Computed tomography chest imaging for the detection of pulmonary hypertension in patients with post-tuberculosis lung disease

M Almubarek,[1] MB ChB, MMed (Int Med), FCP (SA); **E H Louw,**[1] MB ChB, FCP (SA), Cert Pulmonology (SA);
S Griffith-Richards,[2] MB ChB, MMed (Rad D), FC Rad Diag (SA); **C Ackermann,**[2] MB ChB, MMed (Rad D), PhD;
N Baines,[1] BSc; **H Th mson,**[3] MB ChB, BSc, DTM&H, MRCP; **A J K Pecoraro,**[4] MB ChB, FCP (SA), Cert Cardiology (SA), PhD;
C F N Koegelenberg,[1] FCP (SA), FRCP, Cert Pulmonology (SA), PhD; **E M Irusen,**[1] MB ChB, FCP (SA), PhD;
B W Allwood,[1] MB ChB, FCP (SA), Cert Pulmonology (SA), PhD

[1] *Division of Pulmonology, Department of Medicine, Faculty of Medicine and Health Sciences, Stellenbosch University and Tygerberg Hospital, Cape Town, South Africa*
[2] *Department of Radiology, Faculty of Medicine and Health Sciences, Stellenbosch University and Tygerberg Hospital, Cape Town, South Africa*
[3] *Cleveland Clinic, London, UK*
[4] *Division of Cardiology, Department of Medicine, Faculty of Medicine and Health Sciences, Stellenbosch University and Tygerberg Hospital, Cape Town, South Africa*

Corresponding author: *M Almubarek (marwanalmobark89@gmail.com)*

Background. Pulmonary hypertension (PH) after tuberculosis is increasingly recognised as important in high-burden tuberculosis settings. However, the ability of computed tomography (CT) imaging to accurately detect PH remains unclear.
Objectives. To evaluate the performance of standard CT measurements in detecting PH in patients with post-tuberculosis lung disease (PTLD), and to determine the potential role of CT imaging as a screening tool in this population.
Methods. A retrospective study of patients with PTLD was conducted from January 2019 to September 2021. Adult patients with both a CT chest scan and an echocardiogram performed within 9 months of each other were enrolled. A diagnosis of PH by echocardiography was made if the right ventricular systolic pressure (RVSP) was ≥36 mmHg or the peak tricuspid regurgitant jet velocity (TRVmax) >2.8 m/s. Radiological criteria for PH included a pulmonary artery/ascending aorta (PA/AA) diameter ratio >1, pulmonary artery diameter (PAD) ≥29 mm (males) or ≥27 mm (females), and right ventricle/left ventricle (RV/LV) diameter ratio ≥1.28. Spirometry was also performed.
Results. Of 173 patients with PTLD, 52 met the inclusion criteria. Significant correlations were found between the CT-measured PA/AA ratio and RVSP (p=0.0083) and TRVmax (p=0.0582), but not between the CT-measured RV/LV ratio and RVSP (p=0.1729) or TRVmax (p=0.0749). PAD was also significantly correlated with RVSP (p=0.0011) and TRVmax (p=0.0023). The PA/AA ratio identified patients with PH on echocardiography with ~100% sensitivity, 65% specificity and a positive predictive value of 39.1%, indicating a high potential for false-positive diagnosis. The forced vital capacity was 13.7% lower in patients with PH than in those without (p=0.044); however, the forced expiratory volume in 1 second was not statistically different.
Conclusion. A low PA/AA ratio can be used to rule out the diagnosis of PH in PTLD, but a high PA/AA ratio requires further investigation for PH.

Keywords. Post-tuberculosis lung disease, pulmonary hypertension, computed tomography scan, echocardiography, pulmonary artery/ascending aorta ratio.

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Study synopsis
What the study adds. This study investigated the use of computed tomography (CT) chest imaging to detect pulmonary hypertension (PH) in patients with post-tuberculosis lung disease (PTLD). It revealed significant correlations between the CT-measured pulmonary artery/ascending aorta (PA/AA) diameter ratio and pulmonary artery diameter (PAD), and echocardiographic measures of PH. Notably, a low PA/AA ratio effectively rules out PH, while a high ratio warrants further investigation.
Implications of the findings. These findings suggest that CT imaging, particularly PA/AA ratio measurements, could serve as a valuable initial screening tool for ruling out PH in patients with PTLD, particularly in settings with limited access to echocardiography. However, a high PA/AA in PTLD requires confirmation of PH by other means, owing to a low positive predictive value.

Pulmonary hypertension (PH) is a group of diseases characterised by elevated pulmonary artery pressure, often leading to right heart failure and early mortality.[1,2] The definition of PH was revised in 2018, and a lowered threshold of mean pulmonary arterial pressure ≥20 mmHg was adopted.[3,4] Among the different classifications of PH, group 3 PH, secondary to chronic lung disease, is the second most common subtype.[2,3]

Chronic lung diseases such as chronic obstructive pulmonary disease (COPD), interstitial lung disease, and combined pulmonary fibrosis and emphysema are frequently associated with PH, which portends a poorer prognosis.[2,3]

Tuberculosis (TB), globally the leading cause of death from a single infectious agent,[5] primarily affects the lungs. If left untreated, TB can result in long-term damage, including extensive pulmonary destruction, chronic airflow obstruction, and destruction of the pulmonary vascular bed.[6] However, the association between post-TB lung disease (PTLD) and PH is under-reported in the literature.[1,2,6,7]

The development of PH following TB is believed to involve mechanisms such as pulmonary vascular remodelling, parenchymal pathology leading to destruction of the vascular bed, vasculitis, thrombosis of the pulmonary artery, endarteritis obliterans, and fibrosing mediastinitis.[6,8]

Diagnosing PH can be challenging owing to its nonspecific clinical manifestations.[9] However, timely and accurate diagnosis of PH is essential for prognosis and treatment planning.[9] Echocardiography, a non-invasive and widely available imaging modality, plays a key role in screening for and diagnosis of PH.[10,11] Raised peak tricuspid regurgitant jet velocity (TRVmax) together with other echocardiographic findings has been proposed as a reliable indicator of the probability of PH, as endorsed by the European Society of Cardiology (ESC), the European Respiratory Society (ERS) and the 6th World Symposium on Pulmonary Hypertension.[2]

Although echocardiography is the initial screening test of choice,[10,11] it may present challenges in patients with lung disease, owing to lung hyperinflation and difficulty in obtaining clear acoustic windows.[12] In contrast, computed tomography (CT) imaging enables comprehensive evaluation of the heart, pulmonary arteries, parenchyma and mediastinum, making it a valuable tool in the evaluation of suspected PH.[3,4,13] The presence of PH can be identified through anatomical vascular changes, such as a pulmonary artery/ascending aorta (PA/AA) diameter ratio >1, pulmonary artery diameter (PAD) ≥29 mm in males and ≥27 mm in females,[3,4,13] and right ventricle/left ventricle (RV/LV) diameter ratio ≥1.28.[3]

CT measurements predict the presence of PH with 70% sensitivity and 92% specificity compared with right heart catheterisation (RHC).[14] Echocardiography predicts the presence of PH with a sensitivity of 87% and a specificity of 79% compared with RHC.[15]

While the PA/AA ratio has shown promise in predicting PH in COPD,[12] its applicability in PTLD remains unclear.[12] The aim of this study was to investigate whether CT features correlate robustly with estimates of pulmonary artery pressures obtained through echocardiography in patients with PTLD.

Methods

Study design and patient selection

In this retrospective observational study conducted between January 2019 and September 2021, patients previously treated for pulmonary TB were eligible for inclusion if they were >14 years of age and had had an echocardiogram and a CT scan performed within 9 months of each other. This time frame was chosen because many patients have CT chest imaging conducted at secondary hospitals without simultaneous echocardiograms, frequently with subsequent prolonged delays in referral to our institution. The study was undertaken in the Division of Pulmonology at Tygerberg Hospital, a tertiary hospital in Cape Town, South Africa. Potential participants were identified from the departmental PTLD registry or from patients attending follow-up visits. They were excluded if they had an alternative known cause for PH (e.g. COPD, interstitial lung disease, obstructive sleep apnoea, autoimmune diseases, connective tissue disorders, collagen vascular disorders). In addition, potential participants were excluded if they had missing data, poor views or unreliable echocardiographic measurement and assessment, or a scan time interval >9 months, or if their CT scan was deemed uninterpretable for technical reasons (e.g. anatomical distortion).

Each participant had spirometry performed, and echocardiographic and CT chest data were used to assess for features indicative of PH. The data were interpreted by a pulmonologist, a radiologist and a cardiologist. Radiological criteria for diagnosis of PH were a PA/AA ratio >1, PAD ≥29 mm in males and ≥27 mm in females, and an RV/LV ratio ≥1.28. Echocardiographic criteria for PH were defined as a right ventricular systolic pressure (RVSP) ≥36 mmHg and a TRVmax >2.8 m/s. Although the gold standard for the diagnosis of PH is RHC, it was not required for inclusion. Ethical approval to conduct this study was obtained from the Human Research Ethics Committee of Stellenbosch University (ref. no. S22/08/141).

Measuring the PA/AA diameter ratio, PAD, and the RV/LV diameter ratio

Two radiology specialists independently measured the diameters of the PA, AA, RV and LV, and the average of the two measurements was calculated for each diameter and ratio. The diameters of the PA and AA were measured at a level proximal to the bifurcation of the PA, and for measurement of the RV and LV diameters, a perpendicular axis of the ventricular cavities from the endocardium to the interventricular septum on a standard axial image was measured, using the widest diameter for each chamber. In cases where the measurements of PA/AA and RV/LV ratios were discordant, a consensus read was conducted with both radiologists present. A discordant result was defined as a result where the readers' measured values resulted in a PA/AA or RV/LV ratio that was on opposing sides of the cut-points defined above. In these cases, a third consensus measurement was taken, and the final measurement was determined by averaging all three readings. This collaborative approach aimed to resolve discrepancies and improve measurement accuracy.

Echocardiography

Transthoracic echocardiography was used to evaluate cardiac function. Standard 2D and Doppler echocardiography was performed by a trained and certified echocardiography technologist. The ESC guidelines were followed to assess variables including systolic left ventricular function (Simpson biplane method), peak velocities of E wave and A wave, E/A ratio, right atrial pressure, tricuspid annular

plane systolic excursion, inferior vena cava diameter (inspiration and expiration), right ventricular ejection fraction, TRVmax and RVSP.

Spirometry

A trained technologist conducted spirometry based on standardised American Thoracic Society/ERS criteria.[16] Low forced vital capacity (FVC) was defined as values <80% predicted using the Global Lung Function Initiative 2012 reference ranges or below the lower limit of normal.[16]

Statistical analysis

Baseline data were presented as mean values along with their standard deviations (SDs) for normally distributed variables. Spearman correlation coefficients were calculated to assess the associations between PAD, RV/LV ratio, PA/AA ratio, RVSP and TRVmax. To measure the differences in PA/AA ratio, PAD and spirometric data (forced expiratory volume in 1 second (FEV_1), FVC) between groups with and without echocardiographic evidence of PH, an independent *t*-test was conducted, as PH was considered unlikely in patients with an RVSP <36 and a TRVmax <2.8. Further, a multiple linear regression analysis was used to investigate the correlations between CT chest image metrics (specifically PAD, PA/AA ratio and RV/LV ratio), RVSP and TRVmax.

Results

We assessed the records of 173 patients who had previously been treated for pulmonary TB. Of these, 121 patients were excluded, of whom 8 had left heart disease, 57 had alternative known causes for PH, 16 had echocardiography performed >9 months after the CT chest scan, and 40 had various other reasons for exclusion; 52 patients were therefore included (Fig. 1).

Table 1 summarises the baseline clinical characteristics. Participants had a mean (SD) age of 42.15 (12.60) years, with 27 males (52%) and 25 females (48%). Eight (15%) were HIV positive, 1 (2%) had diabetes mellitus, and 5 (10%) had hypertension. Only 14 (30%) were non-smokers, with 17 (36%) reporting current smoking. More than half (52%) reported two or more episodes of TB. The mean (SD) predicted FEV_1 was 42.9% (18.1%) and the mean FVC 59.6% (17.7%).

The 52 patients were separated into two groups, PA/AA ratio >1 (n=26) and PA/AA ratio ≤1 (n=26). There were no significant differences between the PA/AA ≤1 and >1 groups with regard to sex, smoking status, lung function, presence of obesity, HIV, hypertension, diabetes mellitus or number of TB episodes. However, in the high PA/AA group, there were significantly more participants aged 14 - 34 years (p=0.01).

Table 2 summarises the spirometric results and echocardiographic findings.

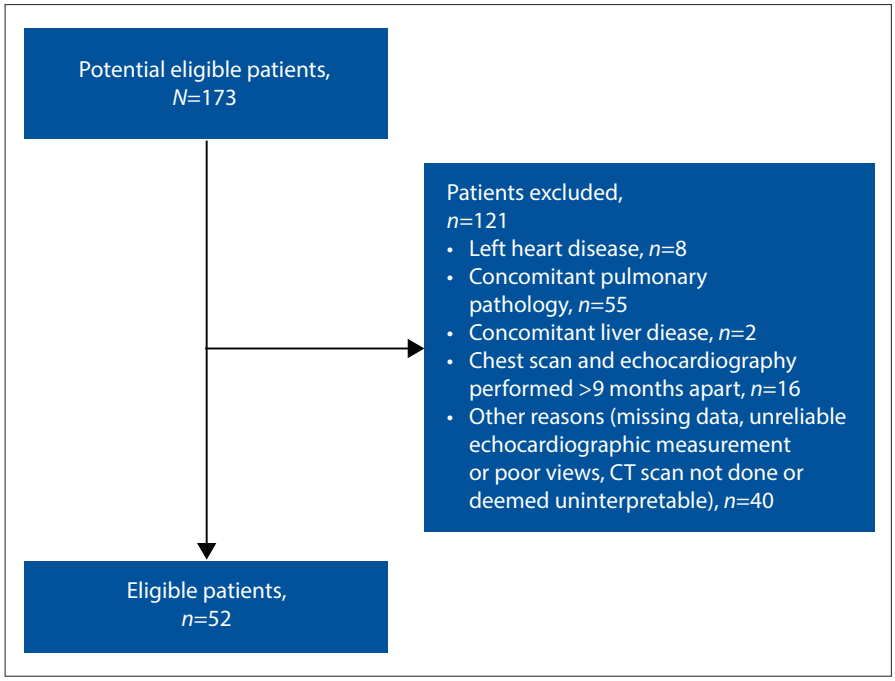

Fig. 1. Consort diagram of patients recruited. (CT = computed tomography.)

Echocardiography-measured RVSP was higher in the PA/AA >1 group than in the group with a ratio ≤1 (mean (SD) 37.5 (30.9) mmHg v. 19.8 (12.2) mmHg, respectively; p=0.05). In 17 patients RVSP could not be measured because no tricuspid regurgitant jet could be seen, and in the absence of alternative echocardiographic criteria, these patients were assumed not to have PH. Five (29%) of these patients had a PA/AA ratio >1.

The CT scan-measured PA/AA ratio correlated linearly with RVSP and TRVmax. A scatter plot displayed a clear positive linear correlation (Spearman's correlation coefficient 0.4420 (p=0.0083) and 0.3444 (p=0.0582), respectively), indicating a moderate positive correlation (Fig. 2). Similarly, RVSP was greater in patients with a raised PAD (p=0.007) and exhibited a significant positive correlation with PAD (Spearman's correlation coefficient 0.5343 (p=0.0011)) (Fig. 2). Additionally, TRVmax showed a robust correlation with PAD (Spearman's correlation coefficient 0.5345 (p=0.0023)).

Patients without echocardiography-measured PH were statistically more likely to have a normal RV/LV ratio on CT scan than those with PH (p<0.001). However, on further analysis there was no direct correlation of CT scan-measured RV/LV ratio with RVSP or TRVmax (Spearman's correlation coefficient 0.2507 (p=0.1729) and 0.3486 (p=0.0749), respectively), indicating at best a weak positive correlation for TRVmax (Fig. 2).

The PA/AA ratio was statistically greater in patients with echocardiography-measured PH compared with those without PH (PA/AA 1.25 v. 0.95, respectively; p<0.001). Similarly, the PAD was significantly greater in the group with PH than in the group without echocardiographic evidence of PH (34.2 mm v. 26.7 mm, respectively; p<0.001) (Fig. 3A).

Patients without echocardiographic PH had a 13.7% higher mean FVC (% predicted) compared with those with PH (mean (SD) 61.3% (16.3%) v. 47.6% (18.3%), respectively; p=0.044). This finding suggests a relationship between the presence of PH and reduced FVC. Interestingly, the same was not found for FEV_1, with no difference in FEV_1 (% predicted) found between those with and without echocardiographic PH (p=0.1543) (Fig. 3B). There was no significant difference in either the FEV_1 (% predicted) or FVC (% predicted) between patients with and

without a raised PA/AA ratio (p=0.1367 and p=0.3889, respectively) (Fig. 3C).

Diagnostic tests demonstrated that a PA/AA ratio >1 had ~100% sensitivity and 65% specificity for predicting the presence of echocardiographic PH, with a positive predictive value (PPV) of 39.1% and a negative predictive value (NPV) of 100%.

The RV/LV ratio demonstrated 50% sensitivity, 97.2% specificity and a PPV of 80%, while the NPV was high at 89.7%. PAD demonstrated 88.8% sensitivity and 42.5% specificity. The PPV was low at 25.8%, and the NPV was 94.4%.

Discussion

We explored the utility of CT chest imaging as a complementary tool to assist in the assessment of PH in patients with PTLD. We found significant correlations between the radiographic measurements of the PA/AA ratio and PAD and the echocardiographic measures of PH, namely RVSP and TRVmax. Patients with a raised PA/AA ratio had a 17.7 mmHg higher RVSP. In our population, the sensitivity of a PA/AA ratio >1 for detecting PH defined according to echocardiographic criteria approached 100%; however, the specificity (65%) and PPV (39.1%) were low, implying high false-positive rates. Additionally, we found significant correlations between some lung function parameters and echocardiographically measured PH, but not the PA/AA ratio.

PTLD often occurs in settings with a lack of access to specialised investigations, in particular echocardiography and RHC. A number of CT scan measurements have been used to predict PH with reported good sensitivity and specificity.[14,17] The PA/AA ratio is one such measurement, and has been tested in other respiratory diseases. In COPD, where echocardiographic measurement can be problematic owing to lung hyperinflation and difficulty in obtaining clear acoustic windows, the PA/AA ratio can predict the presence of PH with 73% sensitivity and 84% specificity.[12] However, in pulmonary fibrosis the predictive value of this CT measurement appears inadequate.[18,19]

In PTLD, no data inform use of the PA/AA ratio to determine the presence of PH.[1,6] Yet the PA/AA ratio is frequently reported in clinical practice with extrapolations of interpretation from other group 3 PH-related diseases, despite potential differences in

Table 1. Baseline characteristics of all participants, and patients with PA/AA ratios >1 and ≤1

Characteristics*	Overall (N=52), n (%)	PA/AA ratio ≤1 (n=26), n (%)	PA/AA ratio >1 (n=26), n (%)	p-value
Age (years) (mean (SD) 42.15 (12.60))				
14 - 34	11 (21)	1 (4)	10 (38)	0.01
35 - 44	20 (38)	11 (42)	9 (35)	
45 - 54	11 (21)	6 (23)	5 (19)	
55 - 74	10 (19)	8 (31)	2 (8)	
Sex				0.17
Male	27 (52)	16 (62)	11 (42)	
Female	25 (48)	10 (38)	15 (58)	
Comorbidities				
Hypertension	5 (10)	2 (8)	3 (12)	0.64
Diabetes mellitus	1 (2)	1 (4)	0 (0)	0.31
HIV	8 (15)	2 (8)	6 (23)	0.12
Obesity	2 (4)	1 (4)	1 (4)	1.00
Dyslipidaemia	3 (6)	3 (12)	0	0.074
Smoking status				0.98
Non-smoker	14/47 (30)	7/24 (29)	7/23 (30)	
Previous smoker	16/47 (34)	8/24 (33)	8/23 (35)	
Current smoker	17/47 (36)	9/24 (38)	8/23 (35)	
Smoking amount (pack-years)				0.64
≤10	12/31 (39)	7/15 (47)	5/16 (31)	
11 - 20	11/31 (35)	5/15 (33)	6/16 (38)	
>20	8/31 (26)	3/15 (20)	5/16 (31)	
Number of episodes of TB				0.20
1	25 (48)	12 (46)	13 (50)	
2	13 (25)	4 (15)	9 (35)	
3	8 (15)	6 (23)	2 (8)	
4	3 (6)	2 (8)	1 (4)	
5	1 (2)	0 (0)	1 (4)	
>5	2 (4)	2 (8)	0	

PA = pulmonary artery; AA = ascending aorta; SD = standard deviation; TB = tuberculosis.

*Percentages are calculated based on available data for each characteristic, as not all information was available for all participants. Denominators are provided for clarity when the totals for the characteristic differ from the column totals.

Table 2. Spirometry results and echocardiographic findi gs for all participants, and patients with PA/AA ratios >1 and ≤1

Characteristics*	Overall (*N*=52), *n* (%)†	PA/AA ratio ≤1 (*n*=26), *n* (%)†	PA/AA ratio >1 (*n*=26), *n* (%)†	*p*-value
Dyspnoea score				0.19
mMRC grade 1	12/40 (30)	8/20 (40)	4/20 (20)	
mMRC grade 2	13/40 (33)	4/20 (20)	9/20 (45)	
mMRC grade 3	15/40 (38)	8/20 (40)	7/20 (35)	
mMRC grade 4	0	0	0	
Spirometry				
FVC (L), mean (SD)	2.2 (0.8)	2.3 (0.8)	2.1 (0.7)	0.39
FVC (%), mean (SD)	59.6 (17.7)	60.6 (18.1)	58.5 (17.8)	0.70
FEV_1 (L), mean (SD)	1.5 (1.6)	1.4 (0.6)	1.6 (2.2)	0.63
FEV_1 (%), mean (SD)	42.9 (18.0)	46.3 (20.8)	39.7 (14.5)	0.23
FEV_1/FVC ratio, mean (SD)	59.6 (21.1)	60.0 (21.5)	59.3 (21.3)	0.91
Echocardiographic findings				
LVEF (%), mean (SD)	57.9 (5.7)	58.2 (3.4)	57.6 (7.4)	0.75
TRVmax (m/s), mean (SD)	2.2 (1.2)	1.8 (0.8)	2.4 (1.4)	0.16
RAP (mmHg), mean (SD)	5.7 (2.6)	5.0 (0.0)	6.4 (3.5)	0.083
RVSP (mmHg), mean (SD)	30.4 (26.4)	19.8 (12.2)	37.5 (30.9)	0.05
TAPSE (mm), mean (SD)	17.9 (3.3)	19.1 (2.7)	16.8 (3.5)	0.014
Tricuspid valve regurgitation absent	30 (58)	18 (60)	12 (40)	0.092
Tricuspid valve regurgitation present	22 (42)	8 (36)	14 (64)	
Pulmonary valve regurgitation absent	41/51 (80)	23/26 (88)	18/25 (72)	0.14
Pulmonary valve regurgitation present	10/51 (20)	3/26 (12)	7/25 (28)	
IVC findings				
IVC size (mm), mean (SD)	15.8 (4.2)	15.8 (3.5)	15.8 (4.6)	0.98
IVC collapse >50%	36/45 (80)	18/24 (75)	18/21 (86)	0.032
IVC dilated	3/45 (7)	0	3/21 (14)	
IVC not measured	4/35 (9)	4/24 (17)	0	
IVC not visualised	2/45 (4)	2/24 (8)	0	
RA size				
Normal size	40/46 (87)	23/23 (100)	17/23 (74)	0.009
Dilated	6/46 (13)	0	6/23 (26)	
RV function				
RVEF normal	37/44 (84)	22/23 (96)	15/21 (71)	0.028
RVEF abnormal	7/44 (16)	1/23 (4)	6/21 (29)	
RV size				
Normal size	40/47 (85)	23/24 (96)	17/23 (74)	0.035
Dilated	7/47 (15)	1/24 (4)	6/23 (26)	
Echocardiographic evidence of PH				<0.001
No	40 (77)	26 (100)	14 (56)	
Yes	9 (17)	0	9 (36)	
Unclear	3 (6)	0	3 (8)	

PA = pulmonary artery; AA = ascending aorta; mMRC = Modified Medical Research Council; FVC = forced vital capacity; SD = standard deviation; FEV_1 = forced expiratory volume in 1 second; LVEF = left ventricular ejection fraction; TRVmax = maximum tricuspid regurgitant velocity; RAP = right atrial pressure; RVSP = right ventricular systolic pressure; TAPSE = tricuspid annular plane systolic excursion; IVC = inferior vena cava; RA = right atrium; RV = right ventricle; RVEF = right ventricular ejection fraction; PH = pulmonary hypertension.
*Percentages are calculated based on available data for each characteristic, as not all information was available for all participants. Denominators are provided for clarity when the totals for the characteristic differ from the column totals.
†Except where otherwise indicated.

underlying pathology. In our population, we found that the PA/AA ratio was helpful when low (≤1.0), and was able to exclude the presence of PH, with an NPV approaching 100%. However, conversely, a raised PA/AA ratio was not able to rule in the diagnosis of PH with certainty, having a PPV of 39.1%. This finding implies that 6 out of 10 patients with a raised PA/AA >1 will not have echocardiographic PH. In our population of PTLD, a normal PA/AA ratio can therefore potentially be used to rule out the need for further investigation of PH. The imaging findings illustrated in Fig. 4 highlight increased PA/AA and RV/LV ratios in patients with and without PH.

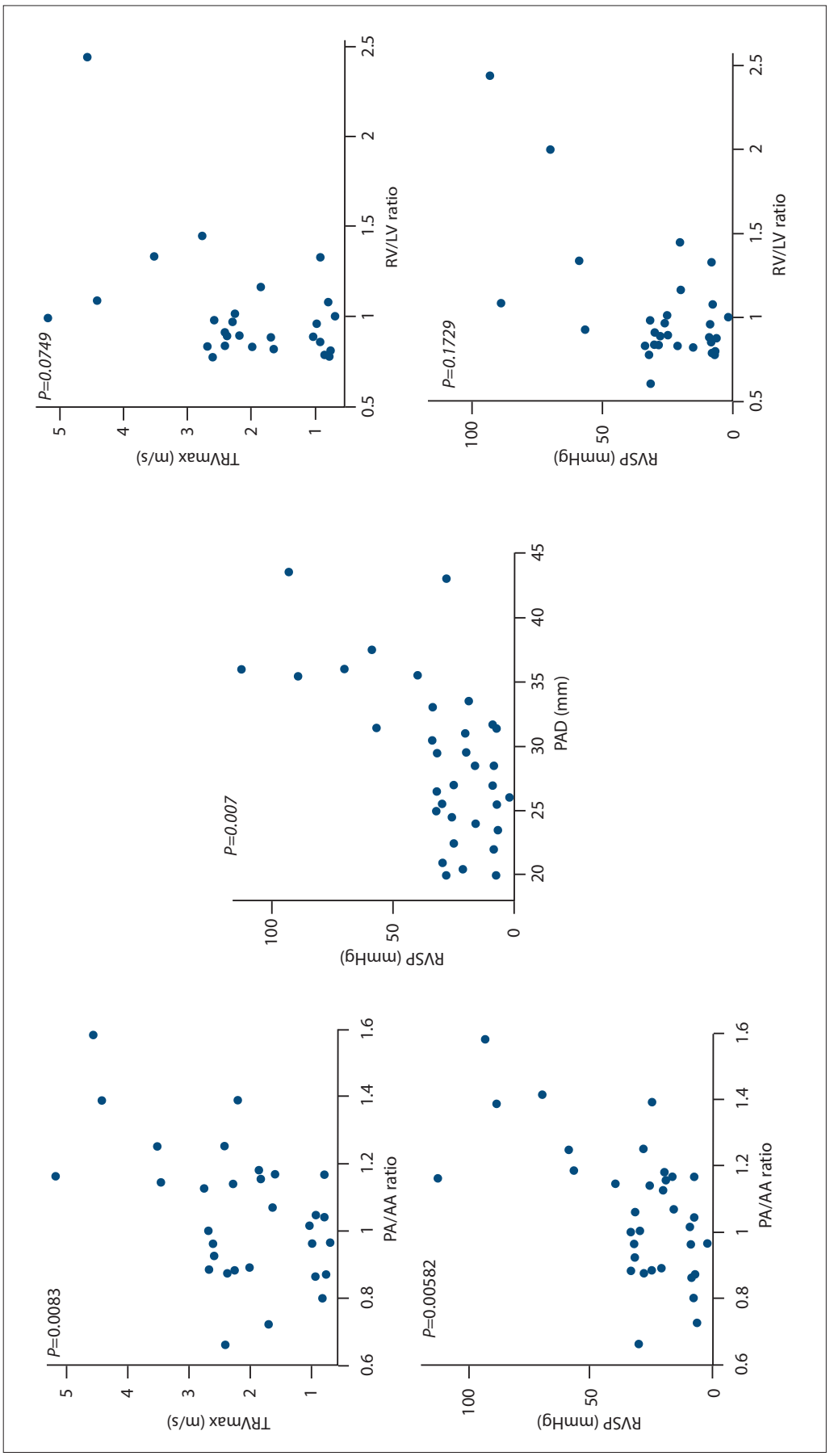

Fig. 2. Correlation relationships between PAD, RV/LV ratio and PA/AA ratio on CT scan and TRV/max on echocardiography. (PAD = pulmonary artery diameter; RV = right ventricle; LV = left ventricle; PA = pulmonary artery; AA = ascending aorta; CT = computed tomography; TRVmax = peak tricuspid regurgitant jet velocity.)

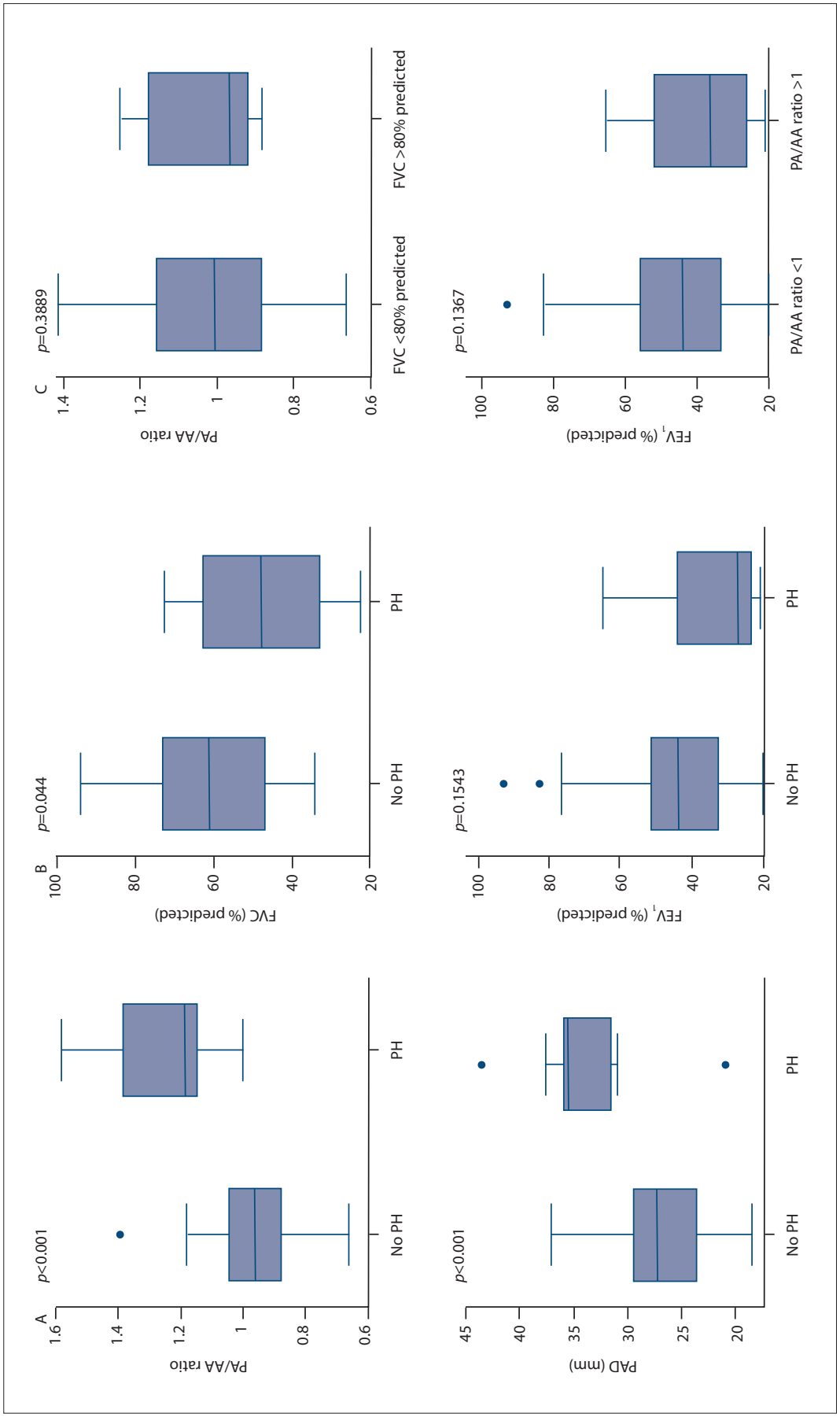

Fig. 3. Box-and-whisker plots comparing (A) PA/AA ratio and PAD in patients with and without PA, (B) FVC and FEV_1 predicted in patients with and without PH, and (C) FVC and FEV_1 predicted in patients with and without a raised PA/AA ratio. (PA = pulmonary artery; AA = ascending aorta; PAD = pulmonary artery diameter; FVC = forced vital capacity; FEV_1 = forced expiratory volume in 1 second; PH = pulmonary hypertension.)

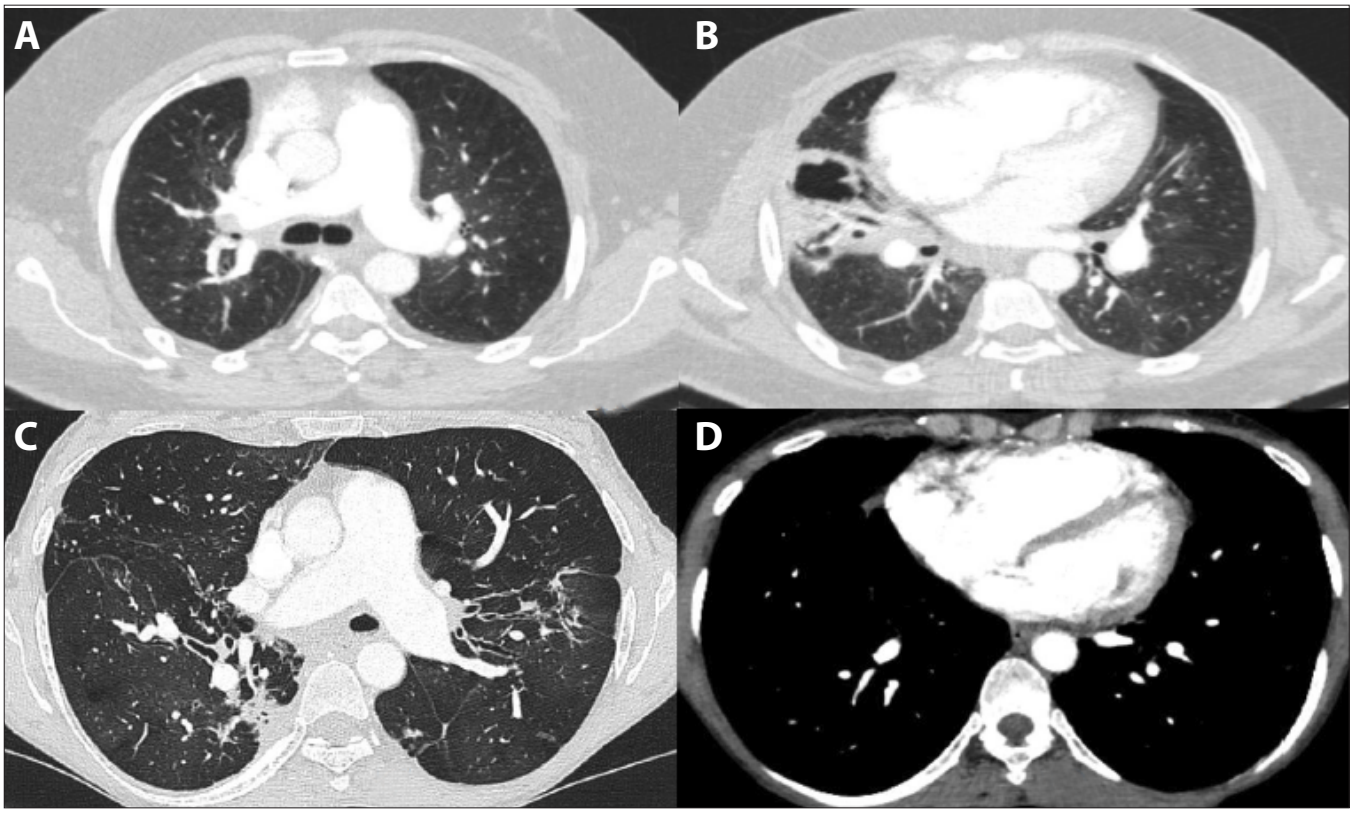

Fig. 4. A 50-year-old woman with one episode of previous TB has PH with a TRVmax of 3.52 m/s and an increased PA/AA ratio (A) and an increased RV/LV ratio (B). A 40-year-old woman with one episode of previous TB but no PH has a TRVmax of 1.85 m/s but an increased PA/AA ratio (C) and an increased RV/LV ratio (D). (TB = tuberculosis; PH = pulmonary hypertension; TRVmax = peak tricuspid regurgitant jet velocity; PA = pulmonary artery; AA = ascending aorta; RV = right ventricle; LV = left ventricle.)

The reason for the high false-positive rate of the PA/AA ratio is not clear. It is possible that the fibrotic destruction of the lung parenchyma adjacent to the pulmonary artery may play a role and cause distortion and dilation of the main pulmonary artery, in the absence of elevated pulmonary artery pressures. Disappointingly, we found no association between spirometric values and PA/AA that would have supported this hypothesis. This lack of association is likely to point to mixed causes of raised PA/AA, being due to raised pressures in some cases and anatomical defects in others, and requires further research. Interestingly, we did find an association between FVC and the presence of PH on echocardiography. This is in contrast to our previous study, which showed no association between spirometric findings and the presence of PH in a non-healthcare-seeking post-TB population.[20] The mechanism for PH after TB is unclear and may include lung fibrosis with loss of the capillary bed, a vasculopathy, increased thrombosis, and even mediastinal fibrosis.[6,8]

Other CT scan measurements have been assessed in the context of PH. Measurement of the RV/LV ratio in a general patient population predicts PH with 85.7% sensitivity and 86.1% specificity,[21,22] but usually requires cardiac gated images, which were not performed in our study. In interstitial lung disease, an increased RV/LV ratio reportedly predicts the presence of PH with 58% sensitivity and 70% specificity and is associated with increased mortality.[23,24]

In our study, the RV/LV ratio had a low sensitivity of 50%, but a specificity of 97.2%. The improved specificity compared with PA/AA is noted and could be helpful to rule in the diagnosis of PH, with a false-positive rate of 20% (PPV 80%).

Studies reported an average sensitivity and specificity for PH based on a dilated PA as 71.9% and 81.1%, respectively.[22,25] In our cohort, the PAD had sensitivity of 88.8%; however, the specificity of 42.5% and PPV of 25.8% were lower, implying high false-positive rates.

From our data, it appears that of the three CT measurements assessed, the PA/AA ratio may be the best screening measurement for excluding the diagnosis of PH in PTLD, but at the cost of a high false-positive rate.

These findings are potentially important in screening for PH in PTLD, as PTLD occurs more frequently in low- and middle-income countries, where access to echocardiography and RHC is limited, and CT scan imaging is marginally more available. Furthermore, CT scan measurements are less operator dependent than echocardiographic measures, and do not require the presence of good acoustic windows, which can be challenging in some patients with PTLD. Although CT imaging cannot replace either echocardiography or RHC in the diagnosis and management of PH, our data show that it may be helpful in ruling out PH and reducing the number of referrals for further investigation.

Study limitations and future directions

Limitations of this study include the small sample size, the retrospective design and the possibility of selection bias. Further, we acknowledge the absence of RHC to confirm PH diagnosis, which is considered the

gold standard test. Additionally, ECG gating on CT scanning, which can minimise motion artifacts, was not routinely performed; however, it is not performed on the majority of CT scans in settings where PTLD is prevalent, and therefore allows a real-world comparison.

Conclusions

This study highlights the potential of CT chest imaging, particularly the PA/AA ratio, as a tool for excluding significant PH in patients with PTLD. However, a raised PA/AA ratio will not be associated with echocardiographic PH in 60% of cases, and other mechanisms causing an increased PA/AA must not be forgotten in PTLD. These findings suggest that CT imaging may have an important role to play in limiting referrals for echocardiography in healthcare-limited settings. Further research with larger cohorts and prospective designs is recommended to validate these correlations and explore treatment options for PTLD patients with PH.

Data availability. The datasets generated and analysed during the present study may be available from the corresponding author (MA) on reasonable request, pending institutional review board approval.

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