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Review/*Thoracic imaging*

Imaging of chronic thromboembolic pulmonary hypertension before, during and after balloon pulmonary angioplasty

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A B S T R A C T

Balloon pulmonary angioplasty (BPA) has recently been elevated as a class I recommendation for the treatment of inoperable or residual chronic thromboembolic pulmonary hypertension (CTEPH). Proper patient selection, procedural safety, and post-procedural evaluation are crucial in the management of these patients, with imaging work-up playing a pivotal role. Understanding the diagnostic and therapeutic imaging algorithms of CTEPH, the imaging features of patients amenable to BPA, all imaging findings observed during and immediately after the procedure and the changes observed during the follow-up is crucial for all interventional radiologists involved in the care of patients with CTEPH. This article illustrates the imaging work-up of patients with CTEPH amenable to BPA, the imaging findings observed before, during and after BPA, and provides a detailed description of all imaging modalities available for CTEPH evaluation.

1. Introduction

Chronic thromboembolic pulmonary hypertension (CTEPH) is a rare (3–5/100,000) and potentially fatal disease, defined as a mean pulmonary arterial pressure (mPAP) > 20 mm Hg and pulmonary vascular resistance (PVR) > 2 Wood units despite anticoagulant therapy ≥ 3 months [1–5]. CTEPH, unlike other forms of pulmonary hypertension (PH), is primarily caused by incomplete resolution of multiple pulmonary artery (PA) thromboembolisms, obstructing blood flow and increasing PVR and mPAP. Additionally, CTEPH causes lung ventilation/perfusion (V'/Q') imbalances, hampering gas exchange and causing symptoms like breathlessness. Prolonged pressure overload triggers right ventricular hypertrophy, dilatation, and ultimately, life-threatening right heart failure and death [6]. CTEPH is classified as group 4 PH and is the only potentially curable form of PH [1]. However, if not treated, it has a poor prognosis, with reported survival without any medical or invasive therapy at three years < 30% [7–9].

The invasive treatment of choice of CTEPH is pulmonary endarterectomy (PEA) [10,11]. Although potentially curative, PEA is a complex surgery to which up to 40% of patients are not amenable [12]. Balloon pulmonary angioplasty (BPA) is a minimally invasive treatment available in patients with distal disease unreachable by surgical means or in inoperable patients [13,14]. BPA improves CTEPH patients' hemodynamic status and clinical condition, with reported improvement in the six-minute walking test of up to 70 m

Abbreviations: 2D-PA, Two-dimensional perfusion angiography; 3D, Three-dimensional; BPA, Balloon pulmonary angioplasty; CBCT-PA, Cone beam CT-pulmonary angiography; CT-PA, Single-energy CT pulmonary angiography; CTEPH, Chronic thromboembolic pulmonary hypertension; DECT-PA, Dual-energy CT-pulmonary angiography; DSA-PA, Digital subtraction pulmonary angiography; HU, Hounsfield unit; LAO, Left anterior oblique; mPAP, Mean pulmonary arterial pressure; NYHA-FC, New York Heart Association functional class; PA, Pulmonary artery; PEA, Pulmonary endarterectomy; PH, Pulmonary hypertension; PVR, Pulmonary vascular resistance; RHC, Right heart catheterization; RLE, Reperfusion lung edema; SPCCT, Spectral photon-counting CT; TDE, Transthoracic Doppler echocardiography; V'/Q', Ventilation/perfusion

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and in the New York Heart Association functional class (NYHA-FC) of up to 0.9 [15–17]. Although severe complication rates reaching up to 10–15%, including hemoptysis and lung injury, were initially reported, recent studies have shown that initiating a BPA program with lower rates of severe complication, ranging between 1% to 5%, is possible [13,18,19]. As a result, BPA has been recently upgraded from class IIb to class I recommendation [1].

Interventional radiologists involved in the management of patients with CTEPH should thoroughly understand the imaging characteristics of patients with CTEPH who are amenable to BPA and be familiarized with intraprocedural imaging findings and imaging follow-up management. The purpose of this article was to discuss the imaging work-up of patients with CTEPH before BPA, illustrate the imaging findings observed during and after BPA, and highlight state-of-the-art imaging modalities introduced for CTEPH evaluation.

2. Chronic thromboembolic pulmonary hypertension imaging assessment

2.1. Chronic thromboembolic pulmonary hypertension diagnosis

Transthoracic Doppler echocardiography (TDE) is the recommended initial imaging test for patients with suspected PH. TDE assigns patients to one of the probability categories, including low, intermediate, or high, Peak tricuspid regurgitation velocity is the primary value to be considered, with a value > 3.4 m/s associated with a high probability of PH. All TDE features for the diagnosis of PH are described in Table 1 [20]. In patients with intermediate and high probability of PH, V'/Q' scintigraphy should be performed [1]. When V'/Q' scintigraphy reveals normal findings, CTEPH can be ruled out with high accuracy (92–95%) and high negative predictive value (98–99%) [21,22]. However, if mismatched V'/Q' defects are reported, a right heart catheterization (RHC) is warranted [3]. Normal values for mPAP and PVR during RHC are 8–20 mmHg and 0.3–2.0 Wood units, respectively. CTEPH is confirmed with an mPAP > 20 mmHg and PVR > 2 Wood units [1]. At this stage, patients should be referred to a center with a specialized CTEPH unit to undergo a thorough evaluation (Fig. 1) [1].

Imaging characterization of chronic thromboembolic lesions is the next step. Several imaging techniques are available in this regard, encompassing conventional or single-energy CT pulmonary angiography (CT-PA), digital subtraction pulmonary angiography (DSA-PA), dual-energy CT-PA (DECT-PA), and cone-beam CT-PA (CBCT-PA) (Tables 2 and 3) [1,23].

2.2. Chronic thromboembolic pulmonary hypertension imaging modalities

CTPA is a non-invasive and readily available test that allows adequate characterization of lesions at the main, lobar, and proximal segmental PAs [24]. However, technique performance drops when assessing segmental and subsegmental disease [25]. Electrocardiogram-gated CT-PA may increase the sensitivity and specificity of the technique, with values up to 99% at the segmental, level compared to DSA-PA [26]. CT imaging also allows an initial assessment of signs of PH severity, lung parenchymal status, anatomical variants, and possible concurrent diseases (Fig. 2) [27].

DSA-PA is still considered as the gold standard imaging modality for CTEPH [23]. DSA-PA also allows the performance of RHC in the same setting and the dynamic assessment of parenchymal perfusion by detecting the presence of lobar, segmental, or subpleural perfusion defects. Also, using DSA-PA, interventional radiologists can immediately assess the intraprocedural impact of BPA on previously compromised vessels, by observing the immediate revascularization of the treated lung parenchyma [23]. The future trend will likely be the combination of DSA-PA with newly available imaging tests such as DECT-PA and CBCT-PA [28].

DECT-PA generates an iodine map superimposable to the CT image, which has been considered as a surrogate marker of pulmonary perfusion [29,30]. DECT-PA offers a "one-stop" assessment of anatomy and perfusion in patients with CTEPH [31,32]. Perfusion defects depicted on DECT-PA correlate with hemodynamic estimates of PH severity [33]. The diagnostic value of DECT-PA iodine map has been recently compared with V'/Q' scintigraphy, with sensitivities and specificities ranging from 97% to 100% and 86% to 92%, respectively [34,35]. However, while DECT-PA can perform effectively, there are numerous instances for which it is unsuitable or is limited by technical issues such as obesity or artifacts. [23]. Furthermore, the DECT-PA iodine map is not as widely recognized outside specialized centers. By contrast, V'/Q' is readily available in most centers and familiar to most healthcare providers [23].

CBCT-PA is a recently implemented technique derived from DSA-PA. Images are obtained during a rotational acquisition with an angio-suite C-arm, while iodinated contrast material is administered through a catheter directly into the common PA [36]. CBCT-PA obtains a greater arterial contrast attenuation than CT-PA, improving the delineation of distal and subsegmental vessels and ultimately enhancing lesion characterization [37,38]. CBCT-PA, while effective for detecting subsegmental lesions, is invasive, patient-dependent, and susceptible to breathing motion artifacts. It requires precise teamwork for optimal imaging and does not yield high-quality lung parenchyma images.

Table 1
Echocardiographic probability of pulmonary hypertension.

Main echocardiographic sign of pulmonary hypertension		
Low peak tricuspid regurgitation velocity (≤ 2.8 m/s)	Intermediate peak tricuspid regurgitation velocity (2.9–3.4 m/s)	High peak tricuspid regurgitation velocity (> 3.4 m/s)
Additional echocardiographic signs suggestive of pulmonary hypertension*		
A: Ventricules	B: Pulmonary artery	C: Inferior vena cava/Right atrium
RV/LV basal diameter/area ratio > 1.0	RVOT AT < 105 ms Mid-systolic notching	IVC diameter > 21 mm with decreased inspiratory collapse (< 50% with a sniff or < 20% with quiet inspiration)
Flattening of interventricular septum (LVEI > 1.1)	Early diastolic pulmonary regurgitation velocity > 2.2 m/s	RA area (end-systole) > 18 cm^2
TAPSE/sPAP ratio < 0.55 mm/mm Hg	PA > AR diameter PA diameter > 25 mm	

* Signs of at least two categories (A/B/C) must be present to alter the level of probability.
AR indicates aortic root. IVC indicates inferior vena cava. LV indicates left ventricle. LVEI indicates left ventricle eccentricity index. PA indicates pulmonary artery. PH indicates pulmonary hypertension. RA indicates right atrium. RV indicates right ventricle. RVOTAT indicates right ventricular outflow tract acceleration time. sPAP indicates systolic pulmonary arterial pressure. TAPSE indicates tricuspid annular plane systolic excursion. TRV indicates tricuspid regurgitation velocity.

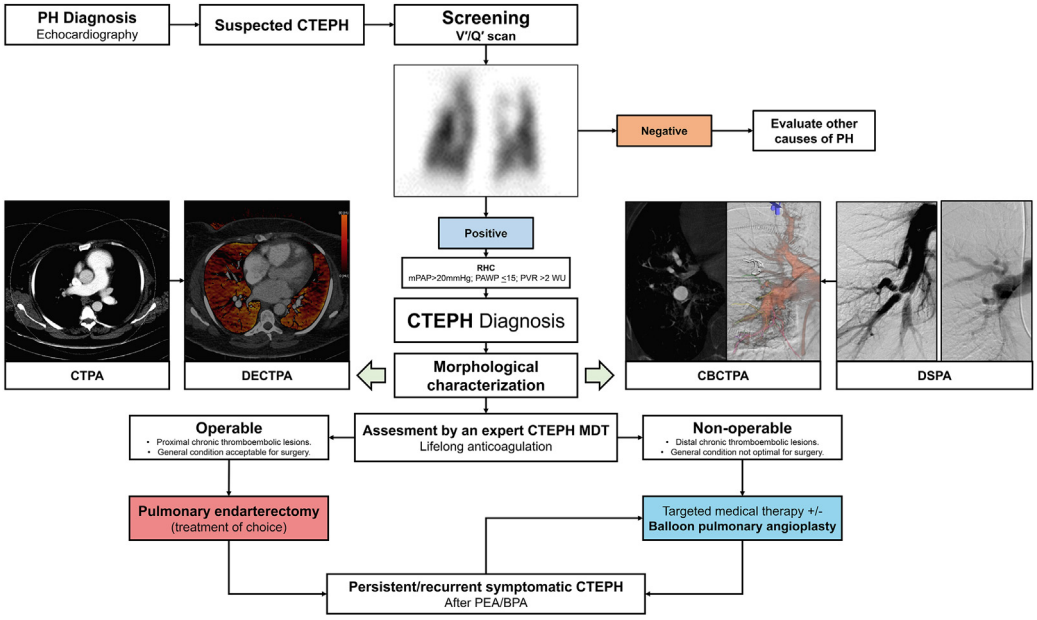

Fig. 1. Diagnostic and therapeutic imaging algorithm for chronic thromboembolic pulmonary hypertension. BPA indicates balloon pulmonary angioplasty; CBCTPA indicates cone beam CT-pulmonary angiography; CTEPH indicates chronic thromboembolic pulmonary hypertension; CTPA indicates single-energy computed tomography pulmonary angiography; DECT-PA indicates dual-energy CT-pulmonary angiography; DSPA indicates digital subtraction pulmonary angiography; PH indicates pulmonary hypertension; MDT indicates medical team; PEA indicates pulmonary endarterectomy; RHC indicates right heart catheterization.

Regarding future directions for imaging management of CTEPH, several advancements are notable. DECT-PA is poised for a significant qualitative improvement with the introduction of spectral photon-counting CT (SPCCT) [39]. SPCCT promises to enhance the sampling of energy dependence in DECT-PA images, offering more comprehensive and accurate analyses. The benefits of SPCCT, particularly for assessing pulmonary perfusion, are currently under development [39]. Additionally, the potential of artificial intelligence-based tools for detecting blood clots and assessing pulmonary hypertension severity scores using three-dimensional (3D) CTPA examinations, leveraging blood clots and cardiac segmentation, has been demonstrated [40]. Further research is necessary to validate the effectiveness of these tools in the CTEPH setting [40].

2.3. Imaging in chronic thromboembolic pulmonary hypertension treatment selection

Therapies available for CTEPH are PEA, BPA and PH-targeted medical treatment. PEA is the treatment of choice of CTEPH [1]. Improvements of up to 65% in PVR, mPAP (46 to 26 mm Hg), median six-minute walking test (362 to 459 m), and NYHA-FC after PEA have been reported [10,12]. In high-volume centers, the in-hospital mortality is now < 5% [10,12,41]. Although variations exist between centers, patients with proximal lesions (main and lobar arteries) are usually considered for PEA.

BPA has shown improvement in the six-minute walking test (+70 meters), NYHA-FC (−0.9 classes), mPAP (−13.2 mm Hg), and PVR (−3.9 Wood units). The most frequent complications include lung injury (incidence ranging between 8% and 30%), defined as the presence of damage to the lung tissue caused by the intervention, and hemoptysis (incidence ranging between 5% and 15%) [15]. Patients with distal segmental or sub-segmental thromboembolic disease are usually selected for BPA [41,42]. Other reasons to consider BPA over PEA are patients in poor general condition, with concurrent pulmonary or cardiac disease, or as a therapeutic alternative in residual CTEPH after PEA [17].

Riociguat, the only approved PH targeted medical treatment for CTEPH, is a soluble guanylate cyclase stimulator that increases cyclic

Table 2
Advantages and limitations of the different imaging modalities used for the assessment of pulmonary hypertension.

Variable	CRX	V'/Q' scan	SE-CTPA	DE-CTPA	DSA-PA	CBCT-PA
PH detection	+	–	+	+	–	+
Lung	+	–	+++	+++	–	+
Heart	+	–	++	++	–	++
PAs	+	+	+++	++++	++	+++++
Mediastinum	–	–	+++	+++	–	++
PH etiology	–	++	+++	++++	++	++
Strengths	Readily available High sensitivity (97%) and specificity (91%)	Screening for CTEPH	Excellent evaluation of etiologies of PH	Assessment of anatomy and lung perfusion (iodine maps)	Planning of invasive treatment	Detailed evaluation of distal segmental and subsegmental PAs
Weaknesesses	Limited role in the assessment of etiology	Needs further imaging to assess the cause of PH	Limited evaluation of distal pulmonary arteries	Needs validation for all DECT technologies	Absence of perivascular structure evaluation Invasive test	Needs validation Invasive test

CBCT-PA indicates cone beam CT pulmonary angiography. CXR indicates chest X-ray radiography. CTEPH indicates chronic thromboembolic pulmonary hypertension. DECT-PA indicates dual-energy CT pulmonary angiography. DSA-PA indicates digital subtraction pulmonary angiography. PAs indicates pulmonary arteries. PH indicates pulmonary hypertension. SECT-PA indicates single energy CT pulmonary angiography. V'/Q' scan indicates ventilation/perfusion scan. "-" indicates no utility. "+" indicates limited utility. "++" indicates moderately useful. "+++" indicates useful. "++++" indicates very useful.

Table 3
Recommended protocols for imaging modalities used for the diagnosis of chronic thromboembolic pulmonary hypertension.

	V'/Q' SPECT	SECT-PA	DECT-PA	DSA-PA	CBCT-PA	MRI
Protocol	Two phases 1. Ventilation 2. Perfusion Multidetector gamma-cameras	Single-enery CE multi-detector CT	Dual-energy CE in a dual phase 2nd generation CT	Digital subtraction angiography with PA (0°), lateral (90°) and oblique (45°) views of both lungs	CBCT angiography with contrast injection directly into the pulmonary trunk through a catheter	4D flow cardiac MRI Native T1 mapping CE T1 mapping Cine-balanced SSFP
Contrast dose	Aerosol ^{99}Tc-DTPA (5–35 mCi) Intravenous ^{99m}Tc-MAA (1–4 mCi)	75–85 mL iodine contrast	75–85 mL iodine contrast	20–24 mL iodine contrast per acquisition	60–80 mL iodine contrast	0.1–0.2 mmol/ kg GBCA (CE-T1-weighted mapping)
Contrast rate (mL/s)	IV injection at low manual rate	5	5	10–12	8–10	N/A
Kv	N/A	120	Low-energy, 80 High-energy, 140	60–80	100–120	N/A
mAs	N/A	100–300	100–300	200–400	100–300	
Delayed scanning time (s)		4–7 s	4–7 s	2 s	4 s	15 min (CE-T1W mapping)
Images/s	N/A	N/A	N/A	7	N/A	
Radiation dose (mSv)	2.2	2–5	3–5	10–30	3–5	None

^{99}Tc-DTPA indicates technetium Tc-99 m pentetic acid; ^{99m}Tc-MAA indicates technetium ^{99m}Tc macro aggregated albumin; CBCT indicates cone beam computed tomography; CE indicates contrast-enhanced; CTPA indicates CT pulmonary angiography; DECTPA indicates dual-energy CTPA; DSPA indicates digital subtraction pulmonary angiography; GBCA indicates gadolinium-based contrast agent; MRI indicates magnetic resonance imaging; N/A indicates not applicable; PA indicates posteroanterior; SECT-PA indicates single energy-CT pulmonary angiography; SSFP indicates steady-state free precession; V'/Q' scan indicates ventilation/perfusion scan.

guanosine monophosphate production, enhancing vasodilation and lessening vascular resistance. Riociguat has also antiproliferative and antifibrotic effects [43]. Riociguat is offered in patients with microvascular disease not amenable to BPA or PEA, as an adjunctive treatment in patients selected for invasive treatment and residual disease after PEA or BPA [44,45].

3. Pre-balloon pulmonary angioplasty imaging findings

3.1. Lesion characterization by location

Lesions location can be classified according to the classification from the University of California (Fig. 3) as follows: level 0 (no evidence of thromboembolic disease), level I (lesions in main PAs), level II (lobar), level III (segmental), and level IV (subsegmental) [41].

CT-PA has shown 98.3% sensitivity and 94.8% specificity for detecting chronic thromboembolic lesions at the level of the main-lobar arteries [24]. However, performance diminishes at level III, with sensitivities decreasing to as low as 70% and specificities ranging from 87% to 90% [46]. Furthermore, different studies have repeatedly shown an acute drop in performance in level IV lesions, with specificities as low as 25% for subsegmental lesion identification [21,22,24,47].

DSA-PA is equivalent to CT-PA in characterizing thromboembolic lesions at the main-lobar level [46,47]. DSA-PA appears superior to CT-PA for distal disease, as it can assess pulmonary perfusion and denote involvement up to the distal subsegmental level. Compared to DSA-PA, CT-PA sensitivity and specificity at level III-IV are 70%–80% and 85%–90%, respectively [23,46,47]. However, Ley et al. demonstrated that electrocardiogram-gated CT-PA can outperform DSA-PA in detecting level III lesions with a sensitivity of 100% *vs.* 75% and specificity of 99% *vs.* 100% [26].

DECT-PA can better detect segmental and subsegmental pulmonary arterial thromboembolic involvement, with an excellent agreement between DECT-PA and V/Q scintigraphy in the diagnosis of

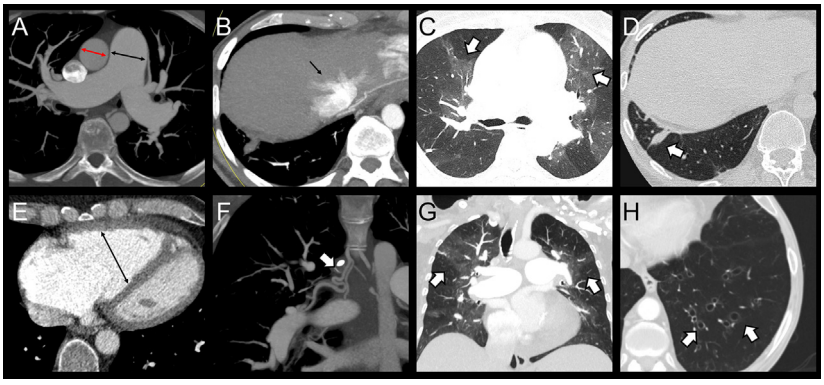

Fig. 2. Extravascular findings of chronic thromboembolic pulmonary hypertension. (**A-F**) 65-year-old woman with suspected chronic thromboembolic pulmonary hypertension (CTEPH). (**A**), CT image in the axial plane obtained after intravenous administration of iodinated contrast material (ICM) shows enlarged pulmonary artery (PA) trunk > 29 mm (black double arrow) and pulmonary artery-aorta ratio > 1 (red double arrow). (**B**), CT image shows reflux and mild enlargement of the hepatic veins (arrow). (**C**), CT image in the axial plane using lung window shows mosaic pulmonary pattern (arrows). (**D**), CT image in the axial plane using lung window shows healed pulmonary infarcts (white arrow). (**E**), CT image shows right ventricular enlargement and rectification of the interventricular septum (black double arrow). (**F**), CT image in the coronal plane shows hypertrophic bronchial arteries (arrow). (**G**), CT image in the coronal plane using lung window showing air trapping (arrows). (**H**), 46-year-old man with confirmed CTEPH. CT image in the axial plane obtained without ICM shows mild bronchiectasis in the lower left lobe (arrows).

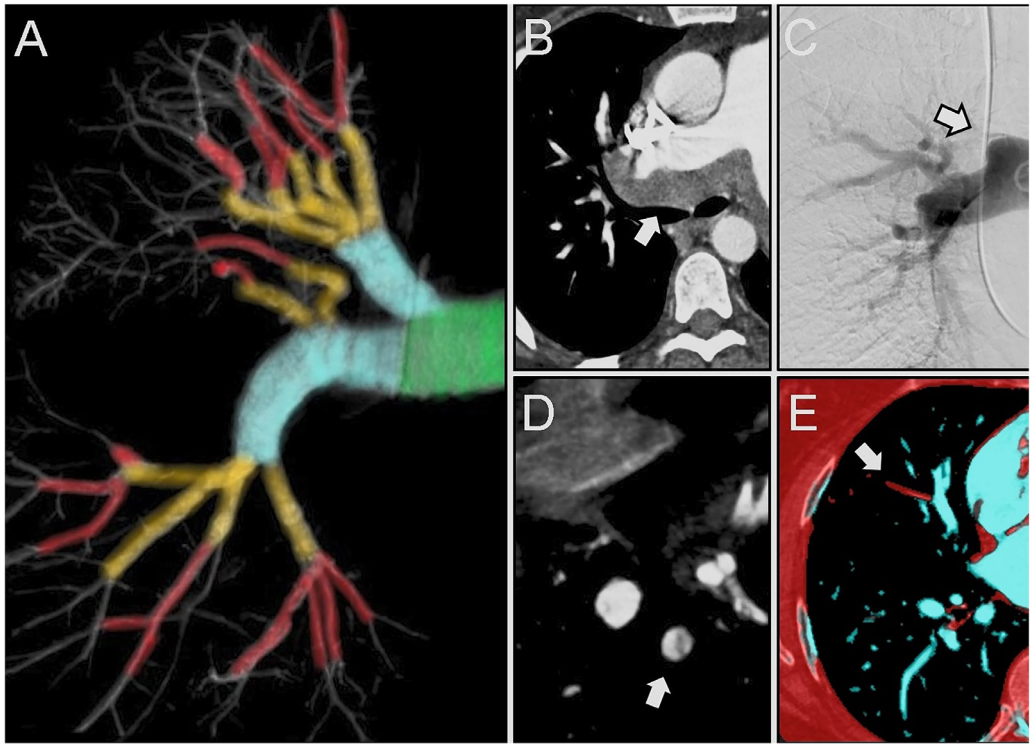

Fig. 3. Location classification of chronic thromboembolic lesions. (**A**) Three-dimensional cone beam CT-pulmonary angiography (CBCT-PA) reconstruction in a 65-year-old man with suspected chronic thromboembolic pulmonary hypertension (CTEPH): *Level I:* lesions beginning in the main pulmonary artery (PA) (green). *Level II:* lobar arteries (blue). *Level III:* segmental level (yellow). *Level IV:* subsegmental level (red). (**B**) Single-energy CT pulmonary angiography image in the axial plane in a 56-year-old man demonstrates involvement at the level of the main PA (arrow, *level I*). (**C**) Digital subtraction pulmonary angiography of another patient (36-year-old woman) shows involvement beginning at the lobar level (arrow, *level II*). (**D**) CBCT-PA in a 72-year-old woman with confirmed CTEPH demonstrates lesions affecting the segmental level (arrow, *level III*). (**E**) Dual-energy CT-pulmonary angiograph iodine map in a 43-year-old-man depicts subsegmental perfusion defects (arrow, *level IV*).

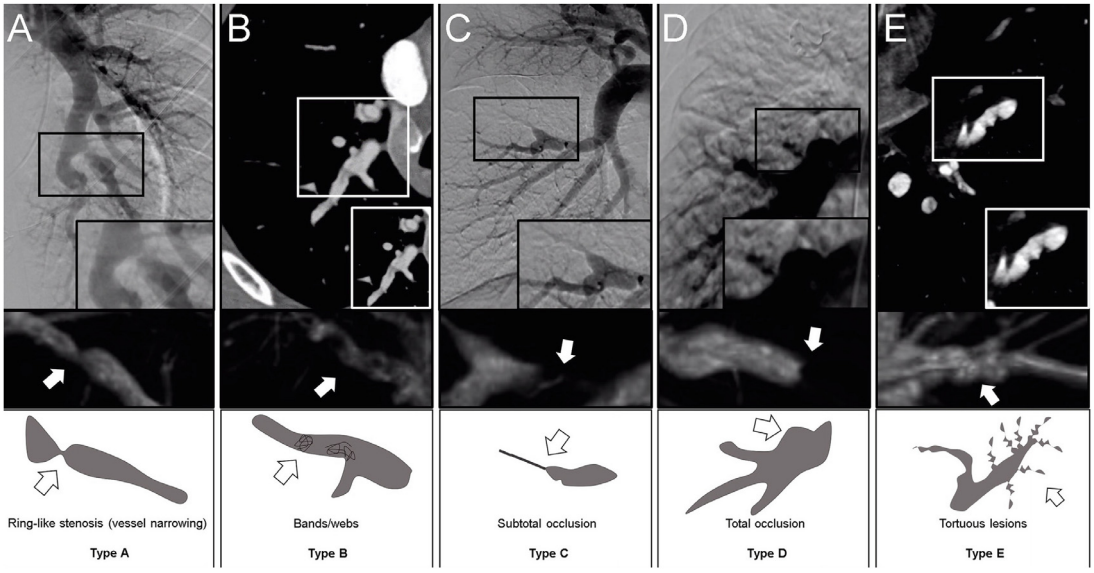

Fig. 4. Morphological classification of chronic thromboembolic lesions using various imaging modalities and three-dimensional schematic reconstructions. Chronic thromboembolic lesions are framed and close-ups in the five images. (**A**) Digital subtraction pulmonary angiography (DSA-PA) in a 64-year-old woman with confirmed chronic thromboembolic pulmonary hypertension (CTEPH) demonstrates a ring-like stenosis lesion in the left posterobasal segmental branch. (**B**) Single-energy CT pulmonary angiography in a 55-year-old man with confirmed CTEPH shows band and web lesions in the superior segmental branches of the right lower lobe and the right anterior basal segmental branch. (**C**) DSA-PA in a 36-year-old woman with confirmed CTEPH shows a subocclusion lesion in the lateral basal segmental branch of the right lower lobe. (**D**) DSA-PA in a 26-year-old woman with confirmed CTEPH reveals total occlusion of the superior lobar branch. (**E**) Cone beam CT-pulmonary angiography in a 56-year-old man with confirmed CTEPH shows tortuous thromboembolic lesion in the left superior lingular branch. Each image of the lower row demonstrates a schematic representation of each lesion using three-dimensional reconstruction (arrows).

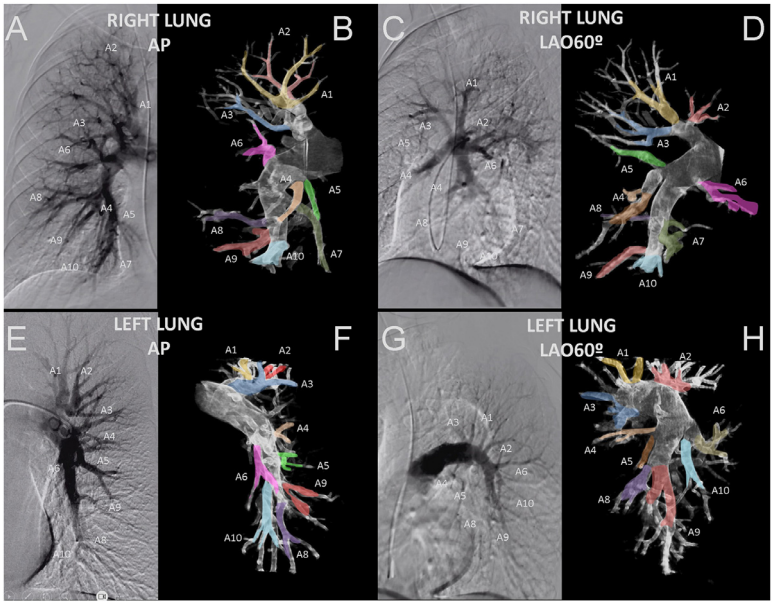

Fig. 5. Pulmonary segments on usually used working angiographic projections during balloon pulmonary angioplasty (PA and LAO 60° projections). Digital subtraction pulmonary angiography (DSA-PA) and three-dimensional reconstruction from cone-beam CT-pulmonary angiography (3D-CBCT-PA) in a 56-year-old woman with confirmed chronic thromboembolic pulmonary hypertension. (**A-D**) demonstrates DSA-PA AP (**A**) and left anterior oblique (LAO) 60° view (**C**), and 3D-CBCT-PA AP (**B**) and LAO 60° view (**D**) images of the right lung. (**E-H**) demonstrates DSA-PA AP (**E**) and LAO60° (**G**), and 3D-CBCT-PA AP (**F**) and LAO60° view (**H**) images of the left lung. Segments are labeled A1 to A10 and marked in colors in the 3D-CBCT-PA images.

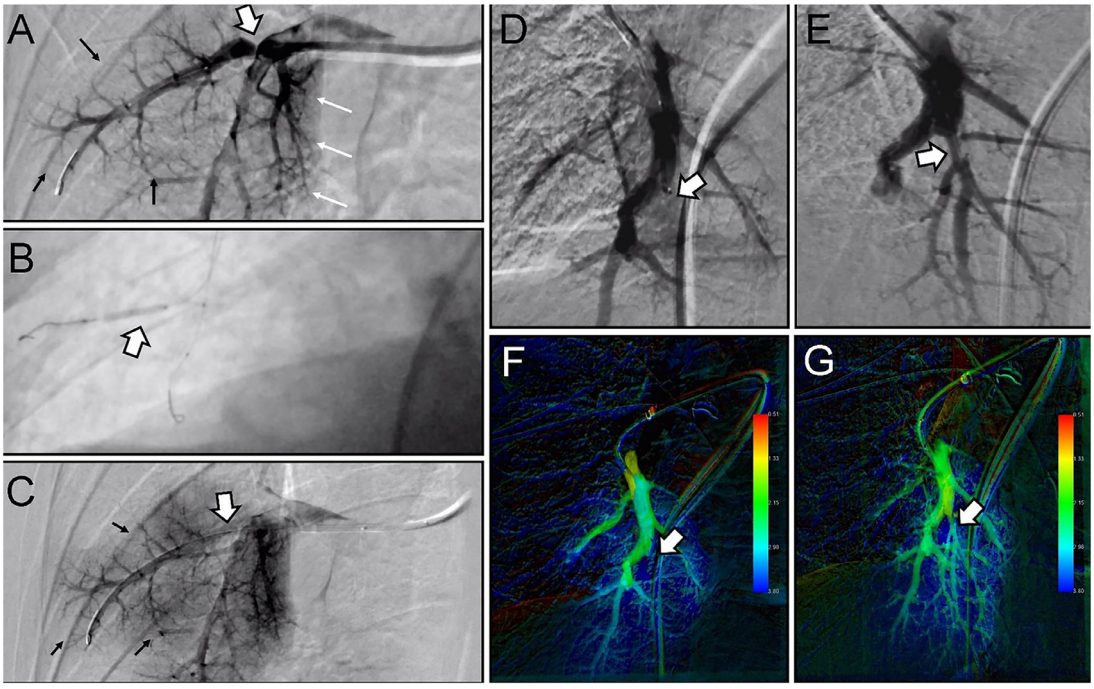

Fig. 6. Pre- and post-balloon pulmonary angioplasty intraprocedural assessment using digital subtraction pulmonary angiography and two-dimensional perfusion angiography. 66-year-old man with confirmed chronic thromboembolic pulmonary hypertension (CTEPH) undergoing his second balloon pulmonary angioplasty (BPA) session. (**A**) Digital subtraction pulmonary angiography (DSA-PA) demonstrates a chronic thromboembolic lesion (ring-like stenosis) (large arrow) in the lateral branch of the middle lobe, with flow reduction (black arrows) compared with the medial segmental branch (small white arrows). (**B**) Same patient session as in A, DSA-PA shows inflated balloon during BPA in the described lesion (arrow). (**C**) DSA-PA demonstrates recovery of the vessel caliber (arrow) and normal pulmonary flow (black arrows) after BPA in the lesion described in A. (**D-G**) 76-year-old woman with confirmed CTEPH undergoing her second BPA session. (**D**) DSA-PA demonstrates complete occlusion of the posterobasal segmental branch of the right lower lobe (arrow). (**E**) DSA-PA after BPA of the lesion described, shows recovery > 50% of the normal vessel caliber (arrow). (**F-G**) Two-dimensional perfusion angiography in the same patient as in **D, E**), before (**F**) and after (**G**) BPA, confirms recovery of pulmonary perfusion.

CTEPH (kappa = 0.80) and a similar diagnosis performance for detecting segmental perfusion defects, with 85% and 92% sensitivities and 99% and 99% specificities, respectively [30]. Furthermore, the diagnostic accuracy of DECT-PA is enhanced by the combined morpho-functional analysis of the iodine map and CTPA [48]. DECT-PA also increases the detection of segments affected by chronic thromboembolic disease by 26.6% compared to CT-PA alone. This ability is essential when characterizing lesions amenable to treatment with BPA [49].

Owing to the use of CBCT-PA, a greater contrast density is obtained in the pulmonary vasculature by comparison with CT-PA (534 Hounsfield units [HU] *vs.* 351 HU) along with a shorter distance between distal vessels and pleura (11.9 mm *vs.* 15.2 mm). Both features enable better delineation and assessment of the distal subsegmental pulmonary vasculature [38]. Moreover, Hinrichs et al. demonstrated that CBCT-PA allowed identify a greater number of lesions at all levels, including segmental and subsegmental, compared to DSA-PA (14.9% *vs.* 6.8%), with an inter-modality agreement between observers significantly higher than with DSA (kappa = 0.96 *vs*. 0.61, respectively) [37]. These findings were further confirmed by Mashke et al. in a study that included 300 patients [28].

3.2. Lesion characterization by morphology

Kawakami et al. proposed a classification based on outcomes and complications using DSA-PA, dividing lesions as follows: ring-like stenosis (type A), bands/webs (type B), subtotal occlusion (type C), total occlusion (type D), and tortuous lesions (type E) (Fig. 4) [50]. Complication rates are < 3% for type A-B lesions but can reach 15.5% for type C and up to 40% for type E lesions [50]. Depending on the type of lesion, technical success is 100% for type A, 98.7% for type B, 86.5% for type C, 52.2% for type D, and 63.6% for type E [50]. Therefore, several guidelines recommend treating type A-B lesions first and then moving on to other lesions [51,52].

CT-PA, specifically electrocardiogram-gated CT-PA, appears to outperform DSA-PA in characterizing lesion morphology, surpassing the latter in depicting webs/bands (sensitivity, 100% *vs*. 60%) and type C lesions (sensitivity, 100% *vs*. 52%) [26]. However, among all the imaging modalities described so far, CBCT-PA has proven particular utility in this regard. CBCT-PA detects more stenosis per web than CTPA, with detection rates of 22% and 7%, respectively [28]. Furthermore, the ability of CBCT-PA to provide better degrees of pulmonary vessel opacification compared to CT-PA may help differentiate associated lesions [38].

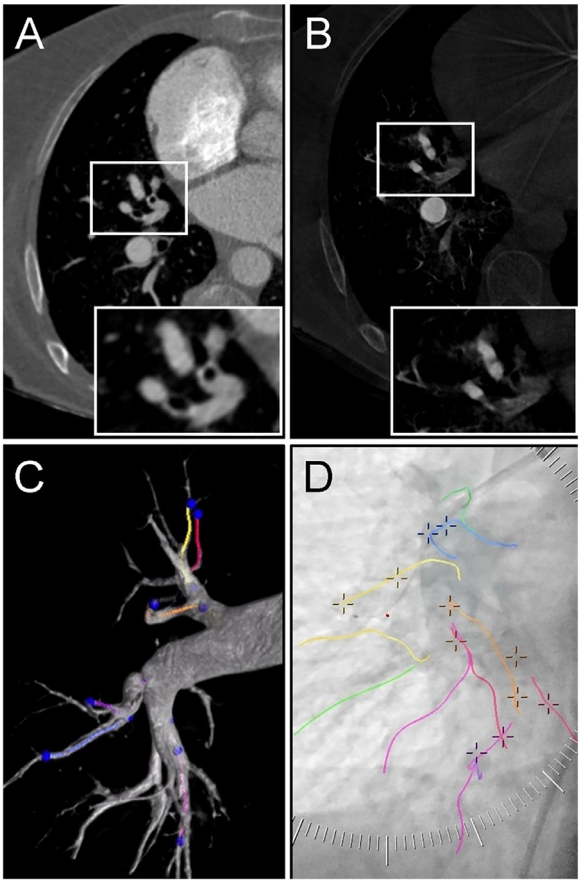

Fig. 7. Balloon pulmonary angioplasty procedural planning using cone beam CT-pulmonary angiography. 62-year-old woman with confirmed chronic thromboembolic pulmonary hypertension (CTEPH) undergoing her first balloon pulmonary angioplasty (BPA) session. (**A-B**) Chronic thromboembolic lesion (band) affecting the segmental medial branch of the middle lobe, is not fully characterizable using single-energy CT pulmonary angiography (CTPA) (**A**), but ultimately seen using cone beam CT-pulmonary angiography (CBCT-PA) (**B**), CBCTPA has the potential to better characterize distal (segmental and subsegmental) chronic thromboembolic lesions compared to CTPA and digital subtraction pulmonary angiography. (**C**) Three-dimensional reconstruction of CBCTPA was performed before starting BPA. This allows procedural planning by marking all lesions to be treated (crosses in **D**) and a road map (colored lines), which can be superimposed on the live fluoroscopic image during the procedure, as demonstrated in (**D**).

4. Balloon pulmonary angioplasty imaging findings and intervention details

4.1. Intervention

BPA is a complex intervention requiring a detailed pulmonary vascular anatomy assessment and a high-capacity angio-suite for proper performance [51]. Through femoral or jugular venous access, the main PA of the lung to be treated is catheterized with a diagnostic catheter through a 7–9Fr/65–90 cm sheath [53]. Pressures are then measured before starting the BPA session [45].

CBCT-PA and DSA-PA are then performed for procedure planning. Working angiographic projections are posteroanterior and left anterior oblique (LAO 60°) views for both lungs, which allow proper visualization of all segments (Fig. 5). Lobar and segmental catheterization is performed using a 6-Fr guiding catheter. Distal segmental and subsegmental through-lesion access is performed using 0.018–0.014″ guidewires. Semi-compliant balloons ranging from 2- to 5 mm below-rated burst pressures are used for angioplasty [45,51,54]. For thromboembolic lesions in lobar or proximal segmental branches, larger balloons can be used over 0.035″ guidewires, even up to 10 mm. After all planned dilatations have been completed, PAP measurement is repeated. Pre- and post-stenosis pressures are generally not measured [55].

Imaging surveillance using DSA-PA is mandatory before and after each angioplasty to confirm balloon location and visualize vessel dilatation and vascular complications [56]. DSA-PA acquisitions should be performed by hand at low flow to avoid reperfusion lung edema (RLE) [52]. The main goal is to improve the affected parenchyma's arterial flow and venous drainage, with the morphological outcome being secondary (Fig. 6). However, a more than 50% residual stenosis is not recommended, in which case angioplasty should be repeated with a balloon of larger caliber [55,57]. Two-dimensional perfusion angiography (2D-PA) is used to quantify tissue perfusion based on specific post-processing of DSA images by generating measured perfusion curves based on contrast flow [58]. These curves can be compared before and after BPA to assess parenchymal reperfusion (Fig. 6) [58]. Motion during image acquisition, such as breathing movements and cardiac motion in BPA, can limit the effectiveness of 2D-PA analysis [59].

4.2. Cone beam CT-pulmonary angiography use in balloon pulmonary angioplasty

Navigating the complex PA system can be challenging. Several groups have started using CBCT-PA to enhance precision during BPA [36,60,61]. Using CBCT-PA acquisition, high-resolution 3D images are generated. Using any of the commercially available emboguidance navigation systems, it is possible to mark the targeted chronic thromboembolic lesions and produce a 3D vascular road map (Fig. 7) [62]. This map can be superimposed on live fluoroscopic images, providing the operator with live guidance throughout the intervention [60,61,63].

This approach improves the efficacy and safety of the intervention. In a comparative study, Lin et al. used CBCT-PA alongside DSA-PA in BPA sessions and demonstrated several benefits compared to DSA-PA alone [60]. The number of vessels treated per session increased (5.8 vessels *vs.* 3.7 vessels), and the dose area product received by the patient (13,901 *vs.* 4682 mGy·cm^2), overall session duration (3.6 hours *vs.* 4.5 hours), and total contrast volume (225 mL *vs.* 293 mL) significantly decreased [60].

4.3. Intraprocedural imaging findings of balloon pulmonary angioplasty-related complications

Hemoptysis is the most common intraprocedural complication, reported in 5% to 15% of all sessions [15]. Predictors of complication are the type of lesion treated (type A, 8% *vs.* type D, 37%), age ≥ 75 years, high PA pressure (> 30 mm Hg), and residual PH after PEA [54,64]. The incidence of vascular injuries related to BPA is approximately 5% and include rupture with contrast material

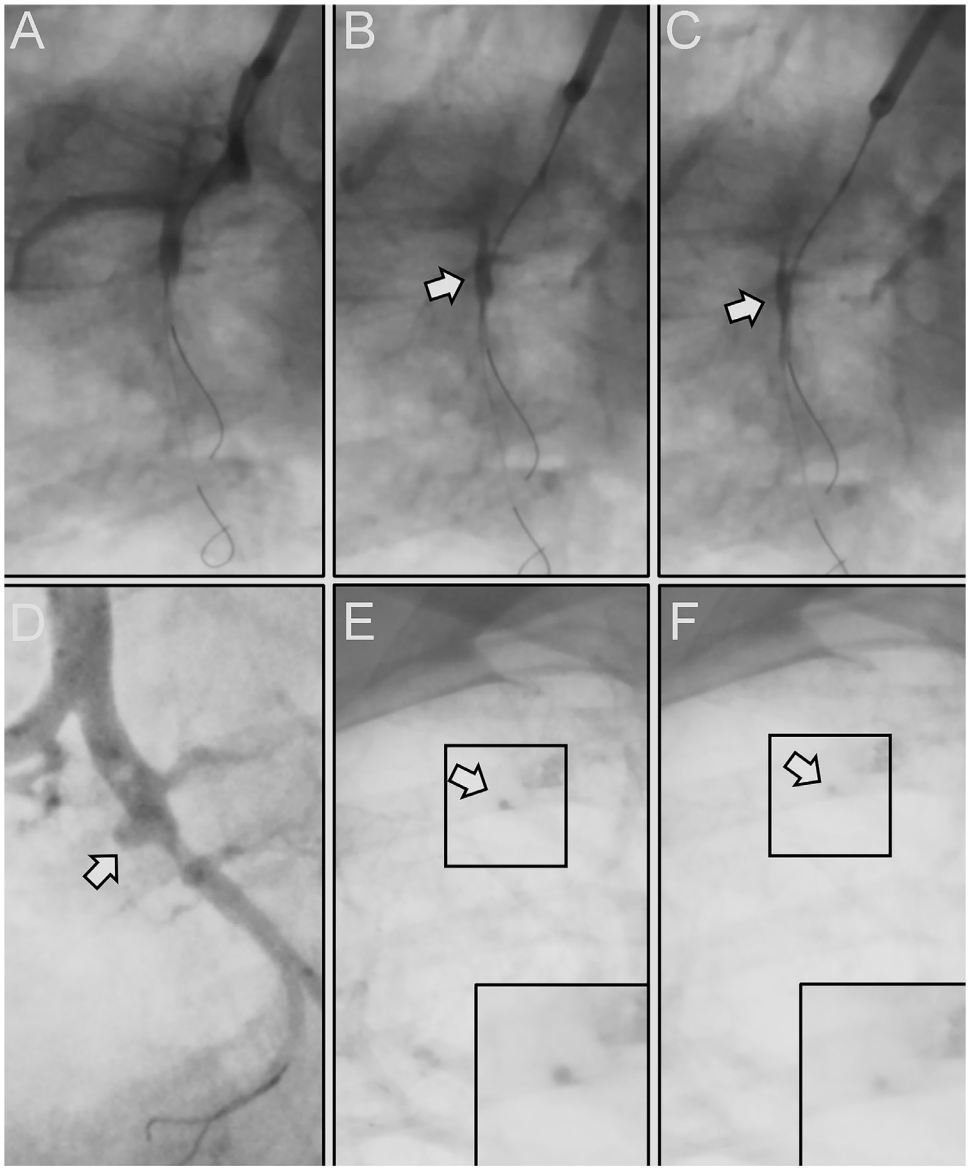

Fig. 8. Intraprocedural vascular lesions related to balloon pulmonary angioplasty. **(A-C)** 76-year-old woman with confirmed chronic thromboembolic pulmonary hypertension (CTEPH) undergoing her fourth balloon pulmonary angioplasty (BPA) session. Intraprocedural digital subtraction pulmonary angiography (DSA-PA) demonstrates a dissection of the posterobasal segmental branch of the right lower lobe. Contrast retention on the vessel lateral wall is not washed out in successive acquisitions (arrow in **B-C**). (**D**) 44-year-old man with confirmed CTEP undergoing his third BPA session. Intraprocedural DSA-PA demonstrates a pseudoaneurysm of the medial basal segmental branch of the right lower lobe (arrow). Balloon was inflated at non-nominal pressure (2 atm) for 10 minutes, with lesion resolution (not shown). (**E-F**) 34-year-old woman with confirmed CTEP undergoing her second BPA session. Minor contrast extravasation post guidewire manipulation (arrow and zoom square in **E, F**). Note the presence of retained contrast in the venous and washout angiographic runs (arrow in **F**), confirming the presence of minor bleeding.

Table 4
Imaging and clinical classification of lung injury.

Grade	Imaging definition	Clinical definition
1	No significant recognition of lung injury on CR Small subsegmental consolidation on CT	Usually asymptomatic
2	Mild or small segmental lung injury on CR.	Self-healing with only a slight increase in oxygen for a few days
3	Moderate lung injury on CR and CT More than a segment but less than a lobe	Elevated concentration of oxygen to maintain arterial saturation at optimum level
4	Moderate to severe lung injury on CR and CT Lobar lesion	Non-invasive positive pressure ventilation with high-concentration oxygen inhalation
5	Extremely severe lung injury on CR and CT More than one lobe or bilateral	Mechanical ventilation

CR indicates chest radiography; CT indicates computed tomography.

extravasation, pseudoaneurysm, and dissection. The main reasons for vascular injury include guidewire perforation, imprecise catheterization, and excessive balloon inflation [65].

Intraprocedural DSA-PA is essential to detect and treat BPA-related vascular injuries (Fig. 8) [52]. When hemoptysis occurs during the intervention, it is imperative to perform a DSA-PA on the last segment treated. Contrast extravasation with or without simultaneous clinical symptoms can be detected in up to 17–21% of BPA sessions on DSA-PA [54,64]. When a vascular lesion is detected, the first approach should be prolonged balloon inflation at the lesion site at low ATM (*e.g.*, 2 atm). If hemoptysis stops, the balloon is deflated, and a new DSA-PA is performed to confirm resolution. If DSA-PA images show no visible vascular injury, inflating the most recently used balloon to 2 atm at the site of the last BPA or guidewire manipulation is still the first recommended approach. When hemoptysis persists or the vascular lesion does not resolve after multiple attempts with this technique, other therapeutic approaches such as gelfoam/coil embolization should be considered [51,66].

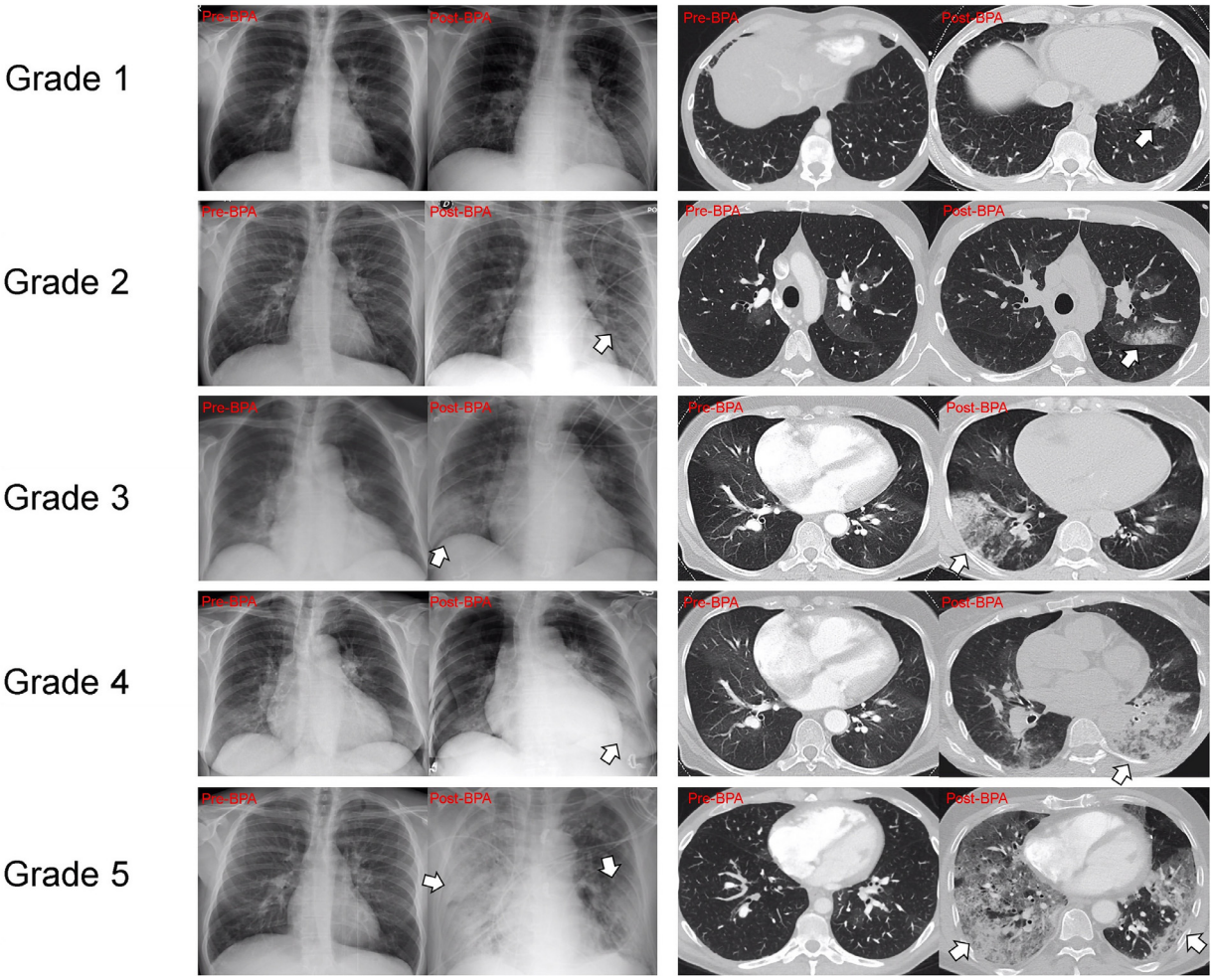

Fig. 9. Post-balloon pulmonary angioplasty lung injury grading. Chest radiograph and non-contrast CT images show the different degrees of lung injury due to balloon pulmonary angioplasty (arrows). Corresponding chest radiograph and CT images before and after the intervention can be seen for each grade: Grade 1 (very mild), with subsegmental involvement; Grade 2 (mild), with segmental involvement; Grade 3 (moderate), with involvement of more than one segment but less than one lobe; grade 4 (severe), with lobar involvement; and grade 5 (extremely severe), with involvement of more than one lobe or bilaterally.

5. Post-balloon pulmonary angioplasty imaging findings

5.1. Immediate post-balloon pulmonary angioplasty imaging findings

An uneventful immediate post-intervention period is reported in 91% to 97% of BPA sessions [15]. However, post-procedural imaging is usually performed at least once during admission. A single chest radiograph, with successive follow-ups with chest radiograph or CT are performed when an adverse event occurs. The choice of the imaging modality depends on the severity and the patient's clinical condition. Lung injury is the most frequent post-BPA complication, and its early detection is the main reason for immediate imaging follow-up. Lung injury can be caused by RLE and lung hemorrhage [52,67]. Currently, RLE and lung hemorrhage cannot be differentiated from each other on any imaging test. RLE and lung hemorrhage manifest as post-procedural lung consolidations on chest radiograph and CT. Lung injury can be classified into five stages according to severity, ranging from grade 1, which corresponds to the absence of abnormalities on chest radiograph and the presence of small subsegmental ground glass opacities on CT, passing through grade 3 (moderate), which is defined as the involvement of more than a segment but less than a lobe, to grade 5 (extremely severe), which affects more than one lobe or bilateral pulmonary involvement (Table 4; Fig. 9) [68].

Lung injury rates vary widely among series, ranging from 8% to 30% [15]. Ejiri et al. demonstrated that vascular injury is the main reason for lung injury after BPA using CT examination [64]. In this study, post post-BPA lung injuries presented as a focal infiltration at the site of BPA, suggesting that the lesions were due to vascular injury rather than to RLE that manifests as a more diffuse infiltration [64]. Using CT examination as a post-BPA imaging test, these researchers also demonstrated that the number of post-BPA lung injuries is underestimated when using chest radiograph alone as an immediate post-treatment imaging modality, with 47% of sessions with lung injury detected by CT *vs.* only 8–30% with chest radiograph [64]. Clinical management of lung injury, whether from lung hemorrhage or RLE, includes immediate stabilization, ensuring a patent airway, sufficient oxygenation, and monitoring vital signs. Diagnostic tools such as chest radiograph and CT determine the type and extent of injury. Treatments address the root cause with strategies such as diuretics for RLE, and reversal of anticoagulation for lung hemorrhage.

5.2. Imaging follow-up and assessment of long-term outcomes

A thorough clinical and pulmonary hemodynamic evaluation is performed between three and six months after completing all sessions [19,69]. The main objective of invasive treatment with BPA is to improve symptoms associated with CTEPH (dyspnea, fatigue, chest pain, exercise intolerance) and pulmonary hemodynamics to normal resting values [69–71]. Clinical evaluation includes reassessing the six-minute walking test and NYHA-FC, aiming to increase the first up to 400 meters and decrease the second to class I-II [15,51]. Tests to assess pulmonary hemodynamics are RHC and TDE. All hemodynamic values, including mPAP, PVR and cardiac output, are measured during RHC. Results are compared with pre-treatment values for assessment. The optimal hemodynamic target is also to normalize all values. TDE can also estimate PAP and provide valuable information on right ventricular function and structural abnormalities [23].

Systematic radiological re-evaluation is unnecessary when clinical and hemodynamic parameters are normalized. However, if residual CTEPH is demonstrated, a complete imaging re-evaluation is warranted. A detailed comparison of pre- and post-BPA residual thromboembolic burden and state of pulmonary perfusion is pivotal for deciding the therapeutic approach (Fig. 10). The median number of BPA sessions reported in the literature is between 4 and 6 [18,19,53,72]. However, sessions are performed until clinical and hemodynamic normalization is achieved, provided the presence of amenable lesions [45].

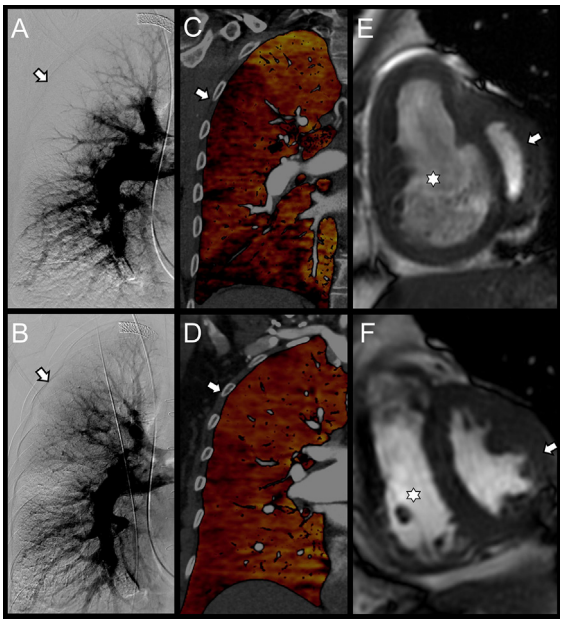

Fig. 10. Post-balloon pulmonary angioplasty imaging follow-up. (**A-D**), Pre- and six-month post-balloon pulmonary angioplasty (BPA) imaging in a 66-year-old woman with confirmed chronic thromboembolic pulmonary hypertension (CTEPH) treated with four BPA sessions due to residual disease after surgery. (**A**) Pre-BPA digital subtraction pulmonary angiography (DSA-PA) and (**C**), dual-energy CT-pulmonary angiography (DECT-PA) iodine perfusion mapping shows anterior and posterior segmental perfusion defects in the right upper lobe (arrow in **A**) and lack of lobar perfusion in the iodine map (arrow in **C**). (**B**), DSA-PA and (**D**), DECT-PA after BPA demonstrate patency of the anterior and posterior segmental branches, with restoration of pulmonary perfusion (arrows). (**E-F**), 76-year-old woman with confirmed CTEPH treated with five BPA sessions due to refusal of surgical treatment who underwent pre- and post-BPA cardiac short-axis cine SSFP MR images. (**E**), Cardiac MR image reveals severe dilatation of the right ventricle (asterisk), as well as hypertrophy of the free wall of the right ventricle and marked flattened interventricular septum (arrow). (**F**), Post-BPA cardiac MR image shows significant improvement in right ventricular dilatation (asterisk) and interventricular septal morphology (arrow).

DSA-PA remains the most widely used imaging tool to assess residual CTEPH after BPA and select patients who need further sessions [37,58]. CBCT-PA can provide more detailed information on vascular structure and obstructive lesions and assess possible significant subsegmental lesions not treated in previous sessions [38]. DECT-PA is helpful in post-BPA evaluation, with sensitivity and specificity levels of iodine mapping virtually identical to those of DSA-PA (sensitivity, 92%; specificity, 99%) and superior to those of V'/Q' SPECT (sensitivity, 85%; specificity, 99%) in this specific scenario [48,73]. Magnetic resonance imaging is a promising tool for evaluating CTEPH after invasive treatment. After BPA, lung parenchymal perfusion and cardiac Magnetic resonance imaging showed improved pulmonary blood flow in treated lobes and, to a lesser degree, in untreated lobes, correlating with changes in hemodynamics and ventricular overload (Fig. 10) [74,75]. Cardiac magnetic resonance imaging has also demonstrated a return to native myocardial T1 values and improved interventricular desynchrony [76,77].

6. Conclusion

Knowledge of imaging findings before, during, and after BPA for patients with CTEPH is essential for proper selection, planning, procedure execution, management of complications, and follow-up. The most widely used imaging modalities in this regard are DSA-PA and CT-PA. Furthermore, CBCT-PA and DECT-PA have gained ground in

the last decade and are likely to take center stage in the imaging work-up of these patients.

Human and animal rights

The authors declare that the work described has been carried out in accordance with the Declaration of Helsinki of the World Medical Association revised in 2013 for experiments involving humans as well as in accordance with the EU Directive 2010/63/EU for animal experiments.

Informed consent and patient details

The authors declare that this article does not contain any personal information that could lead to the identification of the patients.

Contribution of authors

All authors attest that they meet the current International Committee of Medical Journal Editors (ICMJE) criteria for Authorship. All coauthors were involved in inception, data collection, preparation, and editing of this manuscript. The final manuscript has been reviewed and approved for submission by all coauthors involved.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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In loving memory of Alejandro Páez-Carpio, MD.

References

[1] Humbert M, Kovacs G, Hoeper MM, Badagliacca R, Berger RMF, Brida M, et al. 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Respir J 2023;61:2200879.
[2] Hoeper MM, Madani MM, Nakanishi N, Meyer B, Cebotari S, Rubin LJ. Chronic thromboembolic pulmonary hypertension. Lancet Respir Med 2014;2:573–82.
[3] Delcroix M, Torbicki A, Gopalan D, Sitbon O, Klok FA, Lang I, et al. ERS statement on chronic thromboembolic pulmonary hypertension. Eur Respir J 2021;57:2002828.
[4] Escribano-Subias P, Blanco I, López-Meseguer M, Lopez-Guarch CJ, Roman A, Morales P, et al. Survival in pulmonary hypertension in Spain: insights from the Spanish registry. Eur Respir J 2012;40:596–603.
[5] Pepke-Zaba J, Delcroix M, Lang I, Mayer E, Jansa P, Ambroz D, et al. Chronic thromboembolic pulmonary hypertension (CTEPH): results from an international prospective registry. Circulation 2011;124:1973–81.
[6] Lang IM, Dorfmüller P, Vonk Noordegraaf A. The pathobiology of chronic thromboembolic pulmonary hypertension. Ann Am Thorac Soc 2016;13(Suppl 3):S215–21.
[7] Lang I. Chronic thromboembolic pulmonary hypertension: a distinct disease entity. Eur Respir Rev 2015;24:246–52.
[8] Delcroix M, Lang I, Pepke-Zaba J, Jansa P, D'Armini AM, Snijder R, et al. Long-term outcome of patients with chronic thromboembolic pulmonary hypertension: results from an international prospective registry. Circulation 2016;133:859–71.
[9] Riedel M, Stanek V, Widimsky J, Prerovsky I. Longterm follow-up of patients with pulmonary thromboembolism: late prognosis and evolution of hemodynamic and respiratory data. Chest 1982;81:151–8.
[10] Madani MM, Auger WR, Pretorius V, Sakakibara N, Kerr KM, Kim NH, et al. Pulmonary endarterectomy: recent changes in a single institution's experience of more than 2,700 patients. Ann Thorac Surg 2012;94:97–103.
[11] Taboada D, Pepke-Zaba J, Jenkins DP, Berman M, Treacy CM, Cannon JE, et al. Outcome of pulmonary endarterectomy in symptomatic chronic thromboembolic disease. Eur Respir J 2014;44:1635–45.
[12] Jenkins D, Madani M, Fadel E, D'Armini AM, Mayer E, et al. Pulmonary endarterectomy in the management of chronic thromboembolic pulmonary hypertension. Eur Respir Rev 2017;26:160111.
[13] Feinstein JA, Goldhaber SZ, Lock JE, Ferndandes SM, Landzberg MJ. Balloon pulmonary angioplasty for treatment of chronic thromboembolic pulmonary hypertension. Circulation 2001;103:10–3.
[14] Moriarty JM, Khan SN, Kao SD, Saggar R. Balloon pulmonary angioplasty for chronic thromboembolic pulmonary hypertension. Cardiovasc Intervent Radiol 2018;41:1826–39.
[15] Kennedy MK, Kennedy SA, Tan KT, de Perrot M, Bassett P, McInnis MC, et al. Balloon pulmonary angioplasty for chronic thromboembolic pulmonary hypertension: a systematic review and meta-analysis. Cardiovasc Intervent Radiol 2023;46:5–18.
[16] Li W, Yang T, Quan RL, Chen XX, An J, Zhao ZH, et al. Balloon pulmonary angioplasty reverse right ventricular remodelling and dysfunction in patients with inoperable chronic thromboembolic pulmonary hypertension: a systematic review and meta-analysis. Eur Radiol 2021;31:3898–908.
[17] Ito R, Yamashita J, Sasaki Y, Ikeda S, Suzuki S, Murata N, et al. Efficacy and safety of balloon pulmonary angioplasty for residual pulmonary hypertension after pulmonary endarterectomy. Int J Cardiol 2021;334:105–9.
[18] Godinas L, Bonne L, Budts W, Belge C, Leys M, Delcroix M, et al. Balloon pulmonary angioplasty for the treatment of nonoperable chronic thromboembolic pulmonary hypertension: single-center experience with low initial complication rate. J Vasc Interv Radiol 2019;30:1265–72.
[19] Brenot P, Jaïs X, Taniguchi Y, Garcia Alonso C, Gerardin B, Mussot S, et al. French experience of balloon pulmonary angioplasty for chronic thromboembolic pulmonary hypertension. Eur Respir J 2019;53:1802095.
[20] Gall H, Yogeswaran A, Fuge J, Sommer N, Grimminger F, Seeger W, et al. Validity of echocardiographic tricuspid regurgitation gradient to screen for new definition of pulmonary hypertension. E Clin Med 2021;34:100822.
[21] Tunariu N, Gibbs SJ, Win Z, Gin-Sing W, Graham A, Gishen P, et al. Ventilation-perfusion scintigraphy is more sensitive than multidetector CTPA in detecting chronic thromboembolic pulmonary disease as a treatable cause of pulmonary hypertension. J Nucl Med 2007;48:680–4.
[22] Wang M, Wu D, Ma R, Zhang Z, Zhang H, Han K, et al. Comparison of V/Q SPECT and CT angiography for the diagnosis of chronic thromboembolic pulmonary hypertension. Radiology 2020;296:420–9.
[23] Remy-Jardin M, Ryerson CJ, Schiebler ML, Leung ANC, Wild JM, Hoeper MM, et al. Imaging of pulmonary hypertension in adults: a position paper from the Fleischner Society. Radiology 2021;298:531–49.
[24] Dong C, Zhou M, Liu D, Long X, Guo T, Kong X. Diagnostic accuracy of computed tomography for chronic thromboembolic pulmonary hypertension: a systematic review and meta-analysis. PLoS ONE 2015;10:e0126985.
[25] He J, Fang W, Lv B, He JG, Xiong CM, Liu ZH, et al. Diagnosis of chronic thromboembolic pulmonary hypertension: comparison of ventilation/perfusion scanning and multidetector computed tomography pulmonary angiography with pulmonary angiography. Nucl Med Commun 2012;33:459–63.
[26] Ley S, Ley-Zaporozhan J, Pitton MB, Schneider J, Wirth GM, Mayer E, et al. Diagnostic performance of state-of-the-art imaging techniques for morphological assessment of vascular abnormalities in patients with chronic thromboembolic pulmonary hypertension (CTEPH). Eur Radiol 2012;22:607–16.
[27] Castañer E, Gallardo X, Ballesteros E, Andreu M, Pallardó Y, Mata JM, et al. CT diagnosis of chronic pulmonary thromboembolism. Radiographics 2009;29:31–50.
[28] Maschke SK, Werncke T, Becker LS, Dewald CLA, Meine TC, Olsson KM, et al. The value of C-arm computed tomography in addition to conventional digital subtraction angiography in the diagnostic work-up of patients with suspected chronic thromboembolic pulmonary hypertension: an update of 300 patients. Acad Radiol 2022;29(Suppl 2):S1–S10.
[29] Godoy MC, Heller SL, Naidich DP, Assadourian B, Leidecker C, Schmidt B, et al. Dual-energy MDCT: comparison of pulmonary artery enhancement on dedicated CT pulmonary angiography, routine and low contrast volume studies. Eur J Radiol 2011;79:e11–7.
[30] Ameli-Renani S, Rahman F, Nair A, Ramsay L, Bacon JL, Weller A, et al. Dual-energy CT for imaging of pulmonary hypertension: challenges and opportunities. Radiographics 2014;34:1769–90.
[31] Nakazawa T, Watanabe Y, Hori Y, Kiso K, Higashi M, Itoh T, et al. Lung perfused blood volume images with dual-energy computed tomography for chronic thromboembolic pulmonary hypertension: correlation to scintigraphy with single-photon emission computed tomography. J Comput Assist Tomogr 2011;35:590–5.
[32] Dournes G, Verdier D, Montaudon M, Bullier E, Rivière A, Dromer C, et al. Dual-energy CT perfusion and angiography in chronic thromboembolic pulmonary hypertension: diagnostic accuracy and concordance with radionuclide scintigraphy. Eur Radiol 2014;24:42–51.
[33] Meinel FG, Graef A, Thierfelder KM, Armbruster M, Schild C, Neurohr C, et al. Automated quantification of pulmonary perfused blood volume by dual-energy CTPA in chronic thromboembolic pulmonary hypertension. Rofo 2014;186:151–6.
[34] Masy M, Giordano J, Petyt G, Hossein-Foucher C, Duhamel A, Kyheng M, et al. Dual-energy CT (DECT) lung perfusion in pulmonary hypertension: concordance rate with V/Q scintigraphy in diagnosing chronic thromboembolic pulmonary hypertension (CTEPH). Eur Radiol 2018;28:5100–10.

[35] Si-Mohamed S, Moreau-Triby C, Tylski P, Tatard-Leitman V, Wdowik Q, Boccalini S, et al. Head-to-head comparison of lung perfusion with dual-energy CT and SPECT-CT. Diagn Interv Imaging 2020;101:299–310.
[36] Maschke SK, Hinrichs JB, Renne J, Werncke T, Winther HMB, Ringe KI, et al. C-arm computed tomography (CACT)-guided balloon pulmonary angioplasty: evaluation of patient safety and peri- and post-procedural complications. Eur Radiol 2019;29:1276–84.
[37] Hinrichs JB, Marquardt S, von Falck C, Hoeper MM, Olsson KM, Wacker FK, et al. Comparison of C-arm computed tomography and digital subtraction angiography in patients with chronic thromboembolic pulmonary hypertension. Cardiovasc Intervent Radiol 2016;39:53–63.
[38] Hinrichs JB, von Falck C, Hoeper MM, Olsson KM, Wacker FK, Meyer BC, et al. Pulmonary artery imaging in patients with chronic thromboembolic pulmonary hypertension: comparison of cone-beam CT and 64-row multidetector CT. J Vasc Interv Radiol 2016;27:361–8 e2.
[39] Greffier J, Villani N, Defez D, Dabli D, Si-Mohamed S. Spectral CT imaging: technical principles of dual-energy CT and multi-energy photon-counting CT. Diagn Interv Imaging 2023;104:167–77.
[40] Djahnine A, Lazarus C, Lederlin M, Mulé S, Wiemker R, Si-Mohamed S, et al. Detection and severity quantification of pulmonary embolism with 3D CT data using an automated deep learning-based artificial solution. Diagn Interv Imaging 2023. doi: 10.1016/j.diii.2023.09.006.
[41] Madani M, Mayer E, Fadel E, Jenkins DP. Pulmonary endarterectomy: patient selection, technical challenges, and outcomes. Ann Am Thorac Soc 2016;13:S240–7.
[42] Kim NH, Delcroix M, Jais X, Madani MM, Matsubara H, Mayer E, et al. Chronic thromboembolic pulmonary hypertension. Eur Respir J 2019;53:1801915.
[43] Ghofrani HA, Gomez Sanchez MA, Humbert M, Pittrow D, Simonneau G, Gall H, et al. Riociguat treatment in patients with chronic thromboembolic pulmonary hypertension: final safety data from the EXPERT registry. Respir Med 2021;178:106220.
[44] Wiedenroth CB, Ghofrani HA, Adameit MSD, Breithecker A, Haas M, Kriechbaum S, et al. Sequential treatment with riociguat and balloon pulmonary angioplasty for patients with inoperable chronic thromboembolic pulmonary hypertension. Pulm Circ 2018;8:2045894018783996.
[45] Rivers-Bowerman MD, Zener R, Jaberi A, de Perrot M, Granton J, Moriarty JM, et al. Balloon pulmonary angioplasty in chronic thromboembolic pulmonary hypertension: new horizons in the interventional management of pulmonary embolism. Tech Vasc Interv Radiol 2017;20:206–15.
[46] Reichelt A, Hoeper MM, Galanski M, Keberle M. Chronic thromboembolic pulmonary hypertension: evaluation with 64-detector row CT versus digital substraction angiography. Eur J Radiol 2009;71:49–54.
[47] Sugiura T, Tanabe N, Matsuura Y, Shigeta A, Kawata N, Jujo T, et al. Role of 320-slice CT imaging in the diagnostic workup of patients with chronic thromboembolic pulmonary hypertension. Chest 2013;143:1070–7.
[48] Koike H, Sueyoshi E, Sakamoto I, Uetani M, Nakata T, Maemura K. Comparative clinical and predictive value of lung perfusion blood volume CT, lung perfusion SPECT and catheter pulmonary angiography images in patients with chronic thromboembolic pulmonary hypertension before and after balloon pulmonary angioplasty. Eur Radiol 2018;28:5091–9.
[49] Le Faivre J, Duhamel A, Khung S, Faivre JB, Lamblin N, Remy J, et al. Impact of CT perfusion imaging on the assessment of peripheral chronic pulmonary thromboembolism: clinical experience in 62 patients. Eur Radiol 2016;26:4011–20.
[50] Kawakami T, Ogawa A, Miyaji K, Mizoguchi H, Shimokawahara H, Naito T, et al. Novel angiographic classification of each vascular lesion in chronic thromboembolic pulmonary hypertension based on selective angiogram and results of balloon pulmonary angioplasty. Circ Cardiovasc Interv 2016;9:e003318.
[51] Wiedenroth CB, Liebetrau C, Breithecker A, Guth S, Lautze HJ, Ortmann E, et al. Combined pulmonary endarterectomy and balloon pulmonary angioplasty in patients with chronic thromboembolic pulmonary hypertension. J Heart Lung Transplant 2016;35:591–6.
[52] Mizoguchi H, Ogawa A, Munemasa M, Mikouchi H, Ito H, Matsubara H. Refined balloon pulmonary angioplasty for inoperable patients with chronic thromboembolic pulmonary hypertension. Circ Cardiovasc Interv 2012;5:748–55.
[53] Hoole SP, Coghlan JG, Cannon JE, Taboada D, Toshner M, Sheares K, et al. Balloon pulmonary angioplasty for inoperable chronic thromboembolic pulmonary hypertension: the UK experience. Open Heart 2020;7:e001144.
[54] Ikeda N, Kubota S, Okazaki T, Iijima R, Hara H, Hiroi Y, et al. The predictors of complications in balloon pulmonary angioplasty for chronic thromboembolic pulmonary hypertension. Catheter Cardiovasc Interv 2019;93:E349–56.
[55] Kinutani H, Shinke T, Nakayama K, Taniguchi Y, Otake H, Takaya T, et al. High perfusion pressure as a predictor of reperfusion pulmonary injury after balloon pulmonary angioplasty for chronic thromboembolic pulmonary hypertension. Int J Cardiol Heart Vasc 2016;11:1–6.
[56] Coghlan JG, Rothman AM, Hoole SP. Balloon pulmonary angioplasty: state of the art. Interv Cardiol 2020;16:e02.
[57] Mahmud E, Madani MM, Kim NH, Poch D, Ang L, Behnamfar O, et al. Chronic thromboembolic pulmonary hypertension: evolving therapeutic approaches for operable and inoperable disease. J Am Coll Cardiol 2018;71:2468–86.
[58] Maschke SK, Renne J, Werncke T, Olsson KM, Hoeper MM, Wacker FK, et al. Chronic thromboembolic pulmonary hypertension: evaluation of 2D-perfusion angiography in patients who undergo balloon pulmonary angioplasty. Eur Radiol 2017;27:4264–70.
[59] Reekers JA, Koelemay MJ, Marquering HA, van Bavel ET. Functional imaging of the foot with perfusion angiography in critical limb ischemia. Cardiovasc Intervent Radiol 2016;39:183–9.
[60] Lin JL, Chen HM, Lin FC, Li JY, Xie CX, Guo WL, et al. Application of DynaCT angiographic reconstruction in balloon pulmonary angioplasty. Eur Radiol 2020;30:6950–7.
[61] Hinrichs JB, Renne J, Hoeper MM, Olsson KM, Wacker FK, Meyer BC. Balloon pulmonary angioplasty: applicability of C-arm CT for procedure guidance. Eur Radiol 2016;26:4064–71.
[62] Wallace MJ, Kuo MD, Glaiberman C, Binkert CA, Orth RC, Soulez G. Three-dimensional C-arm cone-beam CT: applications in the interventional suite. J Vasc Interv Radiol 2009;20:S523–37.
[63] Ogo T, Fukuda T, Tsuji A, Fukui S, Ueda J, Sanda Y, et al. Efficacy and safety of balloon pulmonary angioplasty for chronic thromboembolic pulmonary hypertension guided by cone-beam computed tomography and electrocardiogram-gated area detector computed tomography. Eur J Radiol 2017;89:270–6.
[64] Ejiri K, Ogawa A, Fujii S, Ito H, Matsubara H. Vascular injury is a major cause of lung injury after balloon pulmonary angioplasty in patients with chronic thromboembolic pulmonary hypertension. Circ Cardiovasc Interv 2018;11:e005884.
[65] Kitani M, Ogawa A, Sarashina T, Yamadori I, Matsubara H. Histological changes of pulmonary arteries treated by balloon pulmonary angioplasty in a patient with chronic thromboembolic pulmonary hypertension. Circ Cardiovasc Interv 2014;7:857–9.
[66] Darocha S, Pietura R, Banaszkiewicz M, Pietrasik A, Kownacki L, Torbicki A, et al. Balloon pulmonary angioplasty with stent implantation as a treatment of proximal chronic thromboembolic pulmonary hypertension. Diagnostics 2020;10:363.
[67] Kataoka M, Inami T, Hayashida K, Shimura N, Ishiguro H, Abe T, et al. Percutaneous transluminal pulmonary angioplasty for the treatment of chronic thromboembolic pulmonary hypertension. Circ Cardiovasc Interv 2012;5:756–62.
[68] Inami T, Kataoka M, Shimura N, Ishiguro H, Yanagisawa R, Taguchi H, et al. Pulmonary edema predictive scoring index (PEPSI), a new index to predict risk of reperfusion pulmonary edema and improvement of hemodynamics in percutaneous transluminal pulmonary angioplasty. JACC Cardiovasc Interv 2013;6:725–36.
[69] Ogawa A, Satoh T, Fukuda T, Sugimura K, Fukumoto Y, Emoto N, et al. Balloon pulmonary angioplasty for chronic thromboembolic pulmonary hypertension: results of a multicenter registry. Circ Cardiovasc Qual Outcomes 2017;10:e004029.
[70] Inami T, Kataoka M, Yanagisawa R, Ishiguro H, Shimura N, Fukuda K, et al. Long-term outcomes after percutaneous transluminal pulmonary angioplasty for chronic thromboembolic pulmonary hypertension. Circulation 2016;134:2030–2.
[71] Aoki T, Sugimura K, Tatebe S, Miura M, Yamamoto S, Yaoita N, et al. Comprehensive evaluation of the effectiveness and safety of balloon pulmonary angioplasty for inoperable chronic thrombo-embolic pulmonary hypertension: long-term effects and procedure-related complications. Eur Heart J 2017;38:3152–9.
[72] Olsson KM, Wiedenroth CB, Kamp JC, Breithecker A, Fuge J, Krombach GA, et al. Balloon pulmonary angioplasty for inoperable patients with chronic thromboembolic pulmonary hypertension: the initial German experience. Eur Respir J 2017;49:1602409.
[73] Martineau L, Branchu A, Boccalini S, Boussel L, Douek P, Si-Mohamed SA. Spectral dual-energy CT: a new tool to monitor lung perfusion recovery in acute pulmonary embolism after mechanical thrombectomy. Diagn Interv Imaging 2023;104:560–1.
[74] Schoenfeld C, Hinrichs JB, Olsson KM, Kuettner MA, Renne J, Kaireit T, et al. Cardio-pulmonary MRI for detection of treatment response after a single balloon pulmonary angioplasty session in chronic thromboembolic pulmonary hypertension. Eur Radiol 2019;29:1693–702.
[75] Broncano J, Bhalla S, Gutierrez FR, Vargas D, Williamson EE, Makan M, et al. Cardiac MRI in pulmonary hypertension: from magnet to bedside. Radiographics 2020;40:982–1002.
[76] Roller FC, Kriechbaum S, Breithecker A, Liebetrau C, Haas M, Schneider C, et al. Correlation of native T1 mapping with right ventricular function and pulmonary haemodynamics in patients with chronic thromboembolic pulmonary hypertension before and after balloon pulmonary angioplasty. Eur Radiol 2019;29:1565–73.
[77] Yamasaki Y, Nagao M, Abe K, Hosokawa K, Kawanami S, Kamitani T, et al. Balloon pulmonary angioplasty improves interventricular dyssynchrony in patients with inoperable chronic thromboembolic pulmonary hypertension: a cardiac MR imaging study. Int J Cardiovasc Imaging 2017;33:229–39.