

Disappearance of myocardial bridging of the left anterior descending coronary artery after inferior myocardial infarction

İnferiyor miyokart enfarktüsü sonrası sol ön inen koroner arterde miyokart köprüleşmesinin kaybolması

Bekir Serhat Yıldız, M.D., Fatma Esin, M.D.,# Yusuf İzzettin Alihanoğlu, M.D., İsmail Doğu Kılıç, M.D., Harun Evrengül, M.D.

Department of Cardiology, Pamukkale University Faculty of Medicine, Denizli;

#Department of Cardiology, Denizli State Hospital, Denizli

Summary– Myocardial bridging (MB) is defined as the intramural course of a major epicardial coronary artery, and is mostly confined to the left ventricle and the left anterior descending coronary artery (LAD). MB is a common congenital abnormality of a coronary artery, and is usually thought to be a benign anatomical variant. Although rare, previous studies have reported that patients with MB may suffer from myocardial ischemia, myocardial infarction (MI), arrhythmias, and even sudden death. Therefore, the diagnosis and treatment of MB are both important. Since MB is congenital, its disappearance is unlikely. We here report a very rare case of disappearance of MB after inferior MI.

Myocardial bridging (MB) is defined as an intramural segment of a coronary artery that normally courses epicardially. The intramyocardial coronary arterial segment is termed a “tunneled segment”. It is considered a congenital anomaly that most commonly affects the mid portion of the left anterior descending artery (LAD). The presence of MB is indicated angiographically by a narrowing of the coronary arteries during systole and normalization during diastole. MB was generally considered a harmless anatomical variant of the coronary arteries.^[1,2] However, some complications, such as ischemia, acute coronary

Özet– Miyokart köprüleşmesi (MK) büyük bir epikardiyal koroner arterin duvar içi seyri olarak tanımlanmaktadır. Çoğunlukla sol ventrikül ve sol ön inen arter (LAD) ile sınırlıdır. MK koroner arterin yaygın bir konjenital anomalisi olup genellikle iyi huylu anatomik bir varyant olarak düşünülmektedir. Nadir olmasına rağmen yapılan çalışmalarda MK bulunan hastalarda miyokart iskemisi, miyokart enfarktüsü, aritmiler ve hatta ani ölüm olabildiği bildirilmiştir. Bu nedenle, MK'nin hem tanısı hem de tedavisi önemlidir. MK doğuştan olduğu için kaybolması pek mümkün değildir. Biz burada inferiyor miyokart enfarktüsü sonrası kaybolan çok nadir görülen bir MK'li olguyu sunuyoruz.

syndromes, coronary spasm, arrhythmias, and sudden death, have been reported. Therefore, the diagnosis and treatment of MB are both important. Since MB is congenital, its disappearance is unlikely.

We here report a very rare case of disappearance of MB after inferior myocardial infarction (MI).

CASE REPORT

A 54-year-old male was admitted to our clinic with the complaint of atypical chest pain. The 12-lead electrocardiogram (ECG) showed sinus rhythm, and a negative T wave was seen on V3-6 derivations (Figure 1a). The chest radiograph was normal. On the physical examination, blood pressure was 130/75 mmHg and heart rate was 68/min without murmurs. Ejection fraction (EF) was found to be normal in the

Abbreviations:

CA	Coronary angiography
CABG	Coronary artery bypass graft
Cx	Circumflex artery
ECHO	Echocardiography
EF	Ejection fraction
LAD	Left anterior descending artery
MB	Myocardial bridging
MI	Myocardial infarction
RCA	Right coronary artery

Presented at the 29th Turkish Cardiology Congress with International Participation (October 26-29, 2013).

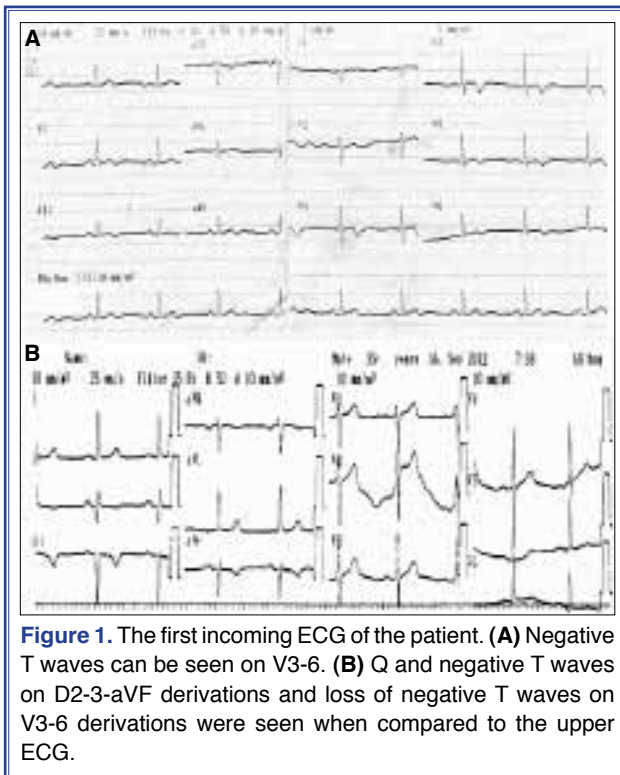
Received: October 27, 2013 Accepted: December 31, 2013

Correspondence: Dr. Bekir Serhat Yıldız, Pamukkale Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, 20100 Denizli.

Tel: +90 258 - 444 07 28 e-mail: bserhatyildiz@yahoo.com

© 2014 Turkish Society of Cardiology



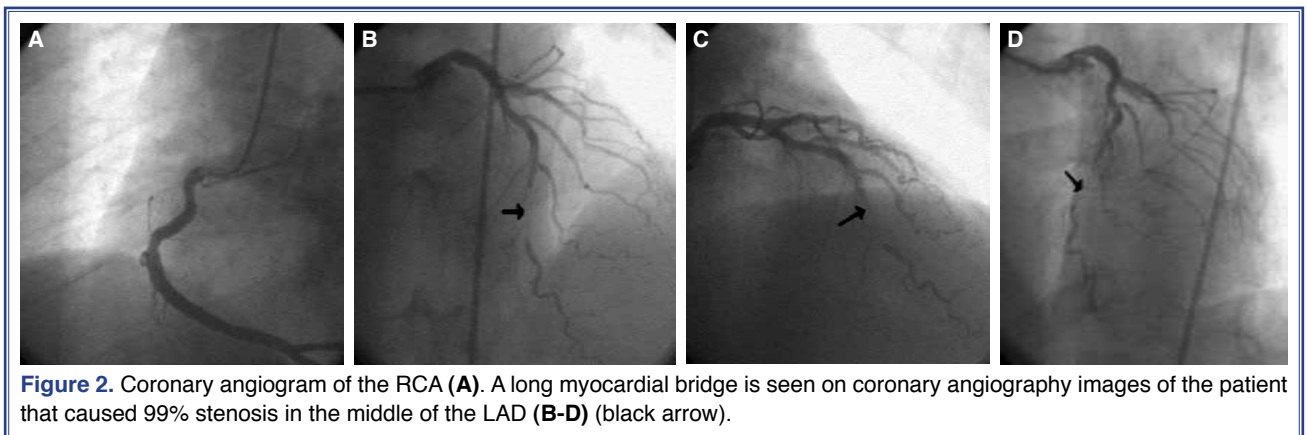


echocardiography (ECHO). He underwent an exercise stress test due to family history and smoking. 2 mm ST segment depression was seen in D1-aVL and V3-6 derivations. Coronary angiography (CA) was performed, which showed plaques in right coronary artery (RCA) and circumflex artery (Cx), along with MB causes of 99% stenosis in the middle of the LAD (Figure 2, Video 1a-c*). Coronary artery bypass graft (CABG) was recommended to the patient, but he refused the operation. He was discharged with medical therapy (beta-blocker - metoprolol 25 mg, aspirin 100 mg). The patient returned to our emergency depart-

ment with the complaint of typical chest pain one year later. Cardiac enzymes were elevated, Q waves and negative T waves were seen on D2-3-aVF derivations, and negative T waves on V3-6 derivations had disappeared (Figure 1b). The patient was admitted to coronary intensive care. Bedside ECHO was performed. Wall motion abnormality was observed in the inferior and posterior wall. EF was 50%. CA was performed due to ongoing chest pain, and showed plaque in Cx and LAD and 100% stenosis in the proximal area of the RCA. The last CA images were compared with the old CA images. Nearly complete disappearance of MB was seen in the middle of the LAD (Figure 3, Video 2a, b*). Stent deployment following balloon angioplasty was done for RCA, and 100% patency of the RCA was achieved after percutaneous coronary intervention (Figure 4, Video 3a, b*). His chest pain was relieved. CA was performed three months later. The RCA was open, and there was no MB in the middle of the LAD as seen on CA images (Figure 5).

DISCUSSION

Myocardial bridging can be seen as an incidental finding on coronary arteriography. Previous studies have reported its prevalence at 1.5-16% when assessed by CA, but in some autopsy series, it is as high as 80%.^[3] LAD was exclusively involved in 70%. Ferreira et al.^[4] described two different types of MB: (i) the superficial type, which crosses the coronary artery perpendicularly or at an acute angle toward the apex, and accounts for the majority of cases; and (ii) muscle fibers arising from the right ventricular apical trabeculae that cross the LAD transversely, obliquely or helically before terminating in the interventricular



septum. We considered our patient as having the second type because total occlusion of the RCA might cause necrosis on muscle fibers that cross the LAD

and change the motion and direction of the muscle fibers. As a result, this change might have been caused by the nearly complete disappearance of the MB of

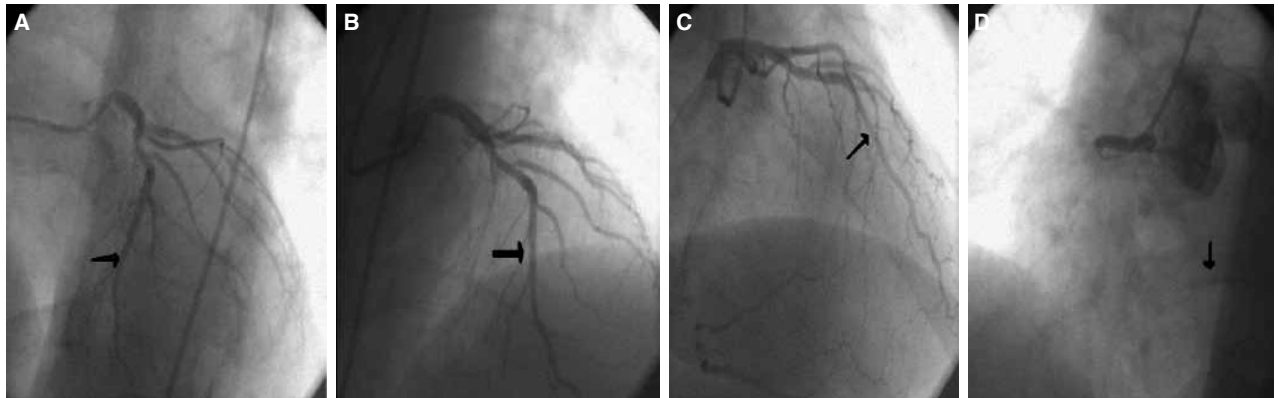


Figure 3. Coronary angiography images of the patient after myocardial infarction. Disappearance of myocardial bridging can be seen in Figures (A) and (B) (black arrow). Occlusion of the proximal RCA and antegrade flow in the distal part of the RCA can be seen in Figure (C) and (D) (black arrow).

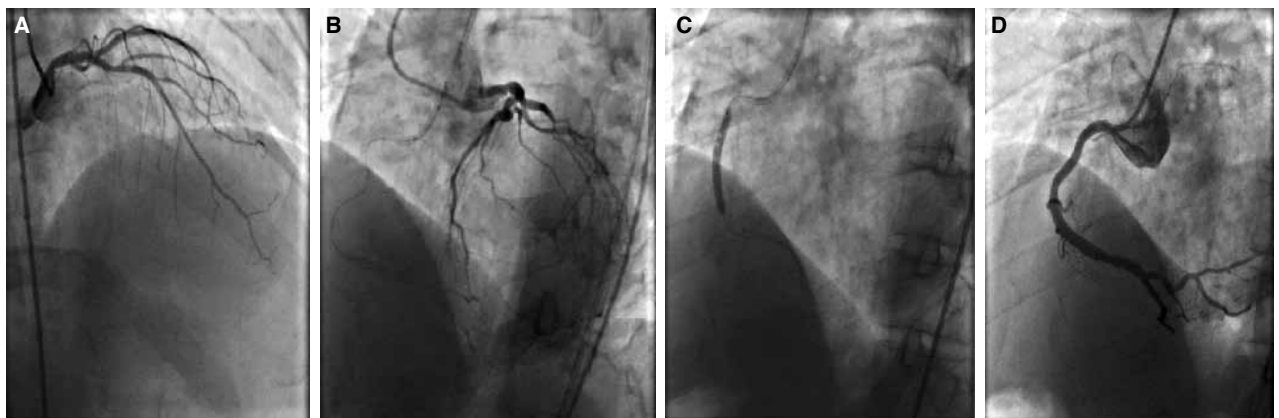


Figure 4. No myocardial bridging in the middle of the LAD on left main coronary artery image can be seen during percutaneous coronary intervention (PCI) of the RCA (A) and (B). 100% patency of the RCA was achieved after PCI (C) and (D).

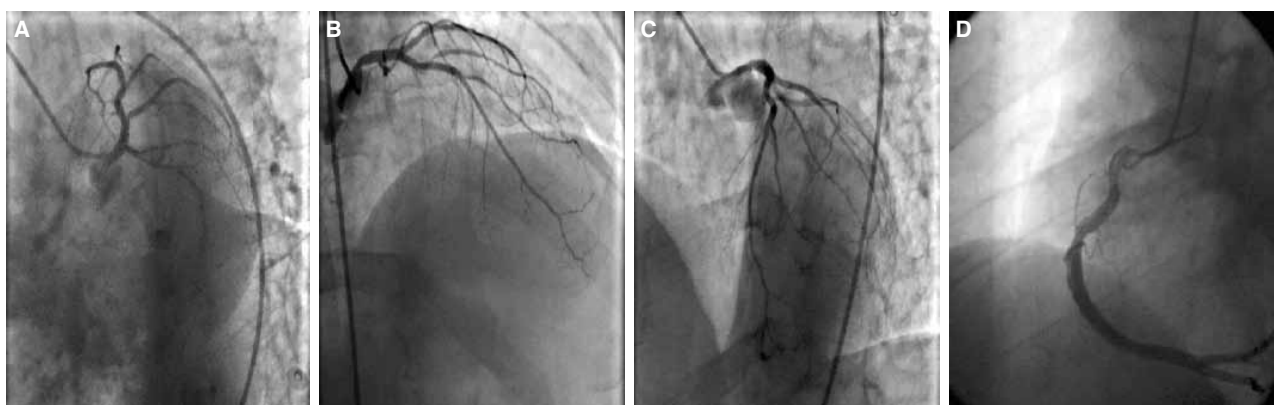


Figure 5. (A-D) Coronary angiography images of the patient taken three months later. The RCA is open and no myocardial bridge in the middle of the LAD can be seen.

the LAD. In addition, beta-blocker uptake, although at a lower dose, and bradycardia might have contributed to reduction of the MB.

Myocardial bridging often occurs without overt symptoms and is generally a benign condition. The clinical significance of the bridge is determined by the anatomy of the tunneled segment, as well as concomitant atheromatous changes and possible myocardial ischemia. During systole, contraction of the overlying myocardium compresses the artery; this compression may persist into diastole, when the majority of coronary blood flow occurs. Increased heart rate, short diastolic perfusion time, increased myocardial contractility and flow velocity, and exercise-induced coronary spasm can all cause ischemia in patients with MB. The length and depth of the intramyocardial segment have previously been correlated with ischemia or sudden death. However, there are no clear criteria of what defines a long or a deep tunneled segment.^[5-7]

In symptomatic patients, management of MBs is usually medical and rarely surgical. Available medications include beta-blockers and calcium channel blockers. The inotropic negative properties of these drugs might explain the decreased bridge-induced systolic coronary compression. Nitrates should generally be avoided because they increase the angiographic degree of systolic narrowing and can lead to worsening of the symptoms. Surgical treatment by dissection of the overlying myocardium (myotomy) or with minimally invasive CABGs should be limited to patients with severe symptoms (intractable angina, recurrent MI) that persist despite medical treatment. A fixed stenosis either before or within the bridged segment may be another indication for revascularization (surgical or percutaneous). Coronary stent placement for MB is a promising technique; however, restenosis and other major periprocedural complications appear in 50% of the cases, including perforation of the artery.^[8]

Kilic et al.^[9] reported a series of 12 cases of transient MB of the LAD in acute MI amongst a population of 64 subjects. MB occurred only in the acute phase of inferior MI and not in the chronic phase. In the acute phase of inferior MI, compensatory hypercontraction of the anterior wall is assumed to occur in response to the decrease in the movement of the infarct-related walls. In the chronic phase, disappearance of the MB was observed due to the resolution of compensatory anterior wall hypercontraction,

as a result of the reperfusion of the infarct-related coronary artery. In contrast, we determined disappearance of the MB of the LAD after inferior MI in our patient.

In conclusion, disappearance of the MB of the LAD after inferior MI is very rare, and no such case has been reported before.

Conflict-of-interest issues regarding the authorship or article: None declared.

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

REFERENCES

1. Noble J, Bourassa MG, Petitclerc R, Dyrda I. Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction? *Am J Cardiol* 1976;37:993-9. [CrossRef](#)
2. Kramer JR, Kitazume H, Proudfit WL, Sones FM Jr. Clinical significance of isolated coronary bridges: benign and frequent condition involving the left anterior descending artery. *Am Heart J* 1982;103:283-8. [CrossRef](#)
3. Rossi L, Dander B, Nidasio GP, Arbustini E, Paris B, Vasanelli C, et al. Myocardial bridges and ischemic heart disease. *Eur Heart J* 1980;1:239-45.
4. Ferreira AG Jr, Trotter SE, König B Jr, Décourt LV, Fox K, Olsen EG. Myocardial bridges: morphological and functional aspects. *Br Heart J* 1991;66:364-7. [CrossRef](#)
5. Ge J, Jeremias A, Rupp A, Abels M, Baumgart D, Liu F, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. *Eur Heart J* 1999;20:1707-16. [CrossRef](#)
6. Morales AR, Romanelli R, Tate LG, Boucek RJ, de Marchena E. Intramural left anterior descending coronary artery: significance of the depth of the muscular tunnel. *Hum Pathol* 1993;24:693-701. [CrossRef](#)
7. Zeina AR, Shefer A, Sharif D, Rosenschein U, Barmeir E. Acute myocardial infarction in a young woman with normal coronary arteries and myocardial bridging. *Br J Radiol* 2008;81:e141-4. [CrossRef](#)
8. Berry JF, von Mering GO, Schmalfuss C, Hill JA, Kerensky RA. Systolic compression of the left anterior descending coronary artery: a case series, review of the literature, and therapeutic options including stenting. *Catheter Cardiovasc Interv* 2002;56:58-63. [CrossRef](#)
9. Kilic H, Akdemir R, Bicer A, Dogan M. Transient myocardial bridging of the left anterior descending coronary artery in acute inferior myocardial infarction. *Int J Cardiol* 2009;131:e112-4. [CrossRef](#)

Key words: Coronary disease; myocardial bridging; myocardial infarction.

Anahtar sözcükler: Koroner hastalık; miyokardiyal köprüleşme; miyokart enfarktüsü.