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Title: Effects of epoprostenol infusion on myocardial inflammation in acute afterload-induced right ventricular failure

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Body: Background: Acute transient pulmonary hypertension may induce a state of persistent right ventricular (RV) failure. Aims and objectives: We hypothesized that this could be related to an activation of inflammatory pathways, and reduced by prostacyclin therapy. Methods: Twenty-three dogs were randomized to a sham- (n=8) or to a 90-min pulmonary artery constriction-operation followed by 30-min banding release (n=15). In 7 dogs with persistent RV failure, epoprostenol infusion was performed 30-min after banding release. Myocardial tissue was finally sampled. Results: Persistent RV failure was associated with increased myocardial expressions of interleukin(IL)-1β, IL-6, monocyte chemoattractant protein(MCP)-1, pro-inflammatory IL-6/IL-10 ratio, and infiltration of neutrophils and macrophages, while heme oxygenase(HO)-1 expression was decreased. These changes were observed in the RV and, to a lesser extent, in the LV. In the RV only, expressions of prostacyclin synthase (PGI2S), IL-10 and IL-33 decreased and expression of vascular cell adhesion molecule(VCAM)1 increased. Epoprostenol decreased RV and LV expressions of IL-1β, VCAM1 and increased IL-10 expression, while MCP-1 decreased only in the RV. Epoprostenol decreased RV IL-6/IL-10 and pro-apoptotic Bax/Bcl-2 ratios, together with decreased neutrophil and macrophage infiltrations. RV ratio of end-systolic to arterial elastances was inversely correlated to RV IL-6/IL-10 ratio, macrophage and neutrophil infiltrations, and correlated to RV expressions of HO-1 and IL-33. Conclusion: Acute afterload-induced persistent RV failure is associated with an activation of inflammation which is limited by an epoprostenol infusion.