To cite this article: Yildiz Ö, Yenigun C. Prognostic value of left ventricular stroke work index in patients with advanced heart failure. Turk J Clin Lab 2022; 1: 9-15.

Original Article

Prognostic value of left ventricular stroke work index in patients with advanced heart failure

İleri evre kalp yetmezliği olan hastalarda sol ventriküler atım iş indeksinin prognostik değeri

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ABSTRACT

Aim: The impact of reduced myocardial contractility on patients' outcomes with advanced heart failure (HF) and the correct measurement units for its measure is most important. The present work aims to evaluate the prognostic value of the left ventricular stroke work index (LVSWI), which is considered a measure of myocardial contractility, in patients with advanced HF.

Material and Methods: Between September 2010 and July 2013, 172 patients with advanced HF admitted to the hospital to guide the specified therapies were included in this study. At baseline, patients were assessed with cardiac catheter-based hemodynamic measurements, ensued by the longitudinal follow-up (median of 52 months) for adverse outcomes (cardiac mortality, ventricular assist device (VAD) placement, and heart transplant (HTx)).

Results: Median LVSWI was 16 cJ/m2. Decreased LVSWI (<16 cJ/m2) was associated with increased adverse outcomes. We observed 50 cardiac deaths, 12 VAD placements and 10 HTx. The prognostic value of LVSWI remained significant after adjustment for age, gender, mean arterial pressure, pulmonary vascular resistance, right atrial pressure and estimated glomerular filtration rate (HR 0.379, 95% CI 0.17-0.84, p=0.017).

Conclusion: Lower LVSWI is an independent predictor of adverse outcomes in patients with advanced HF. Therefore, it could be used for individual risk stratification in these patients to predict clinical outcomes.

Keywords: Left ventricular dysfunction; heart failure; mortality; prognosis.

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Gönderim: 13.08.2021 kabul: 20.09.2021
Doi: 10.18663/tjcl.981827

ÖZ

Amaç: Azalan miyokard kontraktilitesinin ileri evre kalp yetmezliği (KY) olan hastaların sonuçları üzerindeki etkisi ve ölçümü için doğru ölçüm birimi çok önemlidir. Bu çalışma, ileri evre KY olan hastalarda miyokard kontraktilitesinin bir ölçüsü olarak kabul edilen sol ventrikül atım iş indeksinin (LVSWI) prognostik değerini araştırmayı amaçlamaktadır.

Gereç ve Yöntemler: Eylül 2010 ile Temmuz 2013 tarihleri arasında spesifik tedavi yöntemlerini alabilmek için hastaneye başvuran ileri evre KY olan 172 hasta bu çalışmaya dahil edildi. Başlangıçta kardiyak kateter bazlı elde edilen hemodinamik ölçümler longitudinal takipte (median 52 ay) ortaya çıkan olumsuz sonuçlar (kardiyak mortalite, ventriküler destek cihazı (VAD) implantasyonu ve kalp nakli (HTx)) ile değerlendirildi.

Bulgular: Median LVSWI 16 cJ/m2 idi. Azalan LVSWI (<16 cJ/m2), artan olumsuz sonuçlarla ilişkilendirildi. 50 kardiyak ölüm, 12 VAD implantasyonu ve 10 HTx gözlemledik. LVSWI'nin prognostik değeri, yaş, cinsiyet, ortalama arter basıncı, pulmoner vasküler direnç, sağ atriyal basınç ve tahmini glomerüler filtrasyon hızı için düzeltmeler yapıldıktan sonra anlamlı kaldı (HR 0.379, %95 CI 0.17-0.84, p=0.017).

Sonuç: Azalmış LVSWI, ileri evre KY hastalarında olumsuz sonuçların bağımsız bir öngörücüsüdür. Bu nedenle, klinik sonuçları tahmin etmek için bu hastalarda bireysel risk sınıflandırması için kullanılabilir.

Anahtar kelimeler: Sol ventrikül disfonsiyonu; kalp yetmezliği; mortalite; prognoz.

Introduction

The impact of reduced left ventricular myocardial contractility arising from diverse etiologies on therapeutic decision making and prognosis is undisputed. Therefore, it is essential to determine the left ventricular myocardial contractility of the heart accurately. Traditionally, cardiac out (CO) and cardiac index (CI) values are used as myocardial contractility indicators [1].

The left ventricular stroke work index (LVSWI) is a hemodynamic parameter considered the left ventricle's myocardial contractility to maintain blood circulation [2]. Over time, decreased myocardial contractility leads to reduced stroke work and, consequently, to an increased heart rate in patients with advanced heart failure (HF) [3]. Since calculating the LVSWI does not include the variable heart rate, it is more suitable for computing sensitive myocardial contractility changes than CO and CI.

The present research aimed to investigate the prognostic value of LVSWI in patients with advanced HF.

Material and Methods

Study design and study population

Our study cohorts included 172 consecutive patients with advanced HF admitted to a tertiary hospital between September 2010 and July 2013 for medical treatment optimization and evaluation for mechanical circulatory support device implantation and assessment for heart transplantation (HTx). After receiving and understanding all the research-related information, informed consent was obtained from all study populations. Local authorities approved this retrospective cohort study. From the patient's first contact to either undergoing ventricular assist device placement, HTx or cardiac mortality was defined as the length of follow-up time.

Study protocol

At baseline, we performed both right-sided and left-sided heart catheterization for all study patients to evaluate the hemodynamic measurements used for the present analysis. Simultaneously performing right-sided and left-sided heart catheterization, access was obtained by percutaneous puncture of the femoral vein and artery. Following placement of both a catheter into the pulmonary artery and the ascending aorta, a mixed central venous sample and an arterial sample were obtained from the catheter's tip. We computed CO by using Fick's equation, and then we divided cardiac output by the body surface area to obtain the CI. A multipurpose catheter was inserted into the pulmonary artery to monitor right hemodynamics until the wedge position was reached. Then, at the end of expiration, pulmonary capillary wedge pressure (PCWP), pulmonary artery pressure, and right atrial pressure were measured one after the other. To measure mean arterial pressure (MAP) invasively, we inserted a pigtail catheter into the ascending aorta.

The left ventricular contractility was assessed by computation of LVSWI according to the following formula: LVSWI = SVI x (MAP – PCWP) x 0.0133322, where SVI is indexed stroke volume, and the factor 0.0133322 converts volume and pressure units to centijoule (cJ) [4], which is the unit of work and energy measurement used in medicine to express cardiac work.

Patients have been categorized according to their functional capacity as class II or III, or IV in line with the New York Heart Association (NHYA) classification [5]. According to the American Society of Echocardiography guidelines, the left ventricular ejection fraction was determined using the biplane modified Simpson's method [6].

The measurements of the following variables were collected for all patients: Age, gender, left ventricular ejection fraction (LVEF), LVSWI, PCWP, estimated glomerular filtration rate (eGFR), MAP, right atrial pressure (RAP), CO, CI, pulmonary vascular resistance (PVR), mean pulmonary arterial pressure (MPAP), pulmonary arterial systolic pressure (PASP), transpulmonary gradient (TPG). Moreover, the following categorical variables were recorded as available or not available: cardiac resynchronization therapy-defibrillator (CRT-D), ischemic cardiomyopathy (ICMP), valvular heart disease, atrial fibrillation, chronic obstructive pulmonary disease (COPD), hypertension, diabetes mellitus, angiotensin-converting enzyme inhibitors, beta-blockers, mineralocorticoid receptor antagonist medication (MRA), diuretic medication.

Since eGFR less than 60 ml/min/1.73m2 is known to be an indicator of cardiovascular mortality [7,8], eGFR measurements of patients were reported as a dummy variable where 1 indicates that eGFR is below this threshold and 0 indicates otherwise. Measurement units of all variables are indicated in the tables.

Statistical analysis

We fit univariate and multivariate Cox regression models on survival times to investigate all variables' effects on the hazard. We created an LVSWI dummy variable, which is 1 if the LVSWI observation of a patient is above the threshold 16 cJ/m2 and 0 otherwise. The significant variables in the univariate regression models were CI, LVSWI, LVEF, PCWP, eGFR, MAP, RAP, PVR, MPAP, PASP, TPG, MRA and COPD. After this preliminary screening, we carried a stepwise procedure using all the significant variables to build a Cox regression model for studying their adjusted effects on the hazard. We used the well-known forward regression, where the Akaike Information Criterion (AIC) is the model comparison criterion. The base model includes age and gender as demographic variables, and CI is a commonly used variable in the literature. Table 3 displays the order of variables entering the model in the stepwise procedure, which may be regarded as the importance of predictive power variables. After 6th step, adding a new variable does not improve the model. Therefore, the stepwise procedure is terminated.

Results

Out of 172 patients, 11 had to be removed from the study as they did not provide enough observable lifetime due to death at the first contact or having a left ventricular assist device (LVAD) implantation. For the 161 patients in the study, the observed lifetime is the time from the first contact with the patient to an endpoint, which may refer to a cardiac death or some censoring form. A total of 50 cardiac deaths and 111 censoring times are observed, where the censoring times correspond to non-cardiac deaths (n=9), HTx (n=10), LVAD implantations (n=12), and being alive (without HTx or LVAD implantation) at the end of the study (n=80). Table 1 presents the patients' baseline characteristics stratified for LVSWI threshold 16 cJ/m2. Table 2 presents hemodynamics with the same stratification. How this threshold value was obtained will be explained below. In both tables, percentages of the indicated levels are reported for categorical variables, and means and standard deviations (in parenthesis) are reported for numerical variables.

Figure 1 displays the result of our search procedure for a reasonable threshold point for LVSWI, where several threshold values are plotted against the corresponding chi-squared distance between the survival curves of the patients having LVSWI values on either side of the threshold. As displayed in the figure, the largest distance is attained when the LVSWI threshold equals 16 cJ/m2. In other words, when the patients are divided into two strata as having LVSWI below (including) and above the threshold 16 cJ/m2, the log-rank test returns a chi-squared distance of 24.4 ($p<0.0001$), which is the maximum among all possible thresholds. Patients with LVSWI, less than or equal to 16 cJ/m2, have significantly smaller survival probabilities, therefore, may be regarded as high-risk patients. There are 44 patients below and 117 patients above this LVSWI threshold.

Figure 2 displays Kaplan-Meier estimates of survival functions for all patients based on LVSWI strata, along with 95% confidence intervals and numbers at risk by time. As indicated above, survival probability in the high LVSWI strata is significantly higher.

Table 1. Baseline characteristics

Variable	Overall (n=161)	LVSWI≤16 (n=44)	LVSWI>16 (n=117)	p-value
Age (Years)	58.7 (11.2)	56.5 (10.1)	59.5 (11.6)	0.1
Gender (Female)	26.1%	25.0%	26.5%	1
Left ventricular ejection fraction	27.4 (4.74)	24.7 (4.37)	28.5 (4.46)	<0.0001
Chronic obstructive pulmonary disease	13 %	4.5%	16.2%	0.088
Estimated glomerular filtration rate dummy (Low)	16.8%	31.8%	11.1%	0.0044
Cardiac resynchronization therapy-defibrillator	45.3%	52.3%	42.7%	0.365
Ischemic cardiomyophathy	55.9%	54.5%	56.4%	0.9726
Body surface area	1.88 (0.19)	1.83 (0.18)	1.90 (0.2)	0.048
Hypertension	57.8%	47.7%	61.5%	0.161
Diabetes mellitus	34.8%	31.8%	35.9%	0.765
Valvular heart disease	10.6%	18.2%	7.7%	0.1
Atrial fibrillation	8.7%	11.4%	7.7%	0.672
New York Heart Association class				
II	29.2%	11.4%	35.9%	0.004
III	56.5%	54.5%	57.3%	0.895
IV	14.3%	34.1%	6.8%	<0.0001
Medications				
Angiotensin converting enzym inhibitors	73.9%	68.2%	76.1%	0.416
Beta blockers	83.2%	77.3%	85.5%	0.315
Diuretic	68.3%	72.7%	66.7%	0.585
Mineralocorticoid receptor antagonist	64.6%	70.5%	62.4%	0.481

Table 2. Baseline haemodynamic values

Variable	Overall (n=161)	LVSWI≤16 (n=44)	LVSWI>16 (n=117)	p-value
Mean arterial pressure (mmHg)	85.0 (13.6)	79.6 (14.4)	87.1 (12.7)	0.0033
Right atrial pressure (mmHg)	9.71 (5.41)	12.3 (6.18)	8.74 (4.76)	0.0009
Cardiac index (L/min.m2)	2.09 (0.54)	1.63 (0.39)	2.27 (0.49)	<0.0001
Cardiac output (L/min)	3.80 (1.11)	3.01 (0.81)	4.31 (1)	<0.0001
Pulmonary capillary wedge pressure (mm Hg)	21.7 (8.97)	29.0 (9.32)	19.0 (7.14)	<0.0001
Pulmonary vascular resistance (dyn*sec/cm5)	260 (197)	392 (256)	210 (142)	<0.0001
Mean pulmonary arterial pressure (mmHg)	33.0 (12.4)	42.4 (12.8)	29.4 (10.3)	<0.0001
Pulmonary arterial systolic pressure (mm Hg)	50.9 (19.3)	64.9 (20)	45.6 (16.3)	<0.0001
Transpulmonary gradient (mm Hg)	11.3 (6.24)	13.6 (6.83)	10.5 (5.81)	0.00692

Numbers in parantheses are in standard deviations.

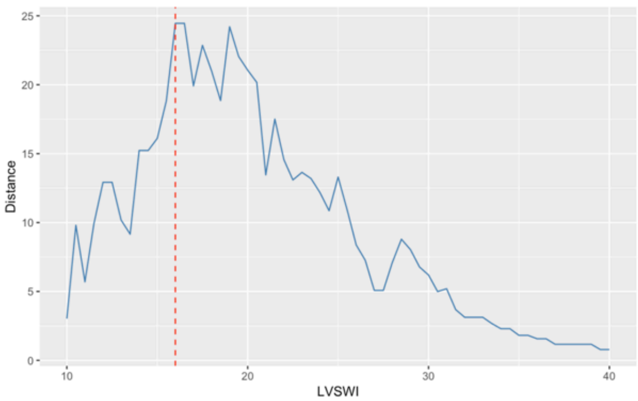

Figure 1. Search for LVSWI threshold value producing the maximum distance between survival curves.

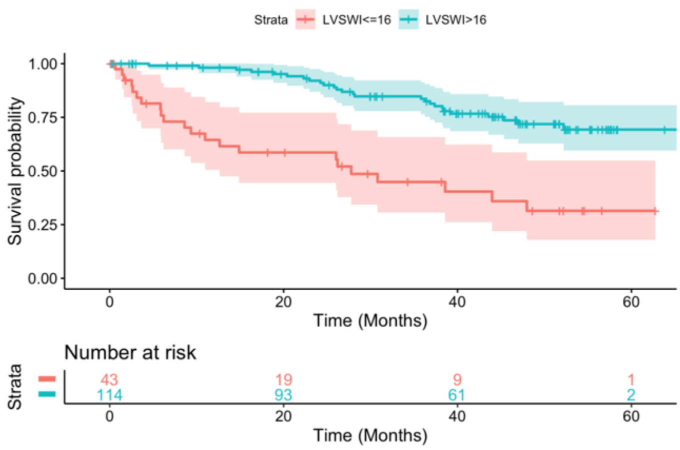

Figure 2. Kaplan-Meier estimates for low and high LVSWI levels.

We fit univariate and multivariate Cox regression models on survival times to investigate all variables' effects on the hazard. We created an LVSWI dummy variable, which is 1 if the LVSWI observation of a patient is above the threshold 16 cJ/m2 and 0 otherwise. The significant variables in the univariate regression models were CI, LVSWI, LVEF, PCWP, EGFR, MAP, RAP, PVR, MPAP, PASP, TPG, MRA and COPD. After this preliminary screening, we carried a stepwise procedure using all the significant variables to build a Cox regression model to study their adjusted effects on the hazard. We used the well-known forward regression, where the Akaike Information Criterion (AIC) is the model comparison criterion. The base model

includes age and gender as demographic variables and CI as it is a commonly used variable in the literature. Table 3 displays the order of variables entering the model in the stepwise procedure, which may be regarded as the importance of predictive power variables. After the 6th step, adding a new variable does not improve the model; therefore, the stepwise procedure is terminated.

Table 3. Forward Stepwise Cox Regression with Akaike Information Criterion (AIC)

	Variable to Enter	AIC
Base Model	Age, gender, cardiac index	448.95
Step 1	Estimated glomerular filtration rate	429.61
Step 2	Left ventricular ejection fraction	413.15
Step 3	Mean arterial pressure	405.0
Step 4	Right atrial pressure	396.78
Step 5	Left ventricular stroke work index	394.04
Step 6	Mineralocorticoid receptor antagonist	393.0

Details of the final adjusted multiple Cox regression model is given in Table 4, along with the unadjusted univariate Cox regression details for each variable in the final model. The reported hazard ratios correspond to the exponentials of regression coefficients, representing the corresponding variables' relative hazard. We see that the hazard ratio of LVSWI equals 0.379, indicating patients with LVSWI greater than the threshold 16 cJ/m2 have about 62.1% less hazard than patients with LVSWI less than or equal to this threshold. Other significant variables are LVEF, RAP and MAP with similar interpretations. Given these four variables, CI, MRA, age and gender do not seem to affect the hazard significantly.

Discussion

The present investigation has several important key findings. These may contribute to our understanding of the clinical implications for assessing a patient's prognosis with advanced HF. First, this is an investigation with an adequate sample size of patients with advanced HF and long-term follow-up. Therefore, we were able to evaluate the clinical impact of LVSWI sufficiently. Second, LVSWI was associated with other common hemodynamic variables of HF severity. Third, we demonstrated a strong association between LVSWI and ventricular assist device- and heart transplant-free survival. Finally, in the present study LVSWI emerged as a significant hemodynamic predictor of adverse outcomes in patients with advanced HF.

The left ventricular stroke work is related to the left ventricle's work during a cardiac cycle to eject the stroke volume into the aorta [4]. As the kinetic energy and friction loss is negligible, it is presented by the area within the left ventricular pressure-volume diagram [9]. The most widely used system of measurement in science is the International System of Units ("Systeme international d'unites"), abbreviated as SI [4]. The Joule is SI derived unit for work or energy. The SI prefix "centi" represents a factor of 10-2 Joules. The calculation with centijoule (cJ) unit is due to small measurements of LVSWI more precise than with Joules, and the result can be easily converted to Joules unit if necessary.

When comparing scientific papers, correct units of measurement are a critical condition. Since different units of measurement and formulas to calculate LVSWI are in use, a comparison of data from various studies is not without problems [10]. Therefore, only a few studies have the correct units of measurement and formulas whose outcomes can be compared.

The concept of LVSWI has been previously studied in investigations with small sample size and event rates. A previous trial of 63 patients with HF demonstrated at a one-year follow-up that patients with LVSWI less than 0,20 J/m2 died of progressive HF while patients with LVSWI 0.4 J/m2 were alive [11]. Looking back to our data, we found for the LVSWI a cut-off value of 16 cJ/m2, which is equal to 0,16 J/m2. This discrepancy could be related to the fact that in contrast to our homogeneous chronic advanced HF population, in the previous trial with small sample size, 26 of 63 patients had acute HF due to myocardial infarction, and the remainder had chronic or acute-on-chronic HF with moderate to severe symptoms [11]. In another prior investigation of 34 patients who underwent non-cardiac surgery, a rapid test with volume infusion was performed [12]. The ability to increase left ventricular work in response to hemodynamic stress as a

Table 4. Cox regression (proportional hazards) models			
Variable	Hazard ratio	95% Confidence Interval	p-value
Unadjusted			
Age	1.009	0.98-1.04	0.498
Gender (1=female, 0=male)	1.077	0.57-2.03	0.82
Cardiac index (L/min.m2)	0.385	0.21-0.69	0.0009
Estimated glomerular filtration rate			
dummy (Low %)	2.261	1.17-4.36	0.015
Left Ventricular Ejection Fraction (LVEF)	0.918	0.86-0.98	0.0085
Mean arterial pressure (mmHg)	0.973	0.95-0.99	0.0159
Right atrial pressure (mmHg)	1.087	1.04-1.14	0.0004
Left Ventricular Stroke Work Index (LVSWI) Dummy (1 if ≤16 0 if >16)	0.264	0.15-0.46	<0.0001
Mineralocorticoid receptor antagonist (%)	2.226	1.11-4.64	0.0242
Adjusted			
Age	0.995	0.96-1.03	0.774
Gender (1=female, 0=male)	0.906	0.42-1.92	0.797
Cardiac index (L/min.m2)	0.812	0.35-1.87	0.626
Estimated glomerular filtration rate dummy (Low %)	1.432	0.63-3.23	0.387
Left Ventricular Ejection Fraction (LVEF)	0.939	0.87-1.01	0.097
Mean arterial pressure (mmHg)	0.961	0.936-0.987	0.0038
Right atrial pressure (mmHg)	1.090	1.03-1.15	0.001
Left Ventricular Stroke Work Index (LVSWI) Dummy (1 if ≤16 0 if >16)	0.379	0.17-0.84	0.017
Mineralocorticoid receptor antagonist (%)	1.669	0.79-3.51	0.177

predictor of perioperative cardiac complications was tested. The patients developing perioperative cardiac complication had an LVSWI of 0.28 J/m2 and who recovered without a cardiac complication had 0.47 J/m2. In a prior study of 31 patients with severe HF, the stroke work index did not predict the one-year follow-up mortality [13]. Compared to the trials mentioned earlier, the present analysis had sufficient sample size and event rates and long-term follow-up period to demonstrate a strong association with LVSWI and mortality.

The renal perfusion is reduced in advanced HF, leading to impaired renal function. An indirect indicator of overall renal function is serum creatinine, strongly associated with HF and mortality progression in patients with cardiac dysfunction [14]. Calculating the eGFR using readily available clinical measurements can help identify advanced HF patients at increased cardiac death risk [8]. In a prior investigation, it has been reported that patients with congenital HF and ventricular dysfunction with reduced CO and eGFR < 60 ml/min/1.73 m2 had a 3-fold increased risk of mortality [15]. Comparably, based on our results associating with eGFR, the LVSWI has an incremental prognostic value for cardio-renal outcomes in patients with advanced HF.

When looking at our data, it is noticeable that both RAP and MAP were also independently associated with cardiac death in multivariate Cox regression analysis. Commonly, the RAP value has been used as a marker of right ventricular dysfunction. Compared to LVSWI, RAP is affected by tricuspid valve regurgitation, which occurs in response to right ventricular dilatation in pulmonary hypertension due to left-sided HF [16]. Hence, LVSWI is a more accurate measure of heart function. Compared to LVSWI, MAP is influenced by a complex interplay between the cardiovascular, renal, and autonomic nervous systems [17], and severe aortic regurgitation. Thus, LVSWI is a more robust and precise representation of cardiac performance. Furthermore, MAP and SVI, and PCWP, are hemodynamic parameters of cardiac function that can predict clinical outcomes, but one can not necessarily predict the other. For instance, cardiogenic shock is marked by decreased SVI and MAP and increased PCWP, whereas decreased SVI and MAP and PCWP mark distributive shock [18]. LVSWI integrates the above-mentioned hemodynamic parameters and, therefore, a more reliable representation of the overall cardiac function.

The loading conditions (preload and afterload) and contractility are essential determinants of cardiac function. Contractility indices should assess the performance potential of the heart without dependent on loading conditions. However, traditional contractility indices such as CO and CI are heavily dependent upon loading conditions and the heart rate. Still, the CI as indicators of myocardial contractility has been demonstrated to correlate well with prognosis [19,20]. According to our data, LVSWI overperformed CI referred to predict adverse clinical outcomes, which could be related to the fact that the LVSWI is independent of heart rate [21].

Study limitations

Based on the retrospective study design, the present investigation's findings should be interpreted with diverse restrictions. The attendance of selection bias for consecutive patients with advanced HF who were referred to a tertiary hospital for optimization of heart failure medication and to be evaluated for circulatory assist device implantation as well as for HTx cannot be ruled out. According to the severity of illness, however, patients with advanced HF are predetermined to compare adverse events. Furthermore, to analyzing the LVSWI at rest only at one point in time, it is obscure whether temporary hemodynamic changes are compatible with clinical outcomes. Due to the severity of the illness, however, no improvement in health can be expected. Furthermore, endpoints such as HTx and LVAD implantation might be biased by selecting donor heart availability and mechanical circulatory support.

Conclusions

The outcome of the present analysis determines LVSWI <16cj/m2 as having an independent association with increased risk of cardiac mortality. This result underlines the potential for LVSWI to be a useful tool for risk stratification in patients with advanced HF. Prospective randomized studies should confirm the prognostic value of LVSWI in patients with advanced HF.

Declaration of conflict of interest

The authors have no conflicts of interest to declare. The authors received no funding for this work.

References

1. Ramsey JD, Tisdale LA. Use of ventricular stroke work index and ventricular function curves in assessing myocardial contractility. Crit Care Nurse 1995; 15: 61-7.
2. Bilchick KC, Mejia-Lopez E, McCullough P, et al. Clinical impact of changes in hemodynamic indices of contractile function during treatment of acute decompensated heart failure. J Card Fail 2018; 24: 43-50.
3. Cooper LB, Mentz RJ, Stevens SR, et al. Hemodynamic Predictors of Heart Failure Morbidity and Mortality: Fluid or Flow? J Card Fail 2016; 22: 182-9.
4. Schramm W. The units of measurement of the ventricular stroke work: a review study. J Clin Monit Comput 2010; 24: 213-7.
5. American Heart Association. AHA medical/scientific statement 1994 revisions to classification of functional capacity and objective assessment of patients with disease of the heart. Circulation 1994; 90: 644-5.
6. Hahn RT, Abraham T, Adams MS, et al. Guidelines for performing a comprehensive transesophageal echocardiographic examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. J Am Soc Echocardiogr 2013; 26: 921-64.
7. Thompson S, James M, Wiebe N, , et al. Cause of Death in Patients with Reduced Kidney Function. J Am Soc Nephrol 2015; 26: 2504-11.
8. Yildiz O, Aslan G, Demirozu ZT, et al. Evaluation of Resting Cardiac Power Output as a Prognostic Factor in Patients with Advanced Heart Failure. Am J Cardiol 2017; 120: 973-9.
9. Pilz G, Appel R, McGinn P. Calculation of the left ventricular stroke work index. Crit Care Med 1993; 21: 1248.
10. Settergren G. The calculation of left ventricular stroke work index. Acta Anaesthesiol Scand 1986; 30: 450-2.
11. Tan LB. Cardiac pumping capability and prognosis in heart failure. Lancet 986; 2: 1360-3.
12. Leung DY, Armstrong G, Griffin BP, et al. Latent left ventricular dysfunction in patients with mitral regurgitation: feasibility of measuring diminished contractile reserve from a simplified model of noninvasively derived left ventricular pressure-volume loops. Am Heart J 1999; 137: 427-34.
13. La Vecchia L, Varotto L, Spadaro GL, et al. Left ventricular stroke work reserve as prognostic marker in severe heart failure. Minerva Cardioangiol 2006; 54: 249-55.
14. Ronco C, McCullough P, Anker SD, et al. Acute Dialysis Quality Initiative (ADQI) consensus group. Cardio-renal syndrome: report from consensus conference of the acute dialysis qualitative initiative. Eur Heart J 2010; 31: 703-11.
15. Dimopoulus K, Diller GP, Koltsida E, et al. Prevalence, predictors, and prognostic value of renal dysfunction in adults with congenital heart disease. Circulation 2008; 117: 2320-8.
16. Rao S, Tate DA, Stouffer GA. Hemodynamic findings in severe tricuspid regurgitation.Catheter Cardiovasc Interv 2013; 81: 162-9.
17. Domanski MJ, Mitchell GF, Norman JE, et al. Independent prognostic information provided by sphygmomanometrically determined pulse pressure and mean arterial pressure in patients with left ventricular dysfunction. JACC 1999; 33: 951-58.
18. Simmons J, Ventetuolo CE. Cardiopulmonary monitoring of shock. Curr Opin Crit Care 2017; 23: 223-31.
19. Franciosa JA. Why patients with heart failure die: hemodynamic and functional determinants of survival. Circulation 1987; 75: 20-7.
20. Szabo B, Marosi EK, Vargova K, et al. Cardiac Index by Transthoracic Echocardiography (CITE) study. PLoS One 2018; 13: 0207269.
21. Ochagavia A, Zapata L, Carrillo A, et al. Evaluation of contractility and postloading in the intensive care unit. Med Intensiva 2012; 36: 365-74.