Published in final edited form as:

J Comput Assist Tomogr. 2014 ; 38(2): 163–168. doi:10.1097/RCT.0b013e3182aa7fc5.

RELATIONSHIP OF MAIN PULMONARY ARTERY DIAMETER TO PULMONARY ARTERIAL PRESSURE IN SCLERODERMA PATIENTS WITH AND WITHOUT INTERSTITIAL FIBROSIS

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Abstract

Objective—To determine the relationship between main pulmonary artery diameter and pulmonary hypertension (PH) in scleroderma patients with and without interstitial lung disease (ILD).

Methods—We retrospectively reviewed 48 subjects with scleroderma who underwent a chest CT and right heart catheterization with six months of each other. Patients were divided into two groups based on the absence or presence of ILD on chest CT. Subset analysis was performed based on available pulmonary function tests (PFTs) and divided into groups by forced vital capacity (FVC). CT scans were scored for extent of fibrosis and ground glass opacity. Pulmonary artery and ascending aorta measurements were obtained by two independent observers. Univariate and multi-variate models were used to evaluate the correlation between main pulmonary artery diameter (MPAD) and mean pulmonary artery pressure (mPAP) measured by right-heart catheterization. Receiver operating characteristic analysis was performed for diagnostic accuracy of the MPAD measurement in predicting PH

Results—Strong correlations between mPAP and MPAD were found in this study regardless of the presence or absence of mild to moderate interstitial fibrosis on chest CT. When dividing patients based on FVC, the correlation between mPAP and MPAD was substantially attenuated. An MPAD value of 30.8 mm yielded the highest sensitivity and specificity at 81.3% and 87.5% respectively.

Conclusion—In scleroderma patients, an enlarged main pulmonary artery (>30 mm) predicts pulmonary hypertension even in the presence of mild to moderate fibrosis although the relationship may be attenuated in the presence of a low % FVC.

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The authors hereby assert they have no other conflicts of interest other than the support provided above.

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INTRODUCTION

Progressive pulmonary dysfunction has become the primary concern of those investigating mortality linked to Systemic Sclerosis (SSc). Indeed, pulmonary complications have steadily replaced scleroderma renal crisis as the primary cause of SSc-related death with approximately 50% of mortalities resulting from an associated decline in lung function¹. Pulmonary involvement in SSc ranges from minor parenchymal fibrosis to severe pulmonary hypertension (PH). To complicate matters, PH in SSc can result from the progression of interstitial lung disease (ILD) or develop as an isolated pulmonary arterial hypertension (PAH), itself a major mortality factor independent of ILD extent². The clinical evaluation of pulmonary hypertension has proven challenging in the SSc population with 20% of connective tissue disease patients having undiagnosed severe PAH³ and 14% of SSc patients developing severe PH in the face of initial echocardiographic evidence to the contrary⁴.

With regard to screening for pulmonary hypertension, both the invasive nature of right heart catheterization (RHC) and the reduced echocardiographic sensitivity have led to an exploration of alternative methods for establishing the diagnosis of PH in general populations at risk of such involvement⁵⁻¹⁰. Computed Tomography (CT) can potentially serve two roles simultaneously assessing the degree of parenchymal lung disease and the degree of pulmonary arterial pressure (PAP)⁶. Many studies support the use of chest CT in predicting PH⁵⁻⁹ with some reporting correlation coefficients as high as 0.83 between main pulmonary artery diameter (MPAD) measurements and PH⁸. Unfortunately, this correlation may be substantially altered by the presence of underlying parenchymal disease. For example, in idiopathic pulmonary fibrosis there is substantially less correlation between MPAD and PAP^{11,12}.

In light of the recent identification of pulmonary fibrosis as a complicating factor and when considering the heterogeneity of clinical presentation in SSc, it is necessary to understand the relationship between MPAD and PH in the scleroderma population. To our knowledge only one study¹³ has evaluated the utility of the MPAD measurement in mixed populations comprised of very few SSc patients. In this study, we retrospectively analyze the correlation between MPAD and PH using CT to assess pulmonary involvement in a sample of scleroderma patients who have undergone RHC to determine whether MPAD is predictive of PH and determine the influence of ILD on the reliability of the MPAD measurement.

MATERIALS AND METHODS

Patient Selection

This retrospective HIPAA compliant study was done under a waiver of consent from our local institutional review board. To be eligible for the study, patients were required to have undergone both RHC and chest CT scan within a designated five year period between November 18, 2003 and November 18, 2008. Additionally, no more than six months was allowed between RHC and chest CT for patients to be included. In the event that a patient had multiple RHCs over the five year period indicated above, only the earliest RHC for which corresponding chest CT data was available was included in the study. A single rheumatology fellow performed electronic chart review to identify those patients meeting the American College of Rheumatology (ACR) criteria for the diagnosis of SSc¹⁴. Exclusions were made for patients whose chest CT showed signs of intubation or lung resection, whose mediastinal vascular delineation was judged as poor or worse (See CT Scoring Section), who had an insufficient number of image levels to determine overall inflammatory and fibrotic lung involvement, and who received nitric oxide prior to RHC.

For purposes of analysis, patients were divided into two groups based on the absence (Group A; n=20) or presence (Group B; n=28) of ILD on chest CT. A subset analysis of patients with available pulmonary function tests (PFTs) were divided into groups based on forced vital capacity (FVC) measurements (n=34). Patients with FVC > 70% predicted (Group C; n=18) were compared to those with FVC ≤ 70% predicted (Group D; n=16).

CT Scoring

All CT images incorporated into this study were initially reviewed by a single thoracic radiologist with over 10 years of experience to determine the extent of ILD. The same observer also graded each image according to the quality of mediastinal vascular delineation. The extent of ILD was measured according to a previously reported protocol¹⁵ with slight variations. Ground glass opacity (GGO) was evaluated using a six point scale (0 = absent, 1 = less than 5% of total lung, 2 = up to 25% of total lung, 3 = 25% to 49% of total lung, 4 = 50% to 75% of total lung, 5 = greater than 75% of total lung) as was interstitial fibrosis (0 = absent, 1 = interlobular septal thickening w/o honeycombing, 2 = honeycombing involving up to 25% of total lung, 3 = honeycombing involving from 25% to 49% of total lung, 4 = honeycombing involving from 50% to 75% of total lung, 5 = honeycombing involving greater than 75% of total lung). Vascular delineation was rated using a Likert scale (1 = very poor, 2 = poor, 3 = reasonable, 4 = good, 5 = very good)⁷.

After eliminating all studies receiving a vascular delineation rating of 2 or less, two independent radiologist observers reviewed the remaining images to measure the diameter of both the main pulmonary artery and the aorta. Measurements were made using computer calipers on mediastinal windows (window width = 390 HU, window level = 60 HU) blinded to clinical data regarding the research subjects. The MPAD was defined as the greatest distance perpendicular to the long axis of the vessel as it passes anteroposteriorly across the base of the heart on supine full-chest sequence. The widest diameter of the aorta was also measured at the same scan level (Figure 1). For study purposes, the MPAD used for correlation with RHC was defined as the average of the two observer's measurements.

Right Heart Catheterization

In general, RHC was performed following the acquisition of right femoral vein access using a 7 French introducer sheath. A 7 French Swan-Ganz balloon tipped catheter was then introduced via the sheath and advanced through the right heart chambers into the pulmonary capillary wedge position. PAPs and pulmonary capillary wedge (PCW) pressures were recorded for all patients. Pulmonary vascular resistance (PVR) was calculated using the following equation: $PVR = (mPAP - mPCW) / CO$ where mPAP is the mean pulmonary artery pressure, mPCW is the mean pulmonary capillary wedge pressure, and CO is the cardiac output measured by either Fick's method or thermodilution.

Statistical Analysis

Categorical data are expressed as proportions while continuous variables are characterized by mean (S.D.) if normally distributed or median (range) if distributed otherwise. For continuous variables, comparisons between groups were made using the Student's t-test or Mann-Whitney U test depending on the distribution of the data. Either the Chi-square test or Fisher's exact test was used to compare categorical data between groups. Correlations were examined using Pearson's coefficient for normal data and Spearman's coefficient if the assumption of normality could not be upheld. Given the limited sample size in this study, four of the following variables were chosen for incorporation into a multivariable linear regression model of mPAP based on the highest univariate correlations: age, gender, ethnicity, aortic diameter, body surface area, and presence or absence of fibrosis on chest CT. In order to investigate any potential interaction regarding the presence of interstitial

fibrosis on chest CT and the relationship between mPAP and MPAD, a separate term was incorporated into the multivariable regression model. Receiver operating characteristic analysis was performed to determine the diagnostic accuracy of the MPAD measurement in predicting the presence of PH.

RESULTS

Patient attributes

After screening our patient base for the previously specified inclusion and exclusion criteria, 48 subjects remained for incorporation into this study. Table 1 contains demographic information as well as clinical data for Group A and Group B. Data was available on a fraction of the patients with regard to several variables including PFT results. Of the 48 patients with suspected scleroderma, 3 patients had undifferentiated connective tissue disease (UCTD), 1 patient had mixed *sine* scleroderma /sarcoidosis, 37 patients met the ACR criteria for the diagnosis of SSc, 2 patients met ACR criteria for SSc while also suffering from concomitant lupus, and 5 patients had insufficient records to accurately determine a diagnosis. No significant difference existed between Group A and Group B when considering CT-determined measurements of the mediastinal vasculature and invasive measures of pulmonary hemodynamics. The average MPAD in Group A was 3.13cm compared to 3.32cm in Group B ($p = 0.15$). Upon evaluation of pulmonary hemodynamics, the average mPAP was found to be 28mmHg in Group A and 31mmHg in Group B ($p = 0.35$). Forced vital capacity data was available in 14 patients from Group A and 20 patients from Group B with mean values of 85.9% predicted and 64.2% predicted respectively ($p = 0.002$). Diffusion capacity for carbon monoxide (DL_{CO}) was also available in 14 patients in Group A, but only 19 patients in Group B. Similar to FVC, a statistically significant difference in DL_{CO} was found between the groups with Group A having a mean value of 50% predicted and Group B having a mean value of 34% predicted ($p = 0.001$).

Overall, 34 patients had available FVC data with Table 2 containing a comparison of important patient characteristics between Group C and Group D. The main differences between FVC-based groups pertain to patient age and MPAD. The average age of patients in Group C was 62 years compared to 53 years in Group D ($p = 0.027$). On average, MPAD was larger ($p = 0.03$) in Group D (3.30cm) versus Group C (3.01cm).

Univariate correlations

Strong correlations between mPAP and MPAD were found in this study regardless of the presence or absence of mild to moderate interstitial fibrosis on chest CT (Table 3). The correlation coefficient between mPAP and MPAD in Group A ($r = 0.68$, $p = 0.001$) was very close to the value of the correlation coefficient found in Group B ($r = 0.70$, $p < 0.0001$). Interestingly, when dividing patients based on FVC, the correlation between mPAP and MPAD was substantially attenuated with Group C ($r = 0.69$, $p = 0.002$) having a higher coefficient value than Group D ($r = 0.42$, $p = 0.105$) (Figure 2). Significant correlations were also present in both Group A ($r = 0.50$, $p = 0.03$) and Group B ($r = 0.47$, $p = 0.01$) between mPAP and the ratio of MPAD to aortic diameter (AD), though these values were somewhat less in magnitude when compared to the correlation between mPAP and MPAD. In contrast, marked variation between Group A ($r = 0.51$, $p = 0.03$) and Group B ($r = 0.09$, $p = 0.67$) can be seen when PVR is correlated with MPAD/AD. This difference is lessened considerably when evaluating the relationship between PVR and MPAD. When the relationship between mPAP and MPAD/AD was evaluated in Group C and Group D, a different pattern emerged than that seen when dividing patients based on CT measurements of interstitial fibrosis. While the correlation in Group C ($r = 0.66$, $p = 0.003$) remained similar to that seen between mPAP and MPAD, Group D ($r = -0.09$, $p = 0.73$) showed no significant correlation between

mPAP and MPAD/AD. Group differences in the correlation between invasive measures of pulmonary hemodynamics and PFT results can also be appreciated from Table 3.

Multivariable linear regression

No relationship between mPAP and ethnicity, AD, or fibrosis group was identified on multivariable linear regression with MPAD serving as the primary measure of vascular dimension. Also, there was no interaction between MPAD and the presence or absence of fibrosis using this model ($\beta^{\text{hat}} = 0.07$, $p = 0.43$).

Diagnostic Accuracy

Utility of the MPAD measurement was assessed by generating a receiver operating characteristic curve (Figure 3) and calculating the area under the curve (AUC = 0.86). An MPAD value of 30.8 mm yielded the highest sensitivity and specificity at 81.3% and 87.5% respectively.

DISCUSSION

In clinical practice, MPAD can be a valuable tool as a marker of potentially elevated PAP with one study reporting a correlation coefficient as high as 0.83⁸ between these two measures. Recent literature^{11,12} has challenged the previously identified relationship between mPAP and MPAD in populations with underlying ILD. This finding was initially reported in a more specific group of idiopathic pulmonary fibrosis patients by Zisman et al.¹¹. Devaraj and colleagues¹² found no correlation between mPAP measured by RHC (mPAP_{RHC}) and MPAD in a generalized population of patients suffering from diffuse pulmonary fibrosis.

Because of the complexity of SSc patients with regard to both ILD and PH, we sought to further analyze and clarify the correlation between mPAP_{RHC} and MPAD. In our study, we did not find the relationship between MPAD and PAP as measured by RHC to be attenuated by the presence of ILD. This suggests that MPAD may be a useful marker for defining the population of SSc patients that may benefit from more aggressive or invasive work up for PH. Our results differ from Condliffe et.al. who evaluated the correlation between mPAP_{RHC} and MPAD in a limited group of SSc patients with chest CT data gathered strictly under a pulmonary embolism protocol¹³. This study showed low correlation ($r=0.345$, $p=0.002$) between mPAP_{RHC} and MPAD in SSc in a group of 81 patients regardless of the extent of ILD. After excluding significant ILD, defined as either a disease extent > 20% according to a high-resolution CT scoring system set forth by Desai et al.¹⁶ or FVC < 70% predicted on PFTs, the remaining subset of 63 patients exhibited an improved correlation between mPAP_{RHC} and MPAD ($r=0.57$, $p<0.001$). Rajaram et. al. showed a similarly low correlation for CT derived MPAD with PH in connective tissue disease (predominately scleroderma) but did not study the extent or influence of ILD¹⁷.

One major difference between our study and that by Condliffe et al.¹³ is the method by which patients were divided prior to analysis of the correlation between mPAP_{RHC} and MPAD. GGO is a more frequent finding in SSc than in patients with other types of lung disease such as idiopathic pulmonary fibrosis¹⁶ and was highly prevalent in our sample. With these issues in mind and when considering the fact that the interstitial fibrosis score allowed for a more uniform division of groups, we chose to primarily evaluate the role of this CT-derived measure of ILD with regard to the previously described attenuation in the correlation between mPAP_{RHC} and MPAD. Our results show that the correlation between mPAP_{RHC} and MPAD is maintained despite the presence of mild to moderate interstitial fibrosis on chest CT as represented by a fibrosis score of 3 or less. Indeed, 75% of our

patients with fibrosis (Group B) had a score of 2, representing the moderate nature of this type of interstitial involvement (data not shown).

Despite the fact that CT-derived mild to moderate fibrosis did not influence the correlation between $mPAP_{RHC}$ and MPAD in our sample, we did find in a subset of subjects that PFT measures did. Values of FVC $\leq 70\%$ predicted were associated with a considerably lower correlation ($r=0.42$, $p=0.11$) between $mPAP_{RHC}$ and MPAD than values of FVC $> 70\%$ predicted ($r=0.69$, $p=0.002$). A possible explanation for this difference in correlation could be that the criterion of FVC $\leq 70\%$ predicted selects out patients with a greater extent of disease at chest CT¹⁸. In fact, 37.5% of our population with an FVC $\leq 70\%$ predicted also had a fibrosis score of 3. Thus, all patients in our study with a fibrosis score of 3, except for one, were incorporated into Group D. Notwithstanding this discrepancy in fibrosis score distribution when grouping patients based on FVC, it is unlikely that the increase in CT-measured fibrosis is solely responsible for the attenuation in correlation between $mPAP_{RHC}$ and MPAD seen in Group D. This is supported by the finding that the correlation coefficient between $mPAP_{RHC}$ and MPAD in patients with a fibrosis score of 3 ($r=0.71$, $p=0.07$) is relatively strong and trends toward statistical significance in spite of an extremely low sample size (data not shown). Therefore the appearance of fibrosis involving up to 50% of the lungs on CT does not appear to influence the correlation between $mPAP_{RHC}$ and MPAD in scleroderma patients. The revelation that FVC attenuates this correlation may reflect the relatively weak association between CT-measured pulmonary fibrosis and clinically available measurements of lung function in this population¹⁹.

It has been suggested that comparing pulmonary artery and aortic diameter may be superior to pulmonary artery diameter alone^{7,12,13}, however, we did not find MPAD/AD to strengthen the correlation with $mPAP_{RHC}$ in any group. MPAD/AD was most noticeably inferior to MPAD as a correlate of $mPAP_{RHC}$ in Group D. Group D also exhibited significantly larger MPAD values than Group C with no difference in AD or $mPAP$ between the groups. Presumably mediastinal traction^{7,11,20} combined with concomitant systemic arterial disease resulted in a larger aortic diameter and decreased ratio leading to the poor performance of MPAD/AD in Group D.

This study has several limitations including a patient sample that was gathered solely from a tertiary care center. Another major limitation was the lack of available PFT data for 14 of the 48 patients in this study. Therefore, subjects with PFT data may represent a subset of patients with more severe disease. In addition, patients were grouped for analysis based primarily on CT-derived measurements of fibrosis alone. As discussed earlier, this grouping was necessary in order to isolate the contribution of fibrosis to the attenuation in correlation between MPAD and $mPAP_{RHC}$. The interval between RHC and chest CT was another potential limitation to this study, although, up to 9 months between tests has been reported in the literature¹².

In summary, this study suggests that the presence of mild to moderate fibrotic lung disease on chest CT scan does not influence the correlation between $mPAP_{RHC}$ and MPAD in scleroderma patients. On the other hand, dividing patients according to FVC may identify those in whom MPAD is a poor predictor of $mPAP_{RHC}$. Based on our results, until FVC falls to 70% predicted, the MPAD measurement may retain utility for predicting PH in scleroderma patients. Furthermore, given a high positive predictive, the measurement of MPAD on chest CT holds promise in screening for PH screening even when mild to moderate fibrosis is present.

Acknowledgments

PJN and RMS were supported in part by a Multidisciplinary Clinical Research Center grant from the National Institute of Arthritis and Musculoskeletal and Skin Diseases, National Institutes of Health (Grant # P60AR049459).

REFERENCE

1. Steen VD. The lung in systemic sclerosis. *J Clin Rheumatol*. Feb; 2005 11(1):40–46. [PubMed: 16357695]
2. Trad S, Amoura Z, Beigelman C, et al. Pulmonary arterial hypertension is a major mortality factor in diffuse systemic sclerosis, independent of interstitial lung disease. *Arthritis Rheum*. Jan; 2006 54(1):184–191. [PubMed: 16385514]
3. Wigley FM, Lima JA, Mayes M, McLain D, Chapin JL, Ward-Able C. The prevalence of undiagnosed pulmonary arterial hypertension in subjects with connective tissue disease at the secondary health care level of community-based rheumatologists (the UNCOVER study). *Arthritis Rheum*. Jul; 2005 52(7):2125–2132. [PubMed: 15986394]
4. Chang B, Schachna L, White B, Wigley FM, Wise RA. Natural history of mild-moderate pulmonary hypertension and the risk factors for severe pulmonary hypertension in scleroderma. *J Rheumatol*. Feb; 2006 33(2):269–274. [PubMed: 16465657]
5. Haimovici JB, Trotman-Dickenson B, Halpern EF, et al. Relationship between pulmonary artery diameter at computed tomography and pulmonary artery pressures at right-sided heart catheterization. Massachusetts General Hospital Lung Transplantation Program. *Acad Radiol*. May; 1997 4(5):327–334. [PubMed: 9156228]
6. Tan RT, Kuzo R, Goodman LR, Siegel R, Haasler GB, Presberg KW. Utility of CT scan evaluation for predicting pulmonary hypertension in patients with parenchymal lung disease. Medical College of Wisconsin Lung Transplant Group. *Chest*. May; 1998 113(5):1250–1256. [PubMed: 9596302]
7. Ng CS, Wells AU, Padley SP. A CT sign of chronic pulmonary arterial hypertension: the ratio of main pulmonary artery to aortic diameter. *J Thorac Imaging*. Oct; 1999 14(4):270–278. [PubMed: 10524808]
8. Kuriyama K, Gamsu G, Stern RG, Cann CE, Herfkens RJ, Brundage BH. CT-determined pulmonary artery diameters in predicting pulmonary hypertension. *Invest Radiol*. Jan-Feb; 1984 19(1):16–22. [PubMed: 6706516]
9. Schmidt HC, Kauczor HU, Schild HH, et al. Pulmonary hypertension in patients with chronic pulmonary thromboembolism: chest radiograph and CT evaluation before and after surgery. *Eur Radiol*. 1996; 6(6):817–825. [PubMed: 8972316]
10. Moore NR, Scott JP, Flower CD, Higenbottam TW. The relationship between pulmonary artery pressure and pulmonary artery diameter in pulmonary hypertension. *Clin Radiol*. Sep; 1988 39(5):486–489. [PubMed: 3180668]
11. Zisman DA, Karlamangla AS, Ross DJ, et al. High-resolution chest CT findings do not predict the presence of pulmonary hypertension in advanced idiopathic pulmonary fibrosis. *Chest*. Sep; 2007 132(3):773–779. [PubMed: 17573485]
12. Devaraj A, Wells AU, Meister MG, Corte TJ, Hansell DM. The effect of diffuse pulmonary fibrosis on the reliability of CT signs of pulmonary hypertension. *Radiology*. Dec; 2008 249(3):1042–1049. [PubMed: 19011195]
13. Condliffe R, Radon M, Hurdman J, et al. CT pulmonary angiography combined with echocardiography in suspected systemic sclerosis-associated pulmonary arterial hypertension. *Rheumatology (Oxford)*. Aug; 2011 50(8):1480–1486. [PubMed: 21447566]
14. Preliminary criteria for the classification of systemic sclerosis (scleroderma). Subcommittee for scleroderma criteria of the American Rheumatism Association Diagnostic and Therapeutic Criteria Committee. *Arthritis Rheum*. May; 1980 23(5):581–590. [PubMed: 7378088]
15. Gay SE, Kazerooni EA, Toews GB, et al. Idiopathic pulmonary fibrosis: predicting response to therapy and survival. *Am J Respir Crit Care Med*. Apr; 1998 157(4 Pt 1):1063–1072. [PubMed: 9563720]

16. Desai SR, Veeraraghavan S, Hansell DM, et al. CT features of lung disease in patients with systemic sclerosis: comparison with idiopathic pulmonary fibrosis and nonspecific interstitial pneumonia. *Radiology*. Aug; 2004 232(2):560–567. [PubMed: 15286324]
17. Rajaram S, Swift AJ, Capener D, et al. Comparison of the diagnostic utility of cardiac magnetic resonance imaging, computed tomography, and echocardiography in assessment of suspected pulmonary arterial hypertension in patients with connective tissue disease. *J Rheumatol*. Jun; 2012 39(6):1265–1274. [PubMed: 22589263]
18. Goh NS, Desai SR, Veeraraghavan S, et al. Interstitial lung disease in systemic sclerosis: a simple staging system. *Am J Respir Crit Care Med*. Jun 1; 2008 177(11):1248–1254. [PubMed: 18369202]
19. Ooi GC, Mok MY, Tsang KW, et al. Interstitial lung disease in systemic sclerosis. *Acta Radiol*. May; 2003 44(3):258–264. [PubMed: 12751995]
20. Alhamad EH, Al-Boukai AA, Al-Kassimi FA, et al. Prediction of pulmonary hypertension in patients with or without interstitial lung disease: reliability of CT findings. *Radiology*. Sep; 2003 260(3):875–883. [PubMed: 21613438]

Figure 1.
Example of measurements made for main pulmonary artery and aortic diameter.

Figure 2.
Plots of Log mPAP vs. MPAD for Various Patient Groups

Figure 3.
ROC Curve for MPAD Measurement

Table 1

Patient Characteristics by Fibrosis Group

Variable	N(Group A:B)	Total	Group A	Group B	P-value
Age, years	20:28	56.3(13.1)	54.3(13.5)	57.7(12.8)	0.38 [¥]
Gender, % female	20:28	81.3	80.0	82.1	1.00 [*]
Race, % Caucasian	20:28	68.8	75.0	64.3	0.43 [∫]
BSA, m ²	20:28	1.8(0.2)	1.8(0.2)	1.8(0.2)	0.53 [¥]
ACR, % meeting criteria	18:25			88.0	0.63 [*]
Limited: Diffuse, %	17:22	66.7: 33.3	64.7: 35.3	68.2: 31.8	0.82 [∫]
MPAD, mm		32.4(4.5)	31.3(4.2)	33.2(4.6)	0.15 [¥]
AD, mm	20:28	31.4(3.5)	30.6(3.0)	31.9(3.7)	0.17 [¥]
mPAP _{RHC} , mmHg ^π	20:28	29.5 (26.3-33.1)	27.5 (22.4-33.9)	30.9 (26.9-35.5)	0.3 [¥]
PH, % with mPAP ≥ 25	20:28	66.7	65.0	67.9	0.84 [∫]
PCW _{RHC} , mmHg	20:28	10(3-35)	9.5(3-17)	11.5(4-35)	0.17 [€]
PAH, % of mPAP ≥ 25 with PCW ≥ 15	13:19	68.8	84.6	57.9	0.14 [*]
PVR _{RHC} , dyn [*] s/cm ⁵	18:26	288(86.5-1097.5)	166.5(94.2-1097.5)	327.7(86.5-744.6)	0.27 [€]
FVC, % predicted	14:20	73.1(20.8)	85.9(19.8)	64.2(16.7)	0.002 [¥]
DL _{CO} ^π , % predicted	14:19	39.8 (35.5-45.7)	50.1 (43.7-58.9)	33.9 (28.8-40.7)	0.001 [¥]

Data analyzed using the following:

[¥] Student's t-test

^{*} Fisher's Exact test

[∫] Chi-Square

[€] Wilcoxin Rank-Sum

^π Variable was log-transformed. Data represented as antilog of mean (confidence interval).

Table 2

Patient Characteristics By FVC Group

Variable	Total	Group C	Group D	P-value
Age, years	57.4(12.3)	61.7(11.7)	52.5(11.3)	0.03 [¥]
Gender, % female	73.5	77.8	68.8	0.7 [*]
Race, % Caucasian	70.6	71.4	70.0	1.00 [*]
BSA, m ²	1.8(0.2)	1.7(0.2)	1.8(0.2)	0.14 [¥]
MPAD, mm	31.4(4.0)	30.1(3.7)	33.0(3.8)	0.03 [¥]
AD, mm	31.3(3.2)	31.4(3.2)	31.3(3.2)	0.92 [¥]
mPAP ^π , mmHg	26.9 (23.4-30.2)	24 (20.4-28.8)	29.5 (24.6-36.3)	0.1 [¥]

Data analyzed using the following:

[¥] Student's t-test

^{*} Fisher's Exact test

^π Variable was log-transformed. Data represented as antilog of mean (confidence interval).

Table 3

Univariate Correlations

Variables	Group A		Group B		Group C		Group D	
	R	P-value	R	P-value	R	P-value	R	P-value
MPAD								
mPAP _{rhc}	0.68	0.001	0.7	<0.0001	0.69	0.001	0.42	0.11
PVR	0.62	0.01	0.49	0.01				
MPAD/AD								
mPAP _{rhc}	0.5	0.03	0.47	0.01		0.003	-0.09	0.73
PVR		0.03	0.09	0.67				
MPAD/BSA								
mPAP _{rhc}	0.45 [*]	0.05	0.59 [*]	0.001				
PVR	0.49	0.04	0.54	0.004				
FVC, % predicted								
mPAP _{rhc}	-0.52	0.06	0.1	0.69				
PVR	-0.43	0.16	0.35	0.14				
DL_{CO}^π, % predicted								
mPAP _{rhc}	-0.63	0.02	-0.08	0.75				
PVR	-0.51	0.09	-0.004	0.99				

mPAP_{RHC} was log-transformed for all univariate correlations.

^{*} Spearman's correlation coefficient.

^πVariable was log-transformed in addition to mPAP_{RHC}.