Evolution of CT findings after anticoagulant treatment for acute pulmonary embolism in patients with and without an ultimate diagnosis of chronic thromboembolic pulmonary hypertension

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Abstract

Introduction The pulmonary arterial morphology of patients with pulmonary embolism (PE) is diverse and it is unclear how the different vascular lesions evolve after initiation of anticoagulant treatment. A better understanding of the evolution of computed tomography pulmonary angiography (CTPA) findings after the start of anticoagulant treatment may help to better identify those PE patients prone to develop chronic thromboembolic pulmonary hypertension (CTEPH). We aimed to assess the evolution of various thromboembolic lesions on CTPA over time after the initiation of adequate anticoagulant treatment in individual acute PE patients with and without an ultimate diagnosis of CTEPH.

Methods We analysed CTPA at diagnosis of acute PE (baseline) and at follow-up in 41 patients with CTEPH and 124 patients without an ultimate diagnosis of CTEPH, all receiving anticoagulant treatment. Central and segmental pulmonary arteries were scored by expert chest radiologists as normal or affected. Lesions were further subclassified as 1) central thrombus, 2) total thrombotic occlusion, 3) mural thrombus, 4) web or 5) tapered pulmonary artery.

Results Central thrombi resolved after anticoagulant treatment, while mural thrombi and total thrombotic occlusions either resolved or evolved into webs or tapered pulmonary arteries. Only patients with an ultimate diagnosis of CTEPH exhibited webs and tapered pulmonary arteries on the baseline scan. Moreover, such lesions always persisted after follow-up.

Conclusions Webs and tapered pulmonary arteries at the time of PE diagnosis strongly indicate a state of chronic PE and should raise awareness for possible CTEPH, particularly in patients with persistent dyspnoea after anticoagulant treatment for acute PE.

Introduction

Acute pulmonary embolism (PE) is a potentially dangerous form of venous thromboembolism and is the third most common cause of cardiovascular death [1, 2]. It is characterised by thromboembolic occlusions of one or more pulmonary arteries and requires anticoagulant treatment for at least 3 months [3]. Despite effective anticoagulant treatment, ∼1–3% of PE patients develop chronic thromboembolic pulmonary

hypertension (CTEPH) [4, 5]. This is the most severe long-term consequence of PE and, if left untreated, may ultimately lead to right-sided heart failure and death [6–8].

Signs of chronic thromboembolism are usually present on initial computed tomography pulmonary angiography (CTPA) of patients with an ultimate diagnosis of CTEPH [9–11]. However, without 3 months of adequate anticoagulant treatment a presumptive diagnosis is made of acute PE, even when chronicity is suspected. Of note, signs of chronicity are often overlooked in the setting of CTPA performed to diagnose acute PE in routine practice [12]. Webs, tapered vessels, mural thrombi and total thrombotic occlusions are generally associated with chronic thromboembolisms, while it is unknown whether these vascular lesions can dissolve after initiation of anticoagulant treatment [9–11, 13, 14]. In addition, particularly mural thrombi and total thrombotic occlusions may also be identified in acute PE patients who will not have signs of chronic thromboembolism at follow-up [15].

The pulmonary arterial morphology of CTEPH patients is diverse and it is unclear how the different vascular lesions evolve after initiation of anticoagulant treatment. A better understanding of the evolution of CTPA findings after the start of anticoagulant treatment may help to better identify those acute PE patients prone to develop CTEPH. We aimed to assess the evolution of various thromboembolic lesions on CTPA over time after the initiation of adequate anticoagulant treatment in individual acute PE patients with and without an ultimate diagnosis of CTEPH.

Methods

Study subjects

As CTEPH develops only in a small proportion of acute PE patients, we performed a case–cohort study. As cases, we selected patients with a documented history of acute PE who were diagnosed with CTEPH in the Amsterdam University Medical Center (Vrije Universiteit Amsterdam, Amsterdam, The Netherlands) between 2014 and 2019 (CTEPH@FU group). According to standard care, CTPA was repeated for the diagnostic work-up for CTEPH after the initial PE. As controls, we selected acute PE patients from a prospective multicentre cohort study (Prometheus), who were subjected to follow-up CTPA 6 months after their initial PE and who had no signs or symptoms of CTEPH in a period of 2.5 years following their initial PE (no-CTEPH@FU group) [16]. Patients were excluded if 1) they had a prior history of oral anticoagulant treatment, 2) the acute PE had been treated with reperfusion therapy, 3) anticoagulation had been discontinued before follow-up CTPA was performed or 4) follow-up CTPA had been performed <3 months after the initial CTPA. This selection resulted in a homogenous population of patients who all underwent CTPA for a first episode of acute PE and who had a follow-up CTPA performed after at least 3 months of continuous anticoagulant treatment. We combined the two prospective cohorts and analysed pulmonary vessel morphology at CTPA at diagnosis of initial PE (baseline) and at follow-up in the CTEPH@FU and no-CTEPH@FU groups. Anticoagulant treatment consisted of low-molecular-weight heparin, direct oral anticoagulants (DOACs) or vitamin K antagonists (VKAs), according to standard care [17].

In this study, CTEPH@FU patients did not fall within the scope of the Dutch Medical Research Involving Human Subjects Act, since the diagnostic procedures were performed for routine clinical purposes. This was confirmed by the Medical Ethics Review Committee of the University Medical Center of the Vrije Universiteit (2017.313). No-CTEPH@FU patients were selected from a previous prospective multicentre study. In that study, institutional ethics review boards of all participating centres approved the study protocol and all participants had provided written informed consent [16].

CTPA data acquisition and assessment

Standard CTPA was performed as described in previous studies [16, 18]. In short, CTPA was performed on multidetector row CT scanners with at least 64 slices from different vendors. CTPAs were excluded from analysis if the available reconstructed slice thickness was >3 mm or if image quality was nondiagnostic.

The presence of bronchial collaterals was defined as the presence of one or more bronchial collaterals with a diameter >2 mm [19]. The right ventricular (RV)/left ventricular (LV) ratio was measured in diastole on the transverse section by defining the widest point from inner wall to inner wall at the valvular level of each chamber. Pulmonary artery diameter was measured at the level of the pulmonary trunk.

Pulmonary arterial morphology was assessed *post hoc* as described previously [18]. In brief, 31 pulmonary arteries were evaluated in each patient, including 11 central pulmonary arteries (five mediastinal and six lobar) and 20 segmental pulmonary arteries. Each artery was scored as normal or affected and was further subclassified as 1) central thrombus, 2) total thrombotic occlusion, 3) mural thrombus, 4) web or

5) tapered pulmonary artery. Morphological pulmonary artery characteristics were presented as percentage of the total central or segmental vasculature. All thrombotic pulmonary arteries were additionally scored for the presence of calcification. Furthermore, lumen size was measured in total thrombotic occlusions that did not resolve after anticoagulant treatment. Two investigators, including at least one expert chest radiologist with specific expertise in CTEPH, reviewed the images and final judgement was achieved by consensus.

Statistical analysis

Data are presented as mean with standard deviation or as percentage of the pulmonary vasculature. All variables were tested for normal distribution by carefully assessing the mean, median and standard deviation. Data that failed the normal distribution were log or square root transformed for analysis. Comparison of characteristics between the groups was performed using the independent t-test for continuous variables and the Chi-squared test for proportions. Comparison of baseline with follow-up characteristics was performed using the dependent t-test. Differences in pulmonary arterial morphology changes over time between the groups were tested using the two-way repeated measures ANOVA test. Missing data were not imputed. Values of $p<0.05$ were considered to reflect statistical significance. Statistical analysis was performed using R version 3.6.3 (https://cran.r-project.org).

Results

Patient characteristics

165 patients with acute PE were included (figure 1). Of those, 41 patients were diagnosed with CTEPH (CTEPH@FU) and 124 patients had no signs suggestive of CTEPH during follow-up (no-CTEPH@FU). Baseline characteristics of the study patients are presented in table 1. CTEPH@FU patients were older (63±14 *versus* 54±16 years in no-CTEPH@FU; $p<0.001$), while the median (interquartile range) time between the initial CTPA and the follow-up CTPA was similar (185 (148–243) days in CTEPH@FU *versus* 184 (176–194) days in no-CTEPH@FU; p=nonsignificant). Anticoagulation treatment in CTEPH@FU patients consisted of either VKAs or DOACs, while no-CTEPH@FU patients were predominantly treated with VKAs. This could be explained by the fact that the no-CTEPH@FU group was diagnosed with acute PE before the introduction of the DOACs. All CTEPH@FU patients fulfilled the diagnostic criteria of pulmonary hypertension at follow-up and had mean±SD pulmonary arterial pressure 46±14 mmHg, pulmonary vascular resistance 679±471 dyn·s·cm^{-5} and cardiac index 2.4±0.7 L·min^{-1}·m^{-2} (table 2).

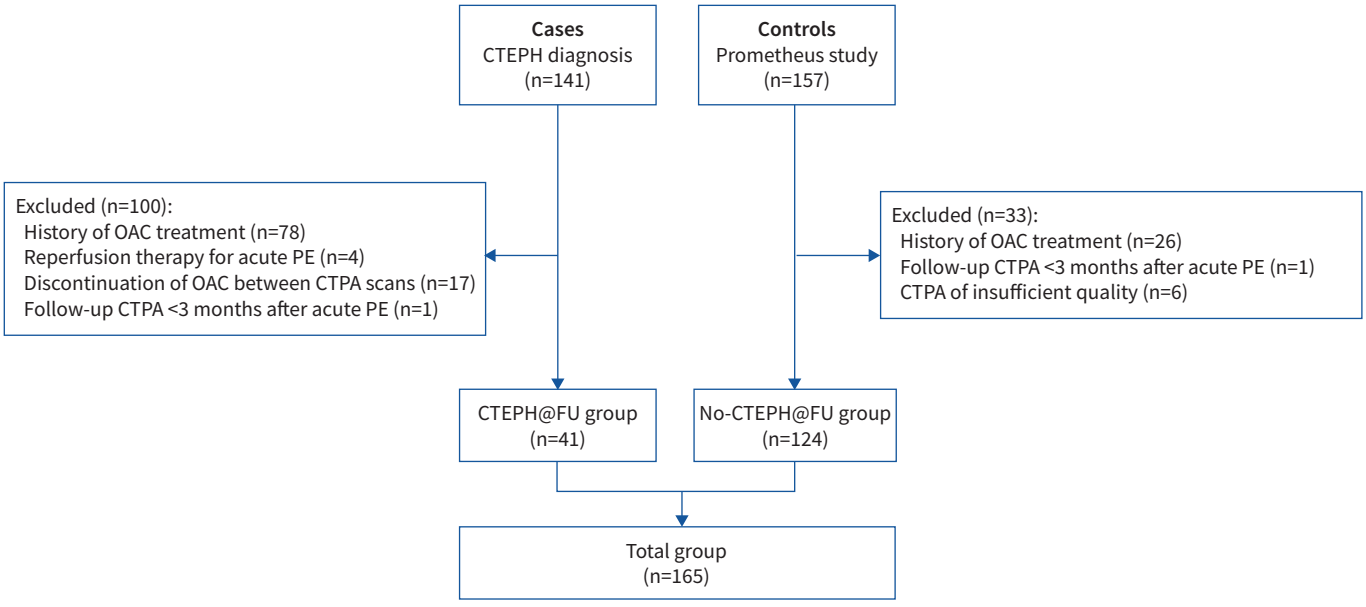

FIGURE 1 Patient flowchart. CTEPH: chronic thromboembolic pulmonary hypertension; OAC: oral anticoagulant; PE: pulmonary embolism; CTPA: computed tomography pulmonary angiography; CTEPH@FU: patients with an ultimate diagnosis of CTEPH at follow-up; no-CTEPH@FU: patients without an ultimate diagnosis of CTEPH at follow-up.

TABLE 1 Baseline characteristics

	Total (n=165)	No-CTEPH@FU (n=124)	CTEPH@FU (n=41)	p-value
Age (years)	56±16	53±16	63±14	<0.01
Male	84 (51)	62 (50)	22 (54)	NS
Anticoagulation				<0.001
VKA	137 (83)	111 (90)	26 (63)	
DOAC	15 (9)	0 (0)	15 (37)	
LMWH	13 (8)	13 (10)	0 (0)	
Anticoagulation time (days)	184 (173–199)	184 (176–194)	185 (148–243)	NS

Data are presented as mean±SD, median (interquartile range) or n (%), unless otherwise stated. CTEPH: chronic thromboembolic pulmonary hypertension; CTEPH@FU: patients with an ultimate diagnosis of CTEPH at follow-up; no-CTEPH@FU: patients without an ultimate diagnosis of CTEPH at follow-up; VKA: vitamin K antagonist; DOAC: direct oral anticoagulant; LMWH: low-molecular-weight heparin. Statistics: independent t-test for continuous variables and Chi-squared test for proportions. NS: nonsignificant.

Imaging analysis

Four segmental pulmonary arteries in a single CTEPH@FU patient could not be analysed due to breathing artefacts. As a result, a total of 451 central pulmonary arteries and 816 segmental pulmonary arteries were scored at baseline and repeatedly at follow-up in this group. In the no-CTEPH@FU group, all 1364 central pulmonary arteries and 2480 segmental pulmonary arteries were scored at baseline and at follow-up. Central and segmental pulmonary arteries were scored as normal or affected and were further subclassified as described in the Methods. Morphological pulmonary artery characteristics were presented as percentage of the total central or segmental vasculature. We observed no calcifications in thrombotic pulmonary arteries.

Bronchial collaterals could not be analysed in five out of 165 patients due to suboptimal contrast timing. Baseline scans from the remaining 160 patients were scored for the presence of bronchial collaterals (table 2). We observed more bronchial collaterals in CTEPH@FU patients compared with no-CTEPH@FU patients (50% *versus* 8%; p<0.001). In both groups, the presence of bronchial collaterals did not change after anticoagulant treatment.

The RV/LV ratio at acute PE diagnosis was higher in CTEPH@FU patients compared with no-CTEPH@FU patients (1.5±0.5 *versus* 1.0±0.3; p<0.001) and did not change after anticoagulant treatment in CTEPH@FU. In contrast, in no-CTEPH@FU patients, the RV/LV ratio decreased significantly after anticoagulant treatment (1.0±0.3 *versus* 0.9±0.2; p<0.001). Accordingly, pulmonary artery diameter at acute PE diagnosis was higher in CTEPH@FU patients compared with no-CTEPH@FU patients (34±5 *versus* 28±4 mm; p<0.001) and did not change after anticoagulant treatment in CTEPH@FU patients. In contrast, in no-CTEPH@FU patients, the pulmonary artery diameter decreased significantly after anticoagulant treatment (28±4 *versus* 26±3 mm; p<0.001).

TABLE 2 Pulmonary haemodynamics

	No-CTEPH@FU (n=124)			CTEPH@FU (n=41)		
	Baseline	Follow-up	p-value	Baseline	Follow-up	p-value
RV/LV ratio	1.0±0.3	0.9±0.2	<0.001	1.5±0.5	1.5±0.5	NS
PA diameter (mm)	28±4	26±3	<0.001	34±5	33±5	NS
Bronchial collaterals	10/122 (8)	10/122 (8)	NS	19/38 (50)	19/38 (50)	NS
mPAP (mmHg)					46±14	
PVR (dyn·s·cm^{-5})					679±471	
mRAP (mmHg)					9±4	
Cardiac index (L·min^{-1}·m^{-2})					2.4±0.7	
S_{vO_2} (%)					62±8	

Data are presented as mean±SD or n/N (%). CTEPH: chronic thromboembolic pulmonary hypertension; CTEPH@FU: patients with an ultimate diagnosis of CTEPH at follow-up; no-CTEPH@FU: patients without an ultimate diagnosis of CTEPH at follow-up; RV: right ventricular; LV: left ventricular; PA: pulmonary artery; mPAP: mean pulmonary arterial pressure; PVR: pulmonary vascular resistance; mRAP: mean right atrial pressure; S_{vO_2}: mixed venous oxygen saturation; NS: nonsignificant.

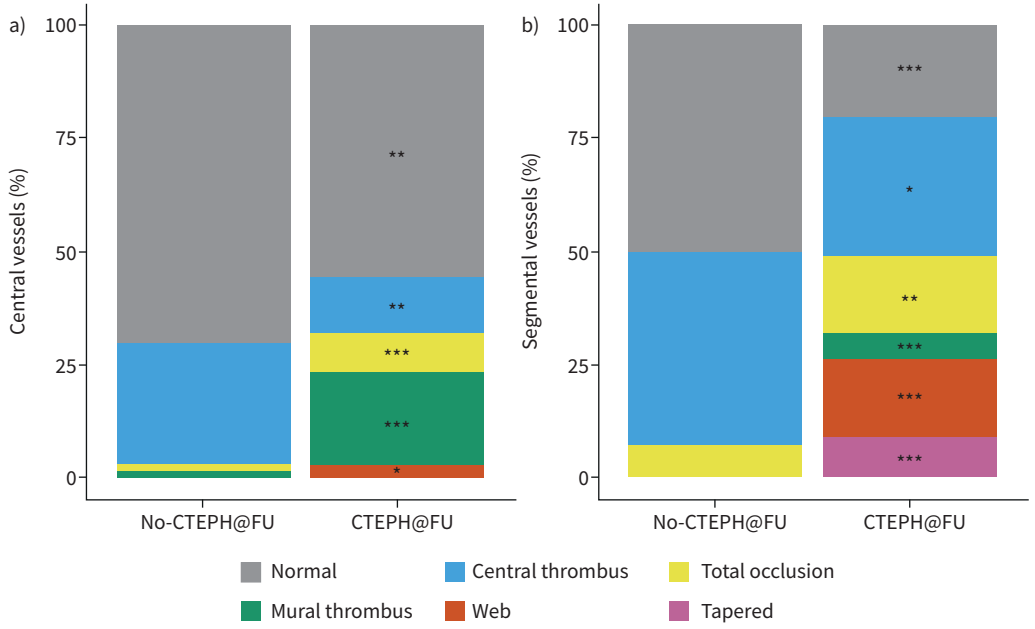

FIGURE 2 Pulmonary arterial morphology at acute pulmonary embolism diagnosis for a) central pulmonary arteries and b) segmental pulmonary arteries for patients with or without an ultimate diagnosis of chronic thromboembolic pulmonary hypertension at follow-up (CTEPH@FU and no-CTEPH@FU). Data are presented as mean percentage. Statistics for the differences between no-CTEPH@FU and CTEPH@FU: independent t-test with Bonferroni correction. *: p<0.05; **: p<0.01; ***: p<0.001.

Pulmonary arterial morphology at acute PE diagnosis

The different types of morphology of central and segmental vessels at the time of the initial acute PE diagnosis are presented in figure 2. CTEPH@FU patients had significantly fewer normal central vessels (55% *versus* 71%; p<0.01) and central thrombi (13% *versus* 27%; p<0.01) at the time of the initial PE diagnosis. In contrast, they showed more total thrombotic occlusions (9% *versus* 2%; p<0.001), mural thrombi (21% *versus* 1%; p<0.001) and webs (3% *versus* 0%; p<0.05) in the central vessels. No tapered pulmonary arteries were observed in the central pulmonary arteries.

Regarding the morphology of segmental vessels at the time of the initial acute PE, CTEPH@FU patients also had significantly fewer normal vessels (20% *versus* 50%; p<0.001) and central thrombi (31% *versus* 42%; p<0.05). In contrast, CTEPH@FU patients had more total thrombotic occlusions (17% *versus* 7%; p<0.01), mural thrombi (5% *versus* <1%; p<0.001), webs (18% *versus* <1%; p<0.001) and tapered pulmonary arteries (9% *versus* 0%; p<0.001).

Central pulmonary arterial morphology after anticoagulant treatment

Changes in central pulmonary morphology after anticoagulant treatment are presented in figure 3a and supplementary table S1. Although both groups showed a significant improvement in the percentage of affected vessels, significantly more vessels remained affected after follow-up in the CTEPH@FU group (24% *versus* 1% in no-CTEPH@FU; p<0.001). In both groups, almost all central thrombi had resolved after follow-up. In addition, the number of total thrombotic occlusions and mural thrombi significantly decreased after oral anticoagulant treatment in both groups ($p_{interaction}$=nonsignificant). However, CTEPH@FU patients had a higher number of residual total thrombotic occlusions (4% *versus* <1%; p<0.01) and mural thrombi (15% *versus* <1%; p<0.001) at follow-up. Although residual total thrombotic occlusions were observed at follow-up, the lumen size of these total occlusions had decreased (13.1±5.1 *versus* 11.8±4.1 mm; p<0.001). The number of webs at follow-up was higher in CTEPH@FU patients (5% *versus* <1% in no-CTEPH@FU; p<0.01) and these webs originated from webs already present at baseline or from central thrombi, total thrombotic occlusions and mural thrombi. None of the central pulmonary arteries were tapered.

Segmental pulmonary arterial morphology after anticoagulant treatment

Changes in segmental pulmonary morphology after anticoagulant treatment are presented in figure 3b and supplementary table S1. In line with findings in the central pulmonary vasculature, both groups showed a

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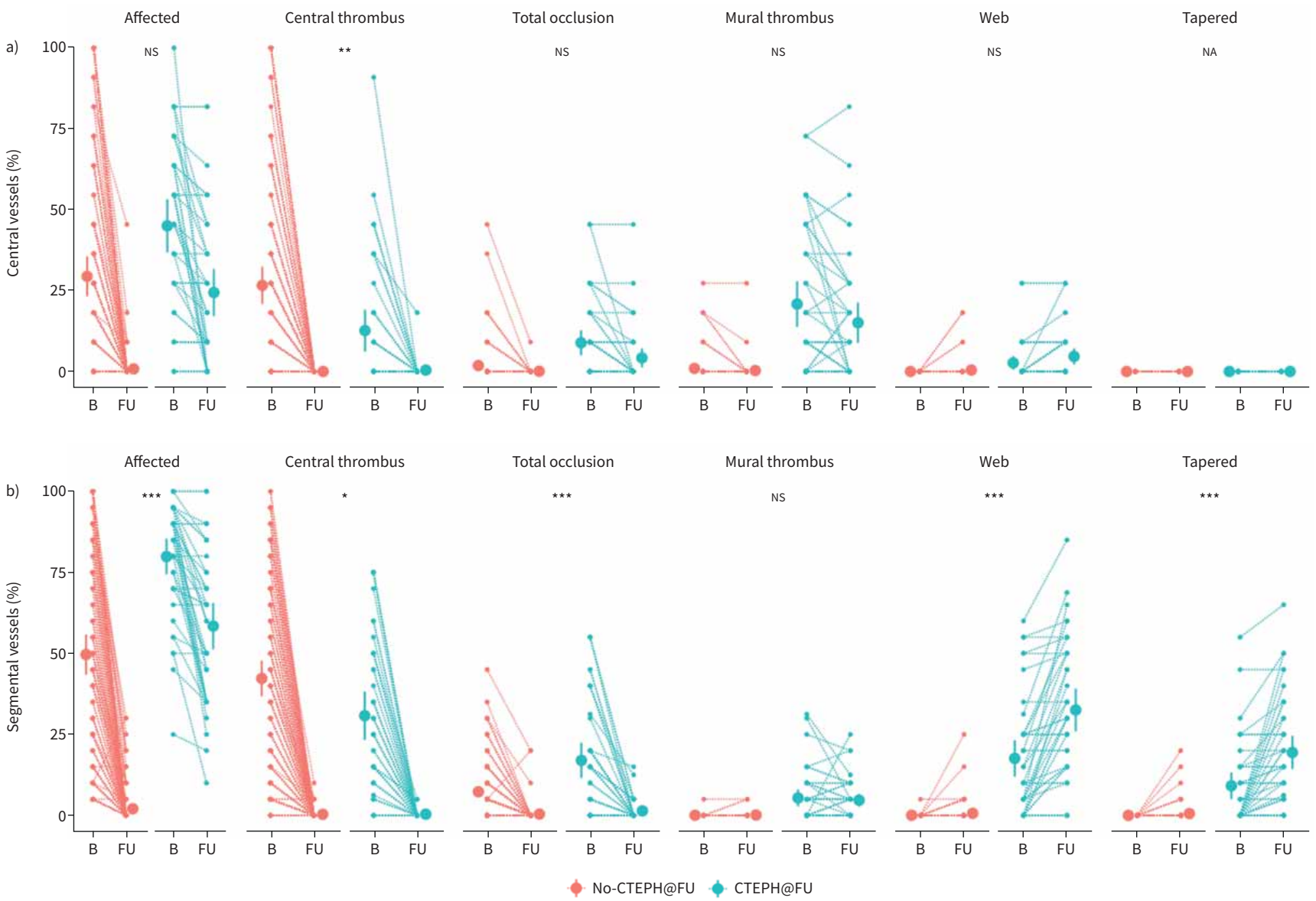

FIGURE 3 Effect of anticoagulant treatment on a) central pulmonary and b) segmental pulmonary arterial morphology for patients with or without an ultimate diagnosis of chronic thromboembolic pulmonary hypertension at follow-up (CTEPH@FU and no-CTEPH@FU). Large circles and error bars indicate mean±SE percentage. B: baseline; FU: follow-up. Statistics: two-way repeated measures ANOVA. *: p<0.05; **: p<0.01; ***: p<0.001; NS: nonsignificant; NA: not applicable.

decrease in percentage of affected vessels. However, more affected vessels persisted after follow-up in CTEPH@FU patients (58% *versus* 2%; p<0.001). After follow-up, almost no central thrombi (<1%) or total thrombotic occlusions (<1%) had persisted, regardless of group. In addition, the lumen size of residual total thrombotic occlusions at follow-up decreased (6.3±2.5 *versus* 5.6±2.1 mm at follow-up; p<0.05). However, mural thrombi did not change their morphology and were mainly observed in CTEPH@FU patients at follow-up (5% *versus* <1%; p<0.001). Remarkably, none of the webs or tapered pulmonary arteries observed at baseline in CTEPH@FU patients had resolved after follow-up (figure 4). In fact, the total number of webs and tapered pulmonary arteries increased in CTEPH@FU patients despite

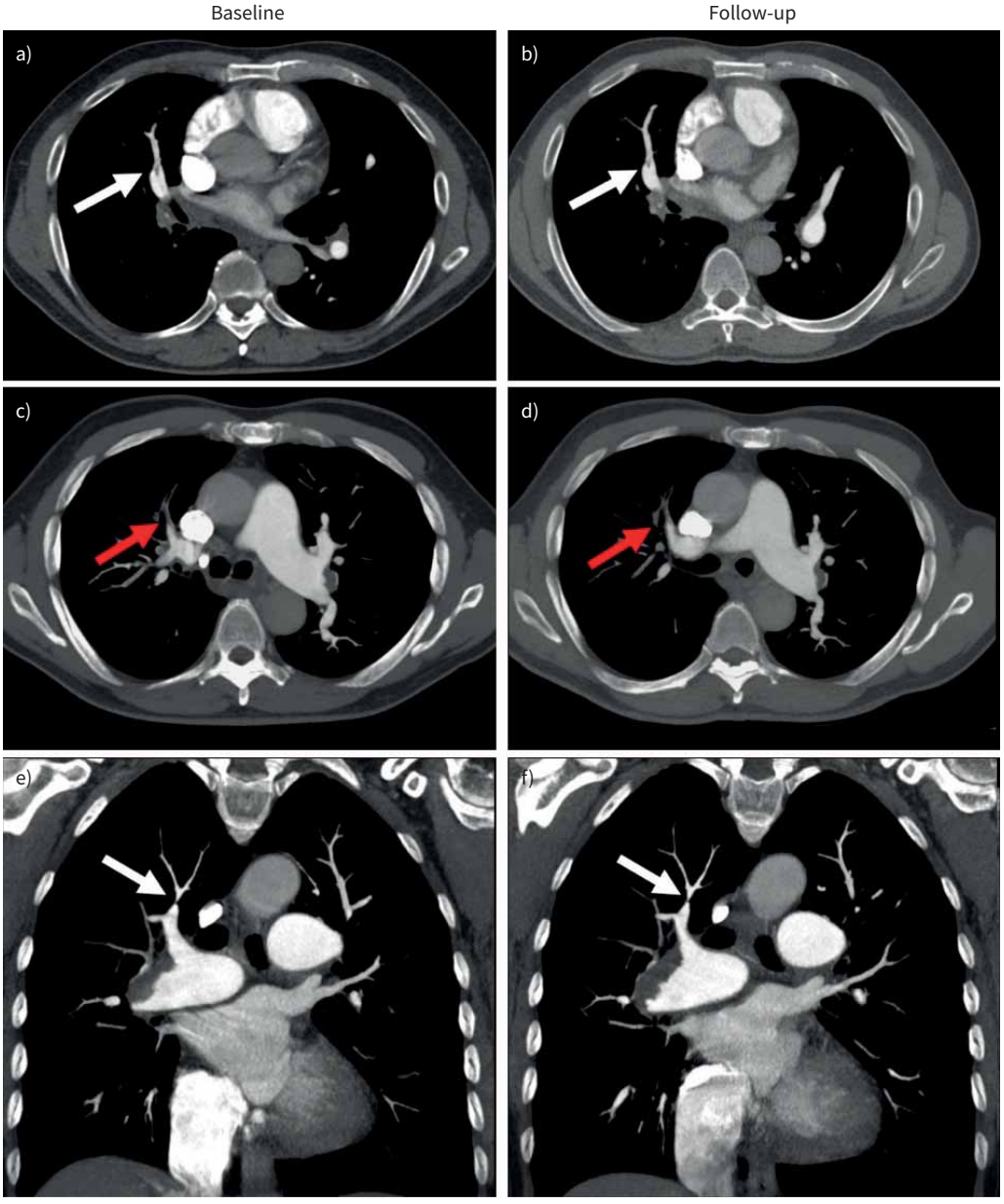

FIGURE 4 Webs and tapered pulmonary arteries did not change morphology after anticoagulant treatment. Computed tomography pulmonary angiography of a chronic thromboembolic pulmonary hypertension patient showing a) a web in the right middle lobe medial segmental pulmonary artery at baseline (white arrow) that was b) unchanged after anticoagulant treatment at follow-up; c) a tapered pulmonary artery detected in the right anterior segmental pulmonary artery at baseline (red arrow) that was d) unchanged after anticoagulant treatment at follow-up; and e) a web detected in the right posterior segmental pulmonary artery at baseline (white arrow) that was f) unchanged after anticoagulant treatment at follow-up.

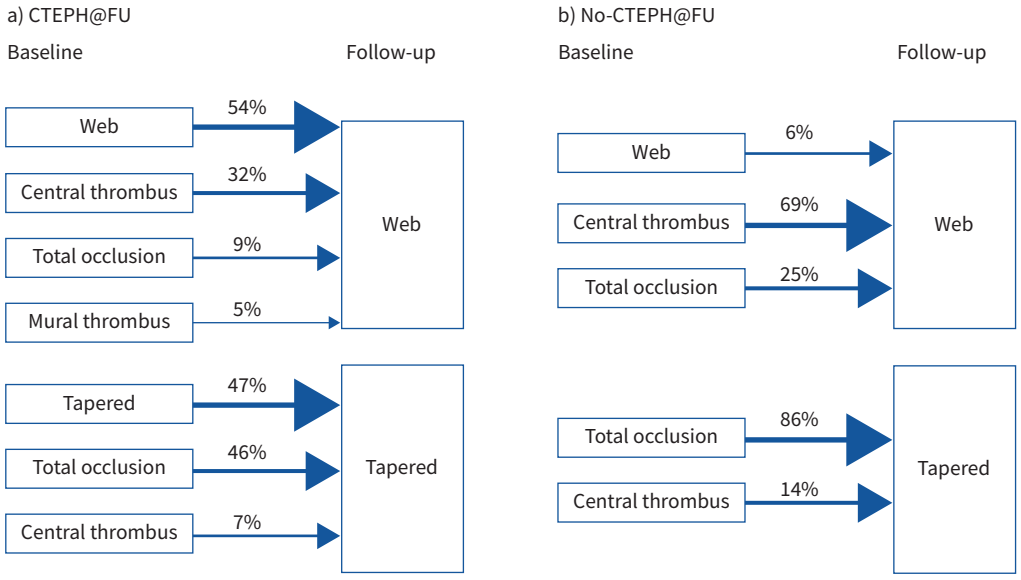

FIGURE 5 Origin of detected webs and tapered pulmonary arteries at follow-up for patients with or without an ultimate diagnosis of chronic thromboembolic pulmonary hypertension at follow-up: a) CTEPH@FU and b) no-CTEPH@FU. Data are presented as mean percentage at follow-up.

anticoagulant treatment (18% to 33% and 9% to 19%, respectively; both p<0.001). Figure 5 represents the origin of webs and tapered vessels detected at follow-up. Webs at follow-up originated either from webs already present at baseline (54% in CTEPH@FU and 6% in no-CTEPH@FU) or from central thrombi (32% in CTEPH@FU and 69% in no-CTEPH@FU), total thrombotic occlusions (9% in CTEPH@FU and 25% in no-CTEPH@FU) and mural thrombi (5% in CTEPH@FU and 0% in no-CTEPH@FU). Tapered pulmonary arteries at follow-up were either already present at baseline (47% CTEPH@FU and 0% no-CTEPH@FU) or evolved from central thrombi (7% CTEPH@FU and 14% no-CTEPH@FU) and total thrombotic occlusions (46% CTEPH@FU and 86% no-CTEPH@FU).

Discussion

To the best of our knowledge, this is the first study describing the morphological changes in pulmonary arteries after anticoagulation therapy for acute PE in patients with and without an ultimate diagnosis of CTEPH. Our study demonstrates that: 1) central thrombi usually resolve during anticoagulant treatment for PE, while mural thrombi and total thrombotic occlusions either resolve or evolve into a web or tapered pulmonary artery; 2) webs and tapered pulmonary arteries observed at baseline do not resolve after anticoagulant treatment; 3) more webs and tapered pulmonary arteries are observed at follow-up than at baseline, because a proportion of central thrombi and total thrombotic occlusions evolve into webs and tapered pulmonary arteries; and 4) patients who later develop CTEPH present with more total thrombotic occlusions, mural thrombi, webs and tapered pulmonary arteries at their initial CTPA than patients who do not develop CTEPH.

CTEPH patients have a different pulmonary arterial morphology at acute PE

Little is known about the morphological evolution of acute and chronic PE over time under the influence of anticoagulant treatment. Previous studies reported on the prediction of CTEPH on CTPA at the time of the initial PE. We previously showed that the presence of webs and tapered pulmonary arteries at the initial PE are the only independent predictors for CTEPH [9]. In an attempt to find additional CTPA biomarkers predicting CTEPH, a recent study excluded all PE CTPAs with webs and tapered pulmonary arteries, and observed that total thrombotic occlusions were also predictive of CTEPH [11]. These findings are in line with our observations, as we found that patients who develop CTEPH have more mural thrombi, total thrombotic occlusions, webs and tapered pulmonary arteries already at the time of their first PE diagnosis. What our study adds to the current literature is a detailed description of the evolution of specific types of vascular morphologies in individual patients from PE diagnosis to 6–7 months of follow-up after initiation of anticoagulant treatment. Our analysis may lead to a better and earlier identification of patients at risk for CTEPH.

Only webs and tapered pulmonary arteries indicate chronic PE

Although chronic PE lesions were mainly observed in the CTEPH@FU group, we also observed some chronic lesions in the no-CTEPH@FU group after anticoagulant treatment. This is in line with our previous studies, where we observed that 16% of all acute PE patients have chronic PE after 6 months of anticoagulant treatment [16]. Chronic PE lesions in CTEPH are thought to result from incomplete clot resolution after acute PE [20]. However, ∼25% of CTEPH patients do not have a history of documented acute PE [21]. This begs the question whether CTEPH is a true long-term complication of acute PE. Alternative explanations for the origin of CTEPH include one or more episodes of "clinically silent PE" and *in situ* thrombosis [22]. In accordance with our observations, it was previously suggested that the chronic PE lesions in CTEPH are often already present at the time of the presumptive first episode of acute PE [4, 10]. In these studies, chronic PE was deemed proven when webs, tapered pulmonary arteries, mural thrombi and total thrombotic occlusions were observed. Webs and tapered vessels are usually associated with chronic PE, central thrombi are regarded to indicate acute PE, whereas mural thrombi and total thrombotic occlusions have been described to occur in acute as well as chronic PE [13, 23]. By analysing the natural evolution of CTPA findings in individual patients, our study demonstrated that webs and tapered pulmonary arteries do not resolve after anticoagulation treatment and sometimes arise from other vascular lesions. Therefore, webs and tapered pulmonary arteries always indicate chronic PE. We also demonstrated that while some mural thrombi and total occlusions persist after anticoagulant treatment, others do resolve after anticoagulant treatment. Therefore, mural thrombi and total thrombotic occlusions cannot be exclusively categorised as chronic lesions.

Strengths and limitations

Strengths of our study are the unique dataset and design leading to a better understanding of the evolution of acute and chronic PE after anticoagulant treatment.

Ideally, a prospective study in all acute PE patients would have avoided a potential selection bias. However, since only 1–3% of acute PE patients develop CTEPH [4], an enormous number of acute PE patients would need a follow-up CTPA to include sufficient cases of CTEPH, which is practically unfeasible. Therefore, we decided to perform a case–cohort study, resulting in a homogenous group of acute PE patients with a follow-up CTPA performed after continuous anticoagulant treatment, resulting in patients with and without CTEPH at follow-up. The no-CTEPH@FU group was selected earlier in time compared with the CTEPH@FU group. Due to changes in the guidelines for acute PE treatment, more patients were treated with VKAs in the no-CTEPH@FU group [3, 24]. However, we assume that different oral anticoagulant treatment would not affect pulmonary vascular morphology differently, as DOACs are shown to be noninferior to VKAs in the treatment of acute PE [25].

The no-CTEPH@FU group did not undergo right heart catheterisation to exclude CTEPH. In this group, the presence of CTEPH was considered unlikely during the 2.5 years following initial PE based on either the absence of persistent dyspnoea or the absence of signs of CTEPH on echocardiography or ventilation/perfusion scanning [16].

Clinical implications

The presence of webs and tapered pulmonary arteries after at least 3 months of anticoagulant treatment is one of the diagnostic criteria for CTEPH according to current guidelines. With this study we demonstrate that webs and tapered pulmonary arteries do not resolve after anticoagulant treatment for acute PE. Therefore, in patients who present with persistent dyspnoea after PE and who had webs and tapered pulmonary arteries at the diagnostic CTPA, careful attention for the presence of CTEPH is required.

Conclusions

During anticoagulant treatment for acute PE almost all central thrombi completely resolved, whereas mural thrombi and total thrombotic occlusions either resolved or evolved into a web or tapered pulmonary artery. Interestingly, none of the webs and tapered pulmonary arteries resolved after anticoagulant treatment. Therefore, webs and tapered pulmonary arteries at the time of PE diagnosis strongly indicate a state of chronic PE and should raise awareness for possible CTEPH, particularly in patients with persistent dyspnoea after anticoagulant treatment for acute PE.

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