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REVIEW

Significance of the Anatomical Properties of a Myocardial Bridge in Coronary Heart Disease

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A myocardial bridge (MB), partially covering the coronary artery, is a congenital anatomical variant usually present in the left anterior descending coronary artery (LAD). MB causes coronary heart disease (CHD) by 2 distinct mechanisms influenced by the anatomical properties of the MB, such as its length, thickness, and location. One is direct MB compression of the LAD at cardiac systole, resulting in delayed arterial relaxation at diastole, reduced blood flow reserve, and decreased blood perfusion. The other is enhancement of coronary atherosclerosis causing stenosis of the LAD proximal to the MB, occurring because of endothelial injury arising from the abnormal hemodynamics provoked by retrograde blood flow up toward the left coronary ostium at cardiac systole. The magnitude of the effect of the 2 distinct mechanisms of the MB on LAD blood flow is prescribed by a common root of the MB-muscle mass volume generated by those properties. Furthermore, the anatomical properties of the MB are closely associated with the choice of treatment and therapeutic outcome in CHD patients having an MB. Thus, the anatomical properties of an MB should be considered in the diagnosis and management of CHD patients with this anomaly.

Key Words: Anatomy; Atherosclerosis; Coronary heart disease; Myocardial bridge

The myocardial bridge (MB) is an anatomical variant that often covers part of the left anterior descending coronary artery (LAD).[1] It occurs almost exclusively in the mid-portion of the LAD (**Figure 1**).[2,3] Muscular contraction of the MB itself alters blood flow within the LAD, as demonstrated by coronary angiography, and influences the distribution of hemodynamic stress.[3–5]

It is widely accepted that an MB sometimes causes coronary heart disease (CHD),[2,5,6] either from direct compression of the MB at cardiac systole or by enhancement of the natural progression of coronary atherosclerosis in the LAD segment proximal to the MB.[2,5,7] Both mechanisms are closely associated with changes in hemodynamic stress driven by the force of the MB contraction through a combination of anatomical properties, such as the location, length, and thickness of the MB.[3,7,8] This review focuses on the relationship between the presence of an MB in the LAD and the occurrence of CHD, specifically addressing the importance of the anatomical properties of the MB as the common root for the 2 distinct mechanisms. In addition, therapeutic approaches to CHD caused by an MB are summarized, along with their outcomes.

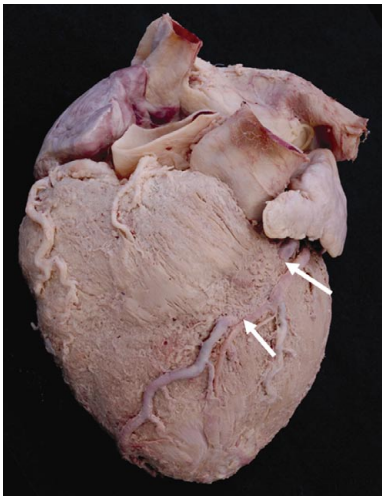

Figure 1. Myocardial bridge in the left anterior descending coronary artery. The epicardial adipose tissue was manually removed after formalin fixation. Arrows indicate the entrance and exit of the bridge.

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Table. Frequency and Anatomic Properties of the MB by Different Methods

First author	No. of cases	Artery	Frequency (%)	Location	Length (min.-max.)	Thickness
Angiography						
Noble J[9]	5,250	LAD	0.5	–	–	–
Ishimori T[10]	313	LAD	1.6	–	–	–
Greenspan M[11]	1,600	LAD	0.9	–	–	–
Rossi L[12]	1,146	LAD	4.5			
	37	LAD		–	(1–5 cm)	–
Kramer TR[13]	658	LAD	12.0	–		
	81	LAD			12.5±6.4 mm	–
Angelini P[2]	1,100	LAD	5.5	–	–	–
Channer KS[14]	1,102	LAD	1.4			
	16	LAD		M 13, D 3	1.5 cm (0.9–4.4)	–
Juilliere Y[15]	7,467	LAD	0.8	–	–	–
Hongo Y[16]	1,644	LAD	2.3	–	–	–
Sorajja P[17]	64	LAD	–	–	14.5±12.8 mm	–
Cay S[18]	25,982	LAD	11.8	–	–	–
Kim JW[19]	81	LAD	–	P 1, M 70, D 10	21.3±6.7 mm	–
Tsujita K[20]	331	LAD	3.0			
	11	LAD		–	16.0±11.4 mm	–
Schwarz ER[21]	157	LAD	–	–	22.6±7.8 mm	–
CT						
Kantarci M[22]	626	LAD	3.5			
	22	LAD		P 8.5%, M 66%, D 25.5%	17 mm (6–22)	2.5 mm (1.2–3.3)
Zeina AR[23]	300	LAD	23.6			
	78	LAD 71+Others 7		–	19.5±5.7 mm (8–30)	2.0±0.6 mm (1–3.1)
Kawawa Y[24]	148	LAD	14.2			
	24	LAD 21+Others 3		P 1, M 19, D 1	20.0±8.6 mm (105–50.2)	1.8±0.7 mm (1.1–3.7)
Lubarsky L[25]	245	LAD	44.0			
	108	LAD		P 8.5%, M 66.0%, D 25.5%	28.7±10.5 mm	–
Leschka S[26]	100	LAD	26.0			
	43	LAD 42+RCA 1		M 22, D 20	24.3±10.0 mm (8–50)	2.6±0.8 mm (1.4–4.8)
Kim PJ[27]	311	LAD	58.0	–	–	–
Bayrak F[28]	990	LAD	16.8			
	265	LAD 166+Others 99		–	14±7 mm	1.6±1.1 mm
Liu S[29]	450	LAD	34.0			
	192	LAD 153+Others 39		P 5, M 112, D 36	20.0±10.1 mm (9–46)	2.1±0.9 mm (0.9–4.8)
Lazoura O[30]	875	LAD	21.0		–	–
	184	LAD		P 125, M 53, D 6	20.9 mm (8–32)	2.6 mm (1.2–4.8)
Kim SY[31]	607	LAD	5.3			
	39	LAD 32+Others 7		P 5, M 20, D 7	16.34±6.26 mm (6.9–30)	1.84±0.77 mm (0.5–3.9)
Autopsy						
Geiringer E[32]	100	LAD	23.0	–	–	–
Edwards JC[33]	276	LAD	4.7			
	13	LAD		P 4, M 7, D 2	(0.5–10.1 cm)	(0.15–0.6 cm)
Polacek P[34]	70	LAD	60.0			
	42	LAD		–	(3–69 mm)	–
Lee SS[35]	108	LAD	58.0			
	63	LAD		4 cm	15 mm	–
Stolte M[36]	711	LAD	22.9			
	163	LAD		33.6 mm	22.5 mm	2.8 mm
Ishii T[37]	642	LAD	45.0			
	50	LAD		–	19.7±1.5 mm	–
Bezerra AJC[38]	50	LAD	52.0	–	–	–
Baptista CAC[39]	82	LAD	35.4			
	29	LAD		–	21.5 mm (5.1–41.8)	–
Ishikawa Y[8]	200	LAD	–	4.39±1.18 cm (1.5–10.0)	1.44±0.95 cm (0.5–5.0)	856.5±676.2 μm (131–4,940)
Loukas M[40]	35	LAD	–	–	32 mm	12 mm
Ishikawa Y[7]	100	LAD with MI	46.0			
	46	LAD with MI		4.8±1.2 cm	1.70±1.15 cm	1,005±703 μm
	100	LAD without MI		4.57±1.3 cm	1.39±0.83 cm	797±526 μm
Cakmak Y[41]	39	All, fetus	38.5	–	–	–

MB, myocardial bridge; min., minimum; max., maximum; LAD, left anterior descending coronary artery; P, proximal; M, middle; D, distal; CT, computed tomography; RCA, right coronary artery; MI, myocardial infarction.
The location of MB indicates the site in the LAD or the distance from the left coronary ostium.

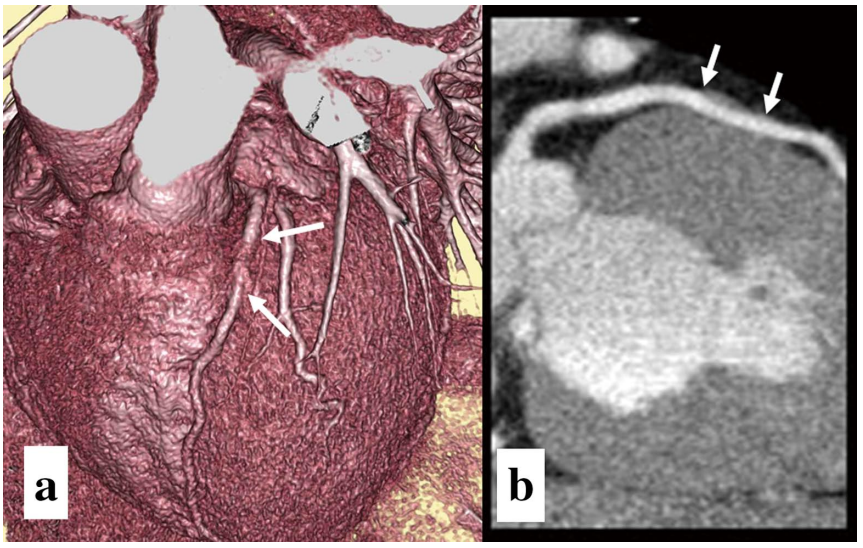

Figure 2. Images of a myocardial bridge (MB) in a 49-year-old man with exertional angina by 16-row multidetector computed tomography. (**a**) Three-dimensional volume-rendered image in which the MB between the 2 white arrows covers the upper middle segment of the left anterior descending coronary artery. (**b**) Curved planar reformat image of the same case as (**a**), in which the MB is 14.5 mm long and 2.2 mm-thick (arrows indicate the entrance and exit of the MB).

Improvement in Detecting an MB in the LAD

Myocardial bridging in the LAD has been detected by angiography, multidetector computed tomography (MDCT), and autopsy, as shown in the Table.[2,7–41] It is more frequently detected by MDCT (3.5–58%) than by angiography (0.4–15.8%). This discrepancy may have resulted from these different methods of MB detection; the increased detection rate is due to the better resolution of MDCT and the direct depiction of MB muscle on imaging; an MB itself is not directly detectable on angiography in which it can be detected only by the milking or squeezing of the artery. In addition, the presence of an MB may often be missed on coronary angiography in cases of a severely stenotic lesion in the coronary segment proximal to the MB, because limited coronary flow to the distal artery masks the typical milking effect.[5] Considering that MB frequency is around 50% by autopsy, the recent introduction of MDCT has remarkably improved the detection rate. The higher frequency of MB detection at autopsy than with MDCT may be due to differences between these methods, because an MB thinner than 200 μm can only be detected by autopsy.

Furthermore, MBs have already been recognized in the LAD of a neonate,[42] an infant suffering a myocardial infarction (MI),[43] and autopsied fetal hearts,[41] indicating that the MB is a chance congenital anatomical structure.

CHD Caused by the MB

The MB has long been considered benign based on angiographic findings, and patients with an MB in the LAD are given a good long-term prognosis.[15] However, many case studies indicate a close association between the presence of an MB and CHD,[2,3,5,32,42] deleterious arrhythmia,[3,42] sudden cardiac death,[43–45] and takotsubo cardiomyopathy.[46,47] Symptomatic cases of MB have been repeatedly described; 183 cases without significant atherosclerosis have been reported from 1968 to 2008.[48] Such cases of CHD exhibit angina, myocardial ischemia, and MI, but most patients have no significant coronary lesion except for the MB around the coronary artery, indicating that the CHD in these cases is induced directly by MB compression of the coronary artery at cardiac systole, irrespective of the presence of coronary atherosclerosis.[48]

Sudden cardiac death related to the presence of MB is also well known, especially in young patients or young athletes. MB in the LAD was detected in 6 of 16 cases (37.5%) of sudden cardiac death in subjects under 35 years of age.[45] In addition, the rate of detecting an MB was 39.3% among 300 individual who died from CHD, and the majority of these (61.0%) were sudden cardiac death.[49] In sports medicine, MB is one of the main causes of sudden cardiac death among young competitive athletes.[50,51] These reports indicate that an MB may cause life-threatening events, even in youth, through vigorous compression of the coronary artery during cardiac systole.

General Anatomical Properties of the MB

The anatomical properties of the MB, such as its location, length, and thickness, have been explored by various methods. On imaging analyses such as coronary angiography and MDCT, an MB is usually found in the middle segment of the LAD (Table). The distance of the MB's entrance from the left coronary ostium has not generally been measured by imaging analyses, except for one study, which reported it as 19.1±10.6 mm by coronary angiography and 30.4±14.0 mm by intravascular ultrasound.[20] The MB length is approximately 1.5–2.5 cm by imaging analyses and autopsy studies, with an average of less than 2 cm in most studies. MDCT imaging techniques permit measurement of the MB's thickness (Figure 2); the average in CHD patients has been often described after the original description by Kantarci et al.[22] Lengths measured on MDCT vary from 0.5 to 3.6 cm (Table), and are similar to those measured at autopsy (0.8–2.8 cm).

It should be mentioned that these anatomical properties are interrelated. Longer MBs tend to be located more proximally in the LAD, and thicker MBs are located at a significantly more proximal position.[37] In addition, MB length usually correlates with MB thickness at autopsy[8,37] and on MDCT.[31]

Two Distinct Mechanisms for CHD by the MB

The occurrence of CHD in patients with an MB is caused by 2 distinct mechanisms: direct compression of the LAD by MB

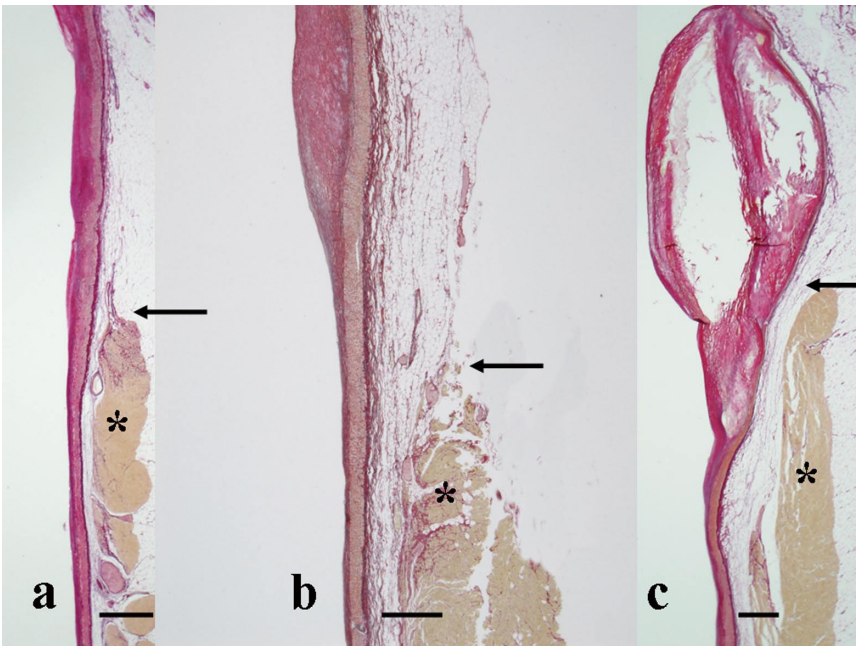

Figure 3. Longitudinal sections of the left anterior descending coronary artery (LAD) with a myocardial bridge (MB) in the normal heart (elastica van Gieson). In each part of the Figure: the lower side is peripheral; (*) MB; arrows show the entrance of the MB; scale bar= 1mm. (**a**) 45-year-old male, (**b**) 62-year-old male, (**c**) 84-year-old male. (**a**, **b**) LAD segment just under the MB shows only mild intimal thickening. (**c**) Atheroma lesion in the LAD segment proximal to the MB, but the intimal lesion development is remarkably suppressed under the MB. (Atheroma core melted out during the process of producing the microscopic slides.)

contraction,[5] and enhancement of the natural history of coronary atherosclerosis in the LAD segment proximal to the MB.[7] The former causes CHD mainly in young patients, and the latter seems to contribute to CHD occurrence in elderly patients. These 2 distinct mechanisms for CHD originate from a common origin based on the anatomical properties of MB.

Direct Compression of the LAD by the MB

Most reported cases of CHD due to the presence of MB exhibit no atherosclerotic lesion in the associated LAD.[48] In such symptomatic MB cases, the occurrence of CHD is considered to be caused by direct MB compression of the LAD.[5] The compression of the LAD by MB contraction causes retrograde blood flow at systole, which leads to increased blood flow velocity at early diastole and delayed diastolic relaxation at the bridged segment.[52] Such abnormal hemodynamics reduce the blood flow reserve for peripheral perfusion and lead to myocardial ischemia.[52,53] This phenomenon in combination with anginal symptoms is further exacerbated by tachycardia[54] as a result of the significantly shortened diastolic period.[6] Tachycardia-induced reduction of coronary blood flow during exercise may cause not only myocardial ischemia, but also sudden cardiac death for young athletes with an MB in the LAD without atherosclerosis.[45,50] In addition, coronary constriction due to vasospasm and subsequent thrombosis at the bridged segment can result in myocardial ischemia and/or infarction.[55,56] Endothelium-dependent vasodilators, such as acetylcholine, enhance the vasoconstriction of the bridged segment, indicating endothelial dysfunction beneath the MB.[5] This endothelial dysfunction stimulates coronary vasospasm and transient platelet aggregation, leading to thrombosis in the segment proximal to or under the MB.[5]

Angiography reveals that patients exhibiting more than 75% narrowing of the LAD from MB compression during cardiac systole experience severe myocardial ischemia.[9] The magnitude of the compressive force exerted by the MB on the coronary artery is closely related to the contractile force of the MB muscle, which is distinctly associated with the anatomical properties of the MB, namely, its length,[20] thickness,[29,57] and location.[58] In fact, coronary angiography combined with IVUS reveals that a longer MB has a significant association with more severe systolic compression,[20] and MDCT shows that the thickness of MB correlates with the severity of systolic compression.[29,57] In hypertrophic hearts, a long MB also leads to significant LAD compression,[14] and a deeply situated intramyocardial LAD with a thick MB is associated with sudden cardiac death.[44] Furthermore, the location of the MB in patients with MI[37,59,60] and symptomatic coronary insufficiency[58] tends to be proximal toward the coronary ostium. These reports indicate the close relationship between the anatomical properties of the MB and the occurrence of CHD. The anatomical properties of the MB therefore have a decisive role on the extent of the abnormal hemodynamics in the LAD.

Alteration of Atherosclerotic Distribution by MB Causing CHD

The presence of an MB in the LAD contributes to altered distribution of atherosclerosis within the entire course of the LAD.[2,3,8] Development of atherosclerotic lesions is suppressed in the intima just under the MB (**Figure 3**), but the LAD segment proximal to the MB is vulnerable to atherosclerosis.[2,4,5,8,37] These changes in the atherosclerotic distribution by presence of an MB have been widely recognized in autopsy,[32,37] angiographic,[20] and MDCT studies.[24,28] LAD compression by the MB during cardiac systole results in an altered hemodynamic force of blood flow, leading to an altered distribution of the atherosclerotic lesions.[3,5] Scanning electron microscopy reveals changes in the shape of the endothelial cells in human LAD intima from flat and polygonal in the segment proximal to the MB to spindle-shaped and engorged under the MB.[4] The intima beneath the MB is hemodynamically regulated by high shear stress, indicating its lack of susceptibility to atherosclerosis, whereas the intima of the segment proximal to the MB is subject to lower shear stress, indicating a susceptibility to disease. This difference

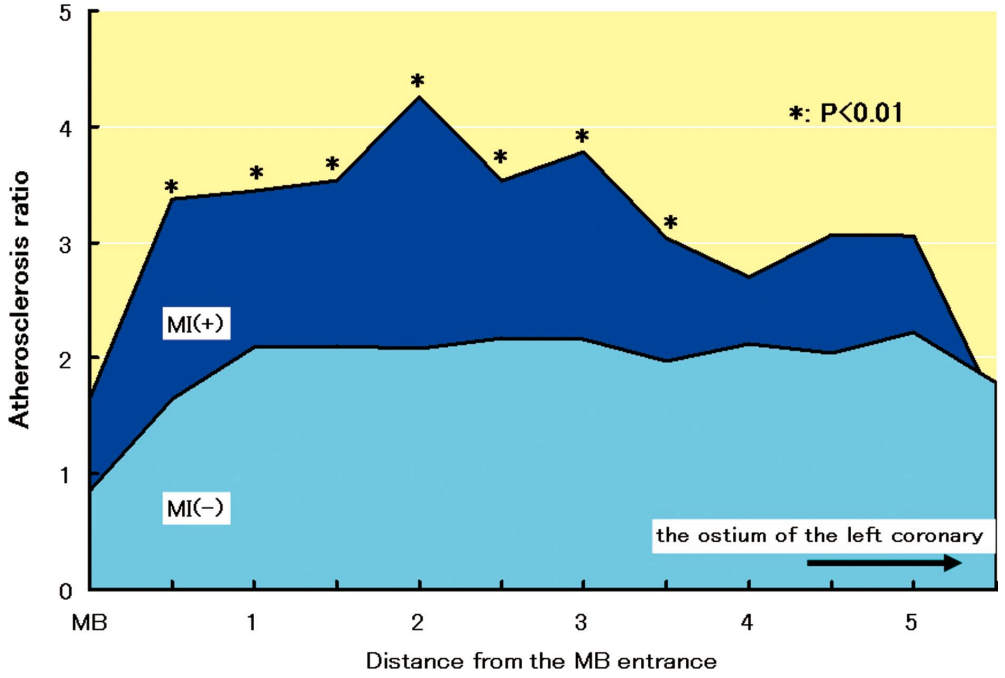

Figure 4. Changes in atherosclerosis ratio (the ratio of intimal area: medial area in the cross-section of the left anterior descending coronary artery) at 5-mm intervals from the entrance to the myocardial bridge (MB) up toward the left coronary ostium in cases of myocardial infarction [MI(+); n=36] and no infarction [MI(–); n=100]. In the MI(+) group, the atherosclerosis ratio increases from the MB entrance up to 2.0 cm and tends to decrease up toward the ostium. In contrast, the atherosclerosis ratio in the MB(–) group shows no significant fluctuation from 1.0 cm up toward the ostium. *Significant difference (P<0.01) between 2 groups.

in endothelial shape between the intima proximal to the MB and that under the myocardial covering is also confirmed in the cholesterol-fed rabbit model.[61] Such hemodynamic alteration caused by MB muscle contraction causes a decrease in the expression of vasoactive proteins, such as endothelial nitric oxide synthase, endothelin-1, and angiotensin-converting enzyme, in the endothelial cells of the LAD under the MB, leading to suppression of atherosclerosis under the MB.[62] In addition, pressure measurement with a catheter pressure transduction system shows that blood pressure in the LAD segment proximal to the MB is higher than aortic pressure.[63] This indicates that the disturbance of intracoronary blood flow and high wall stress proximal to the MB may also enhance the natural history of coronary atherosclerosis by intimal injury.

Such atherosclerotic evolution in the LAD intima proximal to the MB is basically regulated by the anatomical properties of the MB.[3] The more proximal the MB is located to the left coronary ostium, the more turbulent the blood flow becomes in the LAD segment proximal to the MB, thus resulting in the formation of severe atherosclerotic lesions in the segment proximal to the MB.[37] A long and/or thick MB suppresses atherosclerotic evolution in the bridged segment to a greater degree than a short and/or thin MB,[8] and the extent of intimal lesion in the LAD segment proximal to the MB is associated with the thickness and length of the MB.[23] In addition, histopathological analysis of the LAD in autopsied hearts with MI reveals that the presence of an MB with a larger muscle index (multiplication of MB thickness by MB length) is significantly associated with the occurrence of MI through the progression of stenotic atherosclerosis in the LAD proximal to the MB.[7,64] In cases of MI, the location of the LAD segment with the most stenotic lesion in the segments proximal to the MB also significantly correlates with the location of the MB's entrance, and atherosclerotic progression in the LAD proximal to the MB meets the proximal intima 2.0 cm from the MB entrance (**Figure 4**).[7] In addition, the MB muscle index expressed by the 2 anatomical properties has an independent role in shifting the preferential site of atherosclerosis more proximally toward the coronary ostium, leading to MI.[7] The anatomical properties of the MB muscle are important determinants for development of arterial stenotic lesions in the segment proximal to the MB.

Treatment for CHD Caused by the MB

A survey of the literature on therapeutic strategies for patients with symptomatic MB indicates 3 options: medication, stent placement in the bridged segment, and surgical treatment. Coronary angiography shows that patients with ischemic symptoms generally demonstrate systolic narrowing >70–75% at the bridged segment at rest without nitrate administration[9] and/or hemodynamic abnormality,[21] and their abnormal blood flow should be improved by appropriate treatment. The anatomical properties of the MB also influence the method of treatment and the patient's outcome.[65]

Medications

Beta-blocker therapy is the first choice of treatment for symptomatic patients.[66] The β-blocker reduces the arterial compression induced by the MB and lowers the heart rate. It

also increases the diameter of the bridged arterial segment, resulting in a prolonged diastolic period and normalization of the maximal flow velocity within the bridged segment.[67,68] The negative inotropic effect of β-blockers increases coronary perfusion and alleviates symptoms evoked by the MB. However, a calcium-channel blocker is effective for patients with a contraindication to β-blocker therapy or coronary vasospasm.[69] Nitrate is generally useful to relieve anginal symptoms due to coronary vasospasm but also enhances the coronary compression by the MB at cardiac systole resulting in augmentation of angiographic squeezing effect[16] and accentuation of the luminal narrowing at the bridged segment.[9]

Stent Placement

Since the first trial,[70] stent placement has been performed in the bridged segment to resolve the extra-coronary compression by the MB. Intracoronary stent implantation under the MB is used mainly in patients with severe systolic stenosis, complete occlusion at the bridged segment, or resistance to drug therapies. This procedure directly reduces the coronary compression by the MB, which increases the cross-sectional area of the bridged segment and coronary flow reserve by improvement of the intracoronary flow abnormalities.[53,71] The mid-term prognosis of patients within 2–5 years after stenting under the MB is relatively favorable.[21,53,71]

Nevertheless, some complications have been reported during or after stent implantation, such as coronary perforation/rupture immediately after stenting,[56] stent fracture,[72] and in-stent restenosis.[71,73,74] Coronary perforation is caused by an oversized stent,[56] because the coronary artery under the MB generally has a thin arterial wall and a smaller lumen diameter than the coronary segments proximal and distal to the MB.[5] Stent fracture under the MB probably occurs due to mechanical compression by the MB contraction during systole, which may lead to in-stent restenosis and/or in-stent thrombosis.[72] In-stent restenosis under the MB occurs more frequently than in cases with no MB.[74] Although the coronary intima beneath the MB is usually free from atherosclerosis, stent implantation causes mechanical stretch and an intimal/medial injury at the bridged segment, leading to the development of in-stent neointima.[71]

Drug-eluting stent (DES) implantation in the bridged segment has also been attempted for alleviation of in-stent restenosis.[75,76] Compared with bare metal stents, the use of DES in atherosclerotic cases has contributed to a reduction of the in-stent restenosis rate.[77] Short-term follow-up suggests that a DES is a better choice in MB cases.[75] However, there is a report that suggests the use of DES in symptomatic MB patients does not further improve outcome, and that drug therapy remains the mainstay for the treatment of such cases.[76]

To avoid complications from stent placement, it is necessary to choose a suitable stent with a desirable radial strength against the compressive force of the MB, and/or with an appropriate length that completely covers the coronary segment under the MB.[71] In turn, considering the effects of the anatomic properties of the MB in regulating the intracoronary compressive force across it, the development of an effective stent for the bridged LAD is required.

On the other hand, the significance of stent placement to fixed lesions in the LAD segment proximal to the MB should be noted, because the presence of an MB is closely associated with atherosclerotic progression related to CHD.[7] In cases of stenting for a fixed-lesion site proximal to the MB, part of the stent sometimes extends into the bridged segment beyond the obstructive lesion site.[78] Such inadvertent stent placement increases the incidence of late events, mainly within the site of the stent extending into the bridged segment.[78] Further follow-up studies on the relationship between in-stent restenosis and the anatomic properties of the MB are required for patients with stenting at a fixed-lesion site proximal to the MB.

Surgical Treatment

Since 1975, supraarterial myotomy of the MB has also been attempted in patients with repeated symptoms.[79] Myotomy produces a complete relief of coronary compression and improves peripheral blood perfusion, leading to resolution of ischemic reaction.[80] The mid- or long-term outcome of symptomatic patients with surgical relief from MB compression is excellent.[81] Myotomy is also effective for symptomatic patients with a long MB who are unsuitable for stent placement in the bridged segment.[82,83] Furthermore, this technique is favorable for pediatric patients with MB and hypertrophic cardiomyopathy who may suffer from myocardial ischemia due to augmented coronary compression by the MB.[82,84] In young patients with a symptomatic MB, a life-long commitment to medical management is required,[85] and myotomy may contributes to durable normalization of the anatomic environment around the coronary artery.

Surgical procedures may, however, result in some complications, such as a perforation of the right ventricle and coronary injury.[80,81,86] When the LAD is situated deep within the anterior ventricular groove due to a thick MB, the right ventricle may be accidentally perforated during myotomy.[80] For cases of a long and/or thick MB, the coronary artery may be injured during decompressive myotomy,[86] because it is not visible through the epicardial adipose tissue and is thin-walled under the bridged segment.[5] In addition, there is a possibility of postoperative scar formation around the coronary artery, which causes recurrent compression of the coronary artery.[86] Hence, Li et al suggest that surgery be regarded as a limited treatment for patients with an isolated MB of less than 2.5 cm in length and 0.5 cm thickness.[87] Coronary artery bypass grafting rather than myotomy may be better for symptomatic MB patients with not only a thick MB (deeply embedded coronary artery)[88] but also atherosclerotic lesions in the coronary segments proximal to the MB or within the bridged segment.[89] Thus, the surgical application should be carefully decided after consideration of the anatomical properties of the MB and the presence of atherosclerosis in the affected coronary artery.

Conclusions

The anatomical properties of the MB contribute not only to the occurrence of CHD but also to the therapeutic strategies. The actual quantitative values of the anatomical properties of the MB predisposing to CHD or those influencing treatment have so far remained unclear. However, cardiologists as well as pathologists should be aware of the association between the anatomical properties of the MB and the 2 distinct mechanisms leading to the occurrence of CHD in the management of patients with CHD who have an MB.

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