Spontaneous closure of a type 3 coronary artery perforation after percutaneous coronary intervention

Perkütan koroner girişim sonrası tip 3 koroner rüptürün spontan kapanması

Mehmet Vefik Yazıcıoğlu, Elnur Alizade, Göksel Açar, Zeki Şimşek¹ Clinic of Cardiology, Kartal Koşuyolu Heart Education and Training Hospital, İstanbul-*Turkey*

¹Clinic of Cardiology, Corum İskilip Atıf Hoca State Hospital, Corum-*Turkey*

Introduction

Coronary artery perforation (CAP) is a rare, but potentially serious complication of percutaneous coronary artery intervention (PCI). The strategies for management of patients with CAP include prolonged balloon inflation, coil embolization, covered stent implantation, pericardiocentesis, urgent surgery and reversal of heparin with protamine sulfate. We would like to share a case of type 3 perforation of left anterior descending artery (LAD) occurred during PCI, giving rise to cardiac arrest and closed spontaneously after effective cardiopulmonary resuscitation.

Case Report

A 84-year-old man presented to our emergency service with non-ST segment elevation myocardial infarction. There was hypertension, hyperlipidemia on his medical history without diabetes mellitus and previous percutaneous coronary intervention. He was started on aspirin, clopidogrel, enoxaparin, metoprolol and atorvastatin. Coronary angiography revealed multivessel involvement with a severe, eccentric and calcified stenosis of the proximal part of the LAD (Fig. 1A). The potential risks and benefits of the coronary artery by-pass graft (CABG) surgery were extensively discussed with the patient and his family, but they refused the procedure because of that we decided for PCI instead of CABG. The LAD lesion was crossed with a floppy guide wire which after advanced into the LAD with gentle manipulation.

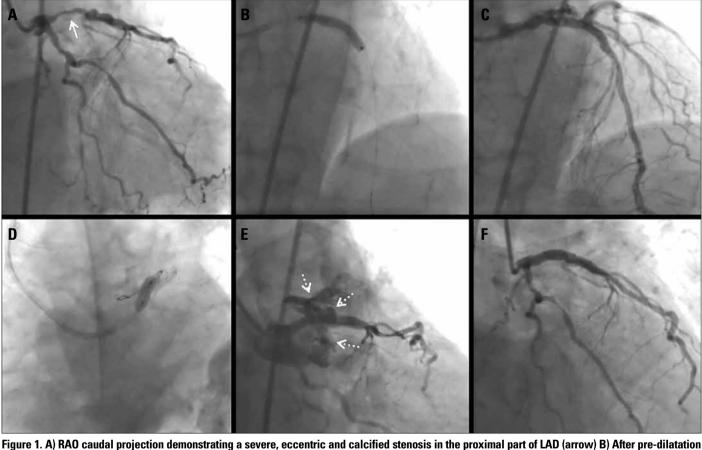


Figure 1. A) RAO caudal projection demonstrating a severe, eccentric and calcified stenosis in the proximal part of LAD (arrow) B) After pre-dilatation with a balloon and deployment of the Ephesus stent (3.5x22 mm) C) Suboptimal result after the stent deployment D) Post-dilatation was performed with a noncompliant balloon (3.5x22 mm) E) Type 3 perforation of the proximal part of LAD with contrast medium leaking into the pericardial cavity (arrows) F) Controlled angiography after two hours later showed no sign of perforation (TIMI flow Grade 0-No perfusion; no antegrade flow beyond the point of occlusion. Grade 1-Penetration without perfusion; contrast material passes beyond the area of obstruction but fails to opacify the entire coronary bed distal to the obstruction for the duration of the cineangiographic filming sequence. Grade 2-Partial perfusion; contrast material passes across the obstruction and opacifies the coronary artery distal to the obstruction. However, the rate of entry of contrast material into the vessel distal to the obstruction or its rate of clearance from the distal bed (or both) is perceptibly slower than its flow into or clearance from comparable areas not perfused by the previously occluded vessel. Grade 3-Complete perfusion; antegrade flow into the bed distal to the obstruction occurs as promptly as antegrade flow into the bed proximal to the obstruction, and clearance of contrast material from the involved bed is as rapid as clearance from an uninvolved bed in the same vessel or the opposite artery)

LAD - left anterior descending artery, RAO - right anterior oblique view

A 2.5x20 mm balloon was inflated at 12 atm for 10 seconds and 3.5x22 mm Ephesus® stent was deployed at 16 atm for 10 seconds with a suboptimal result (Fig. 1B, C). Post-dilatation was performed with a 3.5x22 mm noncompliant balloon at 20 atm for 10 seconds at the proximal part of the stent (Fig. 1D). After performing the last inflation, contrast injection revealed out a jet passing through pericardial cavity from the perforated part of LAD (Fig. 1E, Video 1). The patient's hemodynamic status deteriorated and soon after cardiac arrest occurred. We had started cardiopulmonary resuscitation (CPR) and 2 mg of epinephrine and 2 mg of atropine were used totally. During CPR, we had also concentrated on the management of perforation. Usage of enoxaparin instead of un-fractioned heparin before the procedure; was a mischance for us because lack of complete neutralization of enoxaparin with protamine sulfate. After 8 minutes of effective CPR and getting the pulse, we planned to inflate a balloon in the proximal LAD to stop the perforation. Soon after injecting the contrast into the left coronary system, there were no perforation and no jet image passing through the pericardial cavity. After CPR and resolution of the coronary perforation, the patient had been stabilized with a blood pressure of 135/75 mmHg and heart beat 78/min. Urgent echocardiography showed minimal pericardial effusion with no signs of cardiac tamponade. For this reason, we got back out of performing pericardiocentesis. Serial echocardiographic examinations were performed every 15 minutes, which revealed no change in the amount of pericardial effusion. Two hours later, we performed an angiographic control and we had seen the same miraculous image without the sign of perforation (Fig. 1F, Video 2). As he was stable hemodynamically and echocardiographic findings were normal, the patient was extubated hours after the procedure. The patient had an uneventful course with with successful outcome and was discharged on medical therapy.

Discussion

Coronary artery perforation is a rare complication of PCI which has a poor prognosis and leads to life-threatening conditions because of cardiac tamponade or acute myocardial infarction. Although we were not sure about the cause for cardiac arrest in our case , the distal ischemia could be the leading cause after LAD rupture which was already prone to the ischemia because low TIMI flow grade after PCI. The incidence of CAP in the general population undergone PCI has been reported to range from 0.1% to 0.4% (1, 2). In a meta-analysis of 16 studies by Shimony et al. (3) involving 197.061 PCIs, the pooled incidence of CAP was 0.43%. Among the possible factors predicting its occurrence are clinical, angiographic and technique-associated factors (4). The Ellis classification is commonly used for coronary perforations (1). Type 1 perforation was defined as an extraluminal crater without extravasation, in the absence of angiographic findings suggestive for dissection; type 2 was defined as pericardial or myocardial blush without contrast jet extravasation; type 3 was defined as extravasation through a clearly visible (>1 mm) perforation; and type 4 was defined as extravasation of contrast dye into a cardiac chamber or cavity such as the left ventricle or coronary sinus but not pericardial space. Although there is no consensus about the optimal treatment of CAP, treatment strategy varies according to the clinical settings and Ellis classification. The management includes reversal of heparin, discontinuation of Gp2b/3a inhibitors, platelet transfusion, pericardiocentesis and emergency surgery. Treatment strategies also include prolonged balloon inflation, covered stents, injection of polyvinyl alcohol, coil embolization and intracoronary administration of autologous blood (5-9). Although there is a case report written previously about conservative management of type 3 perforation of a side branch of the left main coronary artery during coronary angiography, there is no report written for spontaneous closure of type 3 CAP located on LAD (which has a higher flow rate compared to side branches) without any intervention (10). The mechanisms for spontaneous closure of type 3 perforation might be explained by following possible mechanisms. Some degree of acute recoil after postdilatation may have a role in the sealing of the perforation. The high concentration of elastic fibers at the proximal part of the coronary arteries compared to the distal parts has been proposed as the possible mechanism of elastic recoil (7). The development of hypotension and cardiac arrest could cause the collapse of the coronary artery and facilitate the spontaneous closure. Small amount of the blood collected in the pericardial cavity in a short time with the help of chest compression during CPR can increase the pressure in pericardial cavity and it slows down the jet velocity from the perforated area without causing cardiac tamponade. These mechanisms might have acted separately or together.

Conclusion

As a conclusion, type 3 CAP has a high mortality and usually requires percutaneous or surgical intervention. As the spontaneous closure is very rare, we intend to report it and discuss the possible mechanisms of spontaneous closure with colleagues with the hopes for offering additional information to the existing literature.

Video 1. Multiple coronary artery perforations following balloon dilation, with extravasation of contrast into the pericardial space. Extravasation through a perforation (>1 mm)

Video 2. Controlled angiography after two hours later showed no sign of perforation

References

- Ellis SG, Ajluni S, Arnold AZ, Popma JJ, Bittl JA, Eigler NL, et al. Increased coronary perforation in the new device era: incidence, classification, management, and outcome. Circulation 1994; 90: 2725-30. [CrossRef]
- Dippel EJ, Kereiakes DJ, Tramuta DA, Broderick TM, Shimshak TM, Roth EM, et al. Coronary perforation during percutaneous coronary intervention in the era of 75 abciximab platelet glycoprotein IIb/IIIa blockade: an algorithm for percutaneous management. Catheter Cardiovasc Interv 2001; 52: 279-86. [CrossRef]
- Shimony A, Joseph L, Mottillo S, Eisenberg MJ. Coronary artery perforation during percutaneous coronary intervention: A systematic review and meta-analysis. Can J Cardiol 2011; 27: 843-50. [CrossRef]
- 4. Tomás C, Fernando P, Carlos L, José M. An alternative treatment for iatrogenic coronary perforation. Rev Esp Cardiol 2009; 62: 328-9. [CrossRef]
- Briguori C, Nishida T, Anzuini A, Di Mario C, Grube E, Colombo A. Emergency polytetrafluoroethylene- covered stent implantation to treat coronary ruptures. Circulation 2000; 102: 3028-31. [CrossRef]
- Cordero H, Gupta N, Underwood PL, Gogte ST, Heuser RR. Intracoronary autologous blood to seal a coronary perforation. Herz 2001; 26: 157-60. [CrossRef]
- Villa M, de la Llera LD, Morán JE, Pérez-Cortacero JA, Fournier JA. Coronary perforation during unprotected left main angioplasty. Management with conservative approach: a case report. Int J Cardiol 2004; 97: 145-6. [CrossRef]
- Yorgun H, Canpolat U, Aytemir K, Oto A. Emergency polytetrafluoroethylene-covered stent implantation to treat right coronary artery perforation during percutaneous coronary intervention. Cardiol J 2012; 19: 639-42.
- Bilge AK, Nişancı Y, Özben B, Yılmaz E, Umman B. Coronary perforation and tamponade during thrombectomy and treatment with PTFE coated stent and autotransfusion: a case report. Anadol Kardiyol Derg 2003; 3: 174-6.
- Özdoğru I, Eryol NK, Taşdemir K, İnanç MT, Doğan A, Kaya MG. Conservative management of the perforation of a side branch of the left main coronary artery during coronary angiography. Int J Cardiol 2008; 126: e55-7. [CrossRef]

E-mail: elnur17@vahoo.com

Address for Correspondence/Yazışma Adresi: Dr. Elnur Alizade,

Kartal Koşuyolu Kalp Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, Denizer Cad. Cevizli Kavşağı, No:2, 34846 Cevizli, Kartal, İstanbul-*Türkiye* Phone: +90 216 459 44 40



Available Online Date/Çevrimiçi Yayın Tarihi: 26.09.2013

© Telif Hakkı 2013 AVES Yayıncılık Ltd. Şti. - Makale metnine www.anakarder.com web sayfasından ulaşılabilir.

© Copyright 2013 by AVES Yayıncılık Ltd. - Available online at www.anakarder.com doi:10.5152/akd.2013.230

Ablation of heterogeneous zone eliminates ventricular tachycardia: Can cardiac MR be a criterion for successful ablation?

Heterojen bölgenin ablasyonu ventriküler taşikardiyi elimine etmiştir: Kardiyak MR başarılı ablasyon için bir kriter olabilir mi?

Kıvanç Yalın, Ebru Gölcük, Ahmet Kaya Bilge, Kamil Adalet Department of Cardiology, İstanbul Faculty of Medicine, İstanbul University, İstanbul-*Turkey*

Introduction

Surviving myocytes in the heterogeneous infarct borders of the scar tissue due to previous myocardial infarction, may provide a critical arrhythmogenic substrate for ventricular tachycardia (VT) (1). Ablation of this critical substrate can eliminate VT. Cardiac magnetic resonance (CMR) imaging can visualize scar tissue, and CMR may allow detailed characterization of infarcts by differentiating the core and peripheral regions. Imaging of heterogeneous zone by CMR has prognostic significance. A clinical study with CMR has reported the associations of the heterogeneous zone with inducible ventricular arrhythmias (2). However, significance of heterogeneous zone ablation has never been studied.

In this report, we present a case of post-myocardial infarction (MI) patient with VT in whom ablation that eliminated the VT caused severe decrease in heterogeneous zone percent seen on CMR.

Case Report

A 75-year-old man was referred to our laboratory due to further evaluation. He experienced inferior myocardial infarction 14 years ago and underwent coronary bypass surgery. A month ago, he was admitted to ER due to palpitation. Electrocardiogram showed monomorphic VT at a rate of 162 beats/min with right bundle branch block morphology and northwest axis (Fig. 1). He was hemodynamically stable. Medical cardioversion attempt with amiodarone failed. He was then electrically cardioverted and oral amiodarone treatment was started. A month after cardioversion he was referred to our institution for further evaluation. The echocardiography showed mild systolic dysfunction (LVEF:45%), inferior and posterior akinesia and basal and mid lateral hypokinesia with normal left ventricular diameters.

CMR was performed in multiple anatomic planes using T1-weighted and cine steady-state free precision sequences. Left ventricular endsystolic volume and end-diastolic volume were 130.10 mL and 72.18 mL, respectively. Gadolinium- enhanced sequences to evaluate early myocardial perfusion and delayed myocardial enhancement were also performed, using 0.1 mL/kg gadobenate dimeglumine. DE-CMRI demonstrated transmural scar in inferior and inferoseptal mid- and basal segments, non-transmural scar in mid-inferolateral segments, which included heterogeneous enhancement pattern in mid inferior segment (Fig. 2A). A custom developed program was used for quantification of the scar core and the heterogeneous zone based on SI thresholds (>3SDs and 2 to 3 SDs above remote normal myocardium, respectively). Coronary angiography showed patent bypass grafts. During electrophysiologic study hemodynamically stable sustained monomorphic VT (CL: 347 msec) was easily induced. Catheter ablation was performed using CARTO (Biosense Webster, New Brunswick, USA). Based on bipolar voltage amplitudes of scar \leq 0.5 mV, scar border 0.5-1.5 mV, and healthy tissue \geq 1.5 mV, an electro-anatomical map of the left ventricle in sinus rhythm revealed an infero-lateral scar (Fig. 3). During sinus rhythm, diastolic potentials and fragmented potentials were detected on the corresponding regions that were represented as heterogeneous zones by CMR. Because of the repetitious termination of the tachycardia by pacing maneuvers, the entrainment mapping could not be performed and pace mapping was preferred. Pace mapping at a border zone revealed

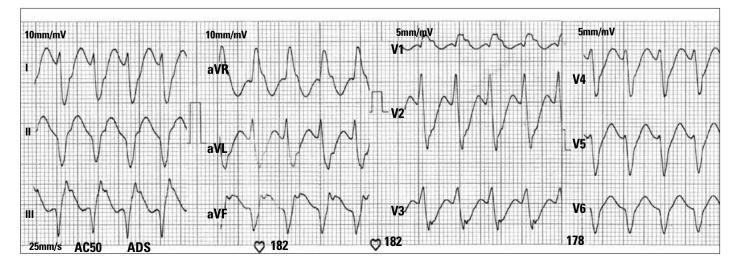


Figure 1. Electrocardiogram during sustained and well-tolerated monomorphic ventricular tachycardia