Left ventricular (LV) twist function, defined as the twirling 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 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, Turkey 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, 6 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.

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 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 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 strain 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 function of patients

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

References