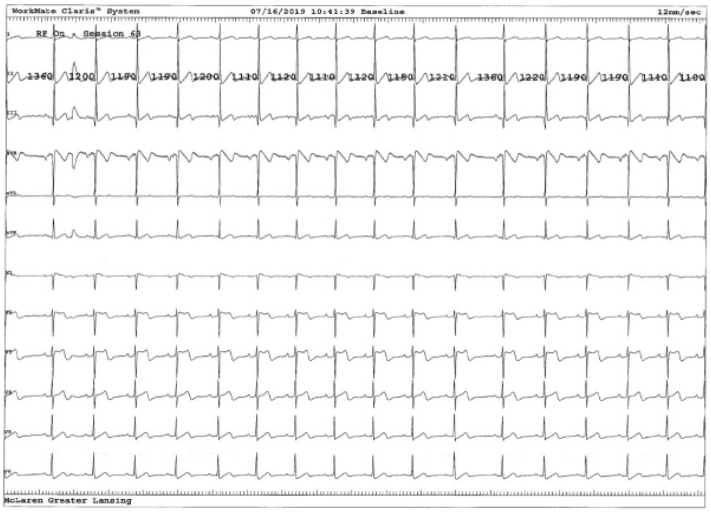

Figure 3: Post Procedure EKG showing resolution of ST deviations.

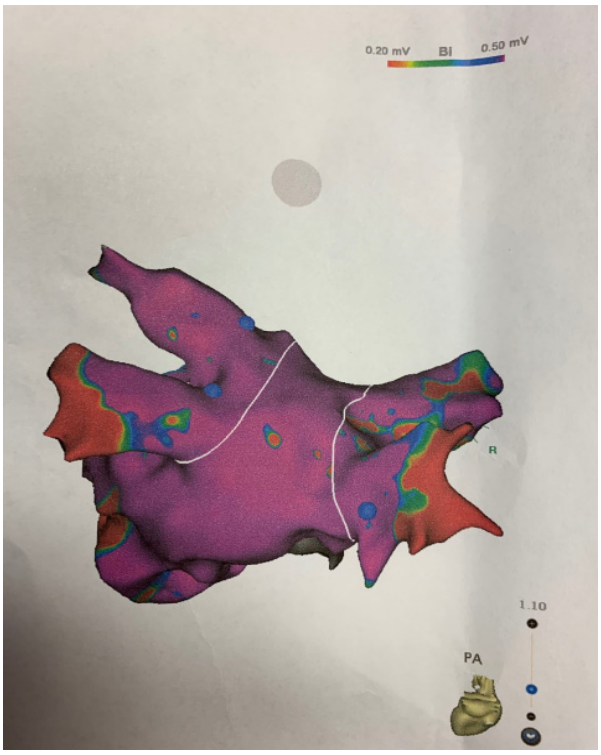

Figure 4: Pre isolation Voltage map of the veins using 3D electroanatomic mapping.

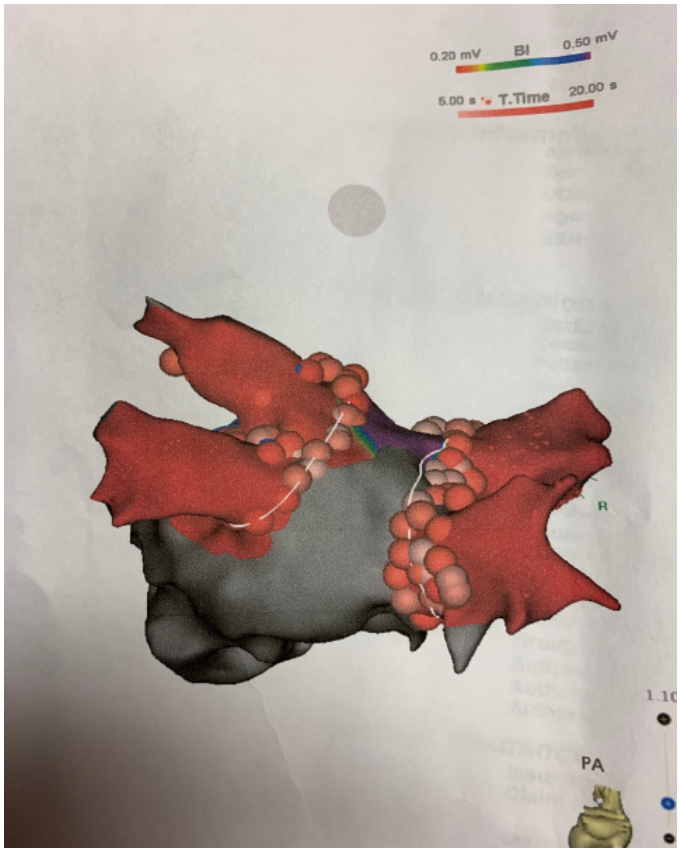

Figure 5: Post pulmonary vein isolation voltage map using 3D electroanatomic mapping showing isolation of pulmonary vein

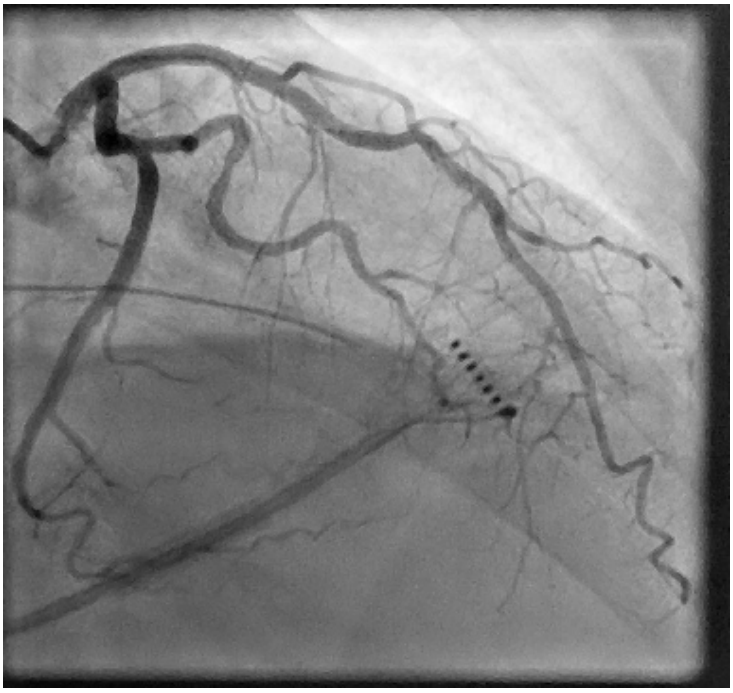

Figure 6: Cardiac Cathetrizations showing no significant left coronary artery and Left circumflex disease.

Although the exact mechanism of ischemic injury and resultant ST elevation during RFA has not been well elucidated the potential mechanisms include direct thermal injury, mechanical injury, thromboembolism, air embolism, and neurohumoral activation.

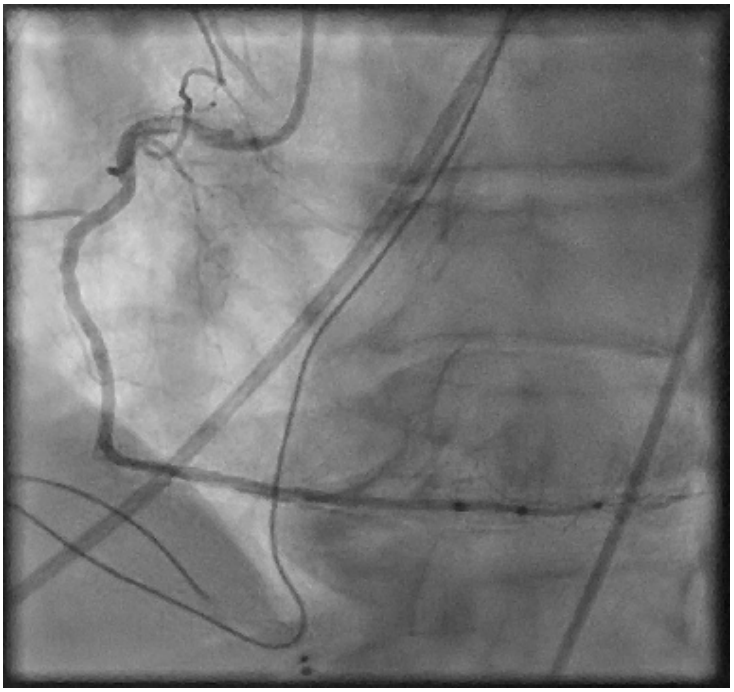

Figure 7: Cardiac Cathetrizations showing no significant right coronary artery.

Direct thermal injury is possible during ablation when a target area is in close anatomic proximity to the coronary vascular system as has been explained during the ablation of Cavo tricuspid isthmus and slow pathway ablation for AVNRT. Mechanical stretch or pressure on the vascular system is a possibility during transeptal puncture. Other potential mechanisms including coronary embolism, air embolism or thermal injury-induced vasospasm. In our patient direct thermal injury and embolism was unlikely because the ACT was maintained between 350 – 400 secs and ablation site was not in close proximity to the arterial system.

It is also possible that the ablation could lead to local neurohumoral activation with the generation of vasospastic neurohormones that can lead to transient vasospasm and ST elevation. In our case, ST-elevation occurred while ablation was performed in the right superior vein in the anterior and superior area of the vein (RSPV). This area is usually rich in ganglionic plexus. Ablating a ganglionic plexus usually results in bradycardia and hypotension and these reflex responses are transient and subside as soon as the ablation is turned off. It is possible that the parasympathetic activation while ablating in the superior area of the RSPV could lead to transient hypoperfusion and ST-elevation through stimulation of ganglionic plexus.

We believe that the mechanism of ST-elevation could have been a reflex phenomenon rather than a direct thermal injury, as there is no major coronary artery close to the anterior superior area of the RSPV. Electrophysiologists have to be careful when encountered with such a situation especially while ablating. We should pay close attention not only to the rhythm but also to the ST segments. In our case the ST elevation was transient, and we were able to complete the procedure without any adverse outcome. However, it is crucial to monitor the

patient closely for any further ST elevation. If recurrent or persistent ST elevation is noted the procedure should be aborted and an urgent cardiac catheterization should be performed. In our patient cardiac catheterization did not show any hemodynamically significant coronary artery disease. Given the normal cardiac catheterization, an air embolism, thrombus or a direct injury to the vessel was unlikely as a mechanism for ST elevation. We believe all patients who have any ST deviation during RFA of AF should be evaluated with cardiac catheterization to rule out any possible vascular injury that would need therapeutic intervention.

Conclusion

Physicians need to be aware that ST elevation can occur during RFA of AF ablation. If transient, ablation can be continued successfully. However, if recurrent or persistent procedure should be aborted and a diagnostic cardiac catheterization should be performed urgently to rule out any possibility of an arterial injury.

References

1. Calkins H, Hindricks G, Cappato R, Kim YH, Saad EB, Aguinaga L, Akar JG, Badhwar V, Brugada J, Camm J, Chen PS, Chen SA, Chung MK, Nielsen JC, Curtis AB, Davies DW, Day JD, d'Avila A, de Groot NMSN, Di Biase L, Duytschaever M, Edgerto JR, Ellenbogen KA, Ellinor PT, Ernst S, Fenelon G, Gerstenfeld EP, Haines DE, Haissaguerre M, Helm RH, Hylek E, Jackman WM, Jalife J, Kalman JM, Kautzner J, Kottkamp H, Kuck KH, Kumagai K, Lee R, Lewalter T, Lindsay BD, Macle L, Mansour M, Marchlinski FE, Michaud GF, Nakagawa H, Natale A, Nattel S, Okumura K, Packer D, Pokushalov E, Reynolds MR, Sanders P, Scanavacca M, Schilling R, Tondo C, Tsao HM, Verma A, Wilber DJ, Yamane T. 2017 HRS/EHRA/ECAS/APHRS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation: Executive summary. J Arrhythm. 2017 Oct;33(5):369-409.
2. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland JC Jr, ContiJB, Ellinor PT, Ezekowitz MD, Field ME, Murray KT, Sacco RL, Stevenson WG, Tchou PJ, Tracy CM, Yancy CW; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. J Am Coll Cardiol. 2014 Dec 2;64(21):e1-76. doi: 10.1016/j.jacc.2014.03.022. Epub 2014 Mar 28. Review. Erratum in: J Am Col Cardiol. 2014 Dec 2;64(21):2305-7.
3. Al Aloul B, Sigurdsson G, Can I, Li JM, Dykoski R, Tholakanahalli VN. Proximity of right coronary artery to cavotricuspid isthmus as determined by computed tomography. Pacing Clin Electrophysiol. 2010;33:1319–1323.
4. Yune S, Lee WJ, Hwang JW, Kim E, Ha JM, Kim JS. Acute myocardial infarction after radiofrequency catheter ablation of typical atrial flutter. J Korean Med Sci. 2014;29(2):292–295. doi:10.3346/jkms.2014.29.2.292
5. Caldwell JC, Fath-Odoubadi F, Garratt CJ. Right coronary artery damage during cavo tricuspid isthmus ablation. Pacing Clin Electrophysiol. 2010;33:e110–e113.
6. Sassone B, Leone O, Martinelli GN, Di Pasquale G. Acute myocardial infarction after radiofrequency catheter ablation of typical atrial flutter: histopathological findings and etiopathogenetic hypothesis. Ital Heart J. 2004;5:403–407
7. Mykytsey A, Kehoe R, Bharati S, Maheshwari P, Halleran S, Krishnan K, Razminia M, Mina A, Trohman RG. Right coronary artery occlusion during RF ablation of typical atrial flutter. J Cardiovasc Electrophysiol. 2010;21:818–821.
8. Cheng YL, Dong JZ, Liu XP, Long DY, Fang DP, Yu RH, Tang RB, Ma CS. Transient ST-segment elevation after transseptal puncture for atrial fibrillation ablation in two cases. Chin Med J (Engl). 2012 Mar;125(5):941-4.
9. Chacko M, Marrouche NF, Bhatt DL. Asymptomatic acute inferior ST-elevation myocardial infarction from thermal injury complicating radiofrequency ablation for atrioventricular re-entrant tachycardia. J Invasive Cardiol. 2004 Sep;16 (9):504-5.
10. Makrides C. Transient ST Elevation in Vagally Mediated Atrial Fibrillation. J Atr Fibrillation. 2012 Jun 15;5(1):487. doi: 10.4022/jafib.487. e Collection 2012 Jun-Jul. PubMed PMID: 28496749;