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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 pathogenetic 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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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.

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How can we reduce complications associated with thrombolysis for prosthetic valve thrombosis?

To the Editor,

We would like to comment on the recent article entitled "Stuck aortic valve treated by reteplase in a Bentall patient." published in Anatol J Cardiol 2015; 15: 339-40 by Tanyeli et al. (1), in which the use of reteplase in a patient with a stuck aortic mechanical valve is reported. We believe there are some major drawbacks to be addressed regarding the diagnostic algorithm and the treatment of choice.

Although guidelines have recommended surgery for PVT (2), we recently reported that low dose (25 mg) and slow infusion (6 h) of recombinant tissue plasminogen activator (t-PA) are very safe and are associated with a very high success in this regard (3, 4). In this study, repeated low-doses and slow infusions of alteplase regimen under the guidance of serial transesophageal echocardiography (TEE) was superior to faster infusion thrombolytic therapy (TT) protocols. In the current report, a patient with aortic PVT was administered double-bolus reteplase, which may be a very rapid TT regimen that may have resulted in a major embolism and/or hemorrhage. Thromboembolism due to rapid TT of PVT is well-recognized, and we respectfully suggest that clinicians should avoid the routine use of such a regimen. Rapid thrombolysis should only be reserved for certain circumstances, including critically ill patients with PVT or those with stroke (5) or acute myocardial infarction. Furthermore, the authors state that they pre-treated the patient with unfractionated heparin (UFH) and acetylsalicylic acid immediately before the first dose of reteplase and that it was continued thereafter. We reported that the safety of thrombolysis is related to prolonged infusion of t-PA without bolus and without concomitant UFH infusion (3, 4). We feel that the rapid infusion of t-PA with bolus dose and concomitant UFH jeopardizes PVT patients who may suffer risks of hazardous consequences (death, embolism, hemorrhage).

TEE should play a central role in every step of the management of patients with PVT, including the initial diagnosis, guiding the therapy, and evaluating the outcome. However, in the current report, the authors used only transthoracic echocardiography for the clinical decision-making of the patient with obstructed aortic PVT, which may be misleading. Fluoroscopy is frequently used to assess the leaflet motion in patients with PVT. However, the detection of the cause of leaflet blockade is not detectable during the catheterization study. Interestingly, the authors stated that they detected a huge thrombus burden resulting in severe aortic stenosis in the catheterization laboratory. The use of TEE is indispensable for the quantitative visualization of thrombus. On the other hand, the evaluation of the severity of obstruction in patients with aortic PVT should almost always include quantitative data beyond the maximum gradient, including the effective orifice area, dimensionless valve index, acceleration time, and acceleration/ejection time.

We believe that the management of patients with PVT should be evidence based, and current evidence strongly suggests the use of low-dose and slow infusion of TT protocols without bolus and without concomitant anticoagulant therapy in patients with PVT. Furthermore, heparin should be continued with warfarin until INR reaches a level of 2.5, rather than only 48 h after successful TT.

While this case is interesting, a good outcome in a single patient certainly does not prove that the approach used is broadly applicable.

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Author`s Reply

To the Editor,

We acknowledge the authors for their kind criticism regarding some complaints about our strategy of the stuck valve in our Bentall