Physiologic Evaluation of Microvascular Damage in Culprit Vessel After Successful Primary Percutaneous Coronary Intervention for ST-elevation Myocardial Infarction Patients

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INTRODUCTION

Comprehensive physiologic assessment can provide additional information about microvascular function in coronary artery disease. Three physiologic indices, fractional flow reserve (FFR), index of microcirculatory resistance (IMR) and coronary flow reserve (CFR) can be easily measured in culprit vessel of ST elevation myocardial infarction (STEMI) patient after successful primary revascularization. Here, we reports 2 cases of STEMI patients who underwent comprehensive physiologic assessment in order to determine microvascular function after successful primary revascularization.

CASE 1

A 56 year old man presented with a 4 hours of ongoing chest pain and admitted via emergency department for
further evaluation and management. He was non-smoker and he had been treated with hypertension and dyslipidemia in local clinic. He had been taking aspirin 100mg, rosuvastatin 10 mg, and olmesartan 40 mg once a day. He had no family history of cardiovascular disease. His initial electrocardiogram (EKG) showed ST segment elevation in lead II, III, aVF, accompanied with reciprocal changes in V4-6 with ST depression (Fig. 1).

In coronary angiography, total thrombotic occlusion with TIMI 0 flow in mid right coronary artery (RCA) without any measurable collateral flow from non-culprit vessels was notified and the culprit lesion was successfully treated with thrombus suction and stent implantation (Synergy 4.0×28 mm) (Fig. 2). The door to balloon time was 55 minutes. The final angiogram showed TIMI 3 flow without residual stenosis (Fig. 3).
Fig. 4. Post–PCI Physiologic assessment in culprit vessel in Case 1. CFR was depressed to 1.1 and IMR was elevated up to 75 U.

Fig. 5. Cardiac MR 5 days after primary PCI in Case 1. (A) Intramyocardial hemorrhage was seen in T2 weighted image, (B) Microvascular obstruction in Late gadolinium enhancement.
Post-percutaneous coronary intervention (PCI) physiologic study in culprit vessel was performed. FFR under maximal hyperemia using intracoronary nicorandil 2 mg bolus injection was 0.97, however, CFR was depressed to 1.1 and IMR was elevated up to 75 U which suggested the presence of overt microvascular damage in culprit vessel territory (Fig. 4).

Cardiac magnetic resonance imaging (MRI), which was performed 5 days after primary PCI showed intramyocardial hemorrhage in T2 weighted image and microvascular obstruction was also notified in infarcted area after late gadolinium enhancement (Fig. 5).

**CASE 2**

A 41 year old man presented with a 1 hour history

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**Fig. 6. Initial EKG in Case 2. There is ST elevation in II, III, aVF with reciprocal changes with V2–4.**

**Fig. 7. Coronary angiography in Case 2. Thrombotic total occlusion was seen in mid right coronary artery (RCA).**

**Fig. 8. Coronary angiography after Successful Revascularization in Case 2. We performed PCI with stent implantation, Synergy 3.5×38 mm.**
of ongoing resting chest pain and admitted via emergency
department for further management. He was current
smoker and he had no remarkable medical history. His
initial EKG showed ST segment elevation in II, III, aVF with
reciprocal changes with ST depression in V2-4 (Fig. 6).

Initial coronary angiography showed thrombotic total
occlusion with TIMI 0 flow in mid-RCA without any
measurable collateral flow from non-culprit vessels. The
culprit vessel was successfully revascularized after
thrombus suction and stent implantation (Synergy 3.5x38
mm). The final angiogram showed TIMI 3 flow without
residual stenosis (Fig. 7, 8). We also performed post-PCI
comprehensive physiologic assessment in culprit vessel.

FFR with use of nicorandil was 0.98, CFR was 2.2 and
IMR was 24 U. The results from physiologic assessment
implied well preserved microvascular function and
non-significant flow limitation through epicardial
 coronary artery as well as microvascular beds (Fig. 9).

Cardiac MRI was taken 2 days after primary PCI and
showed difference pattern with case 1. There were no
intra-myocardial hemorrhage and edema in T2 weighted
image and there was only subtle enhancement in infarcted
area and we couldn’t find microvascular obstruction in
late gadolinium enhancement image (Fig. 10).

![Graph showing FFR and CFR values](image)

**Fig. 9.** Post-PCI Comprehensive Physiologic Assessment in Culprit Vessel in Case 2. CFR was 2.2 and IMR was 24 U.
DISCUSSION

Previously, Fearon et al.\(^1\) showed that elevated IMR value, more than 40 U, is the independent predictive value associated with worse clinical outcome in STEMI patients. And also elevated IMR at the time of STEMI represents larger degree of myocardial and microvascular damage and less recovery of left ventricular function that can be assessed by cardiac enzyme, cardiac MRI, echocardiography, or positron emission tomography.\(^2,3\) Another report from our group presented that not only elevated IMR, but also low CFR was significantly associated with worse clinical outcome in patients with stable coronary artery disease and functionally insignificant epicardial coronary stenosis.\(^4\) These findings emphasize the importance of comprehensive physiologic assessment in patients with coronary artery disease. Similarly another reports from Korea also presented the significant association between microvascular damage, which was defined with depressed CFR as well as elevated IMR, and long-term outcomes in STEMI patients after successful revascularization.\(^5,6\) In this case report, we presented the results from the comprehensive physiologic assessment in culprit vessel after primary PCI in STEMI patients and validated the results using cardiac MR findings. There is acute and overt inflammatory change in myocardial cell and vasculature, especially in acute phase of myocardial infarction. Nevertheless, the reproducibility of IMR measurement and prognostic implication in patients with STEMI, even in the acute phase has been presented by previous studies.\(^7,9\)

Previous studies showed several mechanisms cause overt microvascular dysfunction. Individual susceptibility (e.g. underlying comorbidity, lack of preconditioning, collateral recruitability), degree and extent of ischemic injury and reperfusion injury cause difference in physiologic findings in these two cases. We think that degree of ischemic injury, especially duration of ischemic event (i.e. symptom to door time), is the one of the major causes of difference in these two cases.

In real world practice, most physicians do not perform the comprehensive physiologic assessment after revascularization of epicardial coronary stenosis. We wanted to emphasize the importance of comprehensive physiologic assessment for microvascular function by presenting these 2 cases with substantially different physiologic findings, even after the successful revascularization for the epicardial coronary stenosis.

In conclusion, comprehensive physiologic evaluation at
the time of primary PCI at STEMI is feasible, and low CFR and high IMR highly suggests overt microvascular damage even after the successful revascularization of culprit vessel.

REFERENCES


