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RESEARCH

Assessing the TAPSE/sPAP ratio for prognostic insight in HFpEF patients

KEF-KY hastalarında prognostik bir gösterge olarak TAPSE/sPAP oranının değerlendirilmesi

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Abstract

Purpose: Heart failure with preserved ejection fraction (HFpEF) is closely associated with right heart dysfunction. Our study aimed to elucidate the association between HFpEF and the TAPSE/sPAP ratio.

Materials and Methods: This retrospective observational study was conducted at a tertiary care cardiology clinic. A total of 64 patients diagnosed with HFpEF based on clinical criteria and an H2FPEF score ≥5 were included, along with 62 age- and sex-matched controls without heart failure symptoms. Demographic characteristics, laboratory markers, including NT-proBNP, and echocardiographic measurements were recorded. Particular attention was given to assessing right heart function using the TAPSE/sPAP ratio.

Results: The TAPSE/sPAP ratio was lower in patients with HFpEF than in controls (median 0.76 vs. 0.96). Patients with HFpEF also had higher NT-proBNP levels (median 2136.5 pg/mL vs. 136.5 pg/mL) and higher H2FPEF scores. A TAPSE/sPAP cut-off value of 0.843 was identified, which showed 70.3% sensitivity and 72.6% specificity in distinguishing HFpEF. Correlation analysis revealed that lower TAPSE/sPAP ratios were associated with higher NT-proBNP levels, elevated H2FPEF scores, and increased left atrial volume index.

Conclusion: The evaluation of the TAPSE/sPAP ratio in patients suspected of having HFpEF may be useful. Notably, TAPSE/sPAP predicted HFpEF regardless of EF in the multivariate analysis. In addition, their significant relationship with H2FPEF score and NT-proBNP levels strengthened their prognostic value.

Keywords: Echocardiography, HFpEF, TAPSE, sPAP, right heart dysfunction

Öz

Amaç: Korumalı ejeksiyon fraksiyonlu kalp yetersizliği (KEF-KY), sağ kalp disfonksiyonu ile yakından ilişkilidir. Bu çalışmada, KEF-KY hastaları ile TAPSE/sPAP oranı arasındaki ilişkinin ortaya konması amaçlanmıştır.

Gereç ve Yöntem: Bu çalışma, üçüncü basamak bir kardiyoloji kliniğinde yürütülmüş retrospektif ve gözlemsel bir araştırmadır. Klinik kriterlere ve H2FPEF skoru ≥5 olan toplam 64 HFpEF hastası ile kalp yetersizliği semptomları olmayan, yaş ve cinsiyet açısından eşleştirilmiş 62 sağlıklı kontrol bireyi çalışmaya dahil edilmiştir. Katılımcıların demografik özellikleri, NT-proBNP gibi laboratuvar belirteçleri ve ekokardiyografik ölçümleri kaydedilmiştir. Özellikle TAPSE/sPAP oranı kullanılarak sağ kalp fonksiyonları değerlendirilmiştir.

Bulgular: TAPSE/sPAP oranı, KEF-KY hastalarında kontrol grubuna göre daha düşük bulunmuştur (medyan 0.76'ya karşı 0.96). Ayrıca KEF-KY grubunda NT-proBNP düzeyleri (medyan 2136.5 pg/mL'ye karşı 136.5 pg/mL) ve H2FPEF skorları daha yüksek saptanmıştır. TAPSE/sPAP oranı için belirlenen 0.843 kesim değeri, KEF-KY'yi ayırt etmede %70.3 duyarlılık ve %72.6 özgüllük göstermiştir. Korelasyon analizlerinde düşük TAPSE/sPAP oranları ile yüksek NT-proBNP düzeyleri, artmış H2FPEF skorları ve yükselmiş sol atriyum hacim indeksi arasında anlamlı ilişkiler gözlemlenmiştir.

Sonuç: KEF-KY şüphesi olan hastalarda TAPSE/sPAP oranının değerlendirilmesi yol gösterici olabilir. Özellikle bu oran, ejeksiyon fraksiyonundan bağımsız olarak KEF-KY'yi öngörebilmekte ve H2FPEF skoru ile NT-proBNP ile olan anlamlı ilişkisi, prognostik değerini güçlendirmektedir.

Anahtar kelimeler: Ekokardiyografi, KEF-KY, TAPSE, sPAP, sağ kalp disfonksiyonu

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INTRODUCTION

Heart failure with preserved ejection fraction (HFpEF) is characterized by a left ventricle ejection fraction greater than 50% and elevated filling pressure[1]. It constitutes approximately half of all heart failure (HF) cases globally and is particularly prevalent among elderly individuals[2]. The fundamental pathophysiological mechanisms underlying HFpEF involve increased ventricular stiffness, alterations in myocardial viscoelastic properties, and diastolic dysfunction[3]. Many patients with HFpEF also have right ventricular dysfunction and pulmonary hypertension (PH), which are important to distinguish from other causes of heart failure and pulmonary hypertension[4-5].

Among non-invasive diagnostic tools, the H2FPEF score has emerged as a practical clinical algorithm by combining demographic and echocardiographic variables to estimate the likelihood of HFpEF. This score includes factors such as body mass index, age, atrial fibrillation, and elevated pulmonary artery systolic pressure[6,7].

The TAPSE/sPAP ratio is a non-invasive estimate of right ventrcular-pulmonary arterial pairing. This ratio holds promise for assessing cardiac function by examining the relationship between right ventricular systolic pressure (sPAP) and pressure alterations within the right ventricular ejection tract (TAPSE). Studies have consistently demonstrated that a lower TAPSE/sPAP ratio is associated with worse outcomes, including increased in-hospital major adverse cardiovascular events (MACEs) and higher mortality rates[8,9]. For instance, a TAPSE/sPAP ratio of less than 0.40 mm/mmHg has been independently linked to a higher incidence of in-hospital MACEs, such as all-cause death, resuscitated cardiac arrest, or cardiogenic shock, even after adjusting for comorbidities and clinical severity[10,11]. This threshold has shown incremental prognostic value over traditional risk factors, suggesting its utility in clinical decision-making and risk stratification for patients with AHF. Furthermore, the TAPSE/sPAP ratio has been validated as a predictor of hospitalization and mortality in both acute and chronic heart failure settings. In asymptomatic patients with heart failure, a decrease in the TAPSE/sPAP ratio was associated with a 9% increase in the relative risk of hospitalization, with no adverse events observed above a ratio of 0.47 mm/mmHg. Similarly, in patients with heart failure with reduced ejection fraction (HFrEF) and secondary pulmonary hypertension, the TAPSE/sPAP ratio was a reliable surrogate for right ventricular-pulmonary artery coupling, correlating with parameters of pulmonary vascular load and right ventricular function[12]. The prognostic significance of the TAPSE/sPAP ratio extends to patients with mildly reduced ejection fraction (HFmrEF), where a ratio of 0.46 or lower was associated with higher all-cause mortality and a tendency for increased heart failure re-hospitalization. Overall, the TAPSE/sPAP ratio serves as a valuable non-invasive marker for assessing right ventricular function and predicting adverse outcomes in various heart failure populations, underscoring its potential role in guiding therapeutic strategies and improving patient management in heart failure.

Based on these findings, we evaluated TAPSE/sPAP, a combination of two simple measurements that reflects right heart function in patients with HFpEF diagnosed using the H2FPEF score. This study aimed to evaluate the prognostic utility of the TAPSE/sPAP ratio in patients with HFpEF, particularly its association with clinical and echocardiographic markers such as NT-proBNP and H2FPEF score. We hypothesized that a lower TAPSE/sPAP ratio is independently associated with the presence of HFpEF and may serve as a complementary diagnostic and prognostic marker in routine clinical practice.

MATERIALS AND METHODS

The Institutional Research Ethics Board approved this study following the guidelines of the Helsinki Declaration. The approval was dated 14.12.2023 and identified by the number 94578. All participants provided written consent before participating in the study. Artificial intelligence-supported technologies were not used in this study.

This study was conducted at the Siirt Training and Research Hospital, a tertiary care facility with a dedicated cardiology department and an echocardiography laboratory. The institution maintains an electronic medical record system that ensures secure and traceable data management of the data. All patient files, laboratory results, and echocardiographic measurements were archived systematically, allowing reliable retrospective access

and minimizing the risk of data loss or entry errors. All echocardiographic examinations were performed using high-resolution ultrasound devices (Philips EPIQ) in accordance with the guidelines of the American Society of Echocardiography. Inter-observer consistency was ensured by cross-validation of critical measurements, such as TAPSE and sPAP.

Sample and study design

In this retrospective investigation, a cohort of 64 patients with HFpEF who were previously treated in our clinic, along with 62 controls without signs or symptoms of heart failure, were included. The diagnosis of HFpEF was made according to the current European Society of Cardiology (ESC) and American Heart Association/American College of Cardiology/Heart Failure Society of America (AHA/ACC/HFSA) heart failure guidelines. The H2FPEF score was included in the study in patients of 5 and above. An H2FPEF score of ≥ 5 was selected based on prior validation studies, where a score ≥5 is considered highly suggestive of HFpEF[6]. The control group, on the other hand, was selected from patients who did not contain heart failure symptoms and symptoms and were not diagnosed with HF. The exclusion criteria included individuals under 18 years of age, patients with acute kidney failure, acute coronary syndrome, pulmonary embolism, neurological disorders such as cerebrovascular disease and demyelinating conditions, other rheumatological and autoimmune disorders, and a history of malignancy.

A total of 148 individuals were screened for this study. Of these, 22 were excluded because they met one or more exclusion criteria or had incomplete data. The final study population included 64 patients with HFpEF and 62 control individuals, resulting in a total of 126 participants.

Based on the observed effect size for the TAPSE/sPAP ratio between the HFpEF and control groups (Cohen's d ≈ 0.7), a total sample size of 126 participants (64 in the HFpEF group and 62 in the control group) yielded a statistical power of 86.3% at an alpha level of 0.05. This indicates that the study had sufficient power to detect statistically meaningful differences between the groups regarding the primary outcome.

Procedure

In this randomized study, 126 participants were enrolled, consisting of 64 patients with HF who were monitored in our cardiology clinic between 2023 and 2024 and 62 individuals in a control group matched for age and sex. Demographic profiles, risk factors, and medical treatment histories of the patients were comprehensively assessed. During the hospitalization of the heart failure patients, routine laboratory parameters were documented, and H2FPEF scores were computed and recorded. Heart failure was diagnosed in accordance with the current guidelines.

TTE was performed using a commercially available ultrasound system (Philips EPIQ) equipped with a 2.5–3.5 MHz transducer. All measurements were performed in accordance with the guidelines of the American Society of Echocardiography (ASE). The recorded parameters included ejection fraction (EF, by Simpson's biplane method), left ventricular end-diastolic and end-systolic diameters (LVEDd and LVESd), interventricular septum thickness (IVSd), posterior wall thickness (PWd), left ventricular mass index (LVMI), left atrial volume index (LAVI), E/e' ratio, and relative wall thickness (RWT).

Right ventricular function was assessed by measuring the TAPSE using M-mode imaging in the apical four-chamber view. sPAP was estimated using the peak tricuspid regurgitation velocity, the simplified Bernoulli equation, and the estimated right atrial pressure based on the inferior vena cava diameter and collapsibility. The TAPSE/sPAP ratio was calculated as the primary echocardiographic marker of RV-pulmonary arterial coupling.

Venous blood samples were collected from all participants after overnight fasting for at least 8 h. Laboratory analyses included serum creatinine, sodium, potassium, hemoglobin, white blood cell count, neutrophils, platelets, and lipid profile (LDL, HDL, total cholesterol, and triglycerides). NT-proBNP values were measured in each patient with heart failure.

Statistical analysis

Statistical analyses were performed using SPSS version 18.0 (IBM Corp., Armonk, NY, USA). Categorical variables, such as sex and comorbidities, were expressed as counts and percentages, and differences between the HFpEF and control groups were analyzed using the chi-square (χ^2) test. The normality of the distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Continuous variables with a normal distribution, such

as age and body surface area, are presented as mean ± standard deviation and were compared using the Independent Samples t-test. Non-normally distributed variables, including NT-proBNP, TAPSE/sPAP, LAVI, and H2FPEF scores, were expressed as medians and interquartile ranges and compared using the Mann–Whitney U test.

Correlations between the TAPSE/sPAP ratio and other clinical variables, such as NT-proBNP, H2FPEF score, left atrial volume index (LAVI), and ejection fraction, were evaluated using Spearman's correlation analysis. Receiver Operating Characteristic (ROC) curve analysis was applied to determine the optimal cutoff value of the TAPSE/sPAP ratio for predicting HFpEF. Variables found to be significant in the univariate analysis ($p<0.1$) or deemed clinically relevant (e.g., BMI, NT-proBNP, EF, TAPSE/sPAP) were entered into a binary logistic regression model to identify independent predictors of HFpEF. Statistical significance was set at a two-tailed p-value of <0.05, and all analyses were interpreted with 95% confidence intervals (CIs).

RESULTS

64 heart failure (HF) and 62 healthy controls were studied. The average age of the patients was 66.75±8.81 years, with 65.6% of them being female. The patient group had higher Body Surface Area (BSA) and Body Mass Index (BMI), as well as higher rates of hypertension (HT) and atrial fibrillation. Additionally, the patient group had lower eGFR and Hgb levels and higher creatine and NT-proBNP levels. Specifically, while the TAPSE values alone did not show a significant difference between the HFpEF and control groups ($p=0.13$), the TAPSE/sPAP ratio did demonstrate a statistically significant difference (median 0.76 vs. 0.96, $p<0.001$). Echocardiographic parameters differed between the patient and control groups (Table 1).

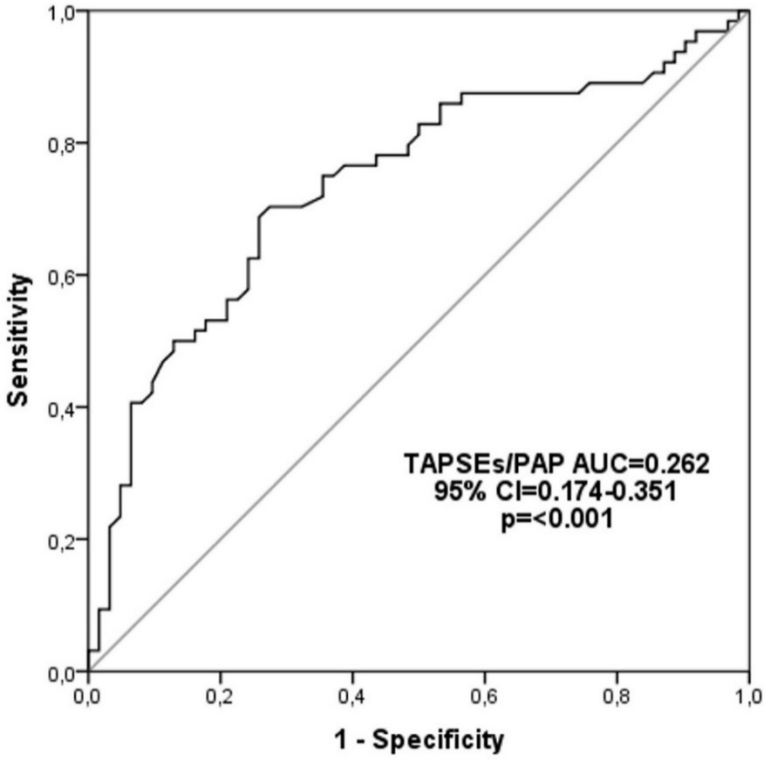

Figure 1. ROC analysis for TAPSE/sPAP

Table 1. Distribution of demographic, clinical, laboratory and echocardiographic parameters in patient and control groups

Variable	HF (n=64)	Control (n=62)	p value
Gender (Female); n (%)	42 (65.6)	31 (50.0)	0.076
Age (years)	66.75±8.81	66.82±6.73	0.959
BMI (kg/m²)	32.10 (24.05-47.06)	29.06 (22.03-44.9)	<0.001
BSA	1.99±0.15	1.91±0.15	0.005
DM; n (%)	28 (43.8)	18 (29.0)	0.086
HT; n (%)	45 (70.3)	31 (50.0)	0.020
AF; n (%)	31 (48.4)	15 (24.2)	0.005
CAD; n(%)	22 (34.4)	22 (35.5)	0.896
Asthma; n (%)	12 (18.8)	10 (16.1)	0.698
eGFR (ml/dk)	61.60±21.79	72.69±21.63	0.005
Creatinine (mg/dL)	1.07 (0.6-2.9)	0.90 (0.48-1.4)	0.042
Na (mmol/L)	138 (128-149)	139(130-145)	0.664
K (mmol/L)	4.38 ± 0.53	4.28 ± 0.45	0.231
Hgb (g/dL)	13.15 ± 1.66	13.67 ± 1.56	0.012
Wbc (10^3/μL)	8.11 (4.4-17.5)	8.02 (4.1-17.2)	0.946
Neu (10^3/μL)	4.40 (2.4-11.0)	4.30 (2.2-8.9)	0.245
Plt (10^3/μL)	270.73±72.83	260.04±59.23	0.369
LDL (mg/dL)	102.50 (44.0-214.0)	105.00 (46.0-227.0)	0.297
HDL (mg/dL)	43.50 (25.0-70.0)	43.60 (25.0-72.5)	0.574
T. Cholesterol (mg/dL)	175.50 (119-348)	176.50 (118-318)	0.515
Triglyceride (mg/dL)	166.00 (54-376)	167.00 (51-357)	0.668
NTproBNP (pg/mL)	2136.5 (556.75-4723.75)	136.5 (78.75-212.00)	<0.001
EF (%)	56.20 (50.9-66.4)	60.35 (51.6-65)	<0.001
LVEDd (mm)	4.80 (4.2-6.0)	4.80 (4.1-5.9)	0.150
LVESd (mm)	3.0 (2.1-4.2)	2.70 (2.1-3.9)	0.022
IVSd	1.20 (0.90-1.45)	1.10 (0.86-1.52)	0.150
PWd	1.10 (0.80-1.30)	1.00 (0.75-1.48)	<0.001
LV Mass (gr)	200.40 (131.0-349.2)	181.02 (124.0-317.0)	0.001
LVMI (gr/m²)	101.75 (57.0-169.0)	93.76 (66.59-153.07)	0.044
RWT	0.41 ± 0.06	0.42 ± 0.06	0.003
E/e' Average	12.32 ± 2.89	7.89 ± 1.96	<0.001
LAVI (ml/m²)	35.86 (26.7-49.8)	28.84 (19.1-43.6)	<0.001
TAPSE	25.19 ± 4.91	26.31 ± 3.29	0.136
RV SM	12.65 (8.0-15.2)	15.00 (10.0-18.0)	<0.001
sPAP	32.40 (22.5-46.9)	27.00 (19.8-57.6)	<0.001
H2FPEF score	5 (5-9)	3 (1-4)	<0.001
TAPSE/sPAP	0.76 (0.40-1.39)	0.96 (0.45-1.42)	<0.001

HF: Heart Failure, BSA: Body Surface Area, DM: Diabetes Mellitus, HT: Hypertension, AF: Atrial Fibrillation, CAD: Coronary Artery Disease, eGFR: Estimated Glomerular Filtration Rate, Hgb: Hemoglobin, Wbc: White Blood Cell, Neu: Neutrophil, Plt: Platelet, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, T. Cholesterol: Total Cholesterol, NTproBNP: N-Terminal pro B-Type Natriuretic Peptide, EF: Ejection Fraction, LVEDd: Left Ventricular End-Diastolic Diameter, LVESd: Left Ventricular End-Systolic Diameter, IVSd: Interventricular Septal Diameter, PWd: Posterior Wall Diameter, LVMAS: Left Ventricular Mass, LWMI: Left Ventricular Mass Index, RWT: Relative Wall Thickness, LAVI: Left Atrial Volume Index, TAPSE: Tricuspid Annular Plane Systolic Excursion, RV SM: Right Ventricular Systolic Motion, sPAP: Systolic Pulmonary Artery Pressure, TAPSE/sPAP: Ratio of Tricuspid Annular Plane Systolic Excursion to Systolic Pulmonary Artery Pressure.

Table 2. Correlation Analysis with TAPSE/sPAP Level in the Study Group

		TAPSE/sPAP
NT-proBNP (pg/mL)	r	-0.307
	p	<0.001
EF (%)	r	0.138
	p	0.122
LAVI (ml/m²)	r	-0.203
	p	0.023
H2FPEF score	r	-0.326
	p	<0.001

r= Spearman Correlation Coefficient

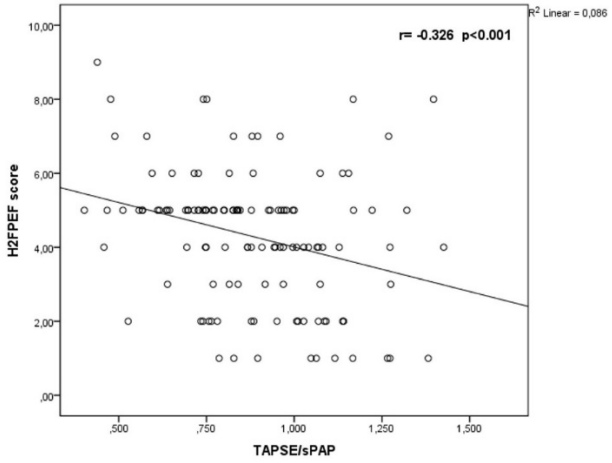

Figure 2. Correlation between TAPSE/sPAP ratio and H2FPEF score.

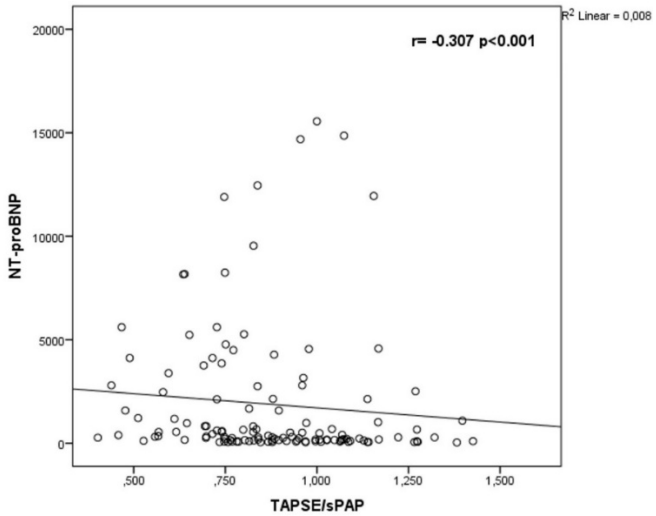

Figure 3. Correlation between TAPSE/sPAP ratio and NT-proBNP levels.

Table 3. Logistic regression analysis for HFpEF patients

	Univariate		Multivariate	
	OR (%95 CI)	**p**	**OR (%95 CI)**	**p**
BMI	1.182 (1.080-1.294)	<0.001	1.200 (1.038-1.388)	0.014
NT-proBNP	1.005 (1.003-1.008)	<0.001	1.005 (1.003-1.008)	<0.001
EF	0.821 (0.744-0.906)	<0.001	0.955 (0.808-1.129)	0.588
TAPSE/sPAP	0.019 (0.003-0.137)	<0.001	0.010 (0.001-0.283)	0.007

Cox&Snell R Square: 0.597, Nagelkerke R Square: 0.796, Accuracy: %90.5

The findings of this study have practical implications for the diagnosis and prediction of HF. Correlation analysis was conducted to evaluate the relationship between the TAPSE/sPAP ratio and the selected clinical parameters in the HFpEF group. A moderate and statistically significant negative correlation was found between the TAPSE/sPAP ratio and both NT-proBNP levels ($r = –0.307$, $p < 0.001$) and H2FPEF scores ($r = –0.326$, $p < 0.001$), suggesting that lower TAPSE/sPAP ratios are associated with increased filling pressures and a higher likelihood of HFpEF. Additionally, a weak but significant negative correlation was observed between the TAPSE/sPAP ratio and left atrial volume index (LAVI) ($r = –0.203$, $p = 0.023$).

No significant correlation was found between the TAPSE/sPAP ratio and ejection fraction ($r = 0.138$, $p = 0.122$), indicating that this parameter provides information independent of the left ventricular systolic function (Table 2). ROC curve analysis was performed to determine the cutoff point of the TAPSE/sPAP ratio in predicting the diagnosis of HF. It was found that TAPSE/sPAP values of ≤ 0.843 could predict the diagnosis of HF with 70.3% sensitivity and 72.6% specificity. The area under the curve was 0.738 ($p<0.001$; 95% CI= 0.649-0.826), indicating the potential utility of this parameter in clinical practice (Figure 1).

The results of our logistic regression analysis for BMI, NT-proBNP, EF, and TAPSE/sPAP parameters to predict HF in patients are presented in Table 3. According to the results of multivariate analysis, a 1-unit increase in the BMI parameter increased the risk of HF by 1,200 times ($p = 0.014$); Increasing the NT-proBNP parameter by 1 unit increased the risk of HF by 1.005 times ($p<0.001$); Increasing the TAPSE/sPAP parameter by 1 unit reduced the risk of HF (OR: 0.010; $p=0.007$). These findings underscore the reliability and precision of our models.

DISCUSSION

Our study highlights the prognostic significance of the TAPSE/sPAP ratio in patients with HFpEF, a condition that continues to challenge clinicians in terms of its diagnosis and management. Our findings demonstrate that the TAPSE/sPAP ratio is significantly lower in patients with HFpEF and exhibits a significant negative correlation with NT-proBNP levels and H2FPEF scores. These findings emphasize the potential of the TAPSE/sPAP ratio as a non-invasive prognostic marker for HFpEF, which is often associated with right heart dysfunction in patients with HFpEF.

The clinical profiles of patients with HFpEF differed from those of the control group, with a higher prevalence of comorbidities such as atrial fibrillation, hypertension, coronary artery disease, diabetes mellitus, and asthma. This is an expected outcome because HFpEF is a complex condition often associated with multiple comorbidities. Many patients with HFpEF also experience right ventricular dysfunction, tricuspid regurgitation, and right-sided heart failure[13]. Over time, distinguishing right heart failure due to HFpEF from other causes can be challenging, and its presence is associated with worse outcomes. More than 80% of patients with HFpEF also have pulmonary hypertension due to chronically elevated left heart pressures, often accompanied by right ventricular dysfunction[14,15]. Differentiating PH secondary to HFpEF from other forms of PH is thus crucial. In this context, our findings regarding TAPSE/sPAP values may contribute to identifying the role of HFpEF in the PH classification system.

TAPSE, which reflects longitudinal right ventricular contraction, is a commonly used measure of RV systolic function. However, as it evaluates only contractile motion without accounting for afterload, it has limitations in assessing RV-pulmonary artery

(PA) coupling. The TAPSE/sPAP ratio overcomes this limitation by integrating contractile function with pulmonary artery systolic pressure, offering a more comprehensive view of RV performance and its interaction with pulmonary circulation.

The TAPSE/sPAP ratio has shown promising associations with chronic kidney disease and survival in patients with systemic sclerosis[16]. Although its optimal cut-off is still under investigation, several studies have suggested thresholds such as 0.35 or 0.39 for risk stratification in pulmonary hypertension[17,18]. The inclusion of the TAPSE/sPAP ratio as an additional echocardiographic marker for PH diagnosis and 1-year mortality risk in the 2022 PH guidelines highlights its growing clinical relevance[19]. It has also been found to predict hospitalization in asymptomatic heart failure patients, with lower values correlating with an increased relative risk [20,21]. Its significance has been confirmed across different heart failure cohorts. For example, Bandera et al. (2019) showed that resting TAPSE/sPAP correlates more strongly with diastolic right heart pressures than pulmonary vascular resistance, indicating its potential as a marker of RV adaptation to pressure overload in HFrEF patients[22]. These findings support the ratio's broader promise as a prognostic indicator in various cardiac conditions.

In our study, although TAPSE alone did not differ significantly between HFpEF and control groups, the TAPSE/sPAP ratio was significantly reduced in HFpEF patients, highlighting its additional diagnostic value. Its correlations with NT-proBNP and H2FPEF scores suggest it reflects not only RV function but also elevated left ventricular filling pressures. A weak yet significant negative correlation with left atrial volume index (LAVI) further supports its association with structural remodeling in the left heart. Our logistic regression analysis confirmed that parameters such as BMI, NT-proBNP, and TAPSE/sPAP had predictive values for identifying patients with HF.

The ROC analysis conducted in this study identified a TAPSE/sPAP cut-off value of 0.843, with 70.3% sensitivity and 72.6% specificity for detecting HFpEF. While these values are moderate, they still support the potential utility of the ratio as a complementary diagnostic tool, especially when interpreted alongside clinical history, biomarkers, and scoring systems like H2FPEF. This could help refine diagnostic accuracy and assist in stratifying patients who may benefit from further evaluation or targeted therapies. Furthermore, the clinical utility of the TAPSE/sPAP ratio may extend beyond diagnosis and prognostic predictions. Incorporating this parameter into the routine evaluation of patients with HFpEF could aid in tailoring therapeutic strategies. In particular, changes in TAPSE/sPAP values during follow-up may provide valuable insight into the right ventricular response to treatment and could serve as a guide for adjusting therapeutic regimens.

These findings underscore the importance of using comprehensive measures, such as the TAPSE/sPAP ratio, rather than isolated parameters, such as TAPSE, when assessing RV function in patients with HFpEF. This ratio offers a more detailed understanding of RV performance in the context of increased pulmonary pressure, which is critical for accurate diagnosis, risk assessment, and clinical management. Solely relying on TAPSE may overlook key pathophysiological changes in HFpEF, whereas the TAPSE/sPAP ratio may more effectively identify at-risk patients and guide therapeutic decisions than TAPSE alone.

The primary strength of our study lies in its well-defined cohort of patients with HFpEF and the use of established diagnostic criteria, such as the H2FPEF score. However, the relatively small sample size is a limitation that may affect the generalizability of our findings. Although non-invasive measurements of these parameters are more convenient for patients, they may not capture the complete range of physiological changes associated with HFpEF. Another limitation of our study is the lack of follow-up measurements of TAPSE/sPAP after treatment. Evaluating the longitudinal changes in this ratio could offer insights into the effects of therapy on RV-PA coupling and help validate its utility in monitoring clinical improvement. As a result, these limitations highlight the need for more comprehensive, multi-center research efforts that involve a larger and more diverse patient population. Until such data are available, the TAPSE/sPAP ratio should be considered a supportive rather than a definitive tool in the diagnostic pathway of HFpEF. Future research should also explore the temporal changes in TAPSE/sPAP under different therapeutic strategies and their association with long-term clinical outcomes such as hospitalization, quality of life, and mortality. Additionally, combining this ratio with other right heart and diastolic function parameters may contribute to the development of a

comprehensive risk stratification model specifically tailored for patients with HFpEF.

The TAPSE/sPAP ratio may serve as a useful and easily obtainable echocardiographic measure that enhances the ability to identify patients with HFpEF. Its correlation with key diagnostic parameters, such as NT-proBNP and H2FPEF, and its capacity to distinguish patients with mild right heart involvement suggest that it could be integrated into standard HFpEF evaluation algorithms.

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