The Prognostic Importance of Right Ventricular Functional Echocardiographic Parameters of Community-Acquired Pneumonia Patients in Intensive Care

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SOUHRN

Cíl: Vztah mezi parametry transtorakální echokardiografie (TTE) a prognózou pacientů hospitalizovaných na jednotce intenzivní péče s diagnózou pneumonie není přesně znám. Cílem této studie bylo prozkoumat vztah mezi funkcemi pravé komory srdeční (PK) a klinickými parametry.
Materiály a metody: Do naší retrospektivní studie byli zařazováni pacienti s pneumonií a s normálními hodnotami ejekční frakce levé komory (EF LK), kteří byli sledováni na jednotce intenzivní péče. Následně jsme porovnávali skóre závažnosti stavu pacientů, výskyt komplikací a mortalitu během sledování s údaji získanými metodou TTE.
Výsledky: Do studie byly zařazeny údaje celkem 107 pacientů; z tohoto počtu se jich 77 (71,9 %) vyléčilo a byli propuštěni z nemocnice (skupina 1), zbývajících 30 (28,1 %) zemřelo (skupina 2). Věk (průměr ± SD) pacientů ve skupinách 1 a 2 byl 67,71 ± 9,65, resp. 76,67 ± 8,52 roku ($p < 0,001$). Žádný statisticky významný rozdíl mezi pohlavími nebyl nalezen (0,357). Hodnoty ukazující na sníženou funkci pravé komory a vyšší incidence komorbidit byly častěji pozorovány u pacientů s nepříznivým výsledkem léčby a s vyšší mortalitou ($p < 0,001$). Výsledkem analýzy zaznamenaných údajů pomocí binární logistické regrese pro modelování nezávislého vztahu mezi hodnotami naměřenými metodou TTE a mortalitou bylo zjištění, že frakční změna plochy pravé komory (right ventricular fractional area change, RVFAC), tlak v plicnici v systole (pulmonary artery systolic pressure, sPAP) a průměr dolní duté žíly (inferior vena cava, IVC) nejsou v přímo úměrném vztahu s mortalitou.
Závěr: Naše studie prokázala, že hodnoty naměřené metodou TTE se – ve srovnání s jinými pacienty – více lišily od hodnot pacientů s těžkým zápalem plic a s nepříznivou prognózou naměřených jinými metodami. Byl zaznamenán i nepříznivý vliv zvýšené zátěže komorbidit na hodnoty naměřené metodou TTE. Ukázalo se, že hodnoty RVFAC, sPAP a IVC, často používané k hodnocení funkcí pravé komory srdeční, mohou napomáhat při predikci mortality pacientů, léčených na jednotkách intenzivní péče se zápalem plic.

ABSTRACT

Purpose: The relationship between transthoracic echocardiography (TTE) parameters and the prognosis of patients hospitalized in intensive care with a diagnosis of pneumonia is not well known. This study planned to investigate the relationship between patients' right ventricular functions (RVF) and clinical outcomes.
Materials and methods: It is a retrospective study including patients with pneumonia and normal left ventricular ejection fraction (LVEF) who were followed up in the intensive care unit. The severity scores of the patients, complications, and mortality rates during follow-up and TTE data were compared.
Results: One hundred and seven patients were included in the study. Seventy-seven (71.9%) of these patients recovered and were discharged (group 1), and 30 (28.1%) died (group 2). The age distributions (mean **± SD) of** group 1 and group 2 patients were 67.71 ± 9.65 and 76.67 ± 8.52, respectively (p <0.001). It did not show statistical significance according to gender distribution (0.357). Parameters indicating decreased RV functions and the presence of increased comorbidities were observed more frequently in patients with adverse clinical outcomes and mortality (p <0.001). As a result of the binary logistic regression analysis performed to model the independent effects of TTE measurements on mortality, it was observed that right ventricular fractional area change (RVFAC), pulmonary artery systolic pressure (sPAP), and inferior vena cava (IVC) variables had independent effects on mortality.

Keywords:
Cardiac imaging
Mortality
Pneumonia
Right ventricular functions
Transthoracic echocardiography

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Conclusion: In our study, it was observed that TTE measurement results deviated more from normal measurement levels in patients with severe pneumonia and poor prognosis compared to other patients. Also the negative impact of increased comorbidity burden on TTE parameters was also observed. It has been determined that RVFAC, sPAP, and IVC measurements, frequently used in evaluating RVF, it is useful in predicting an increase in mortality rates, in patients followed in intensive care due to pneumonia.

Introduction

The mechanisms behind the development of systolic and diastolic right ventricular dysfunction (RVD) have become a matter of curiosity and are becoming better understood day by day. Although RVD is observed quite frequently in patients with heart failure with reduced ejection fraction (HFrEF), it is well known now that RVD is quite common in patients with heart failure with preserved ejection fraction (HFpEF) and contributes to poor prognosis.[1,2] Many diseases such as pulmonary hypertension (PH), atrial fibrillation (AF), chronic obstructive pulmonary disease (COPD), hypertension (HT), obesity, diabetes mellitus (DM), chronic kidney disease (CKD), sleep apnea, rheumatological diseases, and connective tissue diseases, left ventricle (LV) and right ventricle (RV) remodeling processes generally continue together.[1,3,4] The prevalence of RVD has been shown to be between 30–50% in studies using echocardiographic parameters such as right ventricular fractional area change (RVFAC), tricuspid annular plane systolic excursion (TAPSE), RV lateral tricuspid annulus peak systolic velocity (RVS'). Again, in a study using cardiac magnetic resonance (CMR) imaging, RVD was observed in 19% of patients, and a meta-analysis showed that the prevalence of RVD was between 18% and 28%.[5,6] Prevalence data for RVD vary because the severity of comorbidities and the echocardiographic methods, criteria, and thresholds used to evaluate RVD vary in different studies. These criteria have been updated over time.[1,7–9] Despite changing methods and criteria, current data have shown that RVD is more common in HFpEF patients. In some studies, it has been observed that it is the major determinant of clinical prognosis in pathologies accompanied by right ventricular dysfunction and failure.[1,10–12] In addition, it is observed that the clinical prognosis is affected poorly in patients with RVD in the intensive care unit and in cases accompanied by infection and sepsis.[13,14] Pneumonia is a systemic lung parenchymal infection and may present with mild clinical symptoms and signs of the respiratory system but may also lead to sepsis, multiple organ failure complications, and death. Deterioration of right ventricular functions, especially in patients with increased comorbidities, leads to an increase in the frequency of HFpEF in these patients. It has been observed that this situation increases the susceptibility to pneumonia and negatively affects the prognosis.[15] Although rare, data are available between pneumonia and echocardiographic parameters. In addition, since the symptoms and signs of congestive heart failure (CHF) can be masked in patients diagnosed with pneumonia, the diagnosis can often be missed. Although the incidence and prognosis of pneumonia are observed quite frequently in all types of CHF, their course is worse in patients with CHF and those with impaired right heart function.[15,16] This study investigated the effect of transthoracic echocardiography (TTE) parameters on the prognosis and mortality of patients with HFpEF who were followed up in the intensive care unit due to pneumonia.

Materials and methods

Patient selection

The study is a retrospective study including 107 patients who were admitted to the chest diseases and cardiology intensive care unit and followed up between September and November 2024. Patients were divided into two groups: those who recovered and were discharged (group 1) and those who died (group 2). TTE findings, radiological images, laboratory tests, and additional clinical data of the patients were accessed through the hospital automation system. Pregnant women, patients under 18 years of age, patients without intensive care admission, patients with LVEF below 50; advanced heart valve stenosis or insufficiency, restrictive, constructive, and hypertrophic cardiomyopathy, congenital heart disease, malignancy, liver cirrhosis, end-stage renal failure, advanced chronic obstructive pulmonary disease (COPD), idiopathic PH, drug-related PH, hereditary PH and patients who could not be reached TTE data were excluded from the study **(Fig. 1).** Data regarding the TTE images of the patients were obtained from bedside TTE records made on patients consulted to the cardiology clinic during intensive care follow-ups. The pneumonia severity of the patients during their admission to the intensive care unit was graded with pneumonia severity index (PSI) and confusion, blood urea nitrogen, respiratory rate, blood pressure, and age (CURB65) scores.[17]

Echocardiographic measurement and definitions

All patients underwent TTE examination imaging using the Philips EPIQ7 Cardiac Ultrasound System. The main data we use in our study, obtained from TTE records, are as follows: left ventricular ejection fraction (LVEF), left ventricular mass index (LVMi), maximum velocities of the E wave (E) and the A wave (A), mean septal e' velocity/ lateral e' velocity, tricuspid regurgitation velocity (TRV), left atrial volume index (LAVi), average E/e' ratio, right ventricle end-diastolic diameter (RVEDD), left ventricle end-diastolic diameter (LVEDD), inferior vena cava (IVC) diameter, pulmonary artery systolic pressure (sPAP), TAPSE, RV fractional area change (RVFAC), lateral RVS', TAPSE/sPAP ratio, RVEDD/LVEDD. The LVEF was measured using the standard Simpson biplane method in the 4- and 2-chamber views. RVFAC was expressed as the ratio obtained by dividing the difference between the RV end-diastolic area (RVEDA) and the RV end-systolic area (RVESA) by RVEDA. The endocardial contour was traced to calcu-

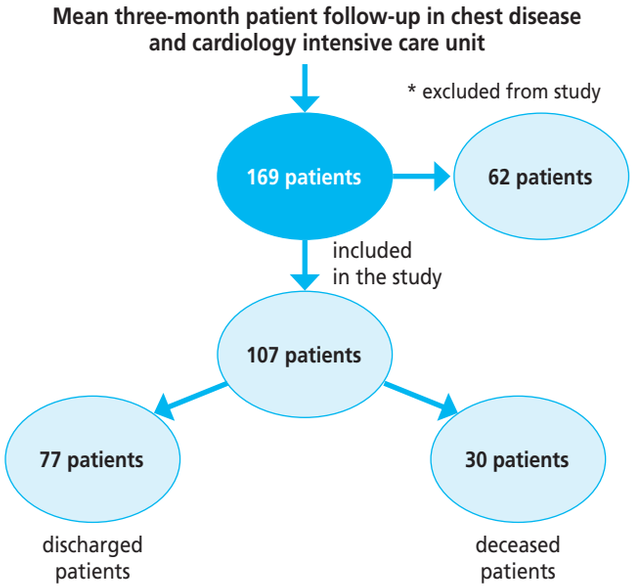

Fig. 1 – A flow chart for inclusion of patients in the our study.
* Pregnant women, patients with LVEF below 50; advanced heart valve stenosis or insufficiency, restrictive, constructive, and hypertrophic cardiomyopathy, congenital heart disease, malignancy, liver cirrhosis, end-stage renal failure, advanced chronic obstructive pulmonary disease (COPD), idiopathic PH, drug-related PH, hereditary PH and patients who could not be reached, TTE data were excluded from the study.

late the RVEDA, RVESA, and RV basal diameter. LV mass was calculated using recommended formula to estimate LV mass from LV linear dimensions and indexed to body surface area LVMi. LA volume was assessed using the modified biplane Simpson's method from apical 2- and 4-chamber views at the end-systole and was indexed to body surface area LAVi. Maximal tricuspid regurgitation velocity (TRV) was used to derive the RV-right atrial (RA) pressure gradient by the simplified Bernoulli equation with continuous-wave Doppler guidance (P = 4 (TRmax2, TRV ≤2.8 m/s velocity value is considered normal). The estimated pulmonary artery systolic pressure (sPAP) was calculated using the tricuspid regurgitation peak gradient (TRPG), IVC diameter, and its variation. According to the recommendations for cardiac chamber quantification by echocardiography in adults, we calculated the estimated right atrial pressure (ERAP) and divided it into 3 pressure grades. An IVC diameter <21 mm and collapses of >50% with a sniff-defined normal pressure of 3 mmHg, IVC diameter >21 mm, and collapses of <50% with a sniff-defined high pressure of 15 mmHg. The other cases were defined as an intermediate pressure of 8 mmHg. Finally, The sPAP was calculated as the sum total of the TRPG and ERAP. Peak mitral E velocity (cm/s) measurement is obtained by measuring diastolic flow velocities between the mitral valve tips in apical four-chamber imaging. E' velocity (cm/s) was obtained from the mitral ring's basal septal or basal lateral region with tissue Doppler. The mean E/e ratio <8 generally indicates normal filling pressures, while >14 is highly specific for increased LV filling pressures. As in the validation study, the sPAP value used for suspicion of PH was taken as 35 mmHg. Peak systolic velocity of the RVS' was measured using tissue Doppler imaging (TDI). A sample volume of pulsed TDI was placed in the tricuspid annulus or in the middle of the basal segment of the RV-free wall. The RVS' velocity was defined as the highest systolic velocity without overgaining the Doppler envelope. RVS' is usually assessed as a categorical variable with a 9.5 cm cut-off for RV dysfunction. An M-mode sampling was placed at the right lateral border of the heart at the tricuspid valve annulus, which generated M-mode active tracings. A TAPSE value was obtained by measuring the vertical height between the peak and trough in a single cardiac cycle to determine the apex-to-base shortening. Other useful indices include RVEDD (>41 mm) and RV hypertrophy (wall thickness >5 mm). TTE was applied to the patients according to the recommendations of the American Society of Echocardiography / European Association of Cardiovascular Imaging guidelines.[9]

Community-acquired pneumonia (CAP): definition and assessment of severity

Although there is no universal consensus on the definition of severe community-acquired pneumonia, currently accepted criteria are based on international clinical practice guidelines. CAP is a condition with diagnostic radiographic findings and no alternative explanation, with 2 or more signs (e.g., temperature >38 °C or ≤36 °C; leukocyte count <4000/μL or >10 000/μL) or symptoms (e.g., new onset or increased cough or shortness of breath). The definition and management of CAP was defined by the Infectious Diseases Society of America / American Thoracic Society.[18] In our study, PSI score and CURB-65 score were used to determine the pneumonia severity. PSI was created by Fine et al., and patients were classified into 5 groups according to demographic factors, comorbidities, physical examination, laboratory, and radiographic findings. As the PSI scores of patients increased, an increase in intensive care unit admission and mortality rates was observed. If we look at the points and mortality rates respectively; (0–50)/(0.1), (51–70)/(0.6), (71–90)/(0.9), (91–130)/(9.3), (131–395)/(27). It is known as the CURB-65 score (confusion, blood urea nitrogen > 7 mmol/L, respiratory rate ≥ 30/min, systolic blood pressure < 90 mmHg or diastolic blood pressure ≤60 mmHg, age ≥65 years). Accordingly, the mortality rate of 1 point was determined as <3%, the mortality rate of 2 points was determined as 9%, and the mortality rate of 3–5 points was determined as 15–40%.[17]

Evaluation of comorbid patients in terms of HFpEF

In our study, the H_2FPEF score, which predicts the possibility of HFpEF and includes many parameters, was also calculated. According to this score, obesity was defined as body mass index (BMI) >30 kg/m^2 (2 points). AF diagnosis was obtained from clinical history or documented electrocardiography (ECG) (3 points). HT was defined as a patient previously diagnosed with HT or using two or more antihypertensive drugs (1 point). sPAP value above 35 mmHg was taken as the limit value (1 point). Age is defined as age 60 and over (1 point). E/e' was evaluated as >9 (1 point). When the total score is 6 or above, the probability of having HFpEF is considered to be 90% or above.[19]

Statistical analysis

In this study, descriptive statistics (number, percentage, mean, standard deviation, median, minimum, and maximum) of the data are given. The assumption of normal distribution was checked with the Shapiro–Wilk test. In cases where the normality assumption was not met, the Whitney U test was applied to compare two independent groups. In testing the relationship between categorical variables, the Pearson Chi-Square test was applied when the sample size assumption (expected value >5) was met, and Fisher's exact test was applied when the sample size assumption was not met. Binary logistic regression analysis with a forward conditional approach was applied to model the categorical dependent variable with independent variables. Analyzes were carried out in the IBM SPSS 25 program.

Results

In our study, 107 patients were followed in the intensive care unit according to the inclusion criteria for 3 months. Seventy-seven (71.9%) of these patients recovered and were discharged, and 30 (28.1%) died. The age distributions (mean ± SD) of the group 1 and group 2 patients were 67.71 ± 9.65 and 76.67 ± 8.52, respectively (<0.001). The rate of female patients was found to be 36 (46.8%) and 17 (56.7%) in both groups and did not show statistical significance (0.357). As a result of the analyses made according to the demographic and clinical characteristics of both patient groups, comorbidities such as HT, DM, coronary artery disease (CAD), ischemic cerebrovascular event (CVE), AF, CKD, COPD were statistically significant in the group 2 (p <0.05). A comparison of demographic and clinical characteristics is given in **Table 1.**

Table 1 – A comparison of demographic and clinical characteristics

	Clinical outcome in intensive care		*p*
	Group 1 (n = 77)	Group 2 (n = 30)	
Age/year ± mean/SD	67.71 ± 9.65	76.67 ± 8.52	<0.001*
Female, n (%)	36 (46.8)	17 (56.7)	0.357
HT, n (%)	20 (26)	18 (60)	0.001*
DM, n (%)	14 (18.2)	13 (43.3)	0.003*
CAD, n (%)	11 (14.3)	12 (40)	0.004*
CVE, n (%)	2 (2.6)	11 (36.7)	<0.001*
AF, n (%)	29 (37.7)	19 (63.3)	0.016*
COPD, n (%)	13 (16.9)	12 (40)	0.003
CRF, n (%)	9 (11.7)	11 (36.7)	0.003*

AF – atrial fibrillation; CAD – coronary artery disease; COPD – chronic obstructive pulmonary disease; CRF – chronic renal failure; CVE – cerebrovascular event; DM – diabetes mellitus; HT – hypertension. * p <0.05.

Table 2 – Laboratory data, and pneumonia severity scores of patients discharged from intensive care and mortality groups

	Clinical outcome in intensive care				*p*
	Group 1 (77)		Group 2 (30)		
	Min.–max.	Mean ± SD (median)	Min.–max.	Mean ± SD (median)	
BMI	16.4–37.8	27.24 ± 5.17 (24.52)	20.82–40.1	29.25 ± 5.03 (27.1)	0.041*
H_2FPEF	0–9	4.4 ± 2.87 (4)	0–9	5.93 ± 2.56 (6)	0.017*
CURB-65	2–5	3.16 ± 0.67 (3)	2–5	4.23 ± 0.77 (4)	<0.001*
PSI	101–197	140.12 ± 17.71 (139)	121–242	165.03 ± 30.49 (154.5)	<0.001*
WBC	853–21365	10111.44 ± 5330.15 (11962)	830–26710	12629.77 ± 8486.4 (12740)	0.119
Hb	0.56–18.2	12.1 ± 2.52 (12.5)	6.8–17.4	11.66 ± 2.7 (11.2)	0.170
Procalcitonin	0.02–7.26	1 ± 0.98 (0.97)	0.1–8.05	3.08 ± 2.44 (2.13)	<0.001*
BNP	12–15561	433.75 ± 1764.4 (121)	3–16589	1351.27 ± 3193.52 (390)	0.008*
D-dimer	23–21417	1428.88 ± 2667.85 (821)	79–25123	3591.23 ± 6185.54 (1489)	0.002*
TnI	0–25000	413.43 ± 2861.87 (12)	0–25000	1284.33 ± 4540.06 (184.5)	<0.001*
Hospitalisation duration	3–34	12.4 ± 7.71 (11)	3–47	11.33 ± 10.42 (9)	0.221

BMI – body mass index; BNP – brain natriuretic peptide; CURB65 – confusion, blood urea nitrogen, respiratory rate, blood pressure, age; Hb – hemoglobin; PSI – Pneumonia severity index; TnI – troponin I; WBC – white blood count. * p <0.05.

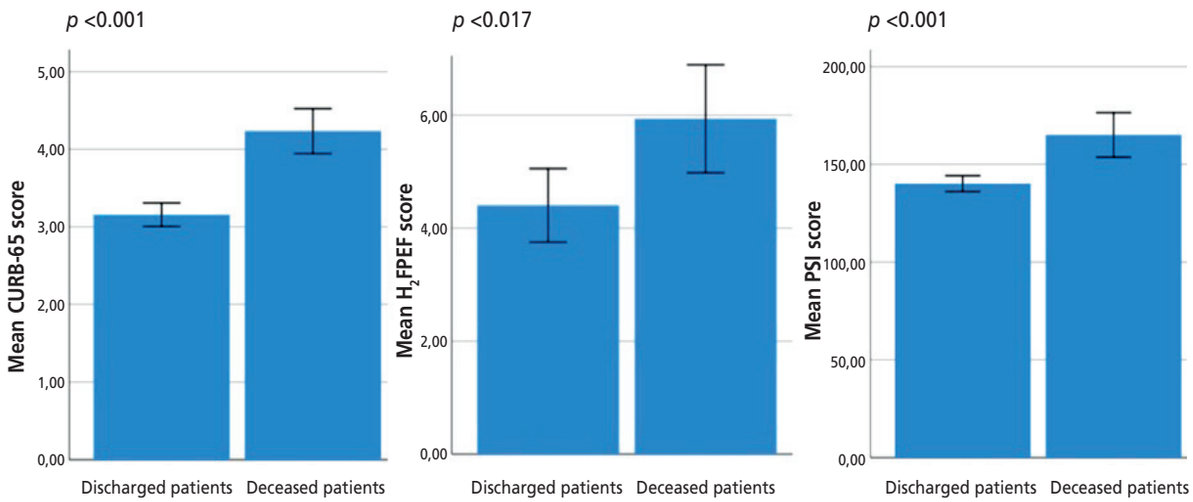

Fig. 2 – The relationship between the patients' HFpEF and pneumonia severity scores averages and their mortality status

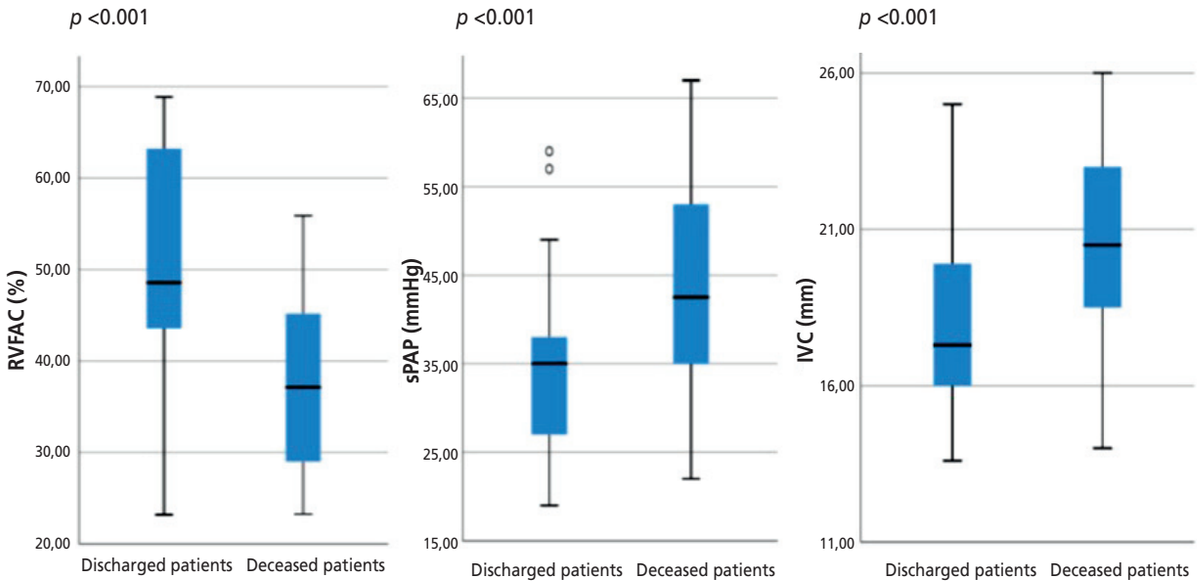

Fig. 3 – Analysis graphs of the distribution of RVFAC, sPAP, and IVC measurements in TTE according to the clinical outcomes.

Table 3 – Comparison of clinical outcomes of patients in intensive care, major complications, and supportive treatments received

	Clinical outcome in intensive care		
	Group 1 (n = 77)	Group 2 (n = 30)	*p*
Peff	9 (11.7)	9 (30)	0.023*
P. embolism	10 (13)	10 (33.3)	0.015*
Pleural EFF	13 (16.9)	11 (36.7)	0.028*
Sepsis	9 (11.7)	10 (33.3)	0.008*
Vasopressor support	10 (13)	16 (53.3)	<0.001*
Steroid use	17 (22.1)	16 (53.3)	0.002*
Mechanical ventilation	43 (55.8)	28 (93.3)	<0.001*

Peff – pericardial effusion; p. embolism – pulmonary embolism; pleural EFF – pleural effusion. * *p* <0.05.

Table 4 – TTE measurements and comparative statistical analysis

	Clinical outcome in intensive care				*p*
	Group 1		Group 2		
	Min.–max	Mean ± SD	Min.–max	Mean ± SD	
LVEF	50–66	56.86 ± 4.02	50–67	55.07 ± 4.31	0.030*
E: cm/s	57–141	93.39 ± 14.67	64–138	93.27 ± 20.23	0.781
Mean e': cm/s	3.6–13.9	9.15 ± 2.56	2.3–13.6	6.83 ± 2.82	<0.001*
A: cm/s	50–114	73 ± 16.07	1.04–114	73.34 ± 28.69	0.779
E/A/ratio	0.76–1.91	1.18 ± 3.8	0.69–2.11	1.21 ± 4.12	0.054
LVMi/ml/bsa	72–119	89.05 ± 10.35	75–122	94.27 ± 14.19	0.117
LAVi/ml/bsa	18–48.1	31.15 ± 6.59	24–49	38.51 ± 7.17	<0.001*
TAPSE/SBAP/ratio	0.17–0.89	0.55 ± 0.21	0.16–0.84	0.38 ± 0.18	<0.001*
RVFAC/ratio	23.19–68.87	50.5 ± 12.68	23.25–55.9	37.68 ± 10.28	<0.001*
E/e' ratio	4.2–17.6	9.73 ± 4.31	5.8–19.5	13.75 ± 4.55	<0.001*
TRV/cm/s	1.4–4	2.62 ± 0.71	1.31–4.3	3.1 ± 0.97	0.002*
TAPSE/cm/s	11.5–28.8	20.15 ± 4.59	12.1–27.1	17.28 ± 4.4	0.001*
sPAB/mmHg	19–59	33.69 ± 8.52	22–67	43.27 ± 12.38	<0.001*
RVS'/cm/s	8.1–23.4	14 ± 6.1	6.2–20.3	11 ± 5.7	<0.009
RV wall thickness/mm	2.7–1.12	5.71 ± 2.12	2.6–1.23	5.87 ± 2.44	0.102
RVEDD/mm	31–47	39 ± 6.8	32–52	42 ± 7.1	0.176
RVEDD/LVEDD ratio	0.51–1.22	0.72 ± 0.19	0.43–1.41	0.82 ± 0.24	0.012*
IVC/mm	13.6–25	17.99 ± 2.67	14–26	20.47 ± 2.98	<0.001*
LVDD (any stage), n (%)	33 (42.9)		22 (73)		0.021*
LV D-shaped, n (%)	10 (13)		10 (33.3)		0.015*

A – A wave; E – E wave; E/A – E wave / Awave ratio; E/e'; E wave/e' ratio; IVC – inferior vena cava; LAVi – left atrial volume index; LVDD – left ventricular diastolic dysfunction; LV D-shaped – left ventricular D-shaped; LVEF – left ventricular ejection fraction; LVMi – left ventricular mass index; mean e'– e' wave; RVEDD – right ventricular end-diastolic diameter; RVFAC – RV fractional area change; RVS' – RV lateral tricuspid annulus peak systolic velocity; RV wall thickness – right ventricular wall thickness; sPAP – pulmonary artery systolic pressure; TAPSE – tricuspid annular plane systolic excursion; TAPSE/sPAP – tricuspid annular plane systolic excursion / pulmonary artery systolic pressure ratio; TRV – tricuspid regurgitation (TR) velocity. * p <0.05.

As a result of the analysis, it is seen that scores expressing the burden of the disease and accompanying comorbidities, such as body mass index (BMI), H_2FPEF, CURB-65 and PSI scores are observed more frequently in the group 2 compared to the group 1 (p <0.05). Similarly, serum procalcitonin, brain natriuretic peptide (BNP), D-dimer, and troponin I (TnI) measurement values were higher in the group 2 compared to the group 1, and statistically significant differences were detected (p <0.05) (**Table 2**). A graphical representation of the distribution of severity scores of these patients according to their clinical outcome is given in **Figure 2.**

Peff**,** P. embolism, Pleural EFF, and sepsis were evaluated as the main clinical complications during the follow-up of the patients, and in the comparison, they were found to be statistically more frequent and significant in the group 2. Again, these patients received more vasopressor support, steroid use, and mechanical ventilation support, and it was seen more in the group2 and was determined to be statistically significant (p <0.05) (**Table 3**)**.**

In our study, measurements of TTE parameters taken at the bedside of the patients were compared. In group2 patients, LAVi, E/e', TRV, SPAP, RVEDD/LVEDD, IVC, RV wall thickness, and RVEDD measurements were higher than in group1. In group 1 patients, EF, e', TAPSE/SBAP, RVFAC, RVS', and TAPSE measurements were higher than in group 2. As a result of comparing the data of both groups, LVEF, e', LAVi, TAPSE/sPAP, RVFAC, E/e', TRV, TAPSE, SPAP, RVEDD/LVEDD, RVS', IVC, left ventricular diastolic dysfunction (LVDD), and the rate of LV D-shaped were statistically significant differences between groups (p <0.05). Comparison data of TTE measurements of both groups are given in **Table 4.**

Binary logistic regression analysis was applied to model the independent effects of all TTE measurements obtained in the study on mortality status. The regression analysis determined that RVFAC, sPAP, and IVC variables had statistically significant effects on mortality (**Table 5**, **Fig. 3**).

Discussion

In our study, it was observed that patients whose RV functions were evaluated as poor had a worse clinical progno-

Table 5 – Binary logistic regression analysis for TTE variables on mortality

			95% confidence interval		
Variables	**SD**	***p***	**OR**	**LL**	**UL**
RVFAC ratio	0.024	0.007*	0.937	0.894	0.982
sPAB (mmHg)	0.029	0.005*	1.085	1.025	1.150
IVC (mm)	0.092	0.002*	1.323	1.106	1.583

Hosmer – Lemeshow; $X^2 = 5.280$ ve $p = 0.727$, Cox and Snell $R^2 = 0.318$ ve Nagelkerke $R^2 = 0.458$. IVC – inferior vena cava; LL – lower limit; OR – odds ratio; RVFAC – RV fractional area change; SD – standard deviation; sPAP – pulmonary artery systolic pressure; UL – upper limit. * $p < 0.05$.

sis and higher mortality rates compared to other patients. In addition, it has been determined that some parameters are useful in predicting an increase in mortality rates in these patients. In our study, patients were divided into two groups according to died or recovery and discharge during intensive care follow-up. In patients who died due to pneumonia during the follow-up period, the prevalence of existing comorbidities and the average age of the patients were observed to be higher and showed statistical significance (p <0.001). H_2FPEF, PSI, and CURB65 scores in patients admitted to intensive care are scores created by the severity of the disease and the burden of accompanying comorbidities and are widely used in the literature to predict clinical prognosis.[17] In our study, the existing scores were observed more frequently in patients with mortality and showed statistical significance (p <0.001). Again, if we look at the laboratory findings of these patients, parameters such as BNP, D-dimer, Tn-I, and procalcitonin, which are correlated with both the severity of the infection and the severity of systemic involvement or cardiac involvement, were observed more frequently in the group 2, and statistical significance was observed (<0.001). Secondary cardiac influence is commonly observed due to increased oxygen demand or inadequate supply in pathologies such as pneumonia, infection, and sepsis. In the group2 during the follow-up of the patients, clinical complications due to pneumonia and intensive care stay or increased severity of the disease (such as Peff, P. embolism, Pleural EFF, sepsis, need for steroids and mechanical ventilation) were observed more frequently (p <001). The findings of our study were quite compatible with the literature and the severity of the disease.[17] Major parameters such as E/e', e,' LAVi, TRV, and LVMi, which have a place in the non-invasive estimation of LV filling pressures, showed statistical significance in the group2 in proportion to disease severity and comorbidity burden (<0.001). The average of the H_2FPEF score (if the score is above 6–9, there is a 90% probability of diagnosis of HFpEF), which is frequently used to evaluate the possibility of HFpEF in the patients in our study, was 5.93 ± 2.56 in the group2 as 4.4 ± 2.87 in the group 1 (p<0.05). Again, the rate of any degree of LVDD in group 2 during follow-up was 22 (73%) and 33 (42.9%) in the other group (p = 0.021). In the post hoc analysis of the PARADIGM-HF (mortality and hospitalization rates of ARNI (sacubitril neprilysin inhibitor and valsartan) compared to enapril in HFrEF and PARAGON-HF (in HFpEF) studies, the frequency of pneumonia was high in patients with CHF, and the incidence of pneumonia, especially in patients with HFpEF, was observed to be approximately 3 times higher than the expected rate.[15] In this study, patients with HFpEF who developed pneumonia were older and had more comorbidities, and pneumonia was associated with significantly higher adverse fatal and nonfatal outcomes. When we look at the echocardiographic parameters in our study; RV dilatation develops due to increasing volume or pressure load in the RV. It was shown that RV dilatation is more common than RV systolic dysfunction and that patients with RV dilatation are more likely to have respiratory failure syndrome and the need for invasive mechanical use.[20] RV is highly sensitive to changes in afterload. This increased afterload load is highly associated with comorbidities.[21] In our study, the average RVEDD was observed to be over 41 mm in the group 2, and the RVEDD/LVEDD ratio was observed to be higher in the group 2 compared to group 1, and it was found to be statistically significant (0.012). Using another parameter, the TAPSE/sPAP ratio, the echocardiographic threshold level of RV-pulmonary artery (PA) coupling was defined. A decreased TAPSE/PASP ratio has been associated with poor prognosis in both HFrEF and HFpEF, and the prognostic cut-off of this ratio has been determined to be <0.36.[22] The reduced TAPSE/sPAP ratio has even been considered a reliable parameter for identifying HFpEF patients likely to have precapillary PH.[23] Again, as seen in our study, the TAPSE/sPAP ratio was lower and statistically significant in the group 2 (<0.001). The most commonly used parameters in the evaluation of RVD are TAPSE, RVFAC, and RVS'. Frequently, these measurements are used together. TAPSE is used quite frequently in HFpEF patients and has been observed to have an independent prognostic value.[1] The currently recommended lower limit for TAPSE is <17 mm, whereas <16 mm has often been used in previous studies.[9] TAPSE has also been shown to be a good predictor of mortality in intubated patients and correlates with D-dimer and increased cardiac Tns.[24] In our study, the mean TAPSE was observed less in the group 2 and showed statistical significance (0.001). It also showed an inversely proportional course with complications and the need for non-invasive mechanical ventilation (p <0.001). Another parameter, RVS', is usually assessed as a categorical variable with a 9.5 cm cut-off for RV dysfunction.[24] In our study, RVS' measurements showed lower values in the group 2, such as TAPSE, and showed statistical significance (p <0.001). RVFAC ra-

tio (lower limit of normal <35%) was also observed as a predictor of all-cause mortality and heart failure hospitalizations in HFpEF.[21] Additionally, based on the 35% lower limit for RVD, D dimer was found to correlate with other abnormal laboratory values and RVFAC predicted mortality.[25] Naturally, in our study, the averages of RVFAC measurement values were significantly lower in the group 2 compared to the group 1. In addition, binary logistic regression analysis was applied to model the independent effects of all TTE measurements obtained in our study on mortality status. As a result of the regression analysis, it was observed that RVFAC was a significant predictor of mortality. A similar effect could not be obtained for TAPSE and RVS'. As a matter of fact, TAPSE and RVS' may be higher than the expected normal value in the case of hyperdynamic LV. This situation is also considered as the stretching effect of the LV on some myocardial muscle fibers that are used in common with the RV.[26] The fact that the patients in our study had normal or above LVEF may support this situation. Similarly, a study evaluating RVD (TAPSE <17 mm and/or RVFAC <35%) showed that RVFAC-based dysfunction was more common than TAPSE-based dysfunction.[27] Another explanation may be a decrease in radial RV systolic function but relative preservation of longitudinal shortening.[28] Accordingly, TAPSE and RVS are thought to provide a compensatory response in cases where radial dysfunction develops. Moreover, since the LV influences the longitudinal function of the RV-free wall, its function may be considered enhanced or preserved due to its attachment to a functioning LV myocardium. Again, in some studies, it has been observed that RVD does not have predictive power for mortality according to some parameters evaluated (according to TAPSE and RVS).[20] As a matter of fact, another study found that RV dysfunction (assessed by TAPSE and RVFAC) was associated with all-cause mortality in patient groups with a high rate of need for mechanical ventilation.[29] Therefore, LV structural and functional functions may need to be considered when evaluating RVD. Another important parameter is PH. In patients with pneumonia, the development of PH is likely multifactorial, and important causes include hypoxic respiratory failure and ARDS, increased pulmonary vascular resistance, and the presence of microthrombus or pulmonary embolism in the lungs.[30] In our study, existing comorbidities and sPAP values were higher and statistically significant in the group 2 patients (p <0.001). In addition to TRV, IVC diameter also significantly contributes to the estimated sPAP measurement in TTE in patients. It indirectly affects sPAP. In addition, according to the binary logistic regression analysis, it was observed that both sPAP and IVC values had effect rates on the mortality of the patients in our study. In conclusion, it was observed that the RV functions of patients with severe pneumonia were worse. However, we do not currently have enough data to evaluate the effect of CAP on RV functions. For this, prospective large-scale studies are needed. Summary of our work: It can be said that in patients with a high probability of HfpEF or pneumonia patients with increased comorbidities, the clinical prognosis worsens, RVF parameters in TTE worsen, and mortality rates increase.

Conclusions

Our study observed that as the comorbidities of the patients increased, their clinical prognosis worsened, and TTE parameters remained at values outside the normal range. Like CURB-65 and PSI scores, which are used to evaluate the severity and prognosis of pneumonia, patients'comorbidities and TTE findings have also been shown to seriously affect prognosis. Additionally, an increased negative effect of increased comorbidity burden on TTE parameters was observed. It has been observed that RVFAC, sPAP, and IVC measurements, frequently used in the evaluation of RDV, are important predictors of mortality rates in patients followed in intensive care due to pneumonia.

Limitation

Our study is a single-center and retrospective study. Since TTE data were obtained in the intensive care environment, attention was paid to sterilization rules. For this reason, detailed and time-consuming measurements were avoided for patient safety. Data from RV strain measurements, 3D TTE, and CMRI imaging could not be compared.

Conflict of interest:
The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as potential conflict of interest.

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Ethical statement
The study was conducted in accordance with the Declaration of Helsinki and approved by the Atatürk University non-invasive clinical research ethics committee (Approval date 27/12/2024, Approval Code:B.30.2.ATA.0.01.00/43).

Informed consent
Informed consent was obtained from all subjects involved in the study.

Data availability statement
The data that support the findings of this study are available from the corresponding author upon a reasonable request.

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