

Transient ST-segment elevation due to coronary slow flow during cryoballoon application

Kriyobalon uygulaması esnasında koroner yavaş akıma bağlı geçici ST segment yükselmesi

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Summary– This report is a description of a rare case of transient ST-segment elevation during cryoballoon application for paroxysmal atrial fibrillation (PAF). A 74-year-old male with symptomatic PAF was referred to the center for ablation. During cryoablation of the left superior pulmonary vein (188 seconds, -48°C), ST-segment elevation in the anterolateral leads was observed suddenly, though without any significant complaint. Upon the increase in the ST segment, the cryoapplication was immediately terminated. Coronary artery angiography was performed less than 5 minutes after balloon deflation and demonstrated coronary slow flow with a corrected Thrombolysis In Myocardial Infarction (TIMI) frame count of 48.4 (normal range: 21 ± 3) in the left anterior descending artery (LAD). There was no significant flow-limiting lesion, coronary vasospasm, thromboembolus, or air embolus in any coronary vessel. The TIMI frame count for the right coronary artery and the circumflex artery was normal. The ST-segment elevation gradually returned to the baseline in 14 minutes without any intervention. A control TIMI frame count of the LAD was 22. A decision was made not to repeat the cryoapplication in this vein because there was both an entrance and an exit block. The other 3 pulmonary veins were then isolated uneventfully. The patient was discharged from the hospital the next day without symptoms or unusual electrocardiogram activity.

Transient ST-segment elevation has been reported as a rare clinical entity during different stages of atrial fibrillation (AF) ablation. Although a great majority of the reported cases are associated with air embolism, coronary vasospasm and thromboembolism are other rare causes.^[1–3] In a previously published article comparing pulmonary vein isolation (PVI) us-

Özet– Burada, paroksizmal atriyum fibrilasyonu (PAF) için kriyobalon uygulaması esnasında geçici ST segment yükselmesi yaşayan nadir bir olguyu sunduk. Şikayete yol açan PAF'lı 74 yaşındaki erkek hasta, ablasyon için merkezimize yönlendirildi. Sol üst pulmoner venin (188 saniye, -48°C 'de) kriyobalon ile ablasyonu sırasında, herhangi bir ciddi yakınma olmaksızın anterolateral derivasyonlarda ST segment yükselmesi geliştiği gözlemlendi. ST segment yükselmesinin artması üzerine kriyobalon uygulaması derhal sonlandırıldı. Balonun indirilmesinin ardından 5 dakikadan daha kısa bir sürede koroner arter anjiyografisi yapıldı ve sol ön inen arterde (LAD) 48.4 (normal aralık 21 ± 3) düzeltilmiş Thrombolysis In Myocardial Infarction (TIMI) kare sayısı ile koroner yavaş akım (KYA) gösterildi. Herhangi bir koroner arterde akımı kısıtlayıcı ciddi lezyon, koroner vazospazmı, trombo-emboli ya da hava embolisi mevcut değildi. Sağ koroner arter ve sirkumfleks arterin TIMI kare sayıları normaldi. Herhangi bir girişime gerek kalmaksızın ST segment yükselmesi 14 dakika içerisinde tedricen başlangıç haline döndü. Sol ön inen arterin kontrol TIMI kare sayısı 22 ölçüldü. Giriş ve çıkış bloğu zaten sağlandığından bu vene tekrar kriyobalon uygulaması yapılmadı. Diğer üç ven olaysız bir şekilde izole edildi. Hasta ertesi gün belirti ve elektrokardiyogram değişikliği olmadan hastaneden taburcu edildi.

ing cryoballoon and radiofrequency for paroxysmal AF, transient ST-segment elevation was detected in 1.5% of patients during first-generation cryoballoon ablation.^[4] The present case is a description of the occurrence of transient ST-segment elevation during cryoballoon ablation secondary to coronary slow flow (CSF).

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CASE REPORT

A 74-year-old male with a history of drug refractory paroxysmal AF was referred to the laboratory for ablation. The left ventricular ejection fraction and left atrial diameter were determined to be within the normal range on his initial transthoracic echocardiographic evaluation. Before the procedure, a transesophageal echocardiogram demonstrated a normal left atrial appendage flow velocity without thrombus formation. Since it is not our routine approach, preprocedural computed tomography was not performed for visualization of the left atrial morphology. Transseptal access was achieved under fluoroscopic guidance using an 8-F transseptal

sheath with a Brockenbrough SL 1 transseptal needle kit (BRK1; St. Jude Medical, St. Paul, MN, USA) and applying the standard technique.^[5] Once transseptal access was obtained, 100 units/kg of unfractionated heparin were administered intravenously, followed by an infusion of 1000 units/hour with the objective of maintaining an activated clotting time (ACT) of >300 seconds.

Abbreviations:

ACT	Activated clotting time
AF	Atrial fibrillation
CSF	Coronary slow flow
ECG	Electrocardiogram
hsTnI	High-sensitivity troponin I
LAD	Left anterior descending artery
PAF	Paroxysmal atrial fibrillation
PV	Pulmonary vein
PVI	Pulmonary vein isolation
TIMI	Thrombolysis in Myocardial Infarction

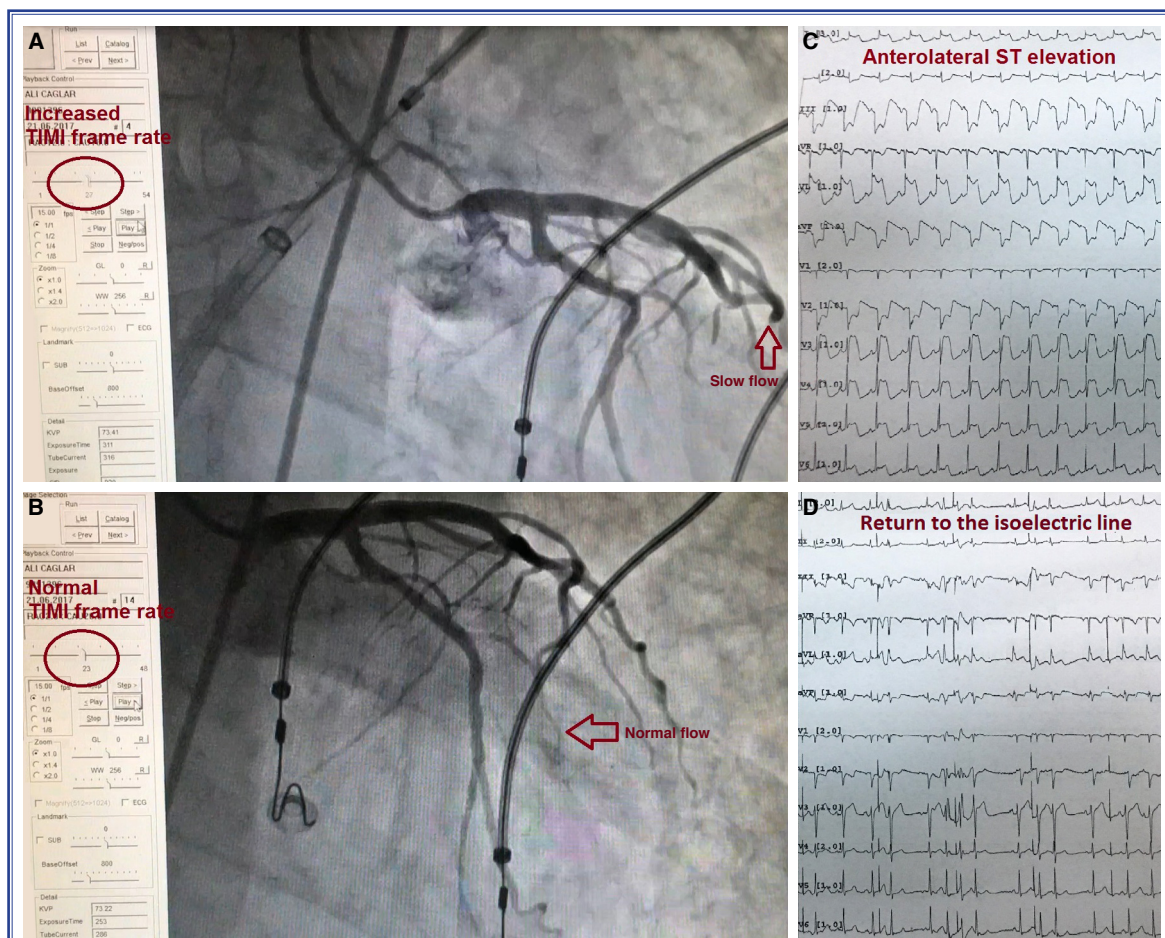


Figure 1. Twelve-lead electrocardiogram (ECG) and angiographic views of the patient during maximum ST-segment elevation and after the ST segment returned to the isoelectric line. **(A)** During maximum ST-segment elevation, a right anterior oblique coronary angiographic view demonstrated no significant stenosis in the epicardial coronary arteries, but coronary slow flow was seen in the left anterior descending artery (LAD). The arrow indicates the delay point of coronary flow. **(B)** After the ST segment returned to the isoelectric line, the right anterior oblique coronary angiographic view showed normal coronary flow in the LAD. **(C)** During cryoballoon ablation, a 12-lead ECG demonstrated significant ST-segment elevation in the anterolateral leads. **(D)** The 12-lead ECG returned to the isoelectric line within minutes after cryoballoon deflation. Please note the quantitative demonstration of coronary slow flow indicated with red circles.

Our routine approach for cryoballoon ablation is a single freeze cycle of 240 seconds in each pulmonary vein (PV). No additional bonus applications are performed once the isolation is achieved. Supported by a circular mapping catheter (Achieve; Medtronic, Inc., Minneapolis, MN, USA), a 28-mm second-generation cryoballoon catheter (Arctic Front; Medtronic, Inc., Minneapolis, MN, USA) was inserted at the ostium of the left superior PV. After a complete seal at the antral aspect of the PV was confirmed with a contrast medium injection, the cryoballoon application was initiated. During the first freezing attempt, at 188 seconds and -48°C , an ST-segment elevation was observed in the V1 and V6 leads, without any complaint. Because the 12-lead surface electrocardiogram (ECG) recording demonstrated ST-segment elevation in the anterolateral leads, the cryoapplication was terminated immediately (Fig. 1). The instantaneous intra-arterial blood pressure was 100/70 mm Hg (pre-procedural blood pressure was 120/80 mm Hg). An emergency bedside transthoracic echocardiogram showed good left ventricular systolic function and no evidence of pericardial effusion. Coronary artery angiography was performed less than 5 minutes after balloon deflation and revealed CSF with a corrected Thrombolysis in Myocardial Infarction (TIMI) frame count of 48 (normal range: 21 ± 3) in the LAD without any significant flow-limiting lesion, coronary vasospasm, thromboembolus, or air embolus (Fig. 1, Video 1*).^[6] The corrected TIMI frame count for the circumflex artery was within the normal range. Since the placement of the ST-segment elevation implied that the left coronaries were the culprit, selective coronary angiography of the right coronary artery was not performed. We had already administered

intravenous heparin treatment after the transseptal puncture. Therefore, we only checked the ACT level. It was greater than 250 seconds, and so an additional heparin bolus application was not performed. The quantity of intravenous fluid drip administered via a lateral line of the Cryocath catheter (Medtronic, Inc., Minneapolis, MN, USA) was increased. The ST-segment elevation started to decrease within 3 minutes and returned to baseline in 14 minutes, without any intervention (Fig. 1). A control coronary artery angiography was performed less than 10 minutes after ST-segment resolution and showed a TIMI-3 blood flow to all 3 major coronary arteries. The TIMI frame count of the LAD was 22 (Fig. 1, Video 2*). It was decided not to repeat the cryoapplication in this vein because there was both an entrance and an exit block. The other 3 PVs were then isolated uneventfully using a cryoballoon (the left inferior PV at 240 seconds, -45°C ; the right superior PV at 240 seconds, -51°C ; and the right inferior PV at 240 seconds, -42°C). The cardiac biomarker levels were checked 4 times over a 24-hour period. The peak level of high-sensitivity troponin I (hsTnI) was 4.4 ng/dL, detected at 12 hours after the procedure. The reference range in our laboratory for hsTnI was 0.0–0.06 ng/mL. The level dropped to 3.5 ng/dL at 24 hours. The patient was discharged from the hospital the next day without symptoms or unusual ECG activity (Fig. 2).

DISCUSSION

The present report highlights the rare complication of ST-segment elevation due to CSF during cryoballoon ablation for AF.

In previous reports describing ST-segment elevation during PVI, the elevation has been attributed to coronary spasm or air embolism.^[2,3] In the present case, a coronary angiogram was performed within a couple of minutes, which was earlier than in the cases reported in the published literature. First, the absence of a vasospasm on the coronary angiography performed at the point the ST-segment elevation was at its peak level excluded vasospasm as a preliminary diagnosis. Second, to avoid an air embolism, we used a continuous saline perfusion of the sheath throughout the procedure and we took extreme care to monitor for any air bubbles entering the system during the exchange of catheters and guidewires. Furthermore, the ST-segment elevation occurred during the cry-

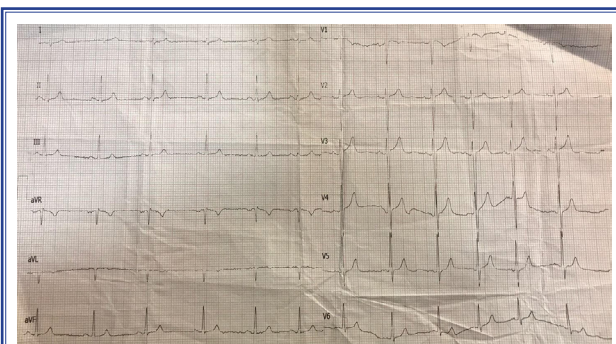


Figure 2. Discharge 12-lead electrocardiogram (ECG) of the patient. There was no ST-segment elevation or depression in any lead on the discharge ECG.

oballoon application, not after the catheter exchange. However, we cannot totally rule out an air embolism being the cause of the CSF.

Various mechanisms have been proposed as etiologies of CSF, such as abnormally high microvascular resistance and widespread atherosclerosis of the coronary arteries.^[7] In rare situations, CSF phenomenon may cause ST-segment elevation as well as myocardial ischemia.^[7] CSF phenomenon as a complication of AF ablation has been reported as occurring during transseptal catheterization. In 1 case, the authors attributed CSF to elevation of vagal tone.^[8] In the present case, a vagal discharge effect from the cryoballoon on the superior left atrial vagal ganglionated plexus, which has a close anatomical relationship with the left superior PV, was considered the primary responsible mechanism for excessive vagal tone.

In coronary interventions, ECG monitoring is usually single-lead, whereas during electrophysiological study, at least 3 leads are used to monitor the patients. We routinely use DI, DII, V1, and V6 leads for a surface ECG. Thus, we promptly realized there was ST-segment elevation in the present case, although the patient had no serious complaint or chest pain.

After balloon deflation, the ST-segment elevation immediately started to decrease in our patient. Neither thrombotic material nor emboli were detected during a control angiography performed within just a few minutes. Since there was no image of coronary spasm in the left system, we did not consider nitroglycerine administration. Although there was no bradycardia, a slight (about 20 mm Hg) reduction in systolic blood pressure was seen during ST elevation. It might be considered a clue to vagal hyperactivation. We did not consider administering atropine since the patient's heart rate was stable. But if we had, perhaps we could have reduced the vagal overactivity sooner. Since we were to begin warfarin therapy after the procedure, we did not start the use of any antiaggregant agent for the ST-segment elevation. The patient had already been heparinized after the transseptal puncture, and therefore, we did not use any additional anticoagulant agent, either.

At this point, another question that may come to mind may be the high troponin value determined after the procedure. However, it has previously been demonstrated by different groups that AF ablation causes significant non-ischemic myocardial damage

and this effect may be detected by an increase in cardiac biomarkers after the procedure.^[9,10] Antolič et al.^[9] demonstrated that elevation in cardiac biomarkers may be greater with a cryoballoon than radiofrequency ablation. In a recently published article, Aksu et al.^[10] demonstrated that this troponin elevation can be as high as a median value of 11.75 ± 5.25 ng/mL and may be related to lower recurrence rates. Although PVI is the main target in paroxysmal AF, substrate abnormality in the PV antrum may play a critical role in the AF mechanism. Additional ablation of the PV antrum after PVI may increase the efficacy of the procedure. Greater troponin release with cryoablation may be linked to more ablation damage in the left atrium compared with a radiofrequency-based PVI procedure. As a finding supporting this hypothesis, it has been recently demonstrated that PVI using a cryoballoon may cause greater complex fractionated atrial electrogram reduction than PVI using radiofrequency energy.^[11] In the present case, hsTnI achieved its maximum level at the 12th hour, whereas in ischemia-related myocardial necrosis, we generally expect to reach the peak troponin value between 18 and 24 hours. Thus, the troponin increase was assumed to be related to myocardial damage due to ablation, not with myocardial necrosis due to prolonged ischemia. However, it is not possible to say for sure that coronary ischemia has no role in a troponin increase.

In conclusion, ST-segment elevation during a cryoballoon application for PVI may be a transient and completely reversible phenomenon. CSF caused by excessive vagal discharge through neurally mediated pathways appears to be a possible explanation for this unexpected complication. However, an air embolism or a coronary vasospasm should always be considered in these cases.

**Supplementary video file associated with this article can be found in the online version of the journal.*

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