Evaluation of the severity of mitral stenosis with a new index: isovolumic myocardial acceleration

Mitral darlığının ciddiyetinin belirlenmesinde yeni bir indeks: İzovolumik miyokardiyal akselerasyon

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Objectives: Although right ventricular (RV) systolic dysfunction is an important indicator for the severity of mitral stenosis (MS), its diagnosis is difficult before systemic signs of venous congestion occur. We assessed the association between tissue Doppler (TDI)-derived isovolumic myocardial acceleration (IVA) and the severity of MS.

Study design: The study included 112 MS patients (79 mild to moderate, 33 severe MS). Two-dimensional and Doppler echocardiographic parameters (mitral valve area, transmitral diastolic gradients, pulmonary artery pressure, RV fractional shortening, pulmonary flow acceleration time, tricuspid valve annular systolic excursion) were calculated. Additionally, TDI-derived systolic velocities of the tricuspid annulus (IVA, peak myocardial velocity during isovolumic contraction - IVV, peak systolic velocity during ejection period - Sa) were recorded. The results were compared with those of 60 age- and sex-matched healthy controls.

Results: All TDI-derived systolic velocities (IVV, Sa and IVA) were significantly decreased in patients with MS ($p<0.0001$). However, IVA was the only parameter to distinguish the severity of MS ($p<0.0001$). It also showed significant correlations with the following parameters with which IVV and Sa were not correlated: mitral valve area ($r=0.79$, $p<0.0001$), mean ($r=-0.54$, $p<0.0001$) and maximum ($r=-0.58$, $p<0.0001$) transmitral diastolic gradients, pulmonary artery pressure ($r=-0.54$, $p<0.0001$), and left atrial diameter ($r=-0.68$, $p<0.0001$). The ROC curve analysis showed that an IVA of <2.9 m/sec^2 predicted MS patients with 86% sensitivity, 87% specificity, and an IVA of <2 m/sec^2 predicted severe MS with 82% sensitivity and 77% specificity.

Conclusion: Tissue Doppler-derived right ventricular IVA may be used as an adjunctive, alternative noninvasive parameter to determine the severity of MS in patients without signs of systemic venous congestion.

Key words: Blood flow velocity; echocardiography, Doppler; heart ventricles; mitral valve stenosis; myocardial contraction; rheumatic heart disease; ventricular function, right.

Amaç: Sağ ventrikül sistolik disfonksiyonu, mitral darlıklı (MD) hastalarda darlığın ciddiyetinin değerlendirilmesinde önemli bir belirteç olmasına rağmen, sistemik venöz konjesyon bulguları ortaya çıkmadan önce tanısı zordur. Bu çalışmada, doku Doppler (DD) ile ölçülen izovolumik miyokardiyal akselerasyonun MD'nin derecesi ile ilişkisi araştırıldı.

Çalışma planı: Çalışmaya MD'li 112 hasta (79 hafif-orta, 33 ciddi MD) alındı. Tüm hastalarda ikiboyutlu ve Doppler ekokardiyografik parametreler (mitral kapak alanı, transmitral diyastolik gradiyentler, pulmoner arter basıncı, sağ ventrikül fraksiyonel kısalması, pulmoner akım akselerasyon zamanı, triküspid kapağın sistolik annular hareketi) ölçüldü. Ayrıca, doku Doppler ile sağ ventrikül triküspid annular sistolik hızları (IVA, izovolumik kasılma sırasındaki zirve miyokard hızı - IVV, zirve sistolik akım - Sa) ölçüldü. Sonuçlar yaş ve cinsiyet uyumlu 60 sağlıklı gönüllüden oluşan kontrol grubuyla karşılaştırıldı.

Bulgular: Tüm doku Doppler miyokardiyal sistolik hız parametreleri (IVV, Sa, IVA) MD'li hastalarda kontrol grubuna göre anlamlı derecede düşük bulundu ($p<0.0001$). Ancak, IVA hafif-orta ve ciddi MD'li hastaları birbirinden ayırabilen tek parametreydi ($p<0.0001$). Ayrıca, IVV ve Sa'nın ilişkili bulunmadığı şu parametrelerle de anlamlı ilişki gösterdi: Mitral kapak alanı ($r=0.79$, $p<0.0001$), ortalama ($r=-0.54$, $p<0.0001$) ve maksimum ($r=-0.58$, $p<0.0001$) transmitral diyastolik basınç gradiyentleri, pulmoner arter basıncı ($r=-0.54$, $p<0.0001$) ve sol atriyum çapı ($r=-0.68$, $p<0.0001$). ROC eğrisi analizinde, IVA'nın <2.9 m/sn^2 değerinin %86 duyarlılık, %87 özgüllük ile MD'li hastaları ayırt ettiği; <2 m/sn^2 değerinin %82 duyarlılık, %77 özgüllük ile ciddi MD'li olguları saptayabildiği görüldü.

Sonuç: Doku Doppler ile hesaplanan sağ ventrikül kaynaklı IVA, sistemik venöz konjesyon bulguları göstermeyen MD'li hastalarda, darlığın ciddiyetinin belirlenmesinde kullanılabilecek noninvaziv ve güvenilir bir seçenektir.

Anahtar sözcükler: Kan akım hızı; ekokardiyografi, Doppler; kalp ventrikülü; mitral kapağı darlığı; miyokard kontraksiyonu; romatizmal kalp hastalığı; ventrikül fonksiyonu, sağ.

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Right ventricular (RV) function is closely related to symptoms, functional capacity, need and timing for interventions, perioperative mortality, and postoperative results in patients with mitral stenosis (MS). In mild degree of MS, secondary pulmonary hypertension occurs due to reactive changes in pulmonary vascular resistance. Although it is reversible in mild MS, long-standing severe MS is associated with fixed pulmonary arteriolar constriction and obliterative changes in vascular bed, giving rise to significant RV afterload and RV dysfunction. Thus, RV dysfunction is an important indicator to evaluate the severity of MS.[1,2]

Evaluation of RV function by conventional transthoracic echocardiography cannot reliably be made due to its asymmetrical shape, narrow acoustic window, and geometrical assumptions for calculation of volumes.[3,4] The gold standard for quantification of RV contractility is the invasive measurement of maximal systolic elastance by catheterization.[5] Right ventricular ejection fraction (EF) can also be derived by three-dimensional echocardiography and magnetic resonance imaging, but load-dependence of EF limits the utility of these methods.[6]

Although ejection phase myocardial velocities measured by tissue Doppler imaging (TDI) have the potential to assess RV contractile function independent of its shape, they have been shown to be preload- and afterload-dependent.[7-9] Recently, a new TDI-derived index of myocardial acceleration during isovolumic contraction (IVA) has been shown to be a reliable and relatively load-independent measure of RV systolic function.[4]

Considering the hypothesis that RV systolic function is impaired in parallel with the progression of MS, we aimed to evaluate the validity of TDI-derived RV systolic velocities to assess the severity of MS.

PATIENTS AND METHODS

Study population. The study included 79 patients with mild to moderate MS (mitral valve area - MVA >1 cm^2) and 33 patients with severe MS (MVA <1 cm^2). Inclusion criteria were as follows: *(i)* pure mitral stenosis of rheumatic origin, *(ii)* nonexisting or mild mitral insufficiency, *(iii)* absence of concomitant hemodynamically significant valvular disease, *(iv)* TDI-derived good quality echocardiographic imaging allowing measurements of tricuspid annular velocities, *(v)* absence of any disease that could affect myocardial function (e.g. coronary artery disease, chronic lung disease, cardiomyopathies), *(vi)* absence of raised jugular venous pressure, enlarged pulsatile liver, *(vii)* no clinical or laboratory evidence for rheumatic activity for the past six months, and *(viii)* absence of atrioventricular conduction abnormalities and atrial fibrillation.

Sixty age- and sex-matched healthy subjects were also enrolled as the control group.

A considerable amount of data from the same patient group was published previously.[10] The study protocol was approved by local ethics committee of our institution and a detailed written informed consent was obtained from each patient. The study was carried out according to the Declaration of Helsinki.

Conventional two-dimensional Doppler echocardiography. All the patients were examined in the left lateral decubitus position by M-mode, two-dimensional Doppler and TDI echocardiography (Vivid 7, GE Vingmed, Horten, Norway) using a 2.5 MHz transducer. Left atrial diameter was calculated from the parasternal long-axis view by M-mode echocardiography. Tricuspid annular plane systolic excursion (TAPSE, mm) was measured in M-mode using the cursor, in apical four-chamber view, at the junction of the tricuspid valve with the right ventricular free wall. Maximum displacement during systole was evaluated.[11] Right ventricular fractional shortening (RVFS, %) and RV free wall thickness (RVW, mm) in end-diastole were also measured. Mitral valve area was expressed as the mean of two values obtained by planimetric measurement and the pressure half-time method.[12] Maximum and mean transmitral diastolic gradients were calculated by Doppler imaging. Pulmonary artery systolic pressure (PAP, mmHg) was estimated by continuous-wave Doppler imaging using the Bernoulli equation. Pulmonary flow acceleration time was measured as the time from the onset of pulmonary flow to the point of peak velocity by Doppler imaging.[13]

Pulsed Doppler tissue imaging. Guided by the two-dimensional four-chamber view, a 5-mm sample volume was placed on the tricuspid annulus at the place of attachment of the anterior leaflet of the tricuspid valve. Settings were adjusted for a frame rate between 120 and 180 Hz and a cine loop of three to five consecutive heart beats were recorded. Special care was taken to obtain an ultrasound beam parallel to the direction of the tricuspid annular motion. The pulsed-wave TDI-derived systolic indices, peak myocardial velocity during isovolumic contraction (IVV, cm/sec), myocardial acceleration during isovolumic contraction (IVA, m/sec^2; defined as the ratio of IVV divided by the acceleration time), and peak velocity during

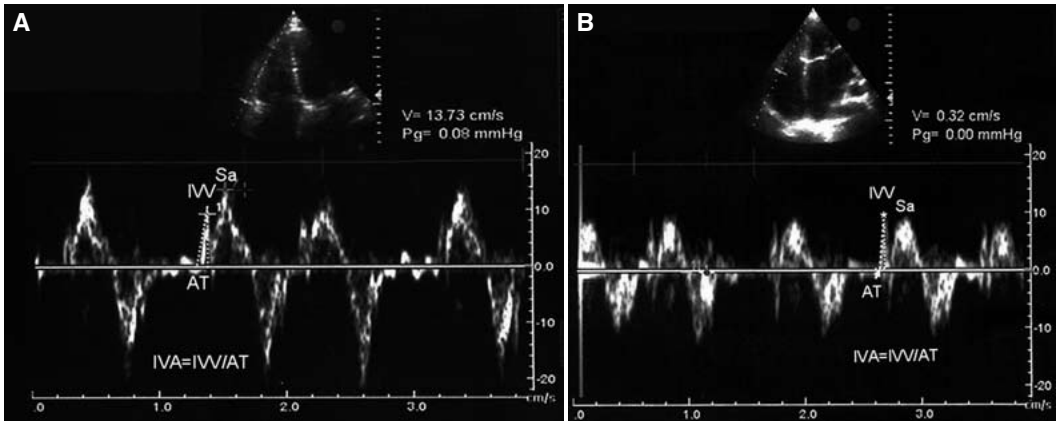

Figure 1. Tissue Doppler-derived myocardial systolic velocities obtained from the tricuspid lateral annulus. IVV, Sa and IVA measurements of two patients **(A)** with and **(B)** without mitral stenosis. IVV: Peak myocardial velocity during isovolumic contraction; Sa: Peak velocity during ejection period of systole; AT: Acceleration time; IVA: Myocardial acceleration during isovolumic contraction.

systolic ejection (Sa, cm/sec) were measured. All the measurements were calculated from three consecutive cycles and the average of three measurements was recorded (Fig. 1).

Reproducibility. Intraobserver and interobserver variabilities of TDI-derived tricuspid lateral annulus systolic velocities were assessed. For interobserver variability, a second observer calculated 20 measurements, and for intraobserver variability, the first observer repeated 20 measurements on another day.

Statistical analysis. All statistical data were processed using the GraphPad Prism V.3 statistical package. The results were expressed as mean and standard deviation (SD). One-way ANOVA analysis was used for comparisons of the groups. The Tukey multiple comparison test was used for comparison of subgroups. The study and control groups were compared using unpaired t-test. The cutoff point of the variable IVA in patients with mild to moderate and severe MS was analyzed in relation to the control group. Sensitivity, specificity, positive and negative predictive values, accuracy, and relative risk values were also calculated. Univariate correlations were sought using the Pearson analysis. Multiple linear regression analysis was used to determine independent factors affecting IVA. The results were considered significant when the *p* value was less than 0.05.

RESULTS

Clinical characteristics. Age, gender, body mass index, and heart rate were similar in both the study and control groups (Table 1).

Echocardiographic parameters. Left atrial diameter, diastolic transmitral gradients (mean/maximum), and estimated pulmonary artery pressure were sig-

Table 1. Clinical characteristics and conventional echocardiographic parameters of the study and control groups

	Mitral stenosis				
	Mild-moderate (n=79)	Severe (n=33)	*p*	Controls (n=60)	*p*
Age (years)	48±10	51±10	0.82	50±11	0.46
Gender (female)	55 (69.6%)	24 (72.7%)	0.75	44 (73.3%)	0.88
Body mass index (kg/m^2)	24±3	23±3	0.72	24±3	0.85
Mitral regurgitation (mild)	24 (30.4%)	10 (30.3%)	0.96	–	
Transmitral diastolic gradient (mmHg)					
Maximum	8.8±3.9	21.9±4.7	<0.0001	–	
Mean	4.1±2.3	11.8±3.2	<0.0001	–	
Mitral valve area (cm^2)	1.7±0.2	0.9±0.2	<0.0001	3.6+1.6	<0.0001
Left atrial diameter (cm)	3.8±0.2	4.5±0.7	<0.0001	3.6±0.3	<0.0001
Systolic pulmonary artery pressure (mmHg)	38.0±9.1	47.5±9.8	<0.0001	24.6±3.3	<0.0001
Right ventricle fractional shortening (%)	55.7±3.6	54.6±3.6	0.39	56.5±4.4	0.14
Right ventricle anterior wall thickness (mm)	2.7±0.4	3.2±0.4	0.78	2.3±0.2	<0.0001
Pulmonary flow acceleration time (msec)	109.1±5.5	109.9±7.5	0.92	128.5±5.1	<0.0001
Tricuspid annulus movement (mm)	20.9±2.9	20.8±2.7	0.89	21.7±2.3	0.17

Table 2. Tissue Doppler-derived myocardial systolic velocities obtained from the tricuspid lateral annulus

	Mitral stenosis				
	Mild-moderate (n=79)	Severe (n=33)	*p*	Controls (n=60)	*p*
Right ventricular Sa (cm/sec)	0.14±0.03	0.13±0.03	0.19	0.19±0.02	<0.0001
Right ventricular IVV (cm/sec)	0.12±0.04	0.11±0.03	0.22	0.15±0.02	<0.0001
Right ventricular IVA (m/sec^2)	2.34±0.45	1.68±0.55	<0.0001	3.21±0.29	<0.0001

IVV: Peak myocardial velocity during isovolumic contraction; Sa: Peak velocity during ejection period of systole; IVA: Myocardial acceleration during isovolumic contraction.

nificantly higher in patients with severe MS. End-diastolic thickness of RVW was significantly greater in patients with severe MS (p<0.0001), but the two MS groups did not differ in this respect (p=0.78). Pulmonary flow acceleration time was markedly reduced in the patient group (p<0.0001), while there was no significant difference between the two MS groups (p=0.92). Tricuspid annular plane systolic excursion was found to be lower in patients with MS, but this was not significant (p=0.17). Right ventricular fractional shortening was also similar in the patient and control groups (p=0.14). Mild mitral regurgitation was detected in 24 patients with mild to moderate MS and in 10 patients with severe MS (Table 1).

Tissue Doppler imaging findings. All TDI-derived tricuspid annular systolic velocities were significantly decreased in MS patients compared to the control group (Table 2). However, in subgroup analyses, the two MS groups did not differ significantly with respect to tricuspid annulus Sa wave (p=0.19) and right ventricular IVV (p=0.22), whereas IVA was markedly lower in patients with severe MS (p<0.0001; Table 2).

Correlation analyses between both traditional and TDI-derived RV systolic indices and the degree of mitral stenosis. Among the traditional parameters of RV systolic function, only left atrial diameter (r=-0.54, p<0.0001) and pulmonary artery pressure (r=-0.64, p<0.0001) showed a significant correlation with the degree of MS. Pulmonary flow acceleration time, TAPSE, and RVFS were not correlated with mitral valve area and transmitral diastolic gradients (Table 3).

Among TDI-derived tricuspid annular systolic velocities, only IVA showed significant correlations with the degree of MS, being in positive correlation with MVA (r=0.79, p<0.0001), and in negative correlation with the mean (r= -0.54, p<0.0001) and maximum (r= -0.58, p<0.0001) transmitral diastolic gradients (Table 3). In addition, IVA was the only parameter correlated significantly with PAP (r=-0.54, p<0.0001) and LA diameter (r=-0.68, p<0.0001).

Additionally, consistent with previous studies, IVA showed significant correlations with pulmonary flow acceleration time (r=0.39; p=0.0001), RVW (r= -0.27, p=0.004), and RV Tei index (r=-0.813, p<0.0001).

On the other hand, IVA was not correlated with Sa (r=0.158, p=0.097), TAPSE (r=-0.03, p=0.73), and RVFS (r=-0.09; p=0.31).

Receiver operating characteristic (ROC) curve analyses for IVA. The overall mean MVA of MS patients was 1.0 cm^2, being ≥1 cm^2 in 72 patients, and <1 cm^2 in 40 patients. The ROC curve analysis in MS

Table 3. Correlations between right ventricular systolic indices and the degree of mitral stenosis

	Mitral valve area		Mean transmitral diastolic gradient		Maximum transmitral diastolic gradient	
	r	*p*	r	*p*	r	*p*
Left atrium diameter	-0.54	**<0.0001**	0.44	**<0.0001**	0.44	**<0.0001**
Right ventricle fractional shortening	0.12	0.23	-0.09	0.37	-0.06	0.50
Pulmonary flow acceleration time	0.09	0.35	0.05	0.58	0.06	0.52
Tricuspid annular motion (mm)	0.03	0.74	0.06	0.54	0.03	0.77
Pulmonary artery pressure	-0.64	**<0.0001**	0.38	**<0.0001**	0.39	**<0.0001**
Tissue Doppler						
Right ventricular Sa	0.12	0.21	-0.09	0.35	-0.08	0.38
Right ventricular IVV	0.13	0.23	-0.15	0.12	-0.16	0.11
Right ventricular IVA	0.79	**<0.0001**	-0.54	**<0.0001**	-0.58	**<0.0001**

IVV: Peak myocardial velocity during isovolumic contraction; Sa: Peak velocity during ejection period of systole; IVA: Myocardial acceleration during isovolumic contraction.

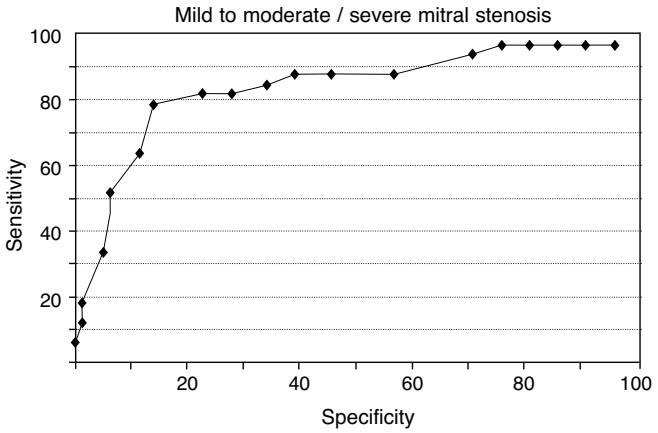

Figure 2. The receiver operating characteristics curve demonstrating the ability of IVA (m/sec²) to predict a mitral valve area of <1 cm². The optimal discriminatory cut point corresponding to an IVA value of 2 m/sec² has 82% sensitivity and 77% specificity.

and control groups showed that IVA of <2.9 m/sec² predicted MS patients with sensitivity, specificity, positive and negative predictive rates of 86%, 87%, 89%, and 83%, respectively. The IVA was <2.9 m/sec² in 89.9% of patients with mild to moderate MS (MVA ≥1 cm²). Therefore, the ROC curve demonstrated a good discrimination between the patients with mild to moderate and severe MS. On the other hand, an IVA value of less than 2 m/sec² predicted severe MS with 82% sensitivity, 77% specificity, 55% positive and 90% negative predictive rates (Fig. 2).

In univariate analysis, age, gender, and PAP were found to be significant parameters affecting IVA ($p<0.0001$). In multiple linear regression analysis, age was found to be the most important parameter responsible for the change in IVA ($\beta= -0.46$, $p=0.018$).

Reproducibility. Interobserver and intraobserver reliability coefficients were good for IVA ($r=0.96$ and $r=0.93$, respectively). The mean interobserver and intraobserver differences for IVA were 0.01±0.20 m/sec² and 0.05±0.22 m/sec², respectively.

DISCUSSION

Determining the severity of MS is important to identify the treatment options and the timing for intervention. A careful clinic evaluation and noninvasive assessment by two-dimensional and Doppler echocardiography usually provide sufficient information in the majority of patients. Cardiac catheterization is recommended in cases in which discrepancy exists between symptoms and hemodynamic data.[14]

M-mode, two-dimensional and Doppler echocardiography is usually used for determination of the severity of MS. However, there are some difficulties with all these conventional methods. M-mode echocardiography may not provide reliable data regarding the actual restrictive orifice.[15] By two-dimensional echocardiography, the stenotic mitral orifice area can accurately be planimetered, which correlates well with hemodynamic data in patients with relatively symmetric involvement. However, asymmetric involvement or irregular orifice shape due to commissurotomy may limit the accuracy of the measurements by planimetry.[16]

Doppler echocardiography is often used to assess the transvalvular gradient from the left atrium to left ventricle, which is known as the most important factor in determining the severity of MS.[17] However, the pressure gradient may be affected by both volume status and heart rate of the patient. Additionally, it has been shown that the value of the anatomic area derived from the pressure half-time calculation is less compared to that of determining pressure gradients and anatomically measured areas.[18] These problems with conventional echocardiographic parameters caused a tendency to search for new modalities in evaluating the severity of MS. Recently, some studies suggested three-dimensional echocardiography and multislice computed tomography as an alternative to assess the severity of the disease.[19,20]

The indication for intervention, either percutaneous or surgical, depends on symptoms, pulmonary artery pressure, and RV function in patients with MS.[9] It is more complicating to decide the need and timing for intervention in MS patients without clinical signs of systemic venous congestion, because RV functions may be impaired before the appearance of clinical signs. Therefore, evaluation of RV systolic function is important in this group of patients. Assessment of RV function is difficult due to its asymmetrical shape and narrow acoustic window. In our study, we used TDI-derived right ventricular IVA. It is a new parameter and has been validated to be a reliable and relatively load-independent measure of RV systolic function.[21,22] The main finding of our study is the evidence for its clinical use in assessing RV systolic function to determine the severity of MS. In many studies, Sa has also been shown to reflect RV systolic function. This parameter was found to have a very good correlation with RV fractional area and RVEF assessed by radionuclide ventriculography.[23] However, Sa is significantly afterload-dependent,[7] whereas IVA reflects RV systolic function during isovolumic contraction. In contrast to Sa, IVA has the

advantage of being relatively preload- and afterload-independent. This parameter has been successfully validated by both experimental and clinical studies. Vogel et al.[5] demonstrated that IVA was an accurate parameter to assess RV systolic dysfunction and was able to measure the force-frequency relation. Pauliks et al.[21] reported that all systolic velocities except for IVA decreased due to load changes during closure of atrial septal defects. Harada et al.[22] showed that Sa was lower in patients after repair of tetralogy of Fallot, compared to the control group. In another study, Toyono et al.[23] reported decreased RV myocardial velocities and IVA after repair of tetralogy of Fallot.

Supporting these results is the demonstration of significant decreases in all TDI-derived tricuspid annular myocardial velocities (Sa, IVV, and IVA) in patients with MS, compared to age- and sex-matched controls ($p<0.0001$). However, Sa and IVV values showed no significant differences between patients with mild to moderate MS and severe MS, whereas IVA was the only parameter that showed a significant decrease in patients with severe MS. This result may be explained by its independency from afterload and preload changes.

We found that IVA of <2.9 m/sec^2 predicted MS patients with 86% sensitivity and 87% specificity. Additionally, our results demonstrated that a cutoff point of 2 m/sec^2 could be used for the prediction of severity in patients with rheumatic MS. Moreover, our results showed a significant positive correlation between IVA and MVA ($p<0.0001$), whereas no correlation existed for Sa and IVV. Similarly, of the three TDI-derived parameters, only IVA was inversely correlated with maximum and mean transmitral diastolic gradients, PAP, and left atrial diameter ($p<0.0001$).

The relationship between traditional parameters for right ventricular systolic function and degree of MS was also analyzed in our study. A previous study showed a significant correlation between TAPSE and right ventricular contractility,[24] but we could not find any relationship between these parameters. Due to similar results obtained in all the groups, RVFS does not seem to be a valuable parameter to evaluate RV function in patients with MS.

We also analyzed pulmonary flow acceleration time which is another useful parameter to evaluate RV systolic function. It was markedly reduced in the patient group, with very similar values in the two MS groups. This may be explained by increased pulmonary vascular resistance in patients with MS. Pulmonary flow acceleration time, in addition, was not correlated with MVA and transmitral diastolic gradients. On the other hand, RVW thickness was not found to be helpful in distinguishing patients with respect to the severity of MS.

Limitations of the study. We did not compare our results with invasive parameters and findings of other new modalities such as three-dimensional echocardiography and multislice computed tomography. Further studies that would present comparative results with new diagnostic modalities are needed to evaluate the diagnostic value of RV IVA in patients with MS.

In conclusion, TDI-derived RV IVA seems to be a quantitative, reproducible, and noninvasive method to determine the severity of MS. It may be used as an adjunctive, alternative noninvasive parameter with definite cutoff points to determine the severity of MS, helping decide the indication and timing for intervention especially in cases in which signs of systemic venous congestion have not yet appeared.

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