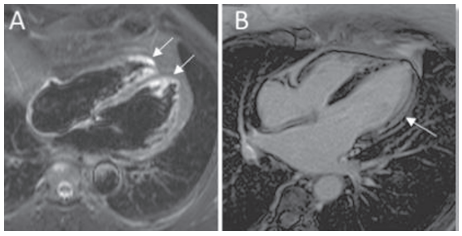
Results: We included 100 consecutive patients: 66 women and 34 men, with a mean age of 43.88±0.88 years. Most of the patients were from Bolivia; with an average time of residence in Spain of 9.73±0.47 years.42% of the patients were asymptomatic and in symtomatic patients the most frequent symptoms were chest pain and palpitations. In the analysis of ECG 37% of patients had normal parameters and 47% had two or more ECG abnormalities; 22% of the patients had alterations echocardiography (6% DTVI greater 55mm, 5% LVEF <50% and 17% alterations of segmental contractility in the inferior face and apex). Other abnormalities detected included: E'lateral <10 (35.4%) and increased VTDVI (31.8%) and increased AI volume (23%). In the analysis of myocardial deformity an overall longitudinal strain <20% was detected in 16% of the patients. In the study using CMR, the most common anomaly was an increase in VTSVI which was higher in men, and alterations of the segmental contractility in the middle and lower apical segments. Significant concordance between volumes and LVEF was detected by ECO and RMC. STIR detected edema in 16% and fibrosis in 18% of patients, more frequent intramyocardial and with apical and inferior predominance. The presence of delayed enhancement was significantly associated with lower LVEF and higher indexed LV end-diastolic volume and worse functional class. Using several complementary exploration methods, up to 92% of the patients presented any abnormality. The combination that most dramatically increased the ability to detect a cardiac abnormality was the ECG with a complete ECO. Adding advanced echocardiographic parameters such as strain or CMR did not clinically increase the rate of patients who presented cardiac abnormalities.



MRI-CHAGAS

Conclusions: The prevalence of cardiac involvement in Chagas disease may range from 61% with conventional criteria to 92% when new imaging parameters are used for assessing anatomy and cardiac function. Cardiac magnetic resonance allows to identify subtle alterations such as myocardial edema or small zones of fibrosis, not identifiable by echocardiography, which could help to identify those patients in whom a closer clinical follow-up should be considered in order to reduce cardiac morbidity and mortality.

P6215
Prognostic value of advanced lung cancer inflammation index in patients with chronic heart failure: a prospective comparative study with cardiac I-123 metaiodobenzylguanidine imaging

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Background: Recently, nutritional status and systemic inflammation are reported as robust prognostic factor in chronic heart failure (CHF) patients. Advanced lung cancer inflammation index (ALI), which is calculated as body mass index × serum albumin / neutrophil to lymphocyte ratio (NLR), is an independent prognostic marker in several types of cancer. On the other hand, cardiac I-123 metaiodobenzylguanidine (MIBG) imaging, which is useful for the estimation of cardiac adrenergic nerve activity, provides prognostic information in CHF patients. However, there is no information available on the comparison of prognostic value of cardiac MIBG imaging and ALI in CHF patients.
Methods and results: We studied 104 CHF outpatients with LVEF <40% in our prospective cohort study. The cardiac MIBG heart-to-mediastinum ratio (H/M) washout rate (WR) were calculated from the chest anterior view images obtained at 20 and 200 min after isotope injection. Abnormal WR was defined as WR≥27%. We also measured laboratory data and echocardiography at entry. During a follow up period of 6.3±4.5 years, 51 patients had cardiac events, defined as readmission for worsening heart failure or cardiac death. At multivariate Cox analysis, ALI (p=0.03), WR (p=0.001), serum sodium level (p=0.02), uric acid level (p=0.004) and LVEF (p=0.002) were significantly associated with cardiac event independently of serum creatinine level. The receiver operator characteristic curve (ROC) analysis revealed that ALI of 49.29 was a fair discriminator for cardiac event (area under the curve 0.743 (95% CI 0.648–0.824)). Kaplan-Meier analysis revealed that patients with low ALI had a significantly greater risk of cardiac event (68% vs 21% p<0.0001, adjusted HR 4.22 [2.05–8.70]). Kaplan-Meier analysis revealed that patients with both low ALI and abnormal WR had a significantly greater risk of the mortality than those with either low ALI or abnormal WR (85% vs 38% p=0.0005, adjusted HR 2.92 [1.56–5.46]). Furthermore, patients with either low ALI or abnormal WR also had a significantly greater risk of the mortality than those with neither low ALI nor abnormal WR (38% vs 14% p=0.016, adjusted HR 3.62 [1.19–11.04]).
Conclusion: The combination of ALI and cardiac MIBG imaging could provide the improved prediction of poor outcome in CHF patients.

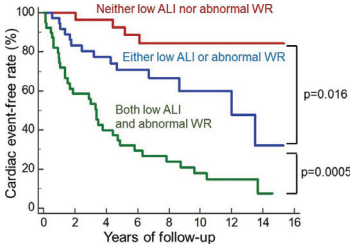

Figure 1

BEST POSTERS IN HEART AND VASCULAR DEVELOPMENT

P6217
Global expression profiling identifies a novel hyaluronan synthases 2 gene in the pathogenesis of lower extremity varicose vein

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Introduction: Lower extremities varicose veins (VV) are among the most easily recognized vascular abnormalities with superficial venous tortuosity and enlargement. The molecular mechanism and genetics of VV are largely unknown.
Purpose: In the present study, we sought to explore the global expressional change of VV and identify novel genes that might play a role in the mechanism of VV.
Methods: We used next-generation ribonucleic acid (RNA) sequence (RNA seq) technology to study the global messenger RNA expressional change in the venous samples of diseased and control patients.
Results: We identified several differentially expressed genes, which were further confirmed by conventional reverse transcription polymerase chain reaction (RT-PCR). Using these significant genes we performed in silico pathway analyses and found distinct transcriptional networks, such as angiogenesis, cell adhesion, vascular injury and carbohydrate metabolisms that might be involved in the mechanism of VV. Among these significant genes, we also found hyaluronan synthases 2 gene (HAS2) played a pivotal role and governed all these pathways. We further confirmed that HAS2 expression was down-regulated in the venous samples of patients with VV. Finally, we used a zebrafish model with fluorescence emitting vasculature and red blood cells to see the morphological changes of the venous system and blood flow. We found that HAS2 knockdown in zebrafish resulted in dilated venous structural with static venous flow.
Conclusions: HAS2 modulates the transcriptional networks of angiogenesis, cell adhesion, vascular injury and carbohydrate metabolisms and down-regulation of HAS2 may underlie the mechanism of VV. Therapeutic strategies targeting on HAS2 may be warranted to identify novel non-surgical treatment for VV.
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P6218
Identification of Latrophilin-2, a novel receptor that specifies cardiac progenitor cells from pluripotent stem cells and is essential for heart development

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The identification of a lineage-specific marker plays a pivotal role in understanding developmental process and is utilized to isolate a certain cell type with high purity for the therapeutic purpose. When mouse pluripotent stem cells were stimulated with BMP4, Activin A, bFGF and VEGF, they differentiated into cardiac cells. To screen cell-surface expressing molecules on cardiac progenitor cells compared to undifferentiated mouse iPS and ES cells, we isolated Flk1+/PDGFRa+ cells at differentiation day 4 and performed microarray analysis. Among candidates, we identified a new G protein-coupled receptor, latrophilin-2 (LPHN2). Here, we report a new cardiac-specific cell surface marker, latrophilin 2 (LPHN2), expressed specifically by cardiac progenitor cells (CPCs) and cardiomyocytes (CMCs) during mouse and human pluripotent stem cells (PSCs) differentiation in vitro and exclusively in the heart during mouse embryonic development. In sorting experiments under cardiac differentiation condition, LPHN2+ cells derived from pluripotent stem cells strongly expressed cardiac-related genes (Mesp1, Nkx2.5, aMHC and cTnT) and exclusively gave rise to beating cardiomyocytes, as compared with LPHN2– cells. Lphn2 knockout in mice is embryonically lethal owing to severe heart, but not vascular, defects. Interestingly, LPHN2+/– heterozygotes were alive and fertile. We also examined the importance of LPHN2 during heart devel-