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# Evaluation of tissue Doppler echocardiography in the assessment of ventricular function in asthmatic children

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## Abstract

**Background**

Bronchial asthma is one of the commonest diseases in children. It not only affects the lung but also other organs, including the heart. Our study aims to assess the effect of bronchial asthma with varying degrees of severity on the ventricular function of the heart using tissue Doppler imaging.

**Patients and methods**

This is a case–control study that was performed at the Pediatric Department of Al-Azhar University Hospital and included 50 patients with bronchial asthma, who were compared with 50 age-matched and sex-matched healthy children as a control group. All children were subjected to full history taking, complete physical examination, measurement of pulmonary function, and echocardiography by conventional and tissue Doppler.

**Results**

Echocardiographic parameters in asthmatic children were nearly similar to those of healthy children. Subgroup analysis showed no significant differences between controlled and uncontrolled asthmatics except for the tricuspid lateral E' velocity, which was lower in uncontrolled asthmatics when compared with the controlled. There were statistically significant differences among mild, moderate, severe asthmatic groups regarding pulmonary function tests, which were lower in severe asthmatic patients when compared with mild asthmatic patients.

**Conclusions**

Although there was no statistically significant difference between asthmatic patients and controls, we noticed that the right ventricular tissue Doppler indices were lower in asthmatic patients than controls, which should be considered, and a statistically significant relation between tissue Doppler echocardiography and the result of the pulmonary function tests. So, the disease severity can be predicted using tissue Doppler echocardiography.

**Keywords:** bronchial asthma, children, tissue Doppler imaging, ventricular function

## Introduction

Asthma is a worldwide chronic respiratory disease, affecting more than 300 million people of all racial groups of all ages [1]. It is a significant global health issue with rising prevalence in many developing countries raising the cost of care and raising the burden on patients and society [2].

Asthma is defined by chronic inflammation of the airways. It is characterized by the history of respiratory symptoms that vary in severity over time, such as tightness of the chest, shortness of breath, wheeze, and cough, as well as the limitation of expiratory airflow [3].

Inflammation of the airways results from interactions between different cellular components and cytokines. Airway inflammation in susceptible individuals can cause recurrent or

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persistent bronchospasm, especially at night, early morning, or after exercise [4].

Asthma, recurrent hypoxemia, and hypercarbia, along with different mediators and cytokines produced as a result of chronic inflammation, contribute to pulmonary vasoconstriction. Moreover, several factors, including the narrowing of pulmonary vessels by parenchymal changes, increased cardiac output, and blood viscosity from polycythemia secondary to hypoxia, also play a role in the development of pulmonary hypertension [5].

One of the most important prognostic factors for idiopathic pulmonary hypertension is right ventricular (RV) dysfunction. The RV is hypertrophied and dilated when chronically pressure-overloaded, which results in both systolic and diastolic dysfunction. In asthmatic patients, RV diastolic dysfunction is the earliest cardiac hemodynamic change owing to increased afterload [6].

The irregular shape of the RV, its retrosternal position, and the frequent coexistence of lung hyperinflation make the standard 'gray-scale' imaging suboptimal for routine assessment of RV [7].

Tissue Doppler echocardiography (TDE) is a tecnology for the quantitative measurement of regional myocardial velocities even if the gray-scale image is not optimum. As TDE may detect subclinical RV abnormalities at a stage when conventional echocardiography parameters are still normal, the use of TDE may be superior to that of conventional echocardiography [8].

Our study aims to assess the effect of bronchial asthma with varying degrees of severity on the ventricular function of the heart using tissue Doppler imaging (TDI).

## Patients and methods

### Participants

This was a case–control study that included 50 patients (6–16 years) with bronchial asthma who were compared with 50 age-matched and sex-matched healthy children as a control group. Patients comprised 29 males and 21 females, whereas the control group comprised 31 males and 19 females.

The study was conducted at the Pediatric Department, Al Zahraa University Hospital, during the period between January 2019 and December 2019.

Children less than 6 years and those with suspected or proven alternative cause for the recurrent wheezing chest as cystic fibrosis or ciliary dyskinesia were excluded from the study. Moreover, children with comorbid diseases such as gastroesophageal reflux, allergic rhinitis, upper and lower respiratory infection, and those with other diseases that can produce TDE changes such as cardiovascular diseases were excluded.

The patient group was classified according to the disease severity based on the recently established guidelines of the global initiative for asthma management and prevention [3].

Group I included 22 patients with mild bronchial asthma.

Group II included 17 patients with moderate bronchial asthma.

Group III included 11 patients with severe bronchial asthma.

Ethical considerations: written informed consent was obtained from all patients and control groups or their parents before being involved in the study. The study was conducted according to the World Medical Association Declaration of Helsinki.

### Methods

The studied population was subjected to the following:

(1) Full medical history, including data regarding age, sex, any other health problems, any history of cough, recurrent wheeze, recurrent difficult breathing, recurrent chest tightness, symptoms occur or worsen at night, hospital admissions for chest conditions, nasal allergy, family history of bronchial asthma or atopic diseases, and therapeutic history about the daily dose of inhaled corticosteroids, need for a rescue inhaler, and symptoms that respond to asthma therapy.

(2) Thorough clinical examination including chest and heart examination.

(3) Pulmonary function testing: it was measured by JAEGER (VIASYS Healthcare GmbH Leibnizstrasse 7, Germany), which measures airflow and lung volumes during the expiratory maneuver and is considered a gold standard airflow test in asthma. It measures forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC), FEV1/FVC ratio, and peak expiratory flow (PEF). It was done by instructing the patients to blow the air into the tube as hard and fast as they could in one long complete breath and performed at least three times, and the best of these results were considered.

(4) Transthoracic echocardiography (TTE): conventional echocardiography was done to exclude any previously undiagnosed congenital or acquired heart disease. TTE was initially performed as a routine diagnostic imaging and included motion mode (M-mode), two-dimensional, pulsed wave (PW), continuous wave as well as color flow Doppler studies in the apical 4 chamber, apical 5 chambers, parasternal long-axis and short-axis parasternal views. TTE was done using the vivid E9 machine (GE Vingmed ultrasound N-3191, Horton, Norway) and the GE Health care, USA. (M5S-D Probe) (S/N 00000 2891, Portugal), and the patient was connected to the ECG tracing of the echocardiographic machine.

(5) Assessment of ventricular dimension function: the following parameters were assessed: left ventricular end-diastolic diameter [left ventricular internal dimension during diastole (LVIDD)], ejection fraction (EF), and shortening fraction (FS) of left ventricle.

(6) Assessment of ventricular diastolic function: pulsed Doppler recordings were used to detect mitral and tricuspid inflow velocities by the measurement of peak early filling (E-wave) velocity, peak atrial filling (A-wave) velocity, and E/A ratio.

(7) Tissue Doppler: by activating the TDI function in the same echocardiographic machine, recordings of the annular velocities were made with PW TDI to obtain the best quality recordings. The filter settings and gains were adjusted at the minimal optimal level to minimize noise and eliminate the signals produced by the transmitral flow. TDI was done to detect the peak systolic velocity (S), peak early diastolic velocity (E), and peak late diastolic velocity (A) waves of the septal and lateral mitral annuli and the lateral tricuspid annulus and to calculate the modified myocardial performance index (Tei index) for both the right and left ventricles [9].

All healthy children were cardiologically evaluated by conventional echocardiography and TDI.

### Statistical analysis

Recorded data were analyzed using the statistical package for social sciences, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were expressed as mean ± SD. Qualitative data were expressed as frequency and percentage. Independent samples *t* test of significance was used when comparing two means. A one-way analysis of variance was used when comparing more than two means. $\chi^2$ test of significance was used to compare proportions between qualitative parameters.

**Table 1. Comparison between asthmatic patients and control groups as regards demographic data, anthropometric measurements, and pulmonary function tests.**

| Variables (Mean±SD) | Patients (*n*=50) | Control (*n*=50) | *t*-test | *P* |
|---|---|---|---|---|
| Demographic Data | | | | |
| Age (years) | 11.46±2.73 | 12.34±3.39 | 2.045 | 0.156 |
| Male | 29 (58%) | 31 (62%) | 0.167 | 0.683 |
| Female | 21 (42%) | 19 (38%) | | |
| Wt (kg) | 36.72±9.51 | 39.42±10.58 | 1.801 | 0.183 |
| Ht (cm) | 138.34±9.38 | 140.84±10.78 | 1.532 | 0.219 |
| Pulmonary function test | | | | |
| FEV1% | 71.86±7.97 | 77.76±6.10 | 4.157 | <0.001** |
| FVC% | 89.30±4.17 | 91.46±2.88 | 3.732 | 8.412 |
| FEV1/FVC% | 79.16±5.04 | 93.75±3.92 | 16.158 | <0.001** |
| PEF% | 73.56±6.99 | 85.90±2.51 | 11.749 | <0.001** |

*P*>0.05 NS; **P*<0.05 S; ***P*<0.001 HS *t*-Independent Sample *t*-test

**Table 2: Comparison between asthmatic patients and control group regarding the mean values of echocardiographic parameters measured by m-mode, posterior wall diameter, and tissue Doppler**

| Variables | Patients (*n*=50) (mean±SD) | Control (*n*=50) (mean±SD) | *t* test | *P* |
|---|---|---|---|---|
| Conventional echocardiographic (m-mode) | | | | |
| LVPWD (cm) | 0.66±0.16 | 0.64±0.14 | 0.637 | 0.427 |
| LVIDD (cm) | 3.82±0.44 | 3.91±0.34 | 1.192 | 0.278 |
| EF% | 69.90±3.18 | 70.14±1.73 | 0.220 | 0.640 |
| FS% | 38.96±2.91 | 38.08±2.78 | 2.385 | 0.126 |
| Conventional echocardiographic (pulse wave Doppler) | | | | |
| Mitral E velocity (m/s) | 0.97±0.15 | 0.94±0.06 | 1.034 | 0.312 |
| Mitral a velocity (m/s) | 0.59±0.11 | 0.61±0.04 | 0.485 | 0.488 |
| E/a ratio | 1.67±0.30 | 1.56±0.33 | 1.136 | 0.108 |
| Tricuspid E velocity (m/s) | 0.91±0.19 | 0.88±0.07 | 1.057 | 0.306 |
| Tricuspid a velocity (m/s) | 0.61±0.13 | 0.64±0.12 | 0.953 | 0.331 |
| E/a ratio | 1.51±0.29 | 1.39±0.36 | 3.099 | 0.081 |
| Tissue Doppler | | | | |
| Septal S (m/s) | 0.08±0.01 | 0.07±0.01 | 1.505 | 0.150 |
| Septal E' (m/s) | 0.18±0.02 | 0.17±0.02 | 1.053 | 0.268 |
| Septal A' (m/s) | 0.07±0.02 | 0.06±0.01 | 0.534 | 0.466 |
| Lateral S (m/s) | 0.13±0.10 | 0.18±0.01 | 0.038 | 0.846 |
| Lateral E' (m/s) | 0.17±0.02 | 0.18±0.01 | 0.095 | 0.759 |
| Lateral A' (m/s) | 0.08±0.01 | 0.07±0.02 | 0.718 | 0.567 |
| Tricuspid lateral S (m/s) | 0.10±0.02 | 0.11±0.02 | 2.397 | 0.125 |
| Tricuspid lateral E' (m/s) | 0.17±0.04 | 0.20±0.04 | 1.150 | 0.267 |
| Tricuspid lateral A' (m/s) | 0.09±0.03 | 0.10±0.02 | 1.877 | 0.174 |

EF, ejection fraction; FS, fractional shortening; LVIDD, left ventricular internal dimension during diastole; LVPWD, left ventricular posterior wall diameter during diastole. *t*, independent sample *t* test. *P*>0.05 nonsignificant.

Pearson's correlation coefficient (*r*) test was used to assess the degree of association between the two sets of variables.

## Results

The results of the present study are demonstrated in the following tables:

Table 1 shows the following:

(1) There was no statistically significant difference between asthmatic patients and control groups regarding their demographic data and anthropometric measurements.
(2) There was a statistically significant decrease in the pulmonary function tests in the patient group compared with the control group.

Table 2 shows that there were no statistically significant differences between asthmatic patients and the control group regarding the conventional echocardiographic parameters measured by m-mode, pulse wave Doppler, and that measured by tissue Doppler.

Table 3 shows that there were statistically significant differences between mild, moderate, severe asthmatic groups regarding pulmonary function tests, which were lower in severe asthmatic than that of the mild asthmatic.

Table 4 shows that there was a statistically significant positive correlation between FEV1/FVC ratio and tricuspid E/A ratio.

There was a statistically significant negative correlation between the following:

(1) FEV1/FVC ratio and mitral lateral S.
(2) PEF and mitral lateral E'.
(3) FEV1/FVC ratio and LVIDD.

Table 5 shows that there was a statistically significant decrease in tricuspid lateral E' velocity in uncontrolled asthmatic patients than controlled.

Table 6 shows that there was a statistically significant positive correlation between the following:

(1) LVIDD and mitral E/A ratio.
(2) Left ventricular posterior wall diameter during diastole and mitral septal S, as well as mitral septal E' and mitral lateral E'.
(3) FS and mitral E velocity.
(4) EF and mitral E velocity.

## Discussion

Bronchial asthma is a significant global health problem in many

**Table 3: Comparison between mild, moderate, and severe asthmatic groups regarding pulmonary function tests and the mean values of echocardiographic parameters**

| Parameters | Mild | Moderate | Sever | ANOVA | *P* |
|---|---|---|---|---|---|
| Pulmonary function test | | | | | |
| FEV1% | 75.00±4.85 | 64.94±1.20 | 59.55±0.69 | 29.146 | 0.024* |
| FVC% | 89.95±3.00 | 88.06±0.97 | 87.91±0.83 | 71.312 | 0.134 |
| FEV1/FVC% | 84.44±2.92 | 72.47±1.28 | 67.65±0.72 | 23.672 | 0.012* |
| PEF% | 78.00±3.35 | 62.76±4.31 | 55.12±1.21 | 37.843 | 0.031* |
| Conventional echocardiographic | | | | | |
| LVPWD (cm) | 0.66±0.14 | 0.64±0.18 | 0.70±0.17 | 0.448 | 0.642 |
| LVIDD (cm) | 3.80±0.39 | 3.85±0.39 | 3.83±0.62 | 0.053 | 0.948 |
| EF% | 69.82±1.56 | 68.59±2.27 | 70.09±5.28 | 0.685 | 0.214 |
| FS% | 38.45±2.02 | 38.35±2.52 | 38.91±4.18 | 3.482 | 0.390 |
| Mitral E velocity (m/s) | 1.04±0.19 | 0.91±0.07 | 0.92±0.06 | 1.612 | 0.203 |
| Mitral a velocity (m/s) | 0.61±0.11 | 0.57±0.07 | 0.60±0.14 | 0.672 | 0.515 |
| E/a ratio | 1.67±0.34 | 1.68±0.25 | 1.66±0.28 | 0.007 | 0.993 |
| Tricuspid E velocity (m/s) | 0.91±0.18 | 0.98±0.22 | 0.88±0.08 | 1.300 | 0.460 |
| Tricuspid a velocity (m/s) | 0.62±0.14 | 0.63±0.16 | 0.59±0.08 | 0.336 | 0.717 |
| E/a ratio | 1.52±0.33 | 1.59±0.29 | 1.37±0.07 | 2.090 | 0.135 |
| Tissue Doppler | | | | | |
| Septal S (m/s) | 0.08±0.01 | 0.08±0.01 | 0.08±0.01 | 1.436 | 0.248 |
| Septal E' (m/s) | 0.19±0.02 | 0.18±0.02 | 0.17±0.02 | 0.115 | 0.310 |
| Septal A' (m/s) | 0.07±0.01 | 0.07±0.02 | 0.06±0.02 | 0.288 | 0.246 |
| Lateral S (m/s) | 0.12±0.02 | 0.15±0.17 | 0.10±0.03 | 0.986 | 0.381 |
| Lateral E' (m/s) | 0.18±0.02 | 0.18±0.02 | 0.17±0.02 | 0.136 | 0.873 |
| Lateral A' (m/s) | 0.08±0.01 | 0.08±0.01 | 0.08±0.01 | 1.448 | 0.245 |
| Tricuspid lateral S (m/s) | 0.12±0.02 | 0.11±0.02 | 0.12±0.02 | 0.282 | 0.246 |
| Tricuspid lateral E' (m/s) | 0.18±0.05 | 0.17±0.03 | 0.17±0.04 | 0.260 | 0.772 |
| Tricuspid lateral A' (m/s) | 0.10±0.03 | 0.10±0.03 | 0.08±0.02 | 1.039 | 0.362 |

ANOVA, analysis of variance; EF, ejection fraction; FEV1, forced expiratory volume in 1 s; FS, fractional shortening; FVC, forced vital capacity; LVIDD, left ventricular internal dimension during diastole; ; LVPWD, left ventricular posterior wall diameter during diastole PEF, peak expiratory flow. *F*, one-way ANOVA. *P*>0.05 nonsignificant. **P*<0.05 significant.

**Table 4: Correlation between the mean values of the measured pulmonary function tests and the mean values of echocardiographic parameters among asthmatic patients**

| Parameters | FEV1% | | FVC% | | FEV1/FVC% | | PEF% | |
|---|---|---|---|---|---|---|---|---|
| | *r* | *P* | *r* | *P* | *r* | *P* | *r* | *P* |
| LVPWD (cm) | −0.146 | 0.312 | −0.110 | 0.448 | −0.160 | 0.268 | −0.033 | 0.820 |
| LVIDD (cm) | −0.019 | 0.894 | −0.015 | 0.919 | −0.476 | 0.005* | −0.106 | 0.462 |
| EF% | −0.233 | 0.103 | −0.192 | 0.182 | −0.267 | 0.061 | −0.133 | 0.359 |
| FS% | −0.365 | 0.009* | −0.318 | 0.024* | −0.385 | 0.006* | −0.196 | 0.172 |
| Mitral E velocity (m/s) | 0.358 | 0.011* | 0.373 | 0.008* | 0.306 | 0.031* | 0.275 | 0.054 |
| Mitral a velocity (m/s) | 0.118 | 0.415 | 0.111 | 0.444 | 0.102 | 0.479 | 0.024 | 0.870 |
| E/a ratio | −0.046 | 0.752 | −0.032 | 0.823 | −0.054 | 0.707 | −0.038 | 0.792 |
| Tricuspid E velocity (m/s) | 0.097 | 0.504 | 0.085 | 0.558 | 0.115 | 0.428 | 0.072 | 0.619 |
| Tricuspid a velocity (m/s) | −0.119 | 0.411 | −0.157 | 0.275 | −0.062 | 0.667 | −0.076 | 0.602 |
| E/a ratio | 0.289 | 0.042* | 0.311 | 0.028* | 0.478 | 0.028* | 0.212 | 0.139 |
| Septal S (m/s) | −0.191 | 0.183 | −0.288 | 0.042* | −0.089 | 0.541 | −0.196 | 0.172 |
| Septal E' (m/s) | −0.336 | 0.017* | −0.341 | 0.016* | −0.315 | 0.026* | −0.372 | 0.008* |
| Septal A' (m/s) | −0.347 | 0.013* | −0.301 | 0.034* | −0.368 | 0.009* | −0.433 | 0.002* |
| Lateral S (m/s) | 0.015 | 0.917 | −0.026 | 0.860 | −0.555 | 0.007* | −0.234 | 0.102 |
| Lateral E' (m/s) | 0.074 | 0.607 | 0.062 | 0.671 | 0.070 | 0.629 | −0.498 | 0.006* |
| Lateral A' (m/s) | −0.117 | 0.419 | −0.144 | 0.320 | −0.084 | 0.560 | −0.082 | 0.573 |
| Tricuspid lateral S (m/s) | −0.318 | 0.025* | −0.315 | 0.026* | −0.288 | 0.042* | −0.347 | 0.014* |
| Tricuspid lateral E' (m/s) | 0.121 | 0.404 | 0.068 | 0.641 | 0.157 | 0.277 | 0.092 | 0.524 |
| Tricuspid lateral A' (m/s) | 0.149 | 0.302 | 0.074 | 0.609 | 0.211 | 0.142 | 0.027 | 0.853 |

EF, ejection fraction; FEV1, forced expiratory volume in 1 s; FS, fractional shortening; FVC, forced vital capacity; LVIDD, left ventricular internal dimension during diastole; LVPWD, left ventricular posterior wall diameter during diastole; PEF, peak expiratory flow. *r*, Pearson correlation coefficient. *P*>0.05 nonsignificant. **P*<0.05 significant. ***P*<0.001 highly signififant.

developing countries affecting all age groups with increasing prevalence [3]. It not only affects the lung but also affects other organs, including the heart. RV systolic and diastolic dysfunctions were found in a considerable percentage of asthmatic children even with mild cases [10].

In this study, our results showed that there were no statistically significant differences between asthmatic patients and control groups regarding their demographic data and anthropometric measurements.

Respiratory function tests, especially FEV1, FEV1/FVC, and PEF, are the best predictors of disease severity in children with asthma. PEF is useful in the routine monitoring of healthy and asthmatic children. In the current study, we found that pulmonary function tests, FEV1, and FEV1/FVC in asthmatic children showed significantly lower values than controls. Our results were in agreement with Ghaderian *et al.* [11], who found that their asthmatic children had lower FEV1 and FEV1/FVC than healthy. Regarding PEF, our asthmatic children showed significantly lower PEF than controls. Our results were in agreement with Shedeed [12], who found that asthmatic children had significantly lower PEF than controls. There were statistically significant differences between mild, moderate, and severe asthmatic groups regarding FEV1, FEV1/FVC, and PEF, which were lower in severe asthmatics when compared with mild asthmatics.

In our study, we assessed the left ventricular dimensions and function among asthmatic children and controls through conventional Doppler echocardiography, and the results were not significant. Moreover, the left ventricular systolic echocardiographic function among cases was insignificantly different from those among controls.

The right and left ventricular diastolic function were evaluated through PW Doppler and revealed that mitral valve peak E velocity, mitral valve peak A velocity, and mitral valve E/A ratio were insignificantly different from those among controls ($P > 0.05$). The RV conventional pulsed Doppler indices (peak E velocity, peak A velocity, and E/A ratio) were insignificantly different among asthmatic patients and control cases (Table 2).

Our results were in agreement with Shedeed [12], who found that conventional findings in echocardiography such as E and A velocity, E/A ratio at mitral and tricuspid valve, LV diameter, EF, and FS did not differ significantly between patients and controls ($P > 0.05$). On the contrary, Abdalla and El Azeem [13] suggested that LV diastolic function is impaired in patients with bronchial asthma, despite there being no effect on RV diastolic function, and they revealed that there were statistically significant differences in the peak E velocity, peak E velocity/peak A velocity ratio, and isovolumetric relaxation time between the two groups ($P < 0.05$).

In our study, TDI was done to evaluate the ventricular function and revealed that mitral septal (peak A' velocity, peak E', and S' velocity), mitral lateral (peak A' velocity, peak E', and S' velocity), and tricuspid lateral (peak E', peak A' velocity, and

**Table 5: Comparison between controlled and uncontrolled asthmatic groups regarding the demographic data, duration of asthma, and the mean values of echocardiographic parameters**

| Parameters | Controlled | Uncontrolled | *t* test | *P* |
|---|---|---|---|---|
| Demographic data | | | | |
| Age (years) | 11.26±2.68 | 12.18±2.89 | 0.988 | 0.325 |
| Weight (kg) | 35.82±9.06 | 39.91±10.80 | 1.606 | 0.211 |
| Height (cm) | 137.67±9.14 | 140.73±10.27 | 0.913 | 0.344 |
| Duration of disease (years) | 6.41±2.10 | 7.91±1.04 | 1.442 | 0.740 |
| Conventional echocardiographic | | | | |
| LVPWD (cm) | 0.65±0.16 | 0.70±0.17 | 0.721 | 0.400 |
| LVIDD (cm) | 3.82±0.39 | 3.83±0.62 | 0.002 | 0.965 |
| EF% | 69.28±1.97 | 72.09±5.28 | 0.613 | 0.818 |
| FS% | 38.41±2.22 | 40.91±4.18 | 0.971 | 0.105 |
| Mitral E velocity (m/s) | 0.98±0.17 | 0.92±0.06 | 1.468 | 0.232 |
| Mitral a velocity (m/s) | 0.59±0.10 | 0.60±0.14 | 0.035 | 0.852 |
| E/a ratio | 1.67±0.30 | 1.66±0.28 | 0.006 | 0.938 |
| Tricuspid E velocity (m/s) | 0.94±0.20 | 0.80±0.08 | 1.327 | 0.064 |
| Tricuspid a velocity (m/s) | 0.62±0.14 | 0.59±0.08 | 0.604 | 0.441 |
| E/a ratio | 1.55±0.31 | 1.37±0.07 | 3.556 | 0.065 |
| Tissue Doppler | | | | |
| Septal S (m/s) | 0.077±0.009 | 0.076±0.005 | 0.039 | 0.844 |
| Septal E' (m/s) | 0.185±0.019 | 0.195±0.023 | 0.775 | 0.474 |
| Septal A' (m/s) | 0.063±0.013 | 0.074±0.019 | 0.613 | 0.368 |
| Lateral S (m/s) | 0.132±0.112 | 0.104±0.029 | 0.663 | 0.419 |
| Lateral E' (m/s) | 0.175±0.020 | 0.175±0.020 | 0.015 | 0.902 |
| Lateral A' (m/s) | 0.077±0.011 | 0.077±0.009 | 0.002 | 0.963 |
| Tricuspid lateral S (m/s) | 0.109±0.022 | 0.112±0.024 | 0.091 | 0.764 |
| Tricuspid lateral E' (m/s) | 0.186±0.040 | 0.171±0.043 | 3.042 | 0.038* |
| Tricuspid lateral A' (m/s) | 0.10±0.03 | 0.08±0.02 | 1.831 | 0.182 |

EF, ejection fraction; FS, fractional shortening; LVIDD, left ventricular internal dimension during diastole; LVPWD, left ventricular posterior wall diameter during diastole. *t*, independent sample *t* test. *P*>0.05 nonsignificant. *P<0.05 significant.

**Table 6: Correlation between the mean values of left ventricular dimensions, function to the mean values of echocardiographic parameters measured by posterior wall diameter, and tissue Doppler imaging among asthmatics**

| Parameters | LVPWD (cm) | | LVIDD (cm) | | EF% | | FS% | |
|---|---|---|---|---|---|---|---|---|
| | *r* | *P* | *r* | *P* | *r* | *P* | *r* | *P* |
| Mitral E velocity (m/s) | −0.115 | 0.426 | −0.036 | 0.932 | 0.476 | 0.017* | 0.640 | 0.012* |
| Mitral A velocity (m/s) | −0.175 | 0.225 | −0.087 | 0.550 | 0.034 | 0.816 | −0.058 | 0.691 |
| E/A ratio | 0.029 | 0.844 | −0.179 | 0.013* | −0.047 | 0.748 | 0.016 | 0.911 |
| Tricuspid E velocity (m/s) | −0.206 | 0.152 | −0.114 | 0.430 | −0.164 | 0.252 | −0.295 | 0.378 |
| Tricuspid A velocity (m/s) | −0.084 | 0.562 | 0.126 | 0.385 | −0.208 | 0.147 | −0.173 | 0.230 |
| E/A ratio | −0.148 | 0.305 | 0.502 | 0.029* | −0.099 | 0.495 | −0.155 | 0.284 |
| Septal S (m/s) | 0.617 | 0.012* | 0.076 | 0.599 | −0.067 | 0.642 | 0.029 | 0.843 |
| Septal E' (m/s) | 0.256 | 0.031* | −0.159 | 0.271 | 0.277 | 0.140 | 0.207 | 0.148 |
| Septal A' (m/s) | 0.205 | 0.154 | 0.058 | 0.688 | −0.241 | 0.091 | −0.174 | 0.227 |
| Lateral S (m/s) | 0.114 | 0.430 | 0.134 | 0.354 | 0.063 | 0.662 | 0.116 | 0.421 |
| Lateral E' (m/s) | 0.291 | 0.040* | −0.045 | 0.910 | −0.207 | 0.149 | −0.213 | 0.137 |
| Lateral A' (m/s) | 0.192 | 0.182 | 0.125 | 0.386 | 0.292 | 0.394 | 0.372 | 0.785 |
| Tricuspid lateral S (m/s) | 0.132 | 0.361 | 0.224 | 0.117 | −0.376 | 0.719 | −0.139 | 0.465 |
| Tricuspid lateral E' (m/s) | 0.009 | 0.952 | 0.078 | 0.592 | −0.241 | 0.092 | −0.131 | 0.275 |
| Tricuspid lateral A' (m/s) | −0.137 | 0.344 | 0.125 | 0.385 | −0.306 | 0.306 | −0.103 | 0.308 |

EF, ejection fraction; FS, fractional shortening; LVIDD, left ventricular internal dimension during diastole; LVPWD, left ventricular posterior wall diameter during diastole. *r*, Pearson correlation coefficient. *P*>0.05 nonsignificant. *P<0.05 significant. **P<0.001 highly significant.

S' velocity) were insignificantly different among asthmatic cases than controls. On the contrary, Shedeed [12] reported that the tissue Doppler study of the RV diastolic function revealed that E'-wave, the A'-wave, and the *S*'-wave velocities

were significantly lower and E'/A' was significantly greater in asthmatic children. A possible hypothesis that may explain subclinical RV dysfunction may be asthmatic children experience bouts of pulmonary hypertension during exacerbations of respiratory symptoms, which may exert a cumulative effect leading to RV hypertrophy. Ozdemir *et al.* [14] reported the same, though there was no difference by conventional echocardiography in asthmatic children. TDI showed subclinical RV dysfunction, and they proposed that TDI was useful in the early detection of some detrimental effects of asthmatic cases.

In this study, our results showed no significant differences between controlled and uncontrolled asthmatics except for the tricuspid lateral E' velocity, which was lower in uncontrolled asthmatics when compared with the controlled.

Several mechanisms may be responsible for diastolic dysfunction in bronchial asthma. The first and most important factor is the significantly increased heart rate in the bronchial asthma group. Tachycardia shortened the diastolic filling period, and atrial contraction may have occurred before the early filling was completed. This tachycardia may be owing to multiple causes including hypoxemia or medications [15]. Ozdemir *et al.* [14] investigated subclinical ventricular dysfunction by using conventional and TDE in asthmatic children without any cardiovascular symptoms. They showed that although the findings of clinical and conventional echocardiography were normal in children with asthma, TDE showed subclinical dysfunction of the RV, which is negatively correlated with PEF. These findings signified the diagnostic value of TDE in the early detection and monitoring of such deleterious effects among asthmatic patients.

In this study, in the asthmatic group, there was a statistically significant positive correlation between FEV1/FVC ratio (reduced FEV1/FVC indicates airflow obstruction) and tricuspid E/A ratio. There was a statistically significant negative correlation between FEV1/FVC ratio and LVIDD and the mitral lateral S velocity. Moreover, there was a statistically significant negative correlation between PEF and the mitral lateral E'. Moreover, in the asthmatic group, there was a statistically significant positive correlation between LVIDD and mitral E/A ratio and between left ventricular posterior wall diameter during diastole and mitral septal S, mitral septal E', and lateral E' velocity. FS and EF had positive correlations with mitral E velocity. Elmasry *et al.* [16] assessed the left ventricular function among asthmatic children both during and after the resolution of acute severe asthma and found that during acute exacerbations of asthma, patients had significantly higher transmitral peak A velocity and lower E/A ratio (i.e. impaired LV diastolic function) during acute asthma exacerbation but disappeared after its resolution and concluded that transmitral inflow velocity patterns during acute severe asthma in children are indicative of altered LV preload owing to an acute transient elevation in the pulmonary artery pressure secondary to the altered lung mechanics.

## Conclusions

From the present study, we concluded that although there was no statistically significant difference between asthmatic patients and controls regarding conventional and TDE, we noticed that the RV tissue Doppler indices were lower in asthmatic patients than controls. Moreover, there was a statistically significant difference between controlled and uncontrolled asthmatic patients in the tricuspid lateral E' velocity, which was lower in uncontrolled asthmatics. We demonstrated also a statistically significant relation between TDE and the result of the pulmonary function tests. So, the disease severity can be predicted using TDE. Identifying patients with increased risk of ventricular dysfunction may have important implications for treatment, and this must be tested in further studies.

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### Conflicts of interest

There are no conflicts of interest.

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