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Pulmonary Artery-to-Pulmonary Artery Collaterals in Patients with Chronic Thromboembolic Pulmonary Hypertension Demonstrated on CT Angiography

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Abstract

Background: The findings of chronic thromboembolic pulmonary hypertension (CTEPH) on computed tomography angiography (CTA) can be divided into vascular and parenchymal changes. Most previous researches examined the systemic collateral branches and there have been no studies examining the prevalence of pulmonary artery-to-pulmonary artery anastomoses using CTA.

Purpose: To evaluate the appearance and prevalence of pulmonary artery-to-pulmonary artery collaterals on CTA of patients with CTEPH.

Methods: Sixty patients who were diagnosed with CTEPH and underwent CTA were included. The collected data including gender, age, underlying diseases, abnormalities in size and appearance of the large pulmonary arteries (main, lobar and segmental), size and uniformity of the small (subsegmental) pulmonary arteries, parenchymal findings, and collaterals which included systemic-to-pulmonary (bronchial and non-bronchial) and pulmonary artery-to-pulmonary artery were reviewed by two radiologists independently. Data were analysed by chi-square test for categoric data and t test for continuous variables. Values of $p < 0.05$ were considered statistically significant.

Results: *Pulmonary artery-to-pulmonary artery anastomoses were found in 7/56 (13%) cases*. There were statistically significant differences in age and mosaic parenchyma between the groups with and without pulmonary artery-to-pulmonary artery collaterals ($p<0.05$). No statistically significant differences in other demographic data and CT findings were found between the groups. The pulmonary artery-to-pulmonary artery anastomoses were significantly younger than those with systemic artery to pulmonary artery anastomosis.

Conclusion: Pulmonary artery-to-pulmonary artery collaterals could be seen on CTA in a small number of CTEPH patients of younger age who showed mosaic lung attenuation, with statistical significance.

Keywords: Pulmonary Artery-to-Pulmonary Artery Collateral Circulation; Chronic; Thromboembolism; Pulmonary Hypertension; Computed Tomography Angiography

Introduction

Chronic thromboembolic pulmonary hypertension (CTEPH) is one of the main causes of pulmonary arterial hypertension. The pathogenesis of this condition is an incomplete resolution of the thrombus or repetition of a thromboembolism in the large and/or middle-sized pulmonary arteries [1], causing decreased perfusion to the lung parenchyma. To maintain viability of the lung portion distal to the thromboemboli, bronchial and non-bronchial systemic-to-pulmonary circulation was developed [2-4].

Pulmonary angiography has long been the gold-standard test for diagnosing CTEPH with sensitivity ranging from 66% at the main/lobar, 76% at the segmental level, and 87% at the subsegmental arteries, and specificity close to 100% [5]. Nowadays, CT angiography plays an important role in patients with CTEPH due to its being a non-invasive technique and its high image quality that results in high sensitivity and specificity to detect thrombi in all main, lobar, segmental and subsegmental arteries. One study found that the sensitivity for diagnosing CTEPH with CT angiography was nearly 100% at the main, lobar and segmental levels, and 80% at the subsegmental arteries, while the specificity at the main, lobar and segmental levels was approximately 99-100 % [5].

The findings regarding CT angiography can be divided into vascular and parenchymal changes. Vascular changes are from either pulmonary arterial hypertension or chronic pulmonary thromboembolisms. The signs of pulmonary arterial hypertension are seen as central pulmonary artery dilatation, right heart chamber enlargement or atherosclerotic changes. The signs of chronic pulmonary thromboembolism appear as ring-like stenoses, intraluminal bands or webs, total occlusions of the pulmonary arteries, or disparity in the segmental vessel size [6]. Parenchymal changes include mosaic lung perfusion and/or peripheral parenchymal opacities [7]. The typical mosaic lung pattern of CTEPH is sharply demarcated geographic or pyramid-shaped areas of decreased and increased attenuation in a segmental or subsegmental distribution with well-defined borders corresponding to the anatomic unit of the secondary pulmonary lobule [6]. The decreased lung attenuation is produced by hypoperfusion in lung areas distal to occluded vessels, whereas increased lung attenuation is produced by a compensatory increase in blood flow to open vessels because of blood flow redistribution and collateral blood flow. The peripheral parenchyma opacities are caused by previous lung infarcts.

An earlier study found abnormally enlarged bronchial and non-bronchial systemic arteries in 73% of chronic thromboembolic pulmonary hypertension patients, compared with 14% of the idiopathic pulmonary hypertension patients [3]. The dilated bronchial artery, more than 1.5 mm in diameter without bronchial wall thickening, is a significant finding of CTEPH, compared with the non-CTEPH [6].

While most previous researchers examined the systemic collateral branches which respond to pulmonary ischemia by enlargement and hypertrophy [8], there has been only one study examining pulmonary artery-to-pulmonary artery anatomizes on angiography, which found a prevalence of approximately 35% (15/43) [4]. Furthermore, there have been no studies examining the prevalence of pulmonary artery-to-pulmonary artery anastomoses using CT angiography. Our aims were to study the prevalence of and describe the appearance of pulmonary artery-to-pulmonary artery anatomizes shown on CT angiography of patients with CTEPH.

Materials and Methods

This study was approved by our Human Research Ethics Committee. Confidentiality of patient data was ensured by using coded numbers as a substitute for patient names and hospital numbers, and concealing all information during the study.

Patients

All patients who were diagnosed as having CTEPH and had CT angiography performed between 1st January 2010 and 31st December 2017 in xxxxx Hospital were recruited into this study by searching the hospital electronic medical record and radiology information systems, using ICD-10: I27-2 other secondary pulmonary hypertension. The diagnostic criteria of CTEPH were mean PAP ≥ 25 mmHg with PAWP ≤ 15 mmHg, mismatched perfusion defects on lung scan and specific diagnostic signs for CTEPH seen on multidetector CT angiography, MR imaging or catheter pulmonary angiography, such as ring-like stenoses, webs/slits and chronic total occlusions (pouch lesions or tapered lesions), according to the 2015 ESC/ERS Guidelines. Recorded data included gender, age, and underlying diseases such as hypertension, diabetes mellitus, dyslipidemia, coronary arterial disease, chronic obstructive pulmonary disease, asthma, and/or other lung and heart diseases.

Image Analysis

Patients whose CT angiograms were of inadequate image quality were excluded. An investigator who did not subsequently participate in image interpretation removed all patient identifying information from the CT scans and previous imaging findings and parameters. At image analysis, all additional patient data including the imaging results were concealed. The interpretations were performed by a 23-year-experienced thoracic radiologist and a 3-year-experienced radiologist independently who recorded the size and appearance of abnormalities in the large pulmonary arteries (main, lobar and segmental), the size and uniformity of abnormalities in the small (subsegmental) pulmonary artery, parenchymal findings, and collaterals which included the bronchial, non-bronchial and pulmonary artery-to-pulmonary arteries.The appearance of the large pulmonary arteries and presence of collateral arteries were evaluated in the arterial phase on a mediastinal window. The size of both the large and small pulmonary arteries, uniformity of the vessel sizes, and parenchymal signs were studied on a lung window.

Terms

The main, lobar and segmental pulmonary arteries were defined as large arteries while the subsegmental pulmonary arteries were defined as small arteries. The appearance of the large pulmonary arteries was considered normal if the broncho-arterial ratio equaled 1 and there was no intraluminal thrombus, webs, band or abnormal wall thickening. Enlargement of the pulmonary artery was recorded if the broncho-arterial ratio was less than 1. The size of the pulmonary arteries was recorded as decreased if the broncho-arterial ratio was greater than 1. Pulmonary arteries were deemed uniform if there was no variation in the diameters of the vessels at the same distance from the hilum in the same lung while they were deemed non-uniformity of the pulmonary arteries if there was vessel size disparity at the same distance from the hilum. Parenchymal signs included subpleural consolidations, defined as pleural-based wedge-shaped or linear opacities, and a mosaic pattern which was defined as sharply demarcated geographic areas of decreased and increased attenuations in a segmental or subsegmental distribution with well-defined borders corresponding to the anatomic unit of the secondary pulmonary lobule. Collaterals were divided into systemic-to-pulmonary artery and pulmonary artery-to-pulmonary artery anastomoses. A systemic-to-pulmonary artery anastomosis was defined as any of the dilated or tortuous systemic arteries (bronchial, intercostal, inferior phrenic or others such as internal mammary arteries) that entered the lung. Pulmonary artery-to-pulmonary artery anastomosis was deemed if the course of one pulmonary artery was not parallel to its corresponding bronchus and communication with the pulmonary artery in the adjacent unit (segment or secondary pulmonary lobule) was shown.

Statistical Analysis

The results were calculated as mean ± SD for continuous variables and frequencies and percentages for categoric variables. Comparative analyses were performed with Fisher exact test or chi-square test for categorical data. For continuous variables, differences between the groups were compared by t test, depending on the distribution characteristics. Values of $p < 0.05$ were considered statistically significant. All statistical analyses were performed with Epidata software.

Results

There were 60 patients who were diagnosed as having CTEPH. Four patients were excluded due to poor image quality. The records of the remaining 56 patients were retrospectively reviewed. Nineteen were male and 37 were female. Their ages ranged between 26 and 88 years with a mean age of 54.4 years. The main underlying diseases were hypertension, diabetes mellitus, dyslipidemia, coronary artery disease, chronic obstructive lung disease, and asthma, as well as other lung and heart diseases such as systemic lupus erythematosus, protein C and protein S deficiency, antiphospholipid syndrome, thalassemia, secondary erythrocytosis, atrial septal defect, patent ductus arteriosus and valvular heart disease. A significant number of the patients (25/56 cases) had heart disease (45%). The demographic data are shown in Table 1. Only 5 CTEPH patients (9%) had lung ventilation and perfusion scans, all of which were positive.

Table 1: Patient demographic data

Characteristic	Total(N=56)	No PA to PA anastomosis(n=49)	PA to PA anastomosis (n=7)	*p*-value
Age (mean±SD)*	54.4± 16.6	56.3± 16.2	40.9± 14.0	0.020
Gender Male Female	19 (33.9%) 37 (66.1%)	18 (36.7%) 31 (63.3%)	1 (14.3%) 6 (85.7%)	0.403
Underlying disease HT DM DLP CAD COPD Asthma Other heart diseases Other diseases	48 (85.7%) 8 (14.3%) 7 (12.5%) 7 (12.5%) 3 (5.4%) 4 (7.1%) 3 (5.4%) 25 (44.6%) 19 (33.9%)	42 (85.7%) 7 (14.3%) 6 (12.2%) 6 (12.2%) 3 (6.1%)4 (8.2%)2 (4.1%)20 (40.8%)16 (32.7%)	6 (85.7%) 1 (14.3%) 1 (14.3%) 1 (14.3%) 0 (0.0%) 0 (0.0%) 1 (14.3%) 5 (71.4%) 3 (42.9%)	1.000 1.000 1.000 1.000 1.000 1.000 0.335 0.223 0.679

Note* Data are means, with standard deviations in parentheses.

PA = Pulmonary artery

HT = Hypertension

DM = Diabetes mellitus

DLP = Dyslipidemia

CAD = Coronary artery disease

COPD = Chronic obstructive pulmonary disease

Statistical Analysis

There were four categories of abnormalities examined in CTEPH patients, namely large pulmonary artery signs, small pulmonary artery signs, parenchymal signs, and collaterals. All patients had abnormalities of the large arteries. Twenty-five out of fifty-six (45%) had all four signs, 44/56 (79%) had at least three signs, 54/56 (96%) had at least two signs, and only 2/56 (4%) had abnormalities only in the large arteries.

The large pulmonary artery signs included abnormal size (56/56, 100%), thickened walls (11/56, 20%), intraluminal thrombi (34/56, 66%), intraluminal webs (16/56, 29%), and calcified walls (8/56, 14%).

Small (subsegmental) pulmonary artery signs were found in 48/56 (86%) patients. They were non-uniform (mixed enlarged and small) in 19/56 (34%), uniformly small in 1/56 (2%) and uniformly enlarged in 28/56 (43%) patients.

Abnormal parenchymal findings, subpleural opacities and mosaic attenuation, were found in 39/56 (70%) cases. No parenchymal findings related to CTEPH were found in 17/56 (30%) patients, four of whom had underlying chronic lung disease and the rest had normal lung parenchyma.

Collaterals were found in 32/56 (57%) patients. Enlarged systemic arteries were found in 30/56 (54%), 26/56 (46%) were bronchial arteries, 9/56 (16%) were intercostal arteries, and 16/56 (29%) were inferior phrenic or internal mammary arteries.

Pulmonary artery-to-pulmonary artery anastomoses were found in 7/56 (13%) cases. One of these seven cases had pulmonary angiography (Figure 1A) performed to rule out the possibility of pulmonary arteriovenous malformation because a CT showed enlarged serpiginous subpleural pulmonary arteries. Bronchial angiography (Figure 1B) in the same setting was also performed because the patient's clinical presentation was hemoptysis.

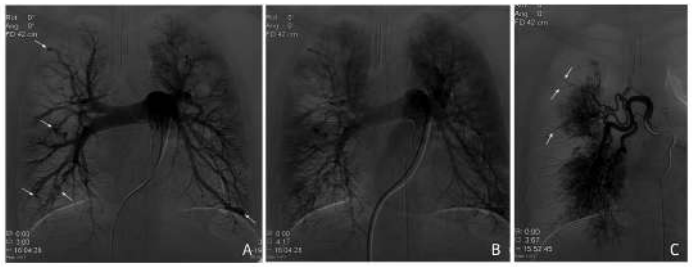

Figure 1: Pulmonary and bronchial circulations demonstrated on angiography of a 25-year-old female who presented with recurrent hemoptysis. (A) Pulmonary arteriography in the early arterial phase showing less branches in the right lung with failure to distal tapering of some arteries (arrows). (B) Pulmonary arteriography in the late arterial phase showing multiple perfusion defects, more in the right lung. (C) Right bronchial angiography demonstrating increased blood supply into the perihilar area that is the perfusion defect in B. There are only a few enlarged branches extending to fill the peripheral pulmonary arteries (arrow)

Direct anastomoses between the parts distal to the point of occlusion of the thromboembolized subsegmental pulmonary arteries with the adjacent patent ones were seen on CT (Figures 2-4) and comparison with superselective angiograms was done (Figures 2,3).

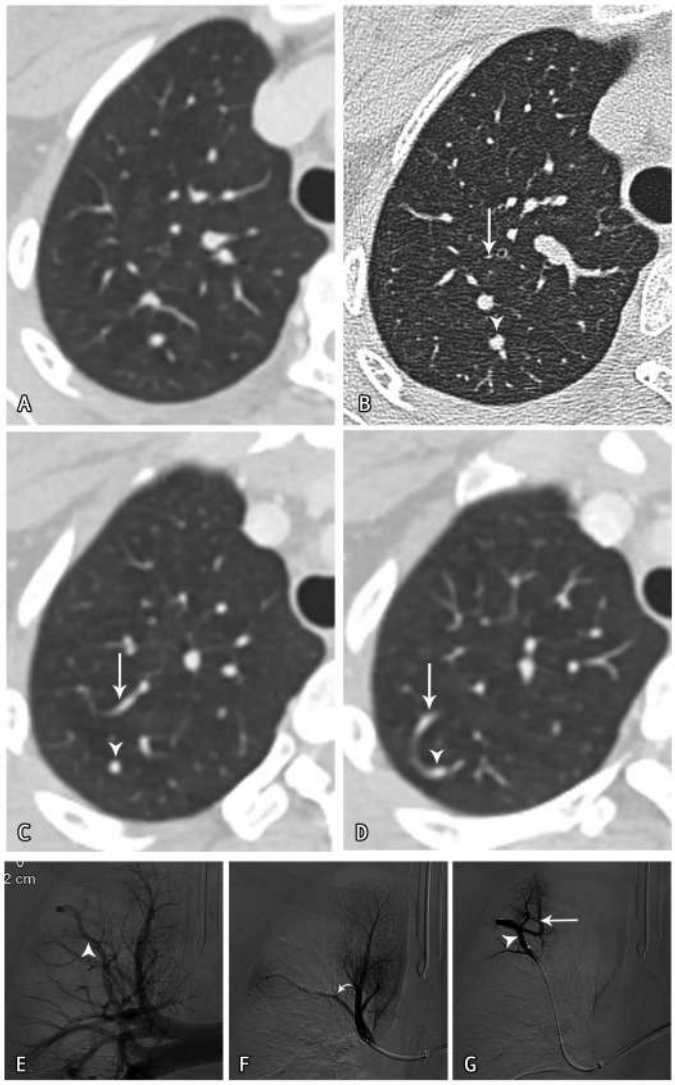

Figure 2: Pulmonary artery-to-pulmonary artery anastomosis in the right upper lobe. CT in lung window (A) and HRCT (B) at a similar level of the right upper lobe showing size disparity in the subsegmental pulmonary arteries in the right upper lobe. The thrombosed artery (arrow) is smaller than the accompanying bronchus while the patent one is dilated (arrowhead). More peripheral (C, D) the distal part of the thrombosed artery (arrow) is receiving supply from the adjacent dilated pulmonary artery (arrowhead). Right pulmonary (E) and selective angiographs of 2 branches of the right upper lobar artery (F, G) showing that the patent pulmonary artery (arrow head) has anastomosed with the distal part of the thrombosed one (arrow) and is supplying the lung parenchyma distal to the thrombosis (curved arrow)

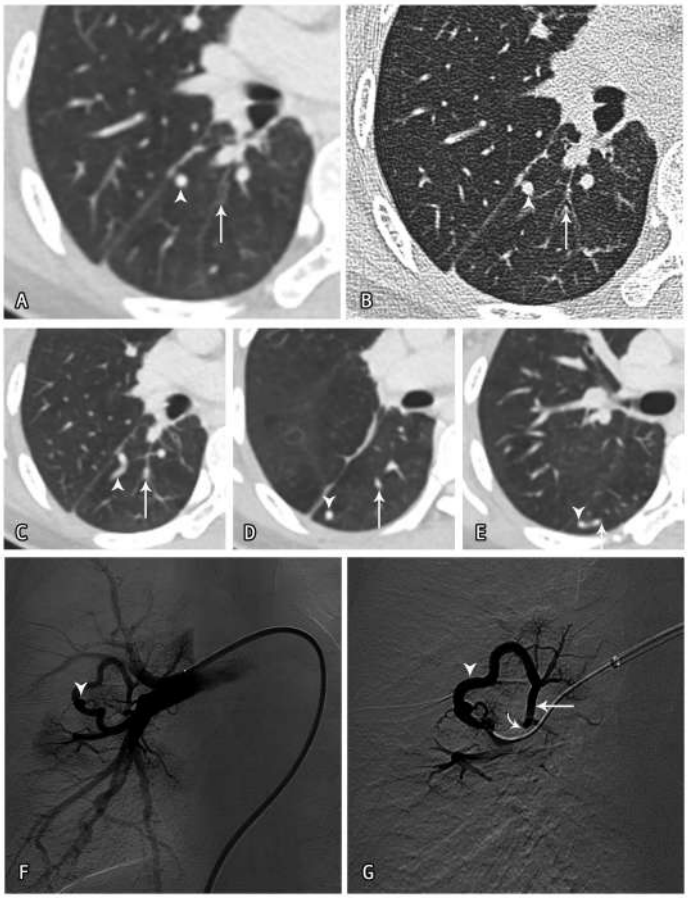

Figure 3: Pulmonary artery-to-pulmonary artery anastomosis in the superior segment of the right lower lobe. CT in lung window (A) and HRCT (B) at a similar level of (A) showing that the thrombosed artery (arrow) is almost absent next to the accompanying bronchus while the patent one is dilated (arrowhead) and compressing the accompanying bronchus. More peripherally (C, D, E) the part distal to the thrombosed artery (arrow) communicates with the adjacent dilated pulmonary artery (arrowhead). Right pulmonary (F) and selective angiographs (G) show that the patent pulmonary artery (arrowhead) has anastomosed with the adjacent artery (arrow) distal to the thrombosis (curved arrow)

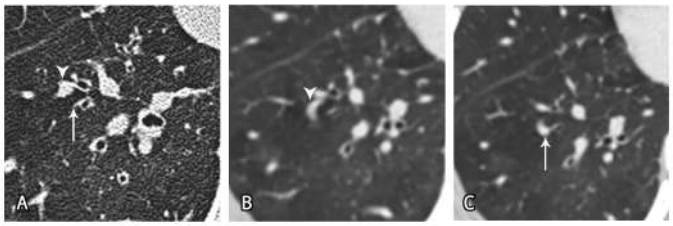

Figure 4: Pulmonary artery-to-pulmonary artery anastomosis in the anterior basal segment of the right lower lobe. An HRCT scan (A) showing that the thrombosed arteries (arrow) are almost absent next to the accompanying bronchi while the patent one is dilated (arrowhead). Two consecutive CT scans in the lung window (B, C) showing an anastomosis between the dilated patent artery (arrowhead) and the adjacent artery distal to the thrombosis (arrow)

The abnormal radiographic findings in CTEPH patients who had and did not have pulmonary artery-to-pulmonary artery collaterals are compared in Tables 2 and 3.

Table 2: Comparison of radiographic CT findings between CTEPH patients with pulmonary artery-to-pulmonary artery collaterals and CTEPH patients without pulmonary artery-to-pulmonary artery collaterals

Characteristic	Total(N=56)	No PA to PA anastomosis(n=49)	PA to PA anastomosis (n=7)	*p*-value
Large PA				-
Abnormal	56 (100.0%)	49 (100.0%)	7 (100.0%)	
Small PA				
Normal	4 (7.1%)	4 (8.2%)	0 (0.0%)	
Abnormal	52 (92.9%)	45 (91.8%)	7 (100.0%)	1.000
Parenchyma				
Normal	17 (30.4%)	17 (34.7%)	0 (0.0%)	
Abnormal	39 (69.6%)	32 (65.3%)	7 (100.0%)	0.088

PA = Pulmonary artery

Characteristic	Total(N=56)	No PA to PA anastomosis(n=49)	PA to PA anastomosis (n=7)	*p*-value
Vascular sign				
Large PA				
Location				
Both left and right sides	56 (100.0%)	49 (100.0%)	7 (100.0%)	-
All main, lobar and segmental branches	51 (91.1%)	44 (89.8%)	7 (100.0%)	-
Abnormality	44 (78.6%)	40 (81.6%)	4 (57.1%)	0.16
Thickened wall	11 (19.6%)	10 (20.4%)	1 (14.3%)	1
Thrombus	37 (66.1%)	34 (69.4%)	3 (42.9%)	0.212
Web	16 (28.6%)	14 (28.6%)	2 (28.6%)	1
Calcification	8 (14.3%)	8 (16.3%)	0 (0.0%)	0.577
Small PA				
Bilateral	51 (91.1%)	44 (89.8%)	7 (100.0%)	1
Abnormality				0.48
Normal	8 (14.3%)	8 (16.3%)	0 (0.0%)	
Non-uniform	19 (33.9%)	15 (30.6%)	4 (57.1%)	
Uniform small	1 (1.8%)	1 (2.0%)	0 (0.0%)	
Uniform large	28 (50.0%)	25 (51.0%)	3 (42.9%)	
Parenchymal sign				
Subpleural consolidation	27 (48.2%)	22 (44.9%)	5 (71.4%)	0.244
Mosaic pattern	29 (51.8%)	22 (44.9%)	7 (100.0%)	0.011
Unevaluable	4 (7.1%)	4 (8.2%)	0 (0.0%)	1

Table 3: Comparison of Vascular and Parenchymal CT Findings between CTEPH Patients With pulmonary artery-to-pulmonary artery collaterals and CTEPH Patients Without pulmonary artery-to-pulmonary artery collaterals

PA = Pulmonary artery

Note: Data in parentheses are 95% confidence intervals.

There were significant differences in age and mosaic parenchyma between the groups with and without pulmonary artery-to-pulmonary artery collaterals, but no other significant differences in terms of demographic data or other CT findings. The patients with pulmonary artery-to-pulmonary artery anastomoses were significantly younger than those with systemic artery-to-pulmonary artery anastomosis (Table 4).

Table 4: Logistic regression to estimate the effect of gender and age on collaterals in CTEPH patients

Collateral	Variable	*Crude analysis*			*Adjust analysis*		
		Crude OR	**95% CI**	***p*-value**	**Adjust OR**	**95% CI**	***p*-value**
PA to PA	Sex of patient (female)	3.484	0.380-31.920	0.269	3.789	0.393-36.570	0.249
	Age of patient	0.936	0.882-0.992	0.027	0.92	0.854-0.991	0.028
Systemic artery to PA	Sex of patient (female)	0.553	0.176-1.734	0.309	0.548	0.176-1.706	0.299
	Age of patient	0.963	0.930-0.998	0.038	0.965	0.932-0.999	0.046
Bronchial artery to PA	Sex of patient (female)	0.496	0.160-1.539	0.225	0.501	0.163-1.542	0.228
	Age of patient	0.967	0.935-1.000	0.052	0.968	0.936-1.001	0.06
Intercostal artery to PA	Sex of patient (female)	0.586	0.136-2.533	0.474	0.562	0.119-2.657	0.468
	Age of patient	0.979	0.934-1.026	0.381	0.98	0.924-1.040	0.511
Other to PA	Sex of patient (female)	0.471	0.084-2.635	0.391	0.455	0.076-2.722	0.388
	Age of patient	1.001	0.957-1.046	0.979	1.009	0.952-1.069	0.764

PA = Pulmonary artery

OR = Odds ratio

Discussion

At one time in the past, it was assumed that there were anastomoses between contiguous pulmonary arteries to maintain circulation distal to thromboembolized arteries, until Liebow et al. [9] in 1950 and Smith et al. [10] in 1964 demonstrated that the pulmonary arteries are true end arteries separated from each other by interlobular septa. Smith et al. [10,11] showed that bronchopulmonary anastomoses occurred at capillary beds and there were no precapillary anastomoses. Since then, changes occurring in the pulmonary and systemic circulations after a thromboembolic event have been studied extensively. The thromoembolized vessels are recanalized by newly formed endotheliazed channels that communicate between the lumen of the pulmonary artery and the hypertrophied vasa vasorum, establishing bronchial artery-to-pulmonay artery anastomose [12]. In addition, systemic arterial supply to the healing infarct can originate from the intercostal arteries in the chest wall or the arteries of the diaphragm. This concept has been strongly supported by many angiographic and histologic experiments. It is now generally accepted that the viability of the lung distal to thromboemboli is maintained by systemic-to- pulmonary artery collaterals, mainly from the bronchial artery, rather than pulmonary artery-to-pulmonary artery anastomoses.

Then in 2005, Hodson et al. [4] observed regions of hyperemia or luxury perfusion arising from normal pulmonary arterial branches which subsequently resulted in retrograde filling of peripheral pulmonary arterial branches in the adjacent oligemic lung during pulmonary angiography. They hypothesized that these indicated pulmonary artery-to-pulmonary artery anastomoses following luxury perfusion and demonstrated these findings in 15 out of 43 patients with CTEPH.

To our knowledge, our study is the first CT report demonstrating direct precapillary pulmonary artery-to-pulmonary artery collaterals in CTEPH patients. However, pulmonary artery-to-pulmonary artery anastomoses were found in only 7 out of 56 (13%) cases in our CTEPH patients, much less than in a prior angiographic study (15/43 patients, 35%) [5]. One possible reason for this could be that the sensitivity of the pulmonary CT angiography in demonstration of these small collaterals is lower than catheter pulmonary angiography. In catheter pulmonary angiography, a dynamic vascular study with higher pressure contrast injection after selective catheterization provides better demonstration of not only direct end-to-end (precapillary) anastomosis, but probably also of anastomoses between small pulmonary arteries through the capillary beds (postcapillary anastomosis) as retrograde opacification of peripheral pulmonary arteries within the adjacent oligemic lung was observed after luxury perfusion in all 15 cases in the prior study [4].

We hypothesize that these pulmonary artery-to-pulmonary artery collaterals were established due to the necessity of providing a blood supply from the hyperemic lobules to the adjacent oligemic ones. With this hypothesis, pulmonary artery-to-pulmonary artery anastomoses should be more common in heterogeneously perfused lungs, which could be seen on CT as mosaic attenuation or non-uniform small pulmonary arteries. In this study, the pattern of lung attenuation was statistically significantly different between the groups with and without pulmonary artery-to-pulmonary artery collaterals. However, the presence of non-uniform small pulmonary arteries was not significantly different between these two groups.

Additional CT findings in our study were similar to those of other studies. The large pulmonary artery signs included abnormal sizes and intraluminal filling defects such as webs, bands, or eccentric thrombi, which were seen in either uniformly enlarged main, lobar or segmental pulmonary arteries, findings which are normally the result of pulmonary arterial hypertension, or, less commonly, pulmonary arteries of decreased sizes.

Most small pulmonary arteries in our CTEPH patients (28/56) were uniformly large, due to pulmonary arterial hypertension. Other characteristics were non-uniform subsegmental pulmonary arteries in 19/56 (34%) patients and uniformly small subsegmental pulmonary arteries in 1/56 (2%). Similar to another study, it was found that the marked variation in segmental vessels size was more specific for CTEPH than it was for non-CTEPH [6].

The parenchymal signs of CTEPH can be seen as subpleural consolidation or mosaic patterns of lung attenuation. The subpleural opacities could appear as parenchymal bands, wedge-shaped consolidations, peripheral nodules, cavities, or irregular peripheral linear opacities. The mosaic pattern of lung attenuation is seen as sharply demarcated regions of decreased and increased attenuation due to irregular perfusion. Bergin et al. [13] found that a combination of mosaic perfusion and segmental vessel size disparity was highly specific for CTEPH.

The systemic-to-pulmonary artery collaterals included in our study were bronchial, intercostal, inferior phrenic and internal mammary arteries. These collaterals were characterized by being enlarged, tortuous or asymmetrical, compared with normal ones, indicating hypertrophy, which is a nonspecific response to stimuli such as reduced pulmonary artery flow or hypoxemia.1 This could be the reason that they are more frequently found in CTEPH patients than in patients with idiopathic pulmonary arterial hypertension. One of the other collaterals that our study did not include was coronary-pulmonary artery collaterals, which were found in 10.8% of CTEPH patients in one study [14].

Even though there seems to be little question that the bronchial circulation plays a major role in supplying the capillary bed distal to a thromboembolus, it is interesting to note that the increased bronchial arterial circulation demonstrated by bronchial angiography in one of our patients (Figure 1) seemed to be concentrated at the perihilar region with little filling of the peripheral small pulmonary arteries. The pulmonary artery-to-pulmonary artery collaterals in our study and the prior angiographic report noted earlier4 were mostly found in the peripheral region of the lung. This difference in forming collaterals in the central and peripheral parts of the lung could be one among many different anatomic and physiologic properties, which is called corticomedullary differentiation and mentioned in the work of Gurney JW [15].

Unlike patients with acute pulmonary embolism who commonly present with acute dyspnea, patients with CTEPH can show various presentations including shortness of breath, chest pain, fainting, fatigue, palpitations, signs of right heart failure, or even hemoptysis [16]. Those who interpret the CT of these CTEPH patients should be familiar with the vascular, parenchymal and collateral signs. Awareness of how combinations of these signs can be significant clues could enhance detection of CTEPH. We found that most patients had at least three signs, and almost all patients had at least two signs. Spectral CT has an increasing role in the diagnosis of CTEPH because of its ability to show lung perfusion more accurately, which increases its diagnostic sensitivity and prediction of the disease severity [17,18]. For patients with hemoptysis, as in one of our patients, recognition of pulmonary artery--to-pulmonary artery collaterals on CT could avoid misinterpretation as an arteriovenous malformation (AVM) and obviate unnecessary pulmonary angiography. In patients with pulmonary AVM, dilated tortuous pulmonary arteries coexist with dilated draining pulmonary veins and there should be no small pulmonary arteries. Careful observation of the bronchoarterial ratio and other signs of CTEPH is the key to differentiating these 2 disease entities.

In conclusion, pulmonary artery-to-pulmonary artery collaterals could be seen when using CT angiography in a small number of CTEPH patients of younger age who showed mosaic lung attenuation, with statistical significance. All of the CTEPH patients had abnormalities in the large arteries. Most patients had at least three signs, and almost all patients had at least two signs.

Conclusion

The findings of chronic thromboembolic pulmonary hypertension (CTEPH) on computed tomography angiography (CTA) can be divided into vascular and parenchymal changes. Most previous researches examined the systemic collateral branches and there have been no studies examining the prevalence of pulmonary artery-to-pulmonary artery anastomoses using CTA. Pulmonary artery-to-pulmonary artery collaterals could be seen on CTA in a small number of CTEPH patients of younger age who showed mosaic lung attenuation, with statistical significance.

Conflict of Interest

The authors of this manuscript declare no relationships with any companies, whose products or services may be related to the subject matter of the article. The authors state that this work has not received any funding.

Ethic Approval

The study was approved by the Ethic Committee of the faculty of Medicine, Prince of Songkla University before the beginning of the study.

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