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Coronary Collaterals Function and Clinical Outcome Between Patients With Acute and Chronic Total Occlusion

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ABSTRACT

OBJECTIVES This study sought to demonstrate how changes in the collateral function and its clinical significance before and after percutaneous coronary interventions (PCIs) are compared between patients with acute coronary syndrome and total or nearly total occlusions (ATOs) and chronic total occlusions (CTOs).

BACKGROUND The functional relevance of the collateral circulation in patients with ATOs and CTOs has not been fully investigated.

METHODS The pressure-derived collateral pressure index (CPI), myocardial fractional flow reserve (FFRmyo), and coronary fractional flow reserve (FFRcor) at maximum hyperemia induced by intravenous adenosine were evaluated in occluded vessels at baseline, after the PCI, and at 1 year in 23 ATO and 74 CTO patients.

RESULTS The FFRmyo and FFRcor were significantly lower, but the CPI was significantly higher in the CTO than ATO patients at baseline and after the PCI. There were significant increases in the FFRmyo ($p < 0.001$) and FFRcor ($p < 0.001$), whereas there was no significant change in the CPI immediately after the PCI in both ATO and CTO patients. In the CTO patients, a post-PCI FFRmyo <0.90 ($p = 0.01$) and post-PCI CPI <0.25 ($p = 0.033$) were independent predictors of the clinical outcome. Patients with a high post-PCI CPI had better clinical outcomes in CTO patients with a low post-PCI FFRmyo (log-rank $p = 0.009$), but not a high post-PCI FFRmyo (log-rank $p = 0.492$).

CONCLUSIONS Recruitable coronary collateral flow did not regress completely immediately after the PCI both in patients with ATOs and CTOs. Despite good collaterals in CTO patients, aggressive efforts to reduce the ischemic burden might improve the clinical outcome. (J Am Coll Cardiol Intv 2017;10:585–93) © 2017 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Coronary collaterals develop during the gradual progression of coronary lesions and have been recognized as alternative conduits of the blood supply to myocardial areas jeopardized by ischemia (1–4). However, the functional relevance of the collateral circulation has remained controversial for many years. Several clinical trials have suggested a rapid regression of the collateral function after percutaneous coronary intervention (PCI) of chronic total coronary occlusions (CTOs) (5–7), which render the myocardium susceptible to an infarction in the event of a subsequent coronary occlusion (8). However, the rate of myocardial infarctions is smaller than the incidence of reocclusion (7,8). This might indicate that collaterals do not regress completely and remain recruitable (9–11), even immediately after a PCI (12). This argument still has an important clinical implication in the current generation drug-eluting stent (DES) era because of higher rates of stent thrombosis in patients with long lesions and CTOs compared with other patient subsets.

Coronary collaterals also develop after an acute coronary occlusion, particularly in patients with latent collaterals. Previous studies have demonstrated the existence of latent collaterals in human subjects (13), and they may open up immediately after an

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ABBREVIATIONS AND ACRONYMS

ATO = acute coronary syndrome with total or nearly total occlusion

CPI = collateral pressure index

CTO = chronic total coronary occlusion

DES = drug-eluting stent(s)

FFRcor = coronary fractional flow reserve

FFRmyo = myocardial fractional flow reserve

MACE = major adverse cardiac event(s)

Pa = aortic pressure

PCI = percutaneous coronary intervention

Pd = coronary distal pressure

Pv = central venous pressure

Pw = coronary wedge pressure

abrupt coronary obstruction (14). However, the time-course of the change in the collateral assessed by a coronary pressure wire during the restoration of the antegrade flow has not been fully demonstrated in the setting of acute coronary syndrome with total or nearly total occlusions (ATOs) (15) and has not been compared with those of CTOs. Therefore, we sought to investigate the time-behavior of the changes in the collateral function using a coronary pressure wire before and after revascularization between the patients with ATOs and CTOs. We also assessed whether the change in the collateral function after the PCI with a DES would have an influence on the long-term clinical outcome including a subsequent myocardial infarction and restenosis.

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METHODS

PATIENT POPULATION. A total of 111 consecutive patients with chest pain were enrolled in this study between March 2011 and April 2014 after successful recanalization of ATOs (n = 31) or CTOs (n = 80). Of those patients, 97 who had both a baseline assessment of their collateral function and a reassessment after the PCI were finally analyzed. The inclusion criteria were: 1) patients with ATOs or CTOs; 2) spontaneously visible collaterals; and 3) no ventricular aneurysm supplied by the occluded artery. An ATO was defined as follows: 1) a duration of the occlusion of <1 week, as determined from a previous angiogram, the date of a previous myocardial infarction, or the onset of symptoms; and 2) a Thrombolysis In Myocardial Infarction coronary flow grade 0 or 1. A CTO was defined as follows: 1) a duration of the occlusion of >3 months, as determined from a previous angiogram, the date of a previous infarction, or the onset of symptoms; and 2) a Thrombolysis In Myocardial Infarction coronary flow grade 0 or 1. The study protocol had been approved by the institutional ethics committee.

CORONARY INTERVENTION. All procedures were performed with standard interventional techniques. The PCI was performed via the femoral route with a 7-F guiding catheter, or radial route with a 6-F guiding catheter. The antiplatelet therapy and periprocedural anticoagulation followed the standard regimen. Before the procedure, all patients received a loading dose of aspirin (300 mg) and clopidogrel (300 mg or 600 mg). In the catheterization laboratory, anticoagulation with a bolus of unfractionated heparin (75 to 100 U/kg) was administered to achieve an activated clotting time >300 s. Glycoprotein IIb/IIIa receptor inhibitors were left to the discretion of the attending interventional cardiologist. Bare metal stents use was prohibited, so all patients received DESs. After the DES deployment, the patients were maintained on aspirin (100 mg) and clopidogrel (75 mg) for at least 12 months with a longer treatment at the operator's discretion.

ANGIOGRAPHIC ASSESSMENT OF THE COLLATERAL FLOW. The collateral supply was angiographically categorized by 2 methods. First, the collateral circulation was graded according to Rentrop et al. (16) as follows: 0, no visible collateral filling; 1, collaterals filling side branches or minimally filling the occluded artery; 2, retrograde partial filling of an epicardial portion of an occluded artery; and 3, filling of all or most of the recipient artery. Second, the anatomic pathway of the collateral supply was categorized as an epicardial pathway (collateral filling via connections on the epicardial surface), intramyocardial pathway (collateral channels via the myocardium, often through the intraventricular septum), or coexistence pathway (both epicardial and intramyocardial pathways) (17). Two blinded investigators evaluated the angiographic assessment independently, and in the case of disagreement a consensus was obtained.

CORONARY PHYSIOLOGIC ASSESSMENT OF THE COLLATERAL FLOW. Before the coronary intervention and pressure measurements, 200 mg of intracoronary nitroglycerin was administered. The mean aortic pressure (Pa), coronary wedge pressure (Pw), and coronary distal pressure (Pd) were obtained at maximum hyperemia induced by intravenous adenosine (140 mg/kg/min) when each measurement was performed. First, at baseline, the lesion was crossed with a conventional guidewire, and an exchange catheter was advanced distal to the occlusion. A 0.014-inch pressure wire (PressureWire, St. Jude Medical, St. Paul, Minnesota) was calibrated, and passed through the target lesion as distal in the coronary artery as possible. The myocardial fractional flow reserve (FFRmyo) was measured by the ratio of the mean distal coronary pressure to the mean aortic pressure at maximal hyperemia: FFRmyo = Pd/Pa. Second, Pw was assessed after at least a 2-min balloon occlusion unless chest pain or ST-segment elevation on the electrocardiogram was noted, and a total occlusion by the balloon inflation was confirmed by a contrast injection. The coronary fractional flow