recommends to be kept high levels of target INR (3.5-4.5) for patients who successfully treated with thrombolytic therapy due to mitral PVT. In our case, INR value was 1.58 and PVT which associated with inadequate anticoagulation has been considered. Due to PVT had not occured in target INR values; our patient was discharged after successful thrombolytic therapy, when our patient's INR value was reached to 3.5.

In the guidelines, there is no consensus about the treatment of patients with PVT. Surgical treatment is recommended in ESC guidelines (1) and thrombolytic therapy is recommended in The Society of Heart Valve Disease guidelines (2). Also, AHA/ACC Valvular Heart Disease guideline (3) that published in March 2014 recommends fibrinolytic therapy for patients with a thrombosed left-sided prosthetic heart valve, recent onset (<14 days) of NYHA class I to II symptoms, and a small thrombus <0.8 cm^2.

In TROIA study, Özkan et al. (4) five different thrombolytic treatment strategies [rapid streptokinase, slow streptokinase, high dose (100 mg) tPA, half-dose slow-infusion (50 mg/6 hour) tPA and low-dose slow infusion (25 mg/6 hour) tPA] were performed to patients with PVT. In this study, treatment success did not differ between the groups. However, the complication rate was found to be significantly lower in the slow-infusion low-dose tPA group than the other groups. In this study, overall complication rate was found significantly higher in the group receiving slow infusion of streptokinase compared to the low-dose slow-infusion tPA group (24.4% vs. 10.5%, p<0.05, respectively). Thus in the development of complications, the type of thrombolytic agent seems to be important as well as the velocity of the infusion.

In our patients, thinking that it was very fresh thrombus, we have applied 25 mg/12 hour tPA therapy. But we have identified this protocol as this patient specific. Therefore, large-scale studies are required to suggest that this protocol to all patients.

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Relation between ABO blood groups and coronary lesion complexity in stable coronary artery disease

To the Editor,

We have read the article "Relation of ABO blood groups to coronary lesion complexity in patients with stable coronary artery disease" written by Kaya et al. (1) with great interest published in February issue of The Anatolian Journal of Cardiology 2014; 14: 55-60. They aimed to investigate relationship between the severity of coronary atherosclerosis assessed by SYNTAX score and ABO blood group in patients with stable coronary artery disease. They concluded that SYNTAX score significantly high non-0 blood group attiribute to ABO gene and ATP-binding cassette 2 (ABCA) gene location in chromosome 9 and lowest von willebrand factor (vWF) antigen levels in O blood group (1).

Stakisaitis et al. (2) showed coronary atherosclerosis and ABO blood groups relationship in women. They found that B blood group can be related with coronary atherosclerosis, 0 blood group can possibly serve as a protective antiatherogenic factor and a blood group is not a risk factor for atherosclerosis in Lithuanian women. Chen et al.(3) contributed that serum cholesterol levels in ABO blood groups as a mediator of an association with coronary artery disease (CAD). They showed that increased low density lipoprotein (LDL) cholesterol, total cholesterol, non-high density lipoprotein (non-HDL) levels in non-O blood groups (3). Biswas et al. (4) found that blood group A risk factor for coronary artery disease and myocardial infarction in young people in Taiwan. They suggested in their study that AB blood group decreases the risk of coronary artery disease (CAD), and risk of CAD due to lower HDL cholesterol levels in Bengali population (4). Karabuva et al. (5) described no association between ABO blood groups and extent of coronary atherosclerosis in Crotian CAD patients. How can we explain these variations between blood groups and CAD in different races? Genetics and/or environment?

Kaya et al. (1) indicated that non-O blood groups had higher SYNTAX score, which evaluate the complexity of CAD but didn't state the interaction between blood groups and cholesterol levels. The relation of SYNTAX score to blood groups might be associated with cholesterol levels, which was showed by Chen et al. (3).

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Author`s Reply

To the Editor,

We would like to thank the authors of the letter for their interest and criticism about our study published in February issue of The Anatolian Journal of Cardiology 2014; 14: 55-60 (1). The relation between ABO blood groups and coronary artery disease is known for many years. But, there are no adequate information about the causes of relation between ABO blood groups and coronary artery diseases. In our study, we tried to discuss the relation between ABO blood groups and the development of coronary artery diseases, also we discussed the mechanism of coagulation attributing vWF and lipid metabolism of ABO blood groups (1). The author summarized the relation between blood groups and CAD in different races (2-5) and wanted our explanation of what might be the causes of variations between our study and the other studies. First of all, it is known that risk factors in the development of coronary artery diseases are different in different races. These differences are claimed to be both genetic and environmental reasons. We think that the variations between these studies and our study as well as these studies each other could be related to both genetic and environmental reasons. Secondly, authors wanted to learn the relation between lipid levels and ABO blood groups. We have re-analyzed our data and HDL (41±12 vs. 40±14), LDL (93±37 vs. 87±36), TG (135±128 vs. 129±131) did not differ between 0 and non 0 groups. However, Chen et al. (3) determined a significant relation between ABO blood groups and lipid levels as noted by the authors. We can list the possible causes of these differences: Both studies were conducted in different races (Turkish vs Chinese), while there were more than 6 thousand patients in the Chen et al. (3) study, there were about 500 patients in our study. In addition in our study 15-20% patients had a history of statin use. All these reasons may explain the differences between the two studies.

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Relation of ABO blood groups to coronary lesion complexity in patients with stable coronary artery disease

To the Editor,

In the past decades, several studies have suggested the possibility of ABO blood groups antigens to participate in pathogenesis of coronary artery disease (CAD), especially acute myocardial infarction and sudden cardiac death (1-3). Association between ABO blood groups and stable CAD has been significantly less investigated. That is why we read the paper of Kaya et al. (4) published in The Anatolian Journal of Cardiology 2014 ; 14 : 55-60 with particular interest, as it is in an important segment associated with our research published several months ago (5).

Contrary to the study by Kaya et al. (4) no association between ABO blood groups and the extent of coronary atherosclerosis in our study was observed. In our opinion, although very similar in the initial idea, our studies have different results, possibly the consequence of the different methodologies used in the atherosclerotic lesions assessment. Contrary to our study, in which the main indicator of CAD severity was the extent of coronary atherosclerosis assessed by modified Gensini scoring system (GS), in the study by Kaya et al. (4) the indicator of CAD severity was complexity of coronary lesions assessed by SYNTAX score (SS).

The SS has been developed as a useful predictor for the outcome of patients undergoing multi-vessel percutaneous coronary intervention, and provides a possibility for choosing optimal revascularization strategies for patients with complex coronary artery disease. This scoring system includes only diameter stenosis ≥50% in vessels ≥1.5 mm long. This is the main difference between the SS and the GS. The GS includes all grades of the narrowing in all cardinal epicardial vessels, including diagonal and obtuse marginal branches, taking into consideration the lesion's position in the coronary arterial tree. For example, an SS patient with five 49% lesions on the proximal segments of all the main epicardial coronary arteries, middle segments of the left anterior descending and right coronary artery, has no complex coronary artery disease so he/she could be excluded from the mentioned study (4). At the same time, according to the GS, this patient would be recognized as the patient with significant atherosclerotic changes (GS=20) and would be included in the analysis.

In that context, the 342 patients excluded from the study by Kaya et al. (4) despite SS=0, might have numerous significant atherosclerotic changes in their coronary arteries. In that case SS=0 only suggests these patients are not yet candidates for coronary interventions. Therefore, it would be very interesting to know if the distribution of the