

Case Report

Fixed Coronary Artery Stenosis in Tunneled Coronary Artery Identified by Intravascular Ultrasound: A Case Report

Yuxiang Dai, Chenguang Li, and Junbo Ge

Shanghai Institute of Cardiovascular Diseases, Zhongshan Hospital, Fudan University, Shanghai, China

Corresponding Author: Junbo Ge; email: jbge@zs-hospital.sh.cn

Received 12 August 2014; Accepted 29 August 2014

Academic Editors: Marco Matteo Ciccone and Elia D. De Maria

Copyright © 2014 Yuxiang Dai et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract. Myocardial bridging is characterized as the compression of a segment of the coronary artery during systole. Significant atherosclerosis within the bridge is almost never seen at pathologic examination because of the absence of synthetic type smooth muscle cells in the intima of the tunneled artery. To date, there have been no reports of significant atherosclerosis at the site of myocardial bridging documented by angiography or intravenous ultrasound. We report a rare case of fixed coronary artery stenosis at the site of myocardial bridging identified by intravascular ultrasound.

Keywords: myocardial bridging, coronary artery stenosis, intravascular ultrasound imaging

1. Introduction

Myocardial bridging is characterized as the presence of strands of myocardial fibers overlying the coronary arteries, which induces compression of a segment of the coronary artery during systole [1–3]. This part of the vessel is called mural coronary artery or tunneled coronary artery, while the myocardial fiber overlying is called myocardial bridge [4, 5]. The incidence of myocardial bridging observed in angiography (1.5–16%) is far less than that observed in autopsies and noninvasive imaging studies using coronary computed tomography (40–80%) [3, 4, 6]. It is currently thought that the muscle bridge must be at least 20 mm in

length and 5 mm in depth to be significant and capable of compressing a coronary artery [4, 6]. Myocardial bridging is thought to be a normal anatomic variant of coronary artery and has been considered as a benign condition; however, severe constriction of a bridged coronary artery occurring in asystole may cause myocardial ischemia or sudden death [6–8].

Significant atherosclerosis within the bridge is almost never seen at pathologic examination because of the absence of synthetic type smooth muscle cells in the intima of the tunneled artery [9, 10]. To date, there have been no reports of significant atherosclerosis at the site of myocardial bridging documented by angiography or intravenous ultrasound. We

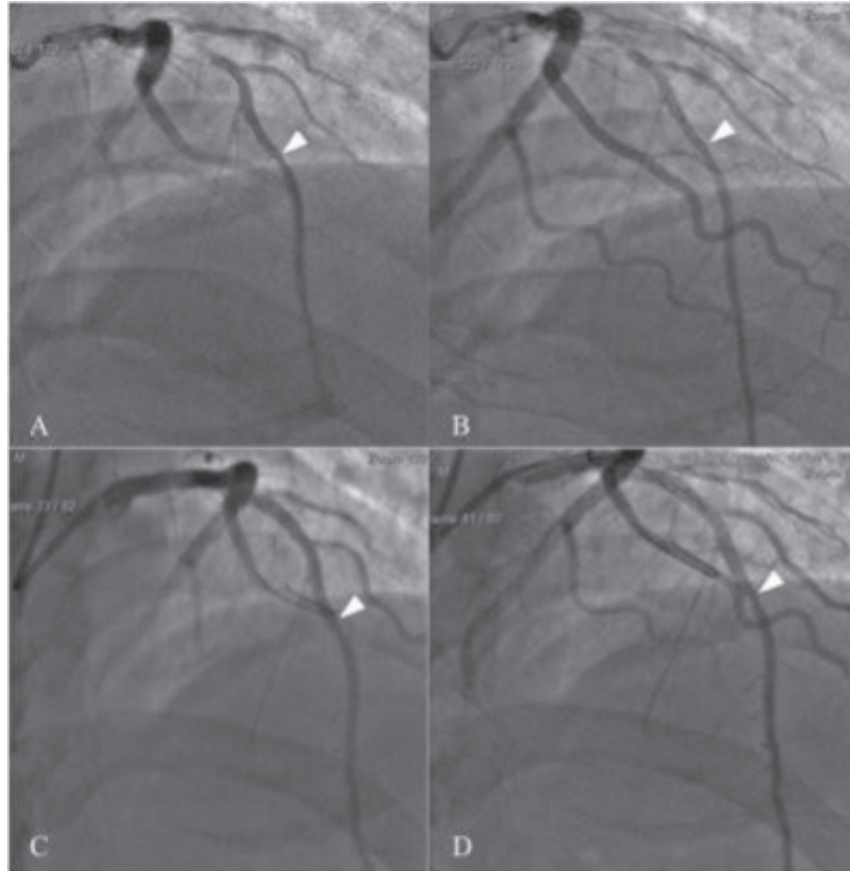


Figure 1: Coronary angiograms show dynamic compression (arrow) of the mid segment of the LAD during **A)** systole and **B)** diastole before stenting and **C)** systole and **D)** diastole after stenting.

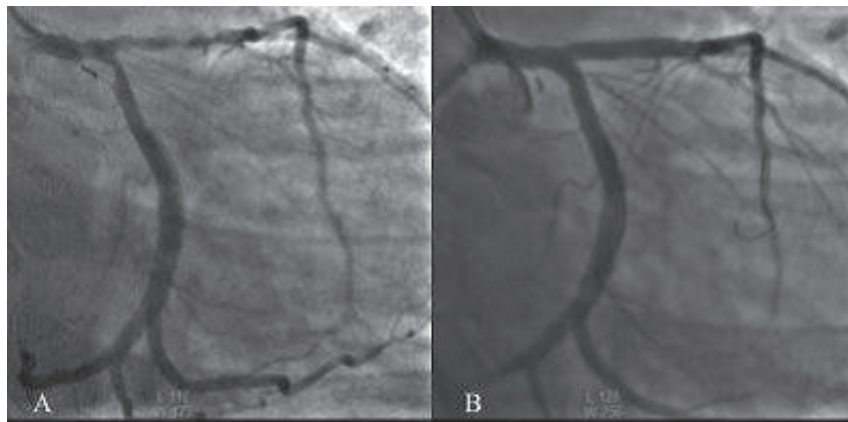


Figure 2: Coronary angiograms of left coronary **A)** before and **B)** after stenting.

report a rare case of fixed coronary artery stenosis at the site of myocardial bridging identified by intravascular ultrasound.

2. Case Report

A 36-year-old male, with a history of hypertension, dyslipidemia, and smoking, presented with a history of chest pain on

exertion class II of half a year duration. Electrocardiogram (ECG) at admission showed negative T waves in leads I-AVL and V5–V6. Transthoracic echocardiography revealed normal LV function. Baseline biochemistry and hematological tests were normal. Coronary angiography (Figure 1 and 2) was done which revealed significant double-vessel disease, with distal 50% stenosis in left main (LM), long lesion in proximal-mid left anterior descending (LAD) with

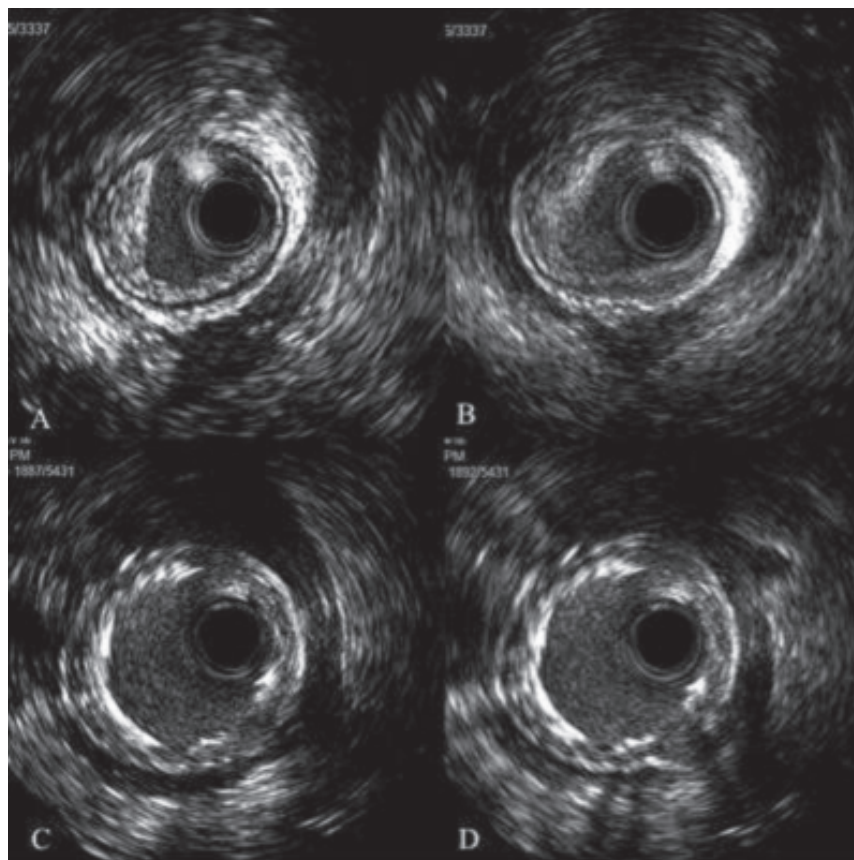


Figure 3: IVUS images of the atherosclerotic plaque informed at the site of myocardial bridging **A)** systole and **B)** diastole before stenting and **C)** systole and **D)** diastole after stenting. A “half-moon”-like area surrounding the tunneled segment is present during the entire cardiac cycle.

95% stenosis and a mid-muscle bridge causing 30% systolic compression, ostial 90% stenosis in the first diagonal branch (D1), proximal 80% stenosis in dominant left circumflex (LCX) and a normal, relative small right coronary artery (RCA).

Subsequently, we performed IVUS examination (Figure 3) to investigate the presence and the extent of coronary stenosis and myocardial bridging. IVUS examination demonstrated diffused plaque formed from mid LAD and LCX to LM, with plaque rupture phenomenon in LM, and a half-moon-shaped, echolucent area surrounding the bridge during the entire cardiac cycle in mid LAD, with atherosclerotic plaque formed in the lumen.

Double-vessel angioplasty for LM, LAD and LCX was planned and Cullote technique was used in the bifurcation lesion. The LAD and LCX stenosis were predilated and then a 3.0×28 mm and a 3.5×28mm DES (XIENCE V, everolimus eluting coronary stent, Abbott Vascular, Santa Clara, CA) were deployed at 12 and 14 atmospheres respectively from the tunneled segment in mid LAD to LM, a 4.0×28 mm DES (XIENCE V) was deployed at 14 atmospheres from proximal LCX to LM. Postdilation was performed at the ostia of the bifurcation.

3. Discussion

Myocardial bridges are most commonly localized in the middle segment of LAD [2, 3]. At present, the gold standard for diagnosing myocardial bridging remains to be coronary angiography with the typical milking phenomenon induced by systolic compression of the tunneled segment [11, 12]. However, intracoronary ultrasound test allows an accurate assessment of vascular anatomy and provide more morphological features [2, 6, 9, 13]. Sometimes, the milking effect may be un conspicuous, and in this situation, IVUS will help to detect the myocardial bridging and improve the prevalence. In our case, the compression of the tunneled segment before stenting was not appreciated angiographically maybe because of the severe stenosis in the proximal LAD, while the myocardial bridging was obviously identified in IVUS.

The occurrence of atheromatous changes in the tunneled coronary segment is still controversial. Traditionally, it was considered that atherosclerotic plaque rarely formed in the tunneled segment, which is supported by studies on a cellular level showing that in contrast to proximal and distal segments, foam cells and modified smooth muscle cells were missing in patients' tunneled segments [6, 10, 14].

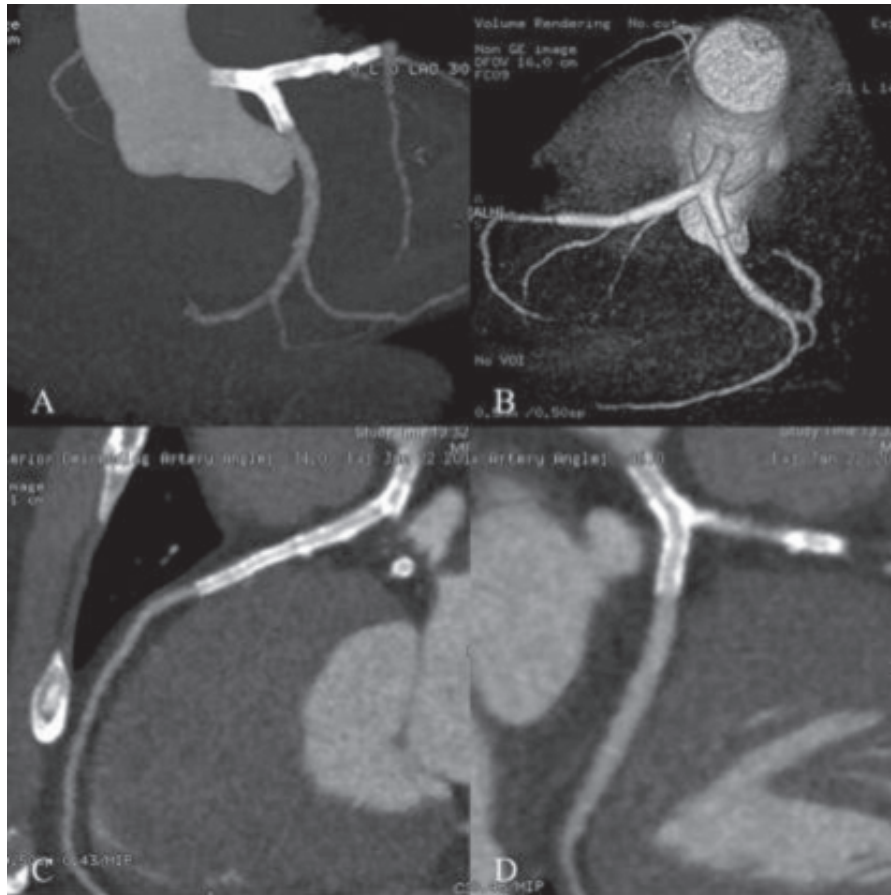


Figure 4: The follow up of coronary CTA showed no restenosis **A)** and **B)**, no late lumen loss in LAD **C)** and LCX **D)**.

Whereas, the segment proximal to the myocardial bridging frequently shows atherosclerotic plaque formation, because of hemodynamic forces, endothelial dysfunction, release of endothelial vasoactive agents, which is supported by autopsy and clinical observations [2, 4, 6, 14]. However, in our case, notable atherosclerotic plaque formation was observed in the tunneled segment. Some other authors also claimed that the atherosclerotic process occurred in the tunneled segment with the same severity and frequency as it did in the epicardial coronary segments [15, 16]. Maybe there lies

some undefined mechanisms and further studies on animal models are needed to make them clarified.

Currently, only symptomatic patients with myocardial bridging are intended to be treated. Therapeutic approaches that have been attempted for myocardial bridging include β -blockers [4, 6], calcium channel blockers [6, 9], stenting [17–19],

minimally invasive coronary artery bypass grafting (CABG) [20], and surgical myotomy [21]. Concern has been raised about stenting in tunneled segment because of two reasons below. First, high inflation pressures may be required for optimal stent implantation, with a higher risk of coronary perforation. Second, previous studies revealed high

restenosis rates, stent thrombosis and stent compression [17–19]. In our case, a decision was made to deploy a stent from the tunneled segment because the muscle bridging lies in the outflow region of the stent and the narrowing was significant, which is prone to lead to stent thrombosis.

One year later, the follow up of coronary computed tomography angiography (CTA) showed no late lumen loss or compression of the stent in the tunneled segment.

There's no restenosis observed in LM, LAD or LCX.

4. Conclusion

Generally speaking, stenting in tunneled segment is not recommended because of high risk of coronary perforation and restenosis. In this case, a stent was placed to eliminate severe stenosis in the segment of myocardial bridging. However, the safety of DES for treatment of the tunneled segment as in this case needs validation in further studies.

References

- [1] E. Geiringer, The mural coronary, *American Heart Journal*, **41**, no. 3, 359–368, (1951).

- [2] P. Angelini, J. A. Velasco, and S. Flamm, Coronary anomalies: Incidence, pathophysiology, and clinical relevance, *Circulation*, **105**, no. 20, 2449–2454, (2002).
- [3] P. Angelini, M. Trivellato, J. Donis, and R. D. Leachman, Myocardial bridges: A review, *Progress in Cardiovascular Diseases*, **26**, no. 1, 75–88, (1983).
- [4] A. G. Ferreira Jr., S. E. Trotter, B. Konig Jr., L. V. Decourt, K. Fox, and E. G. J. Olsen, Myocardial bridges: Morphological and functional aspects, *British Heart Journal*, **66**, no. 5, 364–367, (1991).
- [5] E. Konen, O. Goitein, and E. Di Segni, Myocardial Bridging, a Common Anatomical Variant Rather Than a Congenital Anomaly, *Seminars in Ultrasound, CT and MRI*, **29**, no. 3, 195–203, (2008).
- [6] J. R. Alegria, J. Herrmann, D. R. Holmes Jr., A. Lerman, and C. S. Rihal, Myocardial bridging, *European Heart Journal*, **26**, no. 12, 1159–1168, (2005).
- [7] S. Harikrishnan, K. R. S. Sunder, J. Tharakan, T. Titus, A. Bhat, S. Sivasankaran, and F. Bimal, Clinical and Angiographic Profile and Follow-up of Myocardial Bridges: A Study of 21 Cases, *Indian Heart Journal*, **51**, no. 5, 503–507, (1999).
- [8] M. Agirbasli, G. S. Martin, J. Bunker Stout, H. S. Jennings III, J. W. Lea IV, and J. H. Dixon Jr., Myocardial bridge as a cause of thrombus formation and myocardial infarction in a young athlete, *Clinical Cardiology*, **20**, no. 12, 1032–1036, (1997).
- [9] S. Möhlenkamp, W. Hort, J. Ge, and R. Erbel, Update on myocardial bridging, *Circulation*, **106**, no. 20, 2616–2622, (2002).
- [10] T. Ishii, N. Asuwa, and S. Masuda, et al., The effects of a myocardial bridge on coronary atherosclerosis and ischemia. *J Pathol.*, **185**, 4–9, (1998).
- [11] J. Noble, M. G. Bourassa, R. Petittlerc, and I. Dyrda, Myocardial bridging and milking effect of the left anterior descending coronary artery. Normal variant or obstruction? *American Journal of Cardiology*, **37**, no. 7, 993–999, (1976).
- [12] Y. Juillière, V. Berder, C. Suty-Selton, P. Buffet, N. Danchin, and F. Cherrie, Isolated myocardial bridges with angiographic milking of the left anterior descending coronary artery: A long-term follow-up study, *American Heart Journal*, **129**, no. 4, 663–665, (1995).
- [13] J. Ge, A. Jeremias, A. Rupp, M. Abels, D. Baumgart, F. Liu, M. Haude, G. Gorge, C. Von Birgelen, S. Sack, and R. Erbel, New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler, *European Heart Journal*, **20**, no. 23, 1707–1716, (1999).
- [14] T. Masuda, Y. Ishikawa, and Y. Akasaka, The effect of myocardial bridging of the coronary artery on vasoactive agents and atherosclerosis localization, **193**, 408–414, (2001).
- [15] V. De Melo Lima Jr., J. S. Cavalcanti, and T. Tashiro, Myocardial bridges and their relationship to the anterior interventricular branch of the left coronary artery, *Arquivos Brasileiros de Cardiologia*, **79**, no. 3, 219–222, (2002).
- [16] C. Burnside, J. C. Edwards, A. I. Lansing, and R. L. Swarm, Arteriosclerosis in the intramural and extramural portions of coronary arteries in the human heart., *Circulation*, **13**, no. 2, 235–241, (1956).
- [17] A. Bayés, V. Martí, and J. M. Augé, Coronary stenting for symptomatic myocardial bridging, *Heart*, **80**, no. 1, 102–103, (1998).
- [18] P. K. Haager, E. R. Schwarz, J. Vom Dahl, H. G. Klues, T. Reffelmann, and P. Hanrath, Long term angiographic and clinical follow up in patients with stent implantation for symptomatic myocardial bridging, *Heart*, **84**, no. 4, 403–408, (2000).
- [19] R. H. Stables, C. J. Knight, J. G. McNeill, and U. Sigwart, Coronary stenting in the management of myocardial ischaemia caused by muscle bridging, *British Heart Journal*, **74**, no. 1, 90–92, (1995).
- [20] J. W. Pratt, R. E. Michler, J. Pala, and D. A. Brown, Minimally invasive coronary artery bypass grafting for myocardial muscle bridging., *The heart surgery forum*, **2**, no. 3, 250–253, (1999).
- [21] Y. Atmaca, Ç. Özdöl, G. Pamir, M. Kilickap, and D. Oral, Successful surgical resection of a muscular bridge in a patient with nonobstructive hypertrophic cardiomyopathy - A case report, *Angiology*, **53**, no. 2, 225–227, (2002).