

## Author's Reply

To the Editor,

We appreciate the comments and suggestions made by the authors of the Letter-to-the-editor entitled "Renal dysfunction as a marker of increased mortality in patients with pulmonary thromboembolism" (1), and we would like to thank them for their insightful comments regarding several aspects of our paper published in *Anatol J Cardiol* 2015; 15: 938-43 (2). We have some remarks and specifications to make.

We studied prospectively the factors associated with mortality in 404 consecutive patients with non-high-risk pulmonary thromboembolism followed up for 2 years. The highest 2-year mortality rate (20%) was recorded in patients with moderate renal dysfunction associated with right ventricle dysfunction. We agree that mortality risk stratification in this population is very important and therefore could benefit from further risk stratification.

Chronic kidney disease is associated with increased cardiovascular morbidity and mortality. Renal impairment is a common and independent predictor of stroke and systemic embolism (3). For example, 2 years ago, a novel score for thromboembolic risk (R2CHADS2) in non-valvular atrial fibrillation was proposed (4). This index includes creatinine clearance <60 mL/min/L, and it was shown to have higher discriminating capacity of thromboembolic risk (4). In our study there were no significant differences between the number of patients with atrial fibrillation in non-survivors versus survivors [n=15(45.5%) versus n=138(37.2%); p=0.349]. But thromboembolic risk parameters included in CHA2DS2-VASc like diabetes mellitus, age  $\geq 75$  years, previous deep thrombophlebitis were significantly more frequently in non-survivors versus survivors (see Table 1). Therefore, in our study, thromboembolic risk scores assessment in non-survivors versus survivors is on-going.

In the non-survivors group, there were no patients with cancer; but these patients were older, more frequently females, and with pericardial effusion (known as prognostic factor in patients with pulmonary hypertension) and lower acceleration time (as marker of pulmonary hypertension). In addition, in non-survivors, glomerular filtration rate was significantly lower than in survivors (51.85 $\pm$ 19.08 mL/min/1.73 m<sup>2</sup> versus 71.65 $\pm$ 23.21 mL/min/1.73 m<sup>2</sup>; p=0.000). The causes of death in these patients were related in majority to the more advanced renal and cardiovascular disease (please see Table 1 for GFR, troponin, and BNP values, which are significantly higher in non-survivors than survivors). They also have had more comorbidity, such as diabetes mellitus, coronary heart disease, previous deep thrombophlebitis or varicose veins, COPD, and/or heart failure. Right ventricle dysfunction obviously was an important factor that contributed to a fatal prognostic in these patients. Unfortunately, few autopsies have been performed; therefore, possible recurrences of fatal venous thromboembolism were not diagnosed. The diverse etiologies of death might be more attentively further investigated in our study.

In conclusion, we totally agree that renal dysfunction could be a predictor of both early and long-term increased mortality in patients with acute pulmonary thromboembolism, and also

that this heterogeneous population with non-high-risk pulmonary thromboembolism must be evaluated in further carefully designed clinical studies.

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## 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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## Author's Reply

To the Editor,

We thank the authors of the letter for their valuable comments. In our study entitled "Coronary slow flow: Benign or ominous?" published in *Anatolian Journal of Cardiology* 2015; 15: 531-5 (1), the focus was on the evaluation of characteristics of patients presenting with coronary ischemic symptoms and who happened to only have coronary slow flow phenomenon in coronary angiography; the goal was to understand the natural history of these patients. For this reason, the patients who were admitted for catheterization due to causes other than coronary symptoms were excluded.

Congenital patients have their own specific underlying cardiac pathophysiology, with abnormal coronary anatomies; therefore, they were not taken into account in our study. None of the evaluated patients suffered from specific arrhythmias.

Prescribed drugs might have varied based on individual patient's conditions, but the core components remained constant in the majority of cases.

Regarding echocardiography, echogenicity did not really impose a problem that necessitated the use of contrast material or other modalities, and global left ventricular function was determined in different echocardiographic planes.

Last but not least, we agree with the comment that evaluation for further characteristics, including those parameters mentioned by the authors of the letter, could be related and important in patients with coronary slow flow phenomenon and should become the subject of future studies.

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