Coronary slow flow: Benign or ominous?

To the Editor,

We read the article by Sadrameli et al. (1) entitled "Coronary slow flow: Benign or ominous?" published in Anatolian Journal of Cardiology 2015; 15: 531-5 with great interest. The authors are to be praised for their well-versed study that investigated the clinical features, coronary risk factors, and clinical outcomes relating to 217 patients who had a confirmed diagnosis for coronary slow flow phenomenon (CSFP). This pathology relates to delayed distal vessel opacification as seen on coronary angiography due to reduced blood flow in the absence of significant coronary disease (2). However, we feel there are a number of issues that require further clarification.

First, the authors have not mentioned the number of patients excluded from their initial selection of CSFP patients. Although the exclusion criteria are stated, no clarification is given on deselecting patients with congenital heart disease or specific

arrhythmias, which may contribute to CSFP. Moreover, it is not clear which combination of anti-ischemia and anti-anginal drugs have been prescribed in effectively treating the variable presentations of CSFP, as listed in Table 2 (1). Furthermore, whilst we appreciate that echocardiography is a reliable and reproducible tool for assessing left ventricular function (LVF), it remains sensitive to patient echogenicity (3). It would have been interesting to see if the authors experienced any technical difficulties in evaluating LVF due to poor echocardiographic imaging and whether they attempted to evaluate LVF with the application of contrast-enhanced echocardiography, which would be a more sensitive imaging modality (3).

Second, the authors only used angiography to determine the diagnosis of CSFP according to a myocardial infarction frame count (MIFC) above 27 frames for all vessels, following correction for the length of the left anterior descending artery (1). A study by Nie et al. (4) focused on angiographic features of coronary arteries between control vs CSFP patients. They concluded that CSFP compared with normal subjects was associated with a higher tortuosity index and greater number of distal branches in coronary arteries at end-systole; therefore, the role of coronary angiography may be important to determining the anatomical properties of coronary arteries in CSFP patients compared to an equal selection of normal non-CSFP subjects.

Lastly, the authors could have explored other important demographic variables such as body mass index (BMI) and QT interval ratio, where studies have shown a potential link to CSFP. For instance, Tenekecioğlu et al. (5) showed that QTd, Tp-Te interval, and Tp-Te/QT ratio were markedly prolonged in these patients on electrocardiogram (ECG). This will predispose to future events like angina pectoris, myocardial infarction, and life-threatening arrhythmias. Perhaps an ECG may have been requested to evaluate QT interval relationship especially when 36 patients underwent repeat coronary angiography.

Overall, we praise the authors' useful insight into CSFP; however, we feel a comparative cohort study with normal vs. CSFP subjects, detailed angiography readings, and QT interval ratio measurements may have yielded further information in understanding the pathogenesis of this disease.

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Accepted Date: 11.11.2015

DOI:10.14744/AnatolJCardiol.2015.6777