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Fibrinolytic therapy in prosthetic valve thrombosis

To the Editor,

We have read with great interest the report published by Bayar et al. (1) published in May issue The Anatolian Journal of Cardiology 2014; 14: 297-9., about a woman with diagnosis of prosthetic valve thrombosis (PVT) no obstructive in mitral position, treated successfully with a slow infusion and low dose of tissular plasminogen activator (tPA).

We would like to make some considerations about it.

First we will remain highlighting the importance of treatment and following the anticoagulation in patients with prosthetic heart valve.

As well the author indicates the most frequent cause of PVT is the inadequate anticoagulation, it is essential to take into account all the aspects related with this treatment, fundamentally the pharmacologic interactions that interfere achieving an international normalized ratio (INR) in therapeutic values. The most probable cause of PVT in this case.

The authors affirm that the patient was discharged after a successful thrombolytic therapy with an antiaggregant therapy re-regulated. In this patient is recommended the indication of antagonists of the vitamin K and aspirin to reach an INR goal of 4 (range of 3.5 to 4.5) (2).

In this patient the choice of thrombolytic therapeutic was accurate and successful.

The initial therapeutic decision is difficult and controversial. Clinical practice guidelines express no uniform opinions (3). The European Society of Cardiology proposed surgery as the initial treatment, regardless of clinical status and the size of the thrombus. The Society of Heart Valve Disease recommends that the first choice be thrombolysis in all cases of PVT, unless such treatment is contraindicated.

The American College of Chest Physicians recommends that the main criterion in the therapeutic decision be the size of thrombus, indicating thrombolysis as the treatment choice if the thrombus has an area of 0.8 cm² and surgery in older thrombi. The American Heart Association and American College of Cardiology in the last guidelines published reserve only 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 cm² (Class IIa, Level of Evidence B) (2).

Even with the recommendations of the clinical practice guidelines it is very important to take into account the preference of the patient and the availability of emergency surgery.

In TROIA study, Özkan et al. (4) indicates similar rates of efficacy among the different schemes of thrombolytic treatment utilized. However, is attributed more safety to the scheme of treatment with tPA used by Bayar et al. (1).

Although a higher embolic complication rate has been reported for rtPA, which seems to be related to the higher infusion velocity, rather than with the type of thrombolytic agent (5).

Probably, the efficacy and safety of thrombolytic therapy in the PVT have greater relationship with the precocious diagnosis and the beginning fast treatment with the therapeutic scheme used.

We continued used intravenous recombinant streptokinase (250.000 IU/30 min and continuous infusion at 100.000 IU/hour, up to 72 hours). This approach also appears to be the most widely used and recommended protocol, and our outcomes are with acceptable efficacy rate and a good safety.

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Author`s Reply

To the Editor,

In current guidelines, target INR value is 3.0 for patients who underwent mechanical mitral valve replacement as is known. However, AHA/ACC Valvular Heart Disease guideline that published in March 2014

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 0 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-0 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-0 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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