EFFECT OF NT-PROBNP BETWEEN ACE INHIBITOR AND VALSARTAN FOR HEART FAILURE PROGRESSIVITY PREVENTION IN LEFT TO RIGHT SHUNT CONGENITAL HEART DISEASE

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Background:

Congenital heart disease (CHD) is a disease entity with structural and circulatory abnormalities of the heart presenting at birth or after birth. In some children, CHD frequently manifest as complex disorder that requires fast and precise treatment. Based on 2009 data, mortality rate reached 26.6% in infants born with CHD, 50% of deaths were reported in the first month of life. One of the most common constituents in CHD is heart failure, which strongly associated with ventricular dysfunction, volume overload, and pressure overload. The current diagnosis of heart failure is still guided by a combination of history taking, physical examination, laboratory examination and imaging. Nevertheless, history taking and physical examination are bias in children since influenced by numerous factors such as obesity, malnutrition, and accompanying airway infections. Various laboratory examinations have been developed as biomarkers of heart failure such as natriuretic peptides (i.e. precursor N-terminal pro BNP (NT-proBNP), biologically active form (BNP), cardiac troponin. It is used as one of the key considerations in determining the procedure, monitoring of therapy, and prognosis of a child with heart failure. To date, heart failure therapy in children with CHD is still not satisfactory, although many research for new drugs developed. New strategies are needed to reduce the morbidity and mortality of PJB with heart failure.

Objectives:

To evaluate the differences and mechanisms between the administration of ACE inhibitors and Valsartan to prevent progressivity of heart failure in left to right to left shunt CHD through NT-proBNP, Troponin-t, SOD, and Catalase analysis.

Methods:

A randomized, pre post test control groups trial design was applied. The control group was given captopril (ACE Inhibitor) per oral 3 times a day for 12 weeks, while the treatment group treated with valsartan per oral once a day. To reduce bias, subjects in the treatment group took

1 dose valsartan and 2 placebo doses daily for 12 weeks. Variable evaluation is carried out before and after treatment. The main variables measured were NT-proBNP, Troponin-t, SOD, and Catalase. The measurements were carried out at the beginning of the study for both groups and at the end of treatment.

Results:

There was no statistically significant difference in NT-proBNP levels (p = 0.254), Troponin-t levels (p = 0.411), catalase reactivity (p = 0.965), SOD reactivity(p = 0.826), and echocardiography results (p = 0.266) after the administration of Valsartan or Captopril in the left-to-right shunt CHD group with heart failure. There was a statistically significant difference in clinical outcome (p = 0.02) after 12 weeks of Valsartan administration. However, there was no statistically meaningful difference in clinical outcome (p = 0.083) after 12 weeks of Captopril administration in left-to-right shunt CHD with heart failure.

Conclusion:

There was no statistically significant difference between the use of valsartan or captopril in inhibiting the progressivity of heart failure in left-to-right shunt CHD. The effectiveness of valsartan and captopril therapy are similar in left-to-right shunt CHD with heart failure.