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CLINICAL REVIEW

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ST-segment elevation during arrhythmia ablations—A review

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Abstract

Coronary injury presenting as ST segment elevation (STE) during ablation procedures for different arrhythmias is a rare and most feared complication. There have been multiple reports on STE during various ablation procedures in the recent past. Herein, we review various mechanisms, presentations, and management of STE observed during various ablations, including atrial fibrillation ablation cavotricuspid isthmus and ablation, supraventricular tachycardia ablations, coronary sinus ablation, and ventricular arrhythmia ablations.

KEYWORDS
ablation, arrhythmias, electrocardiogram, elevation, ST-segment

1 | INTRODUCTION

Radiofrequency ablation (RFA) and cryoablation (CryoA) are common electrophysiology procedures performed for the management of various atrial and ventricular arrhythmias. Ablation has revolutionized the treatment of various arrhythmias with very high success rates in supraventricular tachycardia and atrial flutter.[1] Recently, ablation has emerged as an effective treatment for atrial fibrillation and ventricular tachycardia as well. Usually, these procedures are safe with a very low risk of complications, especially in atrial fibrillation where the risk of complications is close to 1%.[1] The most common complications of ablation include vascular injury, retroperitoneal bleeds, cardiac perforation tamponade, stroke, heart blocks, and myocardial infarction.[2] Injury to the coronary vasculature with myocardial infarction is a rare, but a very serious complication, as it needs early recognition and diagnosis for an effective catheter-based treatment for improved outcomes.

2 | METHODS

A literature review was performed by the lead author for articles related to STE and arrhythmia catheter ablations. We searched PubMed, Google Scholar, Cochrane Library, and Ovid MEDLINE using the keywords: "ST" and "elevation" with "catheter", or "ablation", or "arrhythmia", or "transseptal", or "cavotricuspid isthmus", or "coronary sinus," or "ventricular arrhythmia." We then reviewed publications in English between the years 1990-2020. After the search, 23 manuscripts were chosen based on relevance to the topic and the manuscripts were reviewed and agreed upon amongst all the authors. Exclusion criteria consisted of duplicates, abstracts, non-English articles, and works that were unpublished or unrelated to the topic of interest. Of note, no literature review currently exists that reviews STE during multiple different arrhythmias and catheter ablation approaches.

3 | REVIEW

ST-segment elevation (STE) can occur during various stages of different ablation procedures.

3.1 | STE during transseptal catheterizations and ablations

Transient STE has been reported during transseptal catheterization.[2-5] Transseptal catheterization is performed in order to access the left chamber of the heart during ablation of atrial fibrillation and ventricular tachycardia.[4] In a prospective cohort study of 2965 patients who underwent 3452 transseptal catheterizations and ablations, 13 patients had STE during the procedure, indicating an incidence rate of 0.38% for their study.[4] Of these 13 patients, one had persistent atrial fibrillation while the remaining 12 had paroxysmal atrial fibrillation.[4] The only statistically significant difference between the patients who developed STE compared to the patients who did not, was the size of the left atrium.[4] The diameter of the left atrium of patients who developed STE was smaller than the diameter of patients who remained free of transient STE. Otherwise, there was no statistically significant difference in age, gender, or number of repeat ablations. With regards to the timing of the onset of STE, 10 of the 13 patients developed the ECG change directly after transseptal puncture, where the remaining three patients developed STE after pulmonary vein venography. The STE occurred in the inferior leads of II, III, and aVF and lasted for an average of 4.6 minutes.[4] There was no difference in Troponin-I levels between the STE patients and control patients. The ablation procedure was still completed in all 13 patients.[4]

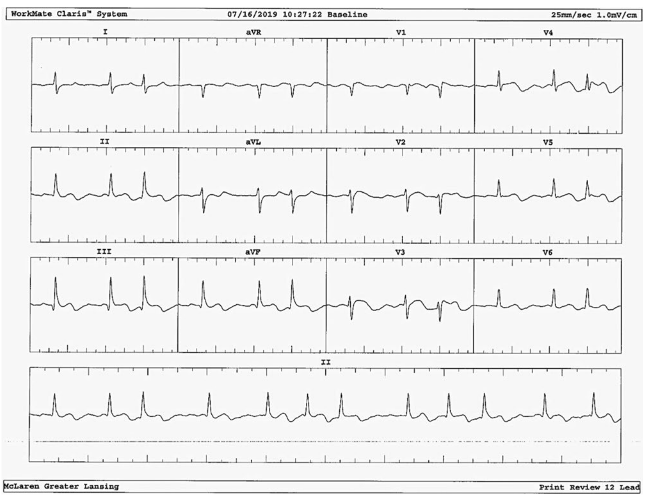

FIGURE 1 EKG showing STE in inferior leads after ablation in the roof of right superior pulmonary vein. Used with permission from Soos M, C Madala M, Kanjwal K. Transient inferior lead ST elevation during radiofrequency ablation of atrial fibrillation. J Atr Fibrillation. 2020;12(6):2293

The pathophysiology of STE during transseptal catheterization remains unclear. The hypothesized pathophysiology has ranged from a possible catheter-induced air embolism affecting the coronary arteries to a Bezold-Jarisch-like-reflex.[4] During transeptal puncture the intraseptal and left atrial ganglionic plexuses may be stimulated by stretch and can lead to autonomic imbalance, coronary vasospasm, and subsequent STE.[5] STE could also be secondary to coronary artery vasospasm caused by direct thermal injury or endothelial dysfunction from existing coronary stents.[6] It has been reported that STE can occur during the left superior ganglionic plexus ablation causing severe coronary vasospasm, relieved with nitroglycerin.[7] When the right superior ganglionic plexus of the left atrium was ablated, multiple coronary spasms occurred leading to STE in lead aVR.[7] In another report, transient STE after ablation in the roof of the right superior pulmonary vein was reported (Figure 1). This area is rich in ganglionic plexii and reflex parasympathetic stimulation resulting in hypotension and bradycardia leading to hypoperfusion could have caused STE as the coronary angiography revealed no evidence of vasospasm, dissection, air embolism, or any stenosis (Figure 2).[8] Transient STE in the inferior leads after protamine administration has been reported as well.[9] The patients received the protamine at the end of the procedure the patient developed hypotension secondary to protamine administration and subsequent STE.[9] An urgent coronary angiography showed the patient had non-obstructive lesion in the right coronary artery. It was postulated that the patient had existing right coronary artery stenosis, and the STE

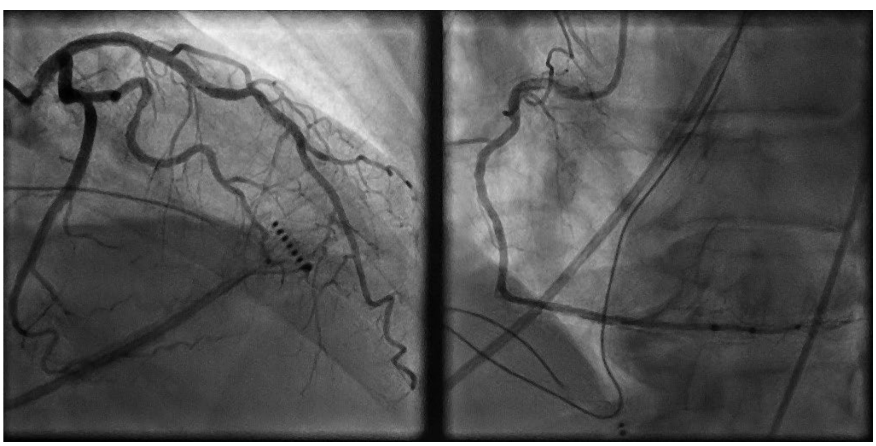

FIGURE 2 Coronary angiogram showing normal coronaries in a patient with transient STE. Used with permission from Soos M, C Madala M, Kanjwal K. Transient inferior lead ST elevation during radiofrequency ablation of atrial fibrillation. J Atr Fibrillation. 2020;12(6):2293

was attributed to lower perfusion distal to the stenosis resulting in transient ischemia.[9]

3.2 | STE during cavotricuspid isthmus ablations

Cavotricuspid Isthmus ablation is a highly successful procedure for the treatment of atrial flutter, with flutter-free rates of 93% two years after ablation.[10] In the first case of acute right coronary stenosis during an atrial flutter cavotricuspid isthmus ablation the patient underwent radiofrequency ablation and developed acute STE in leads II, III, aVF, V5, and V6 with reciprocal depression in anterior leads.[11] The mechanism of STE was thought to be a combination of a thin muscle wall between the endocardium and right coronary artery, thermal injury from radiofrequency ablation, and baseline existing proximal right coronary artery stenosis.[12] Because transmural injury is needed to ensure cavotricuspid isthmus block, the authors subsequently recommended ablating laterally to achieve a transmural lesion with the least amount of power output, titrating power output only if necessary, and using smaller catheter tips sized at 4-5 mm as opposed to 8 mm.[11]

In another study, the proximity of the cavotricuspid isthmus to the right coronary artery was assessed by obtaining electrocardiogram-gated contrast-enhanced computed tomography (CT) scans. The endocardium to right coronary artery thickness along the cavotricuspid isthmus was measured. Along the isthmus, the paraseptal thickness measured 9 ± 3 mm, the central isthmus thickness measure 6 ± 2 mm, and the lateral isthmus measured 5 ± 3 mm.[12]

3.3 | STE during coronary sinus ablations

The coronary sinus is being increasingly targeted for ablation of various arrhythmias including accessory pathways, premature ventricular contractions, and ablation to achieve mitral isthmus block. STE in anterior leads during ablation of premature ventricular complex ablation near the junction between the great cardiac vein and anterior interventricular vein has been reported.[13] Coronary angiography revealed spasm of the left anterior descending artery because of direct thermal injury and the patient had successful ablation.

STE has also been demonstrated after ablation of postero-septal accessory pathways from within the coronary sinus, where efficacy of accessory pathway ablation is higher.[14] Postero-septal accessory pathways account for 34.5% of all accessory pathways.[15] Of these postero-septal accessory pathways, ones that arise from the coronary sinus and connect with the epicardial surface of the left ventricle account for 19%.[16] STE is a risk of posteroseptal venous intraluminal ablation because of the distance of the ideal ablation site relative to the nearest coronary artery. In a study performed by Stavrakis et al of 169 patients who underwent coronary angiography, the distance of the ideal ablation site from a significant coronary artery was less than 2 mm in 100 of 169 patients.[14] Of these 100 patients, only 22 patients underwent ideal site ablation because of the high risk of adverse events. Of the 22 patients, 11 patients developed coronary artery stenosis evidenced by STE with one patient requiring balloon angioplasty for complete occlusion. The risk decreased when the distance between the ideal ablation site and the nearest coronary artery was >2 mm and was negligible when the distance was >5 mm. In patients with accessory pathways undergoing venous intraluminal ablation, there is also a risk of anomaly within the coronary sinus, such as a diverticulum, middle cardiac vein, or a vein opening into the coronary sinus, which can affect the outcome of the ablation.[15,16]

3.4 | STE during ventricular arrhythmia ablations

Transient STE during ablation of ventricular tachycardia was first documented in 2015 in a patient with ischemic ventricular ablation who demonstrated coved-STE in leads V1 and V2 after transseptal puncture.[17] The case report by Hori et al was subsequently re-evaluated by Gottschalk et al, as the STE did not occur in the predicted inferior leads as in other transseptal ablation case reports. The transient STE was determined to be a Brugada phenocopy during ablation of ventricular tachycardia because of coronary vasospasm of unknown etiology.[18] A case of left anterior descending coronary artery dissection during ablation of fascicular left ventricular tachycardia has been reported a swell.[19] The patient developed STE in leads V1 through V5 and the dissection was hypothesized to be because of catheter manipulation after several attempts at crossing the aortic valve.

ST segment elevation can result from other mechanisms besides injury to the coronary artery or hypoperfusion. Cardiac memory (CM) is a known phenomenon which can lead to electrocardiographic changes that can mimic ischemic changes.[20] However, there are few reports of CM that can present as STE. A case of STE in the anterior leads after ablation of premature ventricular contractions (PVCs) near the aortomitral continuity was reported recently.[20] The STE persisted for few hours and the patient was completely asymptomatic and the coronary angiography revealed a normal coronary anatomy without any stenosis, vasospasm, or air embolism (Figure 3).[20] The patient initially presented with bigeminy and the PVCs were discovered to originate in the area of the aortomitral continuity (Figure 4).[20] Once the bigeminy was abolished the CM presented as STE.[20]

3.5 | ST elevations during AVNRT ablations

The anterior-inferior aspect of the coronary sinus ostium is an ablation target for ablation of slow pathway for management of atrioventricular nodal re-entrant tachycardia (AVNRT) patients.[21] STE during ablation of slow pathway has been reported during catheter ablation of AVNRT.[22] In one such report, the patient had spontaneous ventricular fibrillation during the procedure and an emergent coronary

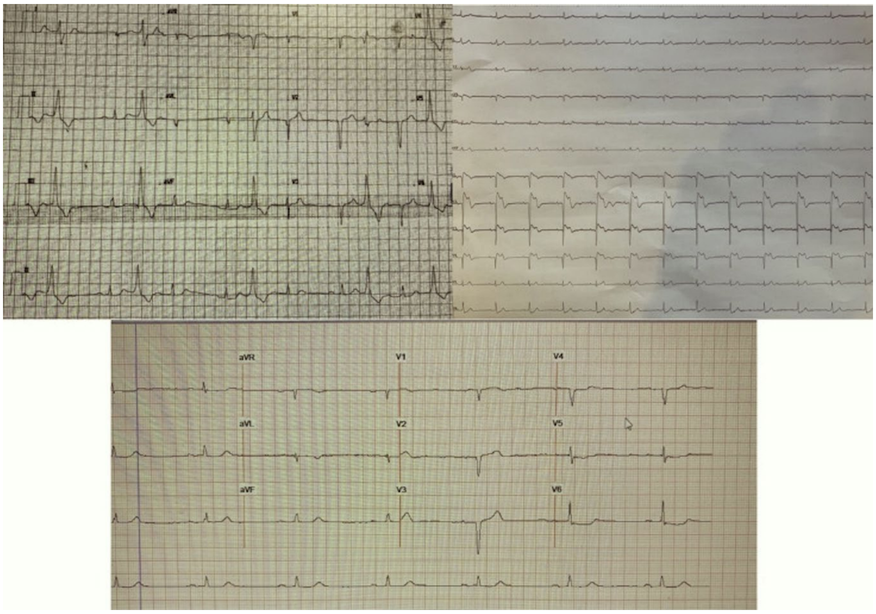

FIGURE 3 ECG Changes before and after premature ventricular contraction (PVC) ablation. Top left: Electrocardiogram (ECG) showed ventricular bigeminy with premature ventricular complexes (PVC) having the morphology of inferior axis and qR pattern in V1 with early transition. Top right: Post–PVC ablation ECG, showing ST elevation mostly noted in anterior leads (V1-V4). Bottom: A repeat ECG 4 h after the ablation, showing sinus bradycardia and complete resolution of ST elevations. Used with permission from Kichloo A, Haji AQ, Kanjwal K. Cardiac memory presenting as ST elevations following premature ventricular complex ablation. HeartRhythm Case Rep. Published November 4, 2020 [Online ahead of print]. https://doi.org/10.1016/j.hrcr.2020.10.011

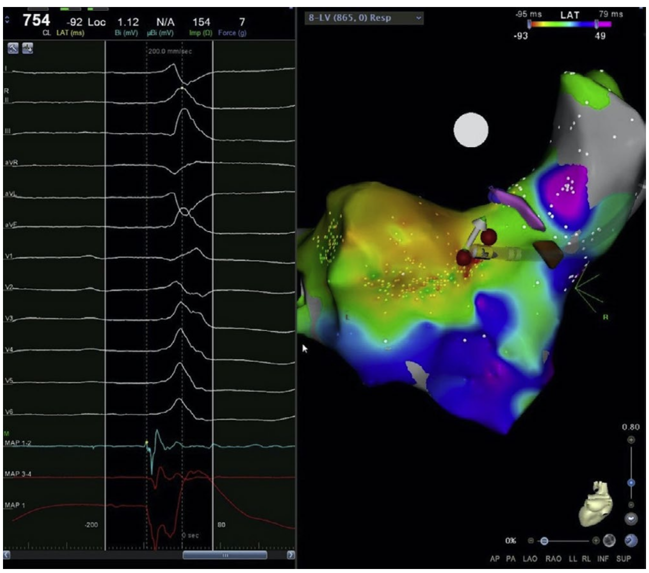

FIGURE 4 Activation map of the left ventricular outflow tract and aortomitral continuity (AMC) showing a successful site of ablation. Used with permission from Kichloo A, Haji AQ, Kanjwal K. Cardiac memory presenting as ST elevations following premature ventricular complex ablation. HeartRhythm Case Rep. Published November 4, 2020 [Online ahead of print]. https://doi.org/10.1016/j.hrcr.2020.10.011

angiography revealed complete occlusion of the posterolateral branch of the right coronary artery.[21] An acute stenosis of the posterolateral branch of the right coronary artery was reported in two other reports following ablation of slow pathway and was treated with intracoronary stent.[22,23]

ST segment elevation during various ablation procedures is frequently being recognized. The mechanism of STE is not exactly known, however, there are various possible mechanisms postulated for these ECG changes. As was previously eluded, some of these mechanisms include direct mechanical injury, coronary spasm, thermal injury, ganglionic plexus activation, Bezold–Jarisch-like-reflex, air embolism, and coronary steal phenomenon. Patients with underlying coronary stenosis are likely to be more susceptible to STE during ablation procedures.

Most ablation procedures in the United States are performed under general anesthesia and patients are not able to express their symptoms Thus, physicians need to pay attention not only to the cardiac rhythm but to ST-segment changes while performing these procedures to ensure proper and early recognition of this complication. Patients undergoing ablation using conscious sedation may report symptoms and these complications may be recognized earlier but many patients may still exhibit impaired coordination and cognition. In such situations relying only on patient-reported symptoms intraoperatively would yield inconsistent results. Furthermore, it has been reported that air emboli can occur after long apnea episodes produced by sedation, right before a loud snore with an increase in negative intrathoracic pressure, causing air to enter via the hemostatic valves or while exchanging circular mapping catheters.[24,25] Air emboli can travel to the coronary arteries or the cerebral arteries and cause hemodynamic collapse or stroke.[24] If sedation is used instead of anesthesia, using sedatives with lesser respiratory depression would minimize the risk of apnea-induced air emboli. Proper catheter exchange techniques and maintaining constant fluid to fluid interface while flushing the sheath or during aspiration of blood from the sheath is a key to the prevention of air embolism. Rarely, air embolism in addition to STE can also cause cerebral air embolism which may require treatment with hyperbaric oxygen. Lastly, removing the catheter from the sheath slowly, once the procedure is finished, to ensure a vacuum is not created in the sheath can avoid air from entering the vasculature.[24,25] These steps, while good practice even with the use of general anesthesia, would help minimize the risk of air emboli in a patient under conscious sedation.

It is also important to know when to stop the procedure to avoid adverse outcomes. The procedure should be aborted if the patient is hemodynamically unstable or develops ventricular arrhythmias. An urgent cardiac catheterization should be performed to rule out any coronary injury that would warrant intervention. If cardiac catheterization reveals normal coronaries the procedure may be continued. However, if any coronary intervention is performed, we recommend aborting the ablation procedure afterward. Also, we recommend aborting the procedure if the STE is recurrent. Physicians also need to be careful while performing ablations in patients with known coronary artery disease as these patients are more susceptible to have an adverse outcomes in case coronary artery injury occurs. There are times when these STE are observed when the procedure is completed, such as in patients who developed these changes following administration of protamine[9] and in a case of CM following ablation of PVC[20]. In such situations, a cardiac catheterization may be still warranted based on the clinical situation and physician's clinical judgment.

4 | CONCLUSION

ST segment elevation during ablation can be a rare complication. Physicians need to have a high level of suspicion and should pay close attention, not only to the cardiac rhythm, but to ST-segments as early recognition of STE may help prevent adverse outcomes.

CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

AUTHORSHIP STATEMENT

All authors contributed equally to the manuscript, including the collection of information for the literature searched, writing of the manuscript, and final approval for all submitted content.

ETHICS APPROVAL

The involved institutions do not require ethical approval for literature reviews.

INFORMED CONSENT

Informed consent was not required for literature reviews at the involved institutions.

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