No-Reflow Fenomeniyle İlişkili Nadir Yerleşimli Miyokardiyal Köprüleme

Rare Myocardial Bridging Associated With No-Reflow Phenomena

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Özet

Miyokardiyal köprülemeyle işlem esnasında gelişebilecek no-reflow fenomeni arasındaki ilişki değerlendirilmiştir. Miyokardiyal köprüleme, epikardiyumda seyreden koroner arterlerin bir bölümünün kas içinde seyretmesi ile ortaya çıkan ve sistolik bası sonucu arterlerde daralmaya yol açan konjenital anomalidir. Neden olduğu sistolik bası ve endotel disfonksiyonu nedeniyle koroner iskemiye yol açarak çeşitli klinik tablolara yol açabilir. Vakamızda alt yüz kalp krizi nedeniyle Sağ Koroner Arter'e (RCA) acil perkütan girişim yapıldı. İşlem sırasında no-reflow gelişmesi nedeniyle hastaya glikoprotein IIb-IIIa infüzyonu verildi. Ardından yapılan kontrol koroner anjiyografide RCA orta bölgede enfarktüsden sorumlu %80 darlık yapan disseke lezyon, takip eden RCA'nın arka inen dalında (PDA) %99 darlık yapan miyokardiyal köprüleme tespit edildi. PDA tutulumlu miyokardiyal köprüleme nadir bir durumdur. Akut koroner sendrom nedeniyle yapılacak perkütan girişimlerde miyokardiyal köprüleme no-reflow gelişme riskini arttırabilir.

Anahtar Kelimeler: Miyokardiyal Köprüleme, Akut koroner sendrom, No-reflow

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Abstract

Myocardial bridge (MB) is a congenital abnormaly that the a part of an epicardial artery follows a route inside myocardium and leads to narrowing with systolic compression. Most commonly seen in left anterior descending artery (LAD). By means of compression and endothelial dysfunction it can lead coronary ischemia and various clinical presentations. Our case presented to our clinic with inferior myocardial infarction and primary percutenous intervention performed for right coronary artery (RCA). No reflow developed during intervention and glycoprotein IIb-IIIa infusion given to patient. Control coronary angiography revealed a dissecting lesion with a 80% stenosis at the distal of RCA after the infarct related lesion and a myocardial bridge with a 99% stenosis at the posterior descending artery (PDA). Myocardial bridging at the PDA is a very rare situation. Percutenous intervention on those lesions during acute coronary syndrome may result with no-reflow phenomena. Myocardial bridging may increase the risk of no-reflow in acute coronary syndromes.

Keywords: Muscular bridge, Acute coronary syndrome, No-reflow

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Introduction

In autopsy series myocardial bridging can be detected in 50% and 0,5-2,5% of angiographic studies but this rate approaches to 40% when provacation performed by nitroglycerine and inotropic agents.[1-3] The condition is mainly confined to the left anterior descending coronary artery. Myocardial bridge of the right coronary artery is rare.[4] Angina pectoris, myocardial infarction, arrythmia can be seen.[1,3] Recently it has been showed that besides mechanical effect they may have tendency to endothelial dysfunction and atherosclerosis.[5]

Case

61 years old female patient without prior cardiac

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symptoms admitted to our hospitals emergency department with chest pain ongoing for 3 hours. She was followed up by a physician with the diagnosis of diabetes, hypertension and hyperlipidemia for 2 years and she was using ramipril, metformin, and atorvastatin. Blood pressure was 110/70 mmHg, pulse 110 beats/min and physical examination findings were normal. Electrocardiogram revealed ST segment elevation on inferior leads and reciprocal ST depressions on anterior leads with ST segment elevations on V4R-V6R. Patient was urgently taken to coronary angiography lab with diagnosis of acute inferior MI with right ventricular involvement. There were no any critical lesions at LAD (Left Anterior Descending) and Cx (Circumflex) arteries but there were thrombotic mid occlusion of RCA. After 600 mg of clopidogrel loading and IV 10.000 units heparin bolus intervention for RCA was planned. RCA was cannulated succesfully with 7F JR4 guiding cathether and lesion was passed by guide wire 0,014 Asashi. On to lesion and distal of lesion consecuitive balloon angioplasties performed with 8-10 atm. pressures with 2,75 – 15 mm balloon. Distal TIMI 0-1 coronary flow was traced because of thrombus load (***Figure 1***). Intervention was ended and patient was taken to coronary care unit. Tirofiban infusion with 0,15 mcg/kg/min started for 24 hours after a 10 mcg/kg with 3 minutes bolus and Acetylsalycylate, Clopidogrel, Atorvastatin, Metoprolol, Ramipril and Enoxoparin medicated for the patient. 24 hours after initial intervention patients hemodynamic status was stabilized and a control angiography performed. There were TIMI-3 flow at RCA, but there were dissecting lesion with 80% stenosis at mid RCA, and there were MB at the following PDA (***Figure 2***). Intervention planned for the mid portion of RCA. RCA cannulated by the 6F JR4 succesfully and distal lesion were passed with 0,014 Asashi guidewire. Ephesos 3,0-20 mm bare metal stent was placed on to lesion with pressure of 17 atm for 15 seconds. TIMI-3 flow was achieved. As the distal flow improved, it has seen that MB at the PDA had a critical stenosis with 99% (***Figure 3***). Treatment continued after intervention. Tirofiban infusion continued untill dose finished. Patient was discharged at the end of fifth day without any complication.

Figure 1 : Total occlusion of RCA mid portion and no reflow development after intervention.

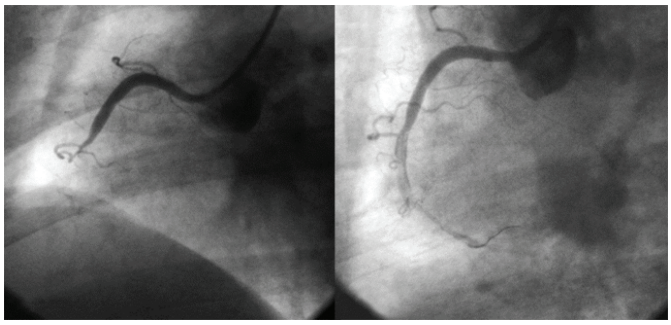


Figure 2 : Image of RCA mid 90_ stenosis and muscular bridge with severe stenosis at PDA after tirofiban infusion. (First image in systolic phase, second image in diastolic phase)

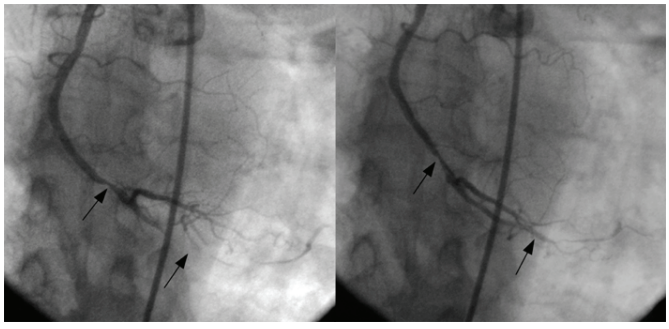
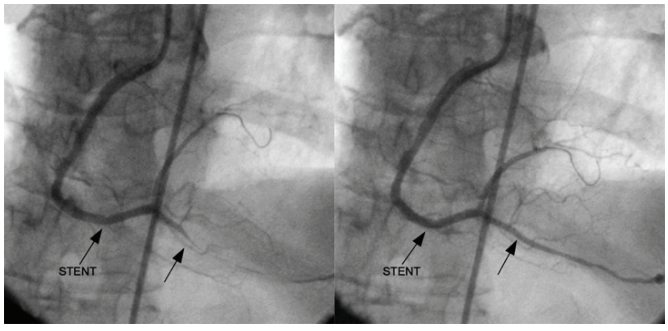


Figure 3 : . Image of RCA mid segment and PDA muscular bridge with sever stenosis after stenting procedure. (First image in systolic phase, second image in diastolic phase)



Discussion

It has been seen that atherosclerotic load at the segment distal to MB was lower than the arterial segments proximal to MB by intravascular ultrasound and histopathologic examinations showed that the caracteristics of endothelium lying the proximal and distal of MB were

different.6-8 It has been proposed that lower shear stress at the segment proximal to MB increases the atherosclerotic process and with at the segment distal to the MB, there is lower atherosclerosis because of increasment of lymphatic drainage with systolic compression.[1,5] Besides this, vasoconstriction and thrombus formation can be seen at the segment distal to the MB because of higher shear stress can lead to tears at the vessel wall, endothelial damage, plaque rupture and thrombocyte activation.[6] MB can lead to ischemia with mechanical compression in early diastole and increased thrombocyte activation if there is prolonged relaxation.[1] No-relow phenomenon is the situation in case of coronary intervention during acute coronary syndomes that is the result of following proposed mechanisms; embolization of distal thrombus and plaque remnants, microvascular neutrophile and inflammatory cell infilitration, increased vasoconstriction and mechanical compression because of myocardial edema, oxidative stress because of ischemia – reperfusion injury.[9] MB can increase the risk of no-reflow phenomena by increasing endothelial dysfunction and thrombocyte activation and distal decrement of blood flow by systolic – early diastolic compression.[6]

No reflow risk can be increased in lesions if there is MB distal to the lesion in case of intervention during acute coronary syndrome as in our case. In order to decrease the risk of no-reflow phenomena, preventing of distal embolization by direct stenting of lesion, thrombus aspiration before stenting, pre and post intervention use of glycoprotein IIb-IIIa inhibitors can be performed.[9]

Concurrent inflammation, thrombosis and distal embolization can lead to no-reflow phenomena in case of acute coronary syndromes. Myocardial bridging is an anomaly that increases the risk of no-reflow phenomena by the hemodynamic and cellular changes in coronary arteries.

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