The Egyptian Cardiothoracic Surgeon

In Press

Original Article

Right Ventricular Dysfunction Post-Surgical Repair of Fallot Tetralogy in Pediatric Age Group: Predictor Factors Analysis

Ihab Mohamed Salah Eldin Elsharkawy, Tarek Salah, John Malaty Fouad, Mahmoud Abdo Omar Abualfa, Ashraf Mostafa Abd Raboh

Department of Cardiothoracic Surgery, Faculty of Medicine, Cairo University, Cairo, Egypt

Abstract

Background: Right ventricular (RV) dysfunction is often detected after Tetralogy of Fallot (TOF) repair. We aimed to analyze the preoperative, intraoperative, and postoperative risk factors for RV dysfunction and to correlate them to the surgical technique used in the repair.

Methods: This prospective cohort study included 26 pediatric patients with TOF. The participants were divided into two groups based on RV dysfunction. Group A included patients with RV dysfunction, and Group B included patients without RV dysfunction. Each patient was assessed clinically and via echocardiography and cardiac magnetic resonance. Participants were followed for 6 months.

Results: The incidence of RV dysfunction was 30.8% of patients. Compared to group B, patients in group A had significantly low preoperative oxygen saturation (p=0.011), high Right ventricular outflow tract pressure gradient (RVOT PG) (p=0.03), operative transannular patch (p=0.011), prolonged intubation time (p=0.017), and pediatric intensive care unit stay (p=0.001), high incidence of inadequate urine output (p=0.014), prolonged inotropic use (p=0.02) as well as low postoperative tricuspid annular plane systolic excursion (TAPSE) (p<0.001) and fractional area change (FAC)(p<0.001), and high RVOT pressure gradient (p<0.001). However, regression analysis showed no statistical correlation between these variables and RV dysfunction.

Conclusion: Physicians should consider low preoperative oxygen saturation, high RVOT PG, operative transannular patch, long intubation time and pediatric intensive care unit stay, high incidence of inadequate urine output, prolonged inotropic use as well as low postoperative TAPSE and FAC, and high RVOT pressure gradient as risk factors for RV dysfunction after TOF repair in pediatrics.

KEYWORDS

Tetralogy of Fallot; Pediatrics; Intensive Care Units; Right Ventricular Dysfunction

Introduction

Teratology of Fallot (TOF) is a prevalent congenital heart disease, accounting for 55-70% of patients. The primary surgical treatment for patients with TOF is complete repair at a young age. Many patients experience a slow recovery after complete repair of TOF, with symptoms including fluid retention, pleural effusion, elevated central venous pressure, ascites, reduced cardiac output, delayed right ventricular (RV) myocardial enhancement, and diastolic dysfunction [1, 2].

Corresponding author: Mahmoud Abdo Omar Abualfa mahmoudabdo140.ma@gmail.com

Increased myocardial stiffness and decreased RV compliance lead to an increase in RV end-diastolic pressure, which is the pathophysiological cause of RV restriction. This RV restriction results in high systemic venous pressure, which is often associated with prolonged pleural effusion and decreased cardiac output, both of which prolong the patient's stay in the intensive care unit [3]

The incidence of impaired relaxation and pseudo-normal filling in the pediatric age group is unknown, especially during intermediate follow-up, but RV restrictive physiology is a well-known phenomenon observed in the immediate postoperative period and during long-term follow-up in postoperative TOF patients [4].

Echocardiography is one of the most important tools for postoperative follow-up of TOF. However, RV diastolic function is often overlooked in favor of focusing on RV systolic performance. Therefore, it is difficult to determine the exact prevalence of RV diastolic dysfunction, especially during intermediate follow-up.

A number of parameters, such as age, weight, oxygen saturation (SaO2), hematocrit, ventricular hypertrophy, right heart enlargement, transannular patch repair, cardio pulmonary bypass (CPB) time, aortic cross-clamping time, ratio of RV pressure to left ventricle pressure, tricuspid annular plane systolic excursion (TAPSE), pulmonary artery systolic pressure (PASP), and TAPSE/PASP ratio, were used to compare restrictive and non-restrictive patients who underwent total correction of TOF [5].

There is a paucity of research focusing on the etiology of RV dysfunction in pediatric age group. Most previous studies have studied the mechanism of RV dysfunction and have been conducted in adolescent or adult patients. Hence, this study was conducted to assess the preoperative, intraoperative, and postoperative factors that lead to RV dysfunction up to 6 months postoperatively and to correlate it to the surgical technique used in the repair.

Patients and Methods

Ethical considerations

The study protocol was approved by the Ethical Committee of the Faculty of Medicine, Cairo University Hospital. Informed consent was obtained from each child's guardian. Participant data were kept confidential.

Study design, duration, and settings

This prospective cohort study included a total of 26 pediatric patients who underwent total repair for TOF at the Pediatric Cardiothoracic Unit of Abu Elriesh Specialized Children's Hospital (CUPH) between May 2023 and August 2024.

Eligibility criteria

Pediatric patients with TOF and favorable anatomy, with a Mac-Goon ratio (greater than 1.6), aged 6 months to 14 years, who underwent single-stage total repair were included. Patients with pulmonary atresia, hypoplastic pulmonary artery (small pulmonary arteries with Mac-Goon ratio less than 1.6) who were not candidates for total repair, and patients with TOF who were candidates for biventricular repair or had abnormal coronary artery anatomy or redo TOF were excluded. Patients with TOF combined with other pathologies and those with absent pulmonary valve were also excluded.

Operative details

The tricuspid valve was used to evaluate the right ventricle through the right transatrial approach. A ventricular septal defect and a large hypertrophy of many septoparietal trabeculations obstructing the RV outflow tract were present, as well as an anterior malposition of the infundibular septum. The hyperthrophied septoparietal trabeculations were removed by trans-tricuspid right ventricular resection. Just lateral to the infundibular septum, the muscle bundles on the anterior, lateral, and posterior walls of the RV outflow tract were removed. The ventricular septal defect was then examined. After transport to the field, the Gore-Tex patch was cut to the appropriate size and shape. It was used to seal the VSD with a 6-0 polypropylene sheath.

The guidewire surrounding the posterior inferior portion of the defect was carefully protected. Upon inspection, the patch was found to be in proper position. The tricuspid valve was

evaluated by inflating the right ventricle with cold saline. If it was found to be incompetent, either commissural suturing or bicuspidization was performed.

The main pulmonary artery, branch pulmonary arteries, pulmonary valve, pulmonary annulus, and RVOT are the areas of focus for the surgeon. The branch pulmonary arteries are examined when the main pulmonary artery is opened longitudinally. The pulmonary valve is inspected; the commissures are not thickened or fused. Hegar is used to measure the pulmonary valve annulus, pulmonary valvotomy is performed, and the pulmonary valve is used to view the right ventricular infundibulum. The pulmonary valve annulus is used to resect any remaining distal right ventricular outflow obstruction, such as the distal parietal insertion of the infundibular septum. The surgeon must determine at this stage of the repair whether the P annulus is large enough to allow all cardiac output to pass through without significantly increasing right ventricular pressure.

To relieve the obstruction and stop excessive regurgitation, the transannular patch must be properly shaped. The patch can be made of PTFE and autologous pericardium and should have an oval shape with blunt corners. The main pulmonary artery incision was supplemented with a custom-made pericardial patch. Running 7-0 polypropylene was used to suture and secure the patch.

To properly size the patch, a Hegar dilator is used that is 2-3 mm larger than the mean normal size of the pulmonary annulus. The maximum width of the patch should be slightly greater than the circumference of the Hegar dilator visible in the open right ventricular outflow tract.

Normalized "Z values" represent the size of the pulmonary valve annulus in relation to the size of the child. In general, if the pulmonary valve annulus is larger than a Z value of -2, a transannular patch can be avoided. The annulus was preserved and the P arteriotomy was closed with a pericardial patch if the P annulus was in good condition and the proper Hegar had flowed through it. In other individuals, however, the proper Hegar did not flow through the P annulus. In these circumstances, the P annulus was cut into the RV infundibulum up to 1 cm in the RVOT until the proper Hegar entered the RVOT. If necessary, additional excision of the hypertrophied RV infundibular muscles was performed in these patients via an RV infundibulotomy. The RV infundibulotomy was closed with the pericardial patch until it reached the P arteriotomy (RVOT transannular patch).

We did not open the P annulus in certain patients whose MPA and P annulus were both normal with Z values + 2. Instead, we made a small incision in the RVOT, less than 1 cm, to complete the RV infundibulotomy from above. The RV infundibulotomy was then closed with a pericardial patch using 6/0 prolene continuous suture.

A limited incision was made in the RVOT, followed by a longitudinal anterior incision in the MPA and its branches, a patch was fashioned on the appropriate size of hegar within the pulmonary arteries, and the RV infundibulotomy was performed using 6/0 prolene continuous suture without interruption to the P annulus if the pulmonary valve annulus was adequate but the mean pulmonary artery and its branches were hypoplastic (Mcgoon ratio less than 1.6 but greater than 1.2).

We started systemic rewarming, A The left heart was de-aired, the lungs were ventilated, the atrial septal defect was mostly closed, and temporary atrial/ventricular pacing wires were placed. The ascending aorta was used for further de-airing, which was confirmed by TEE. After removal of the aortic cross-clamp, sinus rhythm quickly returned to normal. Running polypropylene was used to simulate the right atriotomy, and the caval tourniquets were removed. TEE showed normal tricuspid valve function, no residual ventricular septal defect, and satisfactory velocity across the right ventricular outflow tract. The heart was sequentially decannulated and protamine was administered. Mediastinal chest tubes and a peritoneal dialysis catheter were placed.

If the patient was less than one year old, an incision was made in the peritoneum below the diaphragm to insert a non-tunneled peritoneal dialysis catheter, which was then passed through the abdominal wall into the peritoneal cavity under direct visualization. After irrigation of the mediastinum with antibiotic solution, the mediastinum was aspirated. The skin, fascia, and sternum were all roughly dissected in a typical fashion. After the procedure was completed, the child was stabilized and transferred to the cardiothoracic intensive care unit.

Diagnosis of right ventricular dysfunction

For RV dysfunction, clinical, echocardiography, and cardiac magnetic resonance (CMR) were used to assess RV dysfunction.

Clinically, three symptoms of RV failure are clear lungs, elevated central venous pressure (CVP) > 15 mmHg, and hypotension. In advanced stages of RV failure, arrhythmias and inadequate forward flow can lead to shock, systemic congestion, and multiorgan failure, particularly acute kidney injury [6].

By echocardiography, each patient underwent transthoracic echocardiography with simultaneous recording of respiratory and electrocardiographic waveforms using commercially available echocardiographic equipment. Before surgery, each patient underwent a complete transesophageal echocardiogram in an operating room with respiratory monitoring. The transesophageal echocardiographic probe was positioned according to standard procedures, and echocardiographic specialists performed the examinations in a uniform manner [7]. Right ventricular systolic dysfunction was defined as tricuspid annular space systolic excursion (TAPSE) <17 mm, fractional area change (FAC) <35%, and right ventricular outflow tract pressure gradient (RVOT PG) >40 mmHg.

On CMR, signs of RV dysfunction included large RV volumes such as right ventricular end diastolic volume (RVEDV), right ventricular end systolic volume (RVESV) and right ventricular end systolic volume (RVSV), reduced right ventricular ejection fraction (RVEF) <45% and reduced left ventricular (LV) volume.

Table 1: Baseline patients' characteristics and operative details (Total n = 26). Quantitative variables are presented as mean ± SD. Qualitative variables are presented as frequency (percentage)

Variable	Value
Age, month	
Min. – Max.	6 - 48
Mean±SD	20.77±10.58
Gender	
Male	13 (50%)
Female	13 (50%)
Weight, kg	9.8±1.8
Height, cm	80.99±10.09
BMI, kg/m^2	15.18±1.98
Pre SaO_2 (%)	
Min. – Max.	65 - 96
Mean±SD	83.65±7.18%.
Preoperative echo	
Pre TAPSE, cm	1.83±0.14
Pre RVOT-PG, mmHg	75.15±9.57
TOF repair technique	
Trans annular	13 (50%)
RV PA conduit	7 (26.9%)
RV patch	6 (23.1%)
CPB time, minutes	101.96±14.06
Cross clamp time, minutes	80.62±14.61
Operative time, minutes	222.96±34.33
Need for inotropic support	24 (92.3%)
Rhythm after bypass weaning	
Sinus	16 (61.5%)
Nodal	7 (26.9%)
AF	1 (3.8%)
Heart block	2 (7.7%)

Min.: minimum; Max: maximum; SD: standard deviation; n: number; cm: centimetre; kg: kilogram; BMI: body mass index; SaO_2: oxygen saturation; RV: Right ventricular; TOF: teratology of Fallot; TAPSE: tricuspid annular plane systolic excursion; RVOT PG: right ventricular outflow tract pressure gradient; PA: pulmonary artery; AF: atrial fibrillation

Data collection

Preoperatively, all participants were assessed prospectively for their age, weight, height, body mass index (BMI), and SaO2 at the time of admission.

Intraoperatively, operative technique, ischemic time, total CPB time, total operative time, need for intraoperative inotropic support, and patient rhythm after CPB weaning were recorded.

Table 2: Postoperative clinical and echocardiographic characteristics (Total n = 26)

Results	Mean±SD
Mechanical ventilation, hrs	52.8±41.52
PICU stay, days	10.58±6.8
Chest tube drainage, mL/kg/h	49.53±5.03
Urine output, mL/kg/24 hrs	38.05±3.02
Postoperative inotropic duration, days	4.38±1.77
Post-operative echo parameter	
TAPSE, cm	1.69±0.19
FAC%	35.77±5.05
RVOT PG, mmHg	48.38±11.64

SD: standard deviation; hrs: hours; TAPSE: tricuspid annular plane systolic excursion; FAC: fractional area change; RVOT PG: Right ventricular outflow tract pressure gradient; mmHg: millimetre mercury; PICU: Pediatric intensive care unit

Postoperatively, a standard record of postoperative data was used. It includes the following data, duration of ventilatory support, duration of pediatric intensive care unit (PICU) stays, chest tube drainage in the first 24 hours, urine output, duration of inotropic support, echocardiographic data with clinical indices of RV dysfunction, patient's rhythm in the PICU, need for pacemaker, and duration of hospital stay.

Post-discharge follow-up data

All patients were evaluated by echocardiography, focusing on systolic and diastolic function. Patients who still had RV dysfunction at 6 months post echo were re-evaluated by CMR to confirm RV dysfunction.

Statistical analysis

Data were analyzed using SPSS version 24.0 (IBM/SPSS Inc., Chicago, IL). The Kolmogorov-Smirnov test was used to test the normality of the distribution of numerical variables. Categorical variables are presented as numbers and percentages and were compared using Pearson chi-square ($\chi 2$) and Fisher's exact test, as appropriate. Continuous variables are expressed as mean±standard deviation (SD) and were compared using the independent samples t-test. Binary logistic regression models were used to determine the association between the RV dysfunction and independent risk factors. All reported P values were two-tailed, and a value of $P < 0.05$ was considered statistically significant.

Results

Table 1 shows the patients' characteristics. The mean age was 20.77±10.58 months with equal gender distribution. The mean BMI was 15.18±1.98 kg/m2. The mean pre-operative SaO2 was 83.65±7.18%. The mean preoperative TAPSE and RVOT PG scores were 1.83±0.14 cm and 75.15±9.57 mmHg, respectively. Half of the patients underwent RV patch with trans - annular approach. The mean CPB, CCT, and operative time were 101.96±14.06, 80.62±14.61 minutes, and 222.96±34.33 minutes, respectively. Most of the patients (92.3%) required intraoperative inotropic support after weaning from bypass. After weaning from bypass, most patients (61.5%) had a sinus rhythm.

Postoperatively, all patients were mechanically ventilated for 52.8±41.5 hours with a PICU stay of 10.58±6.8 days. Through the first 24 hours, 26.9% of patients had high chest tube drainage with a mean of 49.53±5.03 cc/kg/hours and inadequate urine output of 33.04±1.09 mL/kg. Inotropic support was required for 4.38±1.77 days. Postoperative Echocardiography revealed that the mean TAPSE score was 1.69±0.19 cm, while the mean FAC and RVOT PG scores were 35.77±5.05% and 48.38±11.64 mmHg (Table 2).

Table 3 demonstrates that 8 patients (30.8%) had post-operative right-sided dysfunction (group A). Group B included 18 patients (69.2%) who had no post-operative right-sided dysfunction. Preoperative SaO2 was significantly lower among patients in group A than group B (78.5±7.85 vs 85.94±5.67, p = 0.011). Preoperative echocardiography showed significantly higher pre RVOT PG in group A than group B (81.25±8.35 vs 72.44±8.99, p = 0.03). Regarding the technique applied for TOF repair, 38.5% of patients operated

Table 3: Comparison of demographic data, technique of repair and operative parameters (Total n = 26). Quantitative variables are presented as mean ± SD. Qualitative variables are presented as frequency (percentage)

Variable	Group A (n=8)	Group B (n=18)	P value
Age, month	22.38±13.39	20.06±9.43	0.616
Gender			
Male	5 (62.5%)	8 (44.4%)	0.673
Female	3 (37.5%)	10 (55.6%)	
Weight	9.9±2.06	9.76±1.68	0.852
Height	80.75±12.73	80.94±9.12	0.965
BMI	15.34±2.42	15.11±1.83	0.794
Pre SaO_2 (%)	78.5±7.85	85.94±5.67	0.011*
Pre TAPSE	1.76±0.16	1.86±0.13	0.128
Pre RVOT PG	81.25±8.35	72.44±8.99	0.03*
RV patch with Trans annular	5 (38.5%)	8 (61.5%)	0.032*
PA conduit	2 (28.6%)	5 (71.4%)	
RV patch	1 (16.7%)	5 (83.3%)	
CPB time	105.63±18.6	100.33±11.79	0.387
CCT	81.38±20.72	80.28±11.69	0.892
Operative time, minutes	235.38±41.65	217.44±30.23	0.226
Need for inotropic support	8 (33.3%)	16 (66.7%)	0.557
Rhythm after bypass weaning			
Sinus	4 (25%)	12 (75%)	0.158
Nodal	2 (28.6%)	5 (71.4%)	
AF	0 (0%)	1 (100%)	
Heart block	2 (100%)	0 (0%)	

Group A: right ventricular dysfunction; Group B: normal right ventricular function; SD: standard deviation; n: number; min: minutes; SaO_2: oxygen saturation; RV: Right ventricle; TAPSE: tricuspid annular plane systolic excursion; RVOT PG: Right ventricular outflow tract pressure gradient; PA: pulmonary artery; AF: atrial fibrillation; CPB: Cardiopulmonary bypass; CCT: aortic cross clamping time

*: Significant at p<0.05

via Trans annular approach developed RV dysfunction, which was significantly more than those subjected to PA conduit and RV patch (28.6% and 16.7% of patients, p = 0.032).

Compared to group B, patients in group A had significantly prolonged duration of mechanical ventilation (3.88±1.2 vs 1.41±0.5 days, p=0.017) and PICU stay (17.88±8.72 vs 7.33±1.33, p=0.001) with high incidence of inadequate urine output (71% vs. 28.6%, p = 0.014) and prolonged postoperative inotropic support (7.01±2.82 vs 3.22±0.83, p = 0.02). Postoperative echocardiographic findings revealed that patients in group A had significantly lower TAPSE and FAC and significantly higher RVOT PG than group B (1.46±0.14 vs 1.78±0.1, 29.13±2.6 vs 38.72±2.14, p<0.001, and 58.7±12.7 vs 43.8±7.6, p=0.001, respectively, Table 4).

Compared to the preoperative echocardiographic findings, there was a significant decline in TAPSE and RVOT PG among patients who developed RV dysfunction postoperatively (1.76±0.15 vs 1.46±0.14, p<0.001 and 81.25±8.35 to 58.75±12.75, p=0.004, respectively) (Table 5).

Table 6 shows the CMR findings among patients with RV dysfunction. Table 7 demonstrates no statistically significant association between preoperative, operative and postoperative parameters and the RV dysfunction.

Table 4: Postoperative follow-up, morbidity, and mortality (Total n = 26). Quantitative variables are presented as mean ± SD. Qualitative variables are presented as frequency (percentage)

Variable	Group A (n=8)	Group B (n=18)	P value
Mechanical ventilation duration, days	3.88±1.2	1.41±0.5	0.017*
PICU stay, days	17.88±8.72	7.33±1.33	0.001*
High drainage	4 (57.1%)	3 (42.9%)	0.149
Urine output			
Good	3 (15.8%)	16 (84.2%)	0.014*
Inadequate	5 (71.4%)	2 (28.6%)	
Postoperative inotropic duration	7.01±2.82	3.22±0.83	0.02*
Post TAPSE, cm	1.46±0.14	1.78±0.1	<0.001*
Post FAC, %	29.13±2.6	38.72±2.14	<0.001*
Post RVOT PG, mmHg	58.7±12.7	43.8±7.6	0.001*
Mortality	0	0	NA
Morbidity			
Heart block	2 (25%)	0	0.334
Pulmonary regurge	1 (12.5%)	0	

Group A: right ventricular dysfunction; Group B: normal right ventricular function; RV: Right ventricle; TAPSE: tricuspid annular plane systolic excursion; RVOT PG: Right ventricular outflow tract pressure gradient; PA: pulmonary artery; SD: standard deviation; n: number; min: minutes

*: Significant at p<0.05

Discussion

The aim of our study was to evaluate preoperative and postoperative risk factors for predicting postoperative RV dysfunction up to 6 months postoperatively and to correlate them with the surgical technique used for repair.

Our main findings were that the incidence of patients with RV dysfunction was 30.8%. Preoperative low SaO2, high RVOT PG, operative transannular patch, long PICU stay and intubation time, and postoperative low TAPSE and FAC, high RVOT PG, and low RVOT PG were significant parameters for RV dysfunction. However, regression analysis revealed no statistically significant association between these parameters and RV dysfunction.

In the current study, 30.8% of the patients had RV dysfunction. Kidwai et al. [4] observed a higher prevalence (51.1%) of RV diastolic dysfunction in postoperative TOF patients in the juvenile age group. The measured subclinical RV impairment is the reason for the discrepancy. Also, Sandeep et al. [5] reported that 58% of their patients had RV restrictive function. In addition, Singh et al. [8] found that 54.2% of patients with a mean age of 7.31±4.74 years who underwent transannular patch repair. These differences in prevalence may be due to the different age groups of the patient populations in the different studies.

Table 5: Pre and postoperative echocardiographic findings (Total n = 26)

Variables	Preoperative	Postoperative	P value
TAPSE	1.76±0.15	1.46±0.14	<0.001*
RVOT PG	81.25±8.35	58.75±12.75	0.004*

SD: standard deviation; TAPSE: tricuspid annular plane systolic excursion; RVOT PG: Right ventricular outflow tract pressure gradient

*: Significant at p<0.05

Table 6: Cardiac magnetic resonance findings among patients with RV dysfunction group (n= 8)

Variable	Value
RVEDV	210.25±19.54
RVESV	92.88±7.28
RVEDVI	129.6±19.2
RVESVI	57.4±16.4
RV EF	42±0.04
RVSV	109.13±16.4

SD: standard deviation; n: number; RVEDV: right ventricular end diastolic volume; RVESV: Right ventricular end systolic volume; RVEDVI: right ventricular end diastolic volume index; RVESVI: Right ventricular end systolic volume index; EF: Ejection fraction. RVSV: right ventricular stroke volume

In the current study, patients with RV dysfunction had significantly low preoperative mean SaO2. Sandeep et al. [5] indicated that the preoperative lower SaO2 was the etiology of RV restrictive physiology. Furthermore, Ji et al. [9] found that low preoperative arterial oxygen saturation was an independent risk factor for late right ventricular systolic dysfunction after correction of TOF.

Patients with RV dysfunction had a significantly high pre-RVOT PG. Terol et al. [10] reported that RV systolic function was reduced with TAPSE. The early postoperative changes are not well understood. Among the factors highlighted are pericardiotomy or pericardial adhesions, CPB, local tissue injury of the thin-walled anterior RV, and reduced RV protection by cold cardioplegia [11]. In addition, hypertension and/or RV volume overload are hallmarks of the preoperative status of patients with Fallot. RV dysfunction appears to persist in Fallot patients during mid-term follow-up [12, 13]. In addition, RV function continues to decline during long-term follow-up. Pulmonary stenosis and/or insufficiency and dyssynchronous contractions exacerbated by QRS prolongation are contributing causes. Clinical variables such as reduced exercise tolerance, arrhythmias, and even cardiovascular mortality have also been associated with this decline [14].

Regarding the repair technique, a significantly high incidence of patients who underwent transannular surgery had RV dysfunction compared to patients who had RV patches and PA conduits.

Table 7: Cardiac magnetic resonance findings among patients with RV dysfunction group (n= 8)

Variable	Exp B	95% CI		P value
		Upper	Lower	
Weight	3.61	0.2	4.2	0.383
Height	0.76	0.43	0.76	0.433
BMI	0.64	0.1	3.9	0.627
SaO_2	0.87	0.71	1.07	0.187
Pre TAPSE	0.07	0.02	3.41	0.551
Pre RVOT-PG	1.15	0.98	1.35	0.771
CPB time	0.986	0.796	1.22	0.901
CCT time	1.03	0.835	1.27	0.778
Method				
Trans annular	3.13	0.28	5.16	0.36
PA conduit	2	0.13	6.18	0.62
Intubation time	1.23	0.05	6.61	0.897
PICU stay	3.34	0.83	10.37	0.089

SD: standard deviation; n: number; CI: confidence interval; minutes; BMI: Body mass index; RV: Right ventricle; AF: atrial fibrillation; CPB: Cardiopulmonary bypass; CCT: aortic cross clamping time; PA: pulmonary artery; TAPSE: tricuspid annular plane systolic excursion; RVOT PG: Right ventricular outflow tract pressure gradient; PICU: pediatric intensive care unit

Sandeep et al. [5] showed a correlation between restricted physiology and transannular patch repair. Moreover, based on the univariate analysis, Ji et al. [9] showed that transannular patches after TOF correction were an independent risk factor for late RVSD. In addition, Ge et al. [15] found that one of the major causes of RV dysfunction was the transannular patch method of RV outflow tract reconstruction. Right ventricular systolic function was significantly decreased by the transannular patch, subsequent tricuspid regurgitation, tricuspid annulus dilatation, and subsequent pulmonary regurgitation. However, age at surgery or clinical follow-up did not affect the ability of the transannular patch to reduce RV systolic function after TOF correction [16]. Also, Norgard et al. [17] found that the anatomic substrate requiring transannular patch repair was more likely to be associated with RV restrictive physiology.

In the present study, patients with RV dysfunction had a significantly prolonged duration of mechanical ventilation and PICU stay. Singh et al. [8] coincided with these findings. Furthermore, Bootsma et al. [18] attributed this to the inadequate cardiac output and the need for more intensive management techniques, all of which make recovery more difficult. In addition, Abdelhameed et al. [19] found that patients who were extubated within the first six hours after surgery had significantly better RV functional parameters on postoperative echocardiograms than patients who were extubated six hours or more after surgery. Abdelhameed and colleagues [19] clarified these findings by understanding the physiological changes in RV function that occur when patients with TOF undergo extensive repair. The pulmonary valve loses its ability to function when the pulmonary annulus is disrupted during TOF repair, resulting in pulmonary regurgitation. Right ventricular volume overflow is the result of postoperative pulmonary regurgitation. After complete repair, the abrupt transition from pressure overload to volume overload leads to RV dysfunction and reduced exercise capacity.

As the RV dilates over time, the electrical properties of the RV myocardium change, leading to atrial and ventricular arrhythmias and possibly abrupt death. Changes in RV myocardial characteristics begin in the early postoperative period. Early extubation reduces the degree of pulmonary regurgitation and improves pulmonary forward flow by limiting the physiological changes induced by mechanical ventilation, particularly the increased pulmonary vascular resistance induced by positive pressure ventilation cycles [20].

According to Abdelhameed et al. [19], variations in lung volume may affect pulmonary vascular resistance and autonomic tone. High lung volumes may also compress the heart in the cardiac fossa in a manner similar to cardiac tamponade. By eliminating the negative effects of mechanical ventilation on cardiac function, early extubation in straightforward total repair of tetralogy may actually improve RV function.

In the current study, inadequate urine production and prolonged inotropic use were significantly more common in those with RVD. Similarly, Karimova et al. [21] found that children who received ventricular assist devices after total repair of TOF showed that low urine output, elevated urea, and creatinine were associated with RV dysfunction. Furthermore, Krishna et al. [22] found a significant relationship between greater postoperative inotropic support and postoperative changes in RV function.

Postoperative echocardiography showed that patients with RV dysfunction had significantly low TAPSE after surgery. Likewise, Romeo et al. [23] found that TAPSE decreased dramatically on postoperative days 1 and 3 compared to preoperative values. TAPSE is readily available and reproducible [24]. Furthermore, Hashimoto and Watanabe [25] reported that TAPSE was subnormal at baseline and decreased dramatically after repair. In addition, Koestenberger et al. [26] showed that although TAPSE values in children with TOF were normal, the values gradually decreased after repair. Romeo et al. [23] explained that due to myocardial injury caused by surgical insult, occult extension of surgery at a later age, or simply altered loading conditions in the early postoperative period.

Furthermore, patients with RV dysfunction had significantly high postoperative RVOT PG. Sandeep et al. [5] found that reduced pulmonary artery systolic pressure could be the cause of RV restrictive physiology. The postoperative hemodynamic changes provide insight into the mechanism by which reduced pulmonary artery systolic pressure contributes to RV restriction physiology. Because of the surgical repair, the RV of patients with TOF must often adapt to altered pressures and volumes. Reduced pulmonary artery systolic pressure may result in inadequate filling of the RV, which would affect diastolic function. The RV may become stiff and less flexible as a result of this poor filling, a feature of restrictive physiology.

Cardiac magnetic resonance provides accurate quantitative data on cardiovascular anatomy, myocardial viability, blood flow measurements, and biventricular size and function. In patients with corrected TOF, CMR has become the noninvasive imaging modality of choice. The RVEDV, RVESV, their indices, RV EF and SV are used to identify RV dysfunction [27].

In our study, CMR was performed in patients with RV dysfunction and showed a large RV volume. The mean RVESV was 92.88±7.28 mL, the mean RVEDV was 210.25±19.54 mL, and the mean EF in patients with RV dysfunction was 42±0.04%. Shin et al. [28] agreed with our findings that the mean RVEF after TOF repair was 53±7%, the mean RVEDV was 187±55 mL, and the mean RVESV was 88±34 mL. In addition, patients with RV dilatation showed a strong trend toward restrictive RV physiology [29]. This suggests that restrictive RV physiology may be the result of an overdistended ventricle rather than a preexisting problem. In addition, patients with restrictive RV physiology have been studied for regional myocardial mechanics, including various echocardiographic features that demonstrate impaired relaxation of a noncompliant RV caused by volume overload [30].

Based on binary regression analysis, there is an insignificant statistical difference between antecedent factors and RV dysfunction, which may be due to the relatively small number of patients.

Limitations

The main strength of this study was that it was a prospective observational study, which helps physicians to recognize the potential factors for RV dysfunction in pediatric patients with TOF undergoing repair surgery. However, this study has several limitations. The generalizability of the results would have been affected by the fact that it was conducted at a single institution with a small sample size, and surgical techniques may differ from center to center, which may affect postoperative RV function. Therefore, our results may not accurately represent the patient characteristics and etiologies observed at other institutions. However, based on sample size calculations, our results may open the door for a larger study to recruit patients.

Conclusion

Low preoperative SaO2, high RVOT PG, operative transannular patch, prolonged intubation time and PICU stay, high incidence of inadequate urine output, prolonged inotropic use, as well as low postoperative TAPSE and FAC and high RVOT PG could be risk factors for postoperative RV dysfunction. Thus, physicians should be aware of these possible factors for RV dysfunction in pediatric patients undergoing TOF repair.

Funding: Self-funded
Conflict of interest: Authors declare no conflict of

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