Left Circumflex Coronary Artery Bridging

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SUMMARY

Myocardial bridging most frequently occurs on the left anterior descending coronary artery and may cause ischemia and related complications. Right coronary artery myocardial bridges (MB) are rare. We report a patient with an unusual coronary bridge, a left circumflex coronary artery bridge, who presented with exercise-induced angina pectoris that was relieved with medical therapy. (Jpn Heart J 2002; 43: 423-427)

Key words: Myocardial bridging, Left circumflex coronary artery, Ischemia, Beta-blocker therapy

MYOCARDIAL bridge (MB) is defined as the intramural course of a major epicardial coronary artery,1) and mostly is confined to the left ventricle and the left anterior descending coronary artery. Several right coronary MBs have been reported,2-5) but only two left circumflex MBs have been reported in the literature.6,7) In this article, we present a case of left circumflex MB and discuss the unusual locations of MB, mechanism of ischemia, and therapeutic approaches.

CASE

A 45-year-old hypertensive Turkish male was admitted to hospital because of chest pain radiating to the left shoulder on exertion for the last 4 months. Chest pain episodes were almost always induced with exercise and rapidly disappeared with resting. He had no risk factors for coronary artery disease except smoking and family history. Blood tests, including CBC, electrolytes, liver enzymes and lipid profile, were in their normal ranges. Physical examination was unremarkable. Blood pressure was slightly high (150/95 mmHg). The resting ECG was normal but after 7 minutes of exercise with the Bruce protocol he developed chest pain associated with 1.5 mm horizontal ST depression in the inferior and lateral leads. Coronary angiography showed normal left main and normal left anterior descending and right coronary arteries. The left circumflex artery was also nor-
mal and without atherosclerotic lesion, although there was complete systolic obliteration and angulation of the proximal segment (approximately 2.5 cm in length) (Figure). Echocardiography revealed mild concentric hypertrophy (IVS 1.3 cm, PW 1.2 cm). Systolic functions were normal and the ejection fraction was calculated to be 65%. Since the bridging segment was relatively long for stent implantation, medical therapy with a beta-blocker (metoprolol 100 mg daily) was chosen. Surgical therapy was considered as the next choice if the non-surgical intervention failed. Exercise induced anginal episodes disappeared following the first week of medical therapy. The patient was asymptomatic during the follow-up for 3 months. Exercise stress test was normal and there was no myocardial perfusion defect on exercise TI-201 SPECT imaging.

**DISCUSSION**

Myocardial bridging is one of the non-atherosclerotic anatomical abnormalities of the coronary arteries. The major coronary arteries pass over the epicardial
surface of the heart and, as described in pathological series, it is not uncommon for a segment of the artery to descend into the myocardium and thus be surrounded by myocardium to form a MB. There is a large difference between the incidence of MBs observed in pathological series and coronary angiography series. While the incidence in autopsy series has been reported to be 15% to 85%, the angiographic incidence has been reported to be 0.5% to 2.5%. Most of the bridges seen in pathological examinations are invisible with angiography because only the deep type of bridges may be apparent angiographically. The bridging appears on angiography as systolic narrowing or complete obliteration of the arterial lumen, while the lumen is normal during diastole. Recent intravascular ultrasound (IVUS) studies in symptomatic patients have showed that vessel compression is not limited to systole, but also persists in diastole, thereby affecting the predominant phase of the coronary perfusion.

The left anterior descending coronary artery is the most frequent location of MB. Right ventricular MB has only been rarely reported. When bridges are found in the right coronary artery they are usually located in the posterior descending artery, and coronary constrictions is related to hypertrophic left ventricular myocardium. Systolic collapse of the right ventricular branches of the right coronary artery in patients with chronic lung disease and pulmonary hypertension has also been reported. Coronary angiography may underestimate the true frequency of the right ventricular myocardial bridge because systolic obliteration of the right ventricular branch is not expected unless there is right ventricular systolic hypertension together with the anatomical abnormality. Thus, the systolic obliteration of a segment of the right ventricular branch can be used as an indirect sign for right ventricular hypertension. Woldow, et al reported a 76 year old patient with chronic obstructive lung disease and dilated cardiomyopathy with multiple etiologies who had systolic constriction of the right coronary artery beyond the crux. To our knowledge, our case is only the third case of angiographically proven significant systolic constriction of the left circumflex artery. Arjomand, et al reported the first case of myocardial bridging of the left circumflex coronary artery associated with acute myocardial infarction. Their patient had presented with sudden onset of shortness of breath and ST segment depression in leads DI, aVL, and V5-V6, and elevation of the cardiac enzymes. Mid-left circumflex bridging has been diagnosed with coronary angiography and treated with stenting of the bridged segment. Garg, et al also reported another occlusive systolic bridging of the left circumflex coronary artery.

Several mechanisms have been postulated to explain ischemia resulting from myocardial bridging of a coronary artery, including vasospasm and systolic kinking of the artery, resulting in direct physical damage to the underlying endothelial cells. Exercise-induced high heart rate, shortened diastolic perfusion
time, increased contractility, compression of the artery, and increased flow velocity may cause ischemia that is relieved by beta-blocker therapy. Schwartz, et al noted that a reduction of vascular compression and maximal flow velocity within the bridged segment of the artery by a short acting beta-blocker alleviates angina and ischemia.\textsuperscript{15)} Recently, phasic systolic compression with a localised peak pressure, persistent diastolic diameter reduction, increased blood flow velocities, retrograde flow, and reduced flow reserve have been shown with IVUS and Doppler studies.\textsuperscript{13)}

Although MB is a relatively common condition, especially in pathological series, controversy exists concerning its clinical and prognostic relevance and the appropriate therapeutic approach for symptomatic patients. There are three potential therapeutic strategies: pharmacological intervention, surgical myotomy and bypass grafting, and percutaneous angioplasty with stent implantation. Coronary vasodilators such as dipyridamole and nitroglycerin may enhance the pressure gradient between the bridge and proximal segments by vasodilatation and reduction of the proximal pressure, resulting in enhanced retrograde flow.\textsuperscript{16,17)} Studies have suggested that tachycardia may worsen ischemia due to a reduction of the diastolic coronary filling duration.\textsuperscript{13)} Negative inotropic agents, especially beta-blockers, are capable of reducing systemic and intramural pressures and thereby the external vessel compression. The accompanying negative inotropic effect mainly prolongs diastole, but also improves coronary perfusion.

Metoprolol successfully relieved the exercise-induced angina in our patient. Since the patient was asymptomatic, and there was no perfusion defect on exercise Tl-201 SPECT imaging with therapy, we did not consider performing any further invasive intervention.

**Conclusion:** Although myocardial bridges most frequently involve the left anterior descending artery, with this case we want to remind clinicians that MB may also occur in the left circumflex coronary and right coronary artery, and that the symptoms may be relieved with beta-blocker therapy.

**REFERENCES**