Transthoracic Echocardiographic Evaluation of Bronchial Asthma Patients

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ABSTRACT

BACKGROUND
Asthma is a chronic inflammatory disease of the airways which is related to airway obstruction and hyper-responsiveness and is characterized by recurrent wheezing, coughing and breathlessness.¹ As asthma is chronic inflammation of airways, many cytokines are produced during early and late phases, which are potent depressants of cardiac contractility. Bronchial asthma affects many organs including the heart. There are many explanations for the occurrence of cardiac dysfunction in asthmatic patients.²⁻⁴ It is suggested that recurrent exposure to hypoxia may cause pulmonary arterial hypertension, which causes RV hypertrophy and/or dilatation.² It is also suggested that recurrent hypoxemia and the release of various mediators and cytokines in bronchial asthma may cause chronic inflammation, which could induce pulmonary vasoconstriction.³⁻⁴ Other hypotheses concluded that the exaggerated respiratory efforts may raise intrathoracic pressure that increases RV afterload and consequently pulmonary hypertension with RV hypertrophy and/or dilatation.³⁻⁵ As a result of chronic pressure overload, the RV hypertrophies, dilates and leads to both systolic and diastolic dysfunction.⁵ We wanted to investigate cardiac dysfunction in bronchial asthma patients.

METHODS
The study was conducted in 50 bronchial asthma patients in 2013-2014. All were subjected to detailed clinical evaluation and trans-thoracic echocardiography.

RESULTS
74% of patients had abnormal echocardiography, out of these, maximum (51.35%) had impaired left ventricular diastolic relaxation. 16.21% patients had pulmonary arterial hypertension and 24.3% of the patients had both.

CONCLUSIONS
2D-Echo may be an important non-invasive tool to rule out any underlying cardiac cause of asthma and PAH or other cardiac co-morbidities in bronchial asthma, and also for further management of the patient.

KEYWORDS
Echocardiography, Bronchial Asthma

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DOI: 10.18410/jebmh/2019/596

Financial or Other Competing Interests: None.

How to Cite This Article:
DOI: 10.18410/jebmh/2019/596

BACKGROUND

In bronchial asthma there is episodic narrowing of airways leading to recurrent exposure to hypoxemia. Hypoxia is one of the mechanisms besides others leading to sustained pulmonary vasoconstriction and narrowing of the pulmonary vasculature. Consequently, pulmonary hypertension develops leading to right heart enlargement with ventricular hypertrophy, and impaired cardiac function, known as cor pulmonale. Mei Lan et al. (2007) reported that Patients with bronchial asthma had a thicker right ventricular free wall than did healthy subjects. As asthma is chronic inflammation of airways, many cytokines are produced during the early and late phases, including interleukins such as IL-1 beta, IL-2, IL-6, IL-8, IL-10, as well as tumour necrosis factor-alpha (TNF-a). These mediators are potent depressants of cardiac contractility. Long-term expression of TNF alpha within the heart may produce cardiac decompensation. The close relationship between the responses of airway and pulmonary vascular smooth muscle cells in the pathological settings of bronchial asthma and PAH, respectively, begins during embryological development of the lung. The pulmonary vascular smooth muscle originates partly from migration of adjacent airway smooth muscle cells and from surrounding undifferentiated mesenchymal cells. Echocardiography is a bedside, innocuous, reliable, reproductive, non-invasive technique for the assessment of pulmonary arterial pressure. Thus, in bronchial asthma, assessment of functional status of the heart particularly right ventricle and pulmonary arterial pressure may be helpful in the better clinical insight and management of these patients by 2D echocardiographic study.

We wanted to assess the cardiac functional abnormality in patients of bronchial asthma and correlate these with duration and severity of the disease if any, which is important for the management of bronchial asthma patient.

METHODS

The present study was carried out in 50 cases of bronchial asthma patients attending to the Department of Respiratory-Medicine, R. D. Gardi Medical College, Ujjain, during 2013-2014. Patients who presented with clinical history suggestive of bronchial asthma which was confirmed by chest x-ray and spirometry were included in the study. Patients with comorbid diseases such as diabetes, hypertension, HIV infection, anaemia, upper or lower respiratory tract infection, allergic rhinitis, gastroesophageal reflux, obesity, chronic cardiovascular or pulmonary diseases were excluded from the study. Transthoracic echocardiography was performed by a single experienced cardiologist, at R. D. Gardi Medical College, Ujjain. All the patients were classified as having mild, moderate, severe asthma based on GINA guideline for assessment of severity of bronchial asthma. Mild persistent asthma- FEV1 above 80% with 12% & above reversibility. Moderate asthma- persistent FEV1 60-80% of predicted with 12% & above reversibility. Severe persistent asthma- FEV1 <60% of predicted with 12% & above reversibility (GINA2015).

All selected patients were subjected for electrocardiography and resting two-dimensional transthoracic doppler echocardiography by cardiologist at Department of medicine R. D. Gardi Medical college Ujjain. The machine used was VIVID 7 model of GE health care system with a multifrequency probe with a range of 2-4.3 MHz. Both 2D and M-Mode studies were done. Echocardiography was reviewed to assess the pericardium, valvular anatomy and function, left and right-side chamber size and cardiac function. Tricuspid regurgitant flow was identified by color flow Doppler technique and the maximum jet velocity was measured by continuous wave Doppler without the use of intravenous contrast. Right ventricular systolic pressure was estimated based on the modified Bernoulli equation and was considered to be equal to the sPAP in the absence of right ventricular outflow obstruction: sPAP (mmHg)= right ventricular systolic pressure= trans-tricuspid pressure gradient (TTPG) + right atrial pressure (RAP), where trans-tricuspid gradient is 4v2 (v= peak velocity of tricuspid regurgitation, m/s). RAP was empirically estimated as 15 mmHg before 1997. Since 1997, RAP was estimated to be 5, 10, or 15 mmHg based on the variation in the size of inferior vena cava with inspiration as follows: complete collapse, RAP= 5 mmHg; partial collapse, RAP= 10 mmHg; and no collapse, RAP= 15 mmHg. Pulmonary hypertension (PH) was defined in this study as sPAP ≥30 mmHg. This value was chosen according to the definition of pulmonary hypertension. PH was classified into mild, moderate, and severe category as sPAP 30-50, 50-70, >70 mmHg, respectively (using Chemla formula, mean pulmonary arterial pressure (MPAP)=0.61 PASP + 2 mmHg and putting value of 25-35, 35-45, and >45 mmHg of MPAP for mild, moderate, and severe pulmonary hypertension, respectively). Right ventricle dimension was measured by M-Mode echo and right ventricular dilation or cor pulmonale was said to be present when it exceeded the normal range of 0.9-2.6 cm. Right ventricle contractility was also noted, and right ventricular systolic dysfunction was said to be present when it was hypokinetic. Left ventricular function was also assessed by using the following parameters: EF (ejection fraction)= measure of how much end-diastolic value is ejected from LV with each contraction (56%-78%). FS (fractional shortening)= it is a percentage change in LV dimension with each LV contraction (28%-44%). LV mass= left ventricular mass (88-224 g).

E/A= diastolic filling of left ventricles usually classified initially on the basis of the peak mitral flow velocity of the early rapid filling wave (E), peak velocity of the late filling wave caused by atrial contraction (A). In normal subjects LV elastic recoil is vigorous because of normal myocardial relaxation, therefore more filling is completed during early diastolic, so left ventricular diastolic dysfunction (LVDD) is said to be present when E/A is <1.3 (age group 45-49 years), <1.2 (age group 50-59 years), <1.0 (age group 60-69 years), and <0.8 (age group ≥70 years).
RESULTS

Out of 50 study patients 40 (80%) were male and 10 (20%) were female. maximum number of patients belong to the age group of 21-40 years in both male as well as female. Mean age of patients were 38.36 years. Majority (58%) of patients were classified as moderate persistent asthma, 10% and 32% of patients had mild and severe persistent asthma respectively on the basis of spirometry. Majority (80%) of patients Electrocardiogram was within normal limit. Other less common finding in electrocardiogram were Right Axis Deviation and Pulmonale 74% of patients had abnormal echocardiography, out of these maximum 51% had impaired left ventricular diastolic relaxation. 16.21% patient had pulmonary arterial hypertension. Since these two abnormalities were common, correlation between these abnormalities and other factors were seen. All the patients of Pulmonary arterial hypertension were having mild PAH.PAH and impairment in left ventricular relaxation were more common in severe persistent asthma. Majority of Patients with PAH were having duration of illness between 5-10 years and patients with impaired left ventricular diastolic relaxation had duration of illness between 1-5 years.

DISCUSSION

There were 40 males and 10 females belong to 11-60 years mean age of the patients were 38.36 years. The various cardiac abnormalities on transthoracic echocardiography were mild pulmonary arterial hypertension, impaired diastolic relaxation of left ventricle, mitral valve prolapse and mild pericardial effusion. Prevalence of mild pulmonary arterial hypertension in bronchial asthma patient in present study was 28%. Rastogi et al \[4,5\] in 2004 found that bronchial asthma was present in 29.48% children with pulmonary arterial hypertension.

Although Meilan KH et al in 2007 concluded that there is right ventricular free wall hypertrophy and dilation of right ventricle due to chronic pressure overload. Soad Sheheed in 2010 reported that bronchial asthma children have significantly higher right ventricular wall thickness than normal control. In the present study diastolic relaxation of left ventricle was impaired in 58% of the patients. Elmasry OA et al \[6\] 2008 also found left ventricular impaired diastolic relaxation during acute exacerbation in children of Egypt. Same author also reported right ventricular hypertrophy in asthmatic patients. In the present study pericardial effusion and mitral valve prolapse was present in 1 and 2 patients respectively. Probably no author has reported the same. In the present study 1 patient also had severe pulmonary arterial hypertension but the same patient had mitral regurgitation also. This may be coincidental finding in this study. Hence it was not considered for correlation. In the present study pulmonary arterial hypertension due to asthma had no correlation with age, educational status, socio-economic status, duration of illness. In the present study PAH has significantly higher prevalence in severe persistent asthma. In the present study left ventricular impaired diastolic relaxation does not have any significant correlation with age, sex, socio-economic status, body mass index, duration of illness. But it was seen more commonly in severe asthma.

CONCLUSIONS

2D-Echocardiography may be an important noninvasive tool to rule out any underlying cardiac cause of asthma and PAH or other cardiac co-morbidities in bronchial asthma, and also for further management of the patient.

REFERENCES


