Kardiologia Polska 2017; 75, 7: 645–654; DOI: 10.5603/KP.a2017.0091 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Changing the strategy of balloon pulmonary angioplasty resulted in a reduced complication rate in patients with chronic thromboembolic pulmonary hypertension. A single-centre European experience

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Abstract

Background and aim: To assess the safety and efficacy of a refined balloon pulmonary angioplasty (BPA) strategy in patients with chronic thromboembolic pulmonary hypertension (CTEPH).

Methods: There were 157 BPA sessions performed in 56 CTEPH patients (47 non-operable, nine after pulmonary endarterectomy; aged 58.6 ± 17.9 years; 28 females) with severely impaired pulmonary haemodynamics (mean pulmonary artery pressure [mPAP]: 51.3 ± 12.2 mm Hg, pulmonary vascular resistance [PVR]: 10.1 ± 3.9 Wood Units). The first 50 sessions aimed to recanalise chronic occlusions and prevent reocclusion with aggressive anticoagulation. The next 107 sessions aimed to relieve "web" and "ring" lesions using reduced tip load guidewires and less intensive anticoagulation.

Results: There was significant reduction in haemoptysis (22% vs. 7%, p = 0.01), vessel injury (30% vs. 13%, p = 0.01), and reperfusion pulmonary injuries (22% vs. 4%, p = 0.01) after changing the BPA strategy. Mortality at 14 days was also reduced (6% vs. 0%; p = 0.05). The cumulative survival rate was 94.6% at 24 months after the first BPA, which was more favourable than medically treated historic controls. In the 31 patients with > 3 BPA sessions, there was significant reduction of PVR (10.3 ± 3.7 vs. 5.9 ± 2.8 Wood Units; p = 0.01), mPAP (50.7 ± 10.8 vs. 35.6 ± 9.3 mm Hg; p = 0.01) and improvement in World Health Organisation functional class (3.19 ± 0.48 vs. 1.97 ± 0.80; p < 0.001).

Conclusions: Balloon pulmonary angioplasty improves haemodynamics and outcome but requires refined strategy to limit early complication rate.

Key words: balloon pulmonary angioplasty, chronic thromboembolic pulmonary hypertension, complications, survival, haemoptysis

Kardiol Pol 2017; 75, 7: 645–654

INTRODUCTION

Until recently, surgical pulmonary endarterectomy (PEA) performed under deep hypothermia with periods of total cardiac arrest was the only effective treatment for chronic thromboembolic pulmonary hypertension (CTEPH) [1]. Despite the increasing expertise of dedicated surgical teams, a significant proportion of patients with CTEPH do not qualify for PEA [2–4]. This is mostly because the distal position of

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Received: 22.03.2017 **Accepted:** 27.04.2017 **Available as AoP:** 10.05.2017

post-thromboembolic lesions is not amenable for surgical endarterectomy. Balloon pulmonary angioplasty (BPA) emerged as a potential therapeutic option for non-operable patients as well as for those with pulmonary hypertension persisting after PEA. The development of BPA was far from rapid. It took 13 years from the first case report to the publication of the first series of 18 patients treated with BPA in the United States [5], and another 13 years until the results of the first European series of 20 patients was reported from Norway [6]. Both reports mentioned specific complications related to this procedure. Meanwhile, several Japanese centres gained their own experience in BPA using slightly different methodologies and claimed reduced complication rates while maintaining high efficacy [7–14]. We started to perform BPA in non-operable CTEPH patients in 2013 [15, 16], motivated by their expected poor survival compared with those offered surgical PEA [2]. Initially, to obtain prompt haemodynamic gain from BPA, we aimed at near-normalisation of the diameter of dilated arteries and approached totally occluded vessels [17]. While rewarding in terms of efficacy, this strategy resulted in some serious complications. Here, we report our results collected before and after modifying the BPA procedure. We also compare the survival related to BPA, as assessed from the very first intervention, with the survival previously reported by our team for historical patients undergoing PEA or remaining on medical therapy alone [2].

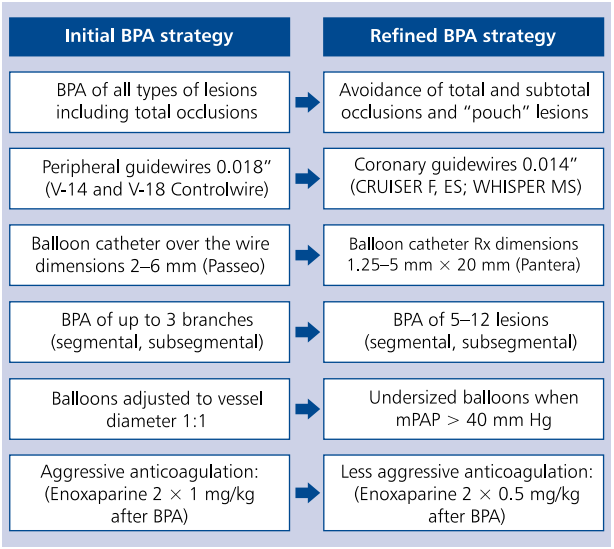

Figure 1. Initial and refined balloon pulmonary angioplasty (BPA) strategies in patients with chronic thromboembolic pulmonary hypertension recruited to the BPA programme; mPAP — mean pulmonary artery pressure

METHODS

Study population

All patients included in the study had a diagnosis of CTEPH. The diagnosis was based on typical post-embolic changes in pulmonary arteries shown on diagnostic imaging (computed tomography angiography, perfusion lung scan, pulmonary angiography) and signs of precapillary pulmonary hypertension identified during right heart catheterisation that persisted despite at least three months of antithrombotic therapy [18]. The treatment was chosen by a multidisciplinary CTEPH-team consisting of a cardiac surgeon with experience in PEA [AB], an interventional cardiologist with experience in BPA [MK], and a cardiologist experienced in using specific treatments [AT] for pulmonary arterial hypertension. Selection of BPA treatment required the consensus of the three specialists.

General BPA strategy

All patients undergoing BPA received standardised written information regarding the nature of the procedure and possible complications, and signed an informed consent form. The Bioethics Committee at the Medical Centre for Postgraduate Education approved the study protocol. The study complies with the Declaration of Helsinki and was registered in the ClinicalTrials.gov database with number NCT0296439. The BPA procedures were performed by two interventional cardiologists [MK, AP] and an interventional radiologist [RP] experienced in right heart catheterisation and coronary and peripheral interventions. The BPA procedure was normally carried out using right femoral vein access. Chronic oral antithrombotic treatment with was discontinued at least 24 h before BPA and unfractionated heparin was administered by IV at a dose of 2000 U/h during the procedure. Patients treated with oxygen chronically received it at the same flow rate. A 6 F coronary guiding catheter (Launcher, Meditronic) was inserted into the right or left pulmonary artery using a 90-cm 6 F vascular sheath (Flexor, Cook). The balloon width and length was adjusted to the type of lesion and degree of stenosis of the pulmonary artery, based on angiography. After inflation, the angiographic effect of the procedure was estimated by the analysis of flow across the treated lesion, peripheral parenchymal perfusion, and venous return from the revascularised segment. In special cases when there were doubts about the nature or significance of a lesion, additional diagnostic tools were used, such as intravascular ultrasound or evaluation of the pressure gradient across the evaluated lesion using a pressure wire. After completion of the procedure, each patient was transferred to the intensive cardiac care unit to monitor vital functions and potential complications for at least 48 h.

After the first 50 procedures of BPA, the safety data were analysed and the treatment strategy was changed in an attempt to reduce the complication rate. The details of the changes in the BPA strategy are depicted in Figure 1.

Initial BPA strategy

The initial phase of the programme (50 BPA procedures) used peripheral guidewires (V-14 and V-18 Controlwire, Boston Scientific) and over the wire balloon catheters with dimensions

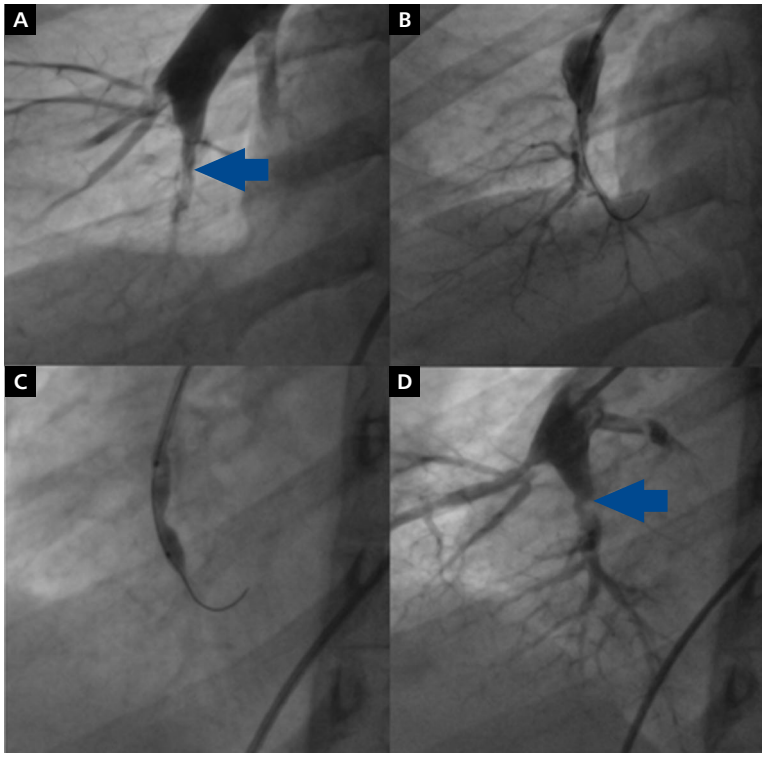

Figure 2. Initial balloon pulmonary angioplasty strategy. Selective angiography presents the subtotal occlusion (blue arrow) in pulmonary artery of right lower lobe (**A**). Panel **B** presented wire penetration across the narrowed vessel. The lesion shaped balloon catheter during dilatation (**C**). Panel **D** presents the final result of angioplasty with post-stenotic lesion (blue arrow).

of 2–6 mm (Passeo, Biotronik). In this phase, interventions were attempted on all types of lesions, including webs, bands, and rings as well as subtotal and total occlusions (Fig. 2). Initially, total elimination of stenosis or occlusion was attempted, regardless of the level of pressure in the pulmonary artery or the type of lesion. During one session, BPA was performed in up to three segmental or subsegmental pulmonary arteries. Early anticoagulation was used to prevent thrombotic reocclusion of dilated vessels and consisted of a full dose of low molecular weight heparin (LMWH) in the first 6–12 h after BPA.

Refined BPA strategy

The peripheral guidewires used in the first phase of the BPA programme were replaced with 0.014-inch coronary guidewires (Cruiser, Biotronik; Whisper MS, Abbott Vascular) with a lower tip load for procedures 51–157. The semi-compliant monorail balloon catheters were sized mainly between 1.25 mm and 5 mm (Pantera, Biotronik). The selection of lesions for BPA interventions was changed in the refined strategy. Total occlusions and pouch lesions were avoided, especially during the first session, while webs, bands, and rings were preferred (Fig. 3). If the mean pulmonary artery pressure (mPAP) was ≥ 40 mm Hg, balloon catheters > 2.5 mm in diameter were avoided. In contrast, the number of segmental and subsegmental arteries treated during one procedure increased to 12. Postoperative anticoagulation was less aggressive — a half of dose of LMWH within 48 h after BPA.

Assessment of complications

A prospective analysis of periprocedural complications was carried out during the BPA according to the following criteria.

Vessel injury was defined as extravasation of contrast outside the lumen, "retention" of the contrast agent within the vessel wall, or prolonged persistence of the contrast agent in the interstitial area vascularised by the injured artery.

Mild haemoptysis was defined as a total haemoptysis volume of < 50 mL and **severe haemoptysis** as > 50 mL over 24 h.

A **reperfusion lung injury** was based on Inami classification [10]. A grade 3 was defined as moderate reperfusion pulmonary oedema that needed an elevated concentration of oxygen administered via an oxygen mask to maintain arterial saturation at the optimum level. Grade 4 was defined as moderate to severe reperfusion pulmonary oedema needing non-invasive positive pressure ventilation with high-concentration oxygen inhalation. Grade 5 was defined as extremely severe reperfusion pulmonary oedema needing mechanical ventilation.

Long-term survival data were obtained from follow-up phone calls made every three months to all patients who completed BPA treatment.

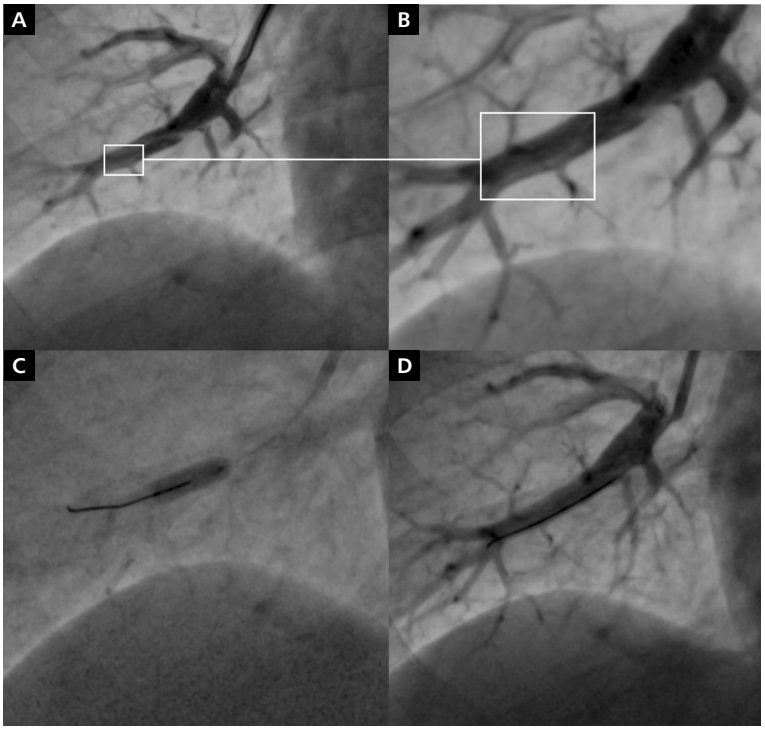

Figure 3. Refined balloon pulmonary angioplasty (BPA) strategy. Selective angiography presents the web lesion in one of the segmental arteries of the right lower lobe (**A**, **B**). Panel **C** presents balloon catheter inflation. The result of BPA presented on control angiography (**D**)

Efficacy measures

Thirty-one patients who either completed BPA treatment or had at least three BPA sessions were eligible for assessment of the efficacy of intravascular therapy. All patients were submitted to the same panel of non-invasive and invasive examinations prior to and 3–6 months after the last BPA session. A Swan-Ganz catheter was used to measure right atrial pressure (RAP), pulmonary artery pressure (PAP), and pulmonary capillary wedge pressure (PCWP). Cardiac output (CO) was measured using a thermodilution technique. The pulmonary vascular resistance (PVR) and cardiac index (CI) were calculated according to the accepted standards [19]. Functional class was assessed using the Word Heath Organisation (WHO) classification. The six-minute walk test (6MWT) was used to assess exercise capacity. Biomarker assessment included the plasma concentrations of troponin (hsTnT), measured with a highly sensitive test (Roche Diagnostics GmbH, Germany), and N-terminal pro B-type natriuretic peptide (NT-proBNP) (Roche Diagnostics GmbH, Germany).

Statistical analysis

Categorical variables were presented as counts and percentages, and continuous variables as means and standard deviations or medians and interquartile range. Depending on the distribution, Student's t-test or Wilcoxon signed rank test was used for statistical analysis of data obtained prior to and following BPA treatment for continuous variables. The χ^2 test with Yates correction (when applicable) was used for comparison of categorical variables. The proportion of patients surviving was estimated by the Kaplan-Meier method. The survival of BPA patients was compared with historical controls by the log-rank test. All statistical analyses were performed using STATISTICA 10 software. A p-value < 0.05 was considered statistically significant.

RESULTS

Patients

We enrolled 56 consecutive patients, and their baseline characteristics are shown in Table 1. The mean age was 58.6 ± 17.9 years, and 28 patients were female (50%). They presented symptoms of WHO functional class II or greater (II/III/IV — 5%/73%/22%). The median interval between CTEPH symptom onset to first BPA was 10.2 months. Mean values of mPAP, CI, RAP, and PVR were: 51.3 ± 12.2 mm Hg, 2.37 ± 0.58 L/min × m^2, 10.7 ± 4.5 mm Hg, and 10.1 ± 3.9 Wood Units, respectively. The mean distance for the 6MWT was 304 ± 164 m, and the median NT-proBNP and hsTNT plasma concentrations were 1656 pg/mL and 0.014 μg/L, respectively. The majority (80%) of patients received specific targeted medical therapy before they started BPA treatment. Sixty-six per cent were on sildenafil and 29% on riociguat monotherapy. Two patients received combined sildenafil and treprostinil therapy.

Table 1. Baseline characteristics of the study population

Variables	BPA population
Patients	56
Females	28 (50%)
Age [years]	58.6 ± 17.9
WHO FC:	
I	0 (0%)
II	3 (5%)
III	41 (73%)
IV	12 (22%)
BMI [kg/m^2]	26.9 ± 5.6
Previous PEA	9 (16%)
Administration of specific pulmonary vasodilators	45 (80%)
Monotherapy:	
PDE5i	30 (54%)
ERA	0 (0%)
sGCs	13 (23%)
PGE2a	0 (0%)
Combined therapy:	
PDE5i + PGE2a	2 (3%)
mRAP [mm Hg]	10.7 ± 4.5
mPAP [mm Hg]	51.3 ± 12.2
PCWP [mm Hg]	11.0 ± 2.2
CO [L/min]	4.32 ± 1.08
CI [L/min×m^2]	2.37 ± 0.58
PVR [Wood Units]	10.1 ± 3.9
6MWT [m]	304 ± 164
Median [IQR]	327 [218–415]
NT-proBNP [pg/mL]	3746 ± 7160
Median [IQR]	1656 [589–3531]
hsTnT [μg/L]	0.020 ± 0.022
Median [IQR]	0.014 [0.007–0.026]

BPA — balloon pulmonary angioplasty; BMI — body mass index; WHO FC — World Health Organisation functional class; PEA — pulmonary endarterectomy; PDE5i — phosphodiesterase type 5 inhibitors; ERA — endothelin receptor antagonists; sGCs — synthetic guanylate cyclase stimulator; PGE2a — prostaglandin E2 analogue; mRAP — mean right atrial pressure; mPAP — mean pulmonary artery pressure; PCWP — pulmonary capillary wedge pressure; CO — cardiac output; CI — cardiac index; PVR — pulmonary vascular resistance; NT-proBNP — N-terminal prohormone of B-type natriuretic peptide; hsTnT — high-sensitivity troponin T; 6MWT — six-minute walking test; IQR — interquartile range

Safety results

The first 50 BPA procedures (group A) were performed using the initial strategy, while the next 107 procedures were performed using the refined strategy (group B). Three patients submitted to strategy A died during the periprocedural period (mortality/session: 6%), two died within 24 h of BPA due to complications of vessel injury, and one died on the seventh day after BPA. The autopsy of this last patient revealed haemorrhagic necrosis in the lobe treated with BPA as the cause of death (Fig. 4).

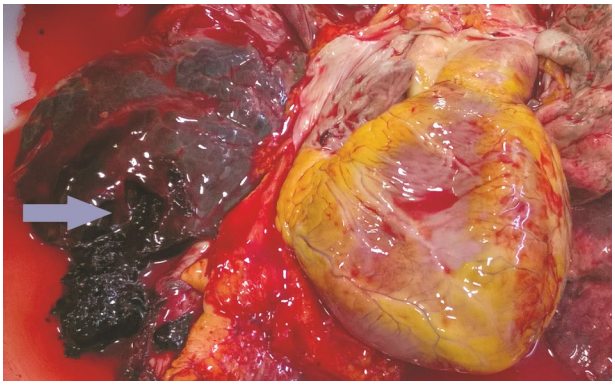

Figure 4. The autopsy examination of the patient who died on the seventh day after balloon pulmonary angioplasty. The haemorrhagic necrosis and rupture (blue arrow) in the treated segments of the right lower lobe was the cause of death

No fatal complications were observed in group B. The frequencies of vessel injuries (13% vs. 30%; p = 0.01), cough (14% vs. 28%; p = 0.02), mild haemoptysis (7% vs. 22%; p = 0.01), and reperfusion pulmonary injuries (11% vs. 4%; p = 0.01) were all lower in group B vs. group A. Severe haemoptysis was infrequent (4%) but remained at the same level regardless of the applied strategy. The type and incidence of complications are presented in Table 2.

Efficacy results

There were 117 BPA sessions performed (average 3.8 sessions/patient) in the subgroup of 31 patients who completed BPA treatment or underwent at least three procedures. Overall, 607 pulmonary segmental or subsegmental arteries underwent angioplasty (19.5/patient). Ninety-five per cent of patients presented symptoms of III and IV functional class before BPA, which reduced to 29% after treatment (p = 0.005). There was significant reduction of mRAP (p < 0.001), mPAP (p < 0.001), and PVR (p = 0.001) and improvement of CI (p = 0.031). In addition, there was significant improvement in the mean value of WHO functional class (p < 0.001), the distance covered in the 6MWT (p < 0.001), and the NT-proBNP concentration (p = 0.001). Before BPA 16 (52%) patients presented elevated hsTnT, and after treatment the number of patients with abnormal hsTnT decreased to 11 (35%; p < 0.001). The final values are compared with baseline in Table 3.

Survival analysis

Three of the 56 (5.4%) patients died, and all survivors were followed for a median of 12.5 months after the first BPA pro-

Table 2. Complications in 157 consecutive balloon pulmonary angioplasty (BPA) procedures using the initial (Group A) and refined (Group B) strategies

Complications	Group A (n = 50); 1–50 BPA	Group B (n = 107); 51–157 BPA	p (test χ^2)
Cough	14 (28%)	15 (14%)	0.02
Haemoptysis	13 (26%)	11 (10%)	0.01
Mild haemoptysis (< 50 mL/24 h)	11 (22%)	7 (6%)	0.01
Severe haemoptysis (> 50 mL/24 h)	2 (4%)	4 (4%)	0.93
Vessel injury	15 (30%)	14 (13%)	0.01
Reperfusion lung injury grade 3,4,5 (Inami class.)	11 (22%)	4 (4%)	0.01
Death related to BPA	3 (6%)	0 (0%)	0.05

Table 3. Efficacy of balloon pulmonary angioplasty (BPA) in 31 patients who completed their BPA treatment or underwent at least 3 sessions

	Population with completed BPA treatment (n = 31)			
	Baseline	Follow-up	Mean % of change	p
Haemodynamic parameters				
Heart rate [bpm]	76.5 ± 18.2	68.4 ± 14.7	–11%	< 0.001
mRAP [mm Hg]	10.4 ± 3.8	6.3 ± 3.5	–39%	< 0.001
mPAP [mm Hg]	50.7 ± 10.8	35.6 ± 9.3	–30%	< 0.001
PCWP [mm Hg]	10.6 ± 2.1	10.5 ± 3.3	–	0.797
CO [L/min]	4.14 ± 1.00	4.51 ± 0.83	+9%	0.044
CI [L/min×m^2]	2.28 ± 0.57	2.51 ± 0.51	+10%	0.031
PVR [Wood Units]	10.3 ± 3.7	5.9 ± 2.8	–43%	< 0.001
Biomarkers				
NT-proBNP [pg/mL]	2571 ± 2719	634 ± 697	–75%	< 0.001*
Median [IQR]	1683 [527–3379]	241 [69–1065]		
hsTnT [μg/L]	0.015 ± 0.011	0.012 ± 0.011	–20%	0.039*
Median [IQR]	0.014 [0.006–0.025]	0.007 [0.004–0.016]		
Functional capacity				
6MWT [m]	306 ± 153	397 ± 123	+30%	< 0.001*
Median [IQR]	333 [220–384]	408 [342–492]		
Mean WHO FC	3.19 ± 0.48	1.97 ± 0.80	–38%	< 0.001
WHO FC:				0.005
I	0 (0%)	10 (32%)		
II	1 (3%)	12 (39%)		
III	23 (74%)	9 (29%)		
IV	7 (23%)	0 (0%)		

*Calculated with Wilcoxon signed rank test; abbreviation as in Table 1

cedure. All deaths occurred in group A patients during the periprocedural period and were related to the first scheduled BPA session. The cumulative survival rate was 94.6% (95% CI 88–100%) at 12 and 24 months. We compared those data with a historical control group of 112 patients with CTEPH, who were treated with PEA (n = 66) or treated with medical therapy alone (n = 46) and followed by our team in the years 1998–2008 (Fig. 5) [2]. The cumulative survival rate after PEA was 90.9% (95% CI 84–97%) at 12 and 24 months (log-rank test vs. BPA group; p = 0.39). Historical patients on

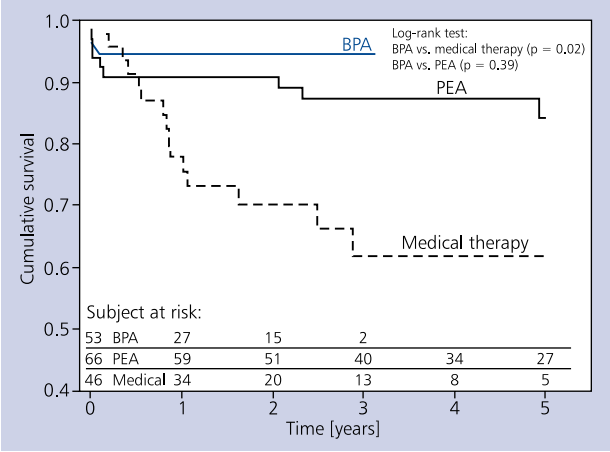

Figure 5. Kaplan-Meier cumulative survival curve for 56 patients who entered the balloon pulmonary angioplasty (BPA) programme (blue line) compared to historical controls with chronic thromboembolic pulmonary hypertension treated with pulmonary endarterectomy (PEA) (solid line) or medical therapy alone (dashed line) and followed by our team in the years 1998–2008 [2]. P estimated by log-rank test

medical therapy alone had survival rates of 78.0% (95% CI 66–90%) and 70.1% (95% CI 56–84%) at 12 and 24 months, respectively. The survival of patients who underwent BPA was more favourable than that of historical patients with CTEPH remaining on medical therapy alone (log-rank test; p = 0.02).

DISCUSSION

The outcome of patients suffering from occlusion of part of their pulmonary arterial bed, due to post-thromboembolic residue, depends on haemodynamic factors. The long-term survival progressively worsens when mPAP increases beyond 30 mm Hg. This is true for patients who do not undergo surgery as well as for those with pulmonary hypertension that persisted despite PEA [20, 21]. While CTEPH is initiated by anatomic occlusion, it is driven by remodelling of distal pulmonary vessels. This is partly due to the redistribution of excessive flow to pulmonary arteries that are still patent. A progressive increase in PVR leads to right ventricular failure, low cardiac output, and death. Any intervention reverting, stopping, or slowing down this vicious circle by reducing PAP and unloading the right ventricle should be beneficial for CTEPH patients.

Interventional cardiology has been successful in dilating vascular stenosis and opening occluded vessels in many vascular areas. There is increasing evidence that percutaneous BPA can serve this purpose for patients with CTEPH. However, the pulmonary environment in which mechanical interventions are necessary differs from that of peripheral, coronary, or extracranial vessels [22]. Pulmonary tissue surrounding pulmonary arteries offers little resistance to bleeding in cases of iatrogenic vascular injury. Extravasated blood may invade either the bronchial system or pulmonary interstitium [6, 9, 10]. Also, dilatation of a significant stenosis of a medium-sized pulmonary artery may result in local reperfusion oedema. All those complications may acutely deteriorate cardiorespiratory function, which is already affected by CTEPH, and result in periprocedural morbidity and mortality [23]. Our paper documents the learning curve we experienced. Observed complications forced us to refine our initial strategy of BPA in CTEPH patients.

Our initial, more aggressive approach to BPA was justified by two factors. The first was the high mortality in our population of non-operated CTEPH patients [2]. Indeed, the first patients on our waiting list for BPA were urgent cases because of the expected high risk of premature death without intervention. Therefore, we thought it was imperative to obtain a significant haemodynamic effect after the first BPA sessions. The second factor was the BPA methodology used by the first European centre that published its experience [6].

After the first 50 sessions, we found our programme to be at a crossroads. We were highly satisfied with the haemodynamic results, particularly visible after 2–3 sessions in individual patients, but we had lost three patients due to complications of BPA. Despite our increasing experience, we could not see any trend for reduction of the significant periprocedural morbidity, such as vascular injury with intrapulmonary bleeding, haemoptysis, and significant desaturations requiring mechanical respiratory support. Moreover, it was difficult to clearly identify why the BPA went smoothly in some sessions and resulted in potentially life-threatening complications in others.

Before accepting that BPA may simply have such unpredictable characteristics, we decided to compare our methodology with procedures used in centres in Japan. This was despite some concerns regarding the different characteristics of Japanese CTEPH patients, who showed less haemodynamic compromise than our European population. In Japanese patients, the immediate efficacy of the first BPA session could have less significance. In addition, Japanese teams could have different PEA qualification strategies. There was some risk that what we learned from Japan may not fully apply to our CTEPH population. Nevertheless, after initiating a direct collaboration with a BPA centre in Okayama, the most important differences in the BPA strategy between Otwock and Japan were analysed and the conclusions applied to our practice.

After more than one year using the modified strategy, we feel the implemented elements that most likely reduced our complication rate were the different selection of vascular lesions, using reduced tip load guidewires and less aggressive anticoagulation in the periprocedural period. A recent report from Japan confirmed the relationship between the type of lesion and the efficacy and the rate of complications in BPA, offering a novel angiographic classification of vascular lesions in CTEPH [23]. This classification identifies five types of le-

sions from A to E: ring-like, web, subtotal occlusions, total occlusions, and tortuous, respectively. As an example, no wire injury was noted when treating 248 type A lesions, while it was reported in 12% (41/342) of the type C and 43% (19/44) of the type E lesions.

Survival in CTEPH according to treatment

The mortality rate related to BPA in our population was 5.4% (3/56 patients), which is not negligible. Initial small series from Unites States and Europe reported 5–10% mortality [5, 6], and Okayama recently reported 4% (4 out of 97 patients) [23]. However, when analysing the whole experience of our team, starting from the very first BPA procedure performed in Otwock in July 2013 [15,17], regardless of the subsequent changes in BPA strategy, the two-year survival of patients treated with BPA is more favourable than the historical survival of our non-operated CTEPH patients [2]. Moreover, it is possible that we are still experiencing a learning curve and the future will bring even better results.

Limitations of the study

We still need to confirm that the refined BPA strategy will be as haemodynamically effective as our initial, more aggressive strategy had been in severely haemodynamically compromised patients. The direct comparison of efficacy is impossible as some patients started treatment with initial strategy and continued with refined strategy. Thus, we focused on safety assessment of particular procedures. The refined strategy will probably require more sessions/patient and may become more time consuming and costly. Moreover, while the reduction of periprocedural morbidity seems obvious, the early mortality in our initial series could be in part due to the characteristics of two of the three cases with fatal outcomes. The patients were technically operable but were disqualified from surgery due to advanced age (85 years) and large bilateral post-mycobacterial intrapulmonary cavities, respectively. In both cases, PEA was extensively discussed but finally considered to be of unacceptable risk due to the particularly severe haemodynamic profiles of the patients. Those patients died in the first 24 h after the procedure because of severe respiratory insufficiency despite mechanical ventilation, but without clinical symptoms of bleeding. The third case was different in terms of truly distal occlusions and a lack of warning of periprocedural symptoms and signs. Death resulted from the oligovolaemic shock due to sudden rupture of a large intrapulmonary haematoma seven days after an apparently uncomplicated procedure. We did not implement all the elements of the methodology used in Japanese centres because there were also some site-specific differences between centres. This was particularly true for the role assigned to intravascular ultrasound and pressure wire in the selection and assessment of results of BPA procedures. Similarly to the team from Okayama, we found this approach to be too time consuming and expensive to be routinely used.

CONCLUSIONS

Balloon pulmonary angioplasty is an effective method for treating patients with CTEPH, who could not benefit from first-line surgical therapy. However, it is not free from potentially life-threatening complications, some of which may be avoided by refining the BPA methodology. A joint effort of BPA teams worldwide will be required before the benefit-to-risk ratio of this promising method reaches its peak and the position of BPA in the treatment algorithm for CTEPH is fully defined. By reporting our experience, we hope to help teams that are considering starting interventional therapy of CTEPH to bypass at least a part of their learning curve and reduce their complication rate by applying a refined BPA strategy.

Acknowledgements

We would like to express our gratitude to Mrs. Małgorzata Suder (certified nurse) for her excellent assistance during the BPA procedures.

Funding: This study was supported by funds from the statutory activity of the Medical Centre of Postgraduate Education in Warsaw, Poland. It was carried out with grant number 501-1-54-01-17.

Conflict of interest: *Marcin Kurzyna reports to have received lecture fees from MSD, Bayer, and Actelion Pharma. Szymon Darocha reports to have received lecture fees from MSD, Bayer, and Actelion Pharma. Radosław Pietura has no conflict of interest to declare. Arkadiusz Pietrasik has no conflict of interest to declare. Justyna Norwa has no conflict of interest to declare. Rafał Mańczak has no conflict of interest to declare. Maria Wieteska reports lectures fee from Actelion Pharma, AOP Orphan and Bayer, outside the submitted work. Andrzej Biederman has no conflict of interest to declare. Hiromi Matsubara reports to have received lecture fees from Bayer Yakuhin, Pfizer Japan, Nippon Shinyaku, Actelion Pharma Japan, GSK, AOP Orphan, and Kaneka Medix, outside the submitted work. Adam Torbicki reports grants and personal fees from Actelion, personal fees from AOP, grants and personal fees from Bayer, and grants and personal fees from MSD, outside the submitted work.*

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Cite this article as: Kurzyna M, Darocha S, Pietura R, et al. Changing the strategy of balloon pulmonary angioplasty resulted in a reduced complication rate in patients with chronic thromboembolic pulmonary hypertension. A single-centre European experience. Kardiol Pol. 2017; 75(7): 645–654, doi: 10.5603/KP.a2017.0091.

Zmiana strategii balonowej angioplastyki tętnic płucnych zmniejsza częstość powikłań u chorych z przewlekłym zakrzepowo-zatorowym nadciśnieniem płucnym. Doświadczenia jednego europejskiego ośrodka

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Streszczenie

Wstęp i cel: Celem pracy była ocena bezpieczeństwa i skuteczności udoskonalonej strategii przezskórnej angioplastyki balonowej (BPA) u pacjentów z przewlekłym zakrzepowo-zatorowym nadciśnieniem płucnym (CTEPH).

Metody: Wykonano 157 zabiegów BPA u 56 osób z CTEPH (wiek 58,6 ± 17,9 roku; 28 kobiet). Pierwszych 50 zabiegów polegało na rewaskularyzacji przewlekłych niedrożności w tętnicach płucnych i zapobieganiu nawrotom zwężeń poprzez intensywną antykoagulację w okresie pozabiegowym. Kolejnych 107 zabiegów polegało na leczeniu zmian o typie „sieci" i „obrączkowatych przewężeń" przy użyciu prowadników ze zredukowanym *tip load* i zastosowaniu mniej intensywnej antykoagulacji.

Wyniki: Po zmianie strategii uzyskano istotną redukcję w zakresie krwioplucia (22% *vs*. 7%, p = 0,01), uszkodzenia naczynia (30% *vs*. 13%; p = 0,01), desaturacji (30% *vs*. 20%; p = 0,02) i uszkodzenia płuca związanego z BPA (38% *vs*. 22%; p = 0,041). 14-dniowa śmiertelność także uległa redukcji (6% *vs*. 0%; p = 0,05). Wskaźnik skumulowanego przeżycia wynosił 94,6% po 24 miesiącach od pierwszego zabiegu BPA i był znacznie korzystniejszy w porównaniu z przeżyciem historycznej grupy leczonej farmakologicznie. U 31 chorych, u których wykonano ponad trzy zabiegi BPA, stwierdzono istotną redukcję naczyniowego oporu płucnego (10,3 ± 3,7 vs. 5,9 ± 2,8 jedn. Wooda; p = 0,01), średnie ciśnienie w tętnicy płucnej (50,7 ± 10,8 *vs*. 35,6 ± 9,3 mm Hg; p=0,01) i poprawę klasy czynnościowej wg Światowej Organizacji Zdrowia (3,19 ± 0,48 *vs*. 1,97 ± 0,80; p < 0,001).

Wnioski: Angioplastyka balonowa tętnic płucnych poprawia parametry hemodynamiczne i czynnościowe, ale wymaga udoskonalenia strategii w celu zmniejszenia częstości groźnych powikłań.

Słowa kluczowe: angioplastyka balonowa tętnic płucnych, przewlekłe zakrzepowo-zatorowe nadciśnienie płucne, powikłania, przeżycie, krwioplucie

Kardiol Pol 2017; 75, 7: 645–654

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Praca wpłynęła: 22.03.2017 r. **Zaakceptowana do druku:** 27.04.2017 r. **Data publikacji AoP:** 10.05.2017 r.