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## The association of FGF23 and inflammation in heart failure with normal kidney functions

**Grander W, Ulmer H** and **Pölzl G** University Teaching Hospital, Austria

**Background:** Fibroblast growth factor-23 (FGF23) produced by oteocytes regulates calcium and phosphate homeostasis which are cornerstones for bone integrity. Recently, FGF23 was also found to be directly related to both severity and prognosis of heart failure. However, the mechanism of FGF23 regulation in heart failure, particularly in patients with preserved renal function is poorly understood.

**Objective:** The association of systemic inflammation (surrogated by CRP) and FGF23 regulation in patients with chronic heart failure and preserved renal function. Furthermore, we analyzed the prognostic ability of FGF23 and CRP in this population.

**Methods:** 221 stable non-ischemic heart failure patients (age  $\geq$  18) with reduced ejection fraction and an estimated glomerular filtration rate of more than 60 ml/min/1.73m<sup>2</sup> were analyzed. Fasting ct-FGF23, high sensitive CRP and a comprehensive panel of further biomarkers, as well as invasive hemodynamic measures from right heart catheterization, were used for univariate and multivariate regression analysis.

**Results:** In bivariate correlation analysis ct-FGF23 was correlated with CO (r=-0.42); NTproBNP (r=0.39); eGRF (r=-0.38) and CRP (r=0.37); for all those p < 0.001. Multivariate linear regression analysis revealed CRP and CO as independently associated with ct-FGF23 (total model fit;  $r^2=0.49$ ; p < 0.001). In time to event analysis both ct-FGF23 and CRP independently predicted transplant-free survival.

**Conclusion:** Our data indicate an association between systemic inflammation and FGF23 elevation in heart failure. Both, FGF23 and systemic inflammation independently predict transplant-free survival in non-ischemic heart failure patients with preserved kidney functions.



wilhelm.grander@tirol-kliniken.at