Early Impairment of Right Ventricular Functions in Patients with Moderate Asthma and the Role of Isovolumic Acceleration

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ABSTRACT

Introduction: Asthma is a common chronic lung disease that affects people all over the world. Pulmonary hypertension and right ventricular (RV) dysfunction are possible complications that may develop in the advanced stages of asthma. However, the number of studies investigating asthma and its implications on new RV parameters are very rare. This study aims to evaluate the RV functions in patients with moderate asthma before the development of pulmonary hypertension.

Patients and Methods: Forty-one patients with moderate asthma and 40 healthy individuals were enrolled in this case-control study. All participants underwent a detailed two-dimensional echocardiographic examination. RV functions were measured through RV isovolumic acceleration (IVA) index in addition to conventional parameters. RV IVA, a tissue doppler derived parameter, was calculated as the ratio between maximum isovolumic myocardial velocity during isovolumic contraction and the time interval from the onset of this wave to the time at its maximum velocity.

Results: There were no significant differences between the two groups in terms of baseline clinical characteristics, laboratory findings and echocardiographic parameters measuring left ventricular functions ($p > 0.05$). In asthmatic patients, RV isovolumic relaxation time and RV myocardial performance index were higher ($p = 0.027$ and $p < 0.001$ respectively), while RV fractional area change, tricuspid annular plane systolic excursion (TAPSE) and RV IVA values were all lower ($p < 0.001$). RV IVA was found to be inversely proportional to asthma duration. TAPSE [$\beta = 0.632$, 95% CI= (0.121) - (0.225), $p < 0.001$] and pulmonary artery systolic pressure [$\beta = -0.188$, 95% CI= (-0.057) - (-0.003), $p = 0.032$] were shown as independent predictors of RV IVA.

Conclusion: Asthma is an important disease that may result in subclinical RV dysfunction even before the development of pulmonary hypertension. RV IVA, an easily obtained and load-independent parameter, may be a useful and reliable index that sensitively analyzes subtle deteriorations in the contractile function of RV in asthmatic patients. RV IVA may also correlate with asthma duration.

Key Words: Asthma; echocardiography; ventricular function; right ventricular dysfunction; pulmonary hypertension

Orta Şiddette Astımı Olan Hastalarda Sağ Ventrikül Fonksiyonlarındaki Erken Bozulma ve İzovolumik Akselerasyonun Rolü

ÖZET

Giriş: Astım, dünya genelindeki insanları etkileyen ve sık görülen bir kronik akciğer hastalığıdır. Pulmoner hipertansiyon ve sağ ventrikül işlev bozukluğuysa astımın ileri evrelerinde görülebilen olası komplikasyonlardır. Ancak, astımın yeni sağ ventrikül parametrelerle ilgili çalışma sayısı oldukça azdır. Bu çalışma, henüz pulmoner hipertansiyon gelişmemiş orta şiddetli astımı olan hastalarda sağ ventrikül işlevlerini değerlendirmeyi amaçlamaktadır.

Hastalar ve Yöntem: Bu vaka-kontrol çalışmasına, orta şiddetli astımı olan 41 hasta ile 40 sağlıklı birey alınmıştır. Katılımcıların tümüne ayrıntılı iki boyutlu ekokardiyografi uygulanmıştır. Sağ ventrikül işlevi, alışılagelmiş ekokardiyografi parametrelerinin yanı sıra sağ ventrikül izovolumik akselerasyon (IVA) indeksiyle de ölçülmüştür. Doku doppler ekokardiyografiyle ölçülen bir parametre olan sağ ventrikül IVA, izovolumik kasılma sırasındaki en yüksek izovolumik miyokardiyal hızın, bu dalganın başlangıcının en yüksek hızına ulaştığı noktaya kadar geçen zaman aralığına oranı olarak hesaplanmıştır.

Bulgular: Gruplar arasında temel demografik özellikler, laboratuvar bulguları ve sol ventrikül sistolik işlevini ölçen ekokardiyografi parametreleri bakımından anlamlı bir farklılık yoktu ($p > 0.05$). Astımlı hastalarda, sağ ventrikül izovolumik gevşeme ve sağ ventrikül miyokard performans indeksi daha yüksekken ($p = 0.027$ ve $p < 0.001$, sırasıyla), sağ ventrikül fraksiyonel alan değişimi, triküspid kapağın anüler planda sistolik yer değiştirmesi (TAPSE) ve sağ ventrikül IVA değerleri daha düşüktü ($p < 0.001$). Sağ ventrikül IVA, astım süresiyle ters orantılı bulundu. TAPSE [$\beta = 0.632$, 95% CI= (0.121) - (0.225), $p < 0.001$] ve pulmoner arter sistolik basıncı [$\beta = -0.188$, 95% CI= (-0.057) - (-0.003), $p = 0.032$] sağ ventrikül IVA'nın bağımsız belirleyicileri olarak gösterildi.

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Sonuç: Astım, pulmoner hipertansiyon gelişmeden önce bile, sağ ventrikül işlev bozukluğuna yol açabilen önemli bir hastalıktır. Kolayca ölçülen ve yükten bağımsız bir parametre olan sağ ventrikül IVA, astımlı hastalarda, sağ ventrikül kasılma fonksiyonlarındaki ufak bozulmaları bile duyarlıca analiz edebilecek, kullanışlı ve güvenilir bir indeks olabilir. Sağ ventrikül IVA, ayrıca astım süresi ile korelasyon gösterebilmektedir.

Anahtar Kelimeler: Astım; ekokardiyografi; ventriküler fonksiyon; sağ ventriküler disfonksiyon; pulmoner hipertansiyon

INTRODUCTION

It is widely known that pulmonary hypertension and subsequent right ventricle (RV) dysfunction are common cardiac complications of chronic obstructive pulmonary disease and diffuse parenchymal lung disorders, both of which are characterized by hypoxia and chronic inflammatory alterations[1,2]. Asthma, on the other hand, is a fast-rising chronic respiratory condition that affects up to 18% of the population in some countries. It's a public health issue characterized by airway inflammation and recurrent attacks that can lead to hypoxemia[3,4]. Despite its frequency, inflammatory and hypoxic nature, studies exploring the detrimental effects of asthma on the pulmonary artery or RV are extremely rare and mainly limited to severe cases[5].

RV is the region that is sensitive to pressure changes in the pulmonary vasculature and lungs. Its prognostic importance in health and disease states was neglected for decades. However, in recent years, growing evidence accelerated our understanding of the crucial role of RV in cardiopulmonary diseases[6-8]. Under normal circumstances, RV acts in an adaptive manner to the pressure changes in the pulmonary circulation (RV afterload) in order to ensure the blood flow through the pulmonary arteries. In the clinical setting, however, if the compensatory mechanisms fail to overcome the alterations in RV afterload, RV dysfunction and subsequent right heart failure may emerge[7,9].

Therefore, evaluating RV functions is critical in identifying the functional state and prognosis of patients with chronic pulmonary diseases. For the assessment of RV structure and functions, two-dimensional transthoracic echocardiography has become the mainstay technique due to its immediate, easily applicable, and safe nature. Quantification of RV functions is based on commonly used conventional echocardiographic parameters each of which has some limitations with respect to the complex ventricular geometry and loading conditions[10]. However, isovolumic acceleration (IVA) is a reliable echocardiographic parameter that is independent of RV shape and loading conditions. Many clinical studies confirmed the validity and utility of IVA in predicting early RV impairment by demonstrating its role even similar to RV strain[11,12]. To our knowledge, there have been relatively few studies examining RV function in patients with non-severe asthma, and there has been no previous work analyzing RV IVA in those patients[13,14].

PATIENTS and METHODS

This prospective case-control study was conducted in outpatient cardiology clinics of our hospital between December 2019 and February 2020. Forty-one patients with moderate asthma were enrolled from our pulmonary diseases outpatient clinic for the case group. The control group consisted of 40 subjects who were age, gender, and BMI-matched with the case group. A pulmonary specialist diagnosed asthma based on pulmonary function test results, symptoms, and clinical findings described in the Global Initiative for Asthma Guidelines[3]. A comprehensive cardiovascular examination, pulmonary function test, two-dimensional transthoracic echocardiography, complete blood count, and routine biochemical blood analysis were performed for each individual. The patients with asthma were further grouped as "<10 years (Group 1) and ≥10 years (Group 2)" according to the duration of asthma.

Exclusion criteria included the following: Being under the age of 18 and over the age of 65, history of an acute asthma attack within the last four weeks, presence of chronic lung disease rather than asthma, collagen vascular disease, pulmonary hypertension, coronary artery disease, moderate to severe valvular heart disease, heart failure, hypertension, obesity, diabetes mellitus, smoking, active infection, pregnancy, malignancy, chronic kidney disease, chronic liver disease, cerebrovascular disease, peripheral artery disease, and suboptimal echogenicity. The same technician administered a pulmonary function test to each participant in a sitting position, using the criteria described in the European Respiratory Society consensus standards[15]. Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), forced expiratory volume in one second/forced vital capacity ratio (FEV_1/FVC) and peak expiratory flow (PEF) were recorded. The study was conducted in accordance with the Declaration of Helsinki and approved by the institutional ethics committee. Written informed consent was obtained from each individual.

Standard Echocardiography and Isovolumic Acceleration

Echocardiographic assessments were performed by an experienced cardiologist who was blinded to the clinical status of the participants. The Philips HD11XE (Andover, US) device was used to evaluate the patients as they were laying in the left lateral decubitus position. Three cardiac cycles were obtained

for the measurement of each echocardiographic parameter during simultaneous single derivation electrocardiographic recording. Parasternal long axis, short axis, apical four-chamber, and two-chamber images were acquired and assessed using M-mode, 2-D, continuous-wave Doppler, and tissue Doppler imaging methods in accordance with the American Society of Echocardiography criteria[16]. Left ventricle (LV) wall dimensions and cavity diameters were measured. LV ejection fraction was determined using Simpson's method. Early (E), late (A) mitral and tricuspid flow velocities, E/A ratio, and deceleration time were calculated. Systolic (S'), early diastolic (E'), and late diastolic (A') velocities were recorded on the septum, lateral wall, and tricuspid annulus by tissue doppler imaging. Isovolumic relaxation time (IVRT), isovolumic contraction time (IVCT), acceleration time, and ejection time (ET) of both ventricles were measured to obtain the myocardial performance index (MPI) using (IVRT + IVCT)/ET formula. RV fractional area change (RV FAC), tricuspid annular plane systolic excursion (TAPSE), and pulmonary artery systolic pressure (PASP) measurements were also obtained.

IVA was obtained using tissue Doppler imaging with the pulsed Doppler sample volume placed in the basal segment of the right ventricular free wall in an apical four-chamber view. It was calculated as the ratio between maximum isovolumic myocardial velocity during isovolumic contraction and the time interval from the onset of this wave to the time at its maximum velocity[10].

Statistical Analysis

Categorical variables were described as frequency and percentage. Continuous variables with normal distribution were expressed as mean ± standard deviation while continuous variables with non-normal distribution were given as median and interquartile range (IQR, range from the 25th percentile to the 75th percentile). The Kolmogorov-Smirnov test was used to verify the normality of the distribution of continuous variables. χ^2 test, independent samples t-test, and Mann-Whitney U test were used for inter-group comparisons. Spearman correlation analysis was performed to analyze the correlation between IVA and other parameters. Multilinear regression analysis was applied to reveal the independent relationship between IVA and the parameters that were significantly correlated with IVA. Receiver operating characteristic (ROC) curve analysis was used to determine the sensitivity, specificity, and cut-off values of IVA in relation with asthma. Two-tailed $p< 0.05$ was considered to be significant. Statistical analysis was performed using SPSS statistical package software (version 20.0, SPSS, Chicago, IL, USA).

RESULTS

The baseline demographic characteristics, laboratory parameters and pulmonary function test results are presented in Table 1. The study group consisted of 41 patients (23 female, 56%; age= 49.83 ± 9.39) while the control group included 40 individuals (23 female, 57.5%; age= 47.98 ± 7.85). The groups were similar in terms of age, sex, body mass index, systolic

Table 1. Baseline demographic, clinical characteristics, laboratory findings, and pulmonary function test results of the study population

	Patients (n= 41)	Controls (n= 40)	p
Age (years)	49.83 ± 9.39	47.98 ± 7.85	0.339
Female, n (%)	23 (56)	23 (57.5)	0.899
BMI (kg/m^2)	27.52 ± 2.14	26.77 ± 1.69	0.086
SBP (mmHg)	122.73 ± 5.47	121.65 ± 5.77	0.389
DBP (mmHg)	79.51 ± 8.54	81.80 ± 6.70	0.185
HR (beats/minute)	78.59 ± 7.94	76.83 ± 7.66	0.313
Leucocytes (G/I)	6.42 ± 1.55	6.81 ± 1.29	0.224
Hemoglobin (g/dL)	14.46 ± 0.71	14.17 ± 0.92	0.112
FEV_1	85 (81.00-89.50)	92 (89.00-97.50)	<0.001*
FVC	83.22 ± 5.43	89.20 ± 5.53	<0.001*
FEV_1/FVC	84.88 ± 7.50	96.05 ± 8.67	<0.001*
PEF	81.73 ± 5.37	84.90 ± 6.04	0.015*

BMI: Body mass index, DBP: Diastolic blood pressure, FEV_1: Forced expiratory volume in 1 second, FVC: Forced vital capacity, HR: Heart rate, PEF: Peak expiratory flow, SBP: Systolic blood pressure; Values are presented as mean ± SD, median (range from the 25th percentile to the 75th percentile) or number and percentage.
*: Indicates significance.

blood pressure, diastolic blood pressure, heart rate, hemoglobin level, and leucocyte count (p> 0.05) while FEV_1, FVC, FEV_1/FVC, and PEF values were found to be significantly lower in asthmatic patients (p< 0.001, p< 0.001, p< 0.001 and p= 0.015, respectively).

The findings of the echocardiographic evaluation are demonstrated in Table 2. LV end-diastolic dimension (LVEDD), LV ejection fraction (LVEF), interventricular septum (IVS) thickness, LV posterior wall (LVPW) thickness, mitral early diastolic flow (E), mitral late diastolic flow (A), mitral E/A ratio, deceleration time (DT), IVRT, LV MPI, LV lateral wall early myocardial diastolic velocity (LV E'), LV lateral wall late myocardial diastolic velocity (LV A') and LV lateral wall myocardial systolic velocity (LV S') did not differ between two groups (p> 0.005).

The following RV structural and function-related findings were discovered: Tricuspid early diastolic flow (E), tricuspid late diastolic flow (A), tricuspid E/A ratio, RV ESA, RV A', RV S' and PASP were found to be similar between two groups (p> 0.05). RV IVRT and RV MPI were significantly higher in asthmatic patients (p= 0.027 and p< 0.001 respectively) while

Table 2. Comparison of echocardiographic parameters in the patient and control groups

	Patients (n= 41)	Controls (n= 40)	p
LVEDD (mm)	4.8 (4.50-4.90)	4.6 (4.20-4.88)	0.100
LVESD (mm)	3.12 ± 0.29	3.07 ± 0.28	0.510
IVS (mm)	0.96 ± 0.10	0.99 ± 0.07	0.116
LVpw (mm)	0.93 (0.87-1.02)	0.95 (0.88-1.01)	0.925
LVEF (%)	62.25 ± 3.72	61.19 ± 3.43	0.188
Mitral E (cm/s)	91.18 ± 10.00	87.69 ± 9.14	0.106
Mitral E/A	1.06 ± 0.13	1.03 ± 0.11	0.244
DT (ms)	188.17 ± 10.43	189.53 ± 10.73	0.566
LV IVRT (ms)	88.89 ± 8.31	87.43 ± 7.95	0.424
LV E' (cm/s)	14.19 ± 1.48	13.81 ± 1.28	0.217
LV A' (cm/s)	10.76 ± 1.21	11.15 ± 1.27	0.157
LV S' (cm/s)	9.73 ± 1.18	10.09 ± 1.43	0.220
LV MPI	0.40 ± 0.02	0.39 ± 0.02	0.741
RV EDA (cm^2)	12.90 ± 2.04	14.14 ± 1.41	0.002*
RV FAC (%)	38.09 ± 4.83	41.53 ± 3.45	<0.001*
Tricuspid E (cm/s)	65.21 ± 5.34	66.09 ± 4.24	0.416
Tricuspid E/A	1.07 ± 0.10	1.09 ± 0.09	0.416
RV IVRT (ms)	53.00 ± 9.24	48.20 ± 9.91	0.027*
RV E' (cm/s)	12.43 ± 1.30	13.18 ± 1.02	0.005*
RV A' (cm/s)	11.45 ± 0.98	11.67 ± 1.20	0.371
RV S' (cm/s)	12.80 ± 1.07	13.00 ± 1.07	0.403
RV MPI	0.41 ± 0.02	0.37 ± 0.02	<0.001*
TAPSE (mm)	16.04 ± 0.92	17.84 ± 1.23	<0.001*
PASP (mmHg)	20.00 (19.00-20.75)	19 (18.58-20.00)	0.062
RV IVA	2.04 ± 0.30	2.37 ± 0.39	<0.001*

LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, IVS: Interventricular septum, LVpw: Left ventricular posterior wall, LVEF: Left ventricular ejection fraction, DT: Deceleration time, IVRT: Isovolumic relaxation time, E': Early myocardial diastolic velocity, A': Late myocardial diastolic velocity, S': Systolic myocardial velocity, RV MPI: Right ventricular myocardial performance index, RV EDA: Right ventricular end-diastolic area, FAC: Fractional area change, TAPSE: Tricuspid annular plane systolic excursion, PASP: Pulmonary artery systolic pressure; Values are presented as mean ± SD or median (range from the 25^{th} percentile to the 75^{th} percentile).

*: Indicates significance.

Table 3. Multilinear regression analysis of RV IVA for each parameter that was significantly correlated with IVA

	RV IVA			
	Univariate		Multivariate	
	β	p	β	p
FEV_1/FVC	0.192	0.086	0.109	0.213
TAPSE	0.698	<0.001	0.632	<0.001*
RV MPI	-0.495	<0.001	-0.109	0.291
PASP	-0.387	<0.001	-0.188	0.032*

RV IVA: Right ventricular isovolumic acceleration, FEV_1/FVC: Forced expiratory volume in 1 second/forced vital capacity ratio, TAPSE: Tricuspid annular plane systolic excursion, RV MPI: Right ventricular myocardial performance index, PASP: Pulmonary artery systolic pressure.
*: Indicates significance.

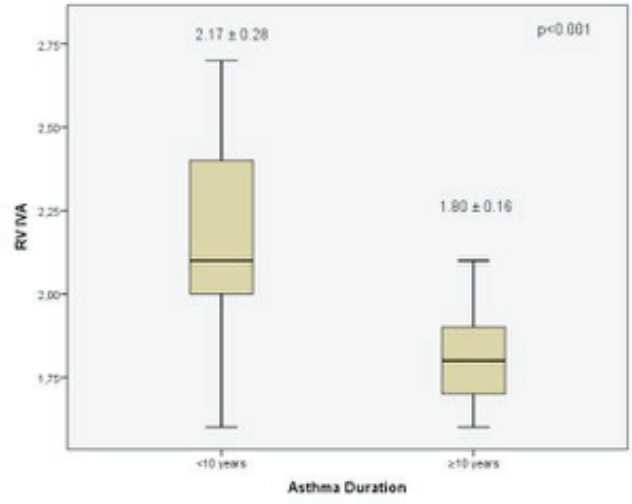

Figure 1. Box plot graph showing the RV IVA in Group 1 (asthma duration <10 years) and Group 2 (asthma duration ≥10 years). RV IVA: Right ventricle isovolumic acceleration.

RV E', RV EDA, RV FAC, TAPSE and RV IVA values were significantly lower in asthmatic patients (p= 0.005, p= 0.002, p< 0.001, p< 0.001, p< 0.001, respectively).

RV IVA was found to be significantly lower in asthmatic patients who had an asthma duration of more than 10 years (Group 2) compared to the asthmatic patients who had an asthma duration of less than 10 years (Group 1), revealing an inverse relationship with asthma duration [2.17 ± 0.28 (Group 1) vs. 1.80 ± 0.16 (Group 2), p< 0.001 (Figure 1)].

In correlation analysis, RV IVA was positively correlated with FEV_1/FVC (r= 0.238, p= 0.033) and TAPSE (r= 0.735, p< 0.001), and negatively correlated with RV MPI (r= -0.455, p< 0.001) and PASP (r= -508, p< 0.001) (Figure 2). When the parameters correlated with RV IVA were further entered into multilinear regression analysis, TAPSE [β= 0.632, 95% CI= (0.121) - (0.225), p< 0.001] and PASP [β= -0.188, 95% CI= (-0.057) - (-0.003), p= 0.032] were determined as the independent predictors of RV IVA.

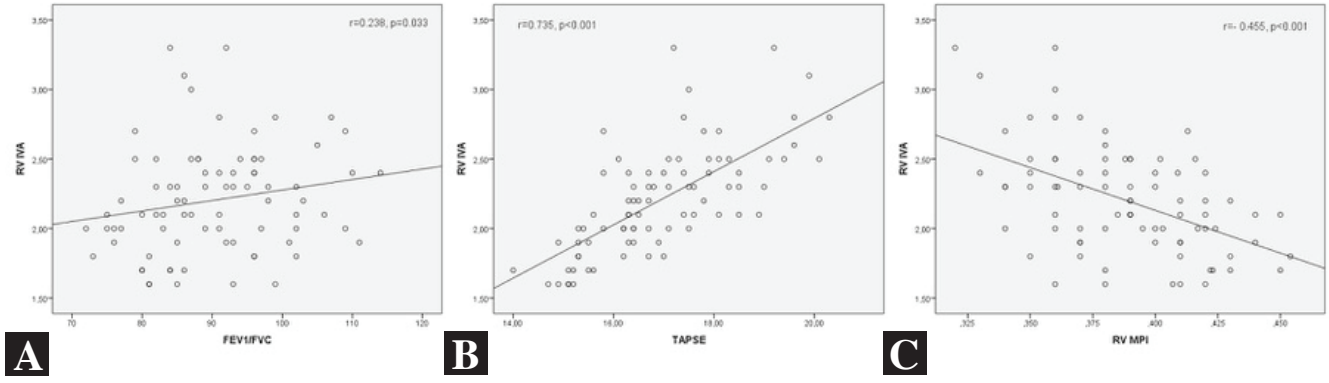

Figure 2. Scatter dot plot graphs revealing correlation analysis between **(A)** RV IVA and FEV_1/FVC, **(B)** RV IVA and TAPSE, **(C)** RV IVA and RV MPI. RV IVA: Right ventricle isovolumic acceleration, TAPSE: Tricuspid annular plane systolic excursion, FEV_1/FVC: Forced expiratory volume in one second/forced vital capacity ratio, RV MPI: Right ventricle myocardial performance index.

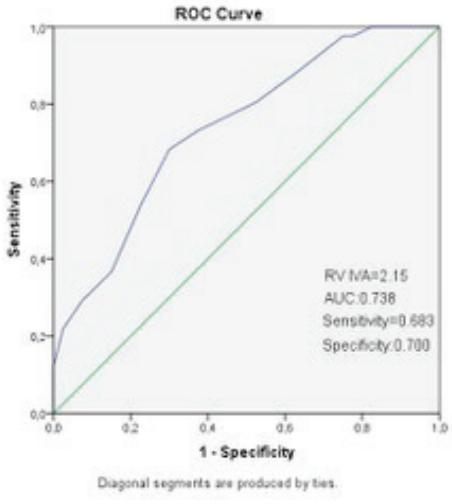

Figure 3. Receiver Operating Characteristics (ROC) curve analysis of RV IVA to predict asthma.
AUC: Area under curve, RV IVA: Right ventricle isovolumic acceleration.

DISCUSSION

The results of this study suggest that in patients with moderate asthma, the parameters of RV E', RV FAC, RV MPI, RV IVRT, TAPSE, and RV IVA decreased, showing subclinical RV dysfunction in the absence of pulmonary hypertension. RV IVA differed significantly among asthmatic patients based on asthma duration. TAPSE and PASP were shown to be independent predictors of RV IVA. Therefore, we propose that moderate asthma may be associated with early RV dysfunction even before the development of pulmonary hypertension and RV IVA may be a reliable and useful parameter to detect subclinical RV systolic dysfunction.

Asthma is a chronic inflammatory disorder originating from heterogenic gene-environment interactions that are not fully understood[17]. Because the inflammatory characteristics of asthma were suggested to have a possible systemic impact, the studies examining the cardiac effects of asthma mainly focused on atherosclerotic disease or arrhythmic disorders[18-20]. However, RV is in a continuum with the lungs and this makes it vulnerable to pressure changes in the pulmonary vasculature. Asthma causes a chronic and long-term hypoxia state which may lead to intimal hyperplasia, fibrosis, and occlusion of pulmonary arterioles. These structural changes may result in increased pulmonary vascular resistance and pulmonary hypertension, which in turn, may lead to cor pulmonale in severe cases[5,6,21].

Despite the aforementioned pathophysiology and the high number of studies reporting an association between RV functions and chronic obstructive pulmonary disease or diffuse parenchymal lung diseases, only a limited number of studies explored a causal relationship between non-severe asthma and RV[1,2,8]. In previous two studies on mild asthmatic patients, impaired RV function detected by speckle tracking echocardiography method and increased pulmonary artery stiffness index was demonstrated as the new findings associated with asthma in adults[13,14]. Likely, our study revealed the presence of subclinical RV dysfunction in asthmatic adults before the development of overt pulmonary hypertension.

There are several echocardiographic parameters assessing RV function, namely, TAPSE, RV S', RV FAC, RV MPI, strain, and strain rate. Of these, TAPSE is load-dependent and may only reflect the longitudinal motion of the RV instead of the global function. RV S' is another measure of longitudinal systolic function of RV and it's less reproducible. RV FAC is limited in reproducibility due to difficulty in endocardial border delineation during volumetric modeling of RV. RV MPI is load-dependent and it's unreliable if right atrial pressure is elevated. Lastly, strain and strain rate are complex measures with a high degree of variability requiring additional software and offline analysis, and are not recommended for routine clinical use[10,22].

RV IVA is an easily obtained, tissue Doppler-derived index of ventricular contractile function. Its validity was firstly demonstrated in pigs in 2002 and then it was confirmed in humans in 2006. It represents the global RV function and estimates the contractile state in a relatively load-independent route. Although its normative data is limited, it's correlated with the severity of diseases affecting right heart function[10,11,23,24].

After its validation in experiments and in humans, RV IVA was evaluated in several clinical settings including the early detection of RV dysfunction in patients with chronic obstructive pulmonary disease, obstructive sleep apnea syndrome, and systemic sclerosis[25-28]. Yang et al.[29] analyzed correlations between cardiac magnetic resonance imaging-derived RV EF and echocardiographic parameters including RV IVA, RV MPI, TAPSE, RV FAC, and RV S' in patients with pulmonary arterial hypertension. Of these parameters, RV IVA was found to be independently associated with RV EF. Similarly, Shahlaee et al.[27] confirmed the correlation of RV IVA to RV EF in patients with heart failure. Sciatti et al.[11] demonstrated that RV IVA had a better prognostic utility compared with RV FAC, TAPSE, and RV S' in patients with heart failure. IVA has also been reported to have a diagnostic value similar to or even better than strain and strain rate[11,12,26]. Its utility in assessing RV function was proved further in patients with surgically corrected tetralogy of Fallot and metabolic syndrome[30,31].

In addition to confirming the validity of RV IVA in the evaluation of several disorders, its utility in monitoring RV functions following therapeutic interventions was also demonstrated. In this manner, RV IVA was shown to be improved as

a response to device closure in patients with atrial septal defect and was found to be a more robust predictor of RV function than S', TAPSE, and free wall strain in the assessment of recovery during the early postoperative period of patients who underwent mitral valve surgery[32,33]. Furthermore, it was demonstrated to be an independent clinical predictor of functional recovery after isolated heart valve surgery[34].

In this study, we used echocardiography to investigate asthmatic patients utilizing M-Mode, two-dimensional, and tissue Doppler imaging techniques, each of which reveals various aspects of RV function. Parameters evaluating RV function were impaired in patients with moderate asthma, indicating the presence of subclinical RV systolic dysfunction. To the best of our knowledge, this is the first study to investigate the role of RV IVA in asthmatic patients, however, there have been some publications on RV IVA in a variety of disorders. Our findings suggest that RV IVA in asthmatic patients is a reliable metric in determining subclinical RV systolic dysfunction.

Limitations

Our study has some limitations. First, this was a single-center study with a limited number of patients. Second, it was not possible to compare the echocardiographic parameters to a gold standard imaging modality (e.g. cardiac magnetic resonance imaging). Third, there were some methodological challenges due to the absence of previous studies on the role of RV IVA in asthmatic patients. Finally, since IVA is an age-dependent parameter, we used an age-matched control group in our study to avoid age-dependent bias.

CONCLUSION

Asthma is a chronic lung disease that may affect RV systolic functions even in the absence of pulmonary hypertension. As a result, it becomes increasingly important to recognize subclinical deteriorations in asthmatic patients to identify those who may benefit from closer monitoring and more aggressive therapy. It was also demonstrated that RV IVA is a robust metric detecting subtle impairments in RV function in correlation with asthma duration. Further studies are needed to evaluate RV IVA and its prognostic implications in large-scale populations.

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Informed Consent: This is retrospective study, we could not obtain written informed consent from the participants.

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