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Combined Balloon Pulmonary Angioplasty and Pulmonary Endarterectomy in Patients with Chronic Thromboembolic Pulmonary Hypertension

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Abstract

Introduction. The combination of pulmonary thromboendarterectomy (PEA) and balloon pulmonary angioplasty (BPA) in patients with chronic thromboembolic pulmonary hypertension (CTEPH) is increasingly performing for the treatment. **Objective:** To present our experience of step-by-step surgical treatment of patients with CTEPH and severe pulmonary hypertension (PH). **Methods:** Retrospective analysis of two-stage treatment 27 patients from January 2019 to March 2024 was conducted. BPA as the first stage of treatment was performed to improve hemodynamic data and reduce surgical risk. After 3 - 4 months, the same patients underwent PEA. **Results:** The median age at diagnosis was 47 (IQR 15 - 78) years, 51.9% males and 48.1% females. Due to New York Heart Association (NYHA), 1 (3.7%) patient belonged to class II, 19 (70.4%) to class III and 7 (25.9%) to class IV. After BPA the mean pulmonary artery pressure (mPAP, mmHg) and pulmonary vascular resistance (PVR, dynes*s*cm^5) significantly decreased (66.2 ± 9.5 vs 53.7 ± 9.5, $P < 0.0001$; 1276 ± 448 vs 772 ± 341, $P < 0.0000001$, respectively). After PEA mPAP (mmHg) and PVR (dynes*s*cm^5) also significantly decreased (51.3 ± 5.5 vs 27.6 ± 4.5, $P < 0.0001$; 776 ± 232 vs 388 ± 141, $P < 0.0000001$). Clinical evidence has improved: NYHA class I was observed in 8 (29.6%) patients, class II—in 16 (59.3%) and class III—in 3 (11.1%) patients. In-hospital mortality rate was 3.7% (one patient died after PEA). **Conclusion:** A staged approach may be an effective surgical tactic in patients with CTEPH and severe PH. However, it is necessary to identify criteria for patient selection, and procedural efficacy.

Keywords

Chronic Thromboembolic Pulmonary Hypertension, Pulmonary Thromboendarterectomy, Balloon Pulmonary Angioplasty, Staged Approach, Residual/Recurrent Pulmonary Hypertension

1. Introduction

Chronic thromboembolic pulmonary disease (CTEPD) with PH, also known as CTEPH, is the feared long-term complication after an acute pulmonary embolism (PE) The incidence of CTEPD with PH is estimated at 2% to 3% of all acute PE [1]-[4]. The incidence of chronic CTEPH may be a consequence of one or multiple acute PE cases without complete thrombotic masses' resolution. It leads to the thrombi organization. As a result, this process eventually leads to increasing of PVR, PH and right heart failure (RHF) [5]. Pulmonary thromboendarterectomy (PEA) has become the standard treatment for CTEPH since it was described for the first time in the San-Diego University College publication in late 1980s. Though, PEA is still the definitive surgical approach [6], but it has always been described as a difficult procedure often burdened by a high mortality [7].

Starting from the initial embolectomy till now there has been no doubt that a complete thrombotic masses extraction is vital for the best outcome [8]. The results of CTEPH treatment have considerably been improved using new anesthesiologic, cardiopulmonary bypass (CBP) protocols and surgical technique. At present, the surgical mortality rate has decreased from 24% to 2.2% [6] [9]. PEA is an open surgical procedure performed with deep hypothermic circulatory arrest (DHPA). A surgeon manually removes potentially curative thrombi. This is the cornerstone of treatment [10] [11] and remains the approach of choice for technically operable patients who are good surgical candidates evaluated at expert CTEPH centers. However, it is offered to only a subset of such patients because there are a number of factors that can prevent or refuse surgery (*there are various depending factors wish may refuse surgery*) [12] [13]. Now PEA is the recommended first-line treatment for operable patients [1] [10] [14]. However, despite continued improvement in the surgical and interventional techniques, PEA remains a challenging procedure. This is particularly the case in patients with elevated PVR, as several studies have previously shown that high pre-operative PVR was associated with an increase in early post-operative mortality [15]-[17].

Percutaneous revascularization of pulmonary vascular obstructions with BPA has emerged as a potential treatment option for individuals who are unsuitable candidates for PEA or have residual obstruction after PEA [18]-[20]. Over the past 5 years, BPA has become a guideline-recommended treatment (class IB) for inoperable CTEPH patients or patients with residual PH after PEA [10]. Not all patients with technically operable disease are amenable to PEA. Treating surgically accessible lesions by BPA leads to significantly less improvement of pulmonary hemodynamics and physical capacity compared to interventional treatment of technically inoperable CTEPH. However, the cumulative survival rates at 1, 5 and 10 years after BPA were not significantly different (92.5%, 86.1%, 84.3% vs 96.5%, 92.9%, 90.1%, respectively) [21] [22]. The intraoperative use of BPA in regions that could not be surgically cleared has already been described, but remains a single-centre experience [23]. Another important finding was that medical pre-treatment reduced the rate of severe complications of BPA in patients with a PVR > 4 Wood Units (WU) in a randomized controlled trial [24]. The goal of optimal BPA is the dilation of all treatable lesions. It has been reported that the improvement in patient hemodynamics correlates with the number of treated segments. Therefore, the BPA treatment is completed over several sessions. Generally, 4 to 8 BPA procedures are necessary to complete the BPA treatment for any individual patient [25]. It has been shown that the number of successfully treated lesions predicts treatment response, and especially the number of successfully treated occlusions (particularly chronic total occlusions) have the largest impact on relative change in mPAP [26].

Identification of the morphology of the pulmonary artery (PA) lesions is crucial for the management of patients with CTEPH because whether PEA, BPA, or medical treatment (MT) should be performed depends on the location of the PA lesions: PEA for the main, lobular and proximal segmental branches; BPA for the distal segmental and subsegmental branches; and MT for the more distal subsegmental branches. Moreover, several recently published reports have recommended combination therapy for patients with residual PAH after PEA [27]. Anatomically, two different vascular lesions participate in the increasing of PVR in patients with CTEPH: an obstruction of pulmonary arteries by unresolved thrombi and a microvasculopathy observed in both obstructed and no obstructed lung areas [28]. A microvasculopathy is also observed in completely obstructed lung areas and is attributed to the development of systemic bronchial arteries anastomoses with pulmonary arterioles /or, and venules [29].

As a certain strategy for patients with high-risk for PEA, pre-operative BPA has been used to decrease right ventricular afterload and thus to reduce the perioperative risk: in CTEPH patients with mixed (proximal and distal) anatomical lesions and baseline PVR >10 WU, all three therapeutic modalities were combined in a sequential multimodal treatment strategy [30]. In some published cases BPA has the beneficial effect of improving right heart function following PA pressure reduction and a subsequent increase in cardiac output. Carried out before PEA BPA may reduce complications after PEA in high-surgical-risk patients with CTEPH [31].

Despite significant advances, BPA remains a procedure in evolution, and several surgical decisions makings must be addressed. First of all, is it possible to perform BPA before PEA as invasive pre-treatments method in patients with

CTEPH and severe PH? Do changes in the structure of the vascular wall after BPA affect the results of PEA and outcome? In observed literature we didn't find any recommendation concerning this idea. This study aims to analyze the results and outcomes of PEA in patients with severe PH and high-surgical-risk who underwent endovascular intervention as the first step of treatment.

2. Methods

2.1. Study Design

Being a retrospective study, ethical approval was waited off. Retrospective data of 27 patients after two-stage (BPA-first, PEA-second) invasive and surgical treatment of the CTEPH from January 2019 to March 2024 at the Department of the Heart Diseases with Progressive Pulmonary Hypertension Surgical Treatment in the Bakulev National Medical Research Center for Cardiovascular Surgery under the Russian Federation Health Ministry (Moscow, Russian Federation) were collected and thoroughly analyzed. Written and informed consents from the patients were received. All the patients were examined before and immediately after BPA and in 3 - 4 months after BPA just before PEA, immediately after operations and at the hospital discharge. After the operation the patients were evaluated with clinical assessment, transthoracic echocardiogram (TEE), computed tomography angiogram (CTA), invasive pulmonary artery pressure (PAP) measurement; the data received were collected. The inclusion criteria suggested by the American College of Chest Physicians were the following: 1) NYHA class symptomatology; 2) preoperative PVR > 300 dynes*s*cm^5; 3) surgical accessibility of thrombi in main, lobar, segmental pulmonary arteries as seen in CTA; 4) no debilitating comorbidities. All the patients with acute PE did not fit PEA undergoing. Demographic variables, PAP (systolic (PAPs), mPAP, diastolic pressure (PAPd)) with the subsequent PVR calculation, right ventricle (RV) disfunction measured by tricuspid annular peak systolic excursion (TAPSE) on 2D echo, central venous pressure (CVP), oxygen saturation (SatO2) and any post invasive and postoperative complications were studied before and after all stages of treatment. PEA was performed with cardiopulmonary bypass (CPB), hypothermia, cardioplegia and without DHCA in patients with type I and II thrombotic masses structure according to Jamieson's classification and with DHCA in patients with type III.

2.2. BPA Anesthetic Management

Pre and intraoperative period

Warfarin was discontinued 3 days before the operation and heparin was prescribed. Right heart catheterization (RHC) was performed at the same time. In the operating room, the peripheral vein and radial artery were catheterized. In addition to standard monitoring and central hemodynamics monitoring, bispectrality index and cerebral oximetry were used. Before and during the intervention, patients were insufflated with oxygen (10 l/min). In order to prevent possible complications, an intubation and resuscitation set was always ready. The ventilator was assembled. During the procedure, the activated partial thromboplastin time was maintained within 60 - 80 sec, and the activated clotting time was within 250 - 300 sec. Propofol 1.5 - 2.5 mg/kg/hour or Dexmedetomidine 0.3 - 0.7 mcg/kg/hour infusion were used for sedation. Opioids were generally avoided due to their depressant effect on the respiratory center. Inhaled nitric oxide (NO) was used to reduce PVR. Any increasing of PVR was avoided, hypoxia, hypercapnia, and acidosis were prevented. Norepinephrine (0.03 - 0.07 mcg/kg/min) was used to maintain peripheral vascular resistance and RV perfusion.

Postoperative period

After completion of procedure all patients were admitted to the intensive care unit (ICU) to exclude the development of reperfusion pulmonary edema (PE). Each patient underwent chest X-ray. Each patient was insufflated with oxygen. Stay in the ICU was 1 day but if any complication is present, this time should be more than 1 day. This time was sufficient for hemodynamic improvement, decreasing of the PVR and stabilization of the RV function. Warfarin was resumed and heparin infusion was continued until warfarin efficacy reached the optimal range.

2.3. Balloon Pulmonary Angioplasty Protocol

2.3.1. Vascular Access

Local anesthesia (e.g., 1 - 2% lidocaine) is administered at the puncture site, followed by percutaneous access to the common femoral vein using the Seldinger technique under ultrasound guidance. An introducer sheath (6 - 8 Fr) is placed to secure vascular access.

2.3.2. Catheter Navigation and Angiography

A hydrophilic guidewire is advanced through the introducer sheath, navigating sequentially through the inferior vena cava, right atrium, RV into the PA. Selective pulmonary angiography is performed by injecting iodinated contrast medium through a pigtail or multipurpose catheter to delineate stenotic or occluded segments.

2.3.3. Balloon Dilation

A 0.014 - 0.035" guidewire is navigated across the target lesion and positioned distally for stability. A balloon catheter, sized at 0.8 - 0.9× the reference vessel diameter, is advanced over the wire and inflated with a contrast-saline mixture (1:1 dilution). Inflation pressure is gradually titrated (4 - 12 atm) under fluoroscopic guidance to avoid complications such as rupture or dissection. Repeat angiography confirms procedural success, residual stenosis (>30%) may necessitate upsizing the balloon or staged interventions.

2.3.4. Post-Procedural Management

The balloon catheter, guidewire, and introducer sheath are removed. Hemostasis is achieved using manual compression or a closure device (e.g., Angio-Seal™), followed by application of a pressure bandage. The patient is advised to maintain bed rest for 6 - 12 hours with close monitoring for hematoma or bleeding. ICU monitoring continues for 24 hours, with surveillance of vital signs, oxygen saturation, and signs of reperfusion injury (e.g., hemoptysis, hypoxia). Anticoagulation (e.g., heparin bridging) is resumed post-procedure as per institutional protocols to ensure patient safety and optimize outcomes.

2.4. Surgical Technic

Methods and peculiarities of CPB, anesthetic management and surgical technic were described previously [32]. During surgery, repeated measurements of pulmonary and systemic hemodynamics were performed using a Swan-Ganz catheter. Cardiac output was measured using the thermodilution method. The type of pulmonary artery lesion was clarified according to Jamieson's classification. After surgery, patients were transferred to the ICU for stabilization.

2.5. Statistical Analysis

For statistical analysis of the results, IBM SPSS Statistics version 26 was used. Quantitative variables following a normal distribution were presented as the mean ± standard deviation; quantitative variables with a non-normal distribution were presented as the median and interquartile range (25th-75th percentiles). To assess the normality of the distribution, the Kolmogorov-Smirnov and Shapiro-Wilk tests were used. Qualitative variables were presented as absolute values and percentages. To compare groups of quantitative variables with a normal distribution, Student's t-test was used. Groups of quantitative variables with a non-normal distribution or with different types of distribution were compared using the Wilcoxon and Mann-Whitney tests. Qualitative variables were analyzed using Pearson's Chi-square test and Fisher's exact test. Differences were considered statistically significant at $p < 0.05$. Levene's test was used to check for homogeneity of variances. If differences between groups were found ($p < 0.05$), pairwise comparisons between these groups were conducted. For variables with a normal distribution, Tukey's test was used for pairwise comparison, while for variables with a non-normal distribution, the Mann–Whitney test was applied. In the case of pairwise comparisons, the Bonferroni correction was used to determine the new significance threshold.

3. Results

The present data analysis included 27 patients who underwent surgery. The median age at diagnosis was 47 (IQR 15 - 78) years, 51.9 % males and 48.1 % females. Due to NYHA, 1 (3.7%) patient preoperatively belonged to class II, 19 (70.4%) to class III and 7 (25.9%) to class IV. Location and extent of thrombi were evaluated by CTA. The baseline demographic and preoperative clinical characteristics of the study population are presented in **Table 1**.

Table 1. Baseline demographic and preoperative clinical characteristics.

Parameters	Values
Gender, n (%)	
Male	14 (51.9)
Female	13 (48.1)
Age, years	47.4 ± 8.0
Weight, kg	80.3 ± 13.7
BSA, m^2	1.91 ± 0.57
BMI, kg/m^2	24.8 ± 1.9
Symptoms, n (%)	
DOE	27 (100)
Palpitation	23 (85.1)
Pedal edema	19(70.3)
Cough	9 (33.3)
Syncope	1 (3.7)
NYHA FC, n (%)	
I	Nill
II	1 (3.7)
III	19 (70.4)
IV	7 (25.9%)
Causes of CTEPH, n (%)	
DVT history	21 (77.8)
Thrombophilia	3 (11.1)
Antiphospholipid syndrome	2 (7.4)
Unknown	1 (3.7)
Thrombus type by Jamieson's classification, n (%)	
I	4 (14.8)
II	16 (59.2)
III	7 (25.9)
IV	Nill

BSA—Body Surface Area. BMI—Body Mass Index. DOE—Dyspnea on Exertion. FC—Functional class. DVT—Deep Venous Thrombosis.

Reason for two-stage surgical approach was based on the high surgical risk associated with PH in all patients. Most patients were treated with PAH targeted therapy: sGC stimulators (74%) PDE5 inhibitors (22%), and endothelin receptor antagonists (4%). The median number of sessions and the median number of treated lesions per patient were 3 (IQR 1 - 5) and 4 (IQR 2 - 8), respectively, to obtain PVR decreasing less than 1000 dynes⋅s⋅cm^{-5} (**Figure 1**). Thoracic complications occurred in 8 patients (29.6%) after BPA procedures with mild complications in 22.2% and moderate complications in 3.7%. The more frequent complication was lung injury defined as reperfusion edema. It was occurred in 5 patients (18.5%). Severe complication (lung injury with mild hemoptysis) requiring intensive care unit treatment occurred in 1 patient (3.7%). No one patient had non-thoracic complications and no one died after BPA. The immediate results showed a significant reduction in mPAP and PVR (**Table 2**) directly after BPA and after a median follow-up of 1 (IQR 1 - 3) month after the last BPA session, but the difference was not significant ($P > 0.05$).

Table 2. Clinical and hemodynamic parameters before and after BPA.

Parameters	Before	After	P value
RAD (cm^2)	34.0 ± 5.0	32.0 ± 6.0	0.48
RVD (cm^2)	36.0 ± 4.0	28.0 ± 6.0	0.0001
RV EDV (mL)	153.4 ± 37.2	147.1 ± 30.7	0.0001
RV EF (%)	40.0 ± 6.3	45.0 ± 4.5	0.05
TAPSE (mm)	14.1±1.7	16.3 ± 2.2	0.05
6MWT (m)	251 ± 43	331 ± 52	0.0001
NT-pro BNP (pg/mL)	312.7 ± 388.3	244.2 ± 316.2	0.0001
Sat O_2 (%)	87.2 ± 1.4	88.7 ± 1.5	0.29
PAPs (mm Hg)	98.6 ± 12.7	90.8 ± 11.4	0.0001
mPAP (mm Hg)	66.2 ± 9.5	53.7 ± 9.5	0.0001
PVR (dynes⋅s⋅cm^{-5})	1276 ± 448	772 ± 341	0.0000001
RAP (mm Hg)	16.2 ± 2.6	12.8± 2.1	0.0001
CO (l/min/ m^2)	1.82 ± 0.4	2.6 ± 0.6	0.05

RAD—right atrial dimension; RVD—right ventricular dimension; RV EDV—right ventricular end-diastolic volume; RV EF—right ventricular ejection fraction; 6MWT—6minute-walking test; NT-pro BNP—terminal part of the brain natriuretic peptide. RAP—right atrial pressure.

Median delay from the last BPA session to PEA was 2 (IQR 2 - 5) months. PEA was only performed on the operable side. The median duration of circulatory arrest and CBP was 18 (IQR 16 - 20), 172 (IQR 161 - 202) min (with DHCA) and 78 (IQR 47 - 102) mm (without DHCA), respectively. All patient operated on without DHCA had type I or II disease according to the new University of California, San Diego Chronic Thromboembolic Disease Surgical Classification [6] [15]. The results of PEA are presented in **Table 3**.

Table 3. Clinical and hemodynamic parameters before and after PEA.

Parameters	Before	After	P value
RAD (cm^2)	33.0 ± 5.0	24.0 ± 2.0	0.0001
RVD (cm^2)	32.0 ± 3.0	25.0 ± 3.0	0.0000001
TV incompetence (+)	3.0 [2.0; 3.0]	1.0 [1.0; 1.0]	0.0000001
RV EDV (mL)	149.2 ± 44.1	112.1 ± 30.1	0.0000001
RV EF (%)	43.0 ± 7.5	50.0 ± 4.5	0.0001
TAPSE (mm)	16.1 ± 1.4	19.3 ± 3.7	0.0001
6MWT (m)	311 ± 42	---	---
NT-pro BNP (pg/mL)	244.2 ± 316.2	89.5 ± 99.4	0.0000001
Sat O_2 (%)	89.1±1.8	96.6 ± 1.7	0.0001
PAPs (mm Hg)	87.8 ± 22.9	40.8 ± 13.5	0.0000001
mPAP (mm Hg)	51.3 ± 5.5	27.6 ± 4.5	0.0001
PVR (dynes·s·cm^{-5})	776 ± 232	388 ± 141	0.0000001
RAP (mm Hg)	12.1 ± 1.8	8.4 ± 1.6	0.0001
CO (l/min/m^2)	2.5 ± 0.6	3.3 ± 0.7	0.0001

TV—Tricuspid valve.

Table 4. Complications in early postoperative period.

Complications	BPA n (%)	PEA n (%)
Lung injury (reperfusion pulmonary edema)	5 (18.5)	8 (29.6)
Vessel injury (hemoptysis)	1 (3.7)	1 (3.7)
Right heart failure	1 (3.7)	6 (22.2)
Residual pulmonary hypertension	---	1 (3.7)
Pneumonia	1 (3.7)	1 (3.7)
Cardiac tamponade	0 (0)	1 (3.7)
Arrhythmia	0 (0)	2 (7.4))
Renal disfunction	1 (3.7)	1 (3.7)
Neurological	0 (0)	0 (0)
ECMO	0 (0)	1 (3.7)
Mortality	0 (0)	1 (3.7)

In 16 (59.3%) patients the postoperative period was uncomplicated. The mean duration of mechanical ventilation was 14 hours [8.0; 20.25] (IQR 6 - 362). The mean ICU length of stay was 1 [1.0; 2.0] days (IQR 1 - 28). Complications after PEA were more often than after BPA and occurred in 11 patients (40.7%) with mild complications in 8 (29.6%), moderate complications in 2 (7.4%) and severe in 1 (3.7%) (Table 4). The more frequent complication was reperfusion pulmonary edema occurred in 8 patients (29.6%) but was severe only in 1 of them, moderate right heart failure in 4 (14.7%) and severe in 2 (7.4%). Residual PH was observed in 1 patient (3.7%). This patient died. The reason of death was the right ventricular dysfunction due to PH. The initial PVR was 1400 dynes·s·cm^{-5} and despite of decreasing after BPA treatment (870 dynes·s·cm^{-5}) the residual PVR was 530 dynes·s·cm^{-5}. The patient was placed on VA-ECMO without any beneficial effect. His long-history of CTEPH was more than 5 years. We hypothesized that this time could be the reason for the development of microvasculopathy with distal changes of pulmonary arteries. Most patients required one inotropic agent, and 14 (51.9%) patients required one vasodilator (NO insufflation) to reduce mPAP and PVR and improve the RV function. No one patient had neurological complications.

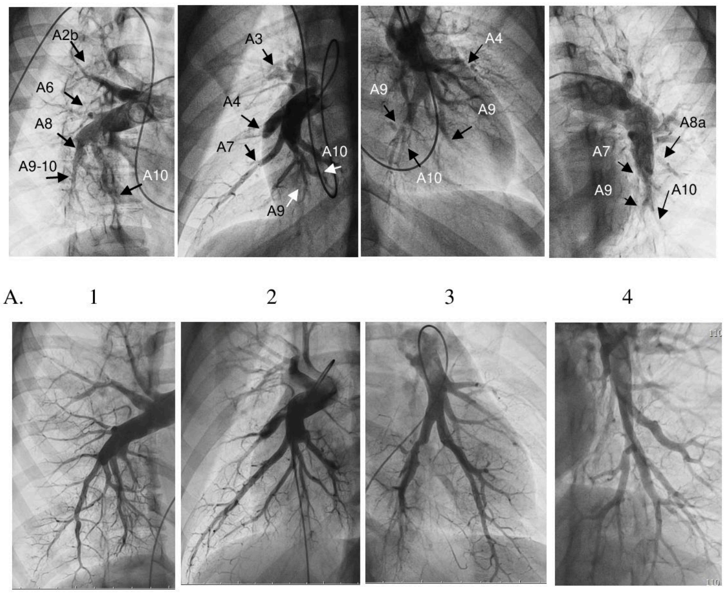
After a median follow-up of 27 (IQR 11 - 46) months post-PEA, PVR had decreased from 1276 (IQR 980 - 1464) to 299 (IQR, 104 - 326) dynes·s·cm^{-5} (p = 0.0001) (Table 5**).** One patient died 3 years after the surgery. The cause of death was coronary artery disease.

Table 5. Long-term clinical and hemodynamic parameters after BPA&PEA.

Parameters	Before BPA&PEA	Long-term > 1 year	P value
RAD (cm^2)	34.0 ± 5.0	22.0 ± 4.0	0.0001
RVD (cm^2)	36.0 ± 4.0	24.0 ± 6.0	0.0001
RV EDV (mL)	153.4 ± 37.2	108.4 ± 14.1	0.0000001
RV EF (%)	40.0 ± 6.3	53.0 ± 4.0	0.0001
TAPSE (mm)	14.1 ± 1.7	19.6 ± 1.4	0.0001
6MWT (m)	251 ± 43	389 ± 62	0.0001
NT-pro BNP (pg/mL)	312.7 ± 388.3	30.6 ± 58.3	0.0000001
Sat O_2 (%)	87.2 ± 1.4	98.4 ± 1.4	0.0001
NYHA FC, n (%)			
I		12 (44.4)	
II		11 (40.7)	
III		3 (11.1)	
IV		Nill	
PAPs (mm Hg)	98.6 ± 12.7	38.7 ± 12.5	0.0000001
mPAP (mm Hg)	66.2 ± 9.5	26.0 ± 6.0	0.0000001
PVR (dynes·s·cm^{-5})	1276 ± 448	299 ± 133	0.0001
RAP (mm Hg)	16.2 ± 2.6	6.3 ± 1.3	0.0000001
CO (l/min/ m^2)	1.82 ± 0.4	3.6 ± 0.7	0.0001

4. Discussion

CTEPH is a long-term chronic disease caused by organized thrombi obstructing the PA. In patients with mPAP exceeding 50 mmHg, the 5-year survival is reported to be as low as 10% [33]. PEA has been one of the effective treatment procedures, and long-term results are well-established by several authors, including the San Diego group [15]. BPA is recommended as a solution for patients with residual PH [34]. However, only a few reports discuss the long-term outcomes of postoperative BPA for eligible patients and their overall survival. In addition, there are only a few reports about possibility and effectiveness of BPA as the first step before endarterectomy, particularly in patients with severe PH [35]. BPA can improve the severity of PH with a significantly reduced incidence of serious complications [36] [37].



B.

Figure 1. Balloon pulmonary angioplasty of a 20-year-old patient.

The first decision remains assessment of operability, and the best improvement in symptoms and survival is achieved by the mechanical therapies, PEA and BPA. With the advances in multimodal therapies, excellent outcomes can be achieved with 3-year survival of >90% [14]. A microvasculopathy is observed in completely or partially obstructed lung areas and is attributed to the development of systemic bronchial arteries anastomoses with pulmonary arterioles and venules [29]. The presence of microvasculopathy is suspected when mechanical obstruction does not correlate with the haemodynamic severity [38]. Based on this, it can be assumed that BPA performing as the first step before PEA can provide significant information about the presence of this complication. When the PAP decreases after BPA, the likelihood of microvasculopathy is significantly lower. Our experience is based on the results obtained after a series of endovascular interventions and successfully performed PEA with normalization of pulmonary hemodynamics in patients with baseline PVR exceeding 1000 dynes∗s∗cm^{-5}. The case presented in our paper is a good example that makes it possible to exclude the presence of microvasculopathy as a cause of high PH and successfully perform operation. **Figure 1** demonstrates an example of balloon pulmonary angioplasty of a 20-year-old patient with thrombophilia and CTEPH. Four sessions were performed with dilatation of 18 lesions over a period of 5 months with a median interval of 28 days (IQR 14 - 48 days). After last session mPAP and PVR had significantly decreased from 62 mmHg to 45 mmHg and from 1944 dynes⋆s⋆cm^{-5} to 600 dynes⋆s⋆cm^{-5}, respectively. Three months after the last session the patient was successfully operated on with a decreasing of mPAP and PVR to 28 mmHg and 270 dynes⋆s⋆cm^{-5}, respectively with good outcome and without complications.

Possibility of the vascular wall` damage during endovascular intervention remains a difficult question. Does it influence on the results of subsequent PEA? Shimahara Y. did not find such a danger in his recently published article. No case of technical difficulty caused by a destroyed PA plane after receiving BPA as a first treatment was observed. Endovascular exposure did not cause additional problems when performing open surgery [39]. A number of other authors are also inclined to this opinion [30] [31]. Conversely, the efficacy of performing BPA first, followed by PEA, is unknown, with only a few reports with small numbers of patients being published [40] [41]. In contrast to BPA in inoperable patients, which is done to relieve PAH and other symptoms, the treatment goal of performing BPA first, followed by PEA, is to reduce the operative risk.

This situation may be attributed to the fact that the plane of the PEA dissection may deteriorate or become distorted during BPA, resulting in an impossible-to-correct dissection and in vascular injuries during PEA. However, our present study did not identify any patient in whom adequate plane dissection was difficult or patients with vascular injuries, although PEA was performed for most lesions that were first treated with BPA. Two recent case reports of patients with high PVR (985 and 1569 dynes·s·cm^{5}, respectively) demonstrated reduced preoperative PVR when BPA preceded PEA, and PEA after BPA resulted in successful outcomes [40] [42]. Conversely, performing BPA for 1 lung (including surgically inaccessible lesions) followed by PEA for the other lung (including proximal lesions) is an alternative strategy. Mercier O. with colleagues [41] reported the outcomes of this strategy in 13 of 418 patients who underwent PEA for CTEPH. Compared with the use of PEA alone, performing BPA first followed by PEA resulted in a lower 90-day death rate, shorter CPB time and a similar percentage decrease in mPAP after PEA. Mechanical vascular injuries, including wire perforation, balloon over-dilatation and high-pressure injection of contrast media, are considered to be the major causes of BPA-related complications [43] [44]. Complications related to performing BPA first, followed by PEA, may aggravate a patient's condition and further complicate the operation. Although 8 patients in our present study experienced minor complications after BPA, no adverse effects of these complications on the operation were observed. However, some patients undergoing BPA may experience major complications, including death or the need for intubation or ECMO [45].

Madani M.M. *et al.* [15] reported that a preoperative PVR of > 1000 dynes⁎s⁎cm^{-5} was associated with a 3 - 4 times higher risk of postoperative death, Saouti N. *et al.* [46] reported preoperative PVR of 645 dynes⁎s⁎cm^{-5} and high mPAP of >46 mmHg as the cut-off values for distinguishing between high- and low-risk cases for postoperative deaths. In addition, residual PAH, postoperative PVR of > 425 - 500 dynes⁎s⁎cm^{-5} or mPAP of > 34 - 38 mmHg, was reported to be related with postoperative deaths [7] [47]. Based on these outcomes, severe PAH with a PVR of > 1000 dynes⁎s⁎cm^{-5} or an mPAP of > 45 mmHg was defined as the first criterion to identify patients at high surgical risk in the present study. After PEA 11 patients experienced complications. In no case did the previous endovascular intervention cause complications. The residual PH as fatality complication and the reason of death was in one case. The initial PVR was 1400 dynes⁎s⁎cm^{-5} and the residual PVR was 530 dynes⁎s⁎cm^{-5}. In patients with PVR more than 1000 dyn·s·cm^{-5} and a clearly established time of medical history more than 3 years, PEA may be effective in reducing of right heart failure and PH, however, the early postoperative period is associated with a higher frequency of complications and a high surgical mortality [48].

5. Conclusion

The efficacy of isolated BPA without PEA for CTEPH has been reported, but unfortunately, the potential of BPA for symptomatic patients has not been fully investigated. Maintaining stable preoperative hemodynamics with lower mPAP and PVR should be more comfortable for surgeons who perform PEA. The results of this study suggested that a CTEPH team can stabilize the hemodynamics of patients with CTEPH by performing BPA before PEA. A staged approach may be an effective and safe surgical tactic in patients with CTEPH and severe PH. However, it is necessary to identify criteria for patient selection, and procedural efficacy.

6. Limitations

The major limitations of the study are a retrospective analysis and a lack of long-term follow-up. However, we believe that this study may provide some useful information for the surgical management of CTEPH patients.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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