Original Article

HEALTH SCIENCES
MEDICINE

DOI: 10.32322/jhsm.1127534
J Health Sci Med 2022; 5(5): 1361-1365

The relationship between pulmonary artery obstruction index and troponin in thorax computed tomography in pulmonary embolism

Ercan Çil[1], Mahmut Çoraplı[2], Gülistan Karadeniz[3], Gökhan Çoraplı[1], Tuğçe Başbuğ Baltalı[4]

[1]Adıyaman University Training and Research Hospital, Department of Chest Diseases, Adıyaman, Turkey
[2]Adıyaman University Training and Research Hospital, Department of Radiology, Adıyaman, Turkey
[3]University of Health Sciences, Dr. Suat Seren Chest Disease and Surgery Training and Research Hospital, Department of Chest Diseases, İzmir, Turkey
[4]Adıyaman University Training and Research Hospital, Department of Internal Medicine, Adıyaman, Turkey

Cite this article as: Çil E, Çoraplı M, Karadeniz G, Çoraplı G, Başbuğ Baltalı T. The relationship between pulmonary artery obstruction index and troponin in thorax computed tomography in pulmonary embolism. J Health Sci Med 2022; 5(5): 1361-1365.

ABSTRACT

Aim: To study the relation between troponin and pulmonary artery obstruction index in thoracic computerized tomography in patients diagnosed with acute pulmonary embolism.

Material and Method: Data obtained from patients hospitalized in the ward and intensive care units with a pulmonary embolism diagnosis between January 2016 and February 2022 were scanned retrospectively. The full blood count, D-dimer, C-reactive protein, procalcitonin, troponin I, thoracic computerized tomography (CT), angiography, and bilateral lower extremity venous Doppler ultrasonography data were extracted. Patients with left heart failure, renal failure, gastrointestinal hemorrhage, sepsis, respiratory system disease, burns, ischemic stroke, or subarachnoid hemorrhage were excluded. The obstruction indices were calculated according to storage defects in the main, right, left, lobar, and segmental pulmonary artery branches in CT angiography.

Results: While 57.0% of the 69 patients included in the study were female, 42.1% were male. The obstruction index in the high troponin-I group was significantly higher than that in the normal troponin-I group (p=0.006). In addition, the obstruction index was significantly higher in patients with bilateral pulmonary embolism than in those with unilateral pulmonary embolism (<0.001). While there was a positive correlation between obstruction index and D-dimer (r=0.310 p=0.041), a significant negative correlation was found with the CRP value (r=-0.268 p=0.042). When the clinical and laboratory data of the patients with normal and high level troponin were examined, the ratios of patients in the ICU and with bilateral embolism were significantly higher in the high troponin group and the CRP value was significantly lower (p=0.004, 0.027, 0.003, respectively).

Conclusion: The pulmonary artery obstruction index shows thrombus load and is a parameter related to troponin.

Keywords: Pulmonary embolism, obstruction index, troponin I

INTRODUCTION

Pulmonary embolism is a disease that occurs as a result of occlusion of the pulmonary artery and/or its branches by fragments of thrombi formed in the deep veins of the legs. It is a disease of advanced age, observed on average among patients aged 50 years and over. With the placement of thrombi in the pulmonary arteries, a series of pathophysiological events occur and clinical findings emerge as a result. The patient's cardiopulmonary reserve, the diameter and number of occluded vessels, the size of the thrombus, reflex vasoconstriction due to pulmonary artery dilatation, and released inflammatory mediators affect these pathophysiological events (1). Pulmonary embolism is of significance for being a disease with high mortality and morbidity that can reappear, but that can be prevented (2).

Cardiac troponin I is an enzyme specific to the cardiac muscles. In cases of acute right heart failure in pulmonary embolism, the need for oxygen is increased due to right ventricle dilatation. Due to the decrease in right coronary artery circulation, microinfarctions could occur and troponin release from this region increases (3). High troponin is known as a bad prognostic marker in pulmonary embolism (4).

Corresponding Author: Ercan Çil, cil.ercan@hotmail.com
Received: 08.06.2022 **Accepted:** 01.08.2022

There are studies in which it is asserted that the benefits of thoracic computerized tomography (CT) angiography in pulmonary embolism are far greater than supposed. One helpful tool in this regard is the pulmonary artery obstruction index (PAOI), which is used to measure the degree of obstruction of the pulmonary arteries in thoracic CT angiography (5). The PAOI is a simple and reproducible index, and the number of studies conducted on it has increased in recent years. The aim of our study was to investigate whether there is a correlation between pulmonary artery obstruction index (PAOI) in acute pulmonary embolism and troponin.

MATERIAL AND METHOD

This study was approved by the Non-Interventional Clinical Research Ethics Committee of Adıyaman University (Date: 19.11.2019, Decision No: 2019/8-18). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The first admission data obtained from patients hospitalized in the ward and intensive care units of Adıyaman University Training and Research Hospital with a pulmonary embolism diagnosis between January 2016 and February 2022 were scanned retrospectively. The full blood count, D-dimer, C-reactive protein (CRP), procalcitonin, troponin I, thoracic CT, angiography, and bilateral lower extremity venous Doppler ultrasonography data were extracted. Normal values were accepted as 0-0.5 for CRP (mg/dL), <0.12 for procalcitonin (ng/mL), <0.001 for troponin (µg/L), and 80-560 for D-dimer (µg/L). High values were accepted as >0.5 for CRP, >0.12 for procalcitonin, >0.001 for troponin, and >560 for D-dimer. Patients with left-sided heart failure, kidney failure, gastrointestinal bleeding, sepsis, respiratory tract disease, burns, ischemic stroke, or subarachnoid hemorrhage of the relevant branch diagnosed by the physician, as both an additional comorbidity in the history of the patients and as a new diagnosis at the time of embolism, were excluded from the study. Patients who died within 120 days (3 months) of the diagnosis of acute pulmonary embolism were considered "nonsurvivors" cases. The Simplified Pulmonary Embolism Severity Index (sPESI) was considered as 0 points=low risk and ≥1 point=high risk with application of the following values: age >80 years=1 point, cancer history=1 point, history of heart failure and/or chronic lung disease=1 point, pulse >110/minute=1 point, systolic blood pressure <100 mmHg=1 point, arterial O_2 saturation <90%=1 point (1).

While complete blood counts were evaluated with the CELL-DYN Ruby System, procalcitonin, D-dimer, troponin I, and CRP values were measured with a Radiometer AQT90 Flex device.

CT Procedure and Image Interpretation

For imaging of all patients, images with 3-mm section thickness were obtained with the patients in supine position using a Toshiba Aquilion 64 CT scanner (Toshiba Medical, Tokyo, Japan). The following parameters were used during the imaging: 80-120 kV tube voltage, 60-120 mAs, and 16 × 0.75 mm beam collimation. The field of view used was approximately 40-50 cm (from lung apex to lung base). The 350 mg/ml contrast material was administered at 4-5 ml/sec via the antecubital vein, and the imaging started when the pulmonary artery exhibited storage with optimal contrast according to the contrast time curve. For pulmonary CT angiography, the patients were administered 80 to 120 ml of contrast material depending on their weight, and imaging was performed enabling the interpretation of images by storing optimal contrast in the pulmonary arteries and their branches. The institutional database system (Oracle database V1.10.43.134) was used in the interpretation of all images. All images were evaluated by an experienced radiologist with eleven years of expertise in the field. With the method used by Quanadli et al. (6), the main, right, left, lobar, and segmental pulmonary artery branches were evaluated in the mediastinal window (WW: 300-500 HU, WL: 50 HU) . On CT, a total of 20 lung segment arteries were given 2 points if the lumen of those with filling defects was fully occluded and 1 point if it was partially occluded. Those without filling defects were given 0 points. The PAOI was obtained by scoring between 1 and 40 while evaluating all CT images in three planes (axial, coronal, and sagittal).

Statistical Analysis

The statistical analyses were conducted using IBM SPSS Statistics 23.0 (IBM Corp., Armonk, NY, USA). The descriptive statistics were expressed as mean±standard deviation (minimum-maximum) and number (percentage). The chi-square test was conducted to compare the categorical data. For comparison of the normally distributed continuous data the independent Student's t-test was used, and the data were expressed as mean±standard deviation. The Mann–Whitney U test was used for comparison of the non-normally distributed data and the data were shown as median (minimum-maximum). Pearson's correlation test was used to determine correlation. P<0.05 values were considered statistically significant.

RESULTS

While 57.9% of the 69 patients included in the study were female, 42.1% were male. There was no statistically significant relation between PAOI and gender (p=0.088). PAOI was significantly higher in the high troponin-I group compared to the normal troponin-I group (p=0.006). In addition, PAOI was significantly higher in the group with bilateral pulmonary embolism than in the group with unilateral pulmonary embolism (<0.001). There were no significant differences in PAOI values between survivors and nonsurvivors cases, presence and absence of relapse, unilateral and bilateral DVT, normal and high CRP values, and normal and high procalcitonin values (p=0.258, 0.966, 0.233, 0.754, and 0.454, respectively). Patients with sPESI scores of 0 had a mean PAOI of 17.90±10.72, and patients with sPESI scores of ≥1 had a mean PAOI of 20.18±11.00 (p=0.433) (**Table 1**).

In Pearson's correlation test, while there was a positive correlation between PAOI and D-dimer (r=0.310 p=0.041), a significant negative correlation was found with CRP value (r= -0.268 p=0.042). No significant correlation was found between PAOI and age, troponin levels, platelet count or procalcitonin or sPESI score (p=0.404, 0.376, 0.429, 0.754, and 0.334, respectively) (**Table 2**).

When the clinical and laboratory data for the patients with normal and high levels of troponin were compared, the ratios of patients in the ICU and with bilateral pulmonary embolism were higher and CRP was significantly lower in the high troponin group (p=0.004, 0.027, 0.003, respectively) (**Table 3**).

Table 1. Obstruction index according to patient demographics

	Obstruction index	p	%95 CI (confidence interval)
Female (n:40) Male (n:29)	21.43±10.42 16.90±11.16	0.088 ¥	-0.697 to +9.754
Survivors (n=4) Nonsurvivors (n=65)	13.50±8.27 19.89±10.97	0.258 ¥	-4.781 to +17.565
Recurrence (+) (n=4) Recurrence (-) (n=65)	19.75±16.52 19.51±10.64	0.966 ¥	-11.524 to +11.039
DVT (+) (n=25) DVT (-) (n=44)	22.44 +8.88 17.86+11.65	0.094 ¥	-9.946 to +0.793
Troponin (µg/L) (High)(n=46) Troponin (µg/L) (normal) (n=23)	22 (2-37) 14 (2-40)	0.006*	Z: -2.740
CRP(mg/dL) (High) (n=50) CRP(mg/dL) (normal) (n=8)	18.90±10.98 20.25±12.93	0.754 ¥	-7.222 to +9.922
Procalcitonin (ng/mL) (High) (n=38) Procalcitonin (ng/mL) (normal) (n=22)	20.34±10.65 18.09±11.98	0.454 ¥	-8.228 to +3.726
Pulmonary embolism (Unilateral) (n=15) Pulmonary embolism (Bilateral) (n=54)	6 (2-28) 22 (2-40)	<0.001*	Z: -3.575
sPESI (Low risk)(n=20) sPESI (High risk)(n=49)	17.90±10.72 20.18±11.00	0.433	-8.068 to +3.500

¥: Independent student t test, mean±SD, *P<0.05; Mann-Whitney U test, median (minimum-maksimum),

Table 2. Obstruction index, age, CRP, troponin, D.Dimer, procalcitonin correlation table

	Age	CRP	Troponin	Platelet	D-dimer	Procalcitonin	sPESI
Obstruction index	r: 0.102 p: 0.404	r: -0.268 p: 0.042	r: -0.108 p: 0.376	r: -0.097 p: 0.429	r: 0.310 p: 0.041	r: 0.027 p: 0.839	r: 0.118 p: 0.334

(Pearson correlation test)

Table 3. Comparison of clinical and laboratory data of patients with normal and high troponin levels

	Troponin (Normal) (n=23)	Troponin (High) (n=46)	p
DVT[a]	10	15	0.432
CRP (mg/dL)[c]	9.40 (0.40-35.80)	2.30 (0.30-21.60)	0.003*
D-dimer (µg/L)[b]	7558±6861	12008±11312	0.196
Thorax CT angio (Unilateral filling defect /Bilateral filling defect)[a]	9/14	6/40	0.027*
Number of patients in the intensive care unit [a]	6 (26.1%)	30 (65.2%)	0.004*
Length of stay in intensive care unit (days)[b]	4.5±2.3	4.5±3.3	0.297
Mortality [a]	1 (4.3%)	3 (6.5%)	0.716
Time to Ex (days)[c]	0 (0-30)	0 (0-120)	0.467
Recurrence [a]	1 (4.3%)	3 (6.5%)	0.709

*P<0.05; a, chi-square test (n,%);b, Independent student t test (mean±SD); c, Mann-Whitney U test (median, minimum-maximum)

DISCUSSION

In our study, the PAOI was significantly higher in patients with high troponin levels and in patients with bilateral pulmonary embolism. Furthermore, there was a significant positive correlation between D-dimer and PAOI. In Shokoohi et al. (7), a correlation was found between troponin and PAOI in patients diagnosed with pulmonary embolism in the emergency department, and a significant correlation was found between high troponin-I values and pulmonary artery involvement. It is asserted that elevated troponin level could predict main pulmonary artery embolism with 53.8% (95% CI, 37.6-66) sensitivity and 92.3% (95% CI, 87-96.4) specificity. We found PAOI levels significantly higher in patients with high troponin. However, we found no significant correlation between troponin and PAOI. We think that the difference in the correlation results in our study compared to the study of Shokoohi et al. may be due to the differences in the characteristics of massive/submassive/nonmassive pulmonary embolism in the patient population.

In Gul et al. (8), troponin levels were correlated with both RV/LV ratio and PAOI. However, PAOI did not predict 30-day mortality. In our study, no difference was found in PAOI between survivors and nonsurvivors cases. There was a significant relationship between PAOI and troponin, but no correlation was found.

In Langroudi et al. (9), PAOI was significantly higher in patients with acute pulmonary embolism who died than in those who did not die (23.6 and 10.4, respectively) ($p<0.001$). In addition, they found that a PAOI value greater than 21.5 predicted hospital mortality with high accuracy. The reason for the lack of a significant difference between the survivors and nonsurvivors cases in our study could be the lower number of nonsurvivors cases. This may be due to the low number of nonsurvivors cases and the 120-day period considered for mortality in our study.

Apfaltrer et al. (10) conducted a prospective study in 50 patients diagnosed with acute pulmonary embolism and they evaluated three different pulmonary artery obstruction scores, called Mastora, Qanadli, and Mastora central. In none of these scores was a significant difference found between patients with and without adverse clinical findings. The authors concluded that the pulmonary artery obstruction scores were not correlated with adverse clinical findings. We found higher PAOI scores in patients with higher troponin values. In addition, we found a positive correlation between PAOI and D-dimer. We think that this correlation could be related to thrombus load. We found a negative correlation between PAOI and CRP. We attribute this finding to the high CRP values in patients with subsegmental embolism, especially in those with infarcted areas in the parenchyma (10), as in central-main pulmonary embolism the CRP values did not elevate significantly, and in contrast PAOI values increased in embolisms in the central-main arteries.

It has been proven that elevated troponin levels in pulmonary embolism are related to decreased survival in the long term, and it is regarded as an estimator of increased mortality (12). In our study as well, the ratios of patients in the ICU and with thrombus in the bilateral pulmonary arteries were significantly higher in the high troponin group. In addition, even though the mortality percentage was higher, it was not statistically significant. The reason for this could have been the smaller number of cases.

In the study of Soares et al. (13), no significant relationship was found between the vascular obstruction index and mortality, which is consistent with our study. In addition, there was no significant relationship between the vascular obstruction index and sPESI. We think that the results related to mortality and PAOI values are different due to differences in methodologies and the characteristics of the patient populations in these studies.

The most important limitation of our study is that it was a retrospective study. Moreover, the population of our study was relatively small. The patients could not be grouped according to massive/submassive/nonmassive pulmonary embolism due to lack of data.

CONCLUSION

The PAOI shows thrombus load and is a parameter related to troponin. D-dimer, another indicator of thrombus load, is positively correlated with the PAOI. In cases where the troponin value is not checked, PAOI can be used, at no additional cost.

ETHICAL DECLARATIONS

Ethics Committee Approval: This study was approved by the Non-Interventional Clinical Research Ethics Committee of Adıyaman University (Date: 19.11.2019, Decision No: 2019/8-18).

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version

REFERENCES

1. Arseven O, Okumuş N.G, Öngen G, Müsellim B. Pulmoner Tromboembolizm tanı ve tedavi uzlaşı raporu. Türk Toraks Derneği 2015: 1-3
2. Büber A, Altunrende Ş.S, Canan A. The relationship of pulmonary artery computed tomography obstruction index ratio, D-dimer and mean platelet volume levels in pulmonary embolism. Turkiye Klinikleri Arch Lung 2017; 18: 1-9
3. Pruszczyk P, Bochowicz A, Torbicki A, et al. Cardiac troponin T monitoring identifies high-risk group of normotensive patients with acute pulmonary embolism. Chest 2003; 123: 1947-52.
4. Becattini C, Vedovati MC, Agnelli G. Prognostic value of troponins in acute pulmonary embolism: a meta-analysis. Circulation 2007; 116: 427-33.
5. Alshumrani G, Al Bshabshe A, Mousa WF. Diagnostic yield of CT pulmonary angiography for pulmonary embolism in clinically suspected patients. Medicine (Baltimore) 2021; 100: 26213.
6. Qanadli SD, El Hajjam M, Vieillard-Baron A, et al. New CT index to quantify arterial obstruction in pulmonary embolism: comparison with angiographic index and echocardiography. AJR Am J Roentgenol 2001; 176: 1415-20.
7. Shokoohi H, Shesser R, Smith JP Hill MC, Hirsch R. The correlation between elevated cardiac troponin I and pulmonary artery obstruction index in ED patients with pulmonary embolism. Am J Emerg Med 2009; 27: 449-53.
8. Gul EE, Can I, Guler I, et al. Association of pulmonary artery obstruction index with elevated heart-type fatty acid binding protein and short-term mortality in patients with pulmonary embolism at intermediate risk. Diagn Interv Radiol 2012; 18: 531-6.
9. Langroudi TF, Shabestari AA, Hekmati S, Pourghorban R. Association between pulmonary arterial obstruction index and right lateral ventricular wall thickness with in-hospital mortality in patients with acute pulmonary embolism. Emerg Radiol 2021; 28: 327-31.
10. Apfaltrer P, Henzler T, Meyer M, et al. Correlation of CT angiographic pulmonary artery obstruction scores with right ventricular dysfunction and clinical outcome in patients with acute pulmonary embolism. Eur J Radiol 2012: 81: 2867-71.
11. Büyükşirin M, Anar C, Polat G, Karadeniz G. Can the Level of CRP in Acute Pulmonary Embolism Determine Early Mortality?. Turk Thorac J 2021; 22: 4-10.
12. Chauin A. The main causes and mechanisms of increase in cardiac troponin concentrations other than acute myocardial infarction (part 1): physical exertion, inflammatory heart disease, pulmonary embolism, renal failure, sepsis. Vasc Health Risk Manag 2021: 17: 601–17.
13. Soares TH, de Bastos M, de Carvalho BV, et al. Prognostic value of computed tomographic pulmonary angiography and the pulmonary embolism severity index in patients with acute pulmonary embolism. Blood Coagul Fibrinolysis 2013: 24: 64-70.