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EXPERIENCE WITH PERCUTANEOUS RIGHT VENTRICULAR SUPPORT IN THE EARLY POST-LEFT VENTRICULAR ASSIST DEVICE IMPLANTATION PERIOD (CLINICAL CASE REPORT AND LITERATURE REVIEWS)

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Implantable left ventricular assist device (LVAD) is a state-of-the-art treatment for adults and children with end-stage heart failure. The early and late period after LVAD implantation can be severely complicated. Right ventricular failure (RVF) still remains a common complication after LVAD implantation. RVF is the cause of reduced post-implant survival. We suggest that an additional temporary or permanent right ventricular assist device (RVAD) is an effective treatment for LVAD-associated RVF. In this clinical case report, we describe the medical history of a pediatric patient (14 years old) with severe heart failure (PediMACS Level 1) against a background of dilated cardiomyopathy. The patient required peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO) prior to urgent LVAD (HM3) implantation. In the early post-LVAD implantation (1 POD) period, the patient presented with hemodynamic and echocardiographic events of acute RVF that was resistant to drug therapy (inotropic/vasopressor support, iNO) and required mechanical circulatory support (MCS) with a preoperatively implanted VA-ECMO. In the LVAD-associated RVF scenario, VA-ECMO as a means of total cardiac bypass is a non-physiological MCS method and, therefore, undesirable. In this clinical situation, our solution was to use a paracorporeal centrifugal blood pump for temporary right heart support. A RVAD was assembled using percutaneous cannulation in two sites and a modification of the pre-existing VA-ECMO circuit. For RVAD, we used an ECMO cannula previously installed through the femoral vein (26 F) and added a reverse venous cannula (22 F) through the right internal jugular vein into the pulmonary trunk. To facilitate the passage of the return cannula into the pulmonary artery, we used a contralateral sheath (6 F, 40 cm) and an Amplatz Super Stiff guidewire under radiological control. The oxygenator was removed from the circuit on day 2 of RVAD. Central hemodynamics (reduction in right atrial pressure (RAP) to 10 mm Hg, increase in pulmonary capillary wedge pressure (PCWP) to 14 mm Hg), as well as right ventricular (RV) and left ventricular (LV) volume characteristics all improved. These observations allowed us to optimize the performance of the implantable LVAD (increase in flow rate to 4.2 l/min or 2.1 l/min/m²). The duration of paracorporeal RVAD after LVAD implantation was 7 days with an average flow rate of 2.3 ± 0.2 l/min. Postoperative treatment in the intensive care unit (ICU) lasted for 15 days. The patient was discharged from the hospital on postoperative day 34.

Keywords: right ventricular assist device, left ventricular assist device, heart failure, VA-ECMO.

INTRODUCTION

Implantable left ventricular assist device (LVAD) has become widely used in the last decade not only for long-term mechanical circulatory support (MSC) before heart transplantation (HT), but also as an independent method of definitive (final) treatment of chronic heart failure (CHF) that is poorly amenable to medication [1]. There is considerable experience of successful long-term application of implantable LVADs both in adults and in children of different age categories in order to survive till HT, restore their own heart or for lifelong MSC [2]. The 2-year patient survival after LVAD implantation has reached and is no longer statistically different from that of heart transplant recipients. With this, the use of

implantable LVADs can be considered as an alternative method of CHF treatment [3].

However, despite the progress and achievement of high efficiency, long-term MCS implemented through implantable LVAD is associated with the risk of both early and late post-implant complications, which can negatively affect patient survival. One of such serious complications is RVF, which can occur in the early post-LVAD implant period and require urgent treatment measures, including application of short-term MCS by various right ventricular support techniques [4]. Chronic right ventricular dysfunction (poorly amenable to medical correction and accompanied by long-term dysfunction of implantable LVAD) in patients with previously

implanted LVAD is an indication for implantation of a second pump in order to implement RVAD and improve central and systemic hemodynamics [5].

The aim of this report is to present our own experience with percutaneous right ventricular support for correction of RVF developing in the early post-LVAD implantation period.

MATERIALS AND METHODS

Between September 2021 and November 2022, LVAD was implanted in 16 pediatric patients (<18 years old). Two (12.5%) of 16 patients developed severe RVF in the early-post LVAD implantation period, requiring additional MCS by paracorporeal RVAD. In one case, RVAD was connected by percutaneous cannulation – an example and discussion are presented in this report.

CLINICAL CASE

Patient A.A.V., 14 years old (height 180 cm, weight 80 kg, body surface area 2.0 m², body mass index 24.7 kg/m²) was admitted at Shumakov National Medical Research Center of Transplantology and Artificial Organs

with the following diagnosis: dilated cardiomyopathy, relative tricuspid and mitral regurgitation. Thromboembolism of lower lobe branch of pulmonary artery of unknown age, multiple organ failure syndrome, CHF class IIB according to the Strazhesko–Vasilenko classification, NYHA functional class 4.

At the time of admission, the patient's condition was considered critical, which was due to severe acute decompensation of CHF with the development of multiple organ dysfunction syndrome (MODS), the leading components of which, apart from impaired heart pumping function, was clinically significant hepatorenal syndrome. Given the instability of systemic hemodynamics, the need for cardiotoxic therapy (dopamine hydrochloride 8 µg/kg/min, epinephrine hydrochloride (adrenaline) 40 ng/kg/min), progression of MODS and metabolic disorders, short-term MCS by peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO) was used. Data from invasive central hemodynamic study, transthoracic echocardiographic and laboratory examination of the patient before the use of peripheral VA-ECMO are presented in Table 1.

Table 1

Results of invasive study of central hemodynamics, laboratory and instrumental examination (clinical case report, patient A.A.V., 14 years old)

Parameter	Investigation stages					
	Before VA-ECMO	Before iLVAD	After LVAD implantation and before RVAD	Against the background of both LVAD and RVAD	After RVAD removal	Before discharge
1	2	3	4	5	6	7
Examination of central hemodynamics						
HR (mins)	118	124	131	124	112	108
SBP / mAP / DBP (mmHg)	79 / 69 / 55	85 / 72 / 63	74 / 71 / 68	84 / 79 / 76	89 / 82 / 76	79 / 75 / 69
RAP (mmHg)	18	10	19	9	12	11
PASP / mPAP / PADP (mmHg)	36 / 33 / 26	34 / 29 / 22	35 / 30 / 26	35 / 26 / 16	37 / 24 / 14	–
PCWP (mmHg)	18	14	6	12	14	–
CO (l/min)	2.2	2.9	2.4	4.4	4.1	–
CI (l/min/m ²)	1.1	1.5	1.4	2.2	2.05	–
Δ PADP – PCWP (mmHg)	8	8	20	4	10	–
SV (ml)	17.8	23.4	18.3	35.5	36.6	–
SVi (ml/m ²)	8.9	11.7	9.2	17.8	18.3	–
TPG (mmHg)	15	15	24	14	10	–
RAP / PCWP	1.00	0.71	3.17	0.75	0.9	–
PVR (Wood units)	6.8	5.17	10.0	3.18	2.17	–
PVR (dynes/sec/cm ⁵)	545	414	800	254	174	–
RVSWI (gm/beat/m ²)	1.8	3.0	1.4	4.1	3.0	–
Dopamine (µg/kg/min)	8	6	6	6	–	–
Dobutamine (µg/kg/min)	–	–	4	2	–	–
Adrenaline (ng/kg/min)	40	10	20	20	6	–
VA-ECMO, Blood flow (l/min)	–	3.3	2.4	–	–	–
Number of pump revolutions (rpm)	–	6000	4900	–	–	–
iLVAD						
Pump Flow (l/min)	–	–	1.8	3.8	3.8	3.6
Pump Speed (rpm)	–	–	4650	4600	4950	4800
Pulse Index	–	–	2.2	4.4	5.0	5.3
Pump Power (watt)	–	–	2.9	3.2	3.3	2.6

End of Table 1

1	2	3	4	5	6	7
Transthoracic/transesophageal echocardiography						
Ao (annulus fibrosus) (cm)	1.9	1.9	1.9	1.9	1.9	1.9
Ao (ascending aorta) (cm)	2.6	2.6	2.6	2.6	2.6	2.6
LA (cm)	4.0	3.9	2.8	3.2	3.3	3.2
LA (ml)	80	74	38	53	56	51
RA (ml)	80	56	92	53	62	64
RV (cm)	3.4	2.9	3.6	3.1	3.2	3.3
RVF1	3.2	2.6	3.4	2.7	3.6	3.7
RVF2	3.0	2.9	3.2	2.5	3.2	3.2
RVF3	6.2	4.4	6.0	4.9	5.8	6.2
IVS (cm)	0.8	0.8	0.8	0.8	0.8	0.8
PW (cm)	0.8	0.8	0.8	0.8	0.8	0.8
LVEDV (ml)	200	182	71	88	84	98
LVESV (ml)	176	151	58	68	66	76
SV (ml)	24	31	13	20	18	22
LVEF (%)	12	17	18	22	21	22
Mitral valve regurgitation (grade)	2.5	3.0	2.5	2.5	2	2
Tricuspid regurgitation (grade)	2.0	2	3.0	2.0	2	2
TAPSE (mm)	18	17	8	11	14	14
Laboratory investigation						
Leukocytes,	13.9	10.7	16.2	17.8	14.5	9.3
stab cells (%)	1.0	3	7	5	1	2.0
segmented cells (%)	82.0	87	81	77	87	68.7
eosinophils (%)	0.5	0.1	0.0	1.4	2	1.5
basophils (%)	0.2	0.1	0.1	0.6	0	0.8
lymphocytes (%)	9.0	8	7	7.3	4	21.0
monocytes (%)	8.0	2	5	8.5	6	6.0
Hemoglobin (g/dL)	10.0	9.0	7.9	8.9	9.0	8.1
Red blood cells ($10^{12}/L$)	3.5	3.14	2.79	2.94	2.97	2.6
Hematocrit (%)	31.0	27.4	24.6	27.4	27.9	26.0
Platelets ($10^9/L$)	72	75	48	41	74	92
Albumin (g/L)	32.3	44.5	42.5	35.6	36.5	35.1
Total protein (g/L)	67.3	71.4	72.3	67.7	66.3	71.3
Total bilirubin ($\mu\text{mol}/L$)	40	32.6	32.8	38.5	36.1	10.9
ALT (U/L)	255	171.3	73.8	27.0	24.8	19.6
AST (U/L)	152	35.2	89.5	32.3	30.4	39.2
Urea, mmol/L	22	21.2	20.1	12.2	15.0	5.6
Creatinine ($\mu\text{mol}/L$)	112	110.8	75.1	78.0	89.1	31.7
PI (%)	64	55	66	65	53	40
INR	1.7	2.05	1.65	1.68	2.15	3.0
Fibrinogen (g/dL)	2.25	1.72	1.50	1.62	1.35	3.93
pH _b	7.274	7.541	7.437	7.499	7.439	7.382
BE _b (mmol/L)	-5.8	10.2	1.9	2.9	3.9	2.2
P _b CO ₂ (mmHg)	47.9	48.6	38.8	43.6	41.9	37.9
P _b O ₂ (mmHg)	27.1	26.4	26.7	27.6	32.6	33.4
S _b O ₂ (%)	48.6	40.4	46.5	49.6	58.7	62.3
Lactate (mmol/L)	3.9	2.1	5.8	1.5	1.6	1.2
Glucose (mmol/L)	5.7	7.1	7.2	6.5	6.7	5.3
K ⁺ (mmol/L)	3.8	4.0	4.6	4.2	3.9	4.3
Na ⁺ (mmol/L)	131	144	138	143	135	138

Note: HR, heart rate; SBP, systolic blood pressure; mAP, mean arterial pressure; DBP, diastolic blood pressure; RAP, right atrial pressure; PASP, pulmonary artery systolic pressure; mPAP, mean pulmonary artery pressure; PADP, pulmonary artery diastolic pressure; PCWP, pulmonary capillary wedge pressure; CO, cardiac output; CI, cardiac index; SV, stroke volume; SVi, stroke volume index; TPG, transpulmonary pressure gradient; PVR, pulmonary vascular resistance; RVSWI, right ventricular stroke work index; VA-ECMO, venoarterial extracorporeal membrane oxygenation; iLVAD, implantable left ventricular assist device; Pump Flow, iLVAD flow rate; Pump Speed, pump rotational speed; Ao, aorta; LA, left atrium; RA, right atrium; RV, right ventricle; RVF1, RVF2, RVF3, end-diastolic size of the RV at the basal and midline sections, and longitudinal size of the RV; IVS, interventricular septum; PW, left ventricular posterior wall; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; TAPSE, tricuspid annular plane systolic excursion; ALT, alanine aminotransferase; AST, aspartate aminotransferase; PI, prothrombin index; INR, international normalized ratio.

The patient was connected to ECMO circuit via percutaneous puncture cannulas inserted into the right femoral vein (venous cannula 26 F) and the right femoral artery (arterial cannula 15 F). To prevent right lower extremity ischemia, the right superficial femoral artery was catheterized using a 15 G single-lumen catheter connected through a perfusion line (trunk) to the arterial part of the VA-ECMO circuit. VA-ECMO parameters were as follows: centrifugal pump speed, 6600 rpm; volumetric blood flow rate, 3.3 L/min or 1.6 L/min/m²; flow gas (sweep gaze) volume rate, 3.5 L/min; flow gas FiO₂, 0.80.

Given the increased risk of heart transplantation (HT) against the background of high pre-transplant pulmonary hypertension (PH) (TPG, 15 mmHg; PVR, 6.8 and multiple organ dysfunction), LVAD was implanted as a method of long-term pre-transplant MCS and regression of PH. From the moment of admission at Shumakov Center until the time of surgery, the patient was on renal replacement therapy by continuous veno-venous hemofiltration, which was then continued in the intra- and post-operative period. The patient was assigned a Pedimacs I preimplantation level. Short-term MCS by peripheral VA-ECMO before LVAD implantation lasted for 5 days. Against the background of VA-ECMO application, metabolic and multiple organ disorders regressed. LVAD was implanted from the median sternotomy approach under cardiopulmonary bypass (CPB) and on a beating heart. Before the operation, the venous cannula of the VA-ECMO circuit was lowered caudally to 35 cm at the level of percutaneous inlet to prevent competition with the CPB venous cannula inserted into the inferior vena cava. During CPB, VA-ECMO flow rate was 0.8–1.0 L/min with a proportional decrease in the volume rate of CPB and the volume rate of flow gas supply to the membrane oxygenator of the CPB circuit. CPB lasted for 67 minutes. An LVAD HeartMate III model by Abbott Corp. (USA) with a centrifugal pump functioning on the principle of magnetic levitation was implanted. Inhaled nitric oxide (iNO) at 20 ppm dose was used as a selective pulmonary vasodilator to reduce increased pulmonary vascular tone and prevent the development of right ventricular failure (RVF) in the early post-implantation period.

At the end of the surgical stage of LVAD implantation, we started a stepwise (by 1 l/min) decrease in the CPB volume rate at a constant VA-ECMO volume rate (0.8–1.0 l/min) against gradual increase in the rate of revolution of the implanted LVAD (by 200 rpm) under control of its flow rate and echocardiographic parameters of filling and contractility of the right and left ventricles of the heart and the interventricular septum location. In the early post-implantation period, despite significant cardiotoxic (dopamine hydrochloride 6.0 µg/kg/min, dobutamine hydrochloride 4.0 µg/kg/min, epinephrine hydrochloride 60 ng/kg/min) and vasodilator

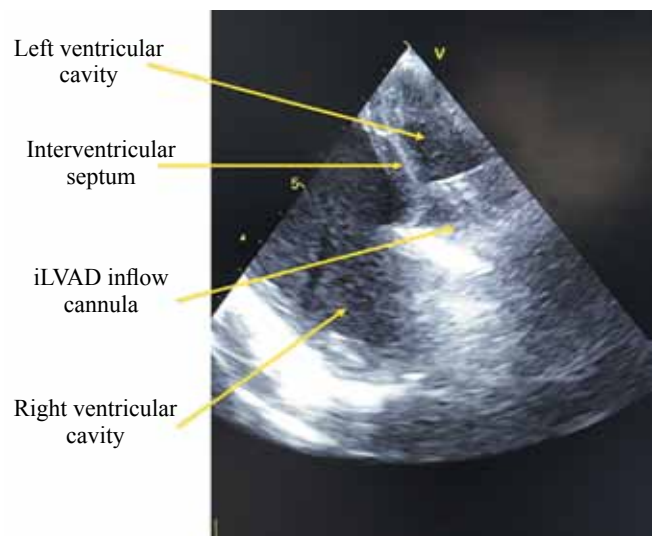


Fig. 1. iLVAD flow cannula suction to the interventricular septum against the background of acute RVF, accompanied by an increase in its volume with a simultaneous reduction in the left ventricular cavity (transesophageal echocardiography). iLVAD, implantable left ventricular assist device

(iNO) therapy at a constant VA-ECMO volume rate (0.8–1.0 L/min), acute right ventricular therapy was noted, which was manifested by increase in RAP to 19 mm Hg, decrease in PCWP to 6 mmHg with a simultaneous decrease in CI to 1.4 L/min/m² and LVAD flow rate to 2.2 L/min or 1.1 L/min/m². Transesophageal echocardiogram revealed increased RV volumetric characteristics, severe hypokinesis of its free wall and outlet section, up to grade 3 tricuspid regurgitation with simultaneous displacement of the interventricular septum toward the left ventricle, sharp reduction in left atrial and left ventricular cavity with LVAD suction cannula to the interventricular septum) (Fig. 1). In order to improve systemic blood flow against the background of acute RVF, the peripheral VA-ECMO volume rate was increased to 3.0 l/min. Given “unphysiological” MCS by peripheral VA-ECMO under LVAD conditions, leading to volumetric underload of the left heart and impaired heart function, it was decided to switch from peripheral VA-ECMO to percutaneous RVAD. The goal was to provide increased blood flow to the left heart and increase LVAD flow rate.

Percutaneous RVAD implantation technique

Under local anesthesia, the puncture and catheterization of the right internal jugular vein was performed, with a J-shaped endovascular contralateral introducer (40 cm, 6 F) with an internal stent (Fig. 2), guided through the right atrium into the RV cavity. Manipulations were performed under fluoroscopic control. A super-stiff metal conduit was passed through the J-shaped introducer sheath into the right heart and then into the right pulmonary artery (Fig. 3). After removal of the

introducer and subsequent dilation of the skin canal with percutaneous dilators, a venous ECMO cannula (22 F) was passed through the right internal jugular vein, the right heart into the pulmonary artery trunk to below 1 cm from the left and right pulmonary veins (Fig. 4). This cannula was connected to the VA-ECMO circuit arterial trunk separated from the arterial cannula to perform percutaneous RVAD according to the inferior vena cava-pulmonary artery scheme (Fig. 5). After RVAD initiation, the arterial cannula was extracted from the lumen of the right femoral artery.

Early post-LVAD implantation period against the background of paracorporeal RVAD

Against the background of RVAD, there was an improvement in central hemodynamics (decrease in RAP to 10 mmHg, increase in PCWP to 14 mmHg) and RV and LV volumetric characteristics. This allowed to optimize LVAD functioning (increase in flow rate to 4.2 l/min or 2.1 l/min/m²). Postoperative MV lasted for 28 hours, postoperative application of renal replacement therapy by continuous veno-venous hemofiltration (CVVH)



Fig. 2. J-shaped contralateral endovascular introducer sheath and super stiff endovascular guidewire for improved percutaneous venous cannulation from the right internal jugular vein into the pulmonary artery

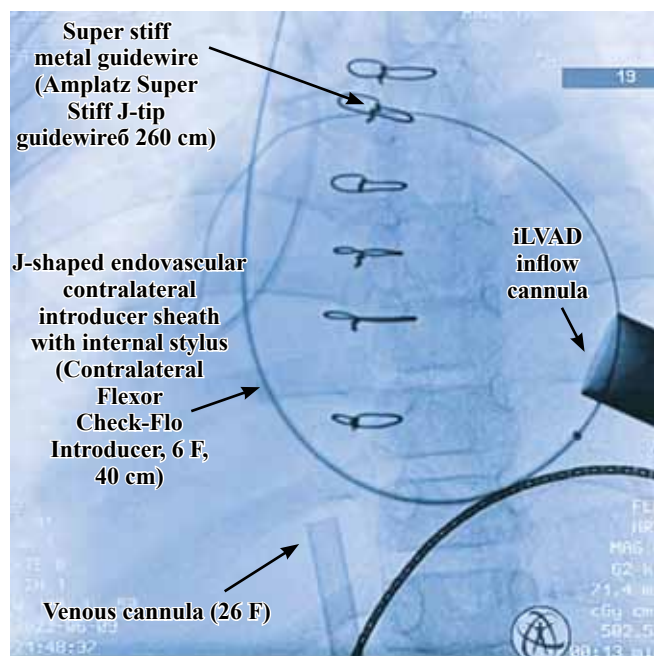


Fig. 3. Passage of a super stiff endovascular guidewire through the J-shaped endovascular contralateral introducer with an internal stylet into the pulmonary artery (fluoroscopic image)

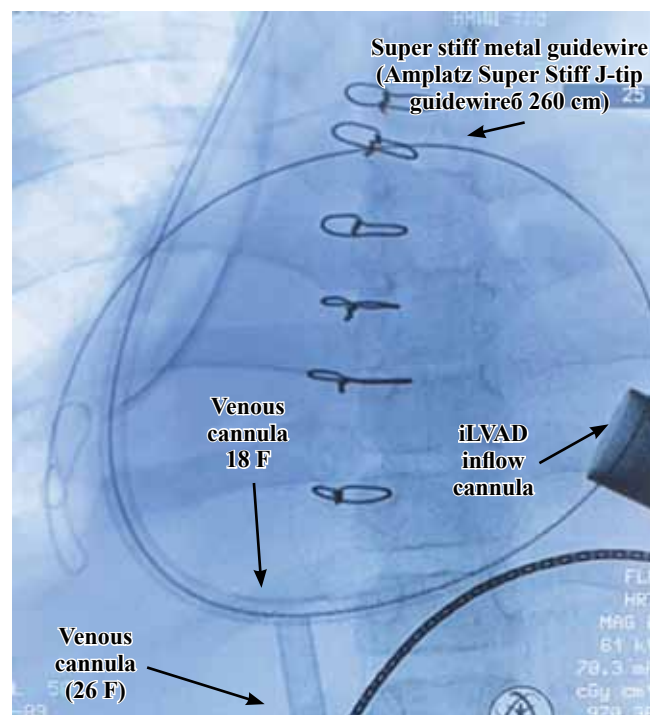


Fig. 4. Insertion of a return venous cannula via a super stiff endovascular guidewire through the right heart into the pulmonary artery (fluoroscopic image)

Acute right ventricular dysfunction that developed after LV assist device implantation and required paracorporeal right ventricular bypass under the “inferior vena cava – pulmonary artery” scheme

M., 14 years old

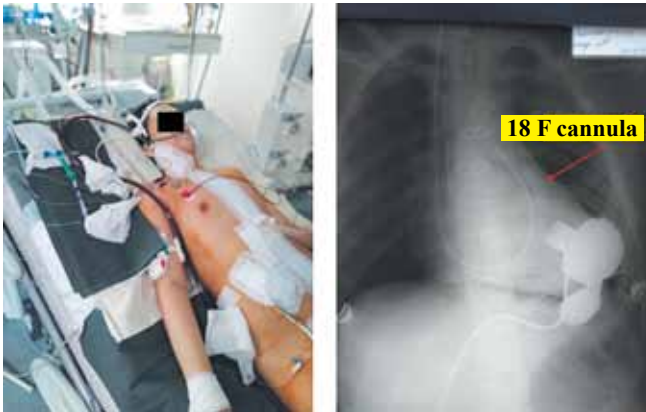


Fig. 5. The patient with percutaneous paracorporeal right ventricular assist device (left photo) and venous (outflow) cannula inserted through the right vena cava into the pulmonary artery (right, radiographic image)

lasted for 3 days. Membrane oxygenator remained integrated in the paracorporeal RVAD circuit for the first 2 days in order to control blood gas composition. The membrane oxygenator was then disconnected from the extracorporeal circuit in the absence of gas exchange function disorders in the lungs. Paracorporeal RVAD after LVAD implantation lasted for 7 days with an average flow rate of 2.3 ± 0.2 l/min. After gradual reduction in RVAD flow rate under the control of central hemodynamics and transthoracic echocardiography, cannulas were removed by purse-string suturing around the percutaneous inlet. After RVAD termination, there were no hemodynamic and echocardiographic signs of clinically severe RVF in the follow-up period (see Table 1). Postoperative treatment in the ICU took 15 days. The patient was discharged from the hospital on postoperative day 34. Baseline post-implantation drug therapy included: ivabradine 5 mg, acetylsalicylic acid 100 mg, warfarin 3.75 mg, sildenafil 25 mg twice daily. Implantable LVAD parameters at the time of discharge from hospital: pump flow 3.6 l/min or 1.8 l/min/m², pump speed 4800 rpm, pump power 2.6 watts, pulse index 5.3. At the time of writing, the patient was under outpatient care without clinical and echocardiographic manifestations of late RV dysfunction and with optimal functioning parameters of the implanted LVAD.

DISCUSSION

RVF is a frequent complication following LVAD implantation. It leads to impaired performance and, accordingly, to lower efficiency of MCS itself, multiple organ failure and death [6]. According to various studies, the incidence of acute RVF in the early period following LVAD implantation ranges from 17% to 50% [7, 8].

There is still no generally accepted definition of acute RVF resulting after LVAD implantation. Potapov E. et al. (2008) defined acute RVF as right ventricular pump function failure developing within the first 48 hours after LVAD implantation in the absence of hemopericardial tamponade. It is accompanied by the following manifestations: mean BP ≤ 55 mmHg, RAP ≥ 16 mmHg, mixed venous oxygen saturation (SvO₂) $\leq 55\%$, CI < 2.0 L/min/m² despite inotropic support [9].

RVF is accompanied by decreased efficiency in the implantable LVAD due to decreased blood flow to the left heart and development of complications specific for this variant of MCS, such as hemolysis, aortic insufficiency, ventricular arrhythmias resulting from creation of increased negative pressure and inflow cannula suction to the left ventricular wall [10]. Besides, when the axial or centrifugal pump turns too frequently, excessive unloading of the left heart occurs, which, even in the absence of preexisting RVF, causes displacement of the interventricular septum, interventricular relationship disorder and its development, especially in hypovolemia [10].

Recently, depending on the period of occurrence after LVAD implantation, acute (< 48 hours), early (> 48 hours and < 14 days) and late (> 14 days) forms of RVF, differing in approaches to its drug and non-drug treatment, have been distinguished [11]. As the life expectancy of patients with implantable LVAD increases, the incidence of late RVF increases, with clinical and instrumental manifestations being detected in more than 40% of cases 2 years after implantation [12]. According to INTERMACs criteria, depending on RAP level, dosage and duration of inotropic and vasodilator therapy, RVAD requirement and risk of death, mild, moderate, severe and acute severe forms of RVF in patients with implantable LVAD are distinguished [13].

There are pre-implantation and peri-implantation risk factors of post-LVAD implantation RVF. Numerous parameters of preoperative laboratory, hemodynamic, echocardiographic and MRI examinations have been suggested to predict the development of RVF both early and late after LVAD implantation (Table 2) [7, 14–16]. High prognostic significance of underlying myocardial imaging (speckle-tracking echocardiography, magnetic resonance imaging) with determination of segmental and global RV deformation to predict its dysfunction in various clinical situations, including LVAD implantation, has been demonstrated [17].

Pre-existing RVF is the leading cause of exacerbation of RV pumping dysfunction after LVAD implantation [8]. Patients with preimplantation low TAPSE value, RV free wall strain, RVFAC and high values of RV/LV end-diastolic area ratios, left atrial (LA) volume index, LA diameter/LV end-diastolic diameter ratio (ratio have an increased risk of RVF after LV implantation [18]. Severe pre-implantation tricuspid regurgitation increases the risk of early RVF 3.5-fold in patients with LVAD [19]. The

relevant prognostic scales – Michigan RV failure risk score and Bayesian model – have been developed for predicting post-LVAD RVF [20, 21]. In patients with high risk of early or late severe RVF, biventricular implantable circulatory support should be considered as the method

of choice for long-term MCS [22]. The outcome of long-term MCS in patients with delayed RVAD implantation is 4.8 times worse than the survival rates of patients with simultaneous LVAD and RVAD implantation [23]. It should be taken into account that RVF can also develop

Table 2

Preoperative clinical, laboratory and instrumental factors of RVF after LVAD implantation

Parameter groups	Factors
Clinical manifestations	Systemic circulatory insufficiency
	Ascites
	Liver dysfunction
	Kidney dysfunction
	Obesity
	Tachycardia
	Inotropic/vasopressor support
	Mechanical circulatory support
	Renal replacement therapy
	MV
Laboratory parameters	Total bilirubin ≥ 2.0 mg/dL
	AST ≥ 80 U/L
	Urea nitrogen > 39 mg/dL
	Blood creatinine ≥ 2.3 mg/dL
	MELD
	Glomerular filtration rate by MDRD 4
Echocardiographic parameters	Right atrial size
	Anteroposterior RV end-diastolic area (D1, D2, D3)
	Indexed RV end-diastolic volume
	Indexed RV end-systolic volume
	RV ejection fraction
	Basal RV/LV end-diastolic area ratio (RVED1/LV) ≥ 0.75
	LA volume index
	LA diameter/LV end-diastolic diameter ratio
	RV fractional area change (RVFAC)
	Tricuspid regurgitation Grade 3.
	TAPSE < 8 mm
	Tricuspid annular peak systolic velocity (TAPSV) < 8 cm/sec
	Pulsed Doppler transmitral E wave (Em) / tissue Doppler lateral systolic velocity (SLAT) ≥ 18.5
	RV strain by speckle tracking technology: – RV global longitudinal strain (GLS) – RV free wall strain (RVFWS) $> -11.8\% \dots -9.6\%$ – RV systolic longitudinal strain rate < 0.6 /sec
Hemodynamic parameters	CVP (RAP) > 15 mmHg
	RAP/PCWP ratio > 0.55
	Pulmonary arterial pulse pressure (PASP – PADP)
	Pulmonary artery pulsatility index: pulmonary arterial pulse pressure / RAP < 2.0
	Pulmonary artery compliance: SV/pulmonary arterial pulse pressure
	Pulmonary effective arterial elastance (PASP/SV)
	Right ventricular stroke work index (RVSWI, right ventricular stroke work index) < 0.25 mmHg/L \times m ²
	Right ventricular systolic working index (RVSWI): pulmonary arterial pulse pressure – RAP/SVi
	Pulmonary vascular resistance (PVR)
	Simple right ventricular contraction pressure index (sRVCPI): TAPSE \times (RV-RA pressure gradient) < 400
MRI parameters	RV global longitudinal strain

Note: MV, Mechanical ventilation; AST, aspartate aminotransferase; MELD, Model for End-Stage Liver Disease; MDRD 4, Modification of Diet in Renal Disease; RV, right ventricular; LV, left ventricular; LA, left atrial; CVP, central venous pressure; RAP, right atrial pressure; PASP, pulmonary artery systolic pressure; PADP, pulmonary artery diastolic pressure; PCWP, pulmonary capillary wedge pressure; SV, stroke volume; SVi, stroke volume index.

in patients without preimplantation risk factors due to perioperative RV myocardial damage of various genesis (embolism, mechanical trauma, etc.) [23].

The strategy for prevention and treatment of acute RVF following LVAD implantation includes tricuspid annuloplasty at the same time as implantation amidst severe insufficiency, strictly controlled volemic load, gradual increase in the implanted pump speed under strict echocardiographic control of the interventricular septum position, use of pulmonary vasodilators (inhaled nitric oxide, prostaglandin E1, prostacyclin, sildenafil) or inodilating effect (dobutamine, milrinone, levosimendan) in patients with high level of pulmonary vascular resistance, correction of hypoxemia and hypercapnia [24–26]. It should be taken into account that in patients without preimplantation pulmonary hypertension, the use of pulmonary vasodilator therapy is ineffective in acute RVF following LVAD implantation [27]. The combination of acute RVF with postperfusion vasoplegic syndrome requiring the use of high vasopressor support is associated with extremely high postoperative mortality [28].

Timely initiated MCS in ineffective drug treatment of acute RVF provides correction of systemic hemodynamic disorders, organ perfusion and prevention of the development of multiple organ disorders [4]. In conditions of implantable LVAD, the use of VA-ECMO should be considered a non-physiological method of MCS in the development of acute RVF, as it is an indirect bypass of the heart and leads to decreased blood flow to the left heart and malfunction of the implanted blood pump. In this scenario of acute RVF development from a hemodynamic point of view, it is more justified to use RVAD, which provides increased blood flow to the left heart, increased performance of the implanted LVAD and improved systemic circulation.

Currently, several extracorporeal RVAD techniques based on the use of external centrifugal pump of various modifications (Biopump Medtronic, RotoFlow, CentriMag, Medos, TandemHeart-RV, etc.) or microaxial catheter pump (Impella-RP) are implemented in clinical practice. Accordingly, the central RVAD connection technique based on the centrifugal pump in the right atrium-pulmonary artery scheme, requiring repeated sternotomy during decannulation, or percutaneous cannulation technique using two single-lumen cannulas or one double-lumen cannula have been developed and are used in practice [29]. Percutaneous separate cannulation can be performed by passing an inflow and outflow cannula through the femoral veins (bifemoral approach) or inflow cannula through the femoral vein and outflow cannula through the right jugular vein. In both cases, the outflow cannula should be passed through the right heart and positioned in the lumen of the pulmonary artery [30]. Recently, the technique of percutaneous RVAD performed with a double-lumen cannula inserted through the right internal jugular vein and the right parts of the

heart into the pulmonary artery (ProtekDuo) has been developed and introduced into clinical practice, to correct acute RVF developed in various clinical situations (heart transplantation, heart attack, acute post-cardiotomy heart attack, LVAD implantation, pulmonary embolism, high pulmonary hypertension, etc.) [31]. The double-lumen cannula facilitates RVAD through unilateral vascular access (internal jugular vein), which makes this MCS technique the least traumatic in acute RVF of postcardiac, postimplantation or posttransplantation genesis [32].

Timely initiation of MCS by RVAD provides better treatment outcomes for acute RVF following LVAD implantation compared to delayed start of assisted circulation [33]. In case of concomitant critical disorders in gas exchange function of the lungs (hypoxemia/hypercapnia), RVAD can be supplemented with ECMO. Separate studies demonstrate better survival rates when RVAD is combined with ECMO [34, 35]. It is necessary to maintain lower RVAD flow rate in relation to LVAD, whose level should not exceed 4.0 L/min in adult patients to ensure prevention of pulmonary edema or pulmonary hemorrhage [36]. The duration of RVAD use for acute RVF following LVAD implantation averages about 5 days [37].

Our own experience with LVAD implantation in pediatric patients (under 18 years of age) demonstrates that severe acute RVF, requiring the use of short-term MCS by paracorporeal centrifugal RVAD, developed in 12.5% of observations. This article provides an example of successful RVAD use in a 14-year-old patient. The possibility of percutaneous cannulation for RVAD (without the need for a re-sternotomy) to install and subsequently remove the inflow and outflow cannulas, carried out through the right femoral vein into the inferior vena cava and the right internal jugular vein into the pulmonary artery respectively, is demonstrated. Application of short-term RVAD improved the central hemodynamics, increased blood flow to the left heart and increased LVAD performance. RVAD duration for 7 days was sufficient to improve RV pumping function and discontinue its use of short-term MCS. In addition, this clinical observation demonstrates the continuity of assisted circulation in a patient with end-stage CHF and preimplantation INTERMACS Level 1 with consecutive transition from VA-ECMO to long-term MCS by implantable LVAD.

CONCLUSION

Short-term MCS by paracorporeal centrifugal RVAD is an effective remedy for hemodynamic disorders caused by early acute post-LVAD RVF. The percutaneous cannulation technique is a less traumatic way to perform right ventricular bypass.

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REFERENCES

1. Frigerio M. Left ventricular assist device: indication, timing, and management. *Heart Fail Clin.* 2021; 17: 619–634.
2. Gustaffson F, Rogers JS. Left ventricular assist device therapy in advanced heart failure: patient selection and outcomes. *Eur J Heart Fail.* 2017; 19: 595–602.
3. Shah P, Yuzefpolskaya M, Hickey GW, Breathett K, Wever-Pinzon O, Khue-Ton V et al. Twelfth Interagency Registry for Mechanically Assisted Circulatory Support report: readmissions after left ventricular assist device. *Ann Thorac Surg.* 2022; 113: 722–737.
4. Noly PE, Kirsch M, Quessard A, Leger P, Pavie A, Amour J, Leprince P. Temporary right ventricular support following left ventricular assist device implantation. *Interact Cardiovasc Surg.* 2014; 19: 49–55.
5. Dandel M, Krabatsch T, Falk V. Left ventricular vs bi-ventricular mechanical support: decision making and strategies for avoidance of right heart failure after left ventricular assist device implantation. *Int J Cardiol.* 2015; 198: 245–250.
6. Saito S, Matsumiya G, Sakaguchi T, Miyagawa S, Yoshikawa Y, Yamauchi T et al. Risk factor analysis of long-term support with left ventricular assist system. *Circ J.* 2010; 74: 715–722.
7. Hadman R, Charif F, Kadri Z. Right ventricle failure in patients treated with left ventricular assist device. *Annales de Cardiologie et al d'Angeologie.* 2022; 69: 51–54.
8. Patlolla B, Beygui R, Haddad F. Right-ventricular failure following left ventricle assist device implantation. *Curr Opin Cardiol.* 2013; 28: 223–233.
9. Potapov EV, Stepanenko A, Dandel M, Kukucka M, Lehmkuhl HB, Weng Y et al. Tricuspid incompetence and geometry of right ventricle as predictors of right ventricular function after implantation of a left ventricular assist device. *J Heart Lung Transplant.* 2008; 27: 1275–1281.
10. Lampert B, Teuteberg JJ. Right ventricular failure after left ventricular assist devices. *J Heart Lung Transplant.* 2015; 34: 1123–1130.
11. Raina A, Patarroyo-Aponte M. Prevention and treatment of right ventricular failure during left ventricular assist device therapy. *Crit Care Clin.* 2018; 34: 439–452.
12. Kapelios CJ, Charitos C, Kaldara E, Malliaras K, Nana E, Patsios C et al. Late-onset right ventricular dysfunction after mechanical support by a continuous-flow left ventricular assist device. *J Heart Lung Transplant.* 2015; 34: 1604–1610.
13. Kirklin JK, Pagani FD, Kormos RL, Stevenson LW, Blume ED, Myers SL et al. Eight annual INTERMACS report: special focus on framing the impact of adverse events. *J Heart Lung Transplant.* 2017; 36: 1080–1086.
14. Dufendach KA, Zhu T, Diaz Castrillon C, Hong Y, Countouris ME, Hickey G et al. Pre-implant right ventricular free wall strain predicts post-LVAD right heart failure. *J Card Surg.* 2021; 36: 1996–2003.
15. Kang G, Ha R, Banerjee D. Pulmonary artery pulsatility index predicts right ventricular failure after left ventricular assist device implantation. *J Heart Lung Transplant.* 2016; 35: 67–73.
16. Kiernan MS, French AL, DeNofrio D, Parmar YJ, Pham DT, Kapur NK et al. Preoperative three-dimensional echocardiography to assess risk of right ventricular failure after left ventricular assist device surgery. *J Card Fail.* 2015; 21: 189–197.
17. Golukhova EZ, Slivneva IV, Mamalyga ML, Marapov DI, Alekhin MN, Skopin II, Antonova DE. Right ventricular free-wall longitudinal speckle tracking strain as a prognostic criterion of adverse outcomes in patients with pulmonary hypertension: a systematic review and meta-analysis. *Russian Journal of Cardiology.* 2021; 26 (4): 4417. (In Russ.).
18. Kiernan MS, Grandin EW, Brinkley M Jr, Kapur NK, Pham DT, Ruthazer R et al. Early right ventricular assist device uses in patients undergoing continuous-flow left ventricular assist device implantation. Incidence and risk factors from the Interagency Registry for Mechanically Assisted Circulation Support. *Circ Heart Fail.* 2017; 10: e003863.
19. Ruiz-Cano MJ, Morshuis M, Koster A, Lauenroth V, Prashovikj E, Gummert J, Paluszkiwicz L. Risk factors of early ventricular failure in patients undergoing LVAD implantation with intermediate Intermacs profile for advanced heart failure. *J Card Surg.* 2020; 35: 1832–1839.
20. Loghmanpour NA, Kormos RL, Kanwar MK, Teuteberg JJ, Murali S, Antaki JF. A Bayesian model to predict right ventricular failure following left ventricular assist device therapy. *JASS Heart Fail.* 2016; 4: 711–721.
21. Matthews JC, Koelling TM, Pagani FD, Aaronson KD. The right ventricular failure risk score a pre-operative tool for assessing the risk of right ventricular failure in left ventricular assist device candidates. *J Am Coll Cardiol.* 2008; 51: 2163–2172.
22. Krabatsch T, Potapov E, Stepanenko A, Schweiger M, Kukucka M, Huebler M et al. Biventricular circulatory support with two miniaturized implantable assist devices. *Circulation.* 2011; 124: S179–S186.
23. Takeda K, Naka Y, Yang JA, Uriel N, Colombo PC, Jorde UP, Takayama H. Outcome of unplanned right ventricular assist device support for severe right heart failure after implantable left ventricular assist device insertion. *J Heart Lung Transplant.* 2014; 33: 141–148.
24. Potapov E, Meyer D, Swaminathan M, Ramsay M, El Banayosy A, Diehl C et al. Inhaled nitric oxide after left ventricular assist device implantation: a prospective, randomized, double-blind, multicenter, placebo-controlled trial. *J Heart Lung Transplant.* 2011; 30: 870–878.
25. Klodell CT Jr, Morey TE, Lobato EB, Aranda JM Jr, Staples ED, Schofield RS et al. Effect of sildenafil on pulmonary artery pressure, systemic pressure, and nitric oxide utilization in patients with left ventricular assist devices. *Ann Thorac Surg.* 2007; 83: 68–71.
26. Theiss HD, Grabmaier U, Kreissl N, Hagl C, Steinbeck G, Sodian R et al. Preconditioning with levosimendan before implantation of left ventricular assist devices. *Artif Organs.* 2014; 38: 231–234.
27. Sabato LA, Salerno DM, Moretz JD, Jennings DL. Inhaled pulmonary vasodilator therapy for management of

- right ventricular dysfunction after left ventricular assist device placement and cardiac transplantation. *Pharmacotherapy*. 2017; 37: 944–955.
28. de Waal EEC, van Zaane B, van der Schoot MM, Huisman A, Ramjankhan F, van Klei WA, Marczin N. Vaso-plegia after implantation of a continuous flow left ventricle assist device: incidence, outcomes and predictors. *BMC Anesthesiol*. 2018; 18 (1): 185.
 29. Dandel M, Hetzer R. Temporary assist device support for the right ventricle: pre-implant and post-implant challenges. *Heart Failure Review*. 2018; 23: 157–171.
 30. Haneya A, Philipp A, Puehler T, Rupprecht L, Kobuch R, Hilker M et al. Temporary percutaneous right ventricular assist support using a centrifugal pump in patients with postoperative acute refractory right ventricular failure after left ventricular assist device implantation. *Eur J Cardiothorac Surg*. 2012; 41: 219–223.
 31. Carrozzini M, Merlanti B, Olivieri GM, Lanfranconi M, Bruschi G, Mondino M, Russo CF. Percutaneous RVAD with the Protect Duo for severe right ventricular primary graft dysfunction after heart transplant. *J Heart Lung Transplant*. 2021; 40: 580–583.
 32. Schmack B, Weymann A, Popov AF, Patil NP, Sabashnikov A, Kremer J et al. Concurrent left ventricular assist device (LVAD) implantation and percutaneous temporary RVAD support via CardiacAssist Protec-Duo TandemHeart to preempt right heart failure. *Med Sci Monit Basic Res*. 2016; 22: 53–57.
 33. Morgan JA, John R, Lee BJ, Oz MC, Naka Y. Is severe right ventricular failure in left ventricular assist device recipients a risk factor for unsuccessful bridging to transplant and post-transplant mortality? *Ann Thorac Surg*. 2004; 77: 859–863.
 34. Leidenfrost J, Prasad S, Itoh A, Lawrance CP, Bell JM, Silvestry SC. Right ventricular assist device with membrane oxygenator support for right ventricular failure following implantable left ventricular assist device placement. *Eur J Cardiothorac Surg*. 2016; 49: 73–77.
 35. Joshi Y, Borjes MC, Aissaoui N, Grinda JM, Bel A, Latremouille C, Jouan J. Percutaneous venopulmonary artery extracorporeal membrane oxygenation for right heart failure after left ventricular assist device insertion. *Interact Cardiovasc Thorac Surg*. 2021; 33: 978–985.
 36. Welp H, Sindermann JR, Deschka H, Martens S, Scherer M. Pulmonary bleeding during right ventricular support after left ventricular assist device implantation. *J Cardiothorac Vasc Anesth*. 2016; 30: 627–631.
 37. Saito S, Sakaguchi T, Miyagawa S, Nishi H, Yoshikawa Y, Fukushima S et al. Recovery of right heart function with temporary right ventricular assist using a centrifugal pump in patients with severe biventricular failure. *J Heart Lung Transplant*. 2012; 31: 858–864.

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