Case Reports

# Effect of Revascularization of Chronic Total Occlusion on Tandem Stenoses in a "Donor" Artery

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The presence, severity, and extent of ischemia predict the outcome of coronary artery disease. Indeed the extent of myocardial ischemia and viability determines the value of revascularization in coronary stenosis after acute myocardial infarction. In multivessel coronary artery disease, noninvasive methods for the evaluation of ischemia are often inadequate to guide percutaneous coronary intervention. It is a common misperception that revascularization might not benefit a myocardial segment in the chronic total occlusion distribution when that segment is supplied by well-developed collateral vessels, because severe ischemia is unlikely to be present under these circumstances.

An 82-year-old man presented with acute coronary syndrome, tandem stenoses in a "donor" artery, and a chronic total occlusion of the right coronary artery. We present a rationale for using fractional flow reserve to determine the existence of ischemia and to aid in deciding the best approach to the treatment of that ischemia. **(Tex Heart Inst J 2014;41(5):547-50)** 

n multivessel coronary artery disease (CAD), noninvasive testing usually underestimates the extent of ischemia—more so when one of the lesions is a chronic total occlusion (CTO). Ischemia, regardless of the method by which it is diagnosed, is a major determinant of outcomes in CAD. We present a case of multivessel CAD wherein the determination of fractional flow reserve (FFR) was useful in revealing the evidence of ischemia, thereby resolving the ambiguity of a stenosis.

## **Case Report**

An 82-year-old man presented with acute onset of dyspnea and chest pain. His history included chronic obstructive pulmonary disease (emphysema and bronchitis), chronic kidney disease, depression, chronic atrial fibrillation, hypertension, congestive heart failure, and intermittent claudication after walking about 10 yards. A year before the current presentation, he had undergone implantation of a biventricular automatic implantable cardioverter-defibrillator (AICD) for symptomatic congestive heart failure, after medical therapy had failed. A dipyridimole nuclear stress test performed before AICD insertion showed an inferior infarction without ischemia (Fig. 1), and the patient's echocardiogram showed severe diffuse global hypokinesis, with a left ventricular ejection fraction (LVEF) of 0.15.

He was hospitalized and diagnosed with myocardial infarction (peak troponin I level, 5.04 ng/mL). After his medical management was optimized, the patient underwent coronary angiography. This showed 40% stenosis of the distal left main coronary artery (LMCA) and 80% stenosis (in tandem) of the left anterior descending coronary artery (LAD), with septal collateral vessels (Fig. 2) from the "donor" LAD to an occluded right coronary artery (RCA) (Fig. 3). He also had occlusion of the right external iliac artery and 70% stenosis of the left external iliac artery, with ankle–brachial indices of 0.45 and 0.8, respectively.

Before making a decision to perform coronary revascularization, we performed a fractional flow reserve (FFR) procedure on the intermediate stenosis in the LMCA. The FFR was 0.58 with the transducer distal to the LAD stenosis, and 0.73 with the transducer between the LMCA and LAD stenoses.

Key words: Acute coronary syndrome/therapy; coronary occlusion/therapy; coronary stenosis/diagnosis; decision making; fractional flow reserve, myocardial; percutaneous coronary intervention; myocardial revascularization/ methods; stents

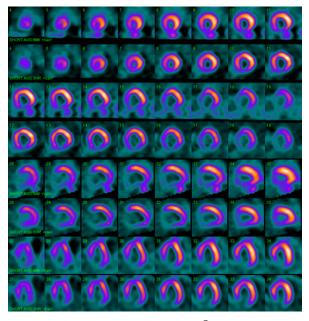
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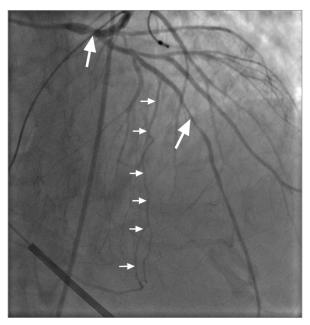
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© 2014 by the Texas Heart® Institute, Houston Because of his multiple comorbidities, the patient was not considered a candidate for bypass surgery. In his first angioplasty procedure, he underwent percutaneous coronary intervention (PCI) of the CTOs of both the RCA and the left external iliac artery. The RCA



**Fig. 1** This result of a Persantine Cardiolite<sup>®</sup> stress test reveals a fixed defect in the inferior wall during stress and rest, which is consistent with infarction.



**Fig. 2** Coronary angiogram (left anterior oblique view with cranial angulation) shows the left main coronary artery with tandem stenoses (indicated by the 2 large arrows), between which are 2 collateral vessels (one continuous, indicated by the small arrows). These collateral vessels served to reconstitute the occluded right posterior descending artery.

was intubated with a Judkins right 4.0 guide catheter. With the support of a  $1.5 \times 12$ -mm over-the-wire balloon catheter, an ASAHI® Fielder XT Coronary Guide Wire (Abbott Vascular, part of Abbott Laboratories; Redwood City, Calif) was advanced carefully through the chronic RCA occlusion. Contrast injection through the balloon catheter confirmed the distal intravascular location of the balloon catheter. Angioplasty was performed with the same balloon. The FFR was then

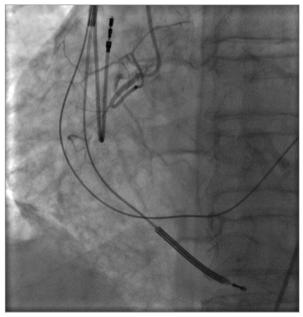


Fig. 3 Coronary angiogram (left anterior oblique view) shows total occlusion of the right coronary artery.

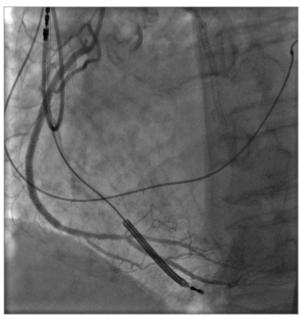
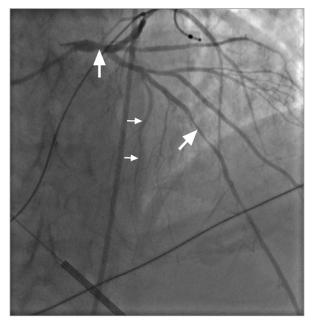


Fig. 4 Coronary angiogram shows the right coronary artery after the deployment of 3 bare-metal stents.



**Fig. 5** Coronary angiogram (left anterior oblique view with cranial angulation) shows the left main coronary artery with tandem stenoses (2 large arrows), between which are 2 septal branches with obliteration of collateral connections (small arrows).

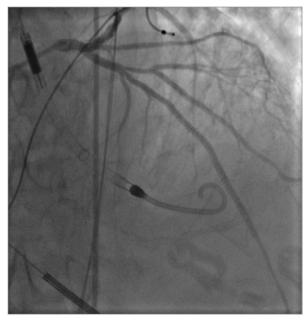
performed with the pressure wire in the posterior descending artery, yielding a value of 0.52. Three baremetal stents were then placed in the RCA (Fig. 4). The final FFR, with the transducer again positioned in the posterior descending artery, was 0.88.

Three weeks later, the patient underwent PCI of the chronic occlusion of the right external iliac artery and the tandem stenoses of the LAD and LMCA. After successful recanalization of the right external iliac artery, we intubated the LMCA with an XB 3.5 guide catheter (Fig. 5). Measurement of the FFR was performed: the numbers were 0.52 with the transducer distal to the LAD stenosis and 0.85 with the transducer between the LAD and LMCA stenoses. Percutaneous coronary intervention was performed in the LAD with 2 baremetal stents. The final FFR was 0.74 with the transducer between the LAD stenosis and the LAD stenosis and the LAD stenosis.

Three weeks afterwards, the patient was taken back to the catheterization laboratory. An Impella<sup>®</sup> device (ABIOMED, Inc.; Danvers, Mass) was inserted via left femoral artery access, and PCI of the LMCA was performed via right femoral artery access. The patient underwent successful stenting of his LMCA stenosis, yielding a final FFR in the distal LAD of 0.93 (Fig. 6). At his 6-month follow-up appointment, the patient was subjectively better, despite no improvement in LVEF.

### Discussion

This case report shows the usefulness of FFR for the evaluation of ischemia in a patient with multivessel



**Fig. 6** Coronary angiogram (left anterior oblique view with cranial angulation) shows the left coronary artery with stented left main and left anterior descending coronary artery lesions and an Impella<sup>®</sup> catheter in the left ventricle.

CAD, including LMCA and LAD stenoses in series and a CTO in the RCA. There are 2 key points about the ambiguity of the LMCA stenosis. First, the initial reversion of FFR from ischemic to nonischemic is secondary to the increase in resistance in the collateral beds after recanalization of the CTO. Second, the drop in FFR across the LMCA stenosis from nonischemic to ischemic is secondary to the recruitment of myocardium after revascularization of one of the high-grade stenoses in series—which then leads to large flow volume across a similar narrow passage.

Initially, in our patient, myocardial viability in the inferior infarcted segment was not evaluated during stress testing before AICD placement. However, this might not have affected our management in accordance with the 2009 appropriateness criteria for coronary revascularization<sup>1</sup>—had we found only the CTO in the RCA.

One could argue that the culprit LAD stenosis should undergo intervention because the inferior wall was infarcted and had a collateral blood supply from the LAD. However, nuclear perfusion imaging has been shown to underestimate the ischemic burden in up to 31% of zones otherwise proven by FFR to be <0.75 (or "totally occluded" on angiography).<sup>2</sup> Further, Werner and colleagues<sup>3</sup> previously found that, in 62 consecutive CTO lesions, evidence of ischemia (FFR, <0.80) during pharmacologic stress was present in all patients, even in those with regional dysfunction. In a study by De Bruyne and associates<sup>4</sup> that evaluated post-myocardial infarction patients who had globally normal LVEFs with or without regional wall-motion abnormalities, a lower FFR (0.52 ± 0.18 versus 0.67  $\pm$  0.16, *P*=0.0079) was found in patients with normal left ventricular systolic function than in those with regional wall-motion abnormality. This suggested that the amount of viable myocardium distal to the stenosis was related to the severity of the FFR abnormality.

This present case also illustrates an important FFR principle. The volume of blood flow across the stenosis determines the degree of pressure loss along a curvilinear pressure-flow relationship. Therefore, a small volume of flow will have a minor effect on pressure loss and a large volume of flow across a similar narrowing will have a major effect on pressure loss. In our patient, after RCA angioplasty with a 1.5-mm balloon, the FFR was 0.52 and the CTO was successfully recanalized, which resulted in the angiographic disappearance of the collateral vessels. Upon repetition of the FFR procedure after angioplasty, the FFR of the LMCA changed to the nonischemic range (from 0.73 to 0.85). That proved the influence of coronary flow volume and myocardial bed size on FFR and the physiologic influence of the increase in collateral resistance.<sup>5</sup> Had the LMCA stenosis been the only stenosis remaining after CTO revascularization, one could have decided upon medical management on the basis of the nonischemic FFR value. However, the flow through the LMCA stenosis in the presence of a 2nd (and in-series) stenosis in the LAD would be submaximal because of tandem-lesion physiology.<sup>6</sup> After our intervention on the LAD stenosis in series, the FFR of the LMCA stenosis dropped again to the ischemic range—a phenomenon explained by the fact that the recruitment of more myocardium leads to greater volume of blood flow through the stenosis. Finally, the uneventful LMCA stenting was accomplished with the help of Impella support, after adequate peripheral access conduits had been established. The final FFR reading across the LMCA and the LAD was nonischemic.

We conclude that the use of FFR to reveal ischemia can help in clinical decision-making in cases of complex multivessel CAD.

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