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Evaluation of Pulmonary Hypertension Using Computed Tomography Measurement of Pulmonary artery in Interstitial lung Disease Among the Patients Attending the Tertiary Care Hospital, South India

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ABSTRACT
Aim of this study is to determine how accurately various measures of the PA, as viewed on HRCT, predict right heart catheterization (RHC)-confirmed pulmonary hypertension. The present study was conducted in the Department of Radiology and 200 patients were included in the study. Patients with a CT scan within 90 days of MRI and RHC were included. In order to meet inclusion criteria, a diagnostic quality CT pulmonary angiogram (CTPA) with a slice thickness of <5 mm was required. 100 (50%) of 200 participants in our study had RHC-verified pulmonary hypertension and 25 mmHg mPAP. The pulmonary hypertension group had higher MPAD, RPAD, LPAD, and PA:Ao during both respiratory cycles than the non-hypertensive group. Non-pulmonary hypertension patients had a larger PA angle. The median MPAD in the pulmonary hypertension subgroup was 34.60 mm during inspiration and 34.65 mm during expiration. In contrast, non-pulmonary hypertension patients had a median MPAD of 30.00 mm during inspiration and 30.50 mm during expiration. The inspiratory MPAD and PA:Ao AUCs for RHC-confirmed pulmonary hypertension (Mpap $\geq$25 mmHg) were 0.741 and 0.750, respectively, for the entire group. The population's diagnostic traits were best when MPAD cut-offs were 32.5 mm and PA:Ao were 0.94. HRCT findings can aid in diagnosing pulmonary hypertension that has been verified by RHC. The sensitivity of MPAD was 29 mm, whereas the specificity of PA:Ao was 1.0. MPAD exhibited higher sensitivity in ILD compared to the overall cohort, while PA:Ao had greater specificity in COPD.

INTRODUCTION

Despite notable progress in the management of pulmonary hypertension (PH), there persists a substantial burden of morbidity and mortality[1,3]. Early identification of pulmonary hypertension (PH) may enhance results due to the availability of more efficient and secure pharmaceutical treatment for PAH[3]. Various screening algorithms have been suggested in order to expedite and enhance the prompt and precise identification of pulmonary hypertension (PH). These algorithms employ a blend of non-invasive techniques, including echocardiographic, physiologic (lung function), and radiologic methods. Prior to undergoing a definitive right heart catheterization (RHC) for confirmation, it is necessary to perform[4,5]. In patients with pulmonary hypertension (PH), computed tomography (CT) chest scans have predominantly replaced chest x-rays. This shift can be attributed, in part, to CT scan's capacity to detect thromboembolism in some instances, as well as their ability to identify diffuse parenchymal lung illnesses that may not be apparent in 15% of chest x-ray cases[6,7]. Due to the progress made in CT technology and its widespread accessibility, efforts have been made to utilise CT for the purpose of predicting the existence of PH. In comparison to the systemic arterial system, the pulmonary artery (PA) has a higher degree of compliance, rendering it more responsive to alterations in pressure and volume. Consequently, there should be a positive correlation between an elevation in mean pulmonary arterial pressure (MPAP) and the diameter of the pulmonary artery. Several dimensions of pulmonary artery (PA) have been investigated to determine if there is a correlation between the presence and severity of pulmonary hypertension (PH). These dimensions include PA diameter, cross-sectional area, diameter-to-bronchus ratio, diameter to pulmonary vein ratio, diameter-to-aortic diameter ratio and multiple regression methods assessing the dimensions of the main and branching pulmonary arteries[8-10].

CT scans are employed for the diagnosis and characterization of suspected ILD and are frequently included in the evaluation of patients with unexplained dyspnea and suspected PH[11]. The identification of dilatation in the main pulmonary artery (PA) or major branch arteries has been recognised as indicators of the existence of pulmonary hypertension (PH) and is frequently the initial imaging observation that indicates the diagnosis[12-16]. Given the prevalent utilisation of CT in the examination of individuals with ILD, it would be advantageous to employ the measurement of pulmonary artery size as a means of screening for the existence of pulmonary hypertension. CT pulmonary angiography is typically conducted without the use of ECG gating. The size of pulmonary arteries undergoes alterations throughout the cardiac cycle. Magnetic resonance imaging (MRI) is commonly employed to monitor the cardiac cycle and enables the evaluation of pulmonary arterial dimensions throughout both systole and diastole phases. Several authors have proposed that when lung fibrosis is present, the main pulmonary artery (PA) diameter may not accurately estimate the mean pulmonary arterial pressure. This is because individuals with pulmonary fibrosis experience dilatation of the main PA even in the absence of pulmonary hypertension (PH)[12,17]. The objective of this study was to assess the predictive accuracy of several parameters of pulmonary artery (PA) as observed on high-resolution computed tomography (HRCT) in relation to right heart catheterization (RHC)-confirmed pulmonary hypertension.

MATERIALS AND METHODS

The current study is a Hospital based observational study which was carried out in the Department of Radiology, over a period of one year with a sample size of 200 patients (Purposive sampling). Those included in the study were patients who underwent a CT scan within 90 days following MRI and RHC. To satisfy the inclusion criteria, it was necessary to get a CT scan of diagnostic quality, with a slice thickness of less than 5 mm. As part of their standard clinical assessment, patients completed a comprehensive evaluation that encompassed clinical assessment, multi-modality imaging, and lung function tests.

Radiographic Assessment: Volumetric CT imaging was performed on all patients during both full-inspiration and end-expiration. The reconstructed CT images were obtained with a slice thickness of 0.75 mm. Scans were obtained at a current range of 50-200 mAs and a peak voltage of 120 kV. The measurement of vessel dimensions was conducted via mediastinal windows. The procedure for measurements was devised, evaluated, and implemented by a radiologist and pulmonologist. Perpendicular to the wall bordering the Ao, the MPAD was measured at the broadest point of the major PA. The Ao was also assessed at this level in order to determine the PA: Ao ratio. The diameters of the left and right PA (LPAD and RPAD, respectively) were measured at their maximum widths following the bifurcation. The PA angle, which represents the angle between the primary PA at the bifurcation, was also measured. We employed different thresholds, such as the standard values MPAD 29 mm and PA: Ao 1.0, to evaluate the diagnostic precision of HRCT measurements for pulmonary hypertension validated by RHC[16,18,19]. A pulmonologist (P.R.) who was unaware of the presence of pulmonary hypertension and the results of the RHC evaluated all HRCT scans. In order to

evaluate the accuracy of measures conducted by the pulmonologist, a chest radiologist (A.O.) reviewed 50 scans that were randomly selected. The chest radiologist was also unaware of the presence of pulmonary hypertension and the findings of the pulmonary hypertension test (RHC).

Statistical Analysis: All statistical calculations were done using SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 24 for Microsoft Windows. Data were statistically described in terms of mean±standard deviation (±SD) and range, or frequencies (number of cases) and percentages wherever applicable. p-values < 0.05 were considered statistically significant.

RHC Haemodynamics: All pressure measurements were performed at end-expiration while patients were in the supine position and breathing spontaneously. We defined pulmonary hypertension as mean PA pressure (mPAP) ≥25 mmHg[18].

RESULTS AND DISCUSSION

This study included 200 scans from 200 patients; 100 (50%) had RHC-confirmed pulmonary hypertension, with mPAP ≥25 mmHg. Compared with the non-pulmonary hypertension group, the group with pulmonary hypertension had greater MPAD, RPAD, LPAD and PA: Ao in both respiratory cycles, whereas the PA angle was greater in the non-pulmonary hypertension group. In the subgroup with pulmonary hypertension, the median MPAD was 34.60 mm in inspiration and 34.65 mm in expiration, while in the non-pulmonary hypertension group it was 30.00 mm in inspiration and 30.50 mm in expiration. For the cohort as a whole, the areas under the receiver operating characteristic curves (AUCs) for inspiratory MPAD and inspiratory PA: Ao (for RHC-confirmed pulmonary hypertension defined as Mpap ≥25 mmHg) were 0.741 and 0.750, respectively. For the cohort as a whole, the cut-offs MPAD ≥32.5 mm and PA: Ao ≥0.94 yielded the most favourable diagnostic profiles. There were weak positive correlations between RHC-measured mPAP and inspiratory MPAD, RPAD, LPAD and (RPAD+LPAD)/2. There was moderate positive correlation between mPAP and PA:Ao. There was weak negative correlation between mPAP and the PA angle. In general, compared with inspiration, correlations were not as strong between mPAP and HRCT measurements taken in expiration. Compared with the ILD subgroup, in the subgroup with COPD, correlations were generally stronger between HRCT measures and Mpap. Pulmonary hypertension is a condition that has the potential to cause significant disablement. It can arise from various underlying conditions, including those that primarily impact the lung, heart, or liver, and can be idiopathic in nature. Pneumonia can also occur as a result of systemic autoimmune illness or recurrent thromboembolism. Despite the proliferation of targeted medicines, the morbidity and mortality associated with pulmonary hypertension continue to be significant[1,2]. Similar to other progressive and potentially curable illnesses, the timely identification and implementation of suitable treatment in pulmonary hypertension are of utmost importance in preserving or enhancing patients' quality of life and extending their lifespan. While right heart catheterisation (RHC) is considered the most reliable method for confirming pulmonary hypertension, it is invasive and not accessible in numerous locations worldwide[20].

We incorporated 200 scans from 200 patients, of which 100 (50%) had verified pulmonary hypertension with RHC, characterised by a mean arterial pressure of 25 mmHg. The group with pulmonary hypertension had higher levels of MPAD, RPAD, LPAD, and PA: Ao in both respiratory cycles compared to the non-pulmonary hypertension group. However, the PA angle was greater in the non-pulmonary hypertension group. The subgroup with pulmonary hypertension had

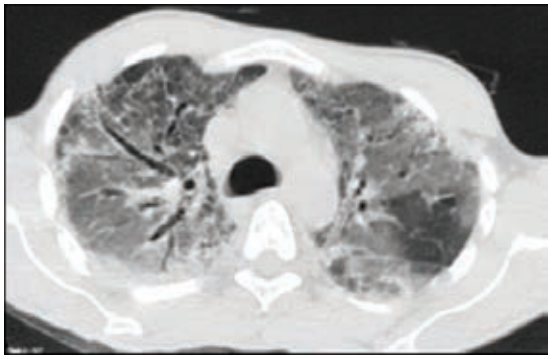

Fig 1: Image showing bilateral diffuse ground glass opacities and bronchiectasis-suggestive of interstitial lung disease

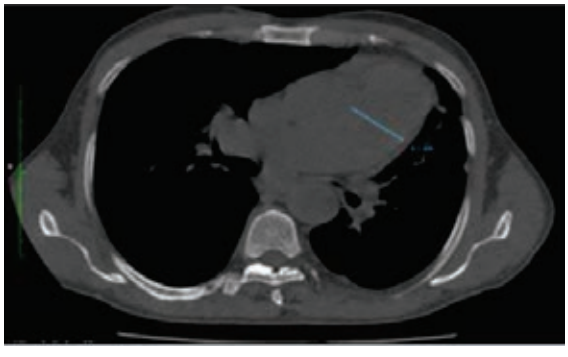

Fig 2: Image Showing Measurement of Main Pulmonary Artery

Table 1: Demographic profile and clinical characteristics of study participants

	Total n=200	Pulmonary hypertension group n=100	Non-pulmonary hypertension group n=100	p-value
Age years	70.01±15.15	71.09±15.67	70.96±14.54	0.760
Male	150	90	60	0.550
Height cm	168.00±14.50	167.10±14.20	169.00±14.99	0.180
Weight kg	83.00±30.71	84.37±30.46	78.47±29.99	0.002
BMI kg·m-2	29.31±9.42	30.03±9.97	28.20±8.46	<0.001
		Smoking history		
Nonsmoker	100	60	40	0.160
Current smoker	10	6	4	
Ex-smoker	90	50	40	
		Underlying disease		
COPD	140	80	60	0.033
ILD	130	70	50	0.384
Embolism	30	18	16	0.432
Heart disease	120	75	45	0.010
		Haemodynamics		
mPAP mmHg	27±13	33±12	20±5	<0.001
CO (TD) L·min-1	4.69±1.93	4.60±2.06	4.80±1.80	0.065
CO (Fick) L·min-1	4.60±1.75	4.59±1.81	4.65±1.63	0.458
PVR (TD) WU	3.02±2.69	4.09±3.28	2.11±1.11	<0.001
PVR (Fick) WU	3.10±2.64	4.00±3.26	2.18±1.31	<0.001
RAP mmHg	7±5	9±6	5±4	<0.001
PCWP mmHg	12±6	14±7	10±5	<0.001
		Inspiratory HRCT median (IQR)		
MPAD mm	32.75 (7.35)	34.60 (7.10)	30.00 (6.50)	<0.001
PA:Ao	0.95 (0.22)	1.01 (0.23)	0.87 (0.16)	<0.001
RPAD mm	25.95 (5.35)	27.00 (4.90)	24.30 (5.10)	<0.001
LPAD mm	25.20 (4.80)	26.15 (4.50)	24.10 (4.70)	<0.001
PA angle deg	84.45 (29.57)	80.17 (28.83)	90.67 (30.62)	<0.001
		Expiratory HRCT median (IQR)		
MPAD mm	32.80 (7.20)	34.65 (7.05)	30.50 (6.00)	<0.001
PA:Ao	0.94 (0.21)	0.99 (0.22)	0.86 (0.16)	<0.001
RPAD mm	25.50 (5.35)	26.60 (5.00)	23.80 (4.70)	<0.001
LPAD mm	25.60 (4.90)	26.10 (4.90)	24.20 (4.70)	<0.001
PA angle deg	94.44 (23.02)	92.99 (22.46)	98.56 (25.96)	<0.001

Table 2: Diagnostic performance of various high-resolution computed tomography-derived measures for pulmonary hypertension defined as mean pulmonary artery (PA) pressure ≥25 mmHg on right heart catheterisation for subgroups with chronic obstructive pulmonary disease (COPD) or interstitial lung disease (ILD)

	Subjects n	Sensitivity %	Specificity %	PPV	NPV
COPD					
Inspiration					
MPAD ≥29 mm	140	88.17	41.86	0.77	0.62
PA:Ao ≥1.0	140	50.54	88.37	0.90	0.45
MPAD and PA:Ao	140	50.54	88.37	0.90	0.45
MPAD or PA:Ao	140	88.17	41.86	0.77	0.62
Expiration					
MPAD ≥29 mm	140	90.11	37.50	0.77	0.63
PA:Ao ≥1.0	140	50.55	85.00	0.88	0.43
MPAD and PA:Ao	140	49.45	85.00	0.88	0.43
MPAD or PA:Ao	140	91.21	37.50	0.77	0.65
ILD					
Inspiration					
MPAD ≥29 mm	130	91.24	37.76	0.67	0.76
PA:Ao ≥ 1.0	130	52.55	80.61	0.79	0.55
MPAD and PA:Ao	130	50.36	84.69	0.82	0.55
MPAD or PA:Ao	130	93.43	33.67	0.66	0.79
Expiration					
MPAD ≥29 mm	130	91.97	32.98	0.67	0.74
PA:Ao ≥1.0	130	45.99	78.72	0.76	0.50
MPAD and PA:Ao	130	45.99	79.79	0.77	0.50
MPAD or PA:Ao	130	91.97	31.91	0.66	0.73

Table 3: Spearman correlation coefficients showing the relationship between various high-resolution computed tomography-derived measures for pulmonary hypertension defined as mean pulmonary artery (PA) pressure ≥ 25 mmHg on right heart catheterization

	Inspiration			Expiration		
	All	ILD	COPD	All	ILD	COPD
MPAD	0.479	0.456	0.574	0.444	0.415	0.534
RPAD	0.335	0.295	0.462	0.327	0.327	0.327
LPAD	0.339	0.323	0. 405	0.273	0.254	0.183
(RPAD+LPAD)/2	0.360	0.329	0.463	0.297	0.269	0.265
PA:Ao	0.507	0.489	0.579	0.488	0.461	0.564
PA angle	-0.241	-0.212	-0.246	-0.241	-0.327	0.316

a median mean pulmonary artery diameter (MPAD) of 34.60 mm during inspiration and 34.65 mm during expiration. In contrast, the non-pulmonary hypertension group had a median MPAD of 30.00 mm

during inspiration and 30.50 mm during expiration. According to a meta-analysis comprising 20 papers, the sensitivity and specificity of CT-measured MPAD in diagnosing pulmonary hypertension verified by right hemisphere cardiomyopathy (RHC) were found to be 79% and 83% respectively. Similarly, PA:Ao had a mean sensitivity of 74% and a mean specificity of 81%[21]. Overall, the meta-analysis of HRCT studies consistently demonstrated that the diagnostic efficacy of HRCT was inferior to that of standard CT or CTA. Our findings align with this observation[22].

Various threshold values have been suggested for detecting pulmonary hypertension confirmed by RHC, ranging from 25-38 mm for MPAD and from 0.84-1.4 for PA: Ao[22]. We choose to utilise the cut-offs suggested in the pulmonary hypertension guideline of the European Society of Cardiology/European Respiratory Society for our primary analysis[19-23]. However, we also conducted analysis utilising various values and determined that alternate cut-off values yielded superior results. Additionally, an analysis was performed to determine a lower threshold of 20 mmHg for mPAP, which might potentially be utilised as the threshold for pulmonary hypertension in subsequent studies[20]. Surprisingly, the overall outcomes were comparable regardless of whether we examined scans conducted within 1 week or within 2 months following the RHC. The outcomes for the subgroups exhibiting COPD or ILD were comparable to those observed for the entire cohort: MPAD exhibited a high level of sensitivity (>90%) for the ILD subgroup, but its specificity was poor. On the other hand, PA: Ao had low sensitivity but high specificity for both subgroups. The most favourable diagnostic profile for people with COPD was obtained using cut-offs of MPAD $\geq$32.5 mm and PA:Ao 0.90. The most favourable diagnostic profiles for people with ILD were obtained using cut-offs of MPAD $\geq$32.5 mm and PA: Ao 0.92. Given that HRCT is predominantly employed in patients with ILD, we suggest utilising these values as cut-off points for this specific patient population. Undoubtedly, the certification of our work will be necessary. ALHAMAD *et al*[17]. found MPAD $\geq$25 mm had a sensitivity of 86%, a specificity of 41% and yielded the largest AUC (0.65) among 100 subjects with various forms of ILD. Among 34 subjects without ILD, including eight with COPD, they found MPAD $\geq$31.6 mm had a sensitivity of 47%, a specificity of 93% and yielded the largest AUC (0.73).

CONCLUSION

The HRCT scan measurements of pulmonary artery diameter (PA) can provide indications of the presence or absence of pulmonary hypertension. These measurements can be classified as extremely sensitive (MPAD) or specific (PA:Ao), but not both simultaneously. MPAD had greater sensitivity in ILD, but PA:Ao demonstrated higher specificity in COPD. In patients undergoing HRCT, the level of worry for pulmonary hypertension may be increased or decreased based on inspiratory assessments for MPAD and PA: Ao. A MPAD measurement of 32.5 mm in a patient with ILD or COPD demonstrates a high level of sensitivity in confirming pulmonary hypertension in the presence of RHC.

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