Stroke Volume Ratio Predicts Redilatation of the Right Ventricle After Pulmonary Valve Replacement

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CONGENITAL HEART

Background. This study aimed to identify the prognostic value of the preoperative stroke volume ratio (right ventricular stroke volume/left ventricular stroke volume) for redilatation of the right ventricle after pulmonary valve replacement in patients with repaired tetralogy of Fallot.

Methods. From April 2004 to November 2013, 20 patients with repaired tetralogy of Fallot underwent pulmonary valve replacement for pulmonary valve regurgitation and right ventricular dilatation. Serial changes in ventricular volume were examined by cardiac magnetic resonance or computed tomography imaging. The redilatation ratio was calculated for right ventricular end-diastolic and end-systolic volume indices by dividing the increment in right ventricular volume from the first (median, 1.1 years) to the second (median, 3.2 years) evaluations after pulmonary valve replacement by the first evaluation. The relationships between the stroke volume ratio and redilatation ratio were assessed. The degree of right ventricular myocardial fibrosis was examined in 13 patients and compared with the stroke volume ratio.

Results. Right ventricular volume (redilatation) significantly increased from a median of 1.1 to 3.2 years after pulmonary valve replacement. Significant positive correlations were detected between the stroke volume ratio and redilatation ratio of the right ventricular end-diastolic ($r = 0.50$. $p = 0.02$) and end-systolic volume indices ($r = 0.49$, $p = 0.03$). The stroke volume ratio also showed a significant positive correlation with the degree of right ventricular myocardial fibrosis ($r = 0.73$, $p = 0.005$).

Conclusions. The preoperative stroke volume ratio can predict redilatation of the right ventricle after pulmonary valve replacement and the degree of right ventricular myocardial fibrosis.

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Pulmonary valve regurgitation (PR) is the most common complication in patients with repaired tetralogy of Fallot (TOF) [1]. The resultant chronic right ventricular (RV) volume overload can lead to RV dilatation, biventricular dysfunction, heart failure symptoms, arrhythmias, and death [2–4]. Tricuspid regurgitation (TR) is often complicated with RV dilatation and might promote deleterious outcomes [5]. Although the precise timing of pulmonary valve replacement (PVR) is still not established, timely PVR has been shown to improve RV dilatation and heart failure symptoms [6–8]. Recent reports have shown favorable outcomes regarding survival and freedom from reintervention after PVR [9]. Therefore, an important issue in this field that should be focused on is late outcomes of RV function.

Since 2005 we have routinely performed image analysis of ventricular volume in patients after PVR at 1 and 3 years postoperatively. Although RV volume is normally decreased after PVR, some patients show an increase in RV volume from 1 to 3 years after PVR (redilatation), even in the absence of any persistent valvular lesions. The current indication of PVR consists of clinical features and the degree of RV dilatation as evaluated by cardiac magnetic resonance (CMR) imaging, and these have been established as a predictor of RV remodeling [10, 11]. However, the predictors of late RV redilatation after PVR have not been investigated.

Previous reports regarding left ventricular (LV) remodeling have shown that the extent of myocardial fibrosis might be useful for predicting progression of LV dilatation [12]. Chronic RV volume overload resulting from PR is likely to lead to RV myocardial fibrosis in patients with repaired TOF [13, 14]. This situation is similar to that in the LV with chronic aortic valve disease [15–17]. We previously reported the development of myocardial fibrosis in the canine RV in response to PR-related volume overload [18]. The degree of RV myocardial fibrosis before PVR has the potential to predict late outcomes of RV function. However, myocardial biopsy with cardiac catheterization is invasive, and the results are inconsistent according to the examined region.

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Abbreviations and Acronyms

CMR = cardiac magnetic resonance
CT = computed tomography
LV = left ventricle
LVEDV = left ventricular end-diastolic volume
LVEDVI = left ventricular end-diastolic volume index
LVESV = left ventricular end-systolic volume
LVESVI = left ventricular end-systolic volume index
PR = pulmonary valve regurgitation
PVR = pulmonary valve replacement
RV = right ventricle
RVEDV = right ventricular end-diastolic volume
RVEDVI = right ventricular end-diastolic volume index
RVESV = right ventricular end-systolic volume
RVESVI = right ventricular end-systolic volume index
SVR = stroke volume ratio
TOF = tetralogy of Fallot
TR = tricuspid regurgitation

The stroke volume ratio (SVR), which was originally calculated as LV stroke volume/RV stroke volume, was initially reported as an index of LV volume overload in patients with aortic valve and mitral valve regurgitation [19]. We hypothesized that the SVR is a sensitive predictor of RV volume overload in patients with PR and TR after repaired TOF without any other valvular lesion. The presented study aimed to identify the predictive value of the SVR for RV redilatation after PVR and pathologic changes in RV myocardium in patients with repaired TOF.

Patients and Methods

The Osaka University Hospital Institutional Review Board approved this retrospective study.

Patients

From April 2004 to November 2013, 20 adult patients (10 men; median age at PVR of 37 years) with repaired TOF underwent PVR for PR and RV dilatation at Osaka University Hospital. Patients with significant pulmonary valve stenosis or left-sided valvular abnormalities were excluded. The median interval from corrective operation was 33 years. Preoperative New York Heart Association Functional Classification was I in 13 patients (65%), II in 5 (25%), and III in 2 (10%). Atrial tachyarrhythmia was observed in 3 patients (15%). Cardiac catheterization was performed in all patients at a median of 81 days before the operation. No patients showed greater than moderate pulmonary stenosis, and the median RV systolic pressure was 44 mm Hg (range, 27 to 58 mm Hg).

Echocardiography was performed concurrently with cardiac catheterization, and greater than moderate TR was found in 11 patients (55%). CMR imaging or computed tomography (CT) was performed at a median of 189 days before the operation and showed that the median RV end-diastolic volume index (RVEDVI) was 176 mL/m^2 (range, 120 to 261 mL/m^2), and the median RV end-systolic volume index (RVESVI) was 83 mL/m^2 (range, 51 to 153 mL/m^2). Baseline and surgical characteristics of the patients are summarized in Table 1.

Surgical Procedure

Our indication for PVR in this study period was symptoms and signs caused by RV volume overload, including palpitation, pedal edema, or atrial tachyarrhythmia. For asymptomatic patients, we scheduled PVR when the RVEDVI as assessed by CMR imaging or CT approached 150 mL/m^2. Three patients underwent PVR based on symptoms without an RVEDVI greater than 150 mL/m^2.

PVR was performed through median sternotomy using cardiopulmonary bypass with mild hypothermia. Cardiac arrest was conducted in 12 patients. A longitudinal incision was performed into the pulmonary trunk or a previously placed patch. Reduction plasty of the infundibulum and complete removal of a previously placed patch was performed, if necessary. PVR was performed using a biologic valve with a continuous suture in all of the patients. Concomitant procedures included tricuspid annuloplasty in 17 patients, the right-side maze procedure in 7, and pacemaker implantation in 1.

To eliminate the effect of histologic changes induced from the previous operation, an RV myocardial biopsy specimen was obtained from a portion of RV outflow that was sufficiently remote from the outflow tract. Thirteen patients agreed to participate in histologic analysis.

Study Methods

RV and LV volume was evaluated with CMR imaging in 12 patients. In 8 patients who had contraindications for CMR imaging, ventricular volume was evaluated with CT, which has been recognized as a comparable modality with CMR for measurements of RV and LV size [20, 21].

CMR studies were conducted using a 1.5-T system (Gyroscan NT; Philips Healthcare, Best, Amsterdam, The Netherlands), and CMR analysis was performed on an offline workstation (View Forum: Philips Medical Systems, Best, The Netherlands). Ventricular volume was calculated by tracing the end-diastolic and end-systolic ventricular slice contours and using the modified Simpson rule method. End-diastolic and end-systolic volumes were defined as the largest and the smallest ventricular cavity, respectively.

CT examinations were obtained using a 320-row CT scanner (Aquilion ONE; Toshiba Medical Systems, Otawara, Japan) at 150 to 550 mA according to body weight, tube voltage of 120 kVp, and a slice collimation of 0.5 mm. The gantry period was 0.35 seconds. Intravenous Landiolol (Ono Pharmaceutical Co, Ltd, Osaka, Japan) was administered as tolerated for a baseline heart rate of 60 beats/min. After obtaining noncontrast localization images, an iodinated contrast agent (300 mg/mL; Iopamiron, Bayer Healthcare, Osaka, Japan) was administered intravenously