

non-infarcted control mice. In contrast, monomorphic or polymorphic VTs could be evoked in 88% of infarcted and vehicle-injected mice ($p < 0.01$). Despite a relatively modest degree of engraftment, transplantation of MNCs and monocytes improved electrical stability within the in vivo electrophysiological examination ($p < 0.05$) and improved conduction velocities in the borderzone ($p < 0.05$). In addition, the decline of mRNA levels of Connexin43 (Cx43) and specific ion channels (Kir2.1, Kv4.2, Nav1.5, Cav1.2) was attenuated in mice treated with MNCs and monocytes. Cryoinfarction resulted in a broad aggravation of left ventricular ejection fraction. All transplanted cell types augmented left ventricular function to a similar extent both with left heart catheterization and echocardiography.

Conclusion: In the present mouse model of myocardial infarction, transplantation of MNCs and monocytes caused an improvement of left ventricular EF and a reduction of inducible VTs by modulating expressions of Cx43 and specific ion channels. Our data suggest next to functional improvement antiarrhythmic effects of specific cellular replacement therapy.

PROGNOSIS IN HEART FAILURE

P1479 | BEDSIDE

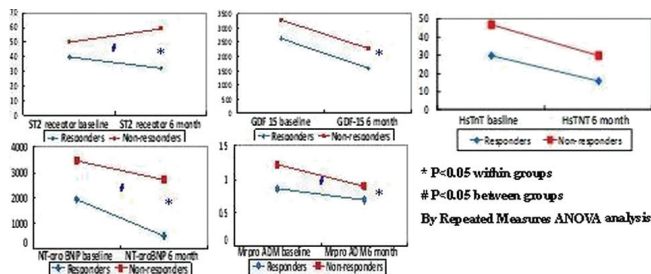
Predictive value of novel biomarkers for response to medical treatment in systolic heart failure

M. Liu¹, A.P.W. Lee¹, A.M. Richards², R. Renneberg³, Q. Zhang⁴, C. Lam², X.R. Huang⁵, H.Y. Lan⁵, B.P. Yan¹, C.M. Yu¹. ¹IVM, Div of Cardiology, Dept of M&T, PWH, The Chinese University of Hong Kong, Hong Kong, Hong Kong SAR, People's Republic of China; ²Dept. of Medicine, University of Otago, New Zealand, Cardiovascular Research Institute, NUHS, Singapore, Singapore; ³Department of Chemistry, Hong Kong University of Science and Technology, Hong Kong, Hong Kong SAR, People's Republic of China; ⁴Department of Cardiology, West China Hospital, Chengdu, Sichuan, China, People's Republic of; ⁵Li Ka Shing Institute of Health Sciences, Chinese University of Hong Kong, Hong Kong, Hong Kong SAR, People's Republic of China

Purpose: Although large scale clinical trials have demonstrated the efficacy and safety of pharmacological treatment for systolic heart failure (HF), HF treatment non-response was a common clinical observation. We sought to evaluate the role of HF biomarkers in prediction of HF treatment response.

Methods: The participating sites from 3 countries have recruited a total of 646 patients with systolic HF, of which 299 patients have completed 6-months follow-up. HF treatment "Non-responders" was defined as death, re-hospitalization for HF, and/or reduction of left ventricular end-systolic volume of $< 10\%$ in 6 months despite standard HF medication therapy. 25 biomarkers related to inflammation, fibrosis or myocyte damage were compared between "Responders" and "Non-responders".

Results: Of 299 patients enrolled during the reporting period, 169 (53.5%) were HF treatment "Non-responders" while 130 (46.5%) were "Responders". 5 of the 25 biomarkers including N-terminal prohormone of Brain Natriuretic Peptide (NT-proBNP), ST2 receptor, Midregional pro-adrenomedullin (MRproADM), Growth differentiation factor 15 (GDF15) and High sensitivity Troponin T (Hs-TNT) were much higher in Non-responders than in Responders. Repeated-measure ANOVA showed differences in the serial changes in response to HF treatment for NT-proBNP, ST2 receptor and MRproADM between "Responders" and "Non-Responders" (All $P < 0.05$). (Figure) In multiple regression analysis, Hs-TNT (Odds Ratios (OR): 1.004, 95% Confidential Interval (CI) 1.000-1.008, $p=0.04$) and ST2 receptor (OR 1.007, 95% CI 1.000-1.013, $p=0.04$) were independent predictors of HF treatment Non-responders.



Conclusions: Hs-TNT and ST2 receptor are potentially useful biomarkers to identify treatment "Non-responders" in mid-term follow-up of HF patients.

P1480 | BEDSIDE

Influence of hospitalization for cardiovascular versus noncardiovascular reasons on subsequent mortality in patients with chronic heart failure across the spectrum of ejection fraction

A.S. Desai¹, B. Claggett¹, M.A. Pfeffer¹, N. Bello¹, P.V. Finn¹, C. Granger², J.J.V. McMurray³, K. Swedberg⁴, S. Yusuf⁵, S.D. Solomon¹. ¹Brigham and Women's Hospital, Boston, United States of America; ²Duke University School of Medicine, Durham, NC, United States of America; ³University of Glasgow, BHF Glasgow Cardiovascular Research Centre, Glasgow, United Kingdom; ⁴Sahlgrenska Academy, University of Gothenburg, Dept. of Molecular & Clinical Medicine, Gothenburg, Sweden; ⁵Population Health Research Institute, McMaster University, Hamilton, Canada

Purpose: Noncardiovascular (non-CV) comorbidities may contribute to hospitalizations in patients with heart failure (HF). We examined the incidence of mortality following hospitalization for cardiovascular (CV) versus non-CV reasons in patients with reduced or preserved ejection fraction (EF) in the CHARM Program. **Methods:** CHARM randomized 7,599 subjects (4576 with $EF \leq 40\%$ and 3023 with $EF > 40\%$) with NYHA class II-IV HF and prior history of cardiac hospitalization to treatment with candesartan or placebo. First hospitalizations for CV or non-CV reasons were related to subsequent risk of all-cause death over median 36.6 month follow up using time-updated proportional hazards models.

Results: 4792 patients experienced a classifiable incident first hospitalization, including 2806 (58.5%) for CV reasons and 1986 (41.4%) for non-CV reasons, while 2802 were not hospitalized. Rates of CV hospitalization were higher for those with $EF \leq 40\%$ than those with $EF > 40\%$ ($p < 0.001$), but rates of non-CV hospitalization did not vary by EF ($p=0.88$, Table). The death rate (per 100-patient years) amongst those not hospitalized was 2.7 compared with 18.3 after CV and 16.2 after non-CV hospitalization (both $p < 0.001$). Mortality at 30-days was higher after CV than non-CV hospitalization ($p < 0.001$, Table). However, amongst 30-day survivors of CV and non-CV hospitalization, rates of subsequent mortality were similar (14.7 vs. 14.3, $p=0.62$). Low EF patients were at higher risk for mortality than high EF patients after both CV and non-CV hospitalization. (both $p < 0.001$).

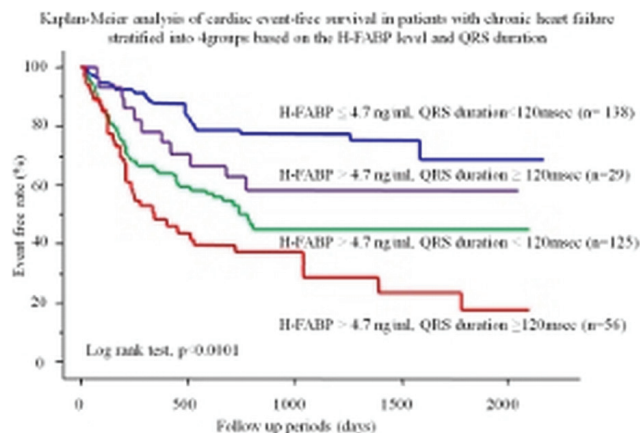
Conclusions: Non-CV reasons for hospitalization are common in HF patients across the spectrum of EF. Hospitalization for any reason is associated with high risk for subsequent mortality, with higher risk in low than preserved EF patients. Early mortality is higher after CV than non-CV hospitalization, but rates of mortality in 30-day survivors are unrelated to the cause of hospitalization.

P1481 | BEDSIDE

Combination of heart-type fatty acid binding protein and QRS prolongation can risk-stratify patients with chronic heart failure

S. Kadowaki, T. Watanabe, T. Narumi, Y. Otaki, Y. Honda, S. Honda, M. Hasegawa, T. Shishido, I. Kubota. Yamagata University School of Medicine, Yamagata, Japan

Background: It was reported that abnormal prolongation of QRS duration is associated with cardiac prognosis in patients with chronic heart failure (CHF). QRS prolongation represents left ventricular hypertrophy, bundle branch block, and intra ventricular conduction disturbance. We and others reported that elevated levels of heart-type fatty acid binding protein (H-FABP), which is a marker of ongoing myocardial damage, can predict poor outcomes in CHF patients. The aim of this study was to elucidate whether a combination of markers for ongoing myocardial damage and electrical disturbance can risk-stratify CHF patients.



Abstract P1480 – Table 1. Incidence of Hospitalization for CV and non-CV Reasons in the CHARM trial and subsequent rates of mortality

	CV Hospitalization			Non-CV Hospitalization		
	CHARM-Overall (N=7599)	LVEF $\leq 40\%$ (N=4576)	LVEF $> 40\%$ (N=3023)	CHARM-Overall (N=7599)	LVEF $\leq 40\%$ (N=4576)	LVEF $> 40\%$ (N=3023)
Incidence of first hospitalization (per 100 p-y, 95% CI)	21.2 (20.4, 22.0)	22.9 (21.9, 24.0)	18.7 (17.6, 19.9)	15.0 (14.4, 15.7)	15.0 (14.2, 15.9)	14.9 (14.0, 16.0)
Death within 30 days of hospitalization (n/N, %)	210/2806 (7.5%)	164/1788 (9.2%)	46/1018 (4.5%)	87/1986 (4.4%)	57/1171 (4.9%)	30/815 (3.7%)
Incidence of Death after 30 days (per 100 p-y, 95% CI)	14.7 (13.7, 15.9)	18.0 (16.5, 19.6)	9.7 (8.4, 11.2)	14.3 (13.1, 15.7)	16.8 (15.1, 18.8)	10.8 (9.1, 12.7)