Poster Session 1 (P1)

Sunday, June 24, 2018

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Assessing the Cause of Pulmonary Hypertension on Echo in the Absence of Tricuspid Regurgitation - a National Echo Database of Australia Study

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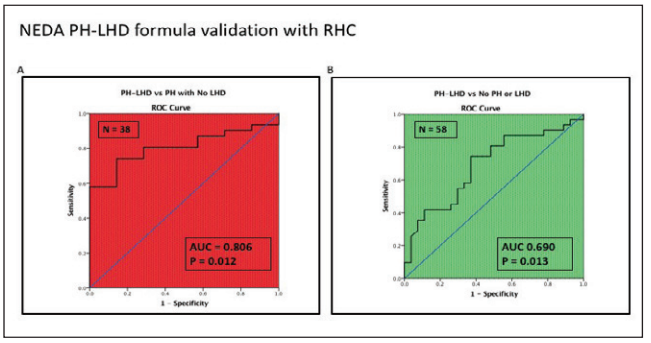
Background: PH-LHD is the most common cause of pulmonary hypertension (PH), associated with a high risk of death. Echo diagnosis of PH usually relies on a measurable TRV, but sufficient TR is absent in up to 40% of patients. Using the NEDA, we have previously described a method for predicting PH-LHD in the absence of a measurable TR velocity. Using age, E' velocity, E/E' ratio, E/A and left atrial volume index (LAVI) we created a model to diagnose PH-LHD. Tested in a cohort of 151,767 echos, our model is highly accurate in diagnosing PH-LHD. The NEDA PH-LHD Constant is ((Con) =-6.649 + (0.035 x Age) + (0.072 x E') + (0.077 x E/E') + (0.509 x E/A) + (0.03 x LAVI)), and the probability of having PH due to left heart disease = [EXP (Con) /[1+ (EXP(Con))] Aim: To validate the NEDA PH-LHD predictive model in patients undergoing right heart catheterisation. **Methods:** We analysed 887 consecutive patients from a West Australian tertiary centre who underwent RHC. They were divided into three groups: Group 1- those with PH-LHD (mPAP ≥ 25mmHg and PCWP ≥ 15mmHg), Group 2 - those with PH not due to LHD (mPAP >25mmHg and PCWP < 15mmHg) and Group 3 - those with no PH or LHD (mPAP <25mmHg and PCWP <15mmHg). To validate the NEDA PH-LHD model we compared Group 1 vs Group 2 in our first analysis and Group 1 vs Group 3 in our second analysis. We then applied these two probability analyses to a ROC curve to establish the accuracy of our model in predicting PH-LHD using RHC. **Results:** Age and the severity of PH was similar across groups, irrespective of causality. PVR was normal in those without PH, but was increased in patients with PH including those with PH-LHD suggesting a degree of pre-capillary PH in these patients. Patients with PH-LHD had higher left atrial filling pressures (PCWP=24.8+/-7mmHg, E/E'=21+/-10, E/A=1.7+/-1.1)(p=<0.0001). Figure 1 illustrates that the accuracy of our formula is 80% predictive in identifying patients with PH-LHD vs those with PH but no LHD, and 70% accurate in predicting PH-LHD when applied to patients with PH-LHD against those with no PH or LHD. **Conclusion:** In the absence of TRV, the NEDA PH-LHD model is 80% accurate in identifying patients with PH-LHD validated against RHC. Using only age and diastolic echo markers, our model is suited as an addition to echo software to automatically identify those likely to have PH who need further investigation.



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Prognosis of Patients with Reverse Stress-Induced Cardiomyopathy When Compared to Other Subtypes

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Background: Stress-induced cardiomyopathy (SCM) is characterized by an acute and transient left ventricular dysfunction. Most of the patients with SCM survive and have a good prognosis. However, the prognosis according to SCM subtypes is unclear since there is limited data for clinical characters and prognostic factors according to the subtypes. We investigated the prognosis of patients with SCM according to the subtypes. **Method:** Patients that had a diagnosis of SCM confirmed by diagnostic coronary angiography and transthoracic echocardiography were retrospectively reviewed from April 2015 to October 2016. We classified the as two groups. One was the reverse SCM group, whose predominate location of the regional left ventricular dysfunction was in the basal LV wall. The other group included classic as well as other SCM subtypes. Clinical and echocardiographic findings were compared between the two groups. The primary outcome was that all-cause death during follow-up. **Results:** Of 52 patients, 39 patients were allocated to the classic and other SCM group while 13 patients reverse SCM group. Among the 52 patients, six patients (11.5%) died during a mean follow-up period of 8.8 ± 5.9 months (median 8.5 months). In addition, 5 patients (9.6%) died within 1 month. The incidence of all cause death was significantly higher in the reverse SCM group compared with the classic and other SCM group (30.8% vs. 5.1%, $p = 0.029$). The incidence of 30-day all cause death was also significantly higher in the reverse SCM group in comparison to the classic and other SCM group (30.8% vs. 2.6%, $p = 0.011$). In addition, the cumulative death free survival rate was significantly lower in the reverse SCM group when compared to the classic and other SCM group (69.2% vs. 94.9%, Log Rank $p = 0.012$). This was also the case for the cumulative 30-day survival rate (69.2% vs. 97.4%, Log Rank p = 0.003). After adjustment for all the possible confounding factors, using the Cox proportional hazard regression analysis, reverse type was shown to have a 13.5 fold increased hazard for all cause death (odds ratio 13.5, 95% CI 1.599-114.554, $p = 0.017$). **Conclusion:** In patients with SCM, all-cause death rate was increased in the reverse subtype as compared to other types. Reverse SCM has been shown to be a good prognostic factor of death, even when taking into account the clinical confounding factors. Patients with SCM need focused evaluation of the predominant left ventricular dysfunction location in order to predict prognosis and appropriate treatment.

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Non-Alcoholic Fatty Liver Disease in Patients without Hypertension is Associated with Decreased Left Ventricular Dimensions and Mass

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Background: Hypertensive individuals with non-alcoholic fatty liver disease (NAFLD) have been reported to have increased left ventricular (LV) mass. Whether similar findings are observed in non-hypertensive individuals is not clear. **Methods:** We compared left heart measurements between adults with and without NAFLD in a cohort of 24,662 individuals with an echocardiogram, and after excluding those with left-sided valve disease, myocardial infarction, decreased left ventricular function, or hypertension. **Results:** There were differences between groups in demographics, comorbidities, and left heart dimensions (Table 1). After multivariate adjustment, individuals with NAFLD (vs. without) had decreased indexed LV end-diastolic diameter (-1.1mm/m^2, 95% CI -0.8 to -1.4, p<0.001), LV end-systolic diameter (-0.9mm/m^2, 95% CI -0.6 to -1.2, p<0.001); septal wall thickness (-0.1mm/m^2, 95% CI -0.03 to -0.3, p=0.01); LV mass (-2.8gm/m^2, 95% CI -1.0 to -4.9, p=0.01), and aortic root diameter (-0.6mm/m2, 95% CI -0.4 to -0.9, p<0.001); there was no difference in indexed posterior wall thickness (p=0.10) or left atrial diameter (p=0.70). Those with NAFLD had lower adjusted peak tricuspid regurgitation gradient (-1.9 mmHg, 95% CI -0.2 to -3.6 mmHg, p=0.03). **Conclusion:** Non-hypertensive individuals with NAFLD had decreased LV dimensions and mass, and lower tricuspid regurgitation gradients when compared to controls. This contrasts with prior findings of increased LV mass in hypertensive individuals with NALFD, and suggests the absence of hypertension is associated with an absence of cardiac remodeling in this population.

Table 1. Left Heart Measurements in Those With versus Without NAFLD

	NAFLD (n=503)	No NAFLD (n=24,159)	p
Demographics and Medical History			
Age	50.9±12.3	51.7±16.7	0.29
Female gender	50.1%	55.8%	0.01
Body mass index	32.8±7.4	28.7±7.3	<0.001
Body surface area	2.0±0.3	1.9±0.3	<0.001
Tobacco use	47.0%	44.4%	0.32
Diabetes	27.3%	7.3%	<0.001
Measurements (mm, gm, or mmHg)			
LV End-Diastolic Diameter	46.8±5.5	46.8±5.6	0.84
LV End-Systolic Diameter	29.7±5.3	30.1±5.4	0.15
Interventricular Septum	9.5±1.7	9.4±2.3	0.16
Posterior Wall	9.4±1.5	9.1±1.8	<0.001
LV Mass	153.5±43.3	149.7±53.3	0.11
Left Atrial Diameter	38.8±5.7	37.0±6.8	<0.001
Aortic Root Diameter	31.5±4.0	31.4±4.5	0.72
Peak Tricuspid Regurgitation Gradient	23.2±6.6	25.8±12.1	0.002
Measurements Indexed to BSA (mm/m² or gm/m²)			
LV End-Diastolic Diameter/BSA	23.1±3.0	24.6±3.4	<0.001
LV End-Systolic Diameter/BSA	14.7±2.7	15.8±3.0	<0.001
Interventricular Septum/BSA	4.8±0.9	5.1±1.2	<0.001
Posterior Wall/BSA	4.7±0.8	4.9±1.2	0.003
LV Mass/BSA	74.7±17.3	77.1±23.2	0.03
Left Atrial Diameter/BSA	19.2±2.9	19.3±3.4	0.32
Aortic Root Diameter/BSA	15.6±2.2	16.5±2.5	<0.001

BSA, body surface area; LV, left ventricle; NAFLD, non-alcoholic fatty liver disease