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Pulmonary-circulation disturbance arises in Glenn patients with high values of n-terminal pro-brain natriuretic peptide

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Backgrounds: We generally consider that values of n-terminal pro-brain natriuretic peptide (NT-proBNP) would be decreased in Glenn circulation. However, high values of NT-proBNP (high NT-proBNP) subsist in Glenn patients. We predict Glenn patients with high NT-proBNP would possess not only cardiac depression but also pulmonary circulation disturbance. **Objective:** We investigated factors related to high NT-proBNP and the feature of pulmonary circulation in high NT-proBNP group.

Methods: The medical records of 125 Glenn patients were reviewed aged from 10 months to 23 years. We defined high NT-proBNP as NT-proBNP 800 pg/ml or over (by Japan heart failure society, n=27). First, indexes were determined which were connected with high NT-proBNP. Second, we compared pulmonary circulation factors between Glenn patients with and without high NT-proBNP.

Results: In monovariate analysis high NT-proBNP was associated with following 6 factors: histories of conduit from right ventricle to pulmonary arteries, ventricular volume on end-systole ($\geq 80\%$) and on end-diastole ($\geq 178\%$), ventricular pressure on end-diastole (≥ 12 mmHg), ventricular ejection fraction ($\leq 35\%$), and strong valve regurgitation of atrio-ventricle. After multivariate analysis high NT-proBNP was independently associated with odds ratio of 4.4 for expanded ventricular volume on end-systole, 3.9 for history of right ventricle conduit, and elevated ventricular pressure on end-diastole. Explanatory coefficient was 0.39. As for pulmonary circulation, pressure of superior vena cava was elevated in high NT-proBNP group (15 vs. 12 mmHg; $p=0.00048$); pressure of pulmonary capillary wedge (or left atrium) was elevated (9.6 vs. 8.0 mmHg; $p=0.041$); the ratio of patients with pulmonary resistance $2.4 \text{ U}\cdot\text{m}^2$ over was higher (30% vs. 9%; $p=0.013$).

Conclusion: High NT-proBNP values in Glenn patients were connected with myocardial stretch, which has been said in Fontan patients. Our study also showed history of conduit from right ventricle was independently related to high NT-proBNP in Glenn patients. As for pulmonary circulation, pressures of pulmonary capillary wedge and superior vena cava were increased in Glenn patients with high NT-proBNP. Depressed cardiac functions would build up afterload on pulmonary arteries. Heart failure therapy might be useful for lowering pressure of pulmonary artery in Glenn patients.