Objectives: Pulmonary hypertension (PH) and vascular resistance in patients with pulmonary arterial hypertension (PAH) are caused by remodeling and thrombosis of small and medium sized pulmonary arteries and arterioles, as well as vasoconstriction. These patients have increased platelet aggregation and the activated platelets are major source of thromboxane A2 which is a strong vasopressor and pro-aggregation molecule. PGI2 and PGE1 have opposite effects. Misbalance in eicosanoids synthesis was observed in patients with PH. The results from vasoreactivity test, in which we measure the hemodynamic response to vasodilator, particularly change in mean pulmonary pressure (mPAP), pulmonary vascular resistance (PVR) and trans-pulmonary gradient (TPG) in patients with PH due to the left heart disease, influence the decision on future course of treatment. The goal: to test whether the change of platelet aggregation during vasoreactivity testing is related to hemodynamic response measured as the change in mPAP, PVR and TPG.

Patients and Methods: Our pilot study included 38 patients with secondary PH due to the left heart disease, 29 men (76%) and 9 women (24%). The right heart catheterization was performed in all patients and vasoreactivity testing with PGE1 in 19 patients (50%). Platelet aggregation induced by addition of AA (ASPI test), ADP (ADP test) and collagen (COL-test) was measured in blood samples from pulmonary artery with Multiplate and repeated after vasoreactivity testing. Results: Patients with the reduction of platelet aggregation in ASPI test had stronger, but insignificant reduction in mPAP (p=0.08) and PVR (p=0.15). Significant reduction was observed in the reduction of TPG (p=0.03). The reduction of platelet aggregation in ADP test was not related to hemodynamic response to PGE1. Patients with the reduction of aggregation in collagen test had stronger, though insignificant reduction in mPAP (p=0.08). However, they had significant reduction in PVR (p=0.01) and TPG (p=0.05) in comparison to those with increased aggregation.

Conclusion: The data analysis in our pilot study shows insufficient size of the sample to reach a final conclusion. However, it suggest a significant potential difference in vasoreactivity among patients who respond with a decrease and those who respond with an increase in platelet aggregation after testing with PGE1. We hypothesize that variations in platelet response to PGE1 modulate its hemodynamic effect on pulmonary circulation.

KEYWORDS: pulmonary hypertension, platelet aggregation, vasoreactivity.