

Left ventricular torsion in clinically stable child heart transplant recipients

Pac F.A.(1), Koca S.(1), Kavurt V. (1), Ece I.(1), Eris D.(1), Kervan U. (2), Sabitoglu S. (2), Sert D. (2), Pac M.(2)

¹ Department of Pediatric Cardiology, Türkiye Yüksek İhtisas Hospital, Ankara

² Department of Cardiovascular Surgery, Türkiye Yüksek İhtisas Hospital, Ankara

Background and aim

Left ventricular (LV) twist function, defined as the wringing motion of the cavity secondary to the counter directional rotation of its apex and base, has an important role for both systolic and diastolic functions (1). LV twist assists ejection, whereas rapid untwist secondary to the release of stored elastic energy during early diastole enhances LV suction by augmenting intraventricular pressure gradients, and hence allowing ventricular filling at relatively low left atrial pressure (2-5). In adult heart transplant recipients, LV twist dynamics are significantly impaired (6). This study aimed at exploring LV twist in our first ten child heart transplant (HT) recipients.

Methods

Patients

Thirteen children underwent heart transplantation at our hospital in the last four years. A total of 10 child who underwent heart transplantation and currently followed at our hospital Ankara, Türkiye were included in this report. All children who had undergone orthotopic human cardiac allograft transplantation, had received standard postoperative care and were on immunosuppressive therapy. All child HT recipients (6 female, 4 male, transplanted at 3 1/12- 17 3/12 years of age, between July 2013 and September 2016) underwent scheduled cardiac catheterization and endomyocardial biopsy at, 2 weeks, 4 weeks, 4 months, 1 year and 1.5 years, and then every 6 months or 1 year after transplant surgery or when rejection was suspected. Time after HT was defined as the time between the heart transplantation and the last echocardiographic examination.

Echocardiography

Two LV short-axis planes obtained at the basal and the apical levels were acquired using a Vivid 7 machine (GE Healthcare, Milwaukee, WI) with an 7S or M4S probe at 75 to 125 frames/s. At each plane, 3 consecutive cardiac cycles were acquired during a breath hold at endexpiration. In all children, 3 cardiac cycles at end-expiration on the respiratory trace were selected. Two dimensional and colour M-mode echocardiography with speckle-tracking analysis were completed in six child HT recipients. By convention, clockwise rotation as viewed from the apex was expressed as a negative angle and counter-clockwise rotation as a positive angle. LV torsion was calculated as the instantaneous net difference in mean rotation between the apical and basal levels.

Results

Main characteristics, two dimensional echocardiography, colour M-mode echocardiography, strain echocardiography and LV torsion analysis were shown in Table 1a and 1b.

LV twist dynamics and strain parameters are normal in our ten HT recipients after one month later from transplantation. But LV twist dynamics significantly impaired after four months later from transplantation, even in comparison with controls. (Figure 1, Table 1b) There was no rejection in any biopsy except one patients first biopsy. This patient therated by pulse steroid succesfully. There was no any biopsy related complication.

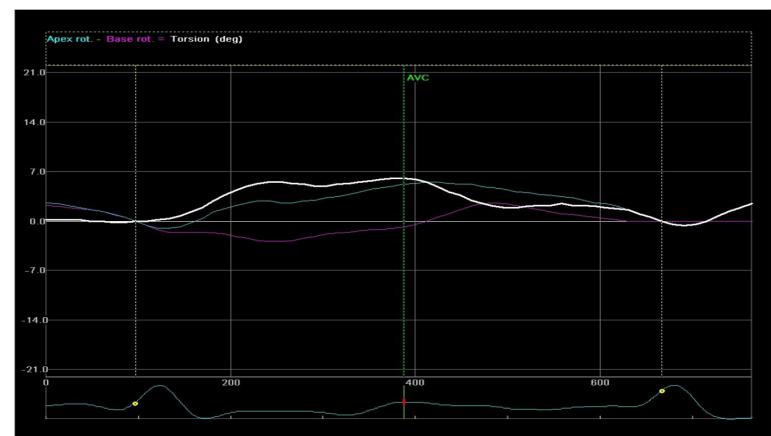


Figure 1: Patient 1 's impaired LV torsion at four months after transplantation

Discussion

Several mechanisms may individually or in combination explain worsening of our HT patients LV torsion. Cardiac denervation with transplantation is often followed by incomplete and heterogeneous cardiac reinnervation, resulting in an inadequate stimulation of LV myocardial betareceptors. As LV twist is sensitive to sympathetic stimuli, it is likely to be blunted early after HT with reduced twist angle and untwist rate. A complex interaction between LV myocardial fibrosis and cellular remodeling, mediated by hypoxia-related pro-angiogenic signals and increased activation of both renin-angiotensin and transforming growth factor-beta systems, may also be a factor.

Moreover, the potential impact of immunosuppressive therapy coupled with pre-transplant vascular and subendocardial dysfunction, frequently occurring in HT subjects via an ischemia-reperfusion injury mechanism, could be a third explanation (6).

LV torsion analysis could be a useful non-invasive approach for early detection of subclinical cellular cardiac rejection also. But further investigation will be necessary to detect the utility of LV torsion derived from strain echocardiography as a screening tool for detecting allograft rejection in child HT recipients.

Conclusion

Although the stain measurements were normal after 4 weeks of heart transplantation, LV twist function is significantly abnormal in stable heart transplant patients. Time after HT was the main predictor of worsening in LV twist dynamics in heart transplant patients.

Table 1a: Main characteristics, cardiac biopsies and left ventricular functions of patients

| | Patient 1 | Patient 2 | Patient 3 | Patient 4 | Patient 5 | Patient 6 | Patient 7 | Patient 8 | Patient 9 | Patient 10 |
|-----------------------|-----------------|------------|------------|-------------|-----------|-----------|-----------|-----------|-----------|------------|
| Age | 14 1/12 | 5 9/12 | 11 8/12 | 13 6/12 | 6 9/12 | 18 1/12 | 13 7/12 | 7 1/12 | 16 8/12 | 12 8/12 |
| Gender | M | M | M | F | F | F | F | F | F | M |
| Height (cm) | 145 | 101 | 122 | 145 | 109 | 158 | 145 | 110 | 155 | 125 |
| Weight (kg) | 34 | 16 | 24 | 34 | 25 | 58 | 45 | 25 | 50 | 40 |
| Time after HT (month) | 33 | 44 | 33 | 27 | 9 | 8 | 7 | 7 | 8 | 6 |
| Cardiac biopsy | 1 mo Gr 2 RJ +* | Gr 1 RJ +* | Gr 0 NRJ - | Gr 0 NRJ - | Gr 1 RJ | Gr 0 RJ | Gr 0 RJ | Gr 0 | Gr 1R | Gr 0 |
| ISHLT 2004 Score | 3 mo Gr 0 RJ - | Gr 1 RJ +* | Gr 1R RJ + | Gr 0 NRJ -* | Gr 0 RJ* | Gr 0 RJ | Gr 0 RJ* | Gr 0 | Gr 0 | Gr 0 |
| ISHLT 2005 Score* | 6 mo Gr 0 RJ - | Gr 0 RJ - | Gr 1R RJ + | Gr 0 NRJ* | Gr 0 RJ | Gr 0 RJ* | - | Gr 0 | - | Gr 1R |
| | 12 mo Gr 0 RJ - | Gr 0 RJ - | Gr 0 NRJ - | Gr 1 RJ - | - | - | - | - | - | - |
| | 18 mo - | - | - | - | - | - | - | - | - | - |
| | 24 mo - | - | - | Gr 0 NRJ - | - | - | - | - | - | - |
| | 30 mo - | - | - | - | - | - | - | - | - | - |
| LV EF (%) | 1 mo 62 | 69 | 61 | 60 | 68 | 64 | 66 | 62 | 66 | 68 |
| | 3 mo 68 | 66 | 67 | 62 | 66 | 62 | 64 | 64 | 75 | 73 |
| | 6 mo 65 | 61 | 63 | 68 | 67 | 67 | 68 | 72 | 64 | 64 |
| | 12 mo 68 | 65 | 59 | 67 | - | - | - | - | - | - |
| | 18 mo 65 | 62 | 62 | 67 | - | - | - | - | - | - |
| | 24 mo 62 | 59 | 64 | 71 | - | - | - | - | - | - |
| | 30 mo 76 | 62 | 75 | - | - | - | - | - | - | - |

LV EF: Left ventricular ejection fraction HT: Heart transplantation

Table 1b: Strain parameters and left ventricular torsion of patients

| | Heart Transplantation Group (mean ± SD) | | | | | | | Control Group (n:10) (mean ± SD) |
|------------|---|-------------|-------------|-------------|-------------|-------------|--------------|----------------------------------|
| | 1.mo (n:10) | 3.mo (n:10) | 6.mo (n:10) | 12.mo (n:4) | 18.mo (n:4) | 24.mo (n:4) | 30. mo (n:3) | |
| GLS | -17.2±3.2 | -18.7±3.3 | -18.1±4.2 | -19.1±4.3 | -17.7±3.8 | -18.6±3.3 | -17.8±2.3 | -19.7±4.8 |
| 4 Ch LS | -17.9±4.2 | -18.7±4.1 | -18.3±3.5 | -18.1±4.8 | -19.1±3.4 | -18.7±4.3 | -18.3±3.2 | -18.9±4.1 |
| 3 Ch LS | -18.1±4.3 | -18.6±5.1 | -19.2±5.1 | -18.7±3.2 | -17.2±6.2 | -16.7±3.4 | -16.3±3.1 | -18.8±4.7 |
| BASAL CS | -21.7±4.9 | -20.1±3.9 | -20.5±4.1 | -19.7±4.8 | -20.1±3.9 | -22.4±3.8 | -21.4±3.3 | -22.5±6.3 |
| APICAL CS | -26.8±6.9 | -25.4±6.4 | -25.3±4.9 | -24.9±3.6 | -26.1±2.8 | -28.7±2.9 | -27.7±2.2 | -27.9±6.7 |
| BASAL RS | -48.4±6.8 | -46.9±9.3 | -47.4±6.3 | -49.3±8.1 | -50.4±4.8 | -48.2±7.5 | -46.2±7.1 | -50.4±9.8 |
| APICAL RS | -38.8±5.2 | -39.4±5.8 | -38.9±3.9 | -39.1±3.2 | -37.4±6.8 | -38.4±2.8 | -37.4±2.2 | -40.4±6.8 |
| LV TORSION | 11.6±1.1 | 9.7±1.1* | 7.6±1.4* | 8.15±0.9* | 7.96±0.3* | 8.16±0.85 | 7.16±0.55* | 12.9±1.9 |

GLS: Global Longitudinal strain, 4 Ch LS: Four chamber longitudinal strain, 3 Ch LS: Three chamber longitudinal strain, CS: Circumferential strain RS: Radial strain

*: p < 0,05

References

- Sengupta PP, Tajik AJ, Chandrasekaran K. Twist mechanics of the left ventricle: principles and applications. JACC Cardiovasc Imaging 2008;1:366-76.
- Beyar R, Sideman S. Effect of the twisting motion on the nonuniformities of transmural fiber mechanics and energy demand—a theoretical study. IEEE Trans Biomed Eng 1985;32:764-9.
- Cameli M, Ballo P, Righini FM, Caputo M, Lisi M, Mondillo S. Physiologic determinants of left ventricular systolic torsion assessed by speckle tracking echocardiography in healthy subjects. Echocardiography 2011 Jul;28(6):641-8.
- Nikolic SD, Feneley MP, Pajaro OE, Rankin JS, Yellin EL. Origin of regional pressure gradients in the left ventricle during early diastole. Am J Physiol Heart Circ Physiol 1995;268:H550-7.
- Notomi Y, Martin-Miklovic MG, Orszak SJ, et al. Enhanced ventricular untwisting during exercise: a mechanistic manifestation of elastic recoil described by Doppler tissue imaging. Circulation 2006;113:2524-33.
- Cameli M, Ballo P, Lisi M, Benincasa S, Focardi M, Bernazzali S, Lisi G, Maccherini M, Henein M, Mondillo S. Left ventricular twist in clinically stable heart transplantation recipients: a speckle tracking echocardiography study. Int J Cardiol. 2013 Sep 20;168(1):357-61.