A Pressure Wire Study on the Effect of Impella 2.5 Support

How this device benefits coronary hemodynamics during high-risk angioplasty.

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The Impella 2.5 (Abiomed, Inc., Danvers, MA) is a percutaneously placed axial flow pump that is capable of delivering 2.5 L/min from the left ventricle (LV) to the ascending aorta. Previous studies have shown that left ventricular unloading by the device improves coronary flow in nonstenotic arteries, but the effects of unloading within a diseased coronary artery are unknown. This study is designed to determine the impact of Impella 2.5 mediated augmentation of cardiac output on coronary flow reserve (CFR) and fractional flow reserve (FFR) during percutaneous coronary intervention (PCI).

CASE REPORT

A 51-year-old man with advanced ischemic cardiomyopathy (LV ejection fraction approximately 15% with chronic systolic heart failure) was referred to the Tulane Heart and Vascular Institute for cardiac transplant evaluation. Coronary angiography showed angiographically nonobstructive disease in the left anterior descending (LAD), severe stenoses within the major diagonal branches, occlusion of a major obtuse marginal branch, and multifocal disease within the right coronary artery (Figure 1). Pressure wire evaluation of the LAD demonstrated the presence of hemodynamically significant disease within the proximal segment, (FFR 0.75, ischemic by definition) (Figure 2). A subsequent viability study showed the presence of viable myocardium in the anterior, anterior-lateral, and posterior segments.

The patient was deemed not suitable to be a candidate for surgical coronary revascularization because of poor graft targets and multiple comorbidities (chronic congestive heart failure (CHF), moderate mitral regurgitation, pulmonary hypertension, and renal insufficiency). During a subsequent admission for acute-on-chronic CHF, the patient was scheduled to undergo multivessel PCI utilizing the Impella 2.5 for intraoperative hemodynamic support.

PROCEDURE DETAILS

Access for the planned PCI was achieved via the right common femoral artery (CFA) using a standard 8-F sheath. A second 8-F sheath was introduced into the left CFA in preparation for the insertion of the Impella 2.5 device. After

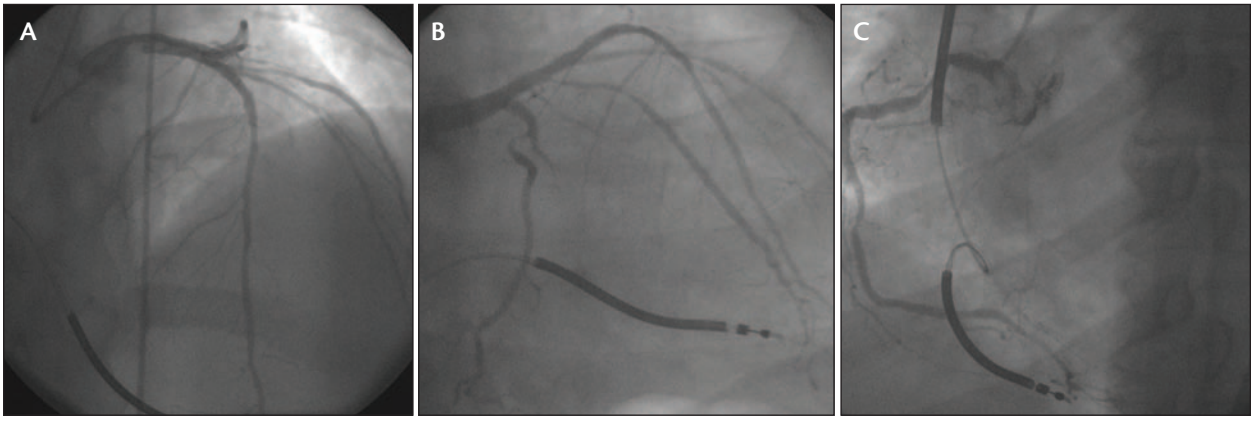

Figure 1. Selective coronary angiography showed nonobstructive LAD disease, severe major diagonal branch stenosis, and occlusion of the obtuse marginal artery. Right coronary artery disease is diffuse.

diagnostic coronary angiography, a coronary pressure wire was inserted and positioned within the mid-LAD. Mean coronary artery (Pd) and mean aortic pressures (Pa) were recorded at baseline and after induction of hyperemia using intravenous adenosine. FFR was calculated from these measurements: FFR = Pd/Pa at maximal hyperemia. CFR was calculated using coronary thermodilution curves recorded at baseline and during hyperemia.

The Impella 2.5 device was inserted via the left CFA using a technique that has previously been described by Raess et al.[1] Briefly, the 8-F sheath was exchanged for a 13-F sheath to accommodate the largest diameter of the device at the motor (12 F). The device catheter (with a smaller diameter of 9 F) was advanced across the aortic valve over an 0.018-inch wire using the monorail technique. The distal tip of the device was positioned in the mid-left ventricle under fluoroscopic guidance. Excessive catheter length was removed, and the catheter was secured at the groin to prevent forward migration.

After confirmation that the Impella catheter was correctly positioned across the aortic valve, mid-LAD FFR and CFR values were recorded at three different levels of support: P3, P6, and P8/P9. The mid-LAD lesion was then treated by placing a drug-eluting stent (DES), utilizing the pressure wire as a guidewire, and the FFR and CFR measurements were repeated at the maximum support level (P8/P9). The first and second diagonal branches and the right coronary artery were then revascularized by DES placement. Impella support was continued for an additional 14 hours after the procedure while the patient was monitored in the coronary care unit. Eventually, the patient was weaned off the Impella catheter over a short period of time and the device was removed. Hemostasis was achieved by manual compression at the groin.

RESULTS

The FFR and CFR values within the LAD for different levels of support are shown in Table 1 (Figure 3). The baseline FFR of 0.76 did not change during augmentation of the cardiac output. Conversely, the CFR improved significantly from a value of 1.6 (abnormal) at baseline to 2.4 (normal) at P6 and was lower at peak augmentation (P8/P9). After LAD revascularization, the FFR increased to 0.85, which is above the ischemic threshold, and the CFR improved to 2.5, which is within the normal range.

DISCUSSION

The Impella 2.5 is a unique device that enables interventional cardiologists to promptly and conveniently implement mechanical circulatory support in the cardiac catheterization lab. The compact design of the catheter allows percutaneous insertion in the lab, thereby eliminating

Figure 2. FFR data demonstrated by hemodynamic significance of an LAD lesion.

the need for major cardiac or vascular surgery in patients who may be too unstable to undergo such surgery. Previous studies have shown that the LV unloading by Impella improves the coronary microcirculatory hemodynamics in nonstenotic arteries. Remmelink's[2] observations in patients undergoing PCI, and Sauren's[3] findings from animal studies, reveal that augmentation of coronary flow occurs at the subendocardial level due to the combination of a direct increase in coronary blood flow and decreased microvascular resistance. Our findings suggest that a similar favorable effect may be seen in stenotic arteries even before revascularization has been achieved. The long-term clinical sequelae of this phenomenon could have profound implications for treating patients with ischemic heart disease, particularly in regards to the selection of periprocedural therapy that would have the most favorable effect on the coronary microcirculation. For example, a recent meta-analysis of intra-aortic balloon counterpulsation (IABP) use during primary PCI for ST-elevation myocardial infarction showed that in randomized controlled studies, IABP therapy had no effect on 30-day survival or the recovery of LV function.[4] Our patient had an ejection fraction of 25% (+10 points) during follow-up at 4 weeks, and he reported a marked improvement in his exertional capacity and CHF symptoms. Similar improvements in EF were reported[5-7] after Impella supported PCI. Ongoing randomized clinical trials comparing the effectiveness of Impella 2.5 and IABP in both high-risk PCI and STEMI patients (Protect II and Recover II) will help to further characterize the effectiveness of this unique percutaneous therapy.

We used a pressure wire in our patient because of the advantages it offers over angiography or intravascular ultrasound in the evaluation of diffusely diseased arteries. When FFR is derived via pressure wire, it allows the functional

TABLE 1. FFR AND CCR DURING IMPELLA SUPPORT					
	Baseline	**P3**	**P6**	**P8/9**	**After PCI**
Pd (mean, mm Hg)	46	47	53	43	58
Pa (mean, mm Hg)	62	62	70	58	68
FFR	0.74	0.76	0.76	0.75	0.85
CFR	1.6	2	2.4	1.9	2.5
Support (L/min)	-	1.3	1.8	2.4	2.4

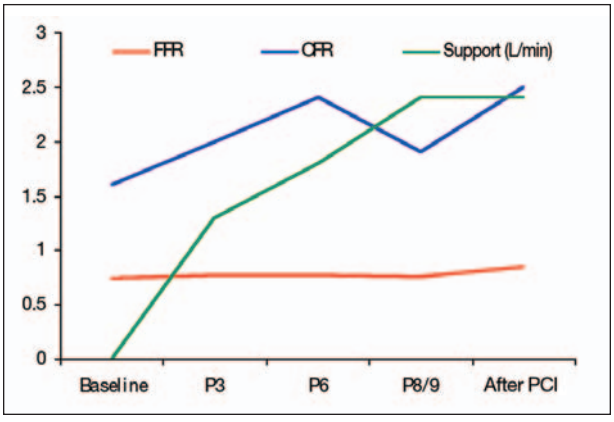

Figure 3. Graph depicting FFR and CCR during Impella support.

assessment of multisegmental atherosclerosis with a very high spatial resolution independent of ventricular loading conditions (such as heart rate, pressure, and contractility). When CFR is derived by pressure wire, it employs the principle of thermodilution to evaluate microcirculatory hemodynamics, and has been validated against CFR that is derived via Doppler (flow) wire. In analysis of our FFR and CFR data, it appears that there was a significant increase in the CFR during the early phases of augmentation, followed by a decline at the maximum support level (P8/P9). This may have been due to the reduction in systemic pressure that was observed after administration of an additional sedative at this point in the procedure. The -12 mm Hg difference in mean aortic pressure between P6 and P8/P9 supports the notion that a decrease in resting blood pressure results in a lower-than-expected CFR reading. It is also possible that not enough time was allowed between measurements at P6 and P8/P9, and that some adenosine receptors were still occupied, resulting in a "partial hyperemic" starting state for the final measurement.

On the other hand, the FFR value remained stable at all levels of augmentation, confirming that this parameter is indeed lesion specific and is not affected by the cardiac output or the changing state of distal circulation (provided maximal hyperemia is achieved). The observation that CFR improved while FFR remained unchanged strongly argues that any improvement in vascular bed reactivity is a direct effect of the Impella device. Also, it is interesting to note that PCI of the LAD resulted in an FFR value that was above the ischemic threshold but yielded a CFR reading that was similar to that achieved by Impella support in the absence of revascularization. To the best of our knowledge, this is the first report of the effects of LV unloading by a cardiac assist device such as the Impella 2.5 on the hemodynamics within a stenotic artery before and immediately after PCI. Additional studies in a larger cohort of patients are warranted to confirm these findings, but the potential for salvage of myocardium before and during Impella-supported PCI is obvious and encouraging. ■

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