Assessment of Severity in Chronic Thromboembolic Pulmonary Hypertension by Quantitative Parameters of Dual-Energy Computed Tomography

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Objective: The objective of this study was to assess the correlation between dual-energy computed tomography (CT) and hemodynamics in patients with chronic thromboembolic pulmonary hypertension.

Methods: Dual-energy computed tomography of 52 chronic thromboembolic pulmonary hypertension patients were evaluated retrospectively. The mean lung perfused blood volume (lung PBV) and the mean pulmonary artery (PA) enhancement measured at pulmonary parenchymal phase were compared with the hemodynamics by Spearman rank correlation coefficient (rs) and receiver operating characteristic analysis.

Results: Lung PBV was correlated with mean pulmonary arterial pressure (r = 0.47, P < 0.001). Pulmonary artery enhancement was correlated with cardiac index (r = -0.49, P < 0.001) and pulmonary vascular resistance (r = 0.48, P < 0.001). The areas under the curves were 0.86 for lung PBV enhancement to predict pulmonary vascular resistance of >1000 dyne·s/cm5. The areas under the curves were 0.86 for lung PBV enhancement to predict mean pulmonary arterial pressure of >50 mm Hg and 0.86 for PA enhancement to predict pulmonary vascular resistance of >1000 dyne·s/cm5.

Conclusions: Lung PBV and PA enhancement could be indicators of hemodynamics.

Key Words: Dual-energy computed tomography, chronic thromboembolic pulmonary hypertension, hemodynamics

ORIGINAL ARTICLE

MATERIALS AND METHODS

Patients

This retrospective study was approved by the institutional review board of Nagoya University Hospital (approval number 2017-0291) with waivers of informed consent from all participants. From April 2014 to July 2017, 58 consecutive patients who underwent DE-CT for the detailed examination or follow-up of CTEPH were treated at our institution. The diagnosis of CTEPH was confirmed by ventilation/perfusion scintigraphy, RHC, and pulmonary arteriography. Patients who underwent pulmonary endarterectomy or balloon pulmonary angioplasty were not included in this study, because of the various time intervals between CT and RHC that were performed before or after treatments. If multiple examinations were performed on a patient during the study period, only the first DE-CT examination was analyzed. This is because multiple examinations become a confounding factor to evaluate CT and RHC parameters. Three patients were excluded because of the presence of pulmonary disease (interstitial pneumonia, atypical mycobacterial disease, or emphysema). One patient was excluded because contrast-enhanced computed tomography (CT) based on the findings of mismatched lung perfusion using pulmonary ventilation/perfusion scintigraphy.

The contrast-enhanced CT findings of CTEPH in the PA are a complete obstruction or partial filling defects such as vessel narrowing, intimal irregularities, and bands and webs caused by chronic organized blood clots. In addition, the dilatation of the main PA diameter is known to be associated with PH. Therefore, the diameter ratio of the main PA to the ascending aorta (rPA) measured on a CT image is widely used as a noninvasive diagnostic method for PH. The rPA is also known to correlate with the mPAP. Recently, dual-energy computed tomography (DE-CT) has enabled us to analyze lung perfused blood volume (lung PBV) and is able to generate an iodine map image similar to that obtained by pulmonary perfusion scintigraphy. Its diagnostic utility for the evaluation of pulmonary perfusion in CTEPH has been reported to be equivalent to pulmonary perfusion scintigraphy.

Right heart catheterization (RHC) is invasive but essential to evaluate the hemodynamics of CTEPH. The mPAP and pulmonary vascular resistance (PVR) obtained by RHC is one of the prognostic factors in CTEPH patients. Therefore, it may be useful to identify high-risk patients by noninvasive CT examination. A few studies reported that quantitative or visual assessment of lung PBV significantly correlated with clinical parameters such as mPAP, PVR, or mosaic attenuation pattern in CTEPH patients. It should be noted however that a correlation between quantitative evaluation using DE-CT and hemodynamic severity has not been demonstrated. Therefore, the purpose of this study was to consider whether contrast-enhanced DE-CT could be used to assess the severity of CTEPH.
the cardiac output (CO) was potentially reduced by a large pericar
dial effusion. Two patients who were previously diagnosed with
CTEPH but did not meet the PH criteria during follow-up were
also excluded. The remaining 52 patients (20 male and 32 female;
median age, 65.5 years) were evaluated retrospectively.

Dual-energy computed tomography and RHC were performed
within 6 months (median, 29.5 days; range, 15–160 days) of each
other. There was one case where the patient started anticoagula-
tion therapy between DE-CT and RHC.

CT Acquisition Protocol

Data acquisition was performed using a dual-source CT
(Somatom Definition Flash; Siemens Healthcare, Forchheim,
Germany) with the following CT scan protocol: tube voltage, 80 kV
and 140 kV (Sn); collimation, 64 × 0.6 mm; gantry rotation speed,
0.33 seconds; helical pitch, 0.65; caudocranial scan direction; and
automatic tube current modulation (CARE dose 4D; Siemens
Healthcare, Forchheim, Germany). The radiation doses (CT dose
index and dose-length product) were recorded for each examina-
tion. Contrast medium was administered into the right antecubital
vein according to the following criteria based on weight: less than
40 kg, 80 mL at a rate of 3.3 mL/s, 300 mg iodine/mL iopromide
(Proscope 300; Alfresa Pharma, Osaka, Japan); 40 to 55 kg, 96 mL
at a rate of 4 mL/sec, 320 mg iodine/mL ioversol (Optiray 320;
Guerbet Japan, Tokyo, Japan); and 55 kg or more, 96 mL at a rate
of 4 mL/s, 370 mg iodine/mL iopamidol (Iopamiron 370; Bayer
Healthcare, Tokyo, Japan), followed by 20 mL saline at 4 mL/s
using a dual-head power injector (Dual Shot GX7; Nemoto Kyorindo,
Tokyo, Japan). Scans were started using a bolus-tracking method
6 seconds after a threshold of 80 Hounsfield units (HU) in the as-
cending aorta was attained. We defined this timing as the pulmo-
nary parenchymal phase, which provided adequate contrast
enhancement from the PA to the pulmonary vein and allowed vi-
ualization of the peripheral PA. In our institution, this scan phase
was used to evaluate CTEPH. Both 80 kV and 140 kV (Sn) im-
ages were reconstructed with a medium soft convolution kernel
(D30f) at 1-mm slice thickness and 1-mm increments.

Image Analysis

The iodine-enhanced images called lung PBV were generated
by the 80 kV and 140 kV (Sn) data sets from the application based
on a 3-material decomposition method. The lung PBV values were
measured using software supplied with DE-CT system (syngo CT
Workplace, lung PBV; Siemens Healthcare, Forchheim, Germany).
The lung PBV value was defined with the following parameters:
Hounsfield scale, air of −1000 HU on both 80 kV and 140 kV
(Sn) data sets, soft tissue of 60 HU on 80 kV and 55 HU on
140 kV (Sn); contrast medium ratio, 3.01; analysis range,
−930
HU to −600 HU; and smoothing process range, 6. Bilateral lungs
were divided into 3 zones (upper, middle, and lower), and the mean
lung PBV values were calculated, except in the right upper zone to
avoid artifacts caused by the presence of the iodine contrast agent in
the superior vena cava and the subclavian vein (Figs. 1A, B). Whole
lung PBV values were also calculated. The mean PA enhancements
were measured by placing a circular region of interest (ROI) in the
pulmonary trunk (Fig. 1C) to evaluate right heart function. The di-
ameter of the PA and the ascending aorta was measured at the level
of the bifurcation of the pulmonary trunk to calculate the diameter
TABLE 1. Patient Characteristics and Measurements

| Parameters | Lung PBV | W-Lung PBV | PA Enhancement | rPA | Body Weight (Contrast Medium) |
|------------|---------|------------|----------------|-----|-----------------------------|
| BMI, kg/m² | 23.8 (16.1–42.1) | 17.8 (16.1–19.4) | 20.4 (16.2–25.5) | 25.4 (18.7–42.1) | NA |
| WHO-FC (II, III, IV) | 21, 28, 3 | 0, 2, 0 | 7, 10, 0 | 14, 16, 3 | 0.411* |
| Age, y | 65.5 (21–80) | 69 (69) | 70 (52–78) | 60 (21–80) | 0.273 |
| BMI, kg/m² | 23.8 (16.1–42.1) | 17.8 (16.1–19.4) | 20.4 (16.2–25.5) | 25.4 (18.7–42.1) | <0.001 |
| WHO-FC (II, III, IV) | 21, 28, 3 | 0, 2, 0 | 7, 10, 0 | 14, 16, 3 | 0.411* |
| Type (central/distal) | 28/24 | 20 | 9/8 | 17/16 | 0.924* |
| mPAP, mm Hg | 42.5 (23–66) | 38 (37–39) | 40 (23–66) | 43 (25–62) | 0.954 |
| sRVP, mm Hg | 73.5 (27–129) | 70.5 (69–72) | 78 (42–129) | 73 (27–95) | 0.644 |
| RAP, mm Hg | 6 (1–16) | 3.5 (2–5) | 5 (1–12) | 7 (1–16) | 0.111 |
| CO, L/min | 4.31 (2.48–9.62) | 3.90 (3.05–4.74) | 4.01 (2.48–9.62) | 4.32 (3.01–7.22) | 0.557 |
| Cardiac index, L/min/m² | 2.55 (1.61–5.80) | 3.00 (2.31–3.68) | 2.68 (1.61–5.80) | 2.43 (1.89–3.69) | 0.222 |
| PVR, dyn·s/cm⁵ | 576 (166–1676) | 667 (573–760) | 581 (166–1676) | 559 (166–1115) | 0.679 |
| Lung PBV, HU | 36.3 (24.0–55.2) | 29.2 (26.8–31.6) | 35.8 (30.4–41.8) | 38.6 (24.0–55.2) | 0.459 |
| W-lung PBV, HU | 36 (24–55) | 27.5 (24–31) | 35 (30–40) | 39 (25–55) | 0.091 |
| PA enhancement, HU | 554 (340–898) | 527 (488–565) | 593 (350–898) | 545 (340–837) | 0.538 |
| ao enhancement, HU | 443 (281–621) | 378 (364–393) | 452 (281–607) | 442 (289–621) | 0.624 |
| PA-Ao enhancement, HU | 116 (–18.3–366) | 148 (94.9–200) | 122 (–18.3 to 291) | 114 (–2 to 366) | 0.545 |
| Scan timing, s | 24.0 (16.0–35.0) | 22.0 (16.0–28.0) | 23.0 (18.2–30.3) | 24.0 (19.0–35.0) | 0.132 |
| rPA | 1.09 (0.73–2.00) | 1.10 (1.03–1.17) | 1.05 (0.73–1.53) | 1.10 (0.82–2.00) | 0.229 |
| CTDI vol, mGy | 10.8 (6.50–16.2) | 7.86 (7.68–8.03) | 9.29 (6.50–11.4) | 11.7 (6.76–16.2) | <0.001 |
| DLP, mGy·cm | 390 (220–684) | 270 (263–276) | 333 (220–401) | 427 (283–684) | <0.001 |

*x² test.

Ao indicates ascending aorta; BMI, body mass index; CTDI vol, CT dose index volume; DLP, dose length product; mGy, mg iodine/mL; NA, not applicable; WHO-FC, World Health Organization functional classification; w-lung, whole lung.

TABLE 2. Correlation Between DE-CT Parameters and Body Weight With Hemodynamics

| Parameters | Lung PBV | W-Lung PBV | PA Enhancement | rPA | Body Weight |
|------------|---------|------------|----------------|-----|-------------|
| mPAP | 0.47 (0.23–0.66) | 0.35 (0.09–0.57) | 0.20 (0.07 to 0.45) | 0.07 (0.21 to 0.34) | −0.13 (0.38 to 0.15) |
| sRVP | 0.44 (0.19–0.63) | 0.3 (0.03–0.53) | 0.14 (0.13 to 0.40) | 0.08 (0.27 to 0.28) | 0.37 (0.47 to 0.05) |
| RAP | 0.32 (0.05–0.54) | 0.34 (0.08–0.56) | −0.09 (0.35 to 0.19) | 0.17 (0.11 to 0.42) | 0.42 (0.16–0.62) |
| CO | 0.22 (0.02–0.42) | 0.03 (0.01–0.13) | −0.59 (0.35–0.19) | 0.533 (0.229–0.08) | 0.002 (0.11–0.58) |
| Cardiac index | −0.03 (−0.29 to 0.25) | −0.14 (−0.30 to 0.24) | −0.74 (−0.74 to −0.37) | −0.20 (−0.20 to 0.34) | 0.007 (0.11–0.58) |
| PVR | 0.31 (0.04–0.53) | 0.21 (0.07 to 0.46) | 0.48 (0.23–0.66) | −0.06 (−0.33 to −0.22) | −0.35 (−0.57 to −0.08) |

W-lung indicates whole lung.

Assessment of Hemodynamic Data

We reviewed the following RHC results: right arterial pressure (RAP), systolic ventricular pressure (sRVP), mPAP, CO,
cardiac index, and PVR. Cardiac output was determined by the thermodilution method, and cardiac index was defined as the CO divided by the body surface area. Pulmonary vascular resistance (dyne·s/cm²) was calculated as follows: (mPAP – pulmonary artery wedge pressure)/CO × 80.

Statistical Analysis

The Mann-Whitney U test was used for comparing the patient characteristics or measurements. The χ² test was used for categorical variables. Spearman rank correlation coefficient (rs) with 95% confidence interval (CI) and P value was used to compare DE-CT quantitative parameters and body weight with the hemodynamics values measured by RHC. The correlation between scan timing and DE-CT parameters was also evaluated, because our image acquisition protocol might have a significant effect on the DE-CT parameters. Furthermore, contrast medium injection protocols were divided based on patient weight, and the correlation coefficient between lung PBV and hemodynamics was analyzed for each group. The group weighing less than 40 kg was excluded because there were only 2 patients. Multivariate linear regression analysis was performed whether DE-CT parameters were independent predictors of hemodynamics in which variables were significantly correlated with hemodynamics in bivariate analysis. In this analysis, body weight was included to the analysis factors to evaluate the effect of the scan protocol.

The data of mPAP and PVR were dichotomized according to the following criteria: mPAP >50 mm Hg and PVR > 1000 dyne·s/cm². The reference values of these prognostic and severity factors were determined based on previous studies.14-16 Areas under the curves (AUCs) for receiver operating characteristic (ROC) analysis were performed to evaluate the ability of mPAP and PVR to perform as prognostic criteria. The Youden index method was used to determine the cutoff value and calculate the sensitivity and the specificity.

Analyses were performed using Microsoft Excel 2013 and the statistical software BellCurve for Excel (version 2.15; Social Survey Research Information Co, Ltd, Tokyo, Japan) and R for windows (version 3.5.1; R Foundation for Statistical Computing, Vienna, Austria). A P value of <0.05 was considered to indicate statistical significance.

RESULTS

Patient characteristics, measurements of RHC, and DE-CT examination are shown in Table 1. An mPAP greater than 50 mm Hg was observed in 9 examinations, and a PVR greater than 1000 dyne·s/cm² was shown in 7 examinations. These patients were considered to have severe CTEPH.

The correlation between DE-CT parameters and body weight with RHC hemodynamics is shown in Table 2. The mean lung PBV was significantly correlated with mPAP (rS = 0.47; 95% CI, 0.23–0.66; P < 0.001) (Fig. 2A), sRVP (rS = 0.44; 95% CI, 0.19–0.63; P = 0.001), rAP (rS = 0.32; 95% CI, 0.05–0.54; P = 0.022), and PVR (rS = 0.31; 95% CI, 0.04–0.53; P = 0.027). There was no significant correlation between lung PBV and CO or cardiac index. Whole lung PBV was correlated with mPAP (rS = -0.35; 95% CI, 0.09–0.57; P = 0.010); however, the correlation coefficient was lower than that for lung PBV calculated while excluding the right upper zone. Pulmonary artery enhancement was positively and significantly correlated with CO (rS = -0.59; 95% CI, -0.74 to -0.37; P < 0.001) and cardiac index (rS = -0.49; 95% CI, -0.68 to -0.25; P < 0.001), and positively and significantly correlated with PVR (rS = 0.48; 95% CI, 0.23–0.66; P < 0.001). The rPA was not correlated with RHC hemodynamic parameters. The body weight was positively and significantly correlated with rAP (rS = 0.42; 95% CI, 0.16–0.62; P = 0.002) and CO (rS = 0.37; 95% CI, 0.11–0.58; P = 0.007), and negatively and significantly correlated with PVR (rS = -0.35; 95% CI, -0.57 to -0.08; P = 0.012). On the other hand, the body weight was not significantly correlated with mPAP, sRVP, and cardiac index. The multivariate linear regression analysis indicated that both the lung PBV and the body weight were significantly correlated with RAP (standardized partial regression coefficient [β] = 0.27, P = 0.033, and β = 0.39, P < 0.001, respectively; Table 3). The PA enhancement was significantly correlated with CO (β = -0.56, 0 < P < 0.001), whereas the body weight was not significantly correlated with CO (β = 0.18, P = 0.121). The PA enhancement was significantly correlated with PVR (β = 0.39, P = 0.121). The body weight was

| Variable | Lung PBV | PA Enhancement | Body Weight |
|----------|----------|----------------|-------------|
|          | B (95% CI) | β     | P        | B (95% CI) | β     | P        | B (95% CI) | β     | P        |
| RAP      | 0.16 (0.01–0.30) | 0.27 | 0.033 | NA | NA | NA | 0.09 (0.03–0.16) | 0.39 | <0.001 |
| CO       | NA | NA | NA | -0.006 (-0.008 to -0.003) | -0.56 | <0.001 | 0.017 (-0.005 to 0.038) | 0.18 | 0.121 |
| PVR      | 9.07 (-3.58 to 21.7) | 0.18 | 0.156 | 0.94 (0.31–1.56) | 0.39 | 0.004 | -0.53 (-11.9 to -1.14) | -0.30 | 0.019 |

B indicates partial regression coefficient; NA, not applicable; β, standardized partial regression coefficient.
than 1000 dyne·s/cm\(^5\), the AUC values were significant for PAHU, 0.78 and 0.86, respectively. At the criterion of PVR greater than 0.59 (0.15; 95% CI, 0.02–0.26; P = 0.071), the AUC values were lung PBV (0.86; 95% CI, 0.74–0.98; P = 0.005), but not for lung PBV (0.67; 95% CI, 0.42–0.92; P = 0.18) and rPA (0.58; 95% CI, 0.32–0.85; P = 0.539). The optimal cutoff value, sensitivity, and specificity of PA enhancement were 614 HU, 0.86 and 0.73, respectively.

Table 4 shows the correlation between lung PBV and hemodynamics in different injection protocols based on body weight in patients with CTEPH. The mean and standard deviation of PBV value was positively correlated with mPAP. There have been few studies comparing the quantitative value of lung PBV with the RHC dataset. Meinel et al.\(^{17}\) reported that lung PBV values relative to PA enhancement were correlated negatively with mPAP. This discrepancy may be caused by a difference in the acquired DE-CT scan phase because of the configuration of the bolus-tracking method. In our study, the bolus-tracking ROI was placed on the ascending aorta. Therefore, the DE-CT scan is acquired in the pulmonary parenchymal phase in which the PA and vein are adequately filled with the contrast medium. The difference in scan timing due to placing the ROI in the pulmonary vascular system. Meinel et al.\(^{17}\) also demonstrated that enhancement of the pulmonary trunk and the enhancement difference between the pulmonary trunk and left atrium significantly influenced PBV values. On the other hand, the scan timing in the pulmonary

**DISCUSSION**

In this study, we examined the correlation between quantitative parameters (lung PBV, PA enhancement, rPA) obtained from DE-CT and hemodynamics in patients with CTEPH. The lung PBV value was positively correlated with mPAP. There have been few studies comparing the quantitative value of lung PBV with the RHC dataset. Meinel et al.\(^{17}\) reported that lung PBV values relative to PA enhancement were correlated negatively with mPAP. This discrepancy may be caused by a difference in the acquired DE-CT scan phase because of the configuration of the bolus-tracking method. In our study, the bolus-tracking ROI was placed on the ascending aorta. Therefore, the DE-CT scan is acquired in the pulmonary parenchymal phase in which the PA and vein are adequately filled with the contrast medium. The difference in scan timing due to placing the ROI in the pulmonary vascular system. Meinel et al.\(^{17}\) also demonstrated that enhancement of the pulmonary trunk and the enhancement difference between the pulmonary trunk and left atrium significantly influenced PBV values. On the other hand, the scan timing in the pulmonary

**FIGURE 3.** The ROCs curves for identifying the criteria of prognostic and severity factors for CTEPH.

![FIGURE 3](image-url)
parenchymal phase was not correlated with lung PBV. Therefore, the lung PBV can be used to directly assess the contrast effect in pulmonary parenchymal. In CTEPH, obstruction of the peripheral PA causes increases in mPAP and PVR, and decreases in circulation from the PA to venous return of the contrast medium. Consequently, the pooling of contrast medium in pulmonary vessels might result in increased lung PBV values. Lung PBV values evaluated using different scan timings may be important to explain the difference in pulmonary circulation in patients with CTEPH.

We performed subgroup analysis to evaluate the correlation between lung PBV and hemodynamics for each weight-based protocol. Although the results showed no statistical significance, lung PBV tended to be correlated with hemodynamics in the group weighing 45 to 55 kg. On the other hand, lung PBV was correlated with mPAP in the group weighing 55 kg or more. This result might have been affected by sample size, because mPAP was not correlated with body weight. Takagi et al\(^{18}\) reported that a high-contrast enhancement of the PA was obtained by injecting a contrast medium at a constant rate of iodine per body weight. Adjusting the contrast medium injection method according to weight may be useful in improving the diagnostic accuracy of lung PBV value.

We also confirmed that PA enhancement was negatively correlated with CO and cardiac index, that is, an increase PA enhancement suggested a decrease in CO associated with right heart failure. Pulmonary hypertension in CTEPH is caused by obstruction of pulmonary arteries with an organized thrombus and vascular remodeling of small vessels.\(^{21,22}\) Progression of PH leads to right heart failure and decreased CO. As a result, the dilution effect of the contrast medium in the pulmonary trunk decreases, and increases the PA enhancement.\(^{23,24}\)

The prognosis of patients with CTEPH is related to the degree of pulmonary arterial pressure. Untreated CTEPH patients with mPAP above 50 mm Hg have an extremely poor prognosis, in which the 2-year survival rate is approximately 20%.\(^{14}\) Pulmonary artery endarterectomy is the first option for curative treatment with CTEPH, although several studies reported that a preoperative PVR of more than 1000 dyne·s/cm\(^5\) was a mortality risk factor.\(^{15,16}\) For this reason, hemodynamic assessment by a noninvasive method is helpful for determining the management of CTEPH. The results of ROC analysis are useful for clinical use of lung PBV and PA enhancement value. Derlin et al\(^{25}\) reported that quantitative assessment of the extent of the perfusion defect in CTEPH on a lung perfusion SPECT/CT with 99mTc–human serum albumin was able to diagnose mPAP greater than 50 mm Hg with a sensitivity of 88% and specificity of 64%. In this study, the lung PBV value was able to diagnose mPAP greater than 50 mm Hg with a sensitivity of 78% and specificity of 87%.

The rPA value showed no significant correlation with mPAP. It has been reported that rPA correlates with mPAP or strongly suggests PH based on the criteria of an rPA larger than 1.\(^{8-10}\) Takagi et al\(^{18}\) report that rPA was positively correlated with mPAP in CTEPH patients; however, approximately 90% of patients were...
treated with pulmonary endarterectomy or balloon pulmonary angioplasty (mean rPA, 0.98; mPAP, 24 mm Hg). The difference between the results might be due to the different target patients. Our result is consistent with a study by Corson et al\textsuperscript{26} that showed that the correlation coefficient between rPA and mPAP was low or not significant only in the PH patients. Therefore, the rPA value is a morphological parameter caused secondarily by high pulmonary arterial pressure and a quantitative parameter established for diagnosing patients with CTEPH, although the lung PBV value that is the hemodynamic parameter might able to diagnose the severity of CTEPH more accurately.

Our study has several limitations. First, this study was performed retrospectively at a single center in a limited number of patients. Second, the right upper zones of the lungs were excluded from the region of measurement because of the inaccuracy of the lung PBV measurement owing to the artifact caused by the dense contrast medium flowing from the subclavian vein to the superior vena cava. However, if huge defects are in the excluded area, the lung PBV value may be imprecise. Hence, it is necessary to confirm visually that there are no huge defects in the right upper zone on the lung PBV images. Third, the scan timing of DE-CT might affect the quantitative values of DE-CT. Our results include the influence of the systemic collateral supply from bronchial arteries or intercostal arteries.\textsuperscript{27–29} When there are widespread defects in lung PBV, regardless of central or distal type of CTEPH, the mean lung PBV value might have been increased by collateral blood supply. It was impossible to assess how much this must have influenced the lung PBV values in this study. If faster DE-CT could scan during both the PA phase and the pulmonary circulation phase, the relationship between the degree of contrast enhancement of the aorta and the potential influence of collateral circulation might be explained. Further study is needed on the degree of influence of the systemic collateral supply on lung PBV.

In conclusion, the lung PBV value was positively correlated with mPAP and PVR, and PA enhancement was negatively correlated with both CO and cardiac index and positively correlated with PVR under the condition that DE-CT scan was performed in the pulmonary parenchymal phase. Furthermore, it was confirmed that the quantitative values of lung PBV and PA enhancement were able to identify severe CTEPH patients specified according to the criterion of mPAP greater than 50 mm Hg or PVR greater than 1000 dyne/s/cm\textsuperscript{2}. In conclusion, lung PBV and PA enhancement could be indicators of hemodynamics, and noninvasive quantitative DE-CT parameters could therefore guide the CTEPH management.

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