

Editorial Comment**Editorial Yorum****Slow coronary flow**

Yavaş koroner akım

Mehmet Agirbasli, M.D.

Marmara University Faculty of Medicine, Department of Cardiology, Istanbul

The main aim of primary angioplasty is to provide instant and continuous normal blood flow through occluded coronary arteries. Although it was previously believed that an unblocked artery was required for a successful percutaneous coronary intervention (PCI), clinical and experimental studies have confirmed that the issue is more complicated. As the importance of microcirculation for clinical results becomes much more understood, the importance of primary angioplasty and reperfusion therapy increases. Reperfusion therapy following primary PCI or angioplasty was reported to be more effective compared to fibrinolytic therapy to preserve the microvascular structure, and less segmental wall motion abnormalities were observed following primary PCI even in patients with normal coronary flow.^[1] An effective reperfusion therapy should also improve tissue perfusion.^[2] The flow should be normalized (TIMI 3) following a successful PCI; however even this flow is not sufficient for a complete improvement in the myocardium. As commonly seen, continuing chest pain despite unblocked epicardial arteries following the procedure, ST-segment elevation, low rate of coronary flow and low penetration of contrast agent to the infarction site indicates an impaired microcirculation in patients undergoing PCI due to acute coronary syndrome.

In recent years, significant researches regarding the biological basis of slow coronary flow (SCF) have been conducted in Turkey. In this issue of Archives of the Turkish Society of Cardiology, Sen et al.^[3] presented an association between SCF and serum gamma-glutamyl-transferase activity.

Impaired microcirculation during acute myocardial infarction results from both embolism of the ruptured

plaque and thrombi of platelets and fibrin. Other causes are irreversible and depend on absence of or delayed formation of tissue-level perfusion. Although one of the causes has been known for a long time to be SCF, effective treatments have not yet been developed.^[4] SCF following angioplasty or the “no-reflow” phenomenon following epicardial reperfusion is still a distress for interventional cardiologists.^[5] In previous studies, congestive heart failure and left ventricular dysfunction which were seen frequently following reperfusion therapy were reported to be associated with slow coronary flow observed in one third of the patients.^[6,7] Similarly, the mortality rate at 6 years was found to be four times higher in patients with SCF documented by angiogram (TIMI ≤ 2 flow) compared to the patients with normal flow (TIMI 3) (37% and 10% respectively, $p=0.002$).^[8]

Slow coronary flow is an angiographic finding and is designed with the number of frames obtained from the beginning to the end point, as the contrast agent passes through the coronary vessels.^[9] The TIMI frame count (TFC) method was developed by Gibson et al.^[9] As well as an angiographic finding, slow coronary flow is also an indicator of existing atherosclerotic burden. Recently, Yilmaz et al.^[10] reported that patients with SCF were more prone to develop metabolic syndrome. Furthermore, Yildiz et al. from Harran University reported an association between SCF and decrease in paraoxonase and antioxidant capacity. On the other hand, Turkoglu et al.^[12] found no difference between diabetics and controls in respect of coronary flow. In another study which was conducted by Abaci et al.^[13] to address these controversies in the literature, it was found that

TIMI frame count, an indicator of SCF, could change depending on nitrate use and heart rate, despite the findings of Gibson et al. in the TIMI-4 study.

Studies conducted using glycoprotein IIb/IIIa inhibitors demonstrated the clinical results of SCH more clearly. Effective inhibition of platelet aggregation in patients with acute coronary infarction may not only be beneficial in preventing patent epicardial artery and the “no-reflow” phenomenon, but also in improving microcirculation. Early studies on this subject demonstrated that normalization of ST-segment elevation, as an indicator of tissue perfusion after acute myocardial infarction, was faster in patients receiving both fibrinolytic (t-PA, streptokinase) and antiplatelet (lamifiban) compared to those receiving a fibrinolytic alone.^[14,15] Effective antiplatelet drug use is a major component for complete reperfusion; however scheduling of antiplatelet drug use is critical. Studies conducted using glycoprotein IIb/IIIa inhibitors have also markedly shown that early use of drugs is crucial for efficiency.^[16] New antiplatelet agents may be useful.

Gibson et al.^[17] developed a new grading system to evaluate microvascular integrity following myocardial reperfusion therapy. The purpose of this system is to evaluate the entrance of contrast agent into the myocardium following the injection of contrast agent in to the epicardium (TMP: tissue myocardial perfusion). Similar to epicardial flow, the mortality rate was found to be lower in patients with normal microcirculation (TMP 3) following reperfusion therapy compared to those with impaired microcirculation.^[17] Kaya et al.^[18] also confirmed the findings of Gibson et al. and suggested that tissue perfusion was a better indicator for long-term mortality compared to coronary flow in 130 patients who underwent primary PCI for acute myocardial infarction. On the other hand, Seyfeli et al.^[19] observed a poor relationship between myocardial vitality and the extent of myocardial perfusion (myocardial blush grade-MBG).

Effective antiplatelet treatment, technical developments in PCI, distal embolic protection catheters or devices during PCI are among the treatment approaches both in the normalization of coronary flow and improvement of tissue perfusion.

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