ABSTRACT

ANALYSIS OF GALECTIN-3 LEVELS IN ACUTE HEART FAILURE PATIENTS THROUGH ACE INHIBITORS THERAPY

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Background: Heart failure is a complex clinical syndrome caused by structural or functional damage to the heart in filling or expelling blood from the ventricles. After a decrease in heart pumping capacity, various compensatory mechanisms are activated. Continuous activation of this system can cause remodeling the left ventricular and subsequent cardiac decompensation. Galectin-3 (Gal-3) is a marker of the inflammatory response in heart failure that can predict hospitalization and death in patients with heart failure. Gal-3 expression appears to stimulate pathological remodeling, specifically by inducing fibroblast proliferation and collagen deposition. Angiotensin-converting enzyme (ACE) Inhibitors are characterized by reducing both afterload and preload as well as being potential to reduce volume. ACE Inhibitors are able to modify heart remodeling.

Objectives: This study aims to analyze changes in Galectin-3 levels in heart failure inpatients through ACE Inhibitors therapy.

Methods: This research is a prospective observational study with pre and post test design. This research was conducted from May to July 2019 and and has approved by the Health Research Ethics Committee of Dr. Soetomo Public Hospital Surabaya. The researcher analyzed 23 blood samples of patients with acute heart failure who met the inclusion criteria. While the pre test on the patient's blood was taken before ACE Inhibitors were given, the post test was conducted on the last day when the patients underwent ACE Inhibitors treatment. Galectin-3 serum levels were examined using the MyBioSource Human Gal-3 ELISA kit and Wilcoxon statistical tests were then performed.

Results: The pre Galectin-3 levels range was 0.2-1.3 ng/mL, the post Galectin-3 levels range was 0.1-1.1 ng/mL, and the average reduction in Galectin-3 levels was 28.99%. Based on Wilcoxon's statistical analysis, there was no significant change in Galectin-3 levels by administering ACE Inhibitors ($p = 0.205$).

Conclusion: There was no significant change in pre and post Galectin-3 levels through ACE Inhibitors means there was no more progression or worsen of fibrosis in myocardium. All patients' cut-off value was ≤17.8 ng/mL so that it can be interpreted as the low risk category and recommended for continuation of regular care with periodic follow-up.

Keywords: Galectin-3, Acute Heart Failure, ACE Inhibitors