

Despite its high prevalence and well-described role in the pathogenesis of cardiovascular disease and its relatively easy treatment, OSA remains largely underdiagnosed. This is a problem especially in patients at a high cardiovascular risk. In a recent study conducted in a population of patients with diabetes mellitus, only 4.2% of the patients were treated for OSA, while the disease was diagnosed in twice as many patients (8.5%); however, the symptoms of daytime sleepiness were reported by as much as 16% of the entire study population. Only approximately 1 in 3 patients with daytime symptoms previously underwent a diagnostic evaluation (4).

The editorial comment on our article "OSACS score - a new simple tool for identifying high risk for Obstructive Sleep Apnea Syndrome based on clinical parameters" provides additional view on some issues addressed in the paper and considers important topics. New OSA risk scores such as OSACS are capable of improving the early diagnosis of the disease. Questionnaires such as the Berlin questionnaire or Epworth Sleepiness Scale were proven to be useful and cost effective. They are also helpful in everyday clinical practice where more advanced screening methods including polysomnography are less available. As it was emphasized in the article, the OSACS score is different from the other scales because it is the first one to be solely based on objective clinical parameters and not subjective symptoms. Moreover parameters included in the OSACS score such as left ventricular mass index, diastolic diameter, intraventricular septal thickness, blood pressure, and body mass index are routinely obtained in acute coronary syndrome patients in whom the scale was addressed. Calculation of the OSACS score does not require any additional diagnostic work-up from the physician; therefore, it is easy to perform and use.

As the Editors stated, the OSACS score needs validation in an external cohort, maybe also in a general population, not only patients with acute coronary syndrome. The external validation would improve the significance of the score and confirm its utility. Nevertheless, all the parameters used in the score were previously described in other studies to be associated with OSA. The first factor, obesity and hypertension (particularly resistant), are one of the most often described OSA predictors, and an increase in body mass is associated with the rising severity of OSA. Additionally, left ventricular geometry is altered in OSA. Some studies show that OSA affects ventricular geometry irrespective of obesity (5). Increased blood pressure values were also described to be independently associated with OSA in numerous studies.

In conclusion, the OSACS score is a non-invasive, simple, and promising tool that may be useful in identifying OSA in acute coronary syndrome patients and in the future, possibly other groups of patients. After external validation, the OSACS score may help in the wider recognition of OSA as a non-classical risk factor. It may help improve the prognosis of patients and therefore reduce the burden of cardiovascular disease.

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Effect of percutaneous mitral balloon valvuloplasty on right ventricular functions in mitral stenosis: Short- and mid-term results

To the Editor,

We read the original investigation entitled "Effect of percutaneous mitral balloon valvuloplasty on right ventricular functions in mitral stenosis: Short- and mid-term results" by Inci et al. (1) published in the *Anatol J Cardiol* 2015; 15: 289-96 with great interest. We would like to touch on some points regarding this article.

A prospective study was conducted in 61 patients (age: 42.7±11.6 years) with isolated rheumatic mitral valve stenosis who underwent percutaneous mitral balloon valvuloplasty (PMBV). The patient population consisted of individuals with notable advanced ages. Although the authors stated clinical, echocardiographic, or angiographic evidence of coronary artery disease as exclusion criteria, there are some unclarified points. Firstly, what percentage of the patients underwent coronary angiography? Furthermore, it should be stated whether the patients with non-critical coronary artery disease were also included in the study.

Secondly, it should also be stated in the text that the clinical characteristics of the patients such as heart rate and systolic and diastolic blood pressures were similar before and after the procedure at the 3rd and 12th months. Otherwise, differences in these parameters will probably affect echocardiographic measurements (deceleration time, E peak, A peak, mean gradient, etc.) (2). In addition, pulmonary flow velocity, right ventricular filling fraction, and A wave, which also reflects right ventricular filling, have already been found to be increased, and right ventricle isovolumetric relaxation time has been found to be prolonged in hypertensive patients. The reduction of pulmonary valve acceleration time index in hypertension should also be noted (3).

Thirdly, mitral valve area assessment using the pressure half-time (PHT) method is not recommended, especially in the early period after

PMBV (4). Because the creation of an iatrogenic atrial septal defect during transseptal catheterization may contribute to a poor agreement between Doppler and Gorlin data after PMBV (5). How do the authors explain the similarity between valve area measurements obtained via the PHT method and planimetry in the early post-PMBV period?

Finally, when increased left atrial diameter (mean, 48 mm; range, 42–57 mm) in the patient population is considered, the development of an arrhythmia such as atrial fibrillation (at least paroxysmal atrial fibrillation) in the 1-year follow-up is highly likely. It is also unclear whether exclusion was performed in the follow-up period because of the development of such an arrhythmia or any other reason. We hope that the authors are willing to comment on these issues.

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DOI:10.5152/AnatolJCardiol.2015.6452

Author's Reply

To the Editor,

We thank you for your interest and positive reviews on our article entitled "Effect of percutaneous mitral balloon valvuloplasty on right ventricular functions in mitral stenosis: Short- and mid-term results" (1) published in the *Anatol J Cardiol* 2015; 15: 289-96.

Firstly, the diagnosis of rheumatoid diseases may have been delayed in some patients because of individual and sociocultural differences in the patient group in the current study and also because the indication for intervention may have been delayed in some patients. As the risk of coronary artery disease (CAD) accompanying this age group is high, we added CAD to the exclusion criteria. At the beginning of the study, the patients were evaluated, and coronary angiography was performed in eight patients. The angiograph was found to be consistent with CAD in six patients, and these were not included in the study. CAG was indicated in the one-year follow-up of five patients, and severe vascular occlusion was detected in three of them; they were excluded from the study. A total of four patients who had non-critical stenosis were included to the study. As these patients would not directly affect the study data, they were not further mentioned in the text.

Secondly, as you have mentioned, the effects of parameters such as systolic and diastolic blood pressures and heart rate on many echocardiographic data are inevitable. Thus, homogeneity was achieved in basal and follow-up parameters. There is no statistically significant difference between the basal and follow-up values in patients included in the study.

Thirdly, as mentioned in the last ACC/AHA valve guideline, measurement of valve area with pressure half-time (PHT) is not recommended immediately after percutaneous mitral balloon valvuloplasty (PMBV) (2) because many factors such as heart rate, cardiac output, left atrial pressure, and mitral regurgitation could affect this measurement (3). Different results have been obtained in previous studies related to this subject. When Chen et al. (4) compared measurements taken immediately after PMBV with the Gorlin formula, they found significant differences, but they also found measurements performed 48–72 h after PMBV close to the Gorlin formula. Pitsavos et al. (5) performed their interventions in a retrograde manner to exclude the left atrial decompression effect of iatrogenic ASD, and they found that PHT measurements taken 48–72 h after the retrograde intervention was similar to the Gorlin measurements; they attributed this to the iatrogenic ASD. In the current study, we also planned to take the measurements 48–72 h after PMBV by considering the differences that could develop during the acute period immediately after PMBV. This may be the reason the PHT and planimetric measurements were similar. The mechanism of this may be acute changes in the left atrium and left ventricle compliance, which develop because of dramatic changes in transmitral gradient immediately after PMBV. We know that iatrogenic ASD produced during the procedure usually has no clinical importance teorically and causes left-to-right shunt in a small percentage of patients. Thus, it is difficult to explain the contribution of iatrogenic ASD to the decrease in transmitral gradient through left atrial decompression. Furthermore, Pitsavos et al. (5) took the measurements 48–72 hours after the procedure.

Finally, to expect AF development in mitral stenosis is not a surprise. In the current study to more homogenously evaluate left ventricle function, we included patients who are in sinus rhythm and we excluded those in whom AF developed during the follow-up.

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Platelet to lymphocyte ratio: a novel and simple predictor of slow coronary flow

To the Editor,

We are grateful to have read with interest the article entitled "Relationship between platelet-to-lymphocyte ratio and coronary slow flow" by Oylumlu et al. published in *Anatol J Cardiol* 2015; 15: 391-5 (1). In this well-presented article, the authors hypothesized that the platelet-to-lymphocyte ratio (PLR) is associated with slow coronary flow (SCF) because an increased PLR was shown to be closely associated with inflammation and atherosclerosis. They demonstrated that PLR was significantly and independently associated with SCF. They suggested that increased PLR is an indicator of underlying inflammation in SCF.

Interventional cardiologists are familiar with the phenomenon of delayed opacification at the distal segments of the major epicardial coronary arteries in the absence of significant epicardial coronary artery stenosis, which is termed as SCF (2). The pathophysiological mechanisms underlying the SCF phenomenon have not been explicitly defined. Endothelial and microvascular dysfunction, inflammation, increased platelet activation, and atherosclerosis have been demonstrated to be closely associated with SCF (2, 3). As a combination of both platelet and lymphocyte counts, PLR recently emerged as a new potential inflammatory marker and predictor of major adverse outcomes in various cardiovascular diseases (4, 5). In the study by Oylumlu et al. (1), PLR was significantly higher in patients with SCF than in those in the control group (135.4±54.1 vs 113.4±31.1, p=0.001). However, other direct and indirect indicators of inflammation including white blood cell count, neutrophil count, neutrophil-to-lymphocyte ratio, and red cell distribution width were similar between the study groups. Additionally, the study lacks any data correlating the conventional biomarkers of systemic inflammation such as C-reactive protein (CRP) with PLR. According to all these findings, it was impossible to highlight the patho-

genetic role of PLR in SCF. In a recent study with a relatively large number of SCF patients (n=221), we reported that PLR, white blood cell, neutrophil, and platelet counts and serum CRP levels were significantly higher in the SCF group than those in the control group (5). Furthermore, PLR was also shown to be positively correlated with serum CRP levels confirmatory to its association with systemic inflammation. Therefore, we proposed that the relationship between PLR and SCF is because of the presence of an ongoing low-grade chronic inflammatory status. Chronic inflammation may cause an enhanced PLR, which would result in an increased risk for SCF.

In conclusion, these results suggest that besides its already known effect on prothrombotic status, a higher PLR level represents the impact of low grade chronic inflammatory state on coronary blood flow. As an easily available and cheap parameter of complete blood count, PLR can be calculated in clinical practice for the prediction of SCF. Further studies are needed to confirm our findings and define the pathophysiological role of PLR in SCF.

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DOI:10.5152/AnatolJCardiol.2015.6460

Author's Reply

To the Editor,

Thank you for your comments. The major limitations of our study were the low sample size and lack of CRP measurements in all patients because of the retrospective design of the study entitled "Relationship between platelet-to-lymphocyte ratio and coronary slow flow" by Oylumlu et al. (1) published in *Anatol J Cardiol* 2015;15:391-5. These may be the reasons for conflicting data with literature.