



## Case Report

## Measurement of fractional flow reserve in a patient with combined myocardial bridging and coronary fixed stenosis

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## ABSTRACT

A segment of coronary arteries traveling through the myocardium is known as myocardial bridging. Several reports have shown reduced fractional flow reserve (FFR) associated with myocardial bridging. We report a case in which FFR was measured in the left anterior descending artery involving both myocardial bridging and fixed stenosis.

**<Learning objective:** We demonstrated a case in which fractional flow reserve (FFR) was measured in the left anterior descending artery involving both myocardial bridging and fixed stenosis. Measurement of FFR was useful to guide PCI for the lesion modified by myocardial bridging.>

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## Introduction

A segment of coronary arteries traveling through the myocardium is known as myocardial bridging [1–10]. Its typical angiographic presentation is the systolic milking effect due to transient myocardial vessel compression. Although myocardial bridging is generally considered benign, it has been shown to cause myocardial ischemia, conduction disturbances, cardiac arrhythmias or sudden death. Measurement of fractional flow reserve (FFR) is increasingly used to evaluate the physiological significance of intermediate coronary stenoses [11–16]. In this report, we describe a case in which FFR was measured in the left anterior descending artery involving both myocardial bridging and fixed stenosis.

## Case report

A 72-year-old man with effort angina underwent treadmill exercise test. Because of positive ischemic changes during exercise, he was admitted for cardiac catheterization. Coronary angiography showed a myocardial bridging in the mid segment and a fixed stenosis in the distal segment of the left anterior descending artery (LAD) (Fig. 1). Systolic compression of the mid segment was seen

with return to a normal caliber during diastole (arrow head). A pressure wire (PressureWire, RADI Medical Systems) was advanced beyond both the myocardial bridging and the fixed stenosis, and a pressure sensor was positioned at the site distal to the stenosis. The ratio of mean distal intracoronary pressure to mean aortic pressure (Pd/Pa) was 0.96. After intravenous adenosine triphosphate to obtain maximal coronary blood flow, FFR was 0.71 suggesting that the combination of the myocardial bridging and the fixed stenosis could cause myocardial ischemia. Percutaneous coronary intervention (PCI) was performed for the fixed stenosis using drug-eluting stent (Fig. 2). After successful PCI, subsequent FFR became 0.82. When the pressure sensor was positioned precisely at the site proximal to myocardial bridging by pullback maneuver, Pd/Pa increased to 0.92. When the pressure sensor was positioned precisely at the proximal segment by further pullback maneuver, Pd/Pa remained to be 0.92. After the procedure, his symptoms disappeared. He was discharged the following day.

## Discussion

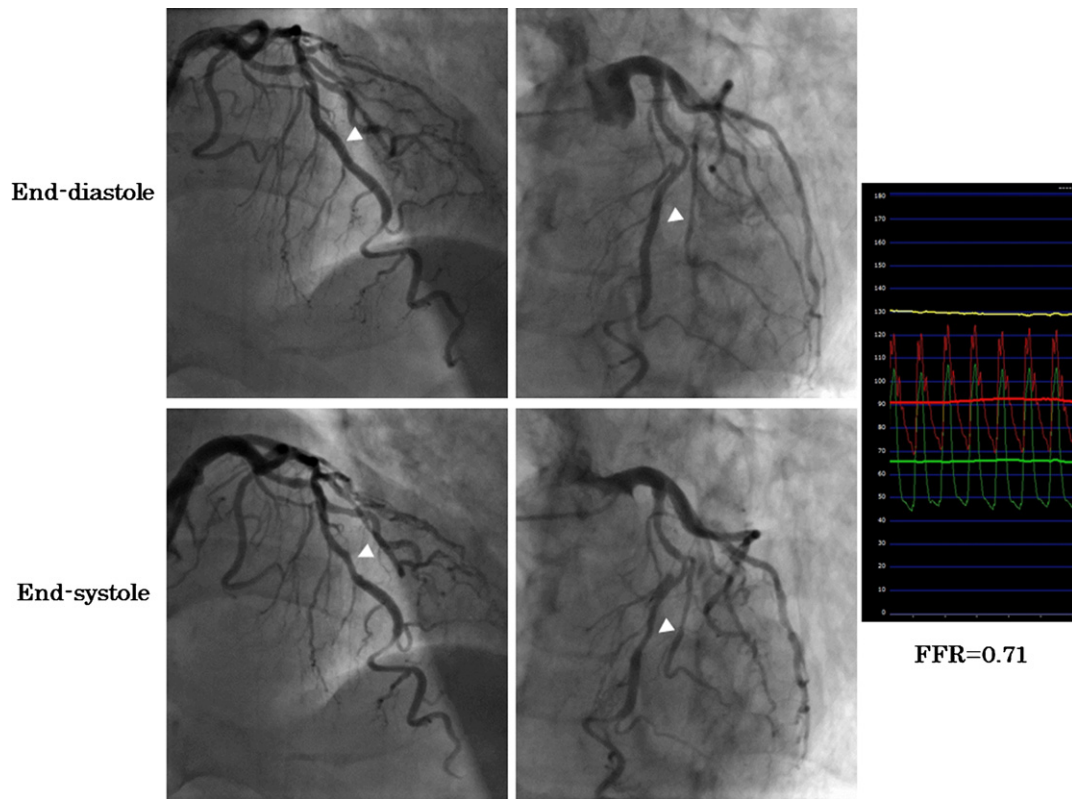
We demonstrated a case in which measurement of FFR was useful to guide PCI for an obstructive LAD modified by myocardial bridging.

Several reports have shown reduced FFR associated with myocardial bridging and its normalization with successful coronary stenting [8,9]. In the current case, initial FFR was 0.71 suggesting that the combination of the myocardial bridging and the fixed stenosis could cause myocardial ischemia in the LAD. After

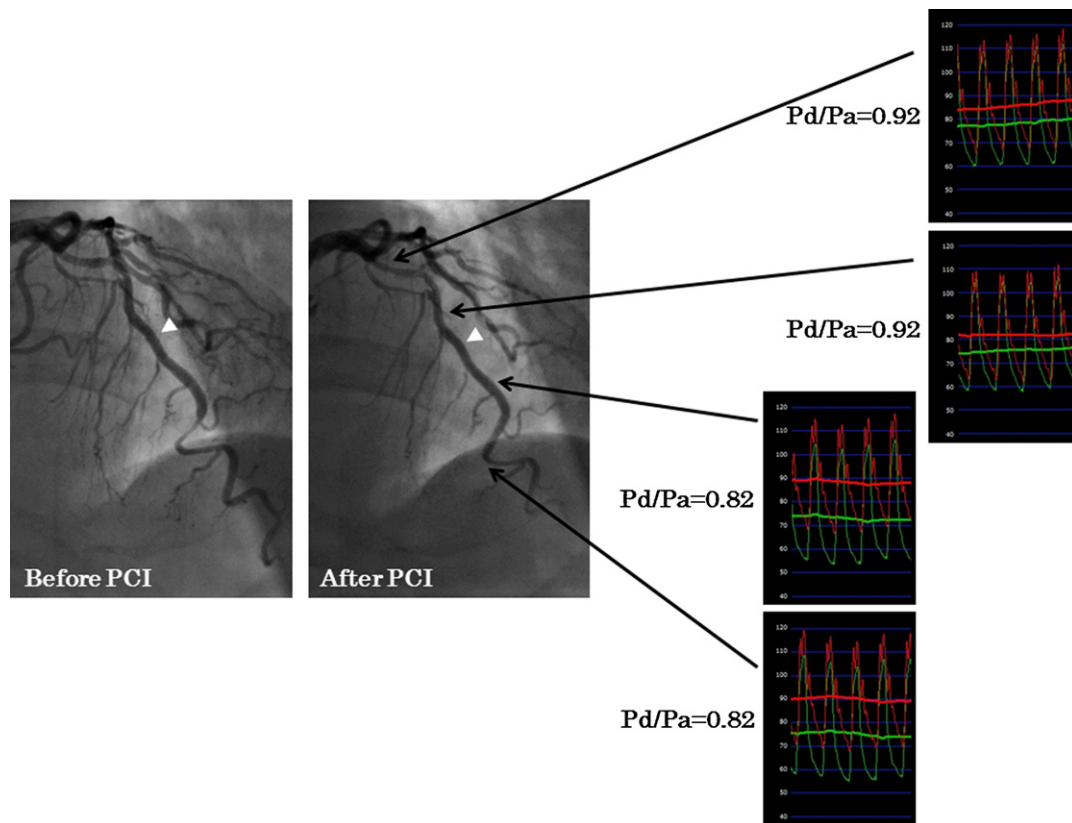
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**Fig. 1.** End-diastolic (upper panels) and end-systolic (lower panels) coronary angiograms. Arrowhead shows the site of myocardial bridging. Fractional flow reserve (FFR) was 0.71.



**Fig. 2.** End-diastolic coronary angiograms before and after percutaneous coronary intervention (PCI). The pressure sensor was positioned by pullback maneuver, and the ratio of mean distal intracoronary pressure to mean aortic pressure (Pd/Pa) was measured during hyperemia at each position. Arrowhead shows the site of myocardial bridging, and arrows show the sites of pressure sensor.

successful PCI, subsequent FFR was 0.82 suggesting that the myocardial bridging caused an intermediate stenosis without physiological significance, and was in part associated with the reduced FFR before PCI. On the basis of these findings, additional PCI for the myocardial bridging was avoided. Schwarz et al. previously showed that myocardial vessel compression in myocardial bridging was not limited to ventricular systole but was also carried on to early and mid-diastole [10]. Because coronary perfusion mainly occurs during diastole, this compression can cause myocardial ischemia. In the current case, if FFR was less than 0.75 after successful PCI for the stenosis, additional PCI for the myocardial bridging should be required. Park et al. recently reported the usefulness of measurement of FFR after intravenous dobutamine in the functional assessment of myocardial bridging [16]. They demonstrated that 2 of the 17 lesions associated with myocardial bridging became functionally significant after intravenous dobutamine. In the current case, we did not use dobutamine, and there was a possibility that myocardial bridging was functionally underestimated.

Therapeutic strategies in symptomatic patients with a myocardial bridging have been varied including calcium channel blockers or beta-blockers. The beneficial effects of beta-blockers are based on their negative inotropic and chronotropic actions, which should lead to a reduction in myocardial vessel compression and a prolongation of diastolic intervals. Further studies are necessary to clarify whether additional these agents reduce adverse cardiac events in patients with both myocardial bridging and fixed stenosis.

In conclusion, measurement of FFR might be useful for lesions accompanied with fixed stenosis and myocardial bridging.

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