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The Area under the Strain Curve Provides Early Prediction of Infarct Size in Patients with Acute Myocardial Infarction

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Background: Early prediction of infarct size directs therapy in patients with acute myocardial infarction (AMI). Global strain by echocardiography describes myocardial deformation and correlates with infarct-size. However, peak strain measures deformation at a single time point, whereas ischemia and necrosis influence deformation throughout the heart cycle. We hypothesized that measurement of myocardial deformation throughout the heart cycle by the area under strain curve (area) is a better expression of myocardial function. The aim was to assess the ability of area to predict infarct size and to identify large infarcts at admission in patients with AMI.

Methods: Echocardiographic measurements were performed at admission in 76 patients with AMI. Strain was assessed in a 16 segments left ventricular (LV) model based on 3 apical planes. Infarct size was measured by contrast MRI after ≥3 months. The ability to identify large infarct (involving ≥6 segments) was calculated by ROC analyses.

Results: Longitudinal globalarea had superior correlation with infarct size compared to longitudinal global strain and LV ejection fraction (r=0.68, r=0.55, r=-0.50, respectively) and better accuracy for identification of large infarcts (Table).

Conclusions: Longitudinal global area provides improved early prediction of infarct size and identification of large infarcts in patients with AMI compared to global strain and LV ejection fraction.

	Area under the curve	Sensitivity	Specificity	Cut-off
Longitudinal global area	0.92*	89%	88%	-8
Longitudinal global strain	0.83	78%	82%	-16%
Left ventricular ejection fraction	0.75	80%	75%	50%

** p<0.05 compared to LV ejection fraction and global strain.*

Transcatheter Aortic Valve Implantation (Tavi) Immediately Improves Global LV Systolic And Diastolic Function

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Background: Transcatheter aortic valve implantation (TAVI) is a treatment option for patients with severe aortic stenosis. We aimed to investigate per procedure changes in LV systolic and diastolic performance during TAVI in high risk patients, and hypothesized that TAVI immediately improves LV systolic and diastolic function.

Methods: Nine (2 women) consecutive patients aged (mean±SD) 77±8 years treated with the transapical aortic valve replacement technique in general anesthesia were studied after induction of anesthesia (BL) and 15 min post TAVI. All had severe symptomatic aortic stenosis (valve area 0.69±0.20 cm2, transvalvular gradient 51±9 mmHg). In all, surgical treatment was declined due to high risk (Log Euroscore 28±14). Four patients had pre procedure PCI. Transoesophageal echocardiography was used to obtain mitral ring systolic velocities (S') and the diastolic mitral inflow deceleration (E DT) and the ration between early (E) and atrial (A) mitral inflow velocities (E/A-ratio). Invasive pressures and continuous cardiac output monitoring were obtained by LiDCO pulse power analysis facilitating estimates of pulse pressure, stroke volume index (SVI) and systemic vascular resistance (SVRI).

Results: The TAVI procedure was successful in all patients and induced an immediate improvement in global LV systolic function, demonstrated by an increase in S' from -2.6±0.9 at BL to -3.6±0.9 cm/s 15 min post TAVI (p=0.003). E DT decreased from 286±131 to 167±78 ms (p=0.011), while E/A-ratio increased from 2.0±1.1 to 3.1±1.6 cm/s (p=0.021), indicating enhanced diastolic relaxation post valve delivery. There was a marked increase in pulse pressure (44±4 mmHg to 60±9 mmHg, p=0.005), despite unchanged SVRI (1775±716 to 1583±359 d.s.cm-5m-2, p=0.43). No significant changes were observed in heart rate (72±15 to 79±17

beats/min), mean arterial blood pressure (65±12 to 70±5 mmHg), central venous pressure (10±4 to 12±2 mmHg) or SVI (34±12 to 37±14 mL/m2).

Conclusions: This study demonstrates that LV systolic contraction and even diastolic relaxation are immediately improved following TAVI in high risk patients. Further studies need to focus on the prognostic value of these results.

Intensive Atorvastatin Treatment Reduces Cardiovascular Events (CVE) in those with Dyslipidemia, Coronary Heart Disease (CHD) And With Mild Or More Advanced Chronic Kidney Disease (CKD): An Analysis Of The TNT, Ideal And Alliance Trials

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Background: Several analyses have demonstrated that statins reduce CVE in CHD subjects with mild renal impairment; however 2 large treatment trials have failed to demonstrate significant reductions in CVE with statins in subjects with end-stage renal disease (ESRD), on dialysis. The effect of more intensive versus less intensive statin therapy on CVE has not been thoroughly examined in subjects with more advanced CKD, without ESRD. Methods: We performed a retrospective analysis of 3 long-term trials in subjects with CHD and dyslipidemia; each of which compared aggressive therapy with atorvastatin (A) to an active comparator (A80 vs. A10 in TNT; A80 vs. simvastatin 20-40 in IDEAL, and A40 [mean dose] vs. usual care in ALLIANCE). The primary endpoint for this analysis was any CVE, defined as CHD death, nonfatal MI, coronary revascularization, hospitalization for angina or heart failure, cerebrovascular event, or PVD.

Results: A total of 20,961 subjects were identified, 6007 (29%) of whom had CKD defined as a MDRD eGFR <60 ml/min/1.73m2 at baseline. Of those with CKD, 71% (4268) had mild CKD (eGFR 55.96 ± 2.80 ml/min/1.73m2) vs 29% (1739) with more advanced CKD (eGFR 43.80 ± 5.58 ml/min/1.73m2). Similar numbers of CKD subjects were randomized to aggressive vs less intensive but lowering therapies, and 2057 CVE occurred in the CKD population. CVE rates were consistently higher in those with more advanced CKD. Statistically significant reductions in CVE were seen with aggressive therapy with A vs comparator therapies in both those with mild or more advanced CKD (mild CKD 28.54% vs 34.87%, HR 0.779 [0.700- 0.867], p<0.0001; advanced CKD 37.25% vs 44.04%, HR 0.795 [0.686-0.922] p=0.0025). Treatment interaction by mild/advanced CKD was nonsignificant.

Conclusion: Aggressive treatment with A was associated with statistically significant reductions in CVE in those with mild and more advanced CKD. Although additional studies are warranted, clinicians and future guidelines should give strong consideration to utilizing or recommending aggressive treatment for dyslipidemia in CHD patients with associated stage IIIb-IV CKD.

Reduced Left Ventricular Global Longitudinal Strain in Heart Transplant Recipients is Associated with an Increased One Year Mortality

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Background: Although a significant proportion of heart transplant (HTx) recipients have reduced left ventricular global longitudinal strain (LVGLS) at the early stages after HTx, no previous study has evaluated the associated risk for mortality. We hypothesized that reduced LVGLS shortly after HTx is associated with increased one year mortality among HTx recipients.

Methods: We included 176 consecutive adult primary single organ orthotopic HTx recipients. Echocardiography was performed 13±6 days post HTx. Peak systolic myocardial strain by 2-D speckle tracking echocardiography was assessed in 16 LV segments, and averaged to global strain - an index of global LV function.

Results: Assessment of strain was feasible in 167 (95%) patients. During the first year, 16 (10%) patients died 82±72 days after HTx. LVGLS was decreased in non-survivors compared to survivors (p<0.01). Only 4 (3 %) patients out of 144 with LVGLS better than -10% (-14.3±2.5 %) died the first year after HTx. In contrast, 11 (48 %) patients out of 23 with LVGLS worse than -10% (-7.8±1.8 %) died during the first year (p<0.05). Importantly, LVGLS was the only significant non-invasiv predictor of 1 year mortality in a Multivariate Cox regression analysis with HR 1.4 (95% CI 1.0-2.0) per 1 % decrease in strain (Table).

Conclusion: Markedly reduced LV function by GLS is related to poor prognosis in HTx recipients. Early assessment of LVGLS might therefore be a predictor of 1 year mortality in these patients.

Univariate and Multivariate Cox Regression Analyses for Survival at 1 Year After HTx (n=176)

	Univariate Cox Regression			Multivariate Cox Regression		
	HR	95% CI	p	HR	95% CI	p
Age (years)	1.08	1.00-1.15	0.044	1.02	0.93-1.11	0.71
Mean Arterial Pressure (mmHg)	0.94	0.91-0.97	<0.001	0.99	0.94-1.05	0.83
Donor Age (years)	1.06	1.10-1.11	0.012	1.00	0.95-1.06	1.00
Mean Pulmonary Artery Pressure (mmHg)	1.13	1.05-1.22	0.001	1.05	0.95-1.16	0.35
Cardiac Index (L/min/m2)	0.24	0.09-0.65	0.005	0.56	0.10-3.30	0.52
Pulmonal Vascular Resistance (WU)	4.60	2.65-7.86	<0.001	2.90	1.30-6.70	0.01
Left Ventricular Ejection Fraction (%)	0.88	0.83-0.93	<0.001	0.96	0.84-1.10	0.50
Left Ventricular Global Longitudinal Strain (%)	1.67	1.40-2.01	<0.001	1.43	1.04-1.95	0.03

Excellent Early Risk Stratification Using Troponins and N-Termnial Pro-Brain Natriuretic Peptides in Chest Pain Patients but no Additional Value of High-Sensitive Troponin T

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Background: Cardiac troponins and N-terminal pro-brain natriuretic peptide (NTproBNP) are useful for identifying chest pain patients at high risk. The aim was to examine whether early serial measurements of high sensitive troponin could further improve early risk stratification.

Methods: Troponin levels were determined by high sensitive troponin T (HsTnT), conventional TnT (Roche Diagnostics) and troponin I (Accu TnI, Beckman) assays on admission and at 2 hours in 231 consecutive chest pain patients with no ST-elevations. Patients were followed regarding death, rehospitalisation because of myocardial infarction(MI) or heart failure(HF).

Results: In ROC analyses, the maximum levels of HsTnT and NTproBNP provided the best diagnostic value. There was no additive value of measuring the change of levels. By combining HsTnT and NTproBNP patients could be divided into low-, intermediate- and high-risk groups (table). After adjusting for differences in baseline data, both log HsTnT (HR:1.4(1.1-1.7)) and log NTproBNP (HR:1.8(1.4-2.3)) predicted long-term outcome. When the prognostic value of HsTnT was compared with that of conventional TnT and TnI I in ROC analyses the AUCs(95%CI) at 3 months (0.78(0.69- 0.88), 0.79(0.70-0.88), 0.75(0.64-0.85))and at 24 months (0.73(0.65-0.80), 0.72(0.65-0.80), 0.69(0.61-0.77)) were similar.

Conclusion: The early use of cardiac troponins and NTproBNP provides excellent prognostic information in chest pain patients, but the additive value of Hs-TnT is limited.

Table

	HsTnT<14 and NTproBNP ≤300 (n=68)	HsTnT<14 and NTproBNP>300 (n=14)	HsTnT ≥14 and NTproBNP ≤300 (n=42)	HsTnT ≥14 and NTproBNP>300 (n=99)
3 months Death, MI, or HF	1(1.5%)	0(0%)	1(2.4%)	17(17.2%)
24 months Death, MI, or HF	1(1.5%)	2(14.3%)	3(7.1%)	34(34.3%)

Sex-Specific Models to Predict Near Term Risk of Myocardial Infarction

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Background: Gender disparities in the prognostic utility of several biomarkers have been reported for coronary heart disease (CHD). We conducted a study to identify biomarkers for assessing 5-yr risk of primary MI, stratified by sex.

Methods: We screened 52 proteins using assays developed de novo in baseline serum from 617 Norwegian adults drawn from the Tromsø Study 4, an observational health study. In this case-control design, subjects were CHD/diabetes-free at baseline (1994-95); 173 cases (57 female, 116 male) experienced a first MI during 5-year follow-up. Proteins contributing prognostic information beyond factors in the Framingham risk score (FRS: age, total cholesterol, HDL, systolic blood pressure, and smoking status) in sex-specific strata were identified using

multivariable logistic regression models selected by minimizing the Bayesian Information Criterion.

Results: The biomarker iCb3 independently predicted MI in women, (OR=0.6; p=0.007) but not men (OR=0.94; p=0.6) when adjusted for the FRS factors. When combined with the FRS factors, iCb3 significantly improved model fit (likelihood ratio test p=0.008) and discrimination (area under the ROC curve AUC=0.797) vs. a model with only FRS factors in women (AUC=0.775; p=0.04). Conversely, lipoprotein profile (LPP) independently predicted MI for men (adjusted OR=1.7; p=0.001), but not women (OR=0.99; p=0.95). In men, LPP improved fit vs. FRS factors alone (p=0.008) and discrimination (AUC=0.741 vs. 0.708; p=0.03). The combined iCb3 model (females) and LPP model (males) predictions had superior discrimination in the whole study (AUC=0.799) vs. the refit FRS model (AUC=0.779; p=0.02), and significant Net Reclassification Improvement (0.101; p<0.01). The FRS as published had an AUC of 0.752. Findings were confirmed using a bootstrap cross-validation technique to correct for possible over-fit.

Conclusions: In this population, the lipoprotein profile added incremental utility to the Framingham risk factors for men. Elevated levels of a serum protein associated with homeostatic control of inflammation, complement iCb3, independently indicated lower risk of MI in women, a novel finding to be verified in other populations.