Role of echocardiographic indicators of right ventricular dysfunction in predicting 30-day mortality in non-high--risk patients with acute pulmonary embolism in different variants of the Bova score

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ABSTRACT

Background: The Bova score is a validated tool for short-term mortality risk stratification in normotensive patients with acute pulmonary embolism (PE). The prognostic value of echocardiographic parameters in this group of patients remains controversial.

Aims: We aimed to assess the role of echocardiographic indicators of right ventricular dysfunction in different variants of the Bova score.

Methods: Patients with PE confirmed by computed tomography pulmonary angiography had a transthoracic echocardiogram performed during the first day of hospitalization and 30-day follow-up.

Results: One hundred eleven consecutive subjects with non-high-risk PE entered the analysis — 55 men (49.6%), at a median age of 69 (58–79) years; 12 patients died during the 30-day follow-up. Among 3 Bova score variants with different echocardiographic criteria used in practice, the original one AD 2014 had the best but, objectively, poor predictive strength — the area under the curve (AUC) of 0.679. The Bova score with the right-to-left ventricle ratio >1 and tricuspid annular plane systolic excursion <16 mm was an even worse indicator (AUC 0.652), whereas the Bova score with free wall longitudinal strain >–19% and Bova 60/60 sign had fair predictability (AUC 0.701 and 0.731, respectively). Still, they were inferior to the simplified Pulmonary Embolism Severity Index (sPESI, AUC — 0.815). The subjects with Bova score variants with points >4 had a higher risk of death (hazard risk of 1.43–1.59) and with an sPESI ≥1 point had a hazard risk of 2.02.

Conclusions: Various echocardiographic markers of right ventricular dysfunction within divergent variants of the Bova score yield different prediction strengths but are all inferior to the sPESI score.

Key words: 60/60 sign, Bova score, echocardiography, pulmonary embolism, simplified Pulmonary Embolism Severity Index

INTRODUCTION

Venous thromboembolic disease with its clinical manifestations of pulmonary embolism (PE) and deep vein thrombosis is one? of the most frequent cardiovascular diseases globally [1]. Its prevalence continues to increase and poses a serious burden to the healthcare system [2]. Acute PE is associated not only with different clinical presentations but also with diverse prognoses. Low-risk PE patients have a mortality rate of marginally under 3%, while those with high-risk PE who experienced cardiopulmonary arrest have a risk of more than 90% [1, 3, 4]. Furthermore, survivors of acute PE might suffer from complications concerning various fields of life, not only re-

WHAT'S NEW?

The Bova score is a validated tool for early mortality risk stratification in normotensive patients with acute pulmonary embolism. In addition to clinical and biochemical variables, it includes echocardiographic parameters of right ventricular dysfunction. Although the prognostic value of echocardiographic markers of right ventricular dysfunction in this group of patients remains controversial, we have demonstrated that an appropriate selection of such parameters might augment the predictive capability of the Bova score. The most efficient echocardiographic criteria comprised the 60/60 sign and right ventricular free wall longitudinal strain >–19%.

lated to health — heart failure or chronic thromboembolic pulmonary hypertension — but also different aspects of life such as employment, the environment, mental health, education, recreation and leisure time and social belonging [5, 6]. Consequently, early diagnosis and accurate risk stratification to determine the appropriate therapeutic management approach are pivotal.

According to the European Society of Cardiology (ESC) guidelines, PE patients without hemodynamic instability should be stratified according to two sets of prognostic criteria: clinical, imaging, and laboratory indicators of PE severity, which are related to the presence of right ventricular (RV) dysfunction and comorbidities and other aggravating conditions that could adversely affect early prognosis. Noteworthy, transthoracic echocardiography (TTE), a non-invasive, widely available tool is not recommended in a routine work-up in hemodynamically stable patients with suspected or diagnosed PE [1]. However, the use of TTE helps diagnose RV dysfunction, even clinically silent, and thus identify patients at increased risk of hemodynamic deterioration and early mortality since short-term outcomes in those patients are closely related to RV failure. Therefore, TTE seems to be an underestimated and underutilized tool in this population [7, 8].

As stand-alone parameters may not suffice to classify PE severity, various combinations of clinical, imaging, and laboratory parameters were used to build prognostic scores that allow at least a semi-quantitative assessment of short-term mortality risk in non-high-risk PE patients. The scores such as the Pulmonary Embolism Severity Index (PESI) and its simplified version (sPESI), Bova score, and FAST score based on heart-type fatty acid-binding protein (H-FABP) or high-sensitivity troponin T have been validated in randomized trials [9–15].

Importantly, it is sPESI that shows the highest discriminatory performance concerning 30-day all-cause mortality in low-to-intermediate PE risk [16]. Interestingly, the only one that incorporates TTE parameters is the Bova score which is intended for non-high-risk PE patients. Notably, various configurations of TTE parameters of RV dysfunction were included in different variants of this score. In principle, the Bova prognostic model includes elevated cardiac troponin (2 points), RV dysfunction (detected on TTE or computed tomography pulmonary angiography with different criteria, 2 points), heart rate ≥110 bpm (1 point), systolic blood pressure 90–100 mm Hg (2 points), with the result of ≤4 points for low risk and >4 points for intermediate-high risk [1]. It is an open question, to what extent TTE might augment the prognostic stratification as part of the Bova score?

This study aimed to assess different echocardiographic indicators of RV dysfunction in predicting 30-day mortality in non-high-risk patients with acute pulmonary embolism using different variants of the Bova score.

MATERIAL AND METHODS

Methodology

This was a cross-sectional observational single-center study. The study group involved consecutive patients with acute PE with low to intermediate risk according to the ESC guidelines, confirmed on computed tomography pulmonary angiography. The patients were recruited between August 1, 2018 and April 30, 2021 in the Internal Medicine Department and the Special Care Cardiac Unit. The treatment regimen followed the ESC guidelines on PE management and was described in detail previously [1, 17, 18].

The exclusion criteria were high-risk PE, recurrent PE, chronic thromboembolic pulmonary hypertension, echocardiograms of inadequate quality in which not all parameters from the unified protocol could be evaluated, severe valvular defects, and tricuspid valve replacement.

A standard diagnostic protocol for all patients included measuring on the day of admission to the ward the laboratory parameters including i.e. serum concentrations of troponin T, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and D-dimer with laboratory methods described earlier [18]. TTE was performed within 24 hours after admission to the ward by an experienced sonographer cardiologist (JW) using echocardiographic systems of Vivid S60N or Vivid S6 (General Electric Company, Boston, MA, US) according to the unified protocol [18–21]. The measurements were taken based on the current guidelines of the European Association of Cardiovascular Imaging with real-time electrocardiographic recording performed to precisely define the phases of the cardiac cycle [22]. The measurements of RV longitudinal strain by two-dimensional speckle-tracking echocardiography were executed within six segments of the RV at the same time in the apical 4-chamber view, as described before [20, 23]. RV free wall longitudinal strain (RVFWLS) as the average value of the

Table 1. Selected echocardiographic parameters in patients with acute pulmonary embolism

	All subjects (n = 111)	Survivors (n = 99)	Non-survivors (n = 12)	*P*-value
LVTD, mm, median (IQR)	44 (39–48)	44 (40–48)	40 (37.75–46.25)	0.22
LVEF, %, median (IQR)	56 (50.5–62)	57 (50.5–62)	55.5 (48.5–60.25)	0.52
RVTD/LVTD, median (IQR)	0.92 (0.83–1.05)	0.92 (0.83–1.05)	0.94 (0.85–1.03)	0.96
TAPSE, mm, median (IQR)	21 (17–24)	21 (17–24)	19.5 (16–20.5)	0.24
RVTD/LVTD>1 and TAPSE<16 mm, n (%)	8 (7.21)	6 (6.06)	2 (16.67)	0.18
60/60 sign, n (%)	21 (18.92)	16 (16.16)	5 (41.67)	0.03
TRPG, mm Hg, median (IQR)	31.36 (25–43.56)	31.36 (25–43.56)	31.4 (23.59–42.28)	0.79
McConnell's sign or RV hypokinesis, n (%)	21 (18.92)	18 (18.18)	3 (25)	0.70
RV FAC, %, median (IQR)	39.09 (30.48–46.38)	39.09 (29.41–47.36)	39.13 (37.16–41.1)	0.97
RVFWLS, %, median (IQR)	–20 (–15 to –24.33)	–20 (–15.33 to –24.33)	–17.83 (–12.75 to –21.58)	0.19
RVGLS, %, median (IQR)	–19 (–15.33 to –22)	–19.08 (–15.46 to –22.04)	–16.17 (–13.75 to –20.08)	0.16

Abbreviations: LVTD, left ventricular transverse diameter; RV FAC, right ventricular fraction area change; RVFWLS, right ventricular free wall longitudinal strain; RVGLS, right ventricular global longitudinal strain; RVTD, right ventricular transverse diameter; TAPSE, tricuspid annular plane systolic excursion; TRPG, tricuspid valve peak systolic gradient; other — see Table 1

strain of 3 RV free wall segments and RV global longitudinal strain (RVGLS) as the average values of the strain of all 6 RV segments (free wall and septal ones) were calculated. Based on previous studies, the TTE parameters whose abnormal values indicate RV dysfunction were included in the analysis [8, 18–21].

Three different Bova score variants with various RV dysfunction criteria used in practice were included:

1. AD 2014 with the original RV dysfunction criteria: end-diastolic diameter >30 mm from the parasternal view or the RV appearing larger than the left ventricle (LV) from the subcostal or apical view (RV:LV >1), hypokinesis of the RV free wall (any view), or peak tricuspid regurgitation velocity 2.6 m/s from the apical or subcostal 4-chamber view [11, 13] and its modified versions:
2. AD 2016: RV dysfunction diagnosis considered when at least two of the following criteria are present: dilatation of the RV (end-diastolic diameter >30 mm from the parasternal view or the RV appearing larger than the LV from the subcostal or apical view — RV:LV >1), hypokinesis of the RV free wall (any view), and estimated systolic pulmonary artery pressure over 30 mm Hg [24]
3. AD 2018: at least one of the following criteria is fulfilled: RV end-diastolic diameter >30 mm (parasternal long-axis or short-axis view), RV/LV end-diastolic diameter — RV:LV >0.9 (apical or subcostal 4-chamber view), RV free wall hypokinesis from any view, tricuspid systolic velocity >2.6 m/s from the apical or subcostal 4-chamber view [25].

For comparison, other Bova score models were added to our analysis, in which echocardiographic criteria with TTE parameters indicating RV dysfunction that distinguished the survivors and non-survivors, with a *P*-value of ≤0.2, were considered (Table 1). Their predictive efficiency was tested along with one of the sPESI scores. The dichotomous approach to score interpretation was adopted from the ESC guidelines along with cut-off values of Bova (≤4 vs. >4 points) and sPESI (0 vs. ≥1 point) scores [1].

The study endpoint was 30-day overall mortality. Data collection during the follow-up of the study was described in detail in the earlier publication [18].

The study protocol was approved by the Bioethics Committee of the Regional Medical Chamber in Tarnow, Poland (No. 3/0177/2019). The study was performed in concordance with the ethical principles of the Declaration of Helsinki.

Statistical analysis

We disproved the normality of distribution with the Shapiro–Wilk test. Subsequently, quantitative variables were expressed as medians with interquartile ranges while the Mann–Whitney U-test was utilized for their comparisons. Qualitative variables were expressed as numbers (percentages) and Fisher's test or χ^2 test was used for their comparisons, when adequate. Standard receiver operating characteristic analysis was performed, and the area under the curve (AUC) was calculated for quantitative parameters and score points. Sensitivity, specificity, accuracy, and the corresponding 95% confidence interval (CI) were determined. The Youden index was used to calculate optimal cut-off values. Cox-proportional hazard models were built; only univariate models were created due to the low number of fatal events. We calculated the hazard risk (HR) for the event of death during a 30-day follow-up.

Two-sided *P*-values <0.05 were considered statistically significant. Statistical analysis was executed with the R Project for Statistical Computing version 4.3.0 (The R Foundation for Statistical Computing, Free Software Foundation Inc., Vienna, Austria).

RESULTS

The study group comprised 132 consecutive patients with confirmed PE. Six patients had high-risk PE. Fifteen subjects had echocardiograms of poor quality. In effect, 111 subjects — 55 men (49.6%), at a median age of 69 (58–79) years — were eligible to enter the analysis. The baseline

Table 2. Clinical characteristics and selected biochemical parameters of the study participants: all patients with acute pulmonary embolism, subgroups of survivors and deceased subjects in 30-day follow-up

	All subjects (n = 111)	Survivors (n = 99)	Non-survivors (n = 12)	*P*-value
Male, n (%)	55 (49.55)	52 (52.53)	3 (25)	0.12
Age, years, median (IQR)	69 (58–79)	67 (57–79)	74 (68.75–83.75)	0.02
Body mass index, kg/m^2, median (IQR)	27.55 (25.09–31.22)	27.55 (25.09–31.12)	27.3 (25.24–32.64)	0.98
Arterial hypertension, n (%)	68 (61.26)	61 (61.62)	7 (58.33)	0.83
Hyperlipidemia, n (%)	40 (36.04)	37 (37.37)	3 (25)	0.53
Diabetes, n (%)	24 (21.62)	20 (20.2)	4 (33.33)	0.29
Coronary artery disease, n (%)	24 (21.62)	22 (22.22)	2 (16.67)	0.87
Chronic heart failure, n (%)	29 (26.13)	24 (24.24)	5 (41.67)	0.24
Atrial fibrillation (present or prior), n (%)	14 (12.61)	12 (12.12)	2 (16.67)	0.65
Stroke, n (%)	2 (1.8)	2 (2.02)	0 (0)	0.81
Smoking, n (%)	10 (9.01)	9 (9.09)	1 (8.33)	0.96
Chronic lung disease, n (%)	8 (7.21)	7 (7.07)	1 (8.33)	0.93
Malignancy, n (%)	22 (19.82)	18 (18.18)	4 (33.33)	0.21
Infection, n (%)	36 (32.43)	31 (31.31)	5 (41.67)	0.47
Troponin T, pg/ml, median (IQR)	22 (10.78–44.78)	19.9 (9.94–44.08)	35.56 (19.75–79.66)	0.04
NT-proBNP, pg/ml, median (IQR)	589 (155.5–2913)	529 (141.5–2630.5)	2476 (1212.25–6106.25)	0.02
D-dimer, ng/ml, median (IQR)	4701 (2226.5–7817)	4701 (2168–8142.5)	4737 (3913–6348.75)	0.91
Creatinine clearance, mL/min, median (IQR)	82.4 (65.5–105.7)	83 (69.2–104.97)	65.5 (45.1–125.6)	0.34
sPESI, points, median (IQR)	1 (0–2)	1 (0–2)	3 (1.75–3.25)	<0.001
Bova score AD 2014, points, median (IQR)	4 (2–4)	4 (2–4)	4 (3.75–5)	0.03

Abbreviations: IQR, interquartile range; NT-proBNP, N-terminal pro-B-type natriuretic peptide; sPESI, Simplified Pulmonary Embolism Severity Index

characteristics and selected biochemical parameters are presented in Table 2.

During 30-day follow-up, 12 patients died. Six patients died due to heart failure. In the next 6 subjects, PE contributed to death by aggravating other decompensated diseases: pneumonia in 3 patients, kidney failure in 1, and disseminated neoplastic disease in 2. None of the study participants required rescue thrombolysis during the follow-up.

The patients who died during the follow-up, compared to survivors, were older and had increased troponin T and NT-proBNP serum concentrations (Table 2).

Echocardiographic parameters

The deceased patients, as compared to survivors, presented more often with the 60/60 sign. In the comparisons between subjects of opposite prognosis 3, other TTE parameters reached the assumed statistical probability values: RV:LV >1 and tricuspid annular plane systolic excursion (TAPSE) <16 mm, RVFWLS, RVGLS (Table 1).

Prediction efficiency

All these TTE markers were then used to build 4 separate Bova score variants and along with the 3 variants of the Bova score used previously in the literature were altogether tested for their prediction efficiency compared to the sPESI. The optimal estimated cut-off value for RVFWLS was –19% and for RVGLS was –17%. All scores, but not the Bova 2016 version, had predictive value in our analysis. The only score with good predictive value (exceeding 0.8) measured with AUC was the sPESI and with fair strength (exceeding 0.7) — the Bova score with the 60/60 sign and the Bova score with RVFWS >–19% [26]. Bova scores showed lower sensitivity and higher specificity while the sPESI showed the opposite. All the scores showed high negative predictive value and low positive predictive value. The proportion of correctly classified patients was low in Bova AD-2014, 2016, and 2018 score variants and higher in the sPESI, Bova scores with the 60/60 sign, with RVFWS >–19% and with RVGLS >–17% (Table 3).

The subjects with Bova score variants with points >4 had a higher risk of death (HR of 1.43–1.59), apart from the 2016 Bova score version, which did not show significant associations, and subjects with the sPESI with ≥1 point had a higher mortality rate with an HR of 2.02 (Table 4).

DISCUSSION

Short-term outcomes in acute PE are mainly determined by patients' hemodynamic status. Not surprisingly, RV dysfunction, defined as the presence of signs of RV pressure overload on imaging examinations (echocardiography or computed tomography) among low-risk patients or as myocardial injury based on elevated cardiac troponins or natriuretic peptides, has been shown to be associated with increased risk of mortality [27]. As for echocardiography, numerous studies have demonstrated consistent associations between various TTE parameters and short-term mortality in unselected patients with acute PE [18, 20, 28–30].

Interestingly, RV dysfunction has no generally accepted definition. In the article by Pruszczyk et al. [31] on 490 normotensive individuals with PE, the combined RV dysfunction criterion of the RV-to-LV ratio >1 with TAPSE <16 mm showed a positive predictive value of 23.3% with a high negative predictive value of 95.6% regarding the composite

Table 3. Efficiency of different clinical scores and their modifications in predicting fatal out-come in non-high-risk patients with acute pulmonary embolism in 30-day follow-up. A dichotomous approach to scores was used (sPESI: 0 vs. ≥1 points; all Bova scores: ≤4 vs. >4 points)

	AUC, value, (95% CI)	Sensitivity, value, (95% CI)	Specificity, %, value, (95% CI)	Positive predictive value, %, value, (95% CI)	Negative predictive value, %, value, (95% CI)	Correctly classified, n (%)	*P*-value
sPESI score	0.815 (0.706, 0.925)	75 (42.81–94.51)	71.72 (61.78–80.31)	71.72 (61.78–80.31)	95.95 (88.61–99.16)	80 (72.07)	<0.001
Bova score AD 2014	0.679 (0.523, 0.835)	44.44 (34.45–54.78)	75 (42.81–94.51)	14.06 (6.64–25.02)	93.62 (82.46–98.66)	53 (47.75)	0.03
Bova score modification AD 2016	0.597 (0.419, 0.775)	41.67 (15.17–72.33)	60.61 (50.28–70.28)	11.36 (3.79–24.56)	89.55 (79.65–95.7)	65 (58.56)	0.26
Bova score modification AD 2018	0.675 (0.518, 0.833)	43.43 (33.5–53.77)	75 (42.81–94.51)	13.85 (6.53–24.66)	93.48 (82.1–98.63)	52 (46.85)	0.03
Bova score with RVTD/LVTD >1 and TAPSE <16 mm	0.652 (0.486, 0.818)	25 (5.49–57.19)	91.92 (84.7–96.45)	27.27 (6.02–60.97)	91 (83.6–95.8)	94 (84.68)	0.03
Bova score with 60/60 sign	0.731 (0.586, 0.877)	41.67 (15.17–72.33)	84.85 (76.24–91.26)	25 (8.66–49.1)	92.31 (84.79–96.85)	89 (80.18)	0.01
Bova score with RVFWLS >–19%	0.701 (0.529, 0.874)	41.67 (15.17–72.33)	81.82 (72.8–88.85)	21.74 (7.46–43.7)	92.05 (84.3–96.74)	86 (77.48)	0.02
Bova score with RVGLS >–17%	0.663 (0.496, 0.83)	16.67 (2.09–48.41)	93.94 (87.27–97.74)	25 (3.19–65.09)	90.29 (82.87–95.25)	95 (85.59)	0.053

Abbreviations: AUC, area under the curve; CI, confidence interval; other — see Tables 1 and 2

Table 4. Hazard risk analysis of different scores in predicting 30-day all-cause mortality in patients with acute pulmonary embolism. A dichotomous approach to scores was used (sPESI: 0 vs. ≥1 points; all Bova scores: ≤4 vs. >4 points)

	Hazard risk	95% confidence interval	*P*-value
sPESI	2.02	1.38–2.95	<0.001
Bova score AD 2014	1.59	1.05–2.38	0.03
Bova score modification AD 2016	1.22	0.89–1.67	0.21
Bova score modification AD 2018	1.58	1.05–2.38	0.03
Bova score with RVTD/LVTD >1 and TAPSE <16 mm	1.46	1.04–2.05	0.03
Bova score with 60/60 sign	1.43	1.10–1.85	0.01
Bova score with RVFWLS >–19%	1.45	1.04–2.02	0.03
Bova score with RVGLS >–17%	1.49	1.02–2.18	0.04

Abbreviations: see Tables 1 and 2

endpoint of PE-related mortality, hemodynamic collapse, or rescue thrombolysis with a significant HR of 6.5 (95% CI, 3.2–13.3; *P* <0.001). Importantly, a recent meta-analysis performed to assess the role of different definitions of RV dysfunction and its parameters as predictors of death demonstrated that TTE RV dysfunction, regardless of its criteria, was associated with increased risk of death (risk ratio 1.49; 95% CI, 1.24–1.79; $I^2 = 64\%$) and PE-related death (risk ratio 3.77; 95% CI, 1.61–8.80; $I^2 = 0\%$) in all-comers with PE, and with death in hemodynamically stable patients (risk ratio 1.52; 95% CI, 1.15–2.00; $I^2 = 73\%$). In patients with PE, an increased RV-to-LV ratio and TAPSE but not increased RV diameter were associated with death, whereas in hemodynamically stable patients the RV-to-LV ratio and TAPSE were not significantly associated with mortality. The authors of that meta-analysis concluded that as the appraisal of RV dysfunction with TTE is a useful tool for risk stratification in all-comers with acute PE and hemodynamically stable patients, the prognostic value of individual parameters of RV dysfunction in hemodynamically stable patients remains controversial [8]. It is noteworthy that those studies focused only on classic TTE parameters including ventricular diameters, interventricular septum flattening, RV hypokinesis, TAPSE, and appraisal of tricuspid valve peak systolic gradient, and their combinations. Neither of the discussed analyses incorporated the 60/60 sign nor longitudinal strain assessment of RV.

The 60/60 sign, which combines a tricuspid regurgitation jet gradient of ≤60 mm Hg and pulmonary ejection acceleration time ≤60 ms, serves as a marker of elevated pulmonary arterial pressure related to the presence of embolic obstacles within pulmonary arteries with subsequent increased pulmonary vascular resistance along with elevated RV wall strain in acute PE [32, 33]. A healthy RV in patients without chronic pulmonary or left heart diseases evoking pulmonary hypertension is usually insufficient to maintain pulmonary artery systolic pressure >60 mm Hg [34]. The prevalence of the 60/60 sign in acute PE ranged from 12.9% to 70.8% in various studies [35, 36]. The sign is highly specific to PE diagnosis but is characterized by poor sensitivity [32]. It is also a useful TTE finding to differentiate acute PE and chronic pulmonary hypertension [37]. Since the 60/60 sign is observed in a small percentage of all-comers with PE, it is seldom included in prognostic assessment after PE. Interestingly, in a recent survey, the 60/60 sign was an independent predictor of short-term

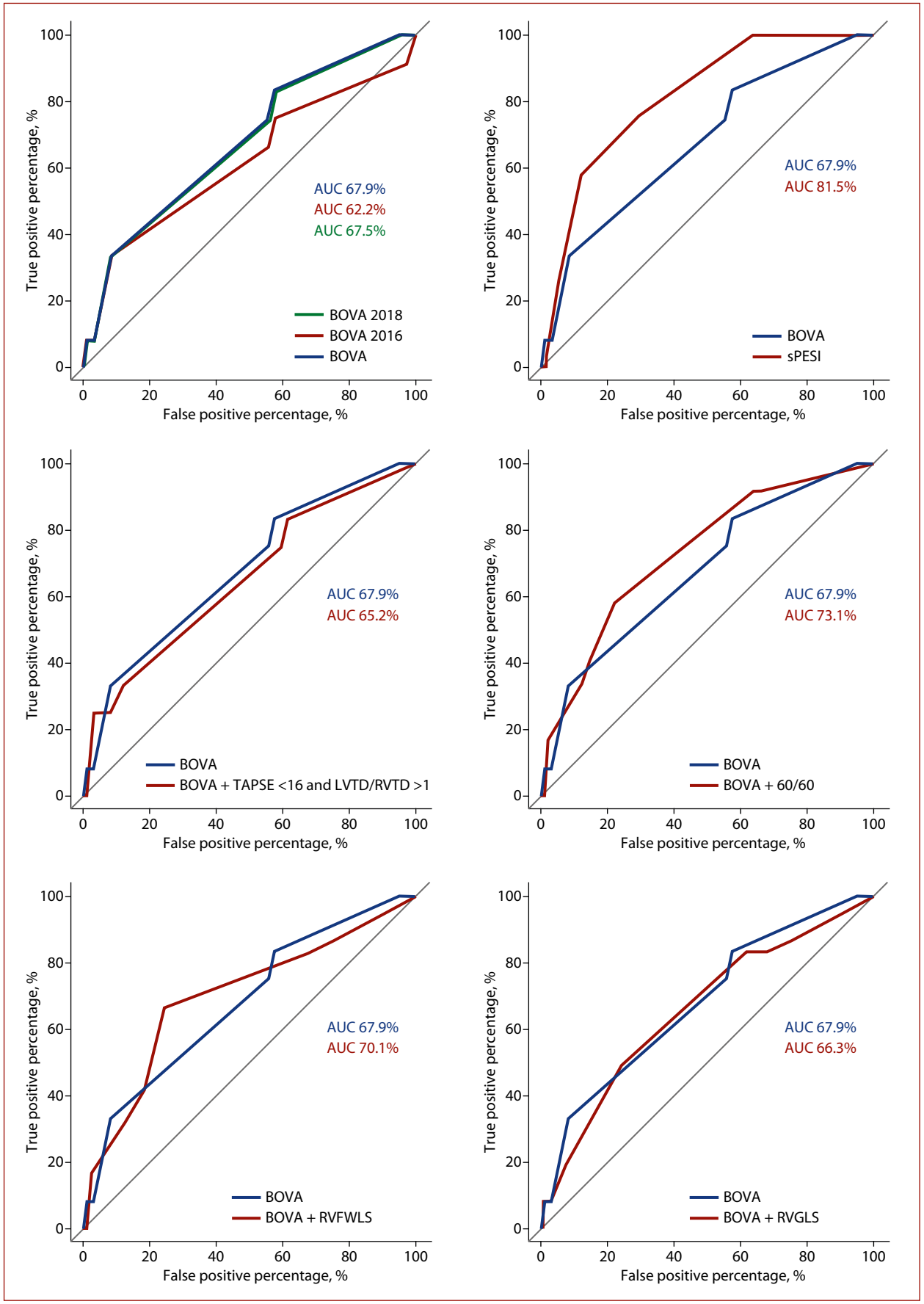

Figure 1. Comparison of receiver operating characteristic curves for simplified Pulmonary Embolism Severity Index (PESI) and different models of the Bova score with various echocardiographic parameters of right ventricular dysfunction

Abbreviations: AUC, area under the curve; other — see Table 2

mortality in patients with acute PE (odds ratio 8.13; 95% CI, 1.11–59.21; $P = 0.034$) [36].

In an Australian study, the difference in RVFWLS between PE subjects and healthy individuals was a great discriminator for PE. In comparative multiple logistic regression models for PE, RVFWLS produced a powerful classifier (AUC 0.966; sample entropy 0.013; $P < 0.022$) with significantly better performance than the model which included traditional measures of RV size and function but without RVFWLS [38]. Moreover, in a recent prospective study, RVFWLS was found to be the most common abnormal echocardiographic marker of RV dysfunction in patients with acute PE. RVFWLS correlated with D-dimer and NT-proBNP concentrations and differed significantly between patients with a sPESI of low risk and those of high risk ($P < 0.001$) [39]. In the publication by Dahhan et al. [29] RVFWLS and RVGLS, in addition to the Tei index, were the only TTE predictors of mortality after acute PE, whereas in the paper by Lee et al. [40], RVFWLS and RVGLS independently predicted in-hospital events: all-cause death, need for additive treatments such as thrombolysis or pulmonary artery thromboembolectomy, and need for inotropic agents due to unstable hemodynamic status. RVFWLS was also a predictor of mortality after acute PE in one-year follow-up [41]. Importantly, compared to RVFWLS, the value of RVGLS is more affected by LV disorders and especially conditions influencing the performance of interventricular septum, including coronary artery diseases and chronic heart failure, thus RVFWLS appears to be more accurate in PE. On the other hand, despite the growing clinical usefulness of longitudinal strain of myocardium appraisal, it is currently not part of routine TTE examinations in many echocardiographic laboratories [38, 42].

Nevertheless, considering predictive value, generally, the performance of TTE parameters is moderate at best when compared to composed clinical scores and their incorporation into clinical scores does not bring the expected benefits [21, 43]. First, RV dysfunction might be also evoked by some preexisting chronic conditions of the heart and lungs not only by acute PE. Second, composed scores include clinical variables that can hugely impact the outcome such as signs of hemodynamic incompetence, age, or chronic diseases with poor prognosis: cancer, chronic heart failure, or chronic pulmonary disease, etc. [44]. Third, what has been recently demonstrated using the PESI as an example, quantitative TTE parameters whose incorrect values reflect RV dysfunction such as TAPSE, RVFWLS, and RVGLS correlated with PESI scores and, therefore, augmented its predictive value to a limited extent when added to the PESI scale. On the contrary, a thrombus in the right heart cavity and the 60/60 sign did not correlate with the PESI score, and as PESI adjuncts, they independently predicted fatal outcomes: the thrombus with an HR of 10.04 (95% CI, 2.81–37.12; $P < 0.001$) and the 60/60 sign with an HR of 4.07 (95% CI, 1.27–12.81; $P < 0.001$) [21].

To summarize, various TTE markers of RV dysfunction within divergent variants of Bova score models in non-high-risk patients with PE yield different prediction strengths but are all inferior to the sPESI score. Among Bova score TTE variants, the most efficient ones include Bova with the 60/60 sign and Bova with RVFWLS >–19%. A holistic approach to assessment of prognosis including clinical characteristics, diagnostic imaging but also biochemical markers is reasonable. Additional clinical information could improve predictability that is not provided by a single scoring system [45].

CONCLUSIONS

Different criteria of RV dysfunction as components of the Bova score in hemodynamically stable patients with acute pulmonary embolism affect its prognostic efficacy but to a limited extent. The assessment of tricuspid regurgitation jet gradient with pulmonary ejection acceleration time and the longitudinal strain of the free wall of the RV provides the most valuable markers of RV dysfunction in the prognostic value of the Bova score. However, they have less strength than the sPESI score.

Study limitation

The study had a relatively low number of participants. The variability of echocardiographic parameters could not be assessed as echocardiograms were not repeated. The prognostic role of biomarkers was not investigated.

Article information

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