toms, functional status, date of HF diagnosis and prior cardiovascular investigations, clinical risk factors, lifestyle factors, socio-economic status, and survey of cultural beliefs, health practices, and attitudes towards device therapy. Centre-level characteristics (case load, referral pattern, specialization, and infrastructure) are also obtained. Patients uniformly undergo standard 12-lead ECG and transthoracic echocardiography at baseline, and are followed over 3 years for outcomes of death or hospitalization

Results: There were 300 HF patient,Mean age was 58±12 years, and 61.4% were male. In our cases, we found that most patients came with worsening heart failure due to acute coronary syndrome (33.3%) and paralleled with ishemic heart condition (66.4%), documented or undocumented by coronary angiography, to be the leading precipitating factor. Other etiologies were dilated cardiomyopathy (9.8%) and valve disease (5.7%). 30-days mortality after discharge and rehospitalization also decreased with optimal dose of ACEI therapy, but not statistically significant (p=0.375 and p=0.184). Subanalysis showed optimal dose of ramipril was more superior than captopril in decreasing inhospital mortality (p=0.032).

Conclusion: The prevalence of heart failure patient higher in men than woman and the most common causes of worsening heart failure were Acute coronary syndrome.

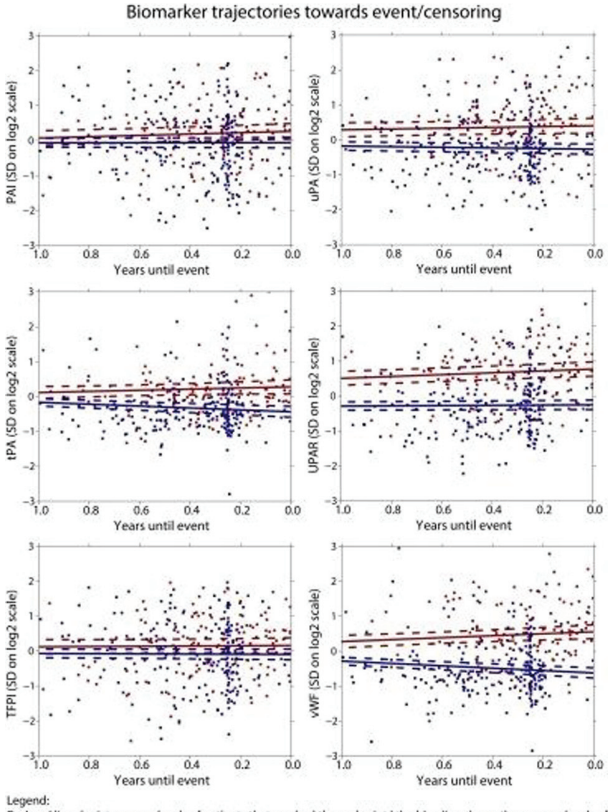
P5665

Coagulation biomarkers and clinical outcomes in patients with chronic heart failure - The bio-shift study

V.J. Van Den Berg[1], E. Bouwens[1], V.A.W.M. Umans[2], O.C. Manintveld[3], K. Caliskan[3], A.A. Constantinescu[3], J.H. Cornel[2], K.M. Akkerhuis[3], E. Boersma[1], I. Kardys[1]. *[1]Erasmus Medical Center, Clinical epidemiology of Cardiology, Rotterdam, Netherlands; [2]Northwest clinics, Cardiology, Alkmaar, Netherlands; [3]Erasmus Medical Center, Cardiology, Rotterdam, Netherlands*

Background: Chronic heart failure (CHF) is a well-known risk factor for thrombotic events, but the predictive value of biomarkers of coagulation in these patients are unknown. We studied 6 biomarkers within the coagulation cascade, and investigated whether their upregulation during the course of CHF is associated with adverse clinical events.

Methods: In a prospective multicenter study, a median of 9 (IQR 5–10) repeated blood samples blood were collected in 263 patients at a three month interval during a median follow-up of 2.2 (IQR 1.4–2.5) years. 70 patients reached the endpoint of cardiovascular death or heart failure admission. Baseline samples were combined with the last two samples before an endpoint, and in event-free patients the last sample available. Plasminogen activator inhibitor 1 (PAI), Urokinase plasminogen activator surface receptor (UPAR), Urokinase-type plasminogen activator (uPA), von Willebrand factor (vWF), Tissue factor pathway inhibitor (TFPI), and Tissue-type plasminogen activator (tPA) were measured using an Olink Proteomics multiplex assay. Associations between the biomarkers and endpoints were investigated by combining mixed models for the longitudinal data and Cox regression for the survival data in a joint model.



Legend:
Dark red line depict average levels of patients that reached the endpoint;l the blue line shows the average levels of patients that did not. The dashed lines are the corresponding upper and lower limits of the 95% confidence interval.

Figure 1

Results: The mean (SD) age of the patients was 67 (13) years, 72% were man, and 74% were in NYHA class I-II. Average biomarker levels of UPAR, uPA and vWF were significantly higher in patients with an endpoint versus event-free patients (figure). The corresponding HR (95% confidence interval) per 1 SD increase difference (in log2 level of an arbitrary measurement unit) were: UPAR: 3.15 (2.30–4.48), uPA: 2.27 (1.49–3.55), and vWF: 5.72 (2.61–16.58) (table).

Table 1. Results joint models

	Crude		Adjusted†	
	Estimate (95% CI)	p-value*	Estimate (95% CI)	p-value
PAI	1.47 (1.01–2.12)	0.042	2.08 (1.30–3.51)	0.004
UPAR	3.15 (2.30–4.48)	<0.001	3.92 (2.48 -6.44)	<0.001
uPA	2.27 (1.49–3.55)	<0.001	1.92 (1.18–3.18)	0.006
vWF	5.72 (2.61–16.58)	<0.001	5.74 (2.27–26.37)	<0.001
TFPI	1.44 (0.95–2.17)	0.096	1.39 (0.88–2.22)	0.152
tPA	1.97 (1.02–4.16)	0.044	2.73 (1.18–8.36)	0.020

*Corrected for multiple testing; level for a significant test was set at (0,05/6=) 0,0083. †Adjusted for gender, age and differences in baseline characteristics between patients that reached the endpoint and patients that have not.

Conclusion: Elevated profiles during the course of CHF of the coagulation biomarkers UPAR, uPA and vWF are strongly associated with clinical outcomes.

Funding Acknowledgements: This work was supported by the Jaap Schouten Foundation and the Foreest Medical School

P5666

Long-term survival analysis of patients with heart failure of different etiologies in a Brazilian cohort of outpatients - a bayesian approach

M. Marcula[1], L.G. Esteves[2], B.A.R. Melo[2], R.A.B. Nunes[1], V. Fossaluza[2], C.A.B. Pereira[2], A.J. Mansur[1]. *[1]Heart Institute (InCor), São Paulo University Medical School, Sao Paulo, Brazil; [2]University of Sao Paulo, Institute of Mathematics and Statistics, Sao Paulo, Brazil*

Purpose: The aim of the study was to evaluate long-term survival in patients with heart failure according to etiology, age, gender, and duration of clinical Picture considering a new statistical model characterized by a convex linear combination of gamma, lognormal and Weibull distributions under the Bayesian perspective. Linear combinations of standard families of distributions are more flexible models than any of its components alone and may be reasonable alternatives to non-parametric methods.

Methods: We studied 1394 patients with heart failure of different etiologies enrolled between April 1991 and July 2003 and followed up to 2016; deaths were ascertained up to 2014 (mean follow-up 9.78 years) in an outpatient care facility of a cardiology hospital. Patients' ages was 44.8 (standard deviation 10.9) years, 1080 (77.5%) were male and 314 (22.5%) female. Etiologies of heart failure were: hypertensive heart disease in 250 (17.9%) patients, ischemic heart disease in 251 (18.1%), Chagas' heart disease in 285 (20.4%), other in 270 (19.4%). In 338 (24.2%) patients heart failure was ascribed to dilated cardiomyopathy. Duration of clinical picture was estimated either from beginning of symptoms (when feasible) or from the first medical visit. Statistical analysis was performed first with traditional survival Kaplan-Meier estimation and then with statistical procedures under the bayesian paradigm according to etiology, age, gender and duration of clinical picture.

Results: We observed that patients with Chagas' heart disease had the lowest survival function estimate and patients with hypertensive heart disease had the highest survival function. There were no significant differences in survival function between patients with heart failure due to ischemic, dilated cardiomyopathy and heart failure due to other etiologies. Male patients had an expected survival time 15% lower than that of female patients. Combining levels of etiology, gender and duration of clinical picture to estimate the probability of survival for more than 10 years, we found the probability of survival for more than 10 years of male

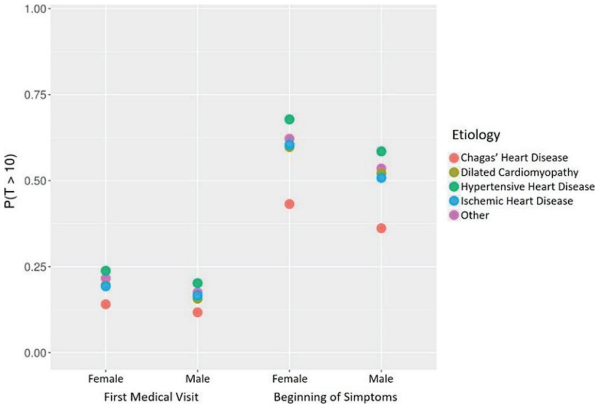


Figure 1. Probability of survival for >10 years