

Letter to the Editor

Editöre Mektup

Association of cardiac adaptations with NT-proBNP level after percutaneous closure of atrial septal defect

To the Editor,

On the June 2019 issue of the Archives of the Turkish Society, Kılıçaslan et al.^[1] reported the relationship between cardiac adaptations and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels after percutaneous closure of atrial septal defects (ASD). ASD is one of the most frequent (5%–10%) congenital heart diseases in adults.^[2] Patients with an ASD have a left-to-right shunt and the consequent chronic volume load of the right atrium, right ventricle, and pulmonary artery can cause heart failure and pulmonary hypertension.^[3] Reduction of right atrial and right ventricular diameter after ASD closure is an expected finding, and in previous studies, it has been demonstrated that closure of an ASD may lead to reduced right heart volumes due to diminished left-to-right shunting.^[4] Brochu et al.^[5] reported that even asymptomatic adult ASD patients experienced a significant increase in their functional capacity after percutaneous closure. NT-proBNP is secreted in response to atrial and/or ventricular stretch and shows a significant positive relationship with the amount of the left-to-right shunt.^[6–8] It doesn't increase only in heart failure or systolic dysfunction, but also in diastolic dysfunction and right ventricular dysfunction.^[7] Previous studies on the natriuretic peptic levels after shunt closure demonstrated significant decreases in plasma BNP levels concomitant with significant improvements in functional capacity, decrease in pulmonary artery systolic pressures and increase in tricuspid annular plane systolic excursion (TAPSE) values one month after the procedure. Increase in TAPSE measurements and decrease in NT-proBNP may be useful for monitoring the success of the intervention.^[8–11] In the Kılıçaslan et al.^[1] study, although the right ventricle and right atrial end diastole diameters and volumes decreased, the BNP level and right ventricle myocardial performance indices increased significantly. This is consistent with the physiological alterations of the right ventricle and is

explained by the lower volume in the ventricle after closure. Of particular interest in the present study was the increase in BNP concentrations after ASD closure.

We think differences in the structural adaptation to volume changes between the right heart chambers and left ventricle may be a cause for the observed increase in NT-proBNP. Additionally, the remodeling of the left ventricle due to volume overload after the closure of the left-to-right shunt may be slower than normal if the ventricular myocardium had been stiffer and had lower compliance which may have resulted in NT-proBNP to remain higher than normal.

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Authors reply

Dear Editor,

First of all, we thank the writers for their interest in our article. According to our results, percutaneous closure of an atrial septal defect (ASD) had multiple beneficial effects on the cardiac anatomy and physiology. A decrease in the size of the right chamber of the heart appeared as early as the first day after the procedure. However, the left side of the heart may be exposed to hemodynamic stress, depending on volume overload after the ASD closure, and left ventricle (LV) volume was found to have increased. The levels of N terminal B-type natriuretic peptide (NT-pro BNP) increased within the first day and were still elevated 30 days after the procedure, which is associated with increased LV diameters and volumes. As we know, the primary stimulus for NT-pro BNP release appears to be LV wall stretching in response to volume and pressure overload.^[1] NT-pro BNP levels are more dependent on the left heart than the right heart.^[2]

Functional measures of the right ventricle, such as tricuspid annular plane systolic excursion (TAPSE) values and basal systolic tissue Doppler velocity, have been generally reported to decrease significantly within 24 hours of closure and to continue to decline over the succeeding 6 to 8 weeks.^[3] Previous studies have demonstrated that the TAPSE value decreased and the myocardial performance index of the RV increased following percutaneous closure of an ASD. It has been proposed that it was a result of the sudden

decrease in the right heart volume load and pressure.^[3,4] Our results were comparable to these data.

As we noted among the limitations of the study, our follow-up period was relatively short and therefore, it is not possible to predict the long-term effects of percutaneous closure of an ASD. Nonetheless, it may be reasonable to envisage normalization of the cardiac structure, functions, and NT-pro BNP level with longer follow-up periods.

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