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CORRELATION OF LABORATORY MARKERS WITH THROMBOTIC BURDEN ASSESSED BY QANADLI SCORE IN ACUTE PULMONARY EMBOLISM

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Abstract. Objective. *Acute pulmonary embolism (APE) is a life-threatening condition and represents the third most common cause of cardiovascular mortality. Computed tomography pulmonary angiography (CTPA) is considered the gold standard for the diagnosis of APE. The primary aim of this retrospective study was to investigate the association between admission laboratory markers in patients with CTPA-confirmed APE and the level of clot burden, as assessed by the modified Qanadli score (mQS), which should not be underestimated as a potential prognostic predictor.* ***Materials and methods.*** *This retrospective, single-center study included 78 consecutive patients with CTPA-confirmed APE admitted to "Sv. Ekaterina" University Hospital, Sofia, Bulgaria. Demographic, clinical, laboratory, echocardiographic, and radiological data were collected. Clot burden was quantified using the mQS, classifying patients into non-massive, submassive, and massive APE. Statistical analyses were used to evaluate associations between laboratory markers and mQS.* ***Results.*** *A total of 78 patients were included in the study. The mean mQS was 15.97. Right ventricular dysfunction (RV/LV >1) was strongly associated with higher clot burden severity (p = 0.0008). Glucose levels at admission were significantly higher in patients with greater clot burden (p = 0.042). In a multivariate analysis, glucose remained an independent predictor of high-severity APE (OR = 1.397, 95% CI 1.013-1.928, p = 0.042). A glucose cut-off of 6.12 mmol/L had a sensitivity of 61.1% and a specificity of 62.5% for identifying patients with high-severity APE. Other laboratory markers, including troponin and D-dimer, did not show statistically significant differences across the severity groups.* ***Conclusion.*** *The mQS is a practical tool that, when combined with laboratory data, may aid risk stratification. Admission glucose levels showed moderate predictive value, suggesting stress-induced hyperglycemia as an indirect marker of disease severity, whereas other laboratory markers were not significantly associated with clot burden. Incorporating clot burden and glucose into prognostic models may facilitate early, individualized management, although glucose should not be used in isolation. The optimal laboratory marker for prognostic prediction in APE remains debated.*

Key words: *acute pulmonary embolism, Qanadli score, clot burden, hyperglycemia, laboratory biomarkers*

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INTRODUCTION

Deep vein thrombosis (DVT) and pulmonary embolism (PE) represent different manifestations within the spectrum of venous thromboembolism (VTE). PE is a potentially life-threatening condition and represents the third most common cause of cardiovascular deaths after myocardial infarction and stroke. The incidence ranges from 39 to 115 per 100,000 population, and the risk increases eight times for persons aged >80 years [1]. The mortality varies from <1% in normotensive patients without right ventricular dysfunction (RVD) to 30% in patients with shock [2-4]. Another delayed but potentially life-threatening complication of PE is chronic thromboembolic pulmonary hypertension (CTEPH). Over the past two decades, the incidence of PE has increased. The frequency rose from 62 per 100,000 in 1998 to 120 per 100,000 in 2016 in the United States. A similar trend is observed in Europe, particularly in high-income countries [5]. Several factors may be responsible for the increasing incidence of PE, including wider use and improved quality of computed tomography pulmonary angiography (CTPA); higher prevalence of comorbidities, such as malignancy, obesity, and immobility; and longer life expectancy, taking into account that there is a significant increase in incidence with advancing age [5]. In contrast to the above, mortality is steadily decreasing in most settings, thanks to advances in early detection, risk stratification, and evidence-based treatment. However, the mortality rate still remains high, and PE causes 300,000 deaths per year in the US and is associated with a significant clinical and economic burden [6].

Assessment of PE severity and risk stratification is crucial in decision-making for further therapeutic strategies. According to current international guidelines, patients with acute PE (APE) should be stratified into risk categories based on predicted short-term mortality and early outcome. Initial risk assessment involves evaluating for hemodynamic instability, which defines high-risk PE and requires urgent reperfusion therapy. Hemodynamically stable patients are further stratified using validated clinical tools, such as the simplified Pulmonary Embolism Severity Index (sPESI), which integrates demographic factors, comorbidities, and clinical findings at diagnosis; imaging modalities (e.g., echocardiography or CTPA) to detect RVD and elevation of cardiac biomarkers, related to RV ischemia (e.g., troponin, BNP) [7].

CTPA is considered the gold standard for the diagnosis of APE, offering high sensitivity and specificity due to its excellent accuracy in detecting emboli in both the main and segmental pulmonary arteries [8]. Beyond confirming the diagnosis, CTPA also plays a prognostic role in APE by assessing RVD; in particular, an RV (right-ventricular)/LV (left ventricular) ratio >1 is associated with increased 30-day mortality [9]. The CT obstruction index (CTOI), referred to as the Qanadli index (QI), is a quantitative method for assessing PE severity by defining clot burden. Its prognostic value remains a subject of debate and inconsistency [8]. Several small studies focused on the association between CTOI and prognosis. Some authors suggested that pulmonary artery obstruction index (PAOI) and RV/LV ratio could predict mortality in PE patients who are hemodynamically stable at presentation [10]. Wu et al. [11] claimed that clot burden is an important predictor of mortality in the setting of PE. In a recent study by Rotzinger et al. [12], a CTOI > 40% was significantly associated with increased mortality in PE patients, excluding those with cardiopulmonary comorbidities or pulmonary malignancy, compared to those with CTOI < 20% ($p < 0.001$). Conversely, other studies argued that clot burden alone is not a reliable predictor of adverse clinical outcome [13].

There is increasing interest in identifying non-invasive predictors of clot burden in APE. Although no single marker has yet proven to be definitive, combinations of clinical parameters, laboratory biomarkers, and imaging data are being actively investigated to improve risk stratification and to guide management, particularly in situations where CT imaging is unavailable or contraindicated.

MATERIALS AND METHODS

This retrospective, single-center study included 78 consecutive patients admitted with a confirmed diagnosis of APE to “Sv. Ekaterina” University Hospital, Sofia, Bulgaria, between January 2018 and January 2020. The diagnosis of APE was established based on clinical presentation and confirmed by CTPA. Relevant patient information was obtained from the hospital’s electronic database and subsequently analyzed.

Inclusion criteria were:

- Age ≥ 18 years
- Radiologically confirmed APE
- Symptoms onset < 12 h
- Availability of complete clinical, laboratory and echocardiographic data

Exclusion criteria included:

- Age < 18 years
- CTEPH
- Incomplete or missing diagnostic data
- Previous diagnosis of APE or chronic anticoagulation therapy

Patients' demographics, comorbidities, symptoms, laboratory data, interventions, Qanadli score, and outcomes were collected. The simplified PESI (sPESI) score was calculated incorporating clinical parameters including age >80 years, prior diagnosis of malignancy, chronic cardiopulmonary comorbidity, heart rate ≥110 bpm, systolic blood pressure <100 mmHg, and arterial oxygen saturation <90% [14]. Upon admission, all patients had blood drawn for a complete blood count, biochemistry including D-dimer and troponin I, and arterial blood gas analysis on room air. The Systemic Immune-Inflammation Index (SII) was defined as: platelet count × neutrophil count)/lymphocyte count [15]. Alveolar–arterial (A-a) oxygen gradient was calculated according to the following formula: $PAO_2 – PaO_2$ gradient (mmHg) = 150 – 1.25 ($PaCO_2 – PaO_2$) [16].

Transthoracic echocardiography was also performed as soon as possible after patient presentation. For the aim of the study, we retrospectively collected data on the pulmonary artery pressure (PAP). All patients underwent Doppler ultrasonography of the lower extremities to detect DVT.

All patients underwent CTPA using a 320-detector row scanner (Aquilion ONE, Canon Medical Systems) with the following parameters: tube voltage 100–120 kV, automated milliampere setting, tube rotation time 0.5 s, and slice thickness 0.5 mm. The protocol included a non-contrast scan, followed by a contrast-enhanced scan in the pulmonary angiographic phase. Scan coverage extended from the lung apices to the diaphragm. For the pulmonary angiographic phase, 45–50 mL of intravenous iodine contrast (dose adjusted according to the Body Mass Index (BMI)) was administered via the cubital vein at a rate of 4.5–5 mL/s, followed by a saline chaser. Scanning was performed in a caudal-to-cranial direction. To optimize pulmonary artery opacification, a bolus tracking technique was used with a threshold of 100 HU, monitored by a region of interest (ROI) placed in the pulmonary trunk. All CT examinations were analyzed using dedicated software on a Vitrea workstation.

A radiologist retrospectively reviewed the CT scans for clot burden assessment. The QS is a semi-quantitative tool that assigns points calculated by: The number of pulmonary artery segments involved (10 per lung or 20 total), multiplied by a weight factor of -1 point for partial occlusion or 2 points for complete occlusion, with a maximum possible score of 40. The CTOI (CT Obstruction Index) or Qanadli index is the sum of the individual scores per artery divided by 40 and expressed as a percentage [17].

For our study, we used the Modified Qanadli score (mQS), where 1 point is given for each occluded segmental artery; no distinction is made between partial or complete obstruction and the maximum score is 20 points (10 segmental arteries per lung × 1 point each). This method is time-saving, reduces interobserver variability, and still correlates strongly with the original QS for severity assessment in APE. According to the results of this clot loading score, our patients are classified in three groups: non-massive APE: score 1–5, submassive APE: score 6–14, and massive APE: score >15 points [18].

STATISTICAL ANALYSIS

Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data or median (range) for non-normally distributed data. Categorical variables were expressed as n (%). Comparisons of continuous variables: normally distributed variables were compared using one-way Analysis of Variance (ANOVA), non-normally distributed variables were compared using the Kruskal–Wallis test. Comparisons of categorical variables were performed using the Chi-square test. A p-value <0.05 was considered statistically significant.

Univariate logistic regression was performed to evaluate the association of each factor with the outcome of interest. Odds ratios (ORs) with 95% confidence intervals (CIs) and p-values were reported. Multivariate logistic regression was conducted to assess the independent effect of each factor while adjusting for potential confounders. Adjusted ORs with 95% CIs and p-values were reported. Variables with p-values <0.05 in univariate analysis were considered statistically significant and were included in the multivariate model. Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the discriminative ability of glucose for predicting the outcome. The area under the curve (AUC) with 95% confidence interval (CI), standard error (SE), and p-value was calculated. The optimal cutoff value was determined to maximize sensitivity and specificity. Sensitivity and specificity corresponding to this cutoff were reported.

RESULTS

A total of 78 individuals, admitted with a diagnosis of APE confirmed by CTPA over 2 years, were included in this study. According to our scoring system, using the mQS, 10 (12.8%) patients fell into the non-massive PE group (Group 1); 14 (17.9%) fell into the submassive group (Group 2) and 54 (69.2%) – into the massive PE group (Group 3). The mean mQS in the study sample was 15.97 (± 5.57).

The mean age of the studied individuals was 64.43 ± 15.52 years (the youngest was 18 years old and the oldest – 91 years old). The study included 37 (47.4%) men and 41 (52.6%) women. Age and gender distribution did not differ significantly across the three groups of APE severity (p-0.268 and p-0.415, respectively).

The most common symptoms were dyspnea (91.2%), fatigue (31.2%), and syncope (12.5%). Mean systolic blood pressure (SBP) was 123 ± 20.5 mmHg, and 8.75% presented with SBP < 100 mmHg. As for

comorbidities and risk factors, in the entire studied population of patients with PE, we found that 70% had hypertension, 57.5% had dyslipidemia, 15% had diabetes mellitus (DM), 17.5% were obese, DVT was present in 39 patients – 48.75%, and a history of malignancy was present in 18 patients – 22.5%. The occurrence of the above-mentioned comorbidities showed no significant association with clot burden severity (all p > 0.1). Regardless of the APE severity assessed by mQS, SBP and sPESI were comparable across the groups (p = 0.235 and 0.433, respectively). As expected, the RV/LV ratio >1 assessed by CT was strongly associated with higher clot burden severity (p = 0.0008), indicating RVD as a marker of more severe embolism. With regard to the echocardiographic findings, we observed that sPAP was significantly higher in patients with more severe APE (p = 0.014).

White blood cell (WBC) count and inflammatory markers, such as CRP and SII, were higher in Group 3 but not significantly different among severity groups. Similarly, D-dimer and serum creatinine levels were similar between groups. High-sensitivity troponin I showed a marked increase in Group 3 (median 0.289 ng/ml), but the difference did not reach statistical significance (p = 0.993). Glucose levels at admission were significantly higher in patients with greater clot burden (p = 0.042), suggesting hyperglycemia may correlate with disease severity. Oxygen saturation (SpO_2) and A-a O_2 gradient did not significantly differ between severity groups. Length of hospital stay was similar across groups (p = 0.375).

In-hospital mortality was low overall, with only 2 deaths (3.7%) occurring in Group 3 (most severe APE).

Associations between demographic, clinical, laboratory, echocardiographic, and CT parameters and APE severity, defined by clot burden on CTPA, were evaluated using the Kruskal–Wallis test for continuous variables and the Chi-square test for categorical variables, as presented in Table 1.

Table 1. Association between the variables and the acute pulmonary embolism severity

	Acute pulmonary embolism severity			
Variable	Group 1 (n = 10)	Group 2 (n = 14)	Group 3 (n = 54)	p-Value
Demographic findings				
Age (years)	71.90 ± 9.17	63.21 ± 15.36	63.22 ± 16.51	0.268
Male gender, n (%)	3(30)	8(57.1)	26(48.1)	0.415
Clinical characteristics				
Systolic BP	132.00 ± 11.60	126.43 ± 16.81	121.30 ± 22.34	0.235
AH, n (%)	8 (80)	8 (57.1)	40 (74.1)	0.376
DM, n (%)	2 (20.0)	4 (28.6)	8 (14.8)	0.482
Dyslipidemia, n (%)	7 (70.0)	5 (35.7)	34 (63.0)	0.136
Obesity, n (%)	4 (40.0)	2 (14.3)	8 (14.8)	0.150
DVT, n (%)	4 (40.0)	7 (50.0)	26 (48.1)	0.874
sPESI score, mean (SD)	90.90 ± 23.72	80.43 ± 19.06	101.15 ± 44.56	0.433
Cancer, n (%)	2 (20.0)	2 (14.3)	13 (24.07)	0.72
Echocardiography findings				
sPAP (mmHg)	30.90 ± 8.17	44.29 ± 19.15	47.22 ± 18.50	0.014
CT findings				
RV/LV>1, n (%)	2 (20.0)	8 (57.1)	41 (75.9)	0.0008
Laboratory findings				
WBC count (x10⁹/L)	8.18 ± 2.32	8.79 ± 2.28	10.19 ± 3.25	0.142
SII	843 (374-1560)	892 (68-4231)	1012 (60-8912)	0.569
Anemia, n (%)	2 (20.0)	0 (0.0)	12 (22.6)	0.146
CRP (mg/L)	36.0 (3.0-108.0)	51.9 (0.0-204.0)	56.1 (2.0-384.0)	0.726
hsTN-I (ng/ml)	0.021 (0,004-0.064)	0.024 (0.005-0.04)	0.289 (0.004-2.99)	0.993
Glu (mmol/L)	6.50 (4.00-8.00)	6.00 (4.00-10.00)	7.00 (4.00-21.00)	0.042
Creat (µmol/L)	86.10 (60-122)	99.14 (67-174)	99.06 (54-319)	0.354
D-dimer (ng/mL)	4.69 (0.47-10.00)	2.51 (0.34-19.00)	4.39 (0.49-17.00)	0.716
SPO2	95.43 ± 1.51	91.00 ± 5.14	91.60 ± 7.00	0.185
A-a 02 gradient	45.17 ± 10.61	50.67 ± 15.51	45.64 ± 17.97	0.880
Outcome				
Length of hospital stay (days)	5.8 ± 3.39	5.5 ± 1.74	6.2 ± 2.78	0.375
In hospital mortality, n (%)	0(0.0)	0(0.0)	2(3.7)	

Abbreviations: APE – acute pulmonary embolism; BP – blood pressure; AH – arterial hypertension; DM – diabetes mellitus; DVT – deep vein thrombosis; sPESI – simplified pulmonary embolism severity index; PAP – pulmonary artery pressure; RV/LV – right ventricle/left ventricle diameter ratio; WBC – white blood cell; SII – systemic immune-inflammation index; CRP – C-reactive protein; hsTnI – high-sensitivity troponin I; Glu – glucose; Creat-creatinine; SpO_2 – peripheral oxygen saturation; A–a O_2 gradient – alveolar–arterial oxygen gradient. Data are shown as the mean±standard deviation or number (percentage), or median (range).

Binary logistic regression was employed to identify independent predictors of severe PE (group 3 vs. groups 1–2). In univariate logistic regression, patients with an RV/LV ratio >1 had significantly higher odds of high-severity APE compared to those with an RV/LV ratio ≤1 (OR = 3.83, 95% CI 1.41–10.46, p = 0.0044). Among laboratory and echocardiographic parameters, elevated glucose levels were also significantly associated with high-severity APE, as each 1 mmol/L increase in glucose is associated with 44% higher odds of high-severity APE (OR = 1.442, 95% CI 1.026–2.026, p = 0.035), whereas sPAP showed a trend toward higher odds but did not reach statistical significance (OR = 1.032, 95% CI 0.998–1.067, p = 0.066). However, each 1 mmHg increase in PAP increased the odds of severe APE by 3.2%. These findings suggest that both right ventricular dilation and hyperglycemia are important markers of increased APE severity. In subsequent multivariate analysis after adjusting for all confounding factor (age, sex, comorbidities – especially DM, AH, cancer, sPESI score, and inflammatory/cardiac biomarkers), glucose remained an independent predictor of high-severity APE (OR = 1.397, 95% CI 1.013–1.928, p = 0.042), while PAP did not retain statistical significance (OR = 1.029, 95% CI 0.994–1.065, p = 0.106) – Table 2.

Table 2.
Logistic regression analyses showing independent predictors of massive acute pulmonary embolism.

Factor	Univariate logistic regression			Multivariate logistic regression		
	OR	95% CI	p	OR	95% CI	p
PAP (mmHg)	1.032	0.998–1.067	0.066	1.029	0.994–1.065	0.106
Glucose (mmol/L)	1.442	1.026–2.026	0.035	1.397	1.013–1.928	0.042

Abbreviations: OR-odds ratio; CI-confidence interval: PAP-pulmonary artery pressure

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the discriminatory ability of admission glucose levels for predicting APE severity. Glucose levels were significantly associated with APE severity. The AUC was 0.673 (SE = 0.068; 95% CI: 0.540–0.805; p = 0.015), indicating a statistically significant but moderate discriminatory capacity. The ideal glucose cutoff value, identified by receiver operating characteristic (ROC) analysis, is 6.12 mmol/L, which provided a sensitivity of 61.1% and a specificity of 62.5% for identifying patients with high-severity APE (Table 3; Fig 1), identifying the glucose levels at admission as a moderate but useful marker, though not strong enough to be used alone.

Table 3. ROC analysis of glucose for predicting acute pulmonary severity according to clot burden.

Variable	AUC (95%CI)	SE	p-value	Cut-off(mmol/L)	Sensitivity	Specificity
Glucose	0.673 (0.540–0.805)	0.068	0.015	6.12	61.1%	62.5%

AUC = area under the curve; 95% CI = 95% confidence interval; SE = standard error.

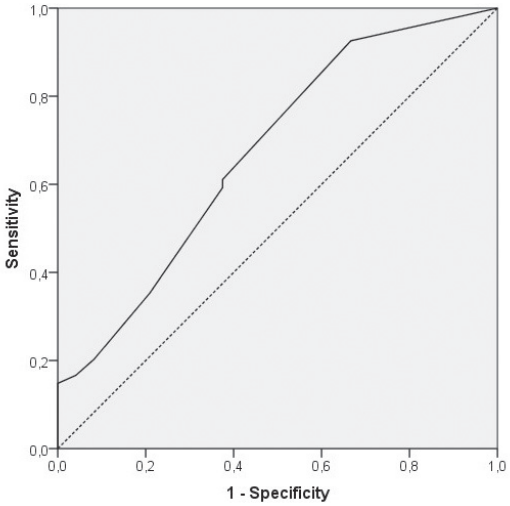

Fig. 1 Receiver operating characteristic (ROC) curve for glucose in predicting acute pulmonary embolism (APE) severity. Solid line (——)-glucose levels; Dotted line (···)-reference line

DISCUSSION

Currently, CTPA is regarded as the primary diagnostic tool for patients with suspected APE as a fast and non-invasive exam, demonstrating very high sensitivity and specificity [19]. CTPA not only confirms or excludes PE, but also provides detailed information on clot location and burden, evaluates right ventricular strain, guides therapy, and identifies alternative diagnoses.

Risk stratification for APE is essential for determining the appropriate therapeutic approach and improving clinical outcomes. According to the current 2019 European Society of Cardiology (ESC) guidelines, risk stratification is primarily based on clinical assessment of hemodynamic stability, biomarkers for myocardial injury, echocardiographic or CT angiographic

evidence of RVD, and clinical scores integrating PE severity and comorbidity – the PESI [7]. Despite these established criteria, the role of clot burden – defined as the volume and location of thrombi within the pulmonary arterial system – is a subject of ongoing scientific debate. Quantitative assessment of clot burden using indices like the QS, Mastora score, and the more recent clot volume and clot ratio provides an objective measure of the degree of pulmonary blood flow occlusion [20]. We believe that the quantitative assessment of clot burden could be a substantial addition to current recommendations, and integrating this assessment could lead to more precise risk stratification, allowing for earlier and more aggressive intervention in patients who, despite their hemodynamic stability, have extensive thrombosis and are at a high risk of future deterioration.

Some studies demonstrate a correlation between clot burden and the severity and outcome in APE. Irmak et al. [21] found that thrombus location and clot burden on CTPA were positively associated with American Heart Association risk stratification and correlated with PAP, D-dimer, the ratio of the pulmonary artery trunk diameter/aorta diameter, and superior vena cava diameter, but association with survival was not found. Higazi et al. [22] proved a significantly higher PAOI in high-risk patients who presented with shock or hypotension, compared to non-high-risk patients who had normal BP. Moreover, the authors state a strong positive relationship between the RVD in the hemodynamically stable patient group and the PAOI ($P < 0.0001$). According to Xi et al. [20], the QS, clot volume, and clot ratio were able to predict patients in the high/intermediate-high risk group as stratified by the 2019 European Guidelines. They also reported that the clot ratio was higher in patients with RVD, although it did not demonstrate predictive value for 30-day clinical outcomes [20]. Another retrospective study involving 201 patients reported that total embolic volume assessed by CT was independently associated with impending shock [23]. In a one-year follow-up period, a retrospective cohort study of 475 patients demonstrated that QS was directly associated with CTEPH after APE with a cutoff value of 14.5 (43.5%) [24]. Wu et al. [11] even demonstrated an association between clot burden and survival, proving that patients with a clot burden exceeding 60% generally have a worse clinical prognosis regarding short-term mortality, and suggest that this finding could stratify mortality risk and guide the use of more aggressive treatments, such as thrombolysis. Van der Meer et al. [25] reported that, at 3-month follow-up, patients with an obstruction index of 40% or higher had an 11.2-fold increased risk of death from PE. In contrast, other authors did not find a significant correlation between clot burden and patients' prognosis [26, 27]. Nevertheless, the location and extent of emboli in APE, as assessed by CTPA, are not recognized as prognostic indicators in current clinical guidelines.

Our retrospective study aims to investigate the association between laboratory markers at admission in patients with CTPA-confirmed APE and the level of clot burden described by the mQS. The use of laboratory tests is easily accessible, rapid, and cost-effective, and can be performed at the patient's bedside, representing a very convenient prognostic tool for risk assessment in the acute phase, especially if imaging methods are not available. Plural studies investigate the predictive role of laboratory markers in risk stratifications and, respectively, adverse events and outcomes, as it may help guide management and therapy strategies.

The ESC guidelines state that elevated cardiac troponin (cTn) levels in patients with APE, in combination with clinical and imaging findings, serve as a prognostic marker for high–risk patients [7]. Moreover, it could be a predictor for longer hospital stay [28]. Numerous studies have demonstrated the negative prognostic role of high cTn levels in patients with APE. A prospective study demonstrated that elevated cardiac troponin T (cTnT) was strongly associated with adverse clinical events, including death, shock, and the need for resuscitation, inotropic support, or mechanical ventilation. Importantly, cTnT remained an independent predictor of 30-day mortality after adjustment [29]. Konstantinides et al. [6] state that elevation of cTnI or cTnT was significantly associated with RVD on echocardiography ($P = 0.001$ and $P < 0.05$, respectively). In addition, increased levels of either marker correlated with the major clinical endpoints of overall mortality and complicated in-hospital course. A meta-analysis by Becattini et al. [30], including data from 20 studies, concluded that elevated cTn levels were strongly associated with short-term mortality, death due to PE, and adverse outcomes. Importantly, even in hemodynamically stable patients, elevated Tn was linked to a significantly increased risk of death.

D-dimer is a fibrin degradation product with high sensitivity and low specificity for the diagnosis of APE. In a meta-analysis, Becattini et al. [31] revealed that D-dimer above the cutoff value is strongly associated with short-term and 3-month mortality in overall PE patients and in particular in hemodynamically stable patients with APE. In the RIETE registry, markedly elevated D-dimer levels (≥ 5,000 ng/mL) were linked to a higher risk of fatal PE and reflected more severe

clinical disease. Other studies claimed that D-dimer ≧ 4.0 μg/mL and WBC ≧ 11,000 mm^3 were defined as strong predictors for in-hospital hemodynamic instability and 30-day mortality [32, 33].

In APE patients, elevated natriuretic peptides (B-type-BNP and its aminoterminal portion-NT-proBNP) are due to increased myocardial shear stress and RVD. A meta-analysis evaluating the relation between elevated BNP or pro-BNP (NT-pro-BNP) and clinical outcome in patients with APE established that high levels were strongly associated with RVD (P < 0.001). Moreover, there is an increased risk of a complicated in-hospital course and 30-day mortality [34].

In recent years, increasing attention has been directed toward the prognostic significance of inflammatory markers in determining disease severity across a range of conditions. In the pathogenesis of pulmonary embolism, hypercoagulability, endothelial injury, and inflammation play key roles. However, there remains considerable interest in whether it represents a cause or merely a consequence of the thromboembolic process [35]. Ozdemir et al. [35] claimed that WBC, neutrophils, NLR (neutrophil-lymphocyte ratio), PLR (platelet-to-lymphocyte ratio), SIRI (systemic inflammatory response index), SII, and CRP could have a predictive role, as their levels were significantly lower in the low-risk group patients with APE compared to the intermediate-low-risk, intermediate-high-risk, and high-risk groups. However, NLR and SII were not independently predictive in multivariate regression. Moreover, in the RIETE cohort, in patients with APE, NLR, PLR, and SII were defined as strong predictors of 30-day mortality, as an NLR >7.0 tripled the risk for all-cause mortality [36]. In the study by Gok et al. [37], which evaluated 442 patients with APE, disease severity was classified according to current guideline criteria. The SII was identified as an independent predictor of massive APE, with a cut-off value of 1,161 yielding 91% sensitivity and 90% specificity. Moreover, a significant association was observed between elevated SII and fatal in-hospital outcomes [37]. CRP is associated with RVD and, accordingly, may serve as a marker for risk classification [38]. This is further confirmed by the findings of Gok et al. [37], who reported that CRP was independently associated with massive APE. Omar et al. [39] analyzed the correlation between several laboratory markers in predicting RVD and 30-day mortality in PE patients. They found a positive relationship for NLR, PLR, CRP, D-dimer, and cTn, with the highest predictive value for A-a O_2 gradient, serum troponin, CRP, D-dimer, NLR, and PLR (P value < 0.001), but none of them were significant by applying multivariate regression regarding 30-day mortality.

Arterial blood gas (ABG) analysis remains a simple and rapid bedside test for patients with APE. The prognostic role of ABG analysis parameters in APE has been studied, although the results are somewhat mixed. In a prospective, multicenter, cohort study with three months of follow-up by Bova et al. [40], hypoxemia, clinical characteristics and TnI predicted the in-hospital mortality, with clinical score, D-dimer, hypoxemia, and troponin I being identified as predictors of 3-month all-cause mortality. In patients with acute PE, an A–a O_2 gradient ≥ 53 mmHg predicted adverse outcomes, with a negative predictive value of 92% for 30-day mortality and 84% for composite events [41]. In elderly PE patients, only lower SO_2 seems to predict 14-day mortality; neither PaO_2, PCO_2, pH, nor alveolar–arterial O_2 gradient seems to identify high-risk patients [4].

Clearly, numerous studies have investigated the relationship between laboratory parameters and the severity of PE, as well as patient prognosis, yet the ideal predictor remains unclear and is still a matter of debate due to conflicting results. On the other hand, data on the correlation between specific laboratory markers and clot burden assessed by CT are more limited. The primary aim of our retrospective study was to investigate this relationship by comparing the relationship between several laboratory markers and thrombotic burden. Our findings are, to some extent, unexpected.

Overall, demographic parameters such as age, sex distribution, and comorbidities (AH, DM, dyslipidemia, obesity, and cancer) did not differ significantly between the three severity groups, suggesting that baseline characteristics alone may not be sufficient for predicting PE severity. Although the difference did not reach statistical significance, there was a tendency toward a higher proportion of cancer patients in group 3, which was also characterized by higher PESI scores. Echocardiographic parameters demonstrated a clear association with disease severity. sPAP was significantly higher in groups 2 and 3 compared with group 1. Moreover, RVD, defined by RV/LV > 1, was strongly associated with severity, defined by clot burden and presented in 75.9% of patients in group 3 compared with only 20% in group 1. These findings confirm the established prognostic role of right ventricular overload as a marker of poor outcome in APE. Rodrigues et al. [42] claim that the relationship between pulmonary vascular involvement and right ventricular burden remains controversial. Similar to our findings report by Huang et al. shows a linear association between the greater TEV and higher sPAP, greater RV diameter, and RV/LV ratio [23]. Similarly, Abdelwahab et al. [43] found significant correlation between clot volume and several parameters of RV dysfunction assessed by pulmonary CTA, including RV diameter, RV/LV diameter ratio, pulmonary artery diameter, the ratio of the main pulmonary artery to ascending aorta diameter, and superior vena cava diameter but, no significant correlation was found be-

tween clot volume and echocardiographic parameters of RVD.

Laboratory results revealed more subtle differences. In terms of inflammatory markers, we observe a tendency toward elevated values in patients with more severe forms of APE, suggesting an enhanced inflammatory response in cases with greater severity. While WBC count and SII tended to be higher in the more severe groups, these differences did not reach statistical significance. Similarly, CRP levels showed an increasing trend but were not significantly different across severity groups. Even more convincing evidence for the association between inflammatory markers and thrombotic burden was provided by Cildag et al. [44], in a study demonstrating significantly higher WBC levels, NLR, and PLR in patients with higher pulmonary arterial computed tomographic obstruction index ratio (PACTOIR), which is similar to QS. These authors also emphasized that WBC count may have greater utility as a prognostic rather than a diagnostic tool. Moreover, NLR and PLR appear to be promising predictors of adverse clinical outcomes.

In contrast, Hajsadeghi et al. [45] revealed non-significant associations between the total QS and variables like WBC and CRP. The exact relationship between CRP levels and the clot burden is complex and may not be a direct one-to-one correlation. There is a lack of information concerning this correlation.

Unexpectedly, cTn and D-dimer, which have been widely regarded as promising predictors of disease severity in APE, did not demonstrate statistically significant differences across the three study groups. Nevertheless, similar findings have been reported by other authors, indicating that the prognostic utility of troponin and D-dimer may be inconsistent and not uniformly reproducible across different patient populations. For example, Hajsadeghi et al. [45], in a retrospective study consisting of 200 patients with APE strong correlation is described between several echocardiographic parameters, suggesting RVD, like RV enlargement, PAH, tricuspid regurgitation severity, and QS, but no significant association was observed for elevated cTn or D-dimer levels.

It is hypothesized that DVT and APE are not two completely separate diseases, but rather two clinical presentations of the same underlying condition – VTE with a similar pathophysiological background. By reviewing the medical literature, it appears that, similarly, in patients with DVT, D-dimer was not related to disease severity, extensive or localized thrombotic burden [46]. On the contrary, higher TEV (strongly correlated with QS ($r = 0.69$, $p < 0.001$) was independently associated with elevated TnI > 0.04 [32]. Several studies stated that cardiac TnI and plasma D-dimer are related to proximal, segmental, larger, and bilateral extension of clots, but such an association was not valid for CRP values [2, 47]. Ghanima et al. [48], studying the association between D-dimer levels and the severity of PE, conducted a multivariate analysis and found that higher D-dimer levels were significantly associated with an increased PAOI, even after adjusting for age, gender, and symptom duration. Both D-dimer and cTnT demonstrated a significant positive correlation with QS and Mastora clot burden score. In contrast, their association with clot volume and clot ratio was not significant according to Xi et al. [20], so the results are still heterogeneous and inconclusive.

Hypoxemia in APE results primarily from the degree of ventilation–perfusion (V/Q) mismatch, along with contributions from right-to-left shunt, reduced cardiac output, and, to a lesser extent, diffusion impairment. In our study, oxygenation parameters, including SpO_2 and alveolar–arterial oxygen gradient, did not significantly discriminate between severity groups. On the contrary, a significant association between (A-a) oxygen gradient and PAOI measured with the QS was observed in a retrospective study by Karakayalı et al. [49]. ABG analysis appears even more promising as a prognostic indicator of PE morphological severity, being a rapid and inexpensive bedside test, with studies confirming significant correlations between all blood gazes values and obstruction index PaO_2 ($r = –0.33$, $p = 0.05$), $PaCO_2$ ($r = –0.34$, $p = 0.05$), (A-a) oxygen gradient ($r = 0.39$, $p = 0.02$), and SaO_2 ($r = –0.35$, $p = 0.04$), suggesting that ABG analysis, together with other clinical parameters, could guide the physician in first medical contact in suspecting severe APE [50].

The association between hyperglycemia and worse outcomes in acute illnesses, including cardiovascular and thromboembolic events, is well-documented [50, 51]. Farrokhi et al. [53] reported that stress hyperglycemia occurs in nearly 40% of critically ill patients, with the incidence rising to 80% among those undergoing cardiothoracic surgery, of whom the majority (80%) had no prior history of DM before admission. Whether hyperglycemia is responsible for a more severe disease course or merely reflects its severity remains unclear, but the relationship is likely bidirectional. The mechanism of stress hyperglycemia in hospitalized patients is complex and is driven by metabolic and hormonal stress. Counterregulatory hormones, released during acute diseases (glucagon, epinephrine, cortisol, growth hormone), increase hepatic glucose production, reduce peripheral glucose utilization, and impair insulin secretion. Pro-inflamma-

tory cytokines, such as TNF-α, IL-6, and IL-1, further promote insulin resistance by disrupting insulin signaling. These processes collectively diminish insulin action, and when pancreatic compensation is insufficient, stress-induced hyperglycemia develops, often worsened by β-cell desensitization. Hyperglycemia, in turn, promotes oxidative stress through reactive oxygen species generation and elevates inflammatory markers, and contributes to endothelial dysfunction by reducing nitric oxide availability and impairing vasodilation. It can trigger cardiac myocyte apoptosis or worsen ischemia-reperfusion injury. Moreover, it disturbs hemostasis by enhancing platelet activation and aggregation and reducing fibrinolysis [53].

In our study, the only laboratory marker that showed a potential predictive value with respect to clot burden was the admission blood glucose level, as patients with higher glucose levels are more likely to belong to the higher severity group of APE, demonstrating a moderate predictive value for adverse clinical outcomes in patients with APE. The ROC analysis showed an AUC of 0.673, suggesting that blood glucose levels can serve as a useful but not highly accurate prognostic marker. A glucose cutoff of 6.12 mmol/L provided a balanced sensitivity of 61.1% and specificity of 62.5%, which highlights its potential role in risk stratification. However, glucose alone should not be used as the only predictor, since its discriminatory ability is **fair but not strong.** Interestingly, despite only moderate predictive accuracy, glucose measurement remains an easily accessible, inexpensive, and rapid bedside test. This provides it with potential clinical utility, especially in emergency and resource-limited settings.

Due to chronic hyperglycemia in diabetic patients with adequate blood glucose control, the criteria for stress hyperglycemia (SH) may need to differ for this subgroup. Therefore, findings in diabetic patients should be interpreted with greater caution. Due to the retrospective nature of our study, small sample size, and somewhat unexpected results, we did not divide patients into subgroups based on the presence of DM. Moreover, our findings indicate that there is no difference between diabetic and non-diabetic patients across the three patient groups, and that DM is not predictive of a higher thrombotic burden. So the diabetic patients were not analyzed separately. Most of the studies have focused on the impact of hyperglycemia on the clinical severity or outcomes, rather than on the anatomical clot burden in patients with APE. For example, Scherz et al. [54] stated that higher admission glucose levels in patients with APE were independently related with increased 30-day mortality – cumulative 30-day mortality increased progressively with rising serum glucose levels: 5.6% (≤110 mg/dL), 8.4% (111–140 mg/dL), 12.0% (141–170 mg/dL), 15.6% (171–240 mg/dL), and 18.3% (>240 mg/dL). Higher admission serum glucose levels were significantly associated with increased odds of mortality in non-diabetic patients, whereas no such association was observed in diabetic patients. Such correlation was not observed regarding the 30-day readmission rate [54]. Liao et al. demonstrated similar results, finding a strong independent association between the higher stress hyperglycemia ratio and the 28-day ICU mortality [55]. There is a lack of data in current literature reports describing the association between SH and clot burden assessed by CTPA. Altabas et al. [51] evaluated the role of SH on embolus size and location in APE in an observational cohort study that included 190 patients. They found that SH was independently associated with embolus location, and this association remained significant after logistic regression. Moreover, logistic regression revealed that SH was independently associated with in-hospital mortality in non-diabetic patients and remained significantly related to in-hospital mortality in the overall cohort, regardless of diabetes status.

Due to the very low number of events (hemodynamic instability, requirement for inotropes, and need for intubation) and the absence of deaths in two of the groups, statistical comparison between groups was not performed, as such analysis would be unreliable and potentially misleading.

Several limitations must be acknowledged. It is a single-centered design, retrospective (limiting the ability to establish causal relationships) study with a relatively small sample size. Very few outcomes, such as deaths, requirement for inotropes, and need for intubation, limit the ability to perform robust statistical analyses regarding adverse events. Additionally, clot burden assessment based on imaging may be subject to interobserver variability. Patients with diabetes were **not analyzed separately** in all analyses due to small numbers and unexpected results. In addition, due to the large number of patients with missing information on BNP and pro-BNP values at admission, this parameter was not included in the statistical analysis, and its prognostic role may have been overlooked. These limitations could be addressed by conducting multicenter studies with larger sample sizes.

CONCLUSIONS

In this study, we evaluated the predictive value of several laboratory markers in determining the severity of APE, as defined by clot burden on imaging. In our view, the QS is readily accessible and, when

combined with other prognostic markers, could serve as a useful tool for risk stratification in patients with PE. Incorporating clot burden into prognostic models may improve PE management, complementing traditional parameters and supporting a more personalized approach in cardiology.

Notably, we found that glucose levels demonstrated moderate predictive value, while other commonly used laboratory markers did not show a significant correlation with sQS. This suggests a possible role for hyperglycemia as an indirect marker of PE severity. This finding highlights the complex interplay between metabolic stress and thromboembolic disease and warrants further investigation in prospective, controlled studies. The identification of glucose as a moderate predictor of clot burden may provide clinicians with an easily accessible and inexpensive tool to aid early risk stratification. However, it should not be used as a standalone marker. While it should not replace imaging, it could be incorporated into initial assessment models, particularly in resource-limited settings or when imaging is delayed.

Although aggressive therapeutic strategies are indicated for hemodynamically unstable patients, the optimal management of those who are hemodynamically stable but exhibit RVD remains uncertain. Additional risk stratification using readily available clinical and laboratory predictors could help identify patients at higher risk and guide management decisions.

However, because most studies have not performed an extensive comparison between all available biomarkers, it remains debated which marker provides the best prognostic value.

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