Myocardial Bridging

Shi-Min Yuan¹, MMed, PhD

Abstract
Myocardial bridging is rare. Myocardial bridges are most commonly localized in the middle segment of the left anterior descending coronary artery. The anatomic features of the bridges vary significantly. Alterations of the endothelial morphology and the vasoactive agents impact on the progression of atherosclerosis of myocardial bridging. Patients may present with chest pain, myocardial infarction, arrhythmia and even sudden death. Patients who respond poorly to the medical treatment with β-blockers warrant a surgical intervention. Myotomy is a preferred surgical procedure for the symptomatic patients. Coronary stent deployment has been in limited use due to the unsatisfactory long-term results.

Keywords: Atherosclerosis. Cardiac Surgical Procedures. Myocardial Bridging. Myocardial Infarction.

INTRODUCTION
Myocardial bridging is systolic compression of a tunneled coronary artery by the overlying myocardial tissue, which disappears completely during diastole¹. This coronary anomaly is usually a benign condition, but it can be associated with a series of serious cardiac events, such as myocardial infarction, arrhythmia and sudden death². Male predominance has been noted in large series of myocardial bridging patients³. The prevalence varies between 0.5% and 86% among different studies with a much higher rate at autopsy than in angiography⁴.

Myocardial bridges are most commonly localized in the middle segment of the left anterior descending coronary artery. Diagonal and marginal branches can be involved in 18% and 40% of cases, respectively. Myocardial bridging can be single or multiple. The multiple ones can occur in a same or different coronary artery or their branches⁵. Ferreira et al.⁶ divided the myocardial bridging into two types: superficial and deep muscle types. The former does not constrict the coronary flow during systole; whereas the latter may compress the coronary artery, reduce the flow and induce myocardial ischemia. Noble et al.⁷ categorized the systolic coronary narrowing into three classes: Class 1 (systolic coronary narrowing <50%), Class 2 (systolic coronary narrowing 50-75%) and Class 3 (systolic coronary narrowing >75%).

The anatomic features of the bridges vary significantly with a length of 2.3-42.8 mm, a thickness of 1.0-3.8 mm and an angle between long axis of muscle fibers and long axis of the crossed vessel of 5°-90°⁸. It was reported that the mean length of the bridges was 14.64±9.03 mm and the mean thickness was 1.23±1.32 mm⁹.

PATHOPHYSIOLOGY
The pathophysiology of myocardial bridging is insufficiently understood. Myocardial bridges are usually small and have no clinical significance. The segment proximal to the region of the myocardial bridging has been associated with atherosclerosis rather than the myocardial bridging segment itself⁴. Both hemodynamic and structural changes, such as blood flow disturbance, myocardial malperfusion, deposits of lipids and mucopolysaccharides and elastic damages, can be noted in the coronary artery segment proximal to a myocardial bridge. All these changes predispose to formation of atherosclerotic plaques in the intima of the coronary artery segment. Obviously, myocardial bridging is associated with degenerations of both myocardium and coronary artery⁶. The pathophysiological studies indicated significant impairment of coronary blood flow based on bridge obstruction as the underlying mechanism of sudden cardiac death in the patients with myocardial bridging⁷.
with heart valve disorder or cardiomyopathies, the patients’
of longitudinal, circumferential and radial strains[20]. Multiple-
echocardiography can detect subtle myocardial dysfunction
diastolic flow disturbance. Three-dimensional speckle-tracking
intravascular ultrasonography have documented a characteristic
mechanisms of atherosclerosis found in the proximal segment[13].
The mechanical stress caused by systolic narrowing at
the myocardial bridging segment may result in endothelial
damage, which, conversely, may induce platelet aggregation,
coronary vasospasm and eventually acute coronary syndrome[14].
Endothelial damage, vasospasm and atherosclerotic processes
developing in the proximal portion of the bridging segment are
alternative causes of ischemia. Hostiuc et al[15] have reported
significant myocardial fibrosis and interstitial edema in the
myocardial bridging segment in patients with later sudden death.

CLINICAL MANIFESTATION

The clinical manifestations of the patients with myocardial
bridging can appear in two ways: 1) by contraction of myocardial
bridge fibers and direct compression of the tunneled segment;
or 2) by stimulation and acceleration of atherosclerosis in the
segment proximal to the myocardial bridging[6]. Significant
myocardial bridging is often associated with total or subtotal
occlusion of the left anterior descending coronary artery during
systole. In patients with myocardial bridging, symptoms often
manifest during exercise and with tachycardia[2]. The patient
may manifest chest squeezing at rest[16]. In young patients with
myocardial bridging, they may have an acute anterior myocardial
infarction due to a subtotal occlusion of the mid-left anterior
descending coronary artery caused by myocardial bridging[6,17].
Symptomatic patients with myocardial bridging may present
with myocardial ischemia, acute coronary syndromes, coronary
spasm, exercise-induced dysrhythmias (such as supraventricular
tachycardia, ventricular tachycardia, or atrioventricular block),
myocardial stunning, transient ventricular dysfunction, syncope,
or even sudden death[18]. When myocardial bridging is associated
with heart valve disorder or cardiomyopathies, the patients’
symptoms can be different. Sustained elevated troponin levels
suggested the presence of myocardial ischemia[19].

Coronary angiography, intracoronary Doppler imaging and
intravascular ultrasonography have documented a characteristic
diastolic flow disturbance. Three-dimensional speckle-tracking
echocardiography can detect subtle myocardial dysfunction
in patients with myocardial bridging in terms of amplitudes
of longitudinal, circumferential and radial strains[20]. Multipleslice
computed tomography, stress single-photon emission
computed tomography and stress echocardiography are
helpful for the diagnosis of myocardial bridging. Multislice
spiral computed tomography defines bridges as segments
surrounded by myocardium and is more helpful in identifying
hemodynamically significant myocardial bridging. On
angiography, diagnosis depends on the change in diameter
between systole and diastole within the bridged coronary
segment. A significant “milking effect” is present when there
is ≥70% reduction in minimal luminal diameter during systole
and persistent ≥35% reduction in minimal luminal diameter
during mid-to-late diastole. Systolic narrowing at the bridge can
be accentuated by intracoronary injection of nitroglycerin by
vasodilating adjacent non-bridged coronary segments[16].

Metrological studies have revealed angiographically a systolic
diameter reduction of 80.6±9.2% and a persistent diastolic
reduction of 35.3±11% within the tunneled segment. Diastolic
flow velocities within the bridging segment were much higher
than those in the proximal and distal portions of the bridging
segment. Coronary flow reserve distal to the bridge was
2.5±0.5[21]. The tunneled artery is significantly thinner (66.3 µm)
than that of the proximal segment[22]. The intracoronary Doppler
revealed a lumen reduction during systole secondary to systolic
compression of the myocardial bridge was 36.4±8.8%[12].

MANAGEMENT AND PROGNOSIS

Pharmacological therapy

Because the patients with myocardial bridging are at
increased risk for atherosclerosis, antiplatelet therapy should be
considered. For symptomatic patients, ß-blockers remain the main
conservative treatment and they may relieve the patients from
hemodynamic impairment caused by the myocardial bridging by
decreasing the heart rate, increasing the diastolic coronary filling
and decreasing the contractility and compression of the coronary
arteries. Calcium channel blockers may have vasodilatory effects
beneficial for the concomitant vasospasm. It is advised that
vasodilating agents including nitroglycerin should be cautiously
prescribed for the patients with myocardial bridging. Nitrates may
exacerbate symptoms by intensifying systolic compression of the
bridged segment and vasodilating segments proximal to the
bridge, and therefore vasodilators should be avoided unless there
is significant coexisting coronary vasospasm.

Percutaneous coronary intervention

Stent implantation in symptomatic patients with myocardial
bridges may alleviate systolic coronary compression and
improves patients’ conditions; however, potential complications
of coronary artery and stent itself in relation to stent deployment
have limited its use. However, drug-eluting stents may be
preferable for the avoidance of future reintervention[23].

Surgery

Surgical intervention involves either supra-arterial myotomy
or coronary artery bypass. Coronary artery bypass is indicated for
the patients with extensive (>25 mm) or deep (>5 mm) myocardial
bridging or when the tunneled coronary segment is unlikely
to be decompressed completely in diastole[24]. The potential
complications of myotomy include wall perforation, ventricular
aneurysm formation and postoperative bleeding[25], while the
major concerns of coronary artery bypass in the patients with
myocardial bridge is lower freedom of angina and graft failure[26].
Rezayat et al.[24] performed surgical myotomy for myocardial
bridging in 26 patients and in one of the patients postoperative
residual narrowing of the left anterior descending coronary artery was noted as the only complication of the patient cohort. Zhu et al. reported their retrospective results of mini-incision myotomy for myocardial bridging of the left anterior descending coronary artery with a systolic narrowing extent of the bridging >60% in all 11 patients. Via a lower partial mid-sternotomy, 10 patients received surgical myotomy with one of them complicated with coronary artery impairment and off-pump coronary artery bypass was performed in this patient. Another patient had myotomy and concurrent repair of left anterior descending coronary artery-pulmonary artery fistula without pump. During the 2-51 month follow-up, one patient with myotomy having recurrent angina received medical treatment and the patient with coronary artery bypass also had recurrent angina and a coronary stent was deployed. Moreover, myotomy through heart-port access for myocardial bridging has also been reported.

CONCLUSION
Myocardial bridging is most often located in the left anterior descending coronary artery. It can be associated with a series of severe cardiovascular events, such as myocardial infarction, arrhythmia and sudden death. Symptomatic patients should be treated conservatively, interventionally or surgically depending on the patients’ conditions. Myotomy is a preferred surgical procedure for relieving the patients’ symptoms, improving the coronary flow and alleviating the coronary artery compression secondary to myocardial bridging.

Author’s roles & responsibilities

SMY Study conception and design; analysis and/or interpretation of data; manuscript writing, final approval of the manuscript

REFERENCES
1. Akdemir R, Gunduz H, Emiroglu Y, Uyan C. Myocardial bridging as a cause of acute myocardial infarction: a case report. BMC Cardiovasc Disord. 2002;2:15.
2. Dursun I, Bahcivan M, Durna K, Ibrahimov F, Erk NH, Yasar E, et al. Treatment strategies in myocardial bridging: a case report. Cardiovasc Revasc Med. 2006;7(3):195-8.
3. 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(5):364-7.
4. Lee MS, Chen CH. Myocardial bridging: an up-to-date review. J Invasive Cardiol. 2015;27(11):521-8.
5. Möhlenkamp S, Hort W, Ge J, Erbel R. Update on myocardial bridging. Circulation. 2002;106(20):2616-22.
6. Noble J, Bouvera MA, Petrie E, Rydyh I. Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction? Am J Cardiol. 1976;37(7):993-9.
7. Kosirski A, Grzybiak M. Myocardial bridges in the human heart: morphological aspects. Folia Morphol (Warsz). 2001;60(1):65-8.
8. Lujinović A, Kulenović A, Kapur E, Gojak R. Morphological aspects of myocardial bridges. Bosn J Basic Med Sci. 2013;13(4):212-7.
9. Ishii T, Asuwa N, Masuda S, Ishikawa Y. The effects of a myocardial bridge on coronary atherosclerosis and ischaemia. J Pathol. 1998;185(1):14-9.
10. Micic J, Nikolić S, Savić S. Sudden cardiac death caused by complicated atherosclerosis of the anterior intraventricular branch of the left coronary artery with a myocardial muscle bridge. Srp Arh Celok Lek. 2003;131(3-4):173-5.
11. Rozenberg VD, Nepomnyashchikh LM. Pathomorphology and pathogenic role of myocardial bridges in sudden cardiac death. Bull Exp Biol Med. 2004;138(1):87-92.
12. 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(23):1707-16.
13. Masuda T, Ishikawa Y, Akasaka Y, Itoh K, Kiguchi H, Ishii T. The effect of myocardial bridging of the coronary artery on vasoactive agents and atherosclerosis localization. J Pathol. 2001;193(3):408-14.
14. Alegria JR, Herrmann J, Holmes DR Jr, Lerman A, Rihal CS. Myocardial bridging. Eur Heart J. 2005;26(12):1159-68.
15. Hostiuc S, Curca GC, Demangiu D, Dervengiu S, Hostiuc M, Rusu MC. Morphological changes associated with hemodynamically significant myocardial bridges in sudden cardiac death. Thorac Cardiovasc Surg. 2011;59(7):393-8.
16. Teragawa H, Fujii Y, Ueda T, Murata D, Nomura S. Case of angiogenic pectoris at rest and during effort due to coronary spasm and myocardial bridging. World J Cardiol. 2015;7(6):367-72.
17. Tauth J, Sullebarger T. Myocardial infarction associated with myocardial bridging: case history and review of the literature. Cathet Cardiovasc Diagn. 1997;40(4):364-7.
18. Corban MT, Hung DY, Estehardi P, Rasoul-Arzrumly E, McDaniel M, Mekonnen G, et al. Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. J Am Coll Cardiol. 2014;63(22):2346-55.
19. Dupont M, Mullens W, De Bruyne B, Vanermen M. Myotomy through heart-port access for myocardial bridging. Int J Cardiol. 2008;124(1):e16-8.
20. Wang D, Sun JP, Lee AP, Ma GS, Yang XS, Yu CM, et al. Evaluation of left ventricular function by three-dimensional speckle-tracking echocardiography in patients with myocardial bridging of the left anterior descending coronary artery. J Am Soc Echocardiogr. 2015;28(6):674-82.
21. Klues HG, Schwartz ER, vom Dahl J, Reffelmann T, Reul H, Potthast K, et al. Disturbed intracoronary hemodynamics in myocardial bridging: early normalization by intracoronary stent placement. Circulation. 1997;96(9):2905-13.
22. Risse M, Weiler G. Coronary muscle bridge and its relations to local coronary sclerosis, regional myocardial ischemia and coronary spasm: a morphometric study. Z Kardiol. 1985;74(12):700-5.
23. Bockera LA, Sukhanov SG, Orekhova EN, Shatakhyan MP, Korotayev DA, Sternik L. Results of coronary artery bypass grafting in myocardial bridging of left anterior descending coronary artery. J Card Surg. 2013;28(3):218-21.
24. Rezayat P, Hassan D, Amirreza S, Susan H. Myocardial bridge. Surgical outcome and midterm follow up. Saudi Med J. 2006;27(10):1530-3.
25. Zhu E, Huang F, Wu Q, Zou Y, Zhang J. Clinical experience of minimally invasive supra-arterial myotomy for myocardial bridging. J Cardiovasc Pulmon Dis. 2014;33(4):548-50,572.