Left ventricle dysfunction in patients with critical neonatal pulmonary stenosis: echocardiographic predictors

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Background: The aim of this study is to identify echocardiographic predictors of transient left ventricle dysfunction after pulmonary valve balloon dilatation (PVBD), in neonates with pulmonary valve stenosis (PVS) and atresia with intact septum (PAIVS) at birth. **Methods:** The study includes patients admitted at the Bambino Gesù Children Hospital from January 2012 to January 2017. Clinical, echocardiographic and cardiac catheterization data before and after PVBD were retrospectively analyzed. **Results:** Twenty-nine infants were included in the study (21 male and 8 female). The median age was 9±6 days. Eight patients developed transient LV dysfunction (3 PAIVS and 5 PVS) and comparing data before and after the procedure, there was no difference in right ventricle geometrical and functional parameters except for evidence of at least moderate pulmonary valve regurgitation after PVBD. **Conclusion:** Moderate to severe degree pulmonary valve regurgitation was significant associated to LV dysfunction ($p<0.05$) in PVS and PAIVS patients.

ORIGINAL ARTICLE

Left ventricle dysfunction in patients with critical neonatal pulmonary stenosis: echocardiographic predictors.

A single-center study

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Introduction

The pulmonary stenosis (PS) and pulmonary atresia with an intact ventricular septum (PAIVS) occur in 25-30% of all congenital heart disease and are forms of cyanotic congenital heart disease where pulmonary blood flow depends on the ductus arteriosus. Pulmonary stenosis (PS) consists in the narrowing of the right ventricle outflow (RVOT) and the most severe form is the pulmonary atresia. **[1]**

Pulmonary atresia with an intact ventricular septum is a complex and uncommon congenital heart defect. It is associated with a relatively high morbidity and mortality, either with no treatment or with surgical or catheter treatment.

The main treatment goal is to achieve anterograde flow across the RVOT, in the neonatal period, to improve the systemic arterial oxygenation.

Trans-catheter intervention with pulmonary valve balloon dilatation (PVBD) with or without previous pulmonary valve perforation represents the primary therapy for infant with critical PS and PAIVS, when it is possible. It is critical that the right ventricle morphology is adequate for PVBD (large, tripartite right ventricle with pure vulvar atresia without coronary artery connections and coronary dependent circulation or with interrupted coronary arteries). **[2]**

It is widely demonstrated that PV perforation and /or BD is characterized by a high rate of initial success with acute gradient reduction, prostaglandin independence and low rate of reintervention. In fact, this procedure permits to obtain a successful growth of both the right ventricle and pulmonary valve.

Despite this, significant left ventricle dysfunction can occur in some infants with PS or PAIVS after PVBD.

Ronai et al. for the first time have given emphasis to this transient effect on left ventricle, supposing it was a consequence of three different hemodynamic effects: the impact of change in size of right ventricle, the loading effect for closure of ductus arteriosus or the acute change in coronary perfusion post-cardiac catheterization. **[3]**

In previous studies the pre-existing left ventricle global and regional dysfunction detected in patients with mild coronary anomalies with PS and PAIVS, before the relief of the RVOT obstruction, have been demonstrated to be predictive risk factors of left ventricle dysfunction after PV perforation and/or BD. **[4-6]**

In this study, we want to contribute to clarify the risk factors and hemodynamics mechanisms in patients with PS and/or PAIVS, with normal left ventricle and without coronary anomalies, for developing transient left ventricle dysfunction after PVBD.

Methods

Study population

We retrospectively enrolled patients admitted at the Bambino Gesù Children Hospital with diagnosis of neonatal PS and/or PAIVS from January 2012 to January 2017 with an available complete echocardiographic examination suitable for strain analysis, angiograms and operative reports.

Data were retrieved from the clinical records and a dedicate database was collected.

Management of patients

The patients, after the diagnosis of PS or PAIVS received intravenous prostaglandin E1 at an initial dose that ranged between 0.005 and 0.1 ng/kg/min.

After clinical stabilization patients were transferred to the catheterization laboratory. The radiofrequency perforation, when necessary, was performed with 2 F cable with 5 W of energy administered for 1-2 sec under fluoroscopic guidance. In case of success, the pulmonary valve was dilated with a balloon measuring 1.2-1.4 times the pulmonary annulus. The procedure was considered effective if the RVOT final gradient was less than 30 mmHg.

Prostaglandin infusion was discontinued some days after the creation of anterograde pulmonary blood flow and with an oxygen saturation > 92%.

Ductal stent placement or a systemic-to-pulmonary shunt were the treatment options if an additional shunt was required for impossibility to wean patients off prostaglandin.

Imaging methods

Patients included in our study underwent complete echocardiographic exam, including two-dimensional and Color-Doppler method using a Philips iE33 (Philips Medical System, Bothell, WA, USA) with 8- and 12-MHz transducers before and after the pulmonary valve balloon dilatation. Images were analyzed with a dedicated off-line review software (Philips Xcelera 4.1 System).

Echocardiographic views used included the subcostal right and left oblique axis, the parasternal long axis, the parasternal short axis at the level of ventricle and great arteries, the left parasternal focused on the right ventricle inflow, the apical 4- and 5-chambers and suprasternal.

Ventricular and great vessels diameters and volumes were evaluated.

The anatomical parameters included end-diastolic and end-systolic LV volume and RV area measured in apical 4-chamber view, tricuspid z score and regurgitation degree associated.

The functional parameters included: manually traced right ventricular fractional area change (e-RVFAC), speckle tracking automatically derive (STAD) RVFAC by 2D (a-RVFAC), Tricuspid Annular Plane Systolic excursion (TAPSE), the systolic wave of the tricuspid valve on the Tissue Doppler Imaging (TDI) and Right and Left Ventricle 2D Systolic Global Longitudinal Strain (RVGLS and LVGLS). According to the American Society Echocardiography guidelines, all right and left ventricular dimensions were evaluated in end-diastolic phase in both apical and parasternal views.

The Echocardiographic FAC (e-RVFAC) was estimated with the following calculation: [(RVEDA-RVESA/RVEDA) x 100] and the automatic FAC (a-RVFAC) was measured with a speckle tracking automatically derived RVFAC by 2D-STAD using a standard 4-chamber apical view. The Ejection Fraction (LVEF) was estimated with the following calculation: [(LVEDV-LVESV/LVEDV) x 100]

Normal RVFAC value was considered greater than 35%. Normal EF value was above 52%.

TAPSE identified the longitudinal right ventricle function obtained positioning the M-mode cursor on the lateral portion of the tricuspid annulus. Normal value was considered above 16 mm.

Tissue Doppler analysis was used to measure systolic tricuspid annulus velocity (Ts'). A velocity below 10 cm/sec was considered as a sign of systolic dysfunction.

Cine-loops recordings were reviewed off-line and analyzed by one single expert operator blinded to diagnosis. Analyses were performed using Philips QLAB software version 10.3 (Philips Andoven, MA, USA). 2D strain was calculated using apical four-chamber view, focused on the right ventricle with a modified apical view, obtaining a complete image of the right ventricle, particularly the free wall. The speckle tracking strain software developed for the left ventricle was applied also to the right one. RVGLS measure derived by averaging only strain curves from the RV free wall (4 RV segment model: basal, median, apical free wall and apex). The normal mean values of RVGLS in children were considered from -20.80% to -34.10 (mean -30.06, 95% CI -32.91 to -27.21) and for LVGLS from -16.7% to -23.6% (mean -20.2%, 95% CI -19.5% to -20.8%). **[7]**

Statistical analysis

Data are expressed as percentage for categorical data and mean ± standard deviation for normally distributed continuous variables.

Comparison between groups (group 1: patients with left ventricle dysfunction; group 2: patients with normal left ventricle function after PVBD) was carried out by Student's t-test for continuous normally distributed variables and the χ^2 test was used to compare categorical variables.

A *p* value less than 0.05 was considered statistically significant.

Statistical analysis was performed by SPSS Statistics for Windows software, version 21.0 (released 2012; IBM Corp., Armonk, NY, USA).

Results

We retrospectively enrolled twenty-nine infant in the study (21 male and 8 female). The median age was 9±6 day. Twenty-five infant with critical pulmonary valve stenosis (PVS) and four with pulmonary atresia with intact ventricular septum (PAIVS) were included.

The pulmonary valve balloon dilation was performed between one and fourteen days of age. The indications for PVBD were standard for neonates with critical PVS and PAIVS. **[8]**

All patients before the procedure had a normal left ventricle ejection fraction (EF) > 57%. After the PVBD, eight patients developed a transient left ventricle dysfunction (5 PVS and 3 PAIVS) with EF < 50% calculated by Simpson biplane method.

Comparing the echocardiographic data before the procedure in patients with transient left ventricle dysfunction, there was no difference about the geometrical parameters of right ventricle, as tricuspid valve annulus diameter Z-score (-0,45±1.3 vs -0.72±1.4, *p*=0.839) and tricuspid regurgitation (1.5±0.8 vs 1.6±0.8, *p*=0.32) and ventricle longitudinal strain that appear equally reduced in both groups for right (-13.9±3.4% vs -14.8±2.5%, *p*=0.192) and left (-27.7±1.95 vs -27.0±2.2%) ventricle. **Table 1-2**

However, pulmonary valve regurgitation more than mild degree after PVBD was statistically significant associated to the left ventricle dysfunction (*p*<0.0001). **Figure 1**

Patients with greater delta right ventricle area (1.2±0.8 vs 0.3±0.9, *p*=0.042) and left ventricle volume (-1.1±0.9 vs -0.1±21, *p*=0.001) calculated before and after PVBD developed left ventricle dysfunction more than patients with less dilation of right ventricle and left ventricle. Moreover, an earlier PVBD (day of life) is associated with left ventricle dysfunction (2.0±1.2 vs 8.7±5.2, *p*=0.04). **Table 3-4-5**

In addition, there was not difference at the cardiac catheterization about the right and left pressure ratio (RV/LV ratio pre: 1.7±0.4 vs 1.5±0.5, *p*=0.122 and RV/LV ratio post: 0.88±0.18 vs 0.80±0.14, *p*=0.350) in patients with and without the transient left ventricle dysfunction. **Table 4**

Discussion

The aim of the study was to investigate the risk factors which predispose neonates with PS or PAIVS to the development of left ventricle dysfunction post-PVBD.

Many studies point the abnormal ventricular-ventricular interaction, present in various conditions like right ventricle pressure and/or volume loading or remodeling, as the reason of the left ventricle dysfunction with an important prognostic effect on mortality. **[9-11]**

Since the right ventricle shares myocardial fibers, interventricular septum and pericardium with the left ventricle, it is intuitive that any changes in geometry and function of one ventricle involves the contralateral one, independent of neural, humoral or circulatory effect, and septum plays a crucial role in mediating this interaction. **[12-13]**

Firstly, Dexter in 1956 reported cases of left heart failure in patients with atrial septal defect for the interventricular dependence. **[14]**

Dale A. Burkett et al. have shown the role of ventricular interdependence in influencing the left ventricle function in patients with pulmonary hypertension. A reduced left ventricle longitudinal and circumferential strain and strain rate, primarily at the basal septum, as consequence of the leftward septal shift of the right ventricle, has been demonstrated in children and young adult with pulmonary hypertension, suggesting direct pressure-loading effects on right and left ventricle performance and hemodynamics. **[15]**

The theory of interventricular interaction and its effect on left ventricle function has been demonstrated also in patients with Tetralogy of Fallot both for volume and pressure overload

Patients with repaired tetralogy of Fallot (rToF) and pulmonary regurgitation (PR) have a different pathophysiological response to RV chronic volume overload but share with PS and PAIVS the indirect effect on left ventricle function worsening.

It being understood that patients with rToF and PR have altered RV longitudinal mechanical performance and a tendency to right systolic dysfunction as shown in a previous study from our institution **[16]**, and that the pulmonary valve replacement in these patients improves global LV strain. **[17-18]**

Moreover, in patients with rToF, the residual RV outflow tract obstruction induces a preservation of RV strain and an early protective effect on RV modeling, but had a negative impact on LV strain. **[19]**

Anyway, in patients with PVS/PAIVS there is a major evidence of RV diastolic dysfunction with a tendency to restrictive physiology for the more ventricular hypertrophy, myocardial disarray and fibrosis compared to patients with repaired Tetralogy of Fallot and PR. **[20]**

In another study, Ronai et colleagues have demonstrated in patients with pulmonary stenosis/atresia after PVBD that the worsening of LV longitudinal and circumferential global and segmental strain (more pronounced in septal segments) in eight patients who subsequently developed LV dysfunction, is predictor of left ventricle dysfunction, while longitudinal RV strain remains unchanged pre- and post-PVBD. Furthermore, in this study for the first time the hypothesis of the right ventricle volume overload as reason of left ventricle dysfunction has emerged.

The mechanism involved in altering myocardial performance in the ventricular septum with negative ventricular-ventricular interaction has been attributed to the RV volume loading following the relief of the right ventricle outflow obstruction. Patients who developed LV dysfunction after PVBD had larger right ventricles but not significantly larger left ventricles **[21].**

In our study left and right ventricular strain before PVBD tend to be reduced, even if not statistically significantly, in patients who develop left ventricle dysfunction, while and in agreement with Ronai's study, the greatest increase mostly of right ventricle area after PVBD in patients with left ventricle dysfunction is statistically significantly. This evidence has confirmed that the right ventricle volume overload (RVVO) after PVBD, due to the iatrogenic development of pulmonary regurgitation, is a predisposing risk factor of transient left ventricle dysfunction.

Our evidences in agreement with the previous study allows us to share the hypothesis, developed by Lin and Louie of how RVVO impacts on left ventricle ejection fraction.

The underlying mechanism depends on the resultant acute right volume overload, regardless of the reduction of pressure load, which can alter right chambers geometry causing left ventricle dysfunction due to the known physiological processes of ventricular interdependence and also the decreased relative contribution of left atrial systole to the left ventricular filling.

The acute right volume overload induce the flattening of the ventricular septum resulting from leftward displacement of the septum toward the center of the left ventricle and it is most marked at end diastole, opposing the normal forces of left ventricle distension. The normal ventricular septal curvature restores at end systole, which opposes the inward motion of the ventricular septum toward the center of the left ventricle during systole contraction. As a result, the net shortening along the ventricular septum-to-posterolateral free wall short axis in RVVO is depressed.

The regional nature of the LV impairment, ventricular septal flattening dependent, refutes strongly a systemic mechanism explanation (loading alteration, neurohumoral interaction, autonomic influence) and confirms the suspicion that the transient left ventricle dysfunction is a consequence of acute RVVO **[22-23]**

In fact, in our study, in all patients the impairment of longitudinal regional strain more in the septum than in the lateral free wall proves that the most accredited theory for transient left ventricle dysfunction leans towards volume overload and interventricular dependence. **Table 6-7**

We have considered also another hypothesis based on the creation of the 'circular shunt'.

In the patient with congenital heart disease, the phenomenon termed 'circular shunt' implies that some shunted blood returns to its chamber of origin through intra-cardiac channels or communications, hence bypassing the systemic capillary bed. In patients with pulmonary stenosis or pulmonary atresia with patent ductus arteriosus the 'circular shunt' implies: left atrium-> left ventricle-> aorta-> ductus arteriosus-> left pulmonary artery->pulmonary circulation-> pulmonary veins->left atrium. It is suggested that retrograde flow through ductus arteriosus from the aorta may impede or limit normal diastolic coronary artery perfusion and may predispose to myocardial ischemia and dysfunction. **[24]**

The theory of the circular shunt contrasts with our data. In fact, there is not statistically significance about the time between ductus closure and left ventricle dysfunction development and the strain segmental analysis shows a reverently dysfunction concentrated in the septum and not uniformly in all segments.

The transient left ventricle dysfunction predominantly in infants undergoing early PVBD led us to think that this probably it depends on the acute overlap of two hemodynamic setting.

In fact, the infant have to find a new balance of adaptation between the increased physiological resistance of the pulmonary circulation and the volume overload for pulmonary regurgitation after PVBD.

Limitations

It was a single-center retrospective study with a small sample size, even for the low percentage of cases due to the rarity of the this congenital heart disease, and the conclusions may be underpowered and it made the study difficult to compare outcomes with other studies. Studies with a larger sample size are needed.

The echocardiographic evaluation of functional and morphological indices is influenced by multiple factors as heart rate, preload and afterload that are potentially selection bias.

Conclusion

Moderate-severe degree pulmonary valve regurgitation predisposes to transient left ventricle dysfunction in patients with PVS and PAIVS after PVBD. The reason is probably due to the acute right ventricle volume overload, which also influences the left ventricle for the known pathophysiological mechanisms of ventricular interdependence.

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Figure 1

Figure 1

Moderate pulmonary valve regurgitation after PVBD was significant associated to LV dysfunction (p<0.0001).

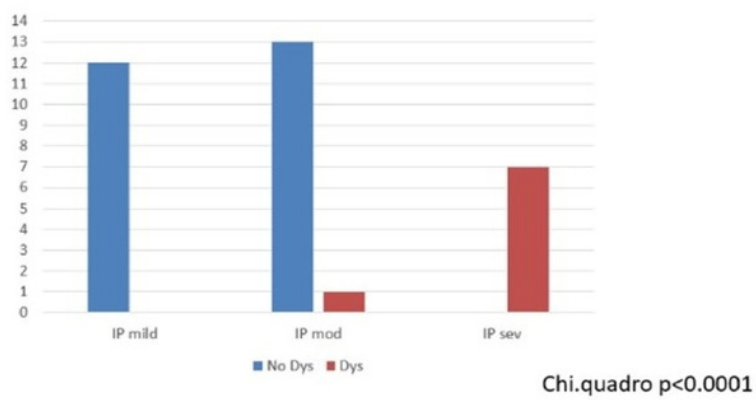

Table 1(on next page)

Table 1

Left Ventricle indices of function

Table 1. Left Ventricle indices of function

	LV dysfunction (n 8)	NO LV dysfunction (n 21)	*p*
EF-PRE (%)	64.2±3.6	64.2±3.6	0.89
EF-POST (%)	41.2±7.1	62.5±2.8	**<0.001**
Delta EF (%)	-23.1±6.9	-1.7±2.4	**<0.001**
LV STRAIN PRE (%)	14.1±1.2	15.5±1.9	0.07

Table 2(on next page)

Table 2

Tricuspid valve indices

Table 2. Tricuspid valve indices

	LV dysfunction (n 8)	NO LV dysfunction (n 21)	*p*
Tricuspid Z-score	-0.45±1.3	-0.72±1.4	0.839
Tricuspid Regurgitation	1.5±0.8	1.6±0.8	0.321

Table 3(on next page)

Table 3

Left Ventricle geometrical indices

Table 3. Left Ventricle geometrical indices

	LV dysfunction (n 8)	NO LV dysfunction (n 21)	*p*
LVED-PRE (mL)	6.6±2.4	6.1±2.0	0.538
LVED-POST (mL)	5.5±2.3	6.0±2.1	**<0.030**
Delta LVED (mL)	-1.1±0.9	-0.1±2.1	**0.001**

Table 4(on next page)

Table 4

Right and left pressure ratio measured during cardiac catheterization

Table 4. Right and left pressure ratio measured during cardiac catheterization

	LV dysfunction (n 8)	NO LV dysfunction (n 21)	*p*
Days of life	2.0±1.2	8.7±5.2	**0.04**
RV/LV ratio Pre	1.7±0.4	1.5±0.5	0.122
RV/LV ratio Post	0.88±0.18	0.80±0.14	0.350

Table 5(on next page)

Table 5

Right Ventricle geometrical indices before and after pulmonary valve balloon dilatation.

Table 5. Right Ventricle geometrical indices before and after pulmonary valve balloon dilatation.

	LV dysfunction (n 8)	NO LV dysfunction (n 21)	*p*
RV area PRE (cm^2)	2.5±0.8	3.0±0.9	0.233
RV area POST (cm^2)	3.7±0.9	3.3±0.8	0.065
DELTA RV area (cm^2)	1.2±0.8	0.3±0.9	**0.042**
RV area PRE (cm^2)	-13.9±3.4	-14.8±2.5	0.192

Table 6(on next page)

Table 6

Regional septum strain analysis before and after pulmonary valve balloon dilatation

Table 6. Regional septum strain analysis before and after pulmonary valve balloon dilatation

All patients (n=29)	LV Regional strain pre-PVBD	LV Regional strain post-PVBD	*p*
Apical Septum	-27.57	-16.11	**<0.04**
Medium Septum	-11.54	-7.62	0.1
Basal Septum	-10.5	-7.6	**<0.001**

Table 7(on next page)

Table 7

Regional left ventricle lateral wall before and after pulmonary valve balloon dilatation

Table 7. Regional left ventricle lateral wall before and after pulmonary valve balloon dilatation

All patients (n=29)	LV Regional strain pre-PVBD	LV Regional strain post-PVBD	*p*
Lateral-Apical	-20.68	-15.6	0.34
Lateral- Medium	-18.14	-16.17	0.37
Lateral- Basal	-12.48	-9.52	0.28