

**P4216**

**Impact of balloon mitral valvuloplasty on left ventricular rotational deformation**

A. Samaan<sup>1</sup>, K. Said<sup>1</sup>, M. Hassan<sup>1</sup>, S. Romeih<sup>2</sup>, W. El Aroussy<sup>3</sup>, M. Fawzy<sup>3</sup>, M. Yacoub<sup>3</sup>. <sup>1</sup>Cairo University, Cardiovascular department, Cairo, Egypt; <sup>2</sup>Aswan Heart Centre, Radiology department, Aswan, Egypt; <sup>3</sup>Aswan Heart Centre, Aswan, Egypt

**Background:** Left ventricular (LV) twisting motion assists in ejecting blood from the LV during systole, while the untwisting motion, through the release of stored elastic energy, may contribute to early diastolic suction. No prior studies have evaluated changes in LV twist following balloon mitral valvuloplasty (BMV).

**Purpose:** To describe changes in LV rotational deformation in patients with rheumatic mitral stenosis (MS) using cardiac magnetic resonance imaging (CMR) 6 months and one year after successful BMV.

**Methods:** Thirty patients (median age 33 years, 22 women) with isolated severe rheumatic MS were studied. CMR myocardial tissue tagging was used for assessment of LV rotational deformation. LV twist was defined as the net difference between apical counterclockwise and basal clockwise rotation. To make the values comparable among different sizes ventricles, LV torsion was calculated as the twist value normalized to the length of the ventricle and multiplied by the mean radius at the base and apex. LV torsion was assessed at base-apex and base-mid levels. All patients had CMR studies before, 6 months and one year after successful BMV.

**Results:** At baseline, patients had a mitral valve area of 0.9 (0.6–1.3) cm<sup>2</sup>, mean pressure gradient of 12.5 (8–24) mmHg across the mitral valve and LV ejection fraction (LVEF) of 57 (range: 45–69) %.

Analysis of LV rotational deformation showed significant improvement in LV base-apex torsion at 6 months (3.3° vs. 2.5°, p<0.001) with a further improvement at one year (4.1° vs. 3.3°, p=0.05) following BMV. Similar pattern of change was seen in LV base-mid torsion with a significant increase at 6 months (3.6° vs. 2.3°, p<0.001) and a further increase at one year (4.7° vs. 3.6°, p=0.007). These changes were associated with a significant increase in LVEF (62% vs. 57%, p<0.001) at one year following BMV.

**Conclusions:** Among patients with isolated rheumatic severe MS, successful BMV is associated with a significant improvement in LV rotational deformation that is accompanied by a significant improvement in LV ejection fraction.

were found to be the best echocardiographic predictor of TR-ACR in this prospective study.

**P4218**

**Polymorphisms in Fc receptor genes are related to antibody-mediated rejection after heart transplantation**

G.M. Marron Linares<sup>1</sup>, L. Nunez Fernandez<sup>1</sup>, E. Alvarez-Lopez<sup>1</sup>, M.G. Crespo-Leiro<sup>2</sup>, E. Barge-Caballero<sup>2</sup>, J. Muniz-Garcia<sup>2</sup>, C.D. Tan<sup>3</sup>, E.R. Rodriguez<sup>3</sup>, J.M. Vazquez-Rodriguez<sup>2</sup>, M. Hermida-Prieto<sup>1</sup>. <sup>1</sup>Instituto de Investigación Biomédica de A Coruña (INIBIC)-CHUAC-UDC, Grupo de investigación en cardiología, A Coruña, Spain; <sup>2</sup>Complejo Hospitalario Universitario de A Coruña (CHUAC), Sergas-INIBIC-UDC, Department of Cardiology, A Coruna, Spain; <sup>3</sup>Cleveland Clinic, Department of Anatomic Pathology, Heart and Vascular Institute, Cleveland, United States of America

**Background:** Heart transplantation (HT) is a well-established life-saving treatment for patients presenting with end-stage cardiac failure. However, antibody-mediated rejection (AMR) represents one of the main problems after HT because of its diagnostic complexity and poor evidences supporting treatments. Fc receptors for IgG (FcγR) are involved in the activation of innate effector cells, antigen presentation, immune-complex-mediated maturation of dendritic cells, and regulation of B-cell activation that produce antibodies. Thus, Fc receptors could influence the development of AMR.

**Methods:** Genetic variants in 5 genes encoded to low affinity immunoglobulins gamma Fc region receptors (FCGR1A, FCGR2A, FCGR2B, FCGR3A, and FCGR3B) were analyzed by next generation sequencing in 46 HT patients, 23 with and 23 without AMR and 28 donors, 14 donors from patients with and 14 donors from patients without AMR.

**Results:** We have identified 3 SNPs [p.Gln63Trp and p.Pro215 (p=) in FCGR2A gene, and p.Leu102His/Arg in FCGR3A gene], which correlates with the development of AMR. Moreover, it has been showed that 2 SNPs in FCGR2A conforms an haplotype TGG-FCGR2A, associated with development of AMR in HT patients.

**Conclusions:** Polymorphisms in the genes related to FcγR could have an important role in the development of AMR.

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**ADVANCED HEART FAILURE: NEW ASPECTS**

**P4217**

**Ruling out acute cellular rejection in heart transplant recipients by classic and emergent echocardiographic factors: a multivariate, prospective, monocentric study**

S. Rodriguez Diego, M. Ruiz Ortiz, M. Delgado Ortega, J. Sanchez Fernandez, F. Carrasco Avalos, J. Lopez Aguilera, A. Lopez Granados, J.M. Arizon Del Prado, N. Paredes Hurtado, A. Luque Moreno, M.J. Oneto Fernandez, E. Martin Dorado, C. Ferreira Quero, M. Pan Alvarez-Ossorio, D. Mesa Rubio. *University Hospital Reina Sofia, Cardiology, Cordoba, Spain*

**Purpose:** Our aim was to find the best echocardiographic parameters for ruling out treatment-requiring acute cellular rejection (TR-ACR).

**Methods:** From September 2014 to October 2017 we performed a comprehensive echocardiographic exam in 37 consecutive adult heart transplant recipients in their first year post-transplantation within 3 hours of the routine surveillance endomyocardial biopsies (EMB) in a single centre. Classic and emergent echocardiographic parameters (including strain analysis), were analyzed and independent predictors of TR-ACR (grade ≥2R) were investigated.

**Results:** A total of 251 pairs of EMB and echo exams were performed, 117 with no rejection (grade 0R), 99 with rejection grade 1R and 35 with rejection grade ≥2R (TR-ACR). Multivariate analysis (Table) identified the sum of lateral mitral annulus systolic (s') and early diastolic (e') velocities, in absolute values, measured by means of tissue Doppler echocardiography (s'+e') as the best single echocardiographic parameter for ruling out TR-ACR, with a C statistic of 0.79 (95% CI 0.71–0.87, p<0.0005) by ROC curve analysis. A s'+e' value ≥23 cm/s, present in 43% of studies, had a negative predictive value of 97.1% for TR-ACR.

| Variable                           | TR-ACR         | Rest of studies | Odds Ratio       | p value |
|------------------------------------|----------------|-----------------|------------------|---------|
| <b>Univariate analysis*</b>        |                |                 |                  |         |
| TAPSE (mm)                         | 10.7±4         | 13.5±4.1        | 0.82 (0.73–0.92) | 0.001   |
| Mitral e'+s' lateral waves (cm/s)  | 16.8±4.3       | 22.6±5.5        | 0.80 (0.74–0.87) | <0.0005 |
| Mitral E/e' septal ratio           | 9.0 (7.7–12.5) | 10.5 (8.4–13.6) | 0.89 (0.80–0.99) | 0.04    |
| LV radial strain (%)               | 28.1±9.3       | 32.4±10.6       | 0.95 (0.91–0.99) | 0.03    |
| RVFW longitudinal peak strain (%)  | -17.5±4.9      | -20.7±5.8       | 1.12 (1.04–1.21) | 0.004   |
| RVFW longitudinal strain rate (/s) | -2.1±0.6       | -2.4±0.7        | 2.19 (1.13–4.24) | 0.02    |
| <b>Multivariate analysis*</b>      |                |                 |                  |         |
| Mitral e'+s' lateral waves (cm/s)  | 16.8±4.3       | 22.6±5.5        | 0.80 (0.73–0.88) | <0.0005 |
| Mitral E/e' septal ratio           | 9.0 (7.7–12.5) | 10.5 (8.4–13.6) | 0.83 (0.72–0.96) | 0.01    |
| RVFW longitudinal peak strain (%)  | -17.5±4.9      | -20.7±5.8       | 1.11 (1.01–1.21) | 0.03    |

Values are mean ± standard deviation if normally distributed or median (interquartile range), otherwise. \*Binary logistic regression (only selected parameters with p<0.05 are shown for univariate analysis). Abbreviations: LV: left ventricle; RVFW: right ventricle free wall; TAPSE: tricuspid annulus plane systolic excursion; TR-ACR: treatment requiring acute cellular rejection.

**Conclusion:** Lateral mitral annulus velocities, a widely available echo parameter,

**P4219**

**Increase of Trypanosoma cruzi parasitic load in endomyocardial biopsies anticipate Chagas disease reactivation after heart transplantation**

L.A. Benvenuti<sup>1</sup>, A. Roggerio<sup>1</sup>, A.S. Nishiya<sup>2</sup>, J.E. Levi<sup>2</sup>. <sup>1</sup>Heart Institute (InCor), University of São Paulo Medical School, São Paulo, Brazil; <sup>2</sup>Molecular Biology Department, Fundação Pró-Sangue/São Paulo Blood Center, São Paulo, Brazil

**Background:** Chagas disease is caused by the protozoan Trypanosoma cruzi. Heart transplantation is a therapeutic option for end-stage chagasic cardiomyopathy, which develops in 20% of symptomatic, chronically infected people. Due to immunosuppression, Chagas disease reactivation (CDR) characterized by inflammatory cell infiltration with T. cruzi parasites in the myocardium can affect the allograft. CDR should be distinguished from acute cellular rejection and promptly treated. Careful histopathological search for T. cruzi parasites is mandatory in endomyocardial biopsies (EMB) of heart-transplanted, chagasic patients and CDR diagnosis is based on the direct detection of parasites.

| Patient #1                           |    |    |         |  |
|--------------------------------------|----|----|---------|--|
| Days before CDR                      | 57 | 50 | 0 (CDR) |  |
| Parasitic load/10 <sup>6</sup> cells | 0  | 0  | 190000  |  |

  

| Patient #2                           |     |    |    |     |     |         |
|--------------------------------------|-----|----|----|-----|-----|---------|
| Days before CDR                      | 125 | 93 | 64 | 22  | 9   | 0 (CDR) |
| Parasitic load/10 <sup>6</sup> cells | 0   | 0  | 0  | 625 | 354 | 6660    |

  

| Patient #3                           |    |    |    |      |      |         |
|--------------------------------------|----|----|----|------|------|---------|
| Days before CDR                      | 96 | 85 | 78 | 58   | 13   | 0 (CDR) |
| Parasitic load/10 <sup>6</sup> cells | 0  | 0  | 0  | 14.7 | 13.5 | 797     |

  

| Patient #4                           |    |    |         |  |
|--------------------------------------|----|----|---------|--|
| Days before CDR                      | 64 | 50 | 0 (CDR) |  |
| Parasitic load/10 <sup>6</sup> cells | 0  | 0  | 22.8    |  |

  

| Patient #5                           |    |    |     |         |
|--------------------------------------|----|----|-----|---------|
| Days before CDR                      | 58 | 31 | 16  | 0 (CDR) |
| Parasitic load/10 <sup>6</sup> cells | 0  | 0  | 237 | 20700   |

  

| Patient #6                           |    |    |         |  |
|--------------------------------------|----|----|---------|--|
| Days before CDR                      | 64 | 57 | 0 (CDR) |  |
| Parasitic load/10 <sup>6</sup> cells | 0  | 0  | 1640    |  |

  

| Patient #7                           |     |    |         |  |
|--------------------------------------|-----|----|---------|--|
| Days before CDR                      | 104 | 70 | 0 (CDR) |  |
| Parasitic load/10 <sup>6</sup> cells | 0   | 0  | 635     |  |

  

| Patient #8                           |     |     |     |    |     |         |
|--------------------------------------|-----|-----|-----|----|-----|---------|
| Days before CDR                      | 134 | 121 | 100 | 70 | 30  | 0 (CDR) |
| Parasitic load/10 <sup>6</sup> cells | 0   | 0   | 0   | 0  | 126 | NA      |

NA: not available  
T. cruzi parasitic load of EMB