► **Special Article** ◀ *Series: Vascular Pathology*

(1) Coronary Events Caused by Myocardial Bridge

Yukio Ishikawa, MD,¹ Yoko Kawawa, MD,² Ehiichi Kohda, MD,² and Toshiharu Ishii, MD¹

Myocardial bridge (MB), which covers a part of the left anterior descending coronary artery (LAD), is a normal anatomical variant structure (45% in frequency by autopsy) in LAD. MB contraction plays the role of a “double-edged sword” on the coronary events, suppressing coronary atherosclerosis under the MB, yet generating abnormal blood flow associated with coronary heart diseases (CHDs). High shear stress driven by MB compression causes the suppression of vascular permeability and vasoactive protein expression such as e-NOS and endothelin-1, which leads to the suppression of atherosclerosis in the LAD segment under the MB. However, despite the prevalent view of MB as benignancy by conventional coronary angiography (5–6% in frequency), with advance of imaging technique such as multislice spiral computed tomography [(MSCT); 16% in frequency], cardiologists are now frequently aware of symptomatic MB occurring not only in hospitalized patients, but also in young athletes free from atherosclerosis. Moreover, the large mass volume of MB muscle induces atherosclerosis evolution at the settled site in LAD proximal to MB and contributes to the occurrence of myocardial infarction.

These events upon the coronary events result from the different pathophysiological mechanisms induced by contractile force of MB, which is solely determined just by the integration of anatomical properties of MB, such as the location, length and thickness of MB in an individual LAD. A recent MSCT provides the objective quantification of the anatomical variables that correlate with the histopathological results in relation to the occurrence of CHD. In this review, we therefore discuss the necessity to explore MB as an inherent chance anatomical risk factor for CHD.

Key words: myocardial bridge, coronary artery, anatomy, atherosclerosis, myocardial infarction

INTRODUCTION

The myocardial bridge (MB) is a normal anatomical variant under which a part of the major coronary arteries usually running in the subepicardial adipose tissue courses intramurally.^{1,2)} It is almost exclusively situated in the mid-portion of left anterior descending coronary ar-

tery (LAD).^{3,4)} Coronary angiography has demonstrated a suppressive effect on coronary atherosclerosis by MB as early as 1960,⁵⁾ whereas intimal lesion progresses in the arterial segment proximal to MB.⁶⁾ However, the relevance of MB to clinical symptoms has been so far controversial. Although the MB presence has long been considered benign for coronary heart diseases (CHDs)^{7,8)} including angina, myocardial ischemia, myocardial infarction and sudden cardiac death, the recent techniques such as intravascular ultrasound (IVUS),⁹⁾ intracoronary doppler (ICD)¹⁰⁾ and multislice spiral computed tomography (MSCT)¹¹⁾ have led to an increase of case reports on symptomatic MB. Several studies have also documented the association of MB with CHDs.^{3,6,12,13)} Moreover, our recent autopsy study elucidated the role of MB on the occurrence of myocardial infarction through histomorphometry for LAD having MB in hearts with myocardial

¹Department of Pathology, Toho University School of Medicine, Tokyo, Japan

²Department of Radiology, Toho University Medical Center, Ohashi Hospital, Tokyo, Japan

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Address for reprint requests to Yukio Ishikawa, MD: Department of Pathology, Toho University School of Medicine, 5-21-16 Omori-nishi, Ota-ku, Tokyo 143-8540, Japan
Tel: +81-3-3762-4151, Fax: +81-3-5493-5414
E-mail: yukio@med.toho-u.ac.jp

Fig. 1 Macroscopic view of a heart after removing the epicardial adipose tissue. There is a myocardial bridge (MB) in the middle segment of the left anterior descending coronary artery (LAD) between the two arrows.

infarction.¹⁴⁾ This review focuses on the pathophysiology in conjunction with changes of extent and localization of atherosclerosis in the LAD caused by MB compression. We also discuss the role of anatomical properties of MB on the occurrence of CHD.

DETECTION OF MB

Autopsy

While the MB occurs almost exclusively in LAD (**Fig. 1**),^{2, 7, 15-17)} it is rarely detected in the right coronary and left circumflex arteries.^{3, 18, 19)} The frequency of MB in LAD has been reported to be 22.2-60.0% in Western countries,^{2, 7, 16, 20)} 52-55.6% in South America,^{18, 21)} and 45-58.3% in the Orient,^{16, 17)} which indicates no significant difference by ethnic group. In addition, no significant difference has been found by age and gender.^{7, 17, 18)} MB is found in the LAD of both a neonate²²⁾ and an infant suffering myocardial infarction.²³⁾ MB is thus considered to be a chance inherent anatomical structure.¹⁵⁾

Coronary Artery Imaging

The clinical diagnosis of MB has been formerly made by coronary angiographic findings of “milking effect” and “step down-step up” phenomena provoked by myocardial contraction of MB itself for the coronary artery during

cardiac systole.^{5, 6, 24)} The frequency of MB in the LAD as demonstrated by angiography is 0.4-5.4%^{8, 25-30)} which is obviously lower than that by autopsy studies.^{2, 7, 15-18, 20, 21)} This discrepancy may be resulted from the nature of these different methods for MB detection. The angiographic results are obtained from the patients under evaluation for suspected CHD, but MB may be underdiagnosed in patients with an LAD demonstrating little systolic compression.³¹⁾ In fact, the frequency of MB detection by angiography increases, when the examiner reviews the same angiographic material with special attention to the possible presence of a bridge.³²⁾

Utilizing recently developed imaging technique, such as IVUS or MSCT, it is possible to obtain highly accurate images including the arterial wall structure and surrounding muscle bridge.^{9-11, 31, 33)} Non-invasive MSCT technique more clearly demonstrates the coronary artery and muscle bridge structure than do angiographic or IVUS examinations (**Fig. 2**).^{11, 33, 34)} A 16-row MSCT technique for patients with CHD reveals MB frequencies in the LAD to be 3.5% in Turkey,¹¹⁾ 15.8% in Japan,³³⁾ and 26% in Israel,³⁵⁾ still lower than that found at autopsy. This discrepancy results from the limited resolution power of MSCT to detect muscle bridge less than 1mm in thickness.³³⁾ Nonetheless, the frequency (15.8%) of MB over 1mm in thickness for the Japanese by MSCT closely approximates the level by autopsy study in Japan (13.3%).³³⁾ The recent development of a 64-row MSCT demonstrates higher frequency of MB detection in the coronaries, such as 32% in USA³⁶⁾ and 58% in Korea.³⁷⁾ The improvement of resolution power of MSCT may be much effective for detection of bridged muscle.³⁷⁾

ATHEROSCLEROSIS SUPPRESSION BY MB

Pathology

Atherosclerosis evolution is significantly suppressed in the LAD segment under the MB regardless of age, whereas the segment proximal to the bridge is vulnerable (**Fig. 3**).^{2, 4, 6, 15, 17, 38, 39)} This effects due to the existence of the MB result from altered hemodynamic force in the segment under the MB.⁴⁾ Scanning electron microscopy reveals endothelial cells proximal to the MB to be polygonal and flat in shape, but those under the MB become spindle shaped, engorged and aligned in the direction of blood flow³⁹⁾ (**Fig. 3**). These changes of endothelial cell shape and alignment between the two segments indicate that the intimal surface of the LAD under the MB is subjected to high shear stress, resulting in a reduced suscep-

Fig. 2 The three-dimensional volume-rendered image (A) and the curved planar reformat image (B) of the LAD having an MB by a 16-row multislice computed tomography in a 62 year-old man with angina pectoris. This MB is situated in the middle segment of the LAD (the two arrows in Figs. A and B indicate the entrance and exit of MB). Low density plaque is shown in the LAD proximal to the MB. The thickness and length of the MB are 2.6 and 29.8 mm, respectively. These figures are diverted from our article of reference no. 33.

tibility to atherosclerotic change.⁴⁰⁾ These phenomena are confirmed in the cholesterol-fed rabbit model in which endothelial permeability for tracer material is significantly suppressed in the segment covered by myocardium.⁴¹⁾ Suppressed endothelial permeability under the myocardial covering leads to atherosclerosis suppression even under the high serum cholesterol level, whereas atherosclerotic lesions consistently progress in the intima of the coronary segment running in the epicardial adipose tissue, as serum cholesterol levels increase (**Fig. 4**).⁴¹⁾ Such hemodynamic change in the human coronary segments under the MB also decreases the expression of vasoactive proteins such as endothelial nitric oxide synthase, endothelin-1, and angiotensin-converting enzyme in the endothelial cells of the arterial intima relative to the segments proximal to the bridge.⁴²⁾ High shear stress also reduces the ability of intimal smooth muscle cells to produce collagen fibers under the MB.^{43, 44)} The alteration of hemodynamic force in the coronary artery is caused by MB compression itself during systole. It is rephrased that such force is practically subjected to the MB thickness and length for regulation of the atherosclerosis suppression in the segment under the MB.³⁸⁾

Imaging Analysis

Imaging analyses by coronary angiography, IVUS and

Fig. 3 Atherosclerosis ratio (ratio of intimal area/medial area of LAD section stained with EVG) at intervals of 5 mm from the ostium of the left coronary artery up to 7 cm. The LADs with no MB [MB (-): n = 100] are selected from 230 autopsied hearts as age and sex matched control for the LADs having MB [MB (+): n = 200]. Black bold line indicated by MB is the portion mainly covered by MB in the MB (+) group, and the values of atherosclerosis ratio in these segments of the MB (+) group are significantly lower than those of the MB (-) group.

Fig. 4 Scanning electron microscopies of the intima of the LAD in a rabbit fed with cholesterol-diet during 20 weeks (serum cholesterol level; 2100 mg/dl). The direction of blood flow is from the top to the bottom of the figures. Fig. A is the intima of the intraepicardial segment running within the adipose tissue, demonstrating many monocytes adhesion on the surface of the intima with partial bulging due to the invasion of monocytes under the endothelial cells. Fig. B is the intima of LAD covered by myocardial tissue in the same rabbit as Fig. A, showing spindle-shaped and engorged endothelial cells along with blood flow axis, but there is no evidence of monocyte attachment to the endothelial surface in spite of hypercholesterolemia. These figures are diverted from our article of *J Jpn Coll Angiol*, 2003; **43**: 691-8.

MSCT reveal that atherosclerotic changes are absent in the arterial intima under the MB, whereas the LAD segment proximal to the MB is usually atherosclerotic.^{9, 10, 33, 35, 45)}

By ICD study, the MB presence reduces the systolic and diastolic dimensions of the segment under the bridge due to the MB compression itself, which leads to the increase of blood flow velocity at both systole and diastole in this segment.⁴⁶⁾ This increase of blood flow velocity under the MB generates high shear in this segment, thus resulting in reduced lipid permeability. By IVUS observation, blood pressure in the segment proximal to the MB is higher than in the aorta, leading to the development of atherosclerosis in the segment proximal to the bridge.⁴⁷⁾ By histopathologic examination, the cross-sectioned cut-surface of atherosclerotic lesions in the segment proximal to the MB is usually eccentric in shape,^{10, 38)} thus leading further support to the premise that this segment is ruled by the turbulent complex blood flow.

CORONARY HEART DISEASE AND SUDDEN CARDIAC DEATH BY AN MB FREE FROM ATHEROSCLEROSIS

Survey of Case Reports

The presence of MB has been long considered benign in the clinical settings, because patients with MB do not

always experience myocardial ischemia or infarction.^{7, 8)} By coronary angiography, the long-term prognosis for patients with MB in the LAD has been considered good.³⁰⁾ However, several studies indicate that the presence of MB is associated with CHDs,^{2, 6, 9, 10, 13, 15)} deleterious arrhythmias,^{4, 13)} and sudden cardiac death.^{12, 48, 49)} In recent decades, symptomatic MB related to cardiac pathology has been described in detail, and such events are widely recognized among the cardiologists. In this review, we hereby summarize the previous case reports describing the symptomatic MB. A total of 180 English language reports indicated in Appendix section (1968-2008) plus three references of no.22, 23, and 46 is tallied for 216 symptomatic MB cases (**Table 1**): 172 cases (79.6%) exhibited CHDs including angina, myocardial ischemia, myocardial infarction, and sudden cardiac death.

This survey clearly indicates that MB is causative of CHD in some cases regardless of coronary atherosclerosis. The average age of symptomatic MB cases is 48.2 ± 16.7 year-old (**Table 1**), obviously younger than that casually encountered at routine clinical practice in Western countries (65.8 year-old in males and 70.4 year-old in females).⁵⁰⁾ There is thus a striking difference in the age of the patient between the two groups. This may be based on those reports having described the only distinctive

Table 1 The profiles of symptomatic MBs

Age	average $\pm$ SD	48.2 $\pm$ 16.7 year-old (0 to 86 year-old) 0–49 year-old: 112 cases (51.9%) 50–86 year-old: 104 cases (48.1%)
Sex ratio (male: female)		155: 61 (71.8%: 28.2%)
Main clinical diagnosis		CHDs* 172 (79.6%) arrhythmia 6 (2.8%) HCM-related diseases 20 (9.3%) sudden death 5 (2.3%) cardiac failure 2 (0.9%) heart transplantation 3 (1.4%) cardiac anomalies 8 (3.7%)
MB location		LAD 200 (92.6%): proximal 23 (10.6%), middle 147 (68.1%), distal 6 (2.8%), not described 24 (11.1%) [†] LCX 5 (2.3%) RCA 11 (5.1%)
Intimal lesion of the coronary artery with MB		
	No	183 (84.5%): thrombus 7, spasm 12 [‡]
	Yes	27 (12.5%): proximal to MB; 16, under MB; 7, distal to MB; 4
	Aneurysm	1 (0.5%): under MB
	Dissection	1 (0.5%): under MB
	Not described	4 (1.9%)

The source of this table is obtained from the 180 English case reports under the search conditions of “myocardial bridge” or “myocardial bridging” which is registered in the online data base of PubMed (U.S. National Library of Medicine) from 1968 to Dec., 2008 (three refs. of nos. 22,23,46 and 177 refs. in Appendix).

MB; myocardial bridge, SD; standard deviation, CHDs; coronary heart diseases, HCM; hypertrophic cardiomyopathy, LAD; the left anterior descending coronary artery, LCX; the left circumflex coronary artery, RCA; the right coronary artery.

*: CHDs including angina, myocardial ischemia, and myocardial infarction; [†]: MB location in the LAD described in each literature; [‡]: findings in the arterial lumen or arterial wall.

MB effect on the LAD, the intima of which was entirely free from atherosclerotic lesions. The additional effect on the occurrence of CHD due to altered hemodynamics by MB may have been masked in the elderly; in such elderly cases, intimal atherosclerotic lesion has already developed in any extent within the segment proximal to the MB as its natural course. Most of these authors might have carefully avoided the documentation of elderly cases because of complicated backgrounds to differentiate the sole MB effect by muscle contraction on myocardial ischemia from casual coronary atherosclerosis. Males (71.8%) predominate over females (**Table 1**), the reasons for which are not known. The vast majority of MB (92.6%) are found in the LAD or its branches, consistent with autopsy results,^{2, 7, 15-17)} and are usually located in the

middle LAD segment. In 183 cases (84.5%), it is noteworthy that no significant lesions are demonstrated in the intima of the coronary arteries only besides the MB presence. Nonetheless most of these patients are affected by CHD. In addition, among 23 CHD patients with MB, MSCT identified three patients lacking any significant coronary wall lesion, finding only the presence of MB.³³⁾

Sudden Cardiac Death

It should be also noted that MB plays a significant role on sudden cardiac death.^{48, 49, 51)} Among the 16 cases of sudden cardiac death in subjects under 35 years-old, caused by non-atherosclerotic coronary diseases, MB was demonstrated in 6 cases (37.5%) in the LAD.⁴⁹⁾ Among 300 incidental cases deceased from CHD, MB was pres-

Fig. 5 The thickness of the MB is varied. In Fig. a of an LAD tissue stained with EVG at 5.5 cm from the ostium of the left coronary artery in a case with no cardiac lesion, the artery is covered by thin MB (270 μm) and reveals no atherosclerotic lesion in its intima. In Fig. b of an LAD tissue at 5.0 cm from the ostium of the left coronary artery in a case with myocardial infarction, the artery is covered by relatively thick MB (1 mm) and lacks no significant intimal lesion except for mild fibrous thickening.

ent in 118 cases (39.3%) and the majority of these (72 cases or 61.0%) experienced sudden cardiac death.⁵¹⁾ Apart from these, in sports medicine, the presence of MB has been established as one of the main causes of sudden cardiac death among young athletes such as basketball, football, and soccer players.^{52, 53)} These findings indicate that an MB undergoing vigorous compression of the LAD during cardiac systole by physiological exertion may cause life threatening events even in the youth.

ANATOMICAL PROPERTIES OF THE MB PRE- DISPOSING TO CHDS

Anatomical Properties of MB

Most symptomatic MB cases have been confirmed by coronary angiography, but the distinct anatomical properties of pathogenic MB such as its location, length and thickness have not been explored. This may sometimes result from difficulty in objective identification of the pathogenic cases under the clinically limited situations.

As for MB location, the LAD usually enters the myocardium 3.4-4.5 cm distal to the left coronary ostium by autopsy studies.^{7, 16, 17, 38)} The closer the bridged segment is to the entrance of left coronary ostium, the greater the extent of intimal thickening in the segment proximal to the bridge.¹⁷⁾ It is likely that the closer the bridged segment is to the left coronary ostium, the more turbulent the blood flow becomes in the proximal segments.⁴⁾ In contrast, the cross-sectional area ratio of intima to media

in the proximal segments of the LAD is significantly lower in cases having MB within 4.5 cm of the left coronary ostium than in cases without MB.³⁸⁾ The discrepancy between these two studies on the relationship between bridge location and intimal lesions^{17, 38)} is probably due to the characteristic cross-sectional shape of the intimal lesions in the segment proximal to the bridge, which is eccentric.³⁸⁾ When the MB is located in the proximal or middle portion of the LAD, the segment proximal to the bridge is vulnerable for atherosclerotic evolution.⁴⁾ In fact, MBs are usually located in the proximal or middle segment of LAD in symptomatic cases (**Table 1**).

MB length and thickness also influence atherosclerotic evolution throughout the LAD intima.^{4, 17, 38)} The long and/or thick bridge segment suppresses the atherosclerosis development in the arterial segment under the bridge to a greater degree than the short and/or thin MB.³⁸⁾ It results from differences in contractile force exerted by MB.³⁸⁾ By autopsy, MB length varies 3-69 mm^{4, 15, 20)} with mean values of 19.7,¹⁷⁾ 14.4,³⁸⁾ and 21.9 mm in males, and 20.3 mm for females.²¹⁾ The mean MB thickness varies by the methods of measurement, being 2.8,⁷⁾ 2.0,²¹⁾ and 0.9 mm,³⁸⁾ respectively (**Fig. 5**). The thickness of MB is generally proportionate to its length.³⁸⁾ In addition, longer bridges tend to be located more proximally in the LAD, and thicker bridges are located significantly more proximally.³⁸⁾ The fundamental anatomical properties of the MB, including the location, length and thickness, constitute the key regulators of atherosclerosis evolution in the

Fig. 6 The changes of atherosclerosis ratio (ratio of intimal area/medial area in the cross-section of LAD) at 5 mm intervals from the MB entrance upward to the left coronary ostium in cases with myocardial infarction [MI (+); $n = 36$] and cases with no infarction [MI (-); $n = 100$]. In MI (-) group, atherosclerosis ratio increases from the MB entrance up to 1.0 cm but shows no significant fluctuation from 1.0 cm upward to the coronary ostium. In contrast, atherosclerosis ratio is larger than that of MI (-) group (*: $P < 0.01$) and increases from the MB entrance upward to 2.0 cm and tends to decrease.

affected LAD.

Anatomical Properties of the MB Predisposing to Myocardial Infarction

It is evident that CHD and related conditions are associated with the anatomical properties of MBs. The long and/or thick MB leads to intensive systolic compression of the LAD, causing an increase of retrograde blood flow toward the proximal LAD segment and disturbance of blood perfusion to the peripheral myocardium.^{38, 45, 48)} In hypertrophic hearts, a long MB covering the LAD may result in clinically significant LAD compression.⁴⁵⁾ A deeply situated intramyocardial LAD with a thick MB appears to be associated with sudden cardiac death.⁴⁸⁾ Patients demonstrating more than 75% narrowing of the LAD during angiography as a result of MB compression during systole experience severe myocardial ischemia.²⁵⁾ This situation is usually associated with a longer or thicker MB.

Recent histopathologic analysis of LADs in the 300 autopsied hearts elucidates that the MB muscle index (thickness X length of MB) in cases with myocardial infarction is significantly larger than that in cases without cardiac lesion.¹⁴⁾ Moreover, in cases with myocardial infarction, the location of segment having the most stenotic

intimal lesion in the LAD proximal to MB significantly correlates with the location of the MB entrance, and the preferential site of LAD lesion causing infarction converges the proximal segment at 2 cm distant to the MB entrance (**Fig. 6**).¹⁴⁾ These findings indicate that the increased mass of MB has an independent role on the occurrence of myocardial infarction through a convergence of atherosclerotic evolution at the settled site of the proximal LAD. However, the specific quantification of the anatomical variables predisposing to myocardial infarction is still necessary. Recent analyses by non-invasive MSCT may enable us to objectively investigate the anatomical properties of the MB and further submit their variables as a risk factor for CHDs.

PATHOPHYSIOLOGY OF CHD BY THE MB

The pathophysiology of CHD provoked by MB has been investigated from a viewpoint of coronary flow disturbance using IVUS and Doppler techniques.^{6, 9, 10, 47, 54)} The arterial compression by the MB leads to retrograde blood flow toward the proximal segment at systole and increases blood flow velocity at early diastole due to delayed compression release at the bridged segment.⁹⁾ In ad-

dition, the diameter and vessel cross-sectional area of this segment are consistently smaller than those in adjacent to the segments proximal and distal to MB at cardiac diastole.^{54, 55)} These abnormal hemodynamic alterations across the bridged segment of the LAD may result in a reduced flow reserve.^{9, 54)} They are paradoxically provoked and enhanced by nitroglycerin injection.¹⁰⁾ On the contrary, the altered hemodynamic changes in the LAD with the MB are normalized through coronary stent placement in the segment under the MB, which results in subsequent improvement of clinical symptoms.⁵⁴⁾ These facts conversely suggest that the MB contraction produces the abnormal coronary flow. Delayed relaxation of the bridged segment at diastole and reduction of coronary flow reserve may be the important mechanisms producing myocardial ischemia.^{6, 9, 10, 54)} In fact, in patients with MB in LAD, coronary flow reserve is decreased, and perfusion defects are recognizable only in the mid-anteroseptal myocardium through TI-201 SPECT.⁵⁶⁾

In addition, tachycardia amplifies the abnormal coronary flow and exacerbate the patient's symptoms.^{25, 27)} Effort-induced ischemia of the heart with MB is attributed to tachycardia which increases oxygen demand of the myocardium and reduces coronary flow during systole,⁴⁹⁾ because an increase in heart rate shortens the diastolic filling period more than the systolic flow time.⁵³⁾ The tachycardia-induced reduction of blood flow in the coronary artery may cause not only myocardial ischemia but also sudden cardiac death for the young athletes during exercise. In addition, vasospasm and subsequent thrombosis occurring in the bridged segment can also cause myocardial ischemia and/or infarction, even at rest (**Table 1**). Endothelium-dependent vasodilator, such as acetylcholine, enhances vasoconstriction for the bridged segment, suggesting endothelial dysfunction in this segment.⁶⁾ This dysfunction potentially causes vasospasm as well as subsequent transient platelet aggregation and/or thrombosis in the segments proximal to or under the MB.⁶⁾

CONCLUSION

The MB plays the role of "double-edged sword," suppressing coronary atherosclerosis under the bridge, yet generating abnormal blood flow and CHD. Both events, such as suppressive effect on coronary atherosclerosis under the MB and atherosclerosis evolution in the settled site in the LAD segment proximal to the MB, result from different pathophysiologic mechanisms, though each originates from a common anatomical structure of the

MB. With the advance of imaging techniques, cardiologists are currently aware of the clinical significance of MB and its relationship to CHD. It remains further necessary to quantify the anatomical properties of MB and clarify their associated risks for CHD.

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APPENDIX

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