Author`s Reply

To the Editor,

We are pleased to see the valuable comments and contribution of our colleagues in response to our article entitled "Antithrombin III deficiency concomitant with atrial fibrillation causes thrombi in all chambers: 2-D and 3-D echocardiographic evaluation" published in the December 2016 issue of the Anatolian Journal of Cardiology (1). We have some points to explain further.

In our report, there were many precipitating factors contributing to the thrombi in all chambers. Antithrombin III (AT) deficiency was proposed as a precipitating factor in addition to coronary artery disease and atrial fibrillation. We are aware of the rarity of arterial thrombosis secondary to AT deficiency; it was for this reason that we reported our case. There are case reports in the literature concerning arterial thrombosis due to AT deficiency (2). Other procoagulant precipitating factors accompanying AT deficiency have a role in the time of clinical incidence, as reported by

Emmanuelle et al. (3). The level of AT activity and the type of AT deficiency determine the clinical picture (4). The occurrence of multiple thrombi at the age of 62 made our case interesting.

The criticism about testing the AT level only once makes sense; however, we only had one chance to test the AT activity in our patient. Due to multiple mobile intracardiac thrombi, intravenous anticoagulation therapy was initiated as soon as possible. The patient did not recover, and was under medical therapy throughout the hospitalization period. Therefore, repeat testing for AT activity while under anticoagulation therapy would be misleading. It is known that AT level decreases as a result of anticoagulation therapy (5).

We agree with the opinion that when someone has inherited natural anticoagulant deficiencies, clinical problems often occur at an early age. On the other hand, as you mentioned, it was presented in a cross-sectional study that 3 patients who were demonstrated to have AT deficiency with repeated tests had no personal or family history of thrombosis (6). Precipitating factors play a major role in these circumstances. In our patient, apart from AT deficiency, atrial fibrillation concomitant with severe apical hypokinesia in the left ventricle due to myocardial infarction exacerbated the situation. It is impossible to link the multiple thrombi to only one of the underlying causes in this case report.

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