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Relation between elevation of brain natriuretic peptide and right ventricular dysfunction can be estimated simply by routine echocardiography in patients with repaired tetralogy of Fallot

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Introduction: Right ventricular (RV) dysfunction resulting from pulmonary valve (PAV) regurgitation is serious problem in patients with reapared tetralogy of Fallot (TOF). Replacement of PAV is triggered by severe RV dysfunction, which has been estimated by magnetic resonance imaging. Brain natriuretic peptide (BNP) is reported to increase by RV dysfunction. Furthermore, RV dysfunction is reported to cause left ventricular (LV) dysfunction by interaction. We investigated whether relation between BNP-elevation and RV dysfunction could be evaluated simply by routine echocardiography in repaired-TOF patients. Methods: Echocardiography and blood sampling were reviewed in 88 ambulatory patients with repaired-TOF. Levels of BNP in the top quartile of 88 patients were defined as BNP-elevation (≥ 42.6pg/ml). Normal end-diastolic dimension of LV (N-LVDd) was calculated with use of body surface: N-LVDd=4.1*(BSA)0.5*10.1mm. We defined %RVDd, %LVDd and %LVSd (end-systolic dimension of LV) as the ratio to N-LVDd. We used increase of %LVSd as index for reduced LV contraction. Results: Patients ranged in age from 2 to 33 years. After multiple logistic regression analysis BNP-elevation was independently associated with odds ratio of 31.7 (p=0.029) for moderate-to-severe PAV regurgitation (≥ II/III), 6.5 (p=0.013) for enlarged %LVSd (≥ 70%), 4.7 (p=0.044) for non-stenosis of pulmonary artery (≤ 2.5m/s), and 3.7 (p=0.04) for enlarged %RVDd (≥ 62%). In monovariate analysis BNP-elevation was additionally related to enlarged %LVDd (≥ 113%) and reduced ejection fraction of LV (≤ 60%). There were no significant associations with following factors: degree of tricuspid valve regurgitation; repair strategy for outflow tract of RV. Of repaired-TOF patients with BNP-elevation, 95% patients had risk factors on RV side; 63% had factors on LV side. Each BNP-elevation in this study could be explained without exception by one or more factors described above. Conclusions: Our study revealed that BNP-elevation in repaired-TOF patients was associated with RV related factors, and LV dysfunction which would be interacted with RV dysfunction. These dysfunctions colud be simply estimated by routine echocardiogram. All BNP-elevation could be accounted for by any one of these risk factors in this study. Based on this simple investigation we should evaluate RV dysfunction in young repaired-TOF patients by cardiac magnetic response.