P1-33

High values of NT-proBNP reflects various right-heart dysfunctions in patients with tetralogy of Fallot after repair

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Background: Values of NT-proBNP in patients with tetralogy of Fallot (TOF) after definitive repair is said to elevate reacting volume overload of right ventricle (RV) followed by pulmonary valve regurgitation (PR) but not reacting pressure overload of RV followed by stenosis of RV outflow tract. We predicted NT-proBNP values would rise highly because of diverse secretory stimulation as well as expanded end-diastolic volume of RV (RVEDVI). We investigated what kind of strain affected high NT-proBNP (hNT-proBNP) in repaired TOF patients. Methods. The medical records of 101 repaired-TOF patients from 1 to 53 years were reviewed. We performed cardiac catheterization to grasp hemodynamic status between 2010 and 2015. We defined hNT-proBNP as NT-proBNP 400pg/ml or over with which patients were potentially in state of heart failure (Japan heart failure society). Cardiac performances were determined which affected hNT-proBNP. Results. In monovariate analysis hNT-proBNP was significantly associated with elevated pressure on right-heart, such as pulmonary capillary wedge (≥ 14 mmHg), end-diastolic ventricle (≥ 14 mmHg), and end-systolic ventricle (pressure of RV/LV≥ 0.79). Similarly, hNT-proBNP was significantly associated with expanded volume on right-heart, such as end-diastolic ventricle (≥ 164 ml/m2), and end-systolic ventricle (≥ 109 ml/m2). Additionally, hNT-proBNP was related to reduced ejection fraction of RV (≤ 31%) and increased pulmonary vascular-resistance (≥ 3.1 units□m2). Cardiac performances on left-heart had no relation to hNT-proBNP. After multivariate analysis hNT-proBNP was independently associated with odds ratio of 11.2 for end-systolic volume of RV, 3.9 for end-systolic pressure of RV, and 3.6 for end-diastolic pressure of RV. Clinically, repaired-TOF patients with hNT-proBNP had some form of symptom more before examinations than those without hNT-proBNP had (45% vs. 17%, p=0.0068). Discussion. Our study showed hNT-proBNP also had relation to diverse RV overload other than expansion of end-diastolic RV in repaired TOF patients. Increased pulmonary resistance and pressure of pulmonary capillary wedge were associated with hNT-proBNP. We could point out RV dysfunctions by hNT-proBNP in no symptomatic patients. We should scan repaired TOF patients with hNT-proBNP, who had no symptom or no left-heart dysfunction, to suspect right-heart dysfunction.