ORIGINAL ARTICLE

Subcostal echocardiographic assessment of tricuspid annular kick (SEATAK): A novel independent predictor of 30-day mortality in patients with acute pulmonary embolism

Jerzy Wiliński[1], Anna Skwarek[1], Radosław Borek[1], Ositadima Chukwu[2], Katarzyna Ciuk[2], Katarzyna Stolarz-Skrzypek[3], Marek Rajzer[3]

[1]Department of Internal Medicine with Cardiology Subdivision, Blessed Marta Wiecka District Hospital, Bochnia, Poland
[2]Jagiellonian University Medical College, Kraków, Poland
[3]1st Department of Cardiology, Interventional Electrocardiology and Arterial Hypertension, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland

Correspondence to:
Jerzy Wiliński, MD, PhD,
Department of Internal Medicine with Cardiology Subdivision,
Blessed Marta Wiecka District Hospital,
Krakowska 31,
32–700 Bochnia, Poland,
phone: +48 14 615 33 17,
e-mail: putamen@interia.pl

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ABSTRACT

Background: The most commonly used parameter of right ventricular (RV) systolic function — tricuspid annular plane systolic excursion (TAPSE) — is unavailable for some patients. Subcostal echocardiographic assessment of tricuspid annular kick (SEATAK) has been proposed as its alternative.

Aim: The study aimed to assess the feasibility of SEATAK use in patients with acute pulmonary embolism (PE) and its value in prognosis after PE.

Methods: The observational study included 164 consecutive patients (45.7% men; average age, 70 years) with a high clinical probability of PE referred for computed tomography pulmonary angiography.

Results: SEATAK was unavailable due to inadequate quality of echocardiogram in 2.8% of patients, whereas TAPSE could not be calculated in 4.9%, both parameters were not estimated only in 0.6%. SEATAK and TAPSE values did not differ between groups of patients with PE (n = 82) and without (n = 82). In the whole study, SEATAK correlated positively with TAPSE (r = 0.71; 95% confidence interval [CI], 0.62–0.78; P <0.001), fractional area change of the RV, left ventricular ejection fraction, and peak systolic tricuspid annular velocity assessed with tissue Doppler imaging. There were only 3 echocardiographic predictors of 30-day all-cause mortality in patients with with PE (n = 10): SEATAK, pulmonary acceleration time, and the 60/60 sign. SEATAK predicted 30-day all-cause mortality with AUC (area under the curve) 0.726 (95% CI, 0.594–0.858; P = 0.01) and 30-day PE-related mortality (n = 4) with AUC, 0.772 (95% CI, 0.506–0.998; P = 0.03).

Conclusions: SEATAK is a promising practicable echocardiographic parameter reflecting RV systolic function and might be an accurate alternative to TAPSE. Moreover, SEATAK could be an independent predictor of all-cause and PE-related 30-day mortality in patients with acute PE.

Key words: echocardiography, pulmonary embolism, tricuspid annular plane systolic excursion, subcostal view

INTRODUCTION

Recent years have brought much interest in the physiology and pathology of the right ventricle (RV) [1]. Echocardiographic assessment of RV systolic function becomes relevant for multiple cardiopulmonary conditions including acute pulmonary embolism (PE) [2, 3]. Since the muscle fiber arrangement in the RV makes its contraction occur primarily in the longitudinal plane, it can be simply assessed with classic echocardiography [4]. The most commonly used parameter of RV systolic function in M-mode is tricuspid annular plane systolic excursion (TAPSE), which was introduced almost 40 years ago, in 1984, by Kaul and colleagues [5]. TAPSE has been demonstrated to be accurate, reproducible, and simple to evaluate. It has its place in

WHAT'S NEW?

Transthoracic echocardiography is an underestimated tool in acute pulmonary embolism. Tricuspid annular plane systolic excursion has been demonstrated to be an accurate, reproducible, and simple-to-evaluate echocardiographic parameter, but due to technical reasons, it is not appropriate for a significant number of patients. We found that a novel tool — subcostal echocardiographic assessment of tricuspid annular kick is an accurate alternative to the conventional tricuspid annular plane systolic excursion, highly reflective of right ventricular systolic function and also is an independent predictor of all-cause and pulmonary embolism-related 30-day mortality in patients with acute pulmonary embolism.

current guidelines as a part of a transthoracic echocardiographic examination (TTE) [6, 7]. Furthermore, TAPSE shows prognostic significance in patients with acute PE and pulmonary hypertension [2, 8].

Nevertheless, TAPSE is dependent on the transducer position and alignment with the tricuspid annulus, often requiring a change of the patient's position to left lateral decubitus. It might be problematic, inter alia, in patients in the intensive care unit, individuals in serious conditions, or during cardiopulmonary resuscitation. Furthermore, inadequate visualization of the tricuspid annulus in the apical view poses another disadvantage. This is commonly encountered in persons with chronic lung diseases, mechanical ventilation, or obesity [4].

The subcostal echocardiographic view is free of some of these limitations. It can be obtained more easily in some patients with chronic lung disease and RV enlargement and in immobilized ones. The assessment of movement of the tricuspid annulus within the subcostal view in PE has never been investigated. The semiquantitative evaluation of RV systolic function using M-mode in the modified subcostal view with the systolic excursion assessment of tricuspid annular kick (SEATAK) was proposed by Díaz-Gómez et al. in 2016 as an alternative to TAPSE in critically ill patients [9]. Thus, SEATAK evaluates the same phenomenon as TAPSE but from a different perspective.

The study aimed to assess the feasibility of SEATAK use in assessment of RV systolic function in patients with PE and the role of this echocardiographic parameter in the short-term prognosis of patients with acute PE.

METHODS

Study group

This was a cross-sectional observational single-center study. The study population included consecutive patients of the Internal Medicine Department and the Special Care Cardiac Unit with a high clinical probability of PE referred for computed tomography pulmonary angiography (CTPA) between August 1, 2018 and August 31, 2020. The treatment followed the guidelines on PE management of the European Society of Cardiology [10, 11]. In summary, unfractionated heparin was used exclusively in high-risk PE patients along with alteplase. All non-high-risk patients and high-risk patients at a later stage of treatment received enoxaparin subsequently replaced with dabigatran, on rare occasions with warfarin or acenocoumarol, alternatively apixaban or rivaroxaban from the beginning of the PE treatment.

The exclusion criteria included recurrent PE, chronic thromboembolic pulmonary hypertension, echocardiograms of inadequate quality, severe valvular defects, and tricuspid valve replacement.

A standard diagnostic protocol comprised determination in all patients on the day of admission to the ward the laboratory parameters including, inter alia, creatinine, estimated creatinine clearance calculated with the Cockcroft-Gault equation, troponin T concentrations determined with high-sensitivity automated sandwich electrochemiluminescence immunoassay (Roche Diagnostics GmbH, Mannheim, Germany), N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels measured using the enzyme-linked immunosorbent assay (ELISA, Roche Diagnostics GmbH, Mannheim, Germany) and D-dimer concentrations using an automated enzyme-linked fluorescent assay (VIDAS D-dimer Exclusion, bioMerieux, Marcy-l'Étoile France).

Echocardiographic assessment

Transthoracic echocardiograms were performed within 24 hours after admission to the ward by an experienced sonographer cardiologist (JW) using commercially available echocardiographic systems of Vivid S60N or Vivid S6 (General Electric Company, Boston, MA, US) according to the same protocol. The measurements were made based on the current guidelines with real-time electrocardiographic recording to precisely define the phases of the heart cycle. The estimation of SEATAK was utilized according to the method by Díaz-Gómez and colleagues from the Mayo Clinic. Briefly, the subcostal four-chamber view was obtained with an average depth of 20 to 24 cm. Then a counterclockwise rotation was applied to acquire the subcostal short-axis view upon which the right atrium, RV, tricuspid annulus, and inferior vena cava could be identified. Subsequently, the cursor was aligned in real-time with M-mode echocardiographic imaging with the tricuspid annulus to obtain a linear measurement from end-diastole to end-systole i.e. SEATAK [9, 12]. The average values of every single echocardiographic parameter were calculated from 3 cardiac cycles using the incorporated sonography software.

Study endpoint

The study endpoints were 30-day overall mortality and 30-day PE-related mortality. Data about mortality was based on hospital records (the only hospital operating in the district), the government electronic system collecting data about individuals covered by public insurance, and phone calls to primary care physicians, patients, and their families. The cause of death was determined mainly based on hospital records, possibly on documentation from the facility where the patient died (other hospitals, nursing homes, etc.) or corresponding general practitioners.

Ethical issues

The study protocol complied with the Declaration of Helsinki and was approved by the Bioethics Committee of the Regional Medical Chamber in Tarnow, Poland (no. 3/0177/2019).

Statistical analysis

Quantitative variables with normal distribution are expressed as mean with standard deviation, whereas quantitative variables with non-normal distribution as median with interquartile range. Student's t-test or the Mann-Whitney U test were accordingly used for their comparisons. Qualitative variables are expressed as numbers (percentage), Fisher's test or the χ^2 test were used for comparisons, when adequate. Pearson or Spearman correlations were calculated to assess the relationship between SEATAK and other RV systolic parameters.

Early mortality and PE-related mortality were treated as right-censored data. Standard Kaplan-Mayer curves were used for 30-day survival analysis, the log-rank test was used for comparisons. Hazard risk was calculated using Cox proportional-hazards regression for early mortality. The proportional hazard assumption was checked with the Grambsch-Therneau test. Due to an insufficient number of events, we withdrew from regression analysis of PE-related mortality. Receiver operating characteristic analysis was performed, and areas under curves (AUC) were calculated. Optimal cut-off values were delineated according to the maximum sensitivity method. Sensitivity, specificity, positive and negative predictive values (PPV and NPV, respectively), and the corresponding 95% confidence interval (CI) were calculated for SEATAK and TAPSE. Two-sided *P*-values <0.05 were considered statistically significant and were not adjusted for multiple testing. Statistical analysis was performed with the R Project for Statistical Computing version 3.6.3 (The R-Foundation for Statistical Computing, Free Software Foundation Inc., Vienna, Austria).

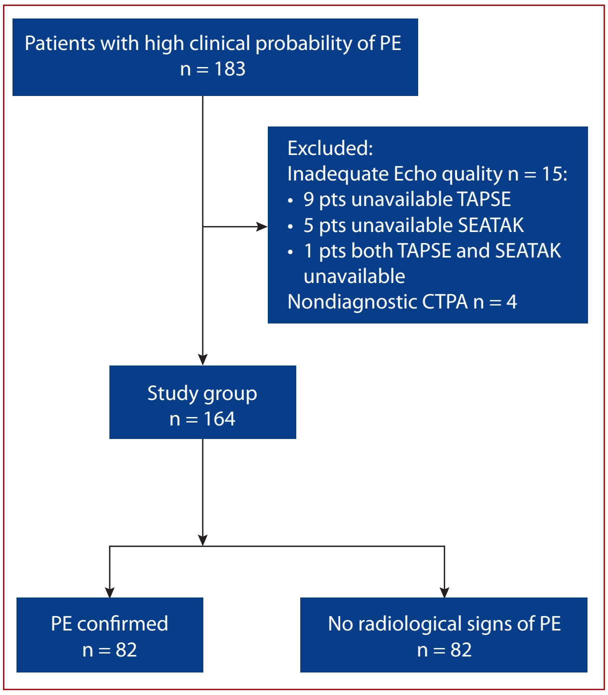

Figure 1. Flow chart of the study

Abbreviations: CTPA, computed tomography pulmonary angiography; PE, pulmonary embolism; SEATAK, subcostal echocardiographic assessment of tricuspid annular kick; TAPSE, tricuspid annular plane systolic excursion

RESULTS

The study comprised 183 consecutive patients. Twelve patients had echocardiograms of poor quality; in 9 (4.9%) individuals TAPSE could not be calculated, in further 5 (2.8%), SEATAK was not available, and in one (0.6%) both parameters were not estimated. Four subjects had nondiagnostic CTPA (Figure 1). Excluded patients had similar clinical characteristics, and there were no significant differences in age, sex, presence of PE, or studied echocardiographic parameters. Finally, 164 individuals were eligible to be enrolled in the study. The baseline characteristics and biochemical parameters of these patients are presented in Table 1. Exactly half of the participants had PE confirmed: 37 subjects had central PE (45.12%), whereas 45 individuals (54.88 %) had peripheral PE. Within this group, 4 patients were classified with high-risk PE, 23 with intermediate-high risk, 32 with intermediate-low risk, and 23 with low-risk PE.

The patients with PE, compared to subjects without PE, had higher body mass index, D-dimer serum concentration, and less often presented with coronary artery disease and chronic heart failure (Table 1).

Ten study participants (12.2%) of the PE group died during the 30-day follow-up. Four patients (4.88%) required thrombolysis within 24 hours from admission to the ward. Two of them (2.44%) died, and two (2.44%) survived. Another 2 patients (2.44%) died due to PE which in effect caused refractory RV heart failure. In the next 6 subjects (7.32%), PE contributed to death by aggravating

Table 1. Clinical characteristics and selected biochemical parameters of the study participants: all patients, subgroups of individuals with and without acute pulmonary embolism, deceased subjects, and survivors within 30-days of observation

	All subjects (n = 164)	Patients with PE (n = 82)	Patients with no PE (n = 82)	*P*-value	Non-survivors (n = 10)	Survivors (n = 72)	*P*-value
Male sex, n (%)	75 (45.73)	40 (48.78)	35 (42.68)	0.43	2 (20)	38 (52.78)	0.09
Age, years, median (IQR)	70 (59.75–80)	70 (58.25–80)	70.5 (60.25–79)	0.90	79.5 (72.25–89.75)	67.5 (57–79.25)	0.006
BMI, kg/m^2, mean (SD)	27.36 (5.95)	28.32 (5.49)	26.43 (6.25)	0.043	28.26 (6.6)	28.33 (5.38)	0.98
Arterial hypertension, n (%)	59 (35.98)	27 (32.93)	32 (39.02)	0.42	6 (60)	21 (29.17)	0.07
Hyperlipidemia, n (%)	43 (26.22)	18 (21.95)	25 (30.49)	0.21	3 (30)	15 (20.83)	0.68
Diabetes mellitus, n (%)	19 (11.59)	12 (14.63)	7 (8.54)	0.22	4 (40)	8 (11.11)	0.04
Coronary artery disease, n (%)	31 (18.9)	8 (9.76)	23 (28.05)	0.003	1 (10)	7 (9.72)	1
Chronic heart failure, n (%)	29 (17.68)	9 (10.98)	20 (24.39)	0.02	3 (30)	6 (8.33)	0.07
Atrial fibrillation (present or prior), n (%)	16 (9.76)	4 (4.88)	12 (14.63)	0.06	0 (0)	4 (5.56)	1
History of stroke, n (%)	3 (1.83)	0 (0)	3 (3.66)	0.23	0 (0)	0 (0)	1
Active smoking, n (%)	19 (11.59)	9 (10.98)	10 (12.2)	1	1 (10)	8 (11.11)	1
Chronic lung disease, n (%)	11 (6.71)	3 (3.66)	8 (9.76)	0.21	1 (10)	2 (2.78)	0.33
Active malignancy, n (%)	35 (21.34)	17 (20.73)	18 (21.95)	0.81	3 (30)	14 (19.44)	0.69
Acute infection, n (%)	25 (15.24)	13 (15.85)	12 (14.63)	0.83	3 (30)	10 (13.89)	0.19
Wells rule — original version, points, median (IQR)	3 (1.5–4.5)	3 (1.5–5.5)	1.5 (1.125–4.5)	0.13	3.75 (1.875–4.75)	3 (0.75–5.5)	0.84
Revised Geneva rule — original version, points, median (IQR)	5 (4–6)	5 (4–7)	5 (4.125–6)	0.57	6 (6–7.25)	5 (4–6.25)	0.07
PESI, median (IQR)	94 (80–118)	97 (81–123)	93 (79.25–116.5)	0.44	126.5 (106–141.5)	94 (79–111)	0.003
sPESI, median (IQR)	1 (0–2)	1 (0–2)	1 (0–2)	0.88	2.5 (2–3)	1 (0–2)	0.004
Troponin T, pg/ml, , median (IQR)	22.18 (12.06–57.87)	27.8 (11.21–65.61)	19 (13–49.93)	0.40	70.65 (52.965–168.885)	22.6 (10.82–51.63)	0.01
NT-proBNP, pg/ml, median (IQR)	1077 (201– 4454)	1232 (155–3623)	947 (249–4813)	0.61	3837 (1839 – 11688)	597 (143–2944)	0.02
D-dimer, µg/ml, median (IQR)	4050 (1991–7301)	5531 (2915–8477)	3197 (1630–5141)	<0.001	8477 (4737–10000)	5369 (2712 –7823)	0.09
Creatinine clearance, ml/min, median (IQR)	82.45 (61.85–103.63)	82.4 (65.5–102)	82.5 (60.1–103.9)	0.68	65.5 (37.7–112.5)	83.55 (69.60–101.48)	0.27

Abbreviations: PE, pulmonary embolism; BMI, body mass index; PESI, Pulmonary Embolism Severity Index; sPESI, simplified Pulmonary Embolism Severity Index; NT-proBNP, N-terminal pro-B-type natriuretic peptide

other decompensated diseases: heart failure in 2 (2.44%), pneumonia in 2 (2.44%), kidney failure in 1 (1.22%), and disseminated neoplastic disease in 1 (1.22%). None of the study participants required rescue thrombolysis in the observational period. The median time of hospitalization was 9 days ranging from 1 to 30 days.

The patients who died in the follow-up, compared to survivors, were older, less frequently had diabetes mellitus; however, they had higher scores in the Pulmonary Embolism Severity Index (PESI) and simplified PESI (sPESI), increased troponin T and NT-proBNP serum concentrations (Table 1).

Echocardiographic parameters

In the whole study group, SEATAK showed smaller values than TAPSE (18.22 ± 5.63 mm vs. 20.17 ± 5.9 mm, P <0.001).

SEATAK and TAPSE did not differ between the groups of patients with and without PE (Table 2).

Patients with PE compared to individuals with no signs of PE upon CTPA had higher values of the ratio of basal right ventricular end-diastolic diameter measured in the transverse view (RVTD) to basal left ventricular end-diastolic diameter measured in the transverse view (LVTD), decreased values of pulmonary artery acceleration time (Act). On the other hand, they presented more frequently with the 60/60 and McConnell signs (Table 2).

Non-survivors had reduced values of SEATAK, TAPSE, RVTD, and Act but more often showed a positive 60/60 sign when compared to the survivors (Table 2).

Relation of SEATAK to other echocardiographic parameters

In the whole study, SEATAK correlated positively with TAPSE (r = 0.71; 95% CI, 0.62–0.78; P <0.001), fractional area change of the RV (FAC) (r = 0.29; 95% CI, 0.02–0.53; P = 0.04), left ventricular ejection fraction (LVEF; r = 0.36; 95% CI, 0.22–0.48; P <0.001), and peak systolic tricuspid annular velocity assessed with tissue Doppler imaging (TSV TDI) (r = 0.47; 95% CI, 0.34–0.58; P <0.001). Neither was SEATAK associated with a Right Ventricular Index of Myocardial Performance (Tei index) measured with tissue Doppler imaging (r = 0.01; 95% CI, –0.15–0.18; P = 0.87) nor with Pulsed-Wave Doppler mode (r = 0.07; 95% CI, –0.1–0.22; P = 0.43).

Echocardiographic predictors of 30-day mortality

The univariable Cox proportional-hazard regression analysis revealed 3 echocardiographic predictors of 30-day all-cause mortality in patients with acute PE: SEATAK, Act,

Table 2. Selected echocardiographic parameters

	Patients with PE (n = 82)	Patients with no PE (n = 82)	*P*-value	Non-survivors (n = 10)	Survivors (n = 72)	*P*-value
SEATAK, mm	17.68 ± 5.71	18.76 ± 5.52	0.22	13.90 ± 3.96	18.21 ± 5.74	0.009
TAPSE, mm	19.93 ± 6.06	20.41 ± 5.77	0.60	16.60 ± 4.12	20.39 ± 6.16	0.02
TASV TDI, cm/s	15.12 ± 4.74	15.72 ± 5.68	0.47	15.44 ± 4.72	15.08 ± 4.78	0.83
RVTD, mm	40 (37–43)	38 (35–42)	0.09	36 (35–38)	40.5 (37–44.25)	0.03
LVTD, mm	42.82 ± 7.1	43.84 ± 7.72	0.39	38.56 ± 6.41	43.38 ± 7.04	0.06
RVTD/LVTD	0.94 (0.83–1.06)	0.87 (0.78–1.03)	0.04	1.03 (0.88–1.1)	0.94 (0.83–1.06)	0.61
Act, ms	70 (55–88)	93 (71.75–115)	<0.001	59 (48–59)	74 (57–90.25)	0.02
TRV, m/s	2.9 ± 0.79	2.8 ± 0.71	0.44	2.95 ± 0.78	2.89 ± 0.8	0.82
TRPG, mm Hg	36 (27–52.5)	36 (27–48)	0.75	40 (34–48)	35.5 (26.25–52.75)	0.64
60/60 sign, n (%)	20 (24.39)	5 (6.1)	0.001	6 (60.00)	14 (19.44)	0.003
McConnell sign, n (%)	9 (10.98)	1 (1.22)	0.02	1 (10)	8 (11.11)	1
IVS flattening, n (%)	4 (4.88)	6 (7.32)	0.75	0	4 (5.56)	1
Distended IVC with diminished inspiratory collapsibility, n (%)	7 (8.54)	4 (4.88)	0.54	1 (10)	6 (8.33)	1
LVEF, %	54.5 (49.25–61.5)	55.5 (45–64.75)	0.96	54 (43.5–59)	54.5 (49.75–62)	0.70

Abbreviations: Act, pulmonary acceleration time; IVC, inferior vena cava; IVS, interventricular septum; LVEF, left ventricular ejection fraction; LVTD, basal left ventricular end-diastolic diameter measured in the transverse view; RVTD, basal right ventricular end-diastolic diameter measured in the transverse view; SEATAK, subcostal echocardiographic assessment of tricuspid annular kick; TAPSE, tricuspid annular plane systolic excursion; TASV TDI, tricuspid annulus' peak systolic velocity measured with tissue Doppler imaging; TRV, tricuspid regurgitation jet velocity; TRPG, tricuspid valve peak systolic gradient; other — see Table 1

Table 3. Univariable analysis of echocardiographic predictors of all-cause 30-day mortality in patients with acute pulmonary embolism (n = 82)

	All-cause 30-day mortality	
	HR (95% CI)	*P*-value
SEATAK, mm	0.86 (0.76–0.98)	0.02
TAPSE, mm	0.90 (0.80–1.00)	0.06
RVTD, mm	0.90 (0.79–1.01)	0.08
LVTD, mm	0.91 (0.82–1.00)	0.06
Act, ms	0.95 (0.91–1.00)	0.04
TRV, m/s	1.11 (0.49–2.54)	0.80
TRPG, mm Hg	1 (0.97–1.04)	0.94
60/60 sign, %	5.39 (1.519–19.11)	0.009
Distended IVC with diminished inspiratory collapsibility, %	1.14 (0.14–8.97)	0.90

Abbreviations: CI, confidence interval; HR, hazard ratio; RVTD, basal right ventricular end-diastolic diameter measured in the transverse view; LVTD, basal left ventricular end-diastolic diameter measured in the transverse view; other — see Table 2

and 60/60 sign. TAPSE did not reach statistical significance. Additionally, the 60/60 sign was present in all the subjects who died of PE (Table 3).

SEATAK and TAPSE as predictors of 30-day mortality

The receiver operating characteristic investigation disclosed that SEATAK is a good predictor of 30-day all-cause mortality (AUC, 0.726) and 30-day PE-related mortality (AUC, 0.772). TAPSE was a predictor of PE-related mortality (AUC, 0.793) and death from any cause (AUC, 0.690) (Figure 2).

Optimal cut-offs for predicting all-cause mortality were <20 mm for SEATAK and <21 for TAPSE. With those cut-offs, both SEATAK and TAPSE showed high sensitivity (100% and 90%, respectively) and PPV (100% and 97%, respectively) but low specificity (43% and 50%, respectively) and NPV (20% both) for adverse prognosis.

Optimal cut-offs for predicting PE-related mortality were <17 mm for both SEATAK and TAPSE. With those cut-offs, both SEATAK and TAPSE showed again high sensitivity (75% and 74%, respectively) and PPV (98% both) but low specificity (54% and 72%, respectively) and NPV (8% and 12%, respectively) for fatal outcomes.

Kaplan-Meier analysis showed favorable outcomes for patients with SEATAK ≥20 mm and TAPSE ≥21 mm in terms of all-cause mortality and for individuals with SEATAK and TAPSE ≥17 mm regarding PE-related death (Figure 3).

SEATAK and TAPSE were neither correlated with age (Spearman correlation coefficient, 0.04 and –0.11, P = 0.63 and 0.17, respectively) nor with D-dimer levels (Spearman correlation coefficients, –0.10 and –0.03; P = 0.26 and 0.68, respectively). In multivariable analysis, SEATAK was a predictor of overall mortality when controlled with age (P = 0.03).

DISCUSSION

TTE is not a mandatory part of routine diagnostics in hemodynamically stable patients with PE [11]. Although short-term outcomes in acute PE are mainly conditioned by the hemodynamic status, RV dysfunction detected, inter alia, in TTE is associated with an increased risk of short-term mortality even in normotensive individuals [13, 14]. Moreover, TTE enables close monitoring to detect hemodynamic decompensation and may help to identify possible candidates for rescue reperfusion therapy. Complex RV geometry precludes determination of a single parameter that could reliably reflect the RV size and function. Dysfunction of RV evoked by acute PE has been evaluated with different echocardiographic techniques, and its criteria differed among studies [11].

The assessment of TAPSE was unsuccessful even in 25% of cases in previous studies, whereas SEATAK was achievable

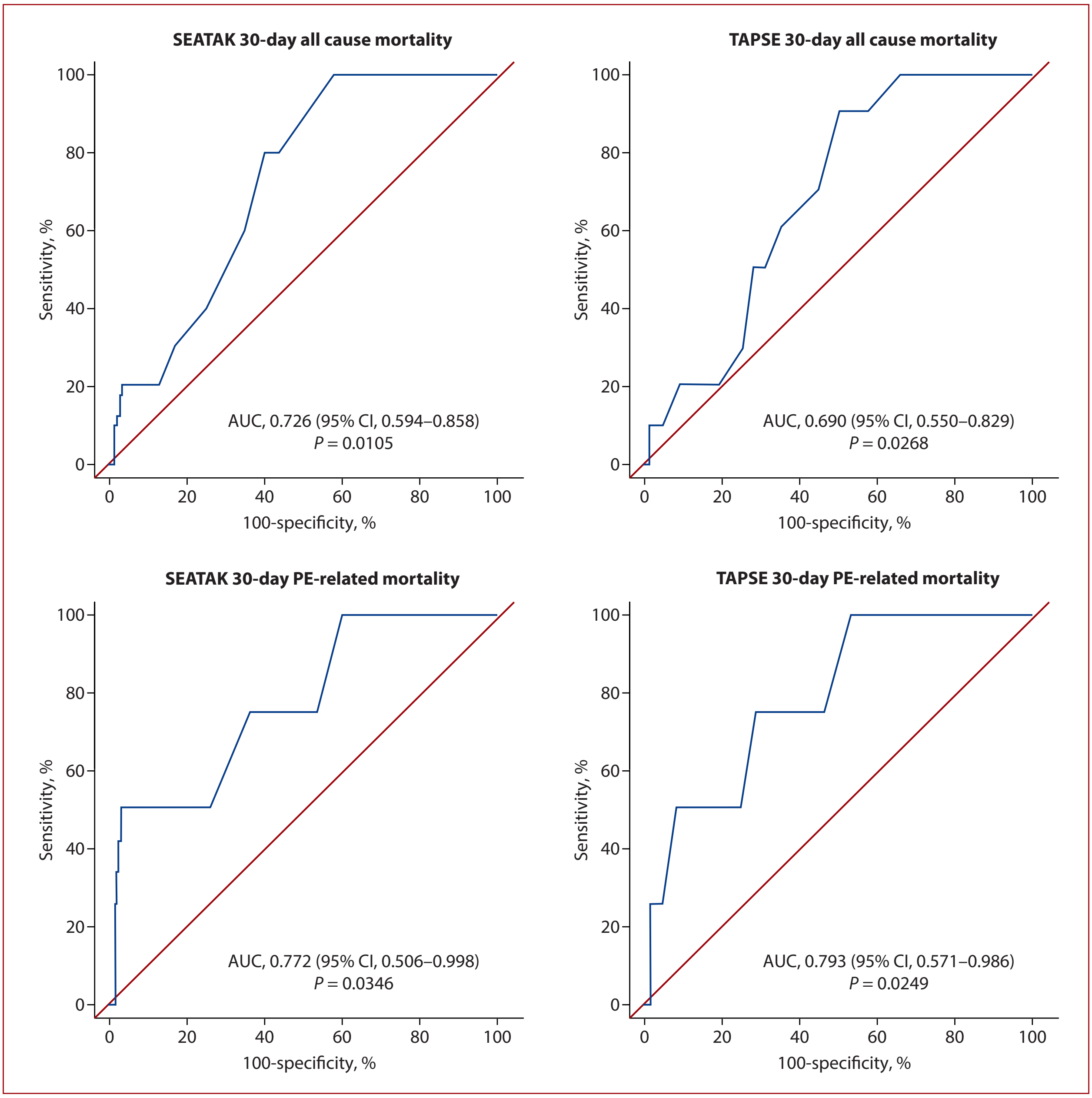

Figure 2. Receiver-operating characteristic (ROC) analysis of tricuspid annular kick (SEATAK) and tricuspid annular plane systolic excursion (TAPSE) in 30-day all-cause mortality and PE related mortality prediction in 82 patients with acute pulmonary embolism (all deaths n = 10; PE-related deaths n = 4)

Abbreviations: AUC, area under the curve; CI, confidence interval; PE, pulmonary embolism

in all subjects [9, 15–17]. In our group rates of failure to determine TAPSE and SEATAK were 4.9% and 2.8%, respectively. Importantly, only in 0.6% of study participants, these two parameters could not be calculated. Thus, SEATAK may be very valuable in RV function appraisal in patients in whom TAPSE is not possible. The estimation of tricuspid annular movement in the subcostal view is getting more attention. “Subcostal TAPSE” has been assessed with anatomical M-mode and B-mode in adult and pediatric populations and proved to be a feasible and accurate alternative to conventional TAPSE with adequate efficacy in the identification of RV dysfunction [18–20]. Nevertheless, it has never been investigated in PE diagnostics and prognosis.

SEATAK showed positive correlations with RV systolic TTE parameters: TAPSE, FAC, TSV TDI, and LVEF, which is in concordance with the results of the studies by Díaz-Gómez et al. on critically ill patients and the SEATAK validation article by Sadek et al. [9, 21] on consecutive subjects with different disorders referred to the echocardiography laboratory. Although TTE examinations were performed in groups of various clinical characteristics, in our analysis, as well as in previous publications, the correlations between SEATAK and TAPSE were all strong (r = 0.71; P <0.001; r = 0.86; P = 0.03 and r = 0.82; P <0.001, respectively). The values of SEATAK were smaller than TAPSE in all these studies with the overall mean difference of 1.9 mm, 1.5 mm, and 2.6 mm,

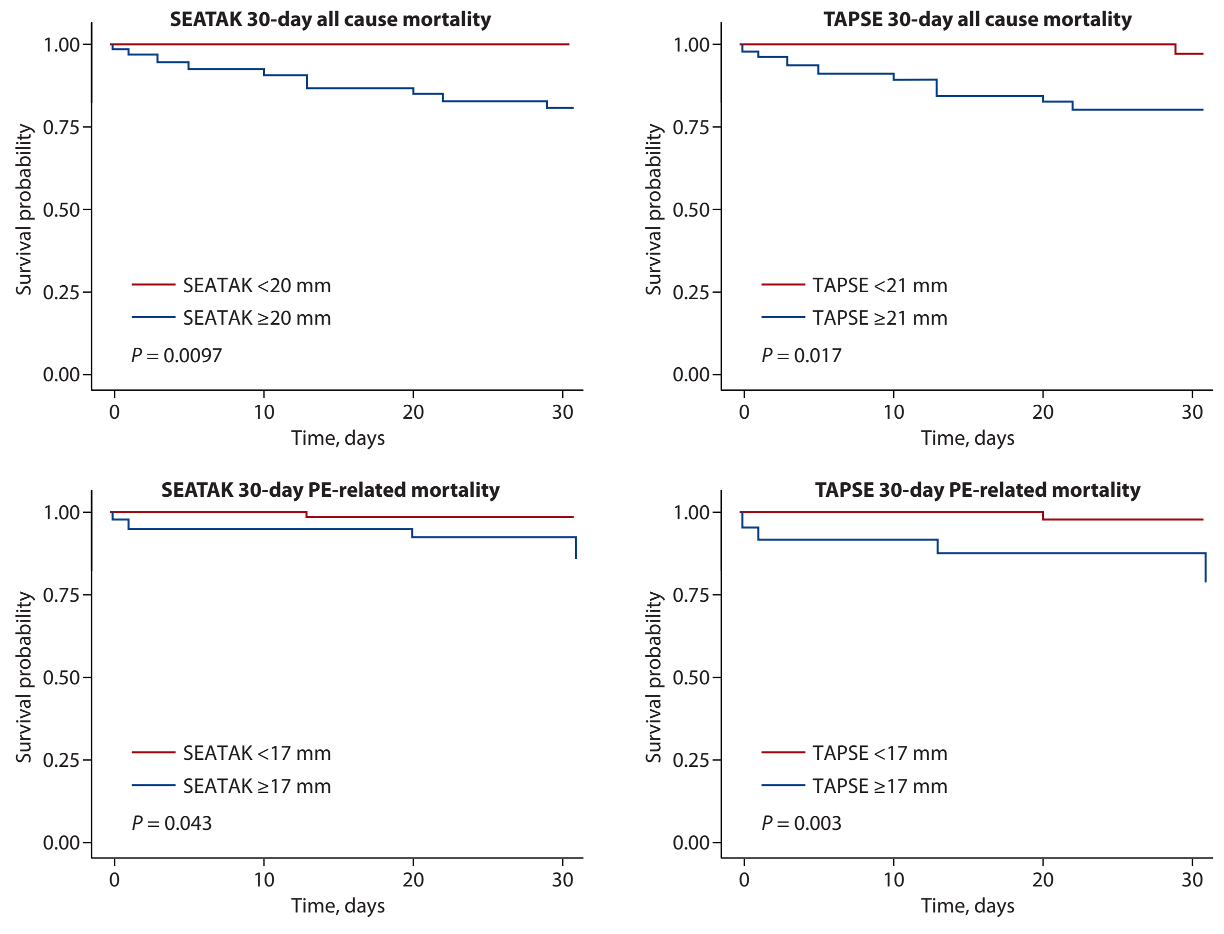

Figure 3. Kaplan-Meier analysis of tricuspid annular kick (SEATAK) and tricuspid annular plane systolic excursion (TAPSE) of 30-day survival in 82 patients with acute pulmonary embolism (PE). N of groups: SEATAK <20 mm 51 pts (62.2%), SEATAK ≥20 mm 31 pts (37.8%), SEATAK <17 mm 37 pts (45.12%), SEATAK ≥17 mm 45 pts (54.88%), TAPSE <21 mm 45 pts (54.88%), TAPSE ≥21 mm 37 pts (45.12%), TAPSE <17 mm 25 pts (30.49%), TAPSE ≥17 mm 57 (69.51%)

Abbreviations: see Tables 1–3

respectively [9, 21]. Notably, during TAPSE calculation in the 4-chamber apical view, in most cases, the M-mode is aligned almost perpendicularly to the tricuspid valve plane while in the subcostal long-axis view it is at an angle, which could explain decreased values of SEATAK. Moreover, in SEATAK calculation, a specific movement of the analyzed lateral part of the tricuspid annulus is observed; a slight rotation towards the left ventricle, which is accurately reflected in the parameter's name with the denotation "kick". This kick might be more dependent on performance of the basic part of the free wall of the RV, while TAPSE is more related to performance of other parts of the RV wall. Thus, in PE where there are, in some patients, regional motion abnormalities of the RV free wall, including subjects with the McConnell sign, the differences between SEATAK and TAPSE could be more pronounced.

Significant positive correlations between TAPSE and other RV systolic TTE parameters and LVEF were reported earlier, inter alia, in a group of 900 patients with different diseases [22]. In the analysis of available hemodynamic profiles, SEATAK correlated with cardiac output, cardiac index, and central venous pressure and showed an inverse relationship with the heart rate and pulmonary arterial occlusion pressure. Díaz-Gómez et al. [9], based on their results, assume that SEATAK might be affected more by preload, whereas TAPSE reflects the isolated intrinsic systolic function of the RV.

Neither in the aforementioned study by Sadek et al. [21] nor in ours, SEATAK correlated with the right ventricular index of myocardial performance, calculated in that article with tissue Doppler imaging and in our study with TDI and Pulsed Doppler of tricuspid inflow and right ventricular outflow tract flow. Importantly, the Tei index is a global estimate of both systolic and diastolic function of the RV, and this diastolic component is most likely the confounding factor [1, 21].

In our study, SEATAK, like TAPSE, showed no utility in PE diagnosis. Both parameters serve as indicators of RV systolic function but not of the presence of thrombi in pulmonary arteries. PE might not affect RV systolic performance or influence it to a different extent, just like other heart disorders, including left heart diseases [11].

Other echocardiographic parameters more specific for PE detection, related to the presence of obstacles in pulmonary arteries and RV pressure overload e.g. shortened Act, the 60/60 sign, increased RVTD to LVTD ratio, and the McConnell sign were different in our subgroups of participants with and without PE . Apart from SEATAK, shortened Act and the 60/60 sign were echocardiographic predictors of unfavorable prognosis in our analysis. Act <81 ms was associated with 30-day mortality in a prospective blinded study [23]. The 60/60 sign was shown to be a good predictor of in-hospital mortality in PE patients (odds ratio [OR], 6.13; 95% CI, 1.11–59.21; $P = 0.03$) [24]. In the analysis of the echocardiographic pattern of 511 consecutive patients with acute PE, the coexistence of the 60/60 sign with the McConnell sign and an enlarged hypokinetic RV was recognized as the most useful echocardiographic criterion for RV dysfunction [25]. Notably, RV dysfunction was superior to the PESI and Bova clinical scores in risk stratification in 571 individuals with acute PE [26].

In our analysis, TAPSE showed good prognostic value in the prediction of 30-day mortality. As reported previously by Pruszczyk and colleagues in a group of normotensive patients with acute PE, TAPSE was the only independent TTE outcome predictor from a broad array of echocardiographic parameters [27]. Similar findings come from the study by Lobo et al. [28]. Moreover, in the article by Kurnicka et al. [29], TAPSE was superior to TSV TDI in the prediction of 30-day adverse outcomes. Notably, assessment of RV function with tissue Doppler imaging correlated with pulmonary artery thromboembolic burden and was successfully utilized to monitor RV performance and filling pressure in PE [30, 31]. In another study, TAPSE was preferable to echocardiographic evaluation of the RV to the left ventricle ratio and the counterpart of this RV pressure overload marker in multidetector computed tomography in 30-day mortality prognosis [32]. The cut-off values for TAPSE considering PE-related outcome measures varied from ≤15 mm to <18 mm as abnormal and ≥18 mm to >20 mm as normal in different studies [2, 27, 28, 32–34]. Our results of TAPSE and SEATAK cut-offs are at a similar level.

A single-center setting with a relatively small number of patients, especially in the non-survivor group, should make our promising results be appraised with caution. Further studies on larger patient groups are advised.

Even though PE is an old topic, new significance is being attached to echocardiography as a useful tool in evaluating this condition and its complications. In the review by Pruszczyk and Konstantinides, elevated echocardiography imaging indexes are included in risk factors that may affect initially normotensive patients with PE and move them to the group of patients with intermediate-risk [35]. Another study aimed to assess usefulness of classic echocardiographic parameters indexed to height and body surface area for prediction of acute PE in patients with a high clinical probability of PE referred for computed tomography pulmonary angiography [36]. The authors of an expert opinion screening for patients with chronic thromboembolic pulmonary hypertension after acute PE claim that TTE is a preferred screening test for chronic thromboembolic pulmonary hypertension and should be performed in any patient with dyspnea of unclear cause after a history of acute PE and at least 3 months of optimal antithrombotic therapy [37].

Study limitations

The main limitation of the presented study is a small number of patients, especially within the non-survivor subgroup. Furthermore, echocardiograms were not repeated, and thus variability of echocardiographic parameters could not be assessed. The prognostic value of biomarkers with different recognized cut-off values was not investigated.

CONCLUSIONS

SEATAK is a promising practicable and useful echocardiographic parameter reflecting RV systolic function and might be an accurate alternative to TAPSE. Moreover, SEATAK could be an independent predictor of all-cause and PE-related 30-day mortality in patients with acute PE.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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