Computed Tomography Angiography in Pulmonary Hypertension

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Abstract

Background: Diseases causing increased pulmonary pressure will subsequently cause a dilation of the pulmonary arteries and right heart chambers.

Objectives: To assess the capability of computed tomography angiography and high resolution CT to diagnose and estimate the severity of pulmonary arterial hypertension as compared with standard means of right heart catheterization, echocardiography and pulmonary function tests.

Methods: The study included 38 patients with PHT who underwent CT angiography and HRCT as part of their routine evaluation. Diagnoses included: primary PHT (n=20), Eisenmenger syndrome (n=6), scleroderma (n=3), thromboembolic disease (n=3), and others (n=6). Mean pulmonary artery pressure was 58 mmHg (range 39–92 mmHg) by catheterization and peak systolic pressure 79 mmHg (range 40–135) by echocardiography. Findings for the diameters of the main pulmonary artery and its main branches, the ascending aorta, the right atria and ventricle as well as the position of the interventricular septum were compared with 22 chest CT scans of patients with no known clinical history of pulmonary hypertension, performed for other reasons (trauma, oncology follow-up) during the study period. Correlations were also calculated with recent right heart catheterization, echocardiography and pulmonary function tests of the study group.

Results: Mean main pulmonary artery diameter in the study group was 3.55 ± 0.66 cm, pulmonary artery/ascending aorta ratio 1.2 ± 0.29, right pulmonary artery 2.63 ± 0.49 cm, left pulmonary artery 2.57 ± 0.5 cm. All diameters were significantly different from the control group (P < 0.0001). Main and right pulmonary artery diameters correlated with the pressure measurement by echocardiography (P = 0.001). Bronchial collaterals were found in 11 patients (30%). The position of the interventricular septum correlated well with the echocardiography study.

Conclusions: The size of the main pulmonary artery on CT angiography has a good predictive value regarding the severity of PHT.

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Pulmonary arterial hypertension is a progressive disease characterized by raised pulmonary artery pressure above 25 mmHg and pathological changes in the pulmonary precapillary vessels. New therapeutic strategies have considerably improved survival. However, the diagnosis of PHT can be missed because of non-specific signs and symptoms. Echocardiography is used for screening and diagnosis, but the gold standard measurement of pulmonary artery pressure and response to vasodilators is done by right heart catheterization. Computerized tomography angiography and high resolution CT are commonly used for the diagnosis of pulmonary embolism and underlying lung parenchymal disease. Chronic elevation of pulmonary artery pressure causes dilation of the pulmonary arteries right atrium and ventricle [1-5] [Figure 1]. Previous studies have reported a correlation between the ratio of the main pulmonary artery/ascending aorta diameters and the pressure measurement by right heart catheterization [6-8]. Asymmetric dilation of the pulmonary arteries, calcified thrombi and bronchial collaterals are also considered signs of chronic thromboembolic disease [8-10]. Deviation of the interventricular septum can be found on echocardiography and was reported on CT as a subjective sign of raised right heart pressure. HRCT of the chest can show a mosaic perfusion pattern, pulmonary scars, and underlying pulmonary disease [4,11].

The emergence of new treatments and the close follow-up required for patients with PHT emphasize the need for non-invasive imaging studies. The aim of this study was to assess the additional capability of CT angiography and HRCT to diagnose and estimate the severity of PHT compared to right heart catheterization, echocardiography and pulmonary function tests.

Patients and Methods

The study group consisted of 38 PHT patients who prospectively underwent CT angiography and HRCT for routine follow-up and evaluation. Diagnoses included primary pulmonary hypertension (n=20), Eisenmenger syndrome (n=6), scleroderma (n=3), chronic thromboembolic disease (n=3), hystiocytosis X (n=1), systemic lupus erythematosus (n=1), hepatopulmonary hypertension (n=1), and others (n=3). The most recent findings on right heart catheterization, echocardiography and pulmonary function test were retrieved from the patient’s files. CT angiography and HRCT were performed for the diagnosis of pulmonary embolism and underlying lung parenchymal disease.

The control group consisted of 22 patients who underwent chest CT with contrast medium during the study period, for reasons other than PHT (trauma and oncology follow-up). None of the control patients was known to have PHT.

Retrospective reading of examinations is under the approval of the Helsinki Committee in our institution, and does not require further institutional review board approval.
Results

The study group consisted of 38 patients, 27 women and 11 men, with a mean age of 52 years (range 20–80). Mean right heart catheterization and peak systolic pulmonary artery pressure measured 58.4 ± 18 and 92.5 ± 25 mmHg, respectively. Estimated systolic pressure by echocardiography was 79 ± 23.5 mmHg. Correlation between the catheterization and echo were noted (r = 0.6, P = 0.001). Other findings were as follows: forced expiratory volume in the first minute 72 ± 32 (% predicted), forced vital capacity 72 ± 59 (% predicted), carbon monoxide diffusion in the lung 58 ± 49 (% predicted), and 6 minute walk 352 ± 42 meters.

The control group comprised 22 patients, 7 women and 15 men, with a mean age of 60.9 (range 18–83). The study group had an older mean age and a higher male percentage.

Mean main pulmonary artery, right pulmonary artery and left pulmonary artery diameters in the study group were 3.55 ± 0.66 cm, 2.63 ± 0.49 cm and 2.57 ± 0.5 cm, respectively. Pulmonary artery/ascending aorta diameter ratio was 1.2 ± 0.29. All diameters were significantly different from the control group (P < 0.0001).

Within the study group, the pulmonary blood pressure measured by echocardiography correlated significantly with the diameters of the main pulmonary arteries, particularly the right main artery [Figure 2A & B]. The pressure measurements by right heart catheterization also correlated with the pulmonary artery diameters, but these findings were less significant [Figure 2C]. Although reverse correlations were found between the diameters and pulmonary function test measurements (FEV1 and FVC) [Figure 2D], the significance of this finding is limited due to the heterogeneity of the underlying disease in the study group.

Bronchial collaterals were detected in 11 of the 38 patients (29%) examined. These collaterals were noted in six patients with primary PHT, one with scleroderma, and four with Eisenmenger syndrome [Figure 3]. Calcifications were noted in 1 of the 38 patients (3%) and filling defects in 2 (5%). Within the limitation of non-cardiac gated CT angiography, we found straightened septum in 26/37 patients (70%), left deviation in 4/37 (10%), and normal right deviation in 7/37 (19%). When comparing the CT findings with the echocardiography reports, in 20 of 28 studies (72%) the echo showed similar findings [Figure 4].

HRCT findings were ground-glass opacities (23/38, 60%), mediastinal lymphadenopathy (14/38, 36%) (when combined with septal lines they may indicate the presence of veno-occlusive disease [13]), and pericardial and pleural effusion (9/38, 23%, and 8/38, 21%, respectively). Other findings were mosaic attenuation (5.2%) (related to differences in vascularity), fibrosis (15%), infiltrates (2.6%), and emphysema (7.9%).

Discussion

A strong correlation exists between the pressure measurements of pulmonary blood pressure measured by the “gold standard” – right heart catheterization and echocardiography – and CT angiography findings [1,15-20]. In addition, and in accordance with prior reports, we found concordance between the sizes of the pulmonary arteries seen on CT angiography and the presence of non-cardiac gated CT angiography, followed by HRCT scan.

Statistical analysis

Population characteristics, CT measurements, pulmonary function tests, pressure measurements by right heart catheterization and echocardiography are expressed as mean with standard deviation. Univariate correlations were evaluated using Pearson's correlation coefficient. P values less than 0.1 were considered significant. Student t-test was used to compare the study and control parameters. P values < 0.05 were considered significant, between 0.05 and 0.01 borderline, and above 0.01 non-significant.

Procedure

CT technique: Helical CT scans were obtained with a 16-slice detector unit (Phillips, CT, USA; Mx 8000 IDT 16). The pulmonary angiogram protocol consisted of intravenous injection of 80–100 ml non-ionic contrast media (iopromide 300, Ultravist; Schering, Berlin, Germany) at a flow rate of 3 ml/sec using a power injector. Slice width was 3 mm, increment size 1.5, and pitch 0.9, followed by HRCT scan.

Interpretation: The CT scans were interpreted by a consensus of two radiologists who were unaware of the patient's diagnosis and pulmonary artery pressure.

Assessment: Diameters of the main pulmonary artery, its right and left main branches, the ascending aorta, right atria and ventricle on CT angiography in the study group were compared with the CT angiography in the control group and correlated with the findings on recent right heart catheterization, echocardiography, pulmonary function tests and 6 minute walk. The position of the interventricular septum was estimated on the CT angiography axial and reconstructed images and graded as normal (deviated to the right ventricle), straight or deviated to the left ventricle [12]. The HRCT findings were summarized as absent or present.
of pulmonary hypertension. The CT findings correlated with the pulmonary blood pressure measurements by echocardiography and right heart catheterization.

We found a high incidence of bronchial collaterals (40% of our patients) including those with primary PHT, scleroderma and Eisenmenger syndrome. According to the literature, the presence of bronchial collaterals raises a high index of suspicion of chronic thromboembolic disease; they are not considered a common finding in primary PHT [13-16]. Ley et al. [15] reported that bronchial collaterals are not visualized on examination of normal patients because of their small diameter (<1.5 mm). However, in patients with chronic thromboembolic disease the flow within these collateral increases, causing them to dilate. Endrys and colleagues [16] found collaterals only in patients with thromboembolic disease and not in patients with primary PHT and concluded that their existence implies an embolic cause for the pulmonary hypertension. Remy-Jardin et al. [17], in a more recent study, found bronchial collaterals in primary PHT, but in a lower percentage than in those with chronic thromboembolic disease. Our relatively high rate of collaterals, therefore, may be attributable to the severity of the disease in our study group. The prominent collateral vessels seen in our patients may have originated as a consequence of the increased resistance to flow in the damaged pulmonary vasculature even in the absence of evidence of thromboembolism seen on CT angiography scan or lung perfusion scan (which was performed in every patient on the initial workup).

The position of the interventricular septum is used as an echocardiographic index of severity of raised pressure in the right atrium and ventricle and is considered a subjective sign in the interpretation of CT angiography. The normal position is slightly deviated to the right owing to the lower pressure in the right heart side. As the pressure rises, the septum straightens and even deviates to the left [12]. We found that deviation of the septum can be seen on CT, even without cardiac gating, and can be used as an additional

Figure 2. Linear (Pearson’s) correlation between pulmonary arteries’ diameters (cm) and pulmonary artery pressure (mmHg) and pulmonary function tests (%).

Figure 3. Bronchial collaterals in a patient with Eisenmenger syndrome [A & B] and a patient with scleroderma [C].
clue to the presence of pulmonary hypertension. The cases of mismatching between the CT estimation of the septal position and the echocardiography results may be attributed to inherent differences of the two imaging modalities and in particular the difference between a dynamic study like echocardiography and a static study like CT angiography. HRCT results were in accord with prior reports [4,11].

Conclusions

In the present series of patients with PHT, dilatation of the pulmonary arteries was very conspicuous and correlated with the severity of the PHT, thereby allowing for a confident diagnosis. We suggest that bronchial collaterals may develop in all types of PHT patients and not only in thromboembolic disease. Another common and reliable sign of severity is the position of the interventricular septum. Thus CT angiography can reliably be used, in addition to echocardiography, for the routine evaluation of patients with PHT. Further studies are needed to assess the utility of CT in the follow-up and in patients’ response to therapy.

References


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Goals are dreams with deadlines

Anonymous