**Original Research Article**

# Pulmonary Arterial Enlargement is Associated with Acute Chest Pain in Patients without Coronary Artery Disease

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## Abstract

**Background:** Pulmonary hypertension (PH) is an uncommon cause for chest pain in patients without significant coronary artery disease (CAD). Therefore, we studied the association between chest pain, right ventricular dimensions (RVDs), and PA size on coronary coronary tomographic angiography (CCTA).
**Materials and methods:** It was a prospective study done from the February 1, 2015 to August 31, 2015. Total of 98 patients were identified, 67 in the chest pain and 31 in the non-chest pain group.
**Results:** Patients with chest pain without CAD showed markedly dilated right atrial and ventricular chambers compared with standard parameters. PAD was measured as 24.81± 0.47 mm in the chest pain group and 21.91 ± 0.41 mm in the control group ($P < 0.05$). Odds ratio between chest pain and a significantly higher PAD was 10.11 (2.76-41.91, $P < .05$), 10.33 (2.15-61.41, $P < .05$) after adjusting for age, sex, BMI, history of HTN, HLP, CHF, COPD, OSA, and smoking. The chest pain group had an RAD1 of 47.19± 0.61 mm, RAD2 of 43.83 ± 1.79 mm, RVD1 of 37.91± 0.75 mm, RVD2 of 30.87± 0.73 mm, and RVD3 of 60.31± 1.1 mm. Based on the existing echocardiographic reference ranges, these measures fall within the upper limits of normal range. When comparing chest pain vs non-chest pain group, respectively, the mean RAD2 measured 39.98 ± 0.73 mm vs 33.78± 1.13 mm ($P = .005$), and the mean RVD2 measured 30.87± 0.73 mm vs 26.71± 1.73 ($P = .03$).
**Conclusion:** In patients presenting with chest pain without CAD on CCTA, there is a strong association between the presence of chest pain and enlarged PAD.

**Key words**

Coronary artery disease (CAD), Computerized coronary tomographic angiography (CCTA), Pulmonary artery.

## Introduction

Pulmonary artery (PA) enlargement is now following increasing trend dragging medical attention. Identification of PA enlargement on computed tomography (CT) of the chest done to assess patients with nonspecific cardiorespiratory symptoms which can raise the possibility of pulmonary hypertension. Earlier detection of PH improves outcomes and timely treatment [7]. Therefore, screening is done using combination of echocardiographic, pulmonary function tests and radiologic non-invasive techniques [1], before proceeding to the invasive yet confirmatory right heart (RH) catheterization. With the radiological advancements in computed tomographic (CT) scan to predict the presence of PH provide a potential for non-invasive methods for the diagnosis of the disease [2-4].

Latest statistics suggest that in India, there are roughly 30 million heart patients and two lakh surgeries are being performed every year. Chest pain is making it the most common complaint nationwide [5]. Patients presenting to the ED with chest pain constitute an important diagnostic challenge so coronary artery disease (CAD) can be ruled out by computerized coronary tomographic angiography (CCTA) based on evidence presented in clinical studies such as Rule Out Myocardial Infarction/Ischemia Using Computer Assisted Tomography (ROMICAT) trial [2]. Pulmonary hypertension (PH) is not a common cause of chest pain accompanying dyspnoea, which resembles angina pectoris in many presentation in location of pain , radiation, intensity, quality, such similarities can lead to misdiagnosis of CAD [3, 4]. Therefore, this study was conducted to investigate the relationship between chest pain and radiologic parameters associated with Pulmonary hypertension and such as pulmonary enlargement and right-sided cardiac morphology.

## Materials and methods

It was a prospectively study conducted in Sree Balaji Medical College and Hospital from the February 1, 2015 to August 31, 2015. The study protocol was approved by the institutional review board.

The study population was categorized into 2 groups: chest pain and non-chest pain (control) groups.

Inclusion criteria for the chest pain group were presented with chest pain, age >18 years, and underwent CCTA showing absence of obstructive CAD (defined as >50% coronary stenosis) and absence of detectable pulmonary embolism.

The non-chest pain group included patients with absence of chest pain or cardiopulmonary symptoms at initial presentation, age >18 years, and received CT chest.

Exclusion criteria for both groups: Patients less than 18 years of age or patients with prior history of obstructive CAD, coronary artery bypass surgery, percutaneous intervention, or acute coronary syndromes.

There was a total of 98 patients identified, including 67 in the chest pain and 31 in the non-chest pain group. Demographic and baseline characteristics collected include age, sex, race, body mass index (BMI), baseline left ventricular ejection fraction, past medical history of medical conditions including congestive heart failure (CHF), hypertension (HTN), hyperlipidaemia (HLP), chronic obstructive pulmonary disease (COPD), OSA, and smoking. Presence of electrocardiographic (ECG) evidence of RH strain was also obtained in the chest pain group including P-pulmonale, RV hypertrophy, right

bundle branch block, RV conduction delay (RVCD), and right axis deviation.

Computerized coronary tomographic angiography image data include PA size, and diastolic right atrial dimension (RAD) and RV dimension (RVD). The CT chest of control group was used to measure PA size using similar methods.

Statistical analyses were performed using SAS 9.4. Comparisons between chest pain and non-chest pain groups were done using contingency tables, $\chi^2$ test for dichotomous variables, and 2-tailed *t*-test for continuous variables. *P* value of <.05 was considered to be statistically significant.

## Results

There was a total of 98 patients participated in study 67 in the chest pain and 31 in the non-chest pain group. The demographic characteristics of the study participants in chest pain and non-chest pain groups were listed as per **Table - 1**. The chest pain group had a significantly higher BMI (P = .001), were more likely to have a history of HTN (P < .001), hyperlipidemia; (P = .03), and smoking (P < .001).

**Table - 1:** Demographic characteristics of the study population.

| Characteristics | Symptoms | | P value |
|---|---|---|---|
| | With chest pain (n = 67) | Without chest pain (n = 31) | |
| Age | 48.98 ± 0.99 | 51.96± 1.35 | 0.45 |
| Sex, % | | | |
| Male | 40 | 44 | 0.63 |
| Female | 60 | 56 | |
| systolic blood pressure | 138± 8.45 | 132± 7.45 | 0.13 |
| diastolic blood pressure | 86± 2.03 | 85± 2.16 | 0.92 |
| BMI | 31 ± 0.97 | 27± 0.88 | .001* |
| History of HTN, % | 55 | 18 | .001* |
| History of HLP, % | 25 | 7 | .03* |
| History of CHF, % | 1.1 | 0 | 0.57 |
| History of COPD, % | 11 | 1 | 0.007* |
| Smoking history, % | 39 | 3 | .0003* |

(HTN - hypertension; HLP - hyperlipidaemia; CHF - congestive heart failure; COPD - chronic obstructive pulmonary disease; OSA - obstructive sleep apnoea)

Pulmonary artery enlargement i.e. dilatation was significantly greater in the chest pain group, even after adjusting for the sex differences. The overall mean PAD was measured as 24.81± 0.47 mm in the chest pain group and 21.91 ± 0.41 mm in the control group ($P < 0.05$). Odds ratio between chest pain and a significantly higher PAD was 10.11 (2.76-41.91, $P < .05$), 10.33 (2.15-61.41, $P < .05$) after adjusting for age, sex, BMI, history of HTN, HLP, CHF, COPD, OSA, and smoking (**Figure – 1**). The chest pain group had an RAD1 of 47.19± 0.61 mm, RAD2 of 43.83 ± 1.79 mm, RVD1 of 37.91± 0.75 mm, RVD2 of 30.87± 0.73 mm, and RVD3 of 60.31± 1.1mm. Based on the existing echocardiographic reference ranges, these measures fall within the upper limits of normal range. When comparing chest pain vs non-chest pain group, respectively, the mean RAD2 measured 39.98 ± 0.73 mm vs 33.78± 1.13 mm ($P = .005$), and the mean RVD2 measured 30.87± 0.73 mm vs 26.71± 1.73 ($P = .03$). The rest of the RV measurements, as outlined in were not found to be significantly different between the 2 groups (**Table – 2**).

**Figure - 1:** Pulmonary artery diameter in both groups.

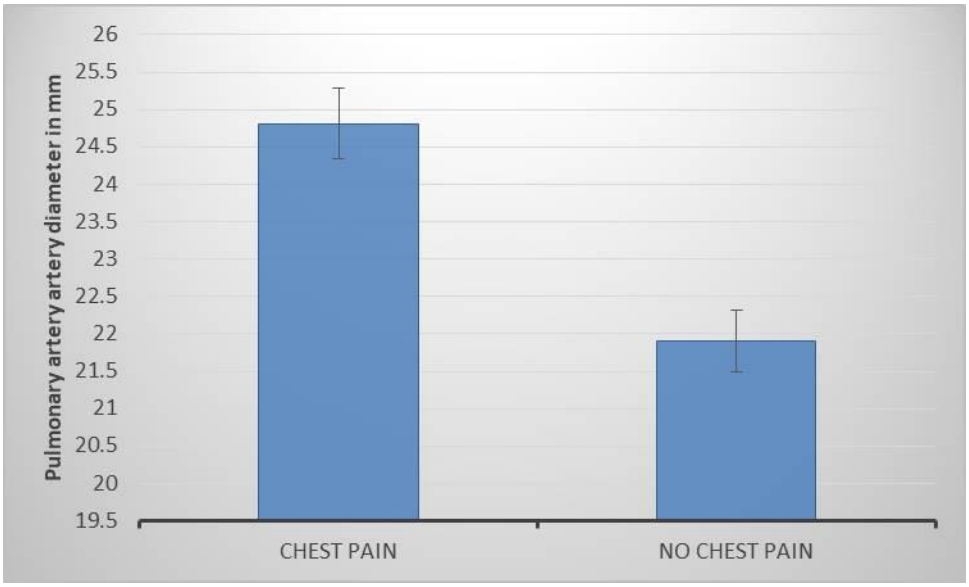


**Table - 2:** The mean PAD, RVD, and RAD of the study participants compared between chest pain vs non-chest pain groups.

| Radiographic measures, mm, ±SEM | With chest pain (n = 67) | Without chest pain (n = 31) | Value |
|---|---|---|---|
| PAD | 24.81± 0.47 | 21.91 ± 0.41 | <0.05* |
| RAD1 | 47.19± 0.61 | 43.83 ± 1.79 | 0.08 |
| RAD2 | 39.98 ± 0.73 | 33.78± 1.13 | .005* |
| RVD1 | 37.91± 0.75 | 34.71 ± 1.6 | 0.21 |
| RVD2 | 30.87± 0.73 | 26.71± 1.73 | .03* |
| RVD3 | 60.31± 1.1 | 55.45 ± 2.87 | 0.09 |

PAD - pulmonary artery diameter; RAD - right atrial dimension; RVD - right ventricular dimension.
RAD1 is a maximal RA cavity diameter measured parallel to tricuspid annulus.
RAD2 is the maximal RA diameter measured perpendicular to the tricuspid annulus.
RVD1 is a basal diastolic diameter of RV cavity at the level of tricuspid annulus.
RVD2 is the mid-RV cavity diastolic diameter.
RVD3 is a measurement of longitudinal extent of RV cavity that extends perpendicularly from tricuspid annulus to the RV apex.

## Discussion

The results of our study demonstrate an association between the presence of acute chest pain and enlarged PA dimensions. In agreement with previously reported studies. Evaluation of RV has also shown elevated diastolic chamber dimensions in patients presenting with acute chest pain and without obstructive CAD on CCTA. Furthermore, the association between the chest pain of possibly non-coronary cause and enlarged main pulmonary dimensions was clearly discernible, even after correcting for potential confounding variables including age, sex, BMI, history of HTN, HLP, CHF, COPD, and history of smoking [6, 7].

Many of the acute chest pain patients that present to the ED with low-to-intermediate probability of CAD which are ultimately ruled out by CCTA are left without an explanation to their clinical symptom or left with an umbrella diagnosis of "non-cardiac chest pain" at disposition. Our

study results illustrate the potential that a portion of these patients' acute chest pain may be related to the increased RH dimensions, especially PAD. Although PAD is not the gold standard method of diagnosing PH, it can serve as a screening tool to identify patients that are high risk for developing PH, thus leading to an earlier diagnosis and identification of the underlying causes, such as OSA. Multiple prior studies have shown that an increased PA size is associated with PH with a specificity of up to 100% [2-7]. One limitation to the outcome of our study is that the chest pain group had an average PAD of $25.92 \pm 0.43$ mm. However, most studies showing a high specificity for predicting PH using PAD used higher cut offs [8, 9].

Many studies like Kuriyama K, et al. [10] proved that non-invasive measurement of the diameter of pulmonary arteries, it can be of value in detecting pulmonary hypertension and estimating mean pulmonary artery pressure.

Burger IA, et al. [11] has showed that main pulmonary artery diameter from low-dose unenhanced multi-slice CT reliably predicts pulmonary hypertension, providing an important added clinical value from attenuation correction (AC) of single-photon emission computed tomography (SPECT) myocardial perfusion imaging (MPI). Ng CS, et al. [12] studied on diameters of the main pulmonary artery and of the ascending aorta (rPA), as assessed on computed tomography (CT), is predictive of pulmonary arterial hypertension. Mahammedi A, et al. [13] studied relationship between pulmonary artery diameter (PAD) as measured on computed tomography (CT) and pulmonary artery pressure (PAP) with the specific goal of assessing the reliability of various measurements on high-resolution chest CT as predictors of pulmonary hypertension (PH).

Although there are multiple causes of PH in the chest pain group (WHO groups I-V), our study examines potential contributory variable for the increased PAD and acute chest pain. This is illustrated by the significant differences in RV and proximal PA dimensions in the chest pain vs non-chest pain groups. Although we attempted to exclude patients with pulmonary emboli as a confounding variable, small pulmonary emboli not detected on CCTA may still be an underlying cause of PH in the chest pain group.

## Conclusion

There is a strong association between the presence of chest pain and enlarged PAD. The results of his novel study highlight the possibility of PH as potential contributors of acute chest pain in patients without obstructive CAD. Further studies using gold standard methods of diagnosing PH are encouraged to demonstrate the direct association between chest pain and these diseases in patients ruled out for obstructive CAD, with the potential for their early detection and treatment.

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