Background: Inflammatory cytokines such as interleukin-6 (IL-6) have been implicated in chronic heart failure (CHF) progression, mediating adverse cardiac remodelling. Neutrophil-to-lymphocyte ratio (NLR) and reactive thrombocytosis are cellular components of systemic inflammation that are regulated by cytokines especially IL-6. In this study, we investigated the prognostic significance of a novel inflammation-based system for CHF, collectively named the CPNR (combination of platelet count and NLR), in predicting outcome in CHF patients.

Methods: A total of 1,557 patients with CHF (mean age 76±11 y, 34% females, 65% IHD; 57% in NYHA III/IV) were recruited as part of the BIOSTAT-CHF Scotland study. Routine laboratory measurements including full blood count was performed at baseline from which NLR and platelet count was determined. The cut-off values of NLR and platelet count were chosen at 3 and 275 respectively so as to maximise model fit, backed up by receiver operating characteristic curve analysis. A patient with both elevated NLR (>3) and platelet count (>275) was allocated a score of 2 (CPNR 2), and a patient shown one or neither was allocated a score of 1 (CPNR 1) or 0 (CPNR 0), respectively. Cox proportional hazard models were used to assess the prognostic impact of CPNR, adjusting for significant covariates.

Results: During a median follow-up period of 1.4 years (IQR 0.6,2.2), there were 23% all-cause deaths, 10% CHF deaths and 12% CVD deaths. Mortality rates (95% CI) were higher in CPNR 2 (all-cause: 251, CHF: 129, & CVD: 116 deaths per 1000 person years) as compared to CPNR 1 (all-cause: 189, CHF: 86, & CVD: 99 deaths per 1000 person years) and CPNR 0 (all-cause: 76, CHF: 28 & CVD: 42 deaths per 1000 person years). A Cox proportional hazard model, adjusted for relevant covariates, showed that CPNR was a significant risk factor for mortality [all-cause, HR=1.5 CI=1.3-1.8; CHF, HR=1.65 CI=1.3-2.2; CVD, HR=1.5 CI=1.2-1.9] and CHF hospitalization (HR=1.3 CI=1.1-1.5).

Conclusion: CPNR is a novel and readily available biomarker of inflammation that could potentially help in risk stratification of CHF patients.