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Determination of Right and Left Ventricular Echocardiographic Parameters that May Predict Instability in Patients with Stable and Unstable Ventricular Tachycardia During Electophysiological Study

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Abstract: Background:SCD constitute a significant part of cardiovascular deaths. More than 90% of such deaths result from VT and/or VF. Despite the use of new pharmalogical agents, RF ablation and ICD therapies, prognosis of patients with ventricular arrhytmias is still poor. Hemodynamic status during VT determines the treatment strategy. **Aim:**We aimed to evaluate RV and LV echo parameters for prediction of hemodynamic instability in patients with stable or unstable VT during EPS.**Material-Metod**: Patients with induced VT during EPS were included. Participants divided into two groups according to the hemodynamic situation as stable or unstable. **Results:** History of cardiovascular disease, diabetes, hyperlipidemia and medication with antiarrhytmic drugs were similar in both groups. However; prevalance of hypertension and the number of patients in NYHA class III were higher significantly more in the unstable VT group. LVEF values of unstable VT group were significantly lower and MPItri values were significantly higher.
Conclusion: Unstable VT may be observed in participants who are elderly, hypertensive, lower LVEF, higher MPItri values, have worse baseline NYHA functional. Participants with these clinical characteristics may need more aggressive pharmacologic therapy and ICD programming approach. More conservative medical therapy and ICD programming approaches may be used in participants without these characteristics.

Keywords: *Echocardiography,electrophysiological study,instability,ventricular tachicardia.*

I. INTRODUCTION

Ventricular arrhythmias may be asymptomatic or have different clinical presentations including dyspnea, chest pain, feeling of weakness, hypotension, syncope, and sudden cardiac death. The primary concern regarding these arrhythmias is the potential risk of a sudden cardiac death (SCD) (1), which accounts for a significant portion of cardiovascular deaths. A very significant proportion of such deaths occur as a result of ventricular tacyhcardia (VT) and/or ventricular fibrilation (VF). Ventricular arrhythmias occur in approximately

20% of patients with acute myocardial infarction and in approximately 50% of patients with heart failure, and these arrhythmias are associated with both very high mortality and morbidity rates.

Despite the use of new pharmacological agents, advances in electrophysiology and the increasing effective use of implantable cardioverter defibrillators (ICDs), the prognosis still remains poor. The effect of ventricular tachycardia on hemodynamics is an important point of distinction in the treatment approach (2). In patients with heart failure, VT has an important role in worsening heart failure and sudden cardiac death (3). In this patient group, it represents a phenotypic expression of multiple complex mechanisms including neurohormonal signaling changes, structural remodeling, and electrophysiological changes (4). It is also associated with many factors involved in its pathophysiology such as ischemia (5), myocardial fibrosis and scar tissue (6), and sympathetic nervous system stimulation (7).

The prognosis of ventricular arrhythmias can be misleading when evaluated in a simplistic manner. One may think that these malignant arrhythmias may be more fatal with increasing morbidities. However, there exists some conflicting data. It has been shown that not all patients with ventricular arrhythmias and coronary artery disease have a poor diagnosis. One study evaluated 124 patients with a history of previous myocardial infarction and hemodynamically stable ventricular tachycardia and found an annual sudden death rate of 2.4%. In another study, Brugada *et al.* observed that only three (2.1%) of 140 patients with a hemodynamically stable VT episode experienced sudden cardiac death during a 26-month follow-up period (8). In this regard, the effect of ventricular arrhythmia on hemodynamics and its determinants gains importance in terms of both prognosis and treatment approach. Based on the hemodynamic status during VT, patients can be categorized into two groups. Patients with pulseless VT and those who exhibit at least two of the following symptoms during VT, namely ongoing or worsening respiratory failure, chest pain, hypotension, shock, pulmonary edema, and altered consciousness, can be classified as the unstable VT group while those patients with VT who do present the aforementioned characteristics can be classified as the stable VT group (9). Electrophysiological study, an important diagnostic and therapeutic method in these patient groups, is performed to determine the risk, to evaluate the need for ICD implantation or the function of implanted devices, and to perform ablation. If hemodynamically unstable tachycardias can be predicted and ICD programming can be performed based on these predictive parameters, it may contribute to the resolution of ICD-related problems (10,11). Ventricular tachycardia may be re-induced in most patients presenting with VT, especially in those with coronary arter disease (12). However, it is unclear which patients with induced VT will be stable or unstable.

In the light of all these data, our aim was to determine the right and left ventricular echocardiographic parameters that may be predictive of instability in patients with stable or unstable ventricular tachycardia during electrophysiological evaluation.

II. MATERIAL AND METHODS

The study was conducted at the Cardiology Department, Faculty of Medicine, Pamukkale University. Patients included those who presented to our clinic with a complaint of cardiac arrhythmia and met the inclusion criteria.

Study Group

The study group included 93 patients scheduled for ICD implantation for primary and secondary prevention. All patients had an indication for class I ICD implantation according to current guidelines. Detailed anamnesis and physical examinations were performed in all patients before the indication for ICD as well as evaluations of routine laboratory tests. They all underwent direct chest radiography and ECG analysis prior to implantation. Patients with potantial reversible causes of VT such as electrolyte imbalance (n=11), acute ischemia (n=11), and history of potential drug-induced QT dispersion (n=19) were excluded. All patients underwent EPS before ICD implantation. Patients who showed no VT induction on EPS and whose VT cycle duration was below 225 msec were excluded. And patients who showed VT induction on EPS were categorized into two groups; those with pulseless VT and who exhibited at least two of the following symptoms during VT, namely ongoing or worsening respiratory failure, chest pain, hypotension, shock, pulmonary edema, and altered consciousness as the unstable VT group; and those with VT who do present the aforementioned characteristics as the stable VT group based on their hemodynamic status during VT in accordance with the American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care.

Two-dimensional transthoracic echocardiography (TTE) was performed in all patients before EPS. Patients without an adequate echocardiographic window (n=9), which would prevent TTE and obtain reliable information were excluded. Patients with severe aortic stenosis, mitral stenosis, aortic regurgitation, moderate or severe mitral insufficiency, atrial septal defect, ventricular septal defect, ascending aortic aneurysm or aortic dissection (n=6) were also excluded. As a result, the study included a total of 93 patients, including 48 hemodynamically stable and 45 hemodynamically unstable patients.

Two-dimensional transthoracic echocardiography and electrophysiological studies of the patients included in the study were evaluated according to the routine clinical procedures. During the electrophysiological study, patients were divided into two groups, including those with stable and unstable ventricular tachycardia. Comprehensive demographic, electrophysiologic and echocardiographic parameters were compared between the two groups.

None of the patients underwent additional test or intervention during the study. This study was approved by the Non-Interventional Clinical Researches Ethics Committee of Pamukkale University.

Statistical Analysis

Statistical analyses were performed using the Statistical Package for Social Sciences – (SPSS) version 16.0 on the Windows Operating System. Continuous variables were expressed as mean ± standard deviation while categorical variables reported as number (percentage). The qualitative data was compared using the Chi-square test and the Student'*s*t*-test was used for comparison of quantitative data. The results were analyzed with a 95% confidence interval and significance level was set at $p \leq 0.05$. Backward logistic regression analysis was employed to examine the variables considered to have an impact on the dependent variable.

III. RESULTS

The ninetythree patients with EPS-induced ventricular tachycardia were divided into two groups as hemodynamically stable (Group I) and unstable (Group II). The mean age of the stable ventricular tachycardia group was 59.92±12.38 years, while the mean age of the unstable ventricular tachycardia group was 67.04±10.40 years (p=0.033). Stable VT group had significantly better functional capacity according to the NYHA classification (NYHA class I-II 76% in Group 1 vs. 24% in Group 2, p=0.01). In terms of risk factors, patients with unstable VT were more hypertensive (24% in Group 1 vs. 56% in Group 2, p=0.021). No statistically significant difference was found between the groups in terms of other demographic data and risk factors (Table 1).

The diameter, area, volume, systolic and diastolic parameters of the heart chambers were measured by TTE in the stable and unstable VT groups and data were compared. In the unstable VT induced group, among left ventricular echocardiographic parameters, left venticular ejection fraction (LVEF) values were significantly lower compared to the stable VT induced group (p=0.039). No statistically significant difference was found in other left ventricular systolic and diastolic parameters and Doppler measurements between the two groups (p>0.05). Among the right ventricular echocardiography parameters, myocardial performance index tricuspid (MPItri) values were higher in the unstable VT induced group than in the stable VT induced group (p=0,027). There was no statistically significant difference between the two groups in other right ventricular echocardiography parameters (Table 2).

The results of regression analysis of the factors involved in the development of hemodynamic instability in patients with EPS-induced VT are shown in Table 3. Backward logistic regression analysis of the parameters involved in development of unstable VT development showed that age (hazards ratio (HR) =4.95 [95% Confidence Interval (CI), 1.03-23.69], p=0.045, MPItri (HR =7.74 [95% CI, 1.51-39.49], p=0.014) were independent risk predictors for the development of unstable VT.

IV. DISCUSSION

Our main finding in this study was that in patients with electrophysiological study induced unstable VT, low ejection fraction determined by echocardiography and myocardial performance index (MPItri) derived from right ventricular measurements of the tricuspid area are important parameters for predicting instability. Age and

pre-procedural NYHA class were significantly higher in patients with induced unstable VT. Finally, the presence of hypertension seems to be a risk factor with predictive value in the induction of unstable VT.

In the patient population with underlying structural cardiac disease and low left ventricular fibrillation and unstable ventricular tachycardia, sudden cardiac death resulting from cardiac arrest secondary to malignant arrhyhtmias such as ventricular fibrillation and unstable ventricular tachycardia is common. Despite significant advances in the treatment of heart failure in recent years, approximately half of patients are lost with sudden cardiac death due to fatal arrythmias or recurrent ischemic episodes (13-15). The most commonly used and best-defined risk factor for major arrhythmic events is the assessment of left ventricular function. Nevertheless, a simple algorithm that can be used to select patients who may benefit from ICD implantation by evaluating echocardiographic measurements and parameters that may be predictive of fatal arryhthmia has not been established (16). As it has been shown in both the Canadian Implantable Defibrillator Study (CIDS) and The Antiarrhythmics Versus Implantable Defibrillators (AVID) Study that low LVEF is effective in predicting the expected benefit from ICD implantation and treatment, most clinicians prefer ICD implantation in patients who experienced ventricular tachycardia and/or ventricular fibrillation in this patient group (17). Another study showed that the frequency of arrhythmic event recurrence correlated with the severity of LV dysfunction in survivors of sustained VT (18). In another study investigating the predictors of malignant arrhythmia, low LVEF and NYHA Class II and above were found to be significant parameters (19).

A review of the scientific literature indicates that there is a clear association between low LVEF and malignant arrhythmias. However, in these studies, ICD indications were questioned, and ventricular arrhythmias were evaluated. The results of our study are in parallel with these data, but also reveal a new perspective. As we determined in our study, low LVEF is important in demonstrating its relation to instability rather than the occurence of VT. In patients with a low left ventricular ejection fraction, it is expected that the limited functioning of the already compromised heart may result in inadequate perfusion to vital organs such as the brain and other organ systems in response to ventricular arrhythmias and these patients cannot tolerate such condition. However, the lack of a consistent correlation between reduced ejection fraction and NYHA class or hemodynamic status raises an intriguing question from a scientific perspective. Studies have demonstrated that LVEF has a relatively low specificity in predicting sudden cardiac death. In a study investigating the use of left ventricular ejection fraction in predicting sudden cardiac death, no significant difference was found in the rates of sudden cardiac death secondary to arrhythmia between the patient group with LVEF below 30% and those with LVEF ranging from 30% to 40% (20). Similarly, in the European Autonomic Tone and Reflexes After Myocardial Infarction (ATRAMI) study, which included 1284 patients with previous myocardial infarction, left ventricular ejection fraction was below 35% in only less than half of the patient population during 21-months follow-up (21).

The myocardial performance index (MPI) is a numeric value obtained using cardiac time intervals. MPItri provides important information about right ventricular systolic and diastolic functions. MPI is known to increase in patients with heart failure. In a study evaluating idiopathic diabetic cardiomiopathy (DCMP), MPI was significantly higher in the patient group (21). In another study, MPI showed a remarkable increase along with the degree of heart failure (22). In our study, the association between increased MPItri measurement and unstable VT induced by EPS was demonstrated. A similar relationship was shown in a study showing the correlation between tricuspid annular plane systolic excursion (TAPSE), which is another right ventricular measurement, and ventricular dyssynchrony and interventricular interaction (23). Another study evaluating the patients with AF found that MPI was an independent risk factor for incerased MACE. This study excluded patients with known HF (24). It seems quite plausible that right ventricular performance may contribute to hemodynamic instability in malignant arrhythmias. Our study has demonstrated the predictive power of MPI for unstable VT regardless of patient's baseline characteristics as it is not affected by blood pressure, heart rate, ventricular geometry, and valvular insufficiency (25) and is easily measureable and reproducible.

It has been shown that ventricular arrhythmias increase with age (26). Similarly, QT and/or QTc increase, as well. Additionally, there is an increment in QT dispersion. Likewise, an increase in comorbidities also accompanying with age, and it is inevitable that they have an impact on predisposition for arrhythmia (27). However, medications with antiarrhymitic effects and health screening have also increased. Therefore, evaluating the potential of ventricular arrhyrthmias based solely on age may be an oversimplified interpretation.

Indeed, as we have demonstrated in our study, age is an independent risk factor predicting unstable VT. In this regard, the loss of elasticity of major arteries with age, decreased beta-adrenergic receptor sensitivity despite arising serum epinephrine levels, decreased end diastolic volume due to decreased venous return and increased frequency of ventricular tachyarrythmias may contribute to the development of increased hemodynamic instability. In this context, our results provide valuable guidance for a clearer understanding of these factors.

Similar to low LVEF, an association was shown between higher NYHA class and more malignant arrythmia and decreased survival in heart failure (26). Major studies have shown that low left ventricular ejection fraction, advanced age and poor NYHA class are clearly associated with mortality in patients with malignant arrhythmias (28). The relationship between the NYHA class and unstable VT in our study appears to be parallel with these data.

One interesting finding in our study is the relationship between hypertension and instability of VT. It has been shown that hypertension increases with age, and elderly hypertensive patients exhibit a higher prevalence of left ventricular hypertrophy, abnormal left ventricular filling pattern and a higher incidence of ventricular arrhythmias in this population (19). Moreover, acute blood pressure elevation is associated with an increase in the frequency of premature ventricular complexes, which are though to be precursors for ventricular tachyarrhythmias, while acute blood pressure decrease is associated with a decrease in the frequency of ventricular complexes; and at the same time the frequency of premature ventricular complexes is decreased with a reduction in the systolic blood pressure obtained in the long-term by oral antihypertensive treatment (29). In hypertensive patients, electrocardiographic changes that may arise from myocardial hypertrophy and remodelling inevitably carry a potential for arrhythmia. It is also known that QT dispersion, which may be associated with ventricular arrhythmia potential (30). Studies have demonstrated the relationship between ECG changes such as strain pattern and fibrosis (31), which should also be considered as an important substrate for ventricular arrhythmias. In our study, the prevalance of hypertension was higher in the group with hemodynamically unstable ventricular tachyarrhytmia compared to the group with hemodynamically stable ventricular tachyarrhytmia. In the light of all these findings, it is not surprising that ventricular arrhythmias increase with hypertension. However, although these data indicate the arryhthmia potential, they do not explain why this patient group is more unstable. In this regard, we believe that our results demonstrating a relationship between hypertension and unstable VT will contribute to further large studies in this respect.

V. CONCLUSION

In this study, we found that patients with EPS-induced unstable VT were those who were older, and hypertensive and had poorer NHYA functional capacity with a lower LVEF and higher MPItri values.

Defining echocardiographic parameters that may predict the risk of ventricular arrhyhtmia instability can theoratically provide valuable information to guide clinicians in planning antriarryhtmic pharmacological treatments and guiding drug switching. Also identifying patients groups that may benefit more significantly from electrophysiological studies and ICD implantation and identifying groups that are not at high risk for hemodynamically unstable ventricular arrhythmias for appropriate ICD programming. For more effective treatment and follow-up of these arrhythmias, our results can serve as a guidance for future studies.

VI. FIGURES AND TABLES

Table 1. A comparison of demographic and clinical data between groups

Demographic and Clinical Data	Stable VT Group (n=48)	Unstable VT Group (n=45)	*p* value
Age (Mean±Std)/year	59.9±12.3	67.0±10.4	**0.033**
Male gender, n (%)	38 (79%)	34 (75%)	0.407
Female gender, n (%)	10 (21%)	11 (25%)	0.882
Height (cm)	167 ± 5	169± 6	0.172

Weight (kg)	72.8 ± 15.2	74.7±16.1	0.226
CH[1], n (%)	27 (%56)	34 (76%)	0.136
HT[2], n (%)	11 (24%)	25 (56%)	**0.021**
DM[3], n (%)	13 (28%)	16 (36%)	0.544
HL[4], n (%)	11 (24%)	14 (32%)	0.269
SD[5], n (%)	27 (56%)	29 (64%)	0.773
NYHA class I/II[6], n (%)	37 (76%)	11 (24%)	**0.010**
NYHA class III[6] n (%)	11 (24%)	34 (76%)	
Beta-Blocker, n (%)	37 (76%)	32 (72%)	1.000
ACE-I/ARB[7], n (%)	17 (36%)	20 (44%)	0.773
Diuretics, n (%)	30 (64%)	40 (88%)	0.098
Spironolactone, n (%)	29 (60%)	23 (52%)	0.776
Amiodarone, n (%)	4 (8%)	13 (28%)	0.138

[1] Cardiac history (significant coronary artery stenosis, previous PCI/CABG), [2]Hypertension, [3] Diabetes mellitus, [4] Hyperlipidemia, [5] Familial history of sudden cardiac death, [6] New York Heart Association functional class, [7] Angiotensin-converting enzyme inhibitors/angiotensin receptor blockers

Table 2. Comparison of Left and Right Heart Two Dimendional and Doppler Echocardiography Findings Between Groups

	Stable VT Group (n=48)	Unstable VT Group (n=45)	p
Two-dimensional echocardiography comparison of left cardiac cavities			
LVEF[1]	36.16±14.65	%29±7.04	**0.039**
LVDSÇ[2]	59.00±10.80	63.00±9.23	0.166
LVSSÇ[3]	46.64±11.33	50.76±9.86	0.177
FS[4]	21±7	19±5	0.324
LVEDV[5]	172.8±54.4	193.5±33.5	0.112
LVESV[6]	114.9±57.1	137.9±31.8	0.087
SV[7]	56.7±15.6	55.2±12.4	0.705
Comparison of Left Heart Doppler Echocardiography Findings			
E[8]	0.74±0.26	0.68±0.22	0.053
OrtEa[9]	0.12±0.11	0.17±0.21	0.072
E/A[10]	1.64±2.03	1.11±0.74	0.479

E/Ea[11]	7.39±2.77	7.93±3.50	0.562
Edes[12]	165.21±32.45	157.30±38.13	0.418
Comparison of Right Heart Doppler Echocardiography Findings			
Etri[13]	0.60±0.20	0.58±0.18	0.775
Atri[14]	0.77±0.25	0.65±0.14	0.115
Etri/Atri[15]	0.89±0.58	0.99±0.43	0.562
MPItri[16]	0.49±0.13	0.56±0.10	**0.027**
TAPSE[17]	14.8±3.79	13.64±4.05	0.301

[1] Left ventricular ejection fraction (%), [2] left ventricular end diastolic diameter, [3] left ventricular end systolic diameter, [4] fractional shortening (%), [5]left ventricular end diastolic volume, [6] left ventricular end systolic volume, [7] stroke volume. [8]mitral early filling velocity (m/sn), [9] mitral annulus mean early filling velocity (m/sn), [10] mitral E/A ratio, [11] mitral E/Ea ratio, [12] mitral early filling deseleration time. [13] tricuspid early filling rate (m/sn), [14] tricuspid late filling rate (m/sn), [15] tricuspid E/A ratio, [16] tricuspid myocardial performance index, [17] tricuspid annular plane systolic excursion.

Table 3. Regression Analysis on Factors Involved in Development of Unstable VT

Variables	HR	95% Confidence Interval	*P*
Age	4.95	1.03-23.69	**0.045**
HT [1] HT (-) (reference) HT (+)	4.16	0.82-21.02	0.085
NYHA class NYHA class I-II (reference) NYHA class III	1.60	0.31-8.06	0.566
LVEF [2] LVEF>35% (reference) LVEF≤35%	0.96	0.89-1.04	0.346
MPItri[3] MPItri<0,55 (reference) MPItri≥0,55	**7.74**	**1.51-39.49**	**0.014**

*Logistic regression was performed by including age, HT, NYHA class, LVEF, and MPItri variables in the model.

** [1] Hypertension, [2] left ventricular ejection fraction, [3] tricuspid myocardial performance index

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