ORIGINAL ARTICLE / ÖZGÜN ARAŞTIRMA MAKALESİ

Clinical Significance of Endocan and Galectin-3 Levels in Massive Pulmonary Thromboembolism

Masif Pulmoner Tromboembolide Endocan ve Galektin-3 Düzeylerinin Klinik Önemi

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ABSTRACT

Aim: Pulmonary thromboembolism (PTE) is one of the most common cardiovascular emergencies in emergency departments. Identifying high-risk PTE and early treatment is crucial. We aim to investigate the effectiveness of Endothelial Cell-specific Molecule-1 (Endocan) and galectin-3 levels in the early diagnosis of PTE and in evaluating PTE severity.

Material and Methods: This prospective observational study was conducted between February 2020 and February 2021, including 52 patients diagnosed with PTE and 26 healthy volunteers. Serum endocan and galectin-3 levels were measured by Enzyme-Linked Immuno Sorbent Assay (ELISA). The severity levels of the PTE were determined according to the American Heart Association 2011 guidelines.

Results: The median serum endocan level was 175 (96-600) ng/L in the patient group and 168 (120-256) ng/L in the control group (U=506,000, p>0.05). The median serum galectin-3 level was 177 (100-768) pg/mL in the patient group and 164 (131-252) pg/mL in the control group (U=512.500, p>0.05). A statistically significant difference in endocan levels (H=71, p=0.003) and galectin-3 levels (H=92, p=0.014) was found among PTE severity groups according to the Kruskal-Wallis test. In massive PTE patients, its sensitivity was 77%, specificity was 89% for serum Endocan levels ≥185.45 ng/mL, its sensitivity was 65%, and specificity 89% for galectin-3 levels ≥179.16 pg/mL.

Conclusion: Endocan and galectin-3 biomarkers are not sufficient for the diagnosis of PTE. However, these biomarkers can guide clinicians in distinguishing massive PTE from submassive and low-risk PTE.

Keywords: Emergency medicine, endocan, galectin-3, pulmonary thromboembolism

ÖZ

Amaç: Pulmoner tromboemboli (PTE), acil servislerde en sık karşılaşılan kardiyovasküler acillerden biridir. Yüksek riskli PTE olgularının tanımlanması ve erken tedavisi hayati önem taşır. Bu çalışmada, Endotelyal Hücreye Özgü Molekül-1 (Endocan) ve Galektin-3 düzeylerinin PTE'nin erken tanısında ve hastalığın şiddetinin değerlendirilmesindeki etkinliğini araştırmayı amaçladık.

Gereç ve Yöntemler: Bu prospektif gözlemsel çalışma, Şubat 2020 ile Şubat 2021 tarihleri arasında gerçekleştirildi. Çalışmaya PTE tanısı alan 52 hasta ve 26 sağlıklı gönüllü dahil edildi. Serum endocan ve galektin-3 düzeyleri Enzim Bağlantılı İmmünosorbent Test (ELISA) yöntemiyle ölçüldü. PTE'nin şiddet düzeyleri, Amerikan Kalp Derneği'nin 2011 kılavuzlarına göre belirlendi.

Bulgular: Hasta grubunda ortanca serum endocan düzeyi 175 (96‑600) ng/L, kontrol grubunda ise 168 (120‑256) ng/L idi (U=506.000, p>0,05). Hasta grubunda ortanca serum galektin-3 düzeyi 177 (100‑768) pg/mL, kontrol grubunda ise 164 (131‑252) pg/mL idi (U=512.500, p>0,05). PTE şiddeti ile endocan düzeyi (H=71, p=0,003) ve galektin-3 düzeyleri (H=92, p=0,014) arasında anlamlı bir ilişki saptandı. Masif PTE hastalarında, serum endocan düzeyinin ≥185,45 ng/mL olması durumunda duyarlılık %77, özgüllük %89; galektin-3 düzeyinin ≥179,16 pg/mL olması durumunda duyarlılık %65, özgüllük %89 olarak bulundu.

Sonuç: Endocan ve galektin-3 biyobelirteçleri PTE tanısı için yeterli değildir. Ancak bu biyobelirteçler, masif PTE'yi submasif ve düşük riskli PTE'den ayırmada klinisyenlere yol gösterici olabilir.

Anahtar Kelimeler: Acil tıp, endocan, galektin-3, pulmoner tromboemboli

Received: 15 April 2025 Accepted: 1 September 2025

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Atıf için/Cited as: Semizoglu IS, Yucel M, Taskin MH, et al. Clinical Significance of Endocan and Galectin-3 Levels in Massive Pulmonary Thromboembolism. Anatolian J Emerg Med 2026;9(1):1-7. https://doi.org/10.54996/anatolianjem.1676577.

Introduction

Pulmonary thromboembolism (PTE) is a disease with high morbidity and mortality. PTE is caused by different types of occlusions in the pulmonary artery and its bronchial branches, leading to impaired blood supply. The disease's most common symptom is dyspnea; the most common finding is tachypnea (1). However, there are no specific symptoms and signs of PTE. Although diagnostic and imaging methods such as arterial blood gas, D-dimer, lower extremity venous doppler ultrasonography, echocardiography, and ventilation-perfusion scintigraphy are used as auxiliary aids in diagnosing PTE, the gold standard for diagnosis is computed tomographic pulmonary angiography (2). PTE can have a clinical course of low or high severity that can be fatal (3). These tests, which are used for the early diagnosis and treatment to reduce the mortality of PTE significantly, can be expensive, involve ionizing radiation, and require the use of contrast material (4-6).

Endocan (Endothelial Cell Specific Molecule-1 (ESM-1)) is a 50 kDa proteoglycan that provides information about endothelial function. It is mainly synthesized from lung and kidney endothelial cells. It is known that cytokines and proangiogenic factors such as tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), and vascular endothelial growth factor (VEGF) increase the secretion of endocan in inflammatory processes (7). It is known that it increases due to vascular endothelial activation, especially in cancers associated with organs such as the lung, kidney, breast, liver, and brain, and is also associated with cancer prognosis (8-11). It is also accepted as a marker of endothelial damage and evaluated as a new biomarker for atherosclerotic cardiovascular diseases (12).

Galectins are lectins with at least one carbohydrate-recognition domain and a high affinity for β-galactosides. Since they interact with extracellular matrix glycoproteins, they are involved in many processes, such as adhesion and migration. Galectin-3 is also a molecule of the galectin family, weighing approximately 30 kDa. It is found on the cell surface and cytoplasm of eosinophils, basophils, mast cells, neutrophils, and monocytes in peripheral blood (13,14). It has been reported that galectin-3 levels and galectin-3 positive cells increase in atherosclerotic lesions close to thrombosed areas. Galectin-3 levels can be used as a biomarker in cardiovascular diseases and heart failure (15,16). In addition, it has been shown that galectin-3 levels increase in diseases such as malignancy, migraine, chronic obstructive pulmonary disease, Lupus, and Behçet's Disease (16-19). Although it is associated with many diseases, galectin-3 can be considered a stable biomarker because it has properties such as being unrelated to age, gender, and body mass index and does not show circadian changes (20).

To the best of our knowledge, there are limited studies examining the relationship between endocan levels and PTE. In addition, we did not find any study examining the relationship between galectin-3 levels and PTE. This study aims to investigate whether serum endocan and galectin-3 levels are effective in diagnosing PTE and determining its clinical severity.

Material and Methods

Ethics committee approval and selection of the patient

This prospective observational study was carried out after obtaining ethical approval from the Samsun Training and Research Hospital Clinical Research Ethics Committee with the protocol number SBUSEAH-KAEK-2020/2/6. A power analysis was performed based on the data reported in the study by Kuluöztürk and colleagues. With a calculated effect size of 1.11, the minimum required sample size was 14 participants per group (α = 0.05, power = 0.80) (21). The study included individuals over 18 who presented to the emergency department between February 2020 and 2021 with a diagnosis of PTE. Those under the age of 18, patients having previous PTE disease or on active treatment for embolism, individuals using active steroids, patients with kidney failure, chronic lung disease or peripheral artery disease, those having a history of malignancy, patients with sepsis or with COVID-19 disease, and also patients with acute coronary syndrome and acute heart failure were excluded from the study. The study's control group consisted of individuals without any disease and a regular drug use history. The written informed consent form was taken from the patients and volunteers.

Recording of patient data

Demographic characteristics of the patients, vital signs (blood pressure, pulse, oxygen saturation), laboratory results obtained before treatment (troponin, D-dimer), imaging results (thorax tomography and echocardiographic findings), and clinical severity scores of PTE patients were recorded.

At the time of admission, the clinical severity levels of PTE patients were arranged according to the AHA 2019 guidelines. The patients were divided into three subgroups: 'massive', 'submassive,' and 'low-risk' (22). In addition, patients were evaluated by echocardiography and divided into three subgroups (normal, slightly dilated, and dilated) according to right ventricular (RV) dilatation status and three subgroups (main, segmental, and subsegmental) according to thrombus localization. Endocan and galectin-3 levels were compared between all subgroups.

Collection of blood samples

Blood samples were taken from patients diagnosed with PTE within 15 minutes after diagnosis and were placed in tubes without anticoagulant; it was centrifuged at 3500 rpm for 10 min. The plasma portion was taken into a separate container and stored at -80° C in the microbiology laboratory of our hospital. The kept samples were taken to room temperature and allowed to dissolve. Then, serum endocan and galectin-3 levels were measured by ELISA. In this process, the Human Endothelial Cell-specific Molecule-1 Elisa Kit (Bioassay Technology Laboratory, Shanghai, China) and Human galectin-3 Elisa Kit (Bioassay Technology Laboratory, Shanghai, China) were used, respectively. After the measurements were made at 450 nm in 10 minutes, the obtained data were recorded.

Statistical analysis

The obtained data were saved in Microsoft Excel and then transferred to SPSS and Medcalc package programs. While performing the statistical evaluation, the conformity of the data to the normal distribution was evaluated with the

Shapiro-Wilk or Kolmogorov-Smirnov test. The mean ± standard deviation for the numerical variables that fit the normal distribution, and the median (minimum-maximum) for the variables that did not fit the normal distribution; number (n) and percentage (%) expressions were also used for categorical variables. The student's t-test was used to compare numerical data with normal distribution in pairwise group comparisons, the Mann-Whitney U test was used to compare data that did not fit, and the Chi-square test was used to compare categorical data. In multi-group comparisons, the ANOVA test was used for data with normal distribution, Kruskal Wallis analysis of variance was used for data that did not fit, and the Mann-Whitney U test was used for Bonferroni correction. Spearman-Brown Correlation Test was used for correlation analysis. Receiver operating characteristic (ROC) analysis was performed to determine the sensitivity and specificity cut-off values. The statistical significance level for all analyses was accepted as p<0.05.

Results

Our study included a total of 78 individuals. Fifty-two were in the patient group, and 26 were in the control group. The mean age of the patients diagnosed with PTE was 68.15 ± 17.27 years, and the mean age of the healthy volunteers in the control group was 64.38 ± 6.4 years. 54% of both the patient and control groups were female. The patient and control groups were similar in age and gender characteristics (p=0.168 and p=0.594, respectively).

Table 1 displays the vital findings, laboratory findings, echocardiography, computed tomographic angiography findings, and severity scores of 52 patients diagnosed with PTE. Of the 52 patients, 9 (17.3%) belonged to the massive embolism clinic, 35 (67.3%) to the submassive class, and 8 (15.4%) to the low-risk class.

Examining the symptom distribution of PTE patients revealed that dyspnea was the most common symptom, accounting for 76.9% (Table 2). There was no significant difference between the patient and control groups regarding endocan and galectin-3 levels (p=0.072 and p=0.083, respectively) (Table 3).

The relationship between clinical severity, computed tomography findings, echocardiographic findings, and serum endocan and galectin-3 levels of PTE patients is shown in Table 4. In the echocardiographic imaging of these patients, endocan levels of patients with normal RV were lower than those with RV dilatation (p<0.05). There was no difference between the localization of thrombus and serum endocan levels. The relationship between the serum endocan levels of the patients and the severity of PTE was examined, and it was determined that the endocan levels of massive PTE patients were higher than both submassive and low-risk PTE patients (p=0.006 and p=0.001, respectively). Endocan levels of submassive PTE patients were also higher than low-risk PTE patients (p=0.007). There was no difference in galectin-3 levels in terms of both echocardiographic images and thrombus localization. Galectin-3 levels were found to be higher in patients with massive PTE than in patients with both submassive and low-risk PTE (p=0.018 and p<0.001, respectively). Galectin-3 levels in submassive PTE patients were also higher than in low-risk patients (p=0.035).

	Parameters		Median (minimum-maximum)
Vital signs	Pulse/min		105 (47-180)
	Saturation (%)		92 (67-99)
	Systolic blood pressure (mmHg)		110 (80-216)
	Diastolic blood pressure (mmHg)		70 (40-108)
Laboratory findings	Troponin (ng/mL)		0.097 (0.097-8.83)
	D-dimer (ng/mL)		4.61 (0.70-41.99)
Cardiac evaluation	PAP (mmHg)		40 (20-70)
	Group		**Frequency n, (%)**
	RV status	Normal	9 (17.3)
		Slightly dilated	21 (40.4)
		Dilated	22 (42.3)
Computed tomographic pulmonary angiography findings	Side of the thrombus	Right	13 (25.0)
		Left	8 (15.4)
		Bilateral	31 (59.6)
	The level of the artery where the thrombus is located	Main	17 (32.7)
		Segmenter	25 (48.1)
		Subsegmenter	10 (19.2)
Severity level of the disease	AHA 2019 Guidelines Classification	Massive	9 (17.3)
		Submassive	35 (67.3)
		Low risk	8 (15.4)

Table 1. Classification of patients with PTE, clinical and laboratory findings
AHA: American Heart Association, PAP: pulmonary artery pressure, PTE: pulmonary thromboembolism

The correlation analysis between the patients' serum endocan and galectin-3 levels and vital signs, pulmonary artery pressures (PAP), troponin and D-dimer results, and PTE risk scoring is presented in Table 5. Accordingly, serum endocan level and RV status (r=0.456, p=0.001), PAP (r=0.317, p=0.022), Wells score (r=0.341, p=0.013), Geneva score (r=0.410, p= 0.003) and heart rate (r=0.303, p=0.029), a moderately significant positive correlation was detected. The serum galectin-3 level was only positively correlated with the Wells score (r=0.298, p=0.032).

Symptoms	Frequency n, (%)
Dyspnea	40 (76.9)
Chest pain	7 (13.5)
Palpitation	6 (11.5)
Syncope	4 (7.7)
Peripheral edema	2 (3.8)
Consciousness change	2 (3.8)
Cough	2 (3.8)
Weakness	2 (3.8)
Nausea	2 (3.8)
Back pain	1 (1.9)

Table 2. Symptom distribution of patients with PTE.
PTE: Pulmonary thromboembolism

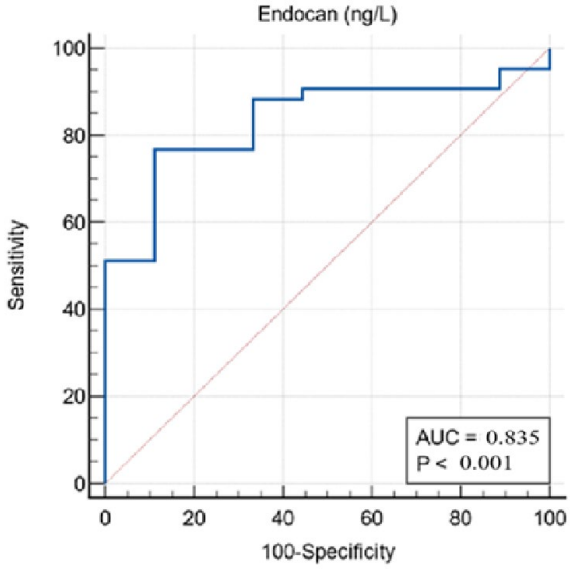

Figure 1. Endocan Levels in Differentiating Massive PTE from submassive and low-risk PTE.

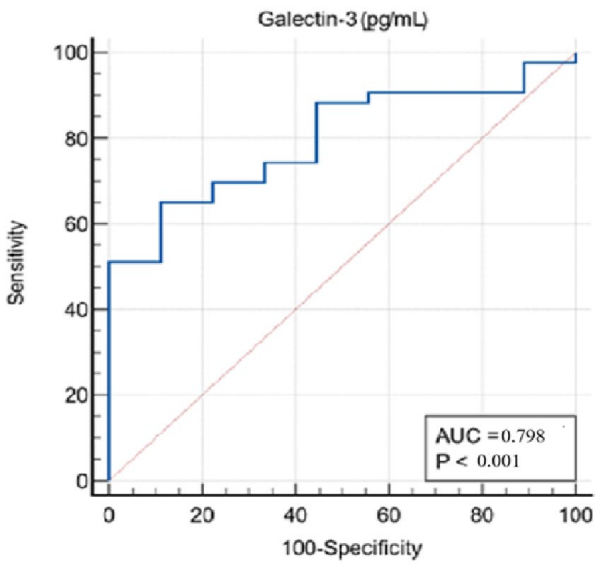

Figure 2. Galectin-3 Levels in Differentiating Massive PTE from submassive and low-risk PTE

In the ROC analysis, the clinical severity of PTE was found to be AUC 0.835 (95% CI: 0.706-0.923; p<0.001) for endocan. Serum endocan levels of ≤185.45 ng/mL distinguished massive PTE patients from submassive and low-risk PTE patients with 76.74% sensitivity and 88.89% specificity (Figure 1).

In the ROC analysis, the clinical severity of PTE was found to be AUC 0.798 (95% CI: 0.664-0.897; p<0.001) for galectin-3. It was determined that serum galectin-3 levels could distinguish massive PTE patients from submassive and low-risk PTE patients with ≤179.16 pg/mL values, 65.12% sensitivity, and 88.89% specificity (Figure 2).

Biomarker	Group	n	Median (minimum-maximum)	p
Endocan (ng/L)	Patient	52	175 (96-600)	0.072
	Control	26	168 (120-256)	
Galectin-3 (pg/mL)	Patient	52	177 (100-768)	0.083
	Control	26	164 (131-252)	

Table 3. Evaluation of Endocan and Galectin-3 levels between patient and control groups

Discussion

In this study, we aimed to investigate the value of serum endocan and galectin-3 levels in determining the diagnosis and clinical severity of PTE. According to the results we obtained, it is determined that although both endocan and galectin-3 are not useful biomarkers in diagnosing PTE, they may be effective biomarkers in determining the clinical severity of PTE. We also found that serum endocan levels were positively correlated with patients' heart rate, RV status, and PAP, and serum galectin-3 levels were also positively correlated with RV status.

Dyspnea is the most common clinical presentation of PTE patients. 97% of patients present with chest pain, tachypnea, and palpitations, in addition to shortness of breath (23). These findings, also known as major symptoms of pulmonary thromboembolism, are not specific to the PTE and may not be seen in almost 10% of PTE patients (24, 25). In our study, the most common presenting symptom was dyspnea. Chest pain was the second most common symptom.

Serum endocan levels are known to increase in endothelial-related diseases. Endocan levels were high, especially in cancers such as renal cell carcinoma and small cell lung cancer that metastasize by the hematogenous route. In addition, it has been observed that the level of endocan is increased in pathologies occurring on a vascular basis, such as atherosclerosis, coronary artery disease, chronic kidney disease, diabetes, migraine, and COVID-19 pneumonia (26). Serum endocan levels have also been studied in diagnosing PTE, and it has been reported that it can be a valuable parameter in the diagnosis phase (21, 27). Despite this, we found that serum endocan levels cannot be used for the diagnosis of PTE in this study. In this respect, our study contradicts the literature data. Therefore, it can be said that there is a need for more comprehensive studies evaluating PTE and endocan levels.

Group			Frequency n, (%)	Endocan (ng/L) median (min-max)	p	Galectin-3 (pg/mL) median (min-max)	p
Cardiac evaluation	Righ ventricle appearance	Normal	9 (17.3)	159.08 (140-174)		157 (121-210)	
		Slightly dilated	21 (40.4)	185.45 (147-472)	**0.005**	182 (137-391)	0.089
		Dilated	22 (42.3)	179.40 (96-600)		179 (100-768)	
Computed tomographic pulmonary angiography	Side of the thrombus	Right	13 (25.0)	172 (155-600)		179 (157-768)	
		Left	8 (15.4)	168 (140-227)	0.562	159 (129-231)	0.321
		Bilateral	31 (59.6)	176 (96-472)		179 (100-408	
	The level of the artery where the thrombus is located	Main	17 (32.7)	176 (96-600)		180 (100-768)	
		Segmental	25 (48.1)	168 (114-465)	0.692	174 (114-408)	0.779
		Subsegmental	10 (19.2)	177 (140-472)		177 (129-373)	
Severity of the pulmonary thromboembolism	AHA 2019 Guidelines Classification	Massive	9 (17.3)	216 (174-465)		219 (174-408)	
		Submassive	35 (67.3)	175 (96-600)	**0.000**	178 (100-768)	**0.002**
		Low risk	8 (15.4)	158 (140-176)		157 (129-174)	

Table 4. The relationship between clinical severity, computed tomography findings, echocardiographic findings, and serum Endocan and Galectin-3 levels of pulmonary thromboembolism patients
AHA: American Heart Association

		Endocan*	Galectin-3*
Systolic blood pressure	r	0.088	0.169
	p	0.533	0.230
Diastolic blood pressure	r	0.086	0.146
	p	0.546	0.301
Pulse/min	r	0.303	0.182
	p	**0.029**	0.197
Saturation (%)	r	0.191	0.088
	p	0.175	0.534
RV status	r	0.456	0.308
	p	**0.001**	**0.026**
PAP (mmHg)	r	0.317	0.256
	p	**0.022**	0.067
Troponin (ng/dL)	r	0.127	0.071
	p	0.368	0.615
D-Dimer (ng/dL)	r	0.069	0.046
	p	0.627	0.745

Table 5. Correlation of Serum Endocan and Galectin-3 Levels of Patients with Various Findings
* Spearman-Brown correlation test, PAP: pulmonary artery pressure, RV: right ventricle.

Our results showed that endocan levels were significantly higher in massive PTE cases, and levels above 185.45 ng/mL may indicate massive presentation with 76.74% sensitivity and 88.89% specificity. These findings may help clinicians in early risk stratification. It is well-known that the early diagnosis and treatment of the PTE positively affect mortality (21).

We recorded echocardiography data, including RV dilatation status and PAP measurements. endocan levels were lower in patients without RV dilatation and showed a significant positive correlation with RV status (r=0.456, p=0.001) and PAP values (r=0.317, p=0.022). These findings suggest that elevated endocan levels may reflect increased thrombotic burden and right ventricular strain. This is consistent with previous studies indicating that endocan may serve as a marker of pulmonary vascular load and right ventricular function (2, 21).

In one study, the endocan levels were not increased in DVT (28), but in other studies, the endocan levels were increased in PTE (21). This was interpreted as endocan having a role in showing damage to the pulmonary vascular bed rather than an indicator of thrombosis. Despite this, in our study, unrelated thrombus localization (main / segmental / subsegmental) and serum endocan levels, and the lack of increased endocan levels in PTE patients are inconsistent with previous studies. In our study, the presence of RV dilatation and the correlation between PAP and endocan levels suggest that endocan is due to the high current thrombus load rather than the presence of thrombus. In our study, the evaluated endocan levels, which were higher in the case of massive PTE compared to patients with non-massive PTE, support our opinion that endocan is a valuable parameter as an indicator of increased thrombus load.

In the study of Kuluöztürk et al., it was found that the blood troponin levels of the patients were not correlated with endocan, and endocan levels were not correlated with D-Dimer levels in deep vein thrombosis (21). In our study, there was no correlation between both troponin and D-dimer and serum endocan levels.

Studies report that galectin-3 levels increase in thrombosis, cardiovascular diseases, and ischemic cerebrovascular disease (13‑16). To our knowledge, no previous study has evaluated galectin-3 levels in PTE. In contrast to those conditions, our study did not find elevated galectin-3 levels in PTE patients overall. However, similar to endocan, galectin-3 levels were significantly higher in patients with massive PTE compared to submassive and low-risk groups. A cut-off value of ≤179.16 pg/mL distinguished massive PTE cases with 65.12% sensitivity and 88.89% specificity. These findings suggest that serum galectin-3 may be useful in assessing the clinical severity of PTE and could help guide treatment decisions, which is crucial for reducing mortality.

When the relationship between the echocardiographic findings of the patient group and serum galectin-3 levels was examined, we found that galectin-3 was not a useful parameter in the group with and without RV dilatation. However, we found a positive correlation between RV dilatation and galectin-3 levels (r=0.308, p=0.026). This situation, which seems to contradict each other, shows us that more comprehensive studies are needed on this subject.

In our study, we found no correlation between the patients' PAP and galectin-3 levels (r=256, p=0.067). In an experimental study by Li et al., high PAP was found to be associated with high galectin-3 levels, contrary to our findings (29). PAP serves as an indicator of increased thrombosis load in the pulmonary vascular bed and reflects the clinical severity of PTE; however, we believe that galectin-3 levels do not support the assessment of PAP.

Limitations

Our study had some limitations. One of these was that the biomarkers were studied from a serum sample obtained from patients simultaneously (at admission). Therefore, a relationship could not be established between endocan and galectin-3 levels and the progression of the PTE. Another limitation was that the mortality did not develop in our patient group, and due to the lack of mortality data, the markers could not be evaluated in relation to clinical outcomes. In addition, the relatively small sample size–particularly in subgroup analyses–limits the statistical power and generalizability of our findings. Lastly, the numerical difference between patient and control groups may have partially limited the interpretation of statistical comparisons.

Conclusion

In conclusion, while endocan and galectin-3 are not effective for diagnosing PTE, they may serve as supportive markers in assessing clinical severity, especially in patients with severe clinical presentation.

Acknowledgements: We would like to thank the Chief Physician's Office of Samsun University Samsun Training and Research Hospital for their financial support and valuable contributions to this study.

Conflict of Interest: The authors declare that there is no conflict of interest.

Financial Support: This study was supported by the Medical Specialty Training Board of Samsun Training and Research Hospital.

Authors' Contribution: ISS: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Writing ‑ original draft (lead), **M Yucel:** Data curation; Formal analysis; Investigation; Methodology; Software; Visualization; Writing ‑ original draft (supporting), **MHT:** Data curation; Investigation; Visualization; Writing ‑original draft (supporting), **M Yadigaroglu**: Data curation; Formal analysis; Investigation; Visualization; Writing - original draft (supporting), **MTD:** Conceptualization; Formal analysis; Methodology; Supervision; Writing - review & editing, **MO:** Conceptualization; Data curation; Methodology; Supervision; Writing - review & editing, **MG:** Conceptualization; Methodology; Supervision; Writing ‑review & editing. All authors read and approved the final submitted version of the manuscript. All authors have agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature.

Ethical Approval: Ethical approval was obtained from Samsun Training and Research Hospital Clinical Research Ethics Committee (protocol number SBUSEAH-KAEK-2020/2/6).

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