International Journal of Cardiology 287 (2019) 48–49

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Letter to the Editor

What would we find behind the darkness to predict patients' fate?

Satoru Mitomo [a], Ozan M. Demir [b], Sunao Nakamura [a,*]

[a] Cardiology Department, New Tokyo Hospital, Chiba, Japan
[b] Department of Cardiology, Hammersmith Hospital, Imperial College Healthcare Trust, London, UK

ARTICLE INFO

Article history:
Received 14 March 2019
Accepted 4 April 2019
Available online 8 April 2019

Keywords:
Non-ST segment elevation myocardial infarction
Plaque composition
Necrotic core
Myocardial tissue-level perfusion

Percutaneous coronary intervention (PCI) is now the most common strategy for treating acute coronary syndrome (ACS) [1]. However, PCI still fails to achieve Thrombolysis In Myocardial Infarction (TIMI)-3 flow in 12–30% of cases [2,3], and moreover, even achieving successful revascularization with an appropriate epicardial coronary flow, sometimes operators could encounter a difficulty to obtain complete myocardial tissue-level reperfusion. The incidences have been reported to be associated with increased cardiac mortality [4,5].

Impaired coronary flow recognized as slow-flow/no-reflow phenomenon during PCI has been previously reported to be associated with lesion characteristics including large plaque burden, lipid-rich plaque, necrotic core or positive vessel remodeling [6]. After balloon inflation or stenting, because of mechanical fragmentation on these plaque, plaque composition especially in the necrotic core including cholesterol clefts, foam cells, microcalcifications would be exposed toward lumen and delivered distally [7], resulting in microembolization impairing myocardial tissue-level perfusion. Consequently, these phenomenon could be evaluated with TIMI flow grade (TFG), TIMI myocardial perfusion grade (TMPG), or TIMI myocardial perfusion frame count (TMPFC) angiographically.

In this study, Gan Yang et al. found that, in NSTEMI patients, intravascular ultrasound (IVUS) findings at the culprit lesion of echo-attenuated plaques and necrotic core (NC) % volume were independent predictors for impaired post-PCI TMPG and TMPFC, and the incidences could be optimally predicted with NC% > 20%. These findings are remarkable as; 1) the authors adopted iMap-IVUS for evaluating plaque composition, which has extremely high correlation with the results of histopathological examination, and 2) the authors evaluated the impact of plaque composition in the non-ST segment elevation myocardial infarction (NSTEMI) culprit lesions on not only epicardial perfusion (i.e. TFG) but also myocardial tissue-level perfusion (i.e. TMPG and TMPFC). However, there are also several important plaque characteristics which have not been evaluated in the current study.

Firstly, the plaque morphology should be considered. The author evaluated NC% in the plaque volume, which could be calculated with NC area in the cross-sectional area derived by iMap-IVUS automatically and then using the Simpson's rule. However, even with the same NC%, their behavior should be different between the different plaque morphology (i.e. focal vs. diffuse, or concentric vs. eccentric) and NC distribution in the plaque (i.e. spotty vs. diffuse), considering plaque deformation or fragmentation after balloon inflation or stenting.

Secondary, thickness of fibrous cap on the NC should be also evaluated. Thin-cap fibroatheroma (TCFA) has been well known as vulnerable plaque, which could be associated with no-reflow phenomenon after stenting [8]. Hong et al. defined TCFA using vertical histology IVUS as a NC ≥ 10% of plaque area in at least three consecutive frames without overlying fibrous tissue in the presence of ≥40% plaque burden, and then found that TCFA within culprit lesions of acute coronary syndrome (ACS) patient was significantly more common in no-reflow phenomenon compared with normal-reflow after stenting [6]. Considering mechanical injury on the plaque during PCI procedure, even the same NC% in the lesion to be treated, thickness of fibrous cap on the NC should have impact on the protrusion of plaque composition toward the lumen and post-PCI myocardial perfusion as well.

In clinical setting, if operators could detect high-risk plaque predicting slow-flow/no-reflow phenomenon, it should be translated to our decision making of PCI strategy how we could prevent or minimize risk of the incidence. Based on the pathophysiological mechanism, utilization of distal protection devices could be potentially considered. However, the efficacy of the device has not been proved sufficiently, therefore, the current guidelines do not recommend routine use of the

DOI of original article: https://doi.org/10.1016/j.ijcard.2019.02.010.
* Corresponding author at: New Tokyo Hospital, Wanagaya 1271, Matsudo, Chiba, Japan.
E-mail address: boss0606@pluto.plala.or.jp (S. Nakamura).

https://doi.org/10.1016/j.ijcard.2019.04.017
0167-5273/

device for STEMI patients [9]. On the other hand, Hibi et al. reported the efficacy of the device for ACS patients [10]. They evaluated two hundred patients with ACS who had native coronary artery lesions and attenuated plaque with longitudinal length ≥ 5 mm on pre-PCI IVUS, which were randomly assigned to undergo PCI with distal protection device or conventional treatment. In this study, utilization of distal protection device was associated with reduced incidence of no-reflow phenomenon and fewer adverse cardiac events including cardiac death, cardiac arrest, and cardiogenic shock after PCI compared with the conventional strategy. The results may suggest that, if the lesions with high-risk plaque could be appropriately selected, the device may effectively reduce or minimize the slow-flow/no-reflow phenomenon. In this regards, stratification of high-risk ACS patients according to the NC% (≥20%) could contribute to decision making of PCI strategy. In order to translate these findings of the current study to our daily practice, it would be required to develop on-line software facilitating automatic plaque characterization intraprocedurally.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

References

[1] W.D. Weaver, R.J. Simes, A. Betriu, C.L. Grines, F. Zijlstra, E. Garcia, et al., Comparison of primary coronary angioplasty and intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review, JAMA 278 (1997) 2093–2098.

[2] R.N. Piana, G.Y. Paik, M. Moscucci, D.J. Cohen, C.M. Gibson, A.D. Kugelmass, et al., Incidence and treatment of 'no-reflow' after percutaneous coronary intervention, Circulation. 89 (1994) 2514–2518.

[3] I. Morishima, T. Sone, S. Mokuno, S. Taga, A. Shimauchi, Y. Oki, et al., Clinical significance of no-reflow phenomenon observed on angiography after successful treatment of acute myocardial infarction with percutaneous transluminal coronary angioplasty, Am. Heart J. 130 (1995) 239–243.

[4] C.M. Gibson, C.P. Cannon, S.A. Murphy, S.J. Marble, H.V. Barron, E. Braunwald, Relationship of the TIMI myocardial perfusion grades, flow grades, frame count, and percutaneous coronary intervention to long-term outcomes after thrombolytic administration in acute myocardial infarction, Circulation. 105 (2002) 1909–1913.

[5] J. Pu, S. Ding, H. Ge, Y. Han, J. Guo, R. Lin, et al., Efficacy and safety of a pharmaco-invasive strategy with half-dose alteplase versus primary angioplasty in ST-segment-elevation myocardial infarction: EARLY-MYO trial (EARLY routine catheterization after alteplase fibrinolysis versus primary PCI in acute ST-segment-elevation MYOcardial infarction), Circulation. 136 (2017) 1462–1473.

[6] Y.J. Hong, M.H. Jeong, Y.H. Choi, J.S. Ko, M.G. Lee, W.Y. Kang, et al., Impact of plaque components on no-reflow phenomenon after stent deployment in patients with acute coronary syndrome: a virtual histology-intravascular ultrasound analysis, Eur. Heart J. 32 (2011) 2059–2066.

[7] A. Nair, B.D. Kuban, E.M. Tuzcu, P. Schoenhagen, S.E. Nissen, D.G. Vince, Coronary plaque classification with intravascular ultrasound radiofrequency data analysis, Circulation. 106 (2002) 2200–2206.

[8] A. Tanaka, T. Imanishi, H. Kitabata, T. Kubo, S. Takarada, T. Tanimoto, et al., Lipid-rich plaque and myocardial perfusion after successful stenting in patients with non-ST-segment elevation acute coronary syndrome: an optical coherence tomography study, Eur. Heart J. 30 (2009) 1348–1355.

[9] B. Ibanez, S. James, S. Agewall, M.J. Antunes, C. Bucciarelli-Ducci, H. Bueno, et al., 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC), Eur. Heart J. 39 (2018) 119–177.

[10] K. Hibi, K. Kozuma, S. Sonoda, T. Endo, H. Tanaka, H. Kyono, et al., A randomized study of distal filter protection versus conventional treatment during percutaneous coronary intervention in patients with attenuated plaque identified by intravascular ultrasound, JACC Cardiovasc. Interv. 11 (2018) 1545–1555.