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LETTER TO THE EDITOR

A case of balloon pulmonary angioplasty as a palliative therapy in chronic thromboembolic pulmonary hypertension

KEYWORDS
Balloon pulmonary angioplasty;
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Chronic thromboembolic pulmonary hypertension (CTEPH) constitutes the only potentially curable cause of pulmonary hypertension. Pulmonary thromboendarterectomy (PEA) is an established therapy for CTEPH in expert centers, but not all patients have surgically accessible disease.[1–3] Balloon pulmonary angioplasty (BPA) has recently been introduced as an adjunctive interventional therapeutic option for patients with inoperable or residual CTEPH after PEA.[4–6]

To the best of our knowledge, we report the first case of BPA in Greece. A 50-year-old male with symptomatic right heart failure (WHO functional class III) for 3 years who was diagnosed with pulmonary hypertension was referred to our center for further evaluation. He had a history of sickle cell disease, liver cirrhosis due to alcohol consumption and permanent atrial fibrillation. No history of pulmonary embolism or deep vein thrombosis was reported. The patient was treated with digoxin, eplerenone, and acenocoumarol. On a 6-min walk test, he achieved 453 m, and lung perfusion scanning revealed ventilation perfusion mismatches in the lower left, upper and middle right pulmonary lobes. Baseline hemodynamic data from right heart catheterization are presented in Table 1. Pulmonary angiography revealed multiple ring-like stenosis, webs and occlusions bilaterally in the lower and middle lobes, confirming CTEPH.

According to the guidelines,[7] surgical consultation was obtained from an internationally recognized PEA center abroad (absence of an official PEA center in Greece). The patient was considered amenable to PEA, with reservations due to his liver cirrhosis, but he categorically refused any surgical intervention. Therefore, he was considered a candidate for a BPA procedure.

The patient was admitted three days prior the procedure so that oral anticoagulation could be bridged with low molecular weight heparin, and he provided written informed consent. The procedure was performed through the right femoral vein. A bolus of 2000 IU of heparin was administered, which was followed by an additional 1000 IU hourly to maintain an ACT (activated clotting time) above 250 seconds throughout the procedure. Initially, standard right heart catheterization was performed, confirming the diagnostic workout values (Table 1). Afterwards, a 6 F JR 4.0 guide catheter (Judkins Right Launcher; Medtronic, Minneapolis, MN, USA) was advanced to the pulmonary artery. Selective pulmonary lobe angiography was performed in an anterior-posterior and lateral projections, depicting, in detail, the lesions identified in the baseline angiogram. Based on a

Table 1 Hemodynamic measurements pre- and post-balloon pulmonary angioplasty.

Hemodynamics	Pre BPA	Post BPA
SvO2 (%)	59	61
sPAP (mmHg)	79	69
dPAP (mmHg)	25	26
mPAP (mmHg)	44	40
RAP (mmHg)	12	11
PCWP (mmHg)	9	8
PVR (Wood units)	7.1	6.2
CI (L/min/m^2)	2.4	2.5

SvO2: mixed venous saturation, sPAP: systolic pulmonary arterial pressure, dPAP: diastolic pulmonary arterial pressure, mPAP: mean pulmonary arterial pressure, RAP: right atrial pressure, PCWP: pulmonary capillary wedge pressure, PVR: pulmonary vascular resistance, and CI: cardiac index.

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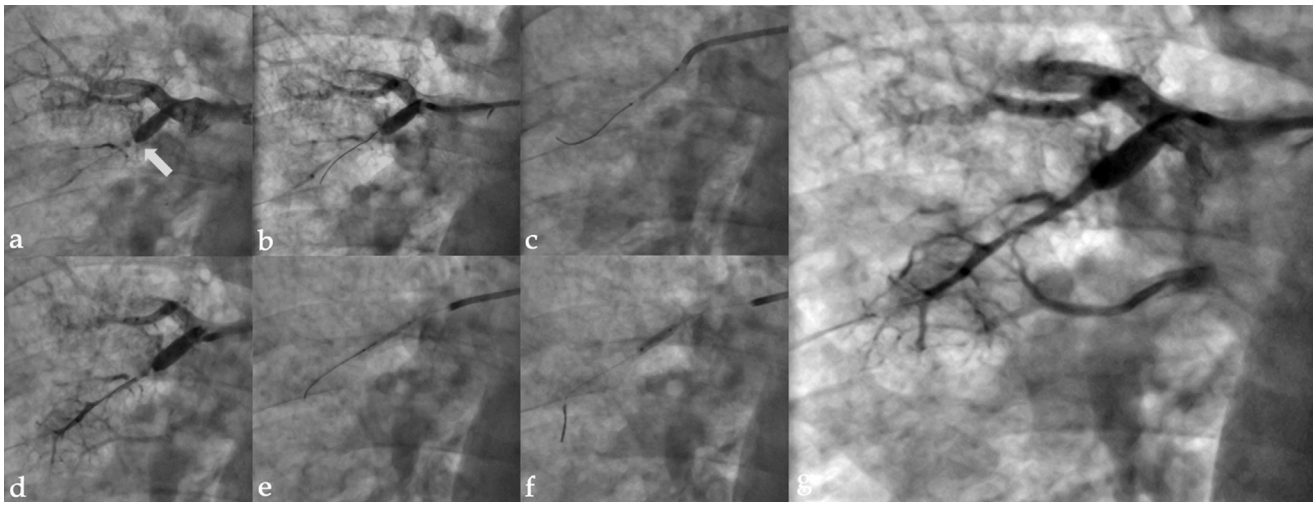

Figure 1 Balloon pulmonary angioplasty on a total pulmonary arterial occlusion. a. Total occlusion of the right middle pulmonary branch with a blunt proximal cap (arrow). b. A Gaia Third wire (Asahi Intecc, Tokyo, Japan) supported by a Finecross microcatheter (Terumo, Tokyo, Japan) advanced through the body of the occlusion. c. Initial dilatation with a Sprinter Legend 2.0x20 mm balloon (Medtronic, Minnesota, USA) at 8 Atm over a Runthrough wire (Terumo, Tokyo, Japan). d. Angiographic result after initial balloon dilatation. e. Subsequent dilatation with a Sprinter Legend 2.5x20 mm balloon at 8 Atm. f. Final dilatation with a Sprinter Legend 3.0x12 mm balloon at 8 Atm. g. Final angiographic result with restoration of antegrade pulmonary flow grade 3 and no signs of complications.

large perfusion defect identified in the right middle lobe, we focused therapeutically on a totally occluded branch that supplies this territory (Figure 1).

The occlusion was initially approached with a Fielder FC wire (Asahi Intecc, Tokyo, Japan) supported by a Finecross microcatheter of 130 cm (Terumo, Tokyo, Japan). This wire could not be advanced through the occlusion; subsequently, composite core wires were used (Gaia First and Gaia Third; Asahi Intecc, Tokyo, Japan). The Gaia Third was successfully advanced through the body of the occlusion and, after the microcatheter advancement, it was exchanged to a floppy wire (Runthrough, Terumo, Tokyo, Japan) to avoid any wire injury at the distal arterial bed. Initial dilatation was performed with a Sprinter Legend 2.0x20 mm balloon (Medtronic, Minnesota, USA), which was followed by a Sprinter Legend 2.5x20 mm. The last dilatation was performed with a Sprinter Legend 3.0x12 mm balloon. Its size was selected to match the 70% of the angiographically estimated diameter (4.5 mm) of the proximal arterial segment, as previously described. The final dilatation reached the 8 atm until full balloon expansion. Pulmonary arterial flow was completely restored (angiographic pulmonary flow grade 3), and there were no signs of arterial rupture or wire injury. Oxygen desaturation did not exceed 4%, and the patient did not develop hemoptysis throughout the procedure. The cumulative radiation dose was 3122 μGym^2 and the contrast media was 400 ml. At the end of the procedure, the mPAP was 40 mmHg, cardiac index was 2.5 $l/min/m^2$ and PVR was 6.2 Wood units (Table 1). As this was our initial experience, we decided against dilating other pulmonary artery lesions and performing further dilatations in a staged fashion over multiple, separate procedures, as previously described.[4] The patient underwent an uneventful post-procedural period and was discharged on the 5th day after BPA.

BPA is a complimentary/alternative approach to the therapeutic management of CTEPH in patients with inoperable or residual disease after PEA, as well in those who refuse surgery or have an unacceptably high surgical risk for PEA,[8] offering marked improvement in both subjective clinical symptoms and hemodynamics.[9] Multiple procedures are usually needed to achieve a significant reduction in pulmonary vascular resistance. Despite technical difficulties and the lack of dedicated devices, BPA, when performed by experienced operators in dedicated centers, has comparable complications to those reported for PEA and is both feasible and efficient for palliative treatment in certain CTEPH patients.[1,10,11] In the first report of BPA, the in-hospital death rate was 5.6%, whereas it was slightly lower, at 0—3.4%, in the subsequent reports.[9] The main complications include lung injury caused by reperfusion edema, pulmonary artery perforation or vessel rupture with a guidewire and contrast nephropathy following multiple procedures. To establish BPA as therapeutic alternative compared to drug therapy (i.e., riociguat), evidence based on randomized controlled data are required to prove the long-term improvement in patients' clinical statuses and survival.

Conflict of interest

The authors declare there are no conflicts of interest related to this manuscript.

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