Coronary slow flow: Electrophysiologic evidence of ischemia?

Zehir et al. (1) found that Tpe (T-wave peak to end time) Interval and Tpe/QT ratio were prolonged in patients with Coronary Slow Flow Phenomenon (CSFP), published in this issue of Anatol J Cardiol. The coronary slow flow phenomenon is an angiographic finding that is characterized by slow progression of the contrast seen during coronary angiography. It can be present in a diverse population from chest discomfort to ST-segment elevation myocardial infarction. The pathogenesis of this phenomenon remains unknown, and it is not clear if it is a primary or secondary phenomena. This phenomenon has been seen in various clinical settings including non-cardiac chest pain, infection and critically ill patients. The incidence of this syndrome thought to be approximately 1% among patients who undergo coronary angiography, particularly in those presenting with acute coronary syndrome (2).

It is probably more prevalent than as described, since many patients could be asymptomatic or have not undergone coronary angiography. It usually occurs in all coronary territories, and therefore, resolved coronary thrombus as the underlying mechanism cannot explain this phenomenon. Transient endothelial dysfunction is probably the main underlying mechanism for its occurrence. In this paper (1), the authors studied the effect of slow flow on the peak to the end of electrocardiographic T wave (Tpe) and its ratio to QT, which was prolonged in these patients. These observations have been seen in patients with acute coronary syndrome (3) and are related to ischemia driven imbalance in repolarization. The fact that these abnormalities can be observed in patients with slow flow coronaries suggests that slow flow is not a benign condition because it causes inhomogeneity in repolarization as the potential trigger for arrhythmias. Small vessel disease, endothelial dysfunction, subclinical atherosclerosis, and inflammation are believed to play a role in this phenomenon (4-6).

There are not many studies that have evaluated the changes in ECG parameters in patients with coronary slow flow phenomena. Atak et al. (7) found QT prolongation in patients with slow flow consistent with the negative effect of this phenomenon on myocardial cells. In this paper, TPe was prolonged. TPe/QT ratio is an index of arrhythmogenesis (8). Therefore, the observation of this study suggests that slow flow phenomena can potentially lead to arrhythmias. Treatment of these patients remains controversial. Intracoronary vasodilator and adenosine will usually normalize flow. However, the beneficial effect of giving intracoronary adenosine on the clinical outcome of these patients has not been studied. Because of the rarity of this syndrome and its occurrence in diverse clinical setting, making a definite conclusion about the pathophysiology of this phenomenon is difficult.

This is an important study showing that slow flow needs to be considered as an ischemia inducing phenomena. However, we need to be cautious in interpreting this study. The authors involved small number of patients, which is a major limitation of their study. However, p values were all statistically significant, suggesting that their results are valid. Further research is needed to understand the pathogenesis of slow flow and the pathogenies of this phenomenon.

M. Reza Movahed
Arizona CareMore Regional Cardiology, Director and Professor of Medicine University of Arizona College of Medicine, Tucson, AZ-USA

References
1. Zehir R, Karabay CY, Kalayci A, Akgün T, Kılıçgedik A, Kırma C. Evaluation of Tpe interval and Tpe/QT ratio in patients with slow coronary flow. Anatol J Cardiol 2015; 15: 00.00. [CrossRef]
2. Goel PK, Gupta SK, Agarwal A, Kapoor A. Slow coronary flow: a distinct angiographic subgroup in syndrome X. Angiology 2001; 52: 507-14. [CrossRef]
3. Eslami V, Safi M, Taherkhani M, Adibi A, Movahed MR. Evaluation of QT, QT dispersion, and T-wave peak to end time changes after primary percutaneous coronary intervention in patients presenting with acute ST-elevation myocardial infarction. J Invasive Cardiol 2013; 25: 232-4. [CrossRef]
4. Cin VG, Pekdemir H, Camsar A, Çiçek D, Akkus MN, Parmaksiz T, et al. Diffuse intimal thickening of coronary arteries in slow coronary flow. Jpn Heart J 2003; 44: 907-19. [CrossRef]
5. Li JJ, Qin XW, Li ZC, Zeng HS, Gao Z, Xu B, et al. Increased plasma C-reactive protein and interleukin-6 concentrations in patients with slow coronary flow. Clin Chim Acta 2007; 385: 43-7. [CrossRef]
6. Camsar A, Pekdemir H, Çiçek D, Polat G, Akkus MN, Döven O, et al. Endothelin-1 and nitric oxide concentrations and their response to exercise in patients with slow coronary flow. Circ J 2003; 67: 1022-8. [CrossRef]
7. Atak R, Turhan H, Sezgin AT, Yetkin O, Senen K, Ileri M, Şahin O, et al. Effects of slow coronary artery flow on QT interval duration and dispersion. Ann Noninvasive Electrocardiol 2003; 8: 107-11. [CrossRef]
8. Gupta P1, Patel C, Patel H, Narayanaswamy S, Malhotra B, Green JT, et al. Tp(e)/QT ratio as an index of arrhythmogenesis. J Electrocardiol 2008; 41: 567-74. [CrossRef]