A 70-year-old woman with a 5-year history of hypertension that was well controlled with benidipine (4 mg/day) and carvedilol (10 mg/day) at 120–130/70–80 mmHg with pulse rate 70–80 beats/min presented to hospital with the chief complaint of exertional chest oppression. Her family history was unremarkable. She had been diagnosed...

Myocardial Bridge With Progression in the Short Term

Tatsuo Masuda, MD; Kazuki Ito, MD; Tsuyoshi Nagao, MD; Yuichiro Ide, MD; Hiroki Tsuboi, MD; Takatoshi Goto, MD

Figure 1. Coronary angiography of the left anterior descending (LAD) artery. (A) The LAD artery in end-diastole in the present patient at 68 years of age. No obvious stenotic lesion was noted. (B) The LAD artery in end-systole in the present patient at 68 years of age. Narrowing of 50–75% was visible and the total length of the bridged segment was 34.5 mm. (C) The LAD artery in end-diastole in the present patient at 69 years of age. No obvious stenotic lesion was observable. (D) The LAD artery in end-systole in the present patient at 69 years of age. Narrowing of 75–90% was seen and the total length extended to 36.2 mm. (E) The LAD artery in end-diastole in the present patient at 70 years of age. No obvious stenotic lesion was observed. (F) The LAD artery in end-systole in the present patient at 70 years of age. Narrowing of 90–100% was documented and the total length further extended to 38.6 mm.
Myocardial Bridge With Progression

Conventional antihypertensive therapy, and the MB was assigned to careful observation. Although treadmill exercise test was not performed before and after the stent placement, the intervention successfully relieved the symptom and improved exercise tolerance, which suggested that the symptom was predominantly attributed to the associated ischemia from right coronary artery stenosis. The exertional chest oppression recurred, however, 1 year later. CAG showed no suspected in-stent restenosis or newly developed organic lesion but 75–90% systolic narrowing of the LAD at the MB site (Figure 1D) with its length extended to 36.2 mm. Because association of coronary spasm with MB is reported, with myocardial bridge (MB), an anatomical variant in which a segment of coronary artery passes intramurally through the myocardium beneath a muscle bridge, at the age of 68 years, when she underwent stent placement in the proximal portion of the right coronary artery to treat a 90% stenotic lesion seemingly responsible for the first chest oppression. The MB was located in the middle portion of the left anterior descending (LAD) artery, and it showed 50–75% narrowing during systole only, with a length of 34.5 mm (Figure 1B) on coronary angiography (CAG). After stent placement, the antiplatelet agents aspirin (100 mg/day) and clopidogrel (75 mg/day), and anti-lipemic agent atorvastatin (5 mg/day) were added to the conventional antihypertensive therapy, and the MB was assigned to careful observation.

Although treadmill exercise test was not performed before and after the stent placement, the intervention successfully relieved the symptom and improved exercise tolerance, which suggested that the symptom was predominantly attributed to the associated ischemia from right coronary artery stenosis. The exertional chest oppression recurred, however, 1 year later. CAG showed no suspected in-stent restenosis or newly developed organic lesion but 75–90% systolic narrowing of the LAD at the MB site (Figure 1D) with its length extended to 36.2 mm.
involvement of coronary spasm was assessed on an ergo-vine loading test, which showed no spasm induced. Given that we presumed that the symptom was attributed to ischemia induced by the progressed MB with tachycardia upon exertion this time, benidipine and carvedilol were increased to 8 mg/day and 20 mg/day, respectively, to reduce the myocardial contraction and oxygen demand. Furthermore, atorvastatin was increased to 10 mg/day to suppress development of an atherosclerotic lesion that was likely to arise just proximal to the MB. The symptom was relieved but recurred approximately 1 year later, which led to admission for further evaluation.

On admission, the patient was stable with blood pressure of 126/76 mmHg and pulse rate of 72 beats/min, and physical findings were insignificant, with no cardiac murmur, jugular venous distension, or lower leg edema. Results of laboratory testing were also not remarkable. Chest X-ray showed no cardiomegaly; a cardiothoracic ratio of 50%; and no pulmonary congestion, with no obvious changes during the past 2 years. Electrocardiogram (ECG) at rest showed regular sinus rhythm and 0.1–0.2-mV ST-segment depression of diastole/systole of 10/13 mm without asymmetry, which had not changed for the past 2 years.

Echocardiogram showed no characteristic findings except for the newly developed mild hypokinesis from the middle to distal anterior wall, with the wall thickness diastole/systole of 10/13 mm without asymmetry, which had not changed for the past 2 years. CAG again showed no restenosis or newly formed organic lesions. The MB, however, completely compressed the middle portion of the LAD for 25 mm during systole. The total length of the narrowing extended to 38.6 mm (Figure 1F). The ergo-vine loading test again failed to show a coronary spasm. Myocardial ischemia was assessed on coronary arterial pressure and coronary flow reserve using a Prime Wire PRESTIGE pressure guide wire (Volcano; CA, USA). Coronary arterial pressure was decreased at and distal to the MB. Adenosine (140 μg·kg⁻¹·min⁻¹) resulted in a coronary flow reserve of 0.72. Pull-back LAD pressure showed an early diastolic dip distal to the bridge that overshot within the bridge, both of which are characteristics of MB (Figure 2B). Left ventriculography showed poor contractility from the middle anterior wall to the apex (Figure 2C). All of these findings suggested worsening of the MB-induced myocardial ischemia. Benidipine was increased to 16 mg/day to suppress non-inducible but possible coronary spasms, and reduce oxygen demand directly by inhibiting myocardial contraction, and indirectly by lowering blood pressure, imidapril (5 mg/day) and spironolactone (50 mg/day) were also given, to utilize the possible inhibitory effects of the angiotensin-converting enzyme inhibitor (ACEI) on angiotensin II receptor blocker (ARB) and aldosterone antagonist against myocardial degeneration.4 This patient is currently followed up on an outpatient basis and has not experienced symptom recurrence.

The present case illustrates a rapidly progressive case of MB. Because correlation of MB length and bridging muscle thickness has been reported, elongation of the MB may indicate progression of “hypertrophy” at the MB site in the present case, although not measured. Although compensatory hypercontractility and possible localized hypertrophy may present Takotsubo-like findings, the current case is different from Takotsubo cardiomyopathy in that: (1) the episodes were not in acute settings and not related to known stress; (2) ECG was not characteristic; and (3) the findings were not transient but persisting on follow-up echocardiogram, which may in turn suggest the mild chronic degenerative changes of the lesion. Nevertheless, any stress might predispose to MB symptom exacerbation, which may manifest the features of Takotsubo cardiomyopathy.

The symptom seemed somewhat well controlled with the current medications in the present case, which suggests the possibility that ACEIs, ARBs and aldosterone antagonists may contribute to prevention of MB progression itself. Although symptom recurrence and/or further worsening of left ventricular wall contractility could be indications for invasive treatment such as coronary stenting, bypass surgery, and/or myotomy, in the present case, the length, narrow diameter of the vessel, progressive nature of the MB, and patency distal to the bridged segment would favor surgical correction of coronary perfusion over coronary stenting.

The prognosis of MB is generally considered to be good and unaffected by the degree of stenosis. Its clinical significance, however, is currently under review because not a small number of patients with MB are reported to have a greater incidence of myocardial ischemia-related events such as acute myocardial infarction, sudden death, and coronary spastic angina. There are no definite factors known to date to predict prognosis or indicate specific treatments. Further accumulation of MB cases and elaboration to establish such factors and treatments are mandatory. For the time being, when definite indications and treatments are not available, because medication alone would not improve overall outcome, the treatment strategy should be well discussed according to the individual’s desired quality of life.

In conclusion, to our knowledge, this is the first radiographic evidence of MB progression of length and degree of narrowing. Although the mechanism of MB progression is not fully elucidated, it is possible that asymptomatic chronic ischemic degeneration may result in ischemic cardiomyopathy.

The use of ACEIs, ARBs and aldosterone antagonists may possibly prevent the progression of ischemic changes in the area supplied by the bridged artery. Further investigation into the mechanism of progression and a possible new strategy against it are warranted.

References