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# TO EVALUATE CARDIAC BIOMARKERS AND ECG CHANGES IN ASSESSING CARDIAC DYSFUNCTION IN CHILDREN WITH ACUTE SEVERE BRONCHIAL ASTHMA

Paediatric Medicine

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## ABSTRACT

**Introduction:** Asthma is a chronic inflammatory disease of the airways, characterized by recurrent episodes of airflow obstruction resulting from edema, bronchospasm, and increased mucus production. There are an estimated 300 million people who have asthma worldwide, with a significant geographic variation of prevalence, severity, and mortality. The exact etiology of asthma remains unclear and appears to be multifactorial. Both genetic and environmental factors seem to contribute. **Aim and Objective:** To evaluate role of cardiac biomarkers and ECG changes in assessing the cardiac dysfunction in children with acute severe bronchial asthma. **Material and Methods:** This was Prospective, observational, Hospital based study at department of pediatric medicine, SPINPH (JK Lone hospital), SMS Medical College, Jaipur, Rajasthan. The study includes 51 patients admitted during study period of 18 months. Sample size was calculated of 95% confidence level, α error of 0.05 expecting 15% abnormal CK-MB levels as one of the biomarker in children with acute severe bronchial asthma. **Result:** The study included Sample size of 51 Patients in each group. In our study, mean age of study participants was 8.8±2.5 years. Out of the 51 participants, 37 were in age of 5-10 years and 14 in age above 10 years. Male participants were 32 and female were 19. Mean FEV1 of study participants was 77.7±5.7%. **Conclusion:** Our study concluded that at the time of admission, children with acute severe bronchial asthma exhibited abnormal CPK-MB in 21.6%, abnormal trop-I in 58.8%, and abnormal BNP in 11.6%. The most common ECG finding in these patients was sinus tachycardia.

## KEYWORDS

Cardiac Biomarkers, ECG Changes, Cardiac Dysfunction, Acute Severe Bronchial Asthma.

## INTRODUCTION

Asthma is a chronic inflammatory disease of the airways, characterized by recurrent episodes of airflow obstruction resulting from edema, bronchospasm, and increased mucus production. Commonly associated with seasonal allergies (allergic rhinitis) and eczema (atopic dermatitis), these three conditions form what is known as the atopic triad.[1]There are an estimated 300 million people who have asthma worldwide, with a significant geographic variation of prevalence, severity, and mortality.

The exact etiology of asthma remains unclear and appears to be multifactorial. Both genetic and environmental factors seem to contribute. One of the most well-studied risk factors during the prenatal period is maternal smoking, which does appear to increase the risk of wheezing in childhood and likely increases risk for the development of asthma. Many childhood exposures have also been the target of research. Tobacco smoke exposure appears to increase the risk of development of asthma and is also a known trigger for exacerbations in those already diagnosed with the disease.

Classic symptoms of asthma include cough, wheezing, chest tightness, and shortness of breath. Symptoms are often episodic and can become triggered by numerous factors, including upper respiratory tract infections, exercise, exposure to allergens, and airway irritants such as tobacco smoke. They may also be worse at night.

Unless otherwise specified, the term "cardiac biomarkers" in the clinical setting refers to cardiac troponins and natriuretic peptides, which have become increasingly popular in recent years, particularly for the diagnosis and risk stratification of numerous cardiovascular diseases all over the world.

Enhanced right ventricular (RV) strain, which is primarily caused by acute increases in pulmonary vascular resistance (PVR), severe hypoxemia, systemic inflammation (cytokine impact, etc.), hemodynamic alterations (tachycardia, etc.), high blood pressure, and other factors may all contribute to high levels of cardiac biomarkers in acute pulmonary conditions. The majority of which may be somewhat related to the severity of the underlying acute pulmonary disease and the prognosis in general.[2,3]

Based on cardiac biomarker levels at admission, the goal of the current study was to determine the prevalence of myocardial dysfunction in patients with acute severe bronchial asthma and to determine whether the dysfunction persisted even after the patient's stabilization or was temporary.

## MATERIAL AND METHOD

This was Prospective, observational, Hospital based study at department of pediatric medicine, SPINPH (JK Lone hospital), SMS Medical College, Jaipur, Rajasthan. The study includes 51 patients admitted during study period of 18 months. Sample size was calculated of 95% confidence level, α error of 0.05 expecting 15% abnormal CPK-MB levels as one of the biomarker in children with acute severe bronchial asthma.

### Inclusion Criteria:

**1.** Children with the age of >5 years admitted in JK Lon hospital with acute severe bronchial asthma. **2.** Severe acute asthma was defined as a condition in which children with acute asthma exacerbation and respiratory distress did not respond to standard dosage of bronchodilators (three nebulizations of salbutamol at interval of 20 minutes or equivalence) and who either had not received corticosteroids as an outpatient or continue to experience respiratory distress despite outpatient treatment.

### Exclusion Criteria:

1. Consent not available 2. Children <5 years of age 3. Children with any other chronic lung disease 4. Children with congenital or acquired heart diseases.

## RESULT

In our study, mean age of study participants was 8.8±2.5 years. Out of the 51 participants, 37 were in age of 5-10 years and 14 in age above 10 years. Male participants were 32 and female were 19. Mean FEV1 of study participants was 77.7±5.7%..

**Figure 1: Distribution of participants according to CK-MB at the time of admission:**

| CK-MB | Frequency | Percent |
|---|---|---|
| Normal (0-25 pg/ml) | 40 | 78.4 |
| Abnormal (>25 pg/ml) | 11 | 21.6 |
| Total | 51 | 100.0 |

In our study, out of the 51 participants, 11 had abnormal CK-MB at the time of admission. In our study, out of the 51 participants, 6 had abnormal BNP at the time of admission.

**Figure 2: Comparison of CK-MB (U/L) at admission and at the time of discharge**

| | CK-MB (U/L) at admission | CK-MB (U/L) at the time of discharge |
|---|---|---|
| Mean | 23.296 | 14.990 |
| Median | 18.000 | 12.000 |
| SD | 17.4403 | 10.2260 |
| Minimum | 5.0 | 5.3 |
| Maximum | 89.0 | 57.0 |
| p-value | 0.0001 | |

In our study, mean CK-MB at admission and at the time of discharge was 23.3±17.4 U/L and 14.9±10.2 U/L This decline of CK-MB was found to be statistically significant.

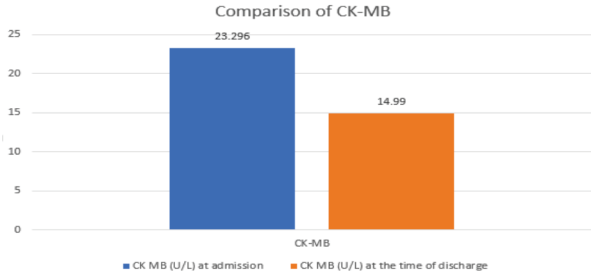



**Figure 3: Comparison of Tn I at admission and at the time of discharge**

| Trop-I | Tn I At admission | | Tn I At the time of discharge | | p-value |
|---|---|---|---|---|---|
| | Frequency | Percent | Frequency | Percent | |
| Normal (0-12 pg/ml) | 21 | 41.2 | 41 | 80.4 | 0.001 |
| Abnormal (>12 pg/ml) | 30 | 58.8 | 10 | 19.6 | |
| Total | 51 | 100.0 | 51 | 100.0 | |

In our study, out of the 51 participants, 30 had abnormal and 21 had normal trop-I at the time of admission while 10 had Tn I abnormal and 41 had normal Tn I at the time of discharge. This difference was found to be statistically significant.

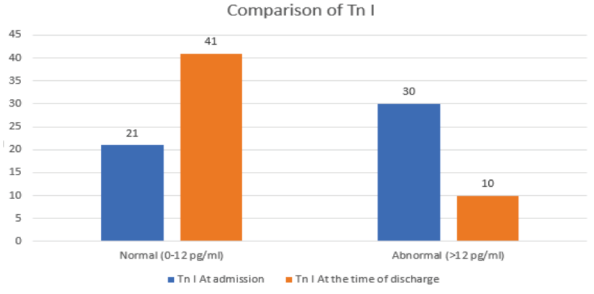



**Figure 4: Comparison of NT-prBNP (pg/ml) at admission and at the time of discharge**

In our study, mean BNP (pg/ml) at admission and at the time of discharge was 86.3±10.13 pg/ml and 50.0±27.0 pg/ml. This decline of BNP was found to be statistically significant.

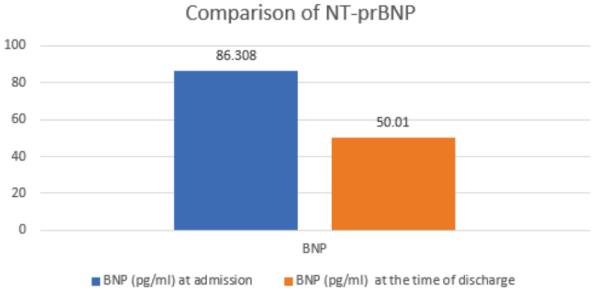
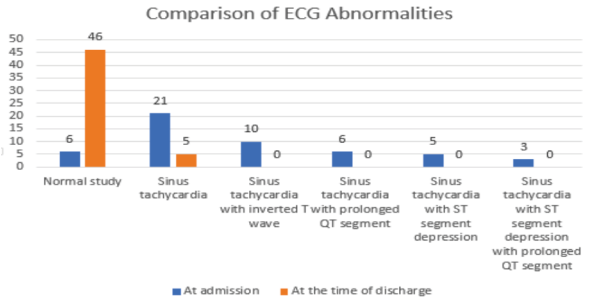



**Figure 5: Comparison of ECG findings at admission and at the time of discharge**

In our study, out of the 51 participants, 6 participants had normal ECG finding and rest were had abnormal (sinus tachycardia) ECG finding at the time of admission while out of the 51 participants, 46 participants had normal ECG finding and rest were had abnormal (sinus tachycardia) ECG finding at the time of discharge. This difference was found to be statistically significant. In our study, mean EF of study participants was 65.4±5.1%.




## DISCUSSION:

Significant acute asthma is distinguished by severe respiratory distress due to increased bronchoconstriction, which results in ventilation perfusion mismatch, hypoxia, respiratory muscle exhaustion, carbon dioxide retention, and respiratory acidosis.

### Cardiac markers and severe acute bronchial asthma:

### CPK-MB:

In our study, out of the 51 participants, 11 (21.6%) had abnormal CPK-MB at the time of admission and mean CPK-MB at admission and at the time of discharge was 23.3±17.4 U/L and 14.9±10.2 U/L. This decline of CPK-MB was found to be statistically significant. (p-0.0001)

Jain M et al[4] conducted a study in which cardiac biomarkers upon admission revealed 15% had abnormally elevated CK-MB levels, similar to our study.

Lovis C et al[5] in a study of 15 patients with acute asthma, 5 of 15 patients had an elevation of CK and CK-MB similar to our study. No patient, however, had an elevation of cardiac troponin-T, indicating no myocardial injury. Plasma myoglobin levels correlated only poorly with total CK elevation.

### Troponine I (Tn I):

In our study, out of the 51 participants, 30 (58.8%) had abnormal Tn I at the time of admission while 10 (19.6%) had Tn I abnormal at the time of discharge. This difference was found to be statistically significant. (p-0.001)

Kalyanaraman M et al[6] studied serial troponin concentrations as a marker of cardiac toxicity in children receiving intravenous terbutaline for status asthmaticus, as well as whether troponin concentrations are affected by asthma severity and risk factors for severe asthma. Cardiac troponin I values ranged from 20 to 4255, with 8 patients having elevated Cardiac Trop I levels (>40mg/ml) on 9 occasions.

Fagbuyi DB et al[7] in a study of 50 patients enrolled, 23 patients (46%; 95% C.I. 32, 60) had either a cTnI elevation or ECG S-T segment change, 12 patients (24%; 95% C.I. 12, 36) had elevated troponin and 15 patients (30%; 95% C.I. 17, 43) had ECG S-T segment change. Four patients (8%; 95% C.I. 1, 15) had both elevated troponin and ECG S-T segment change which parallels the results obtained in our study.

### Brain natriuretic peptide (BNP):

In our study, out of the 51 participants, 6 (11.6%) had abnormal BNP at the time of admission and mean BNP (pg/ml) at admission and at the time of discharge was 86.3±101.3 pg/ml and 50.0±27.0 pg/ml. This decline of BNP was found to be statistically significant. (p-0.0001) Hawkins NM et al[8] looked at BNP levels in patients with acute exacerbation of COPD and found higher BNP levels during the phase of acute exacerbation, similar to our study.

Budweiser *et al*[9] in a study showed a much-generalized role of BNP in assessing disease severity of dyspneic patients. In the study, NT-pro BNP was measured and found to be significantly elevated in patients with chronic hypercapneic respiratory failure compared to healthy controls.

### ECG Findings:

In our study, out of the 51 participants, 45 (88.5%) were had abnormal (sinus tachycardia) ECG finding at the time of admission while out of the 51 participants, 5 (9.8%) had abnormal (sinus tachycardia) ECG finding at the time of discharge. This difference was found to be statistically significant. (p-value-0.001)

Jain M et al[4] in their study on acute severe asthma patients, discovered sinus tachycardia in 90% of their patients, T wave inversion in 35%, ST segment depression in 25%, and QT prolongation in 20%. At the time of discharge, all of the patients had normal ECGs, indicating that the changes were transient and unrelated to overt myocardial dysfunction. We also obtained similar results in this study.

J. Efthimiou et al[10] did a study on acute severe bronchial asthma patients in which twenty-two patients (34%) had inferior lead T-wave inversion on ECGs performed within 1 h of admission. Apart from sinus tachycardia this was the most common ECG abnormality. They concluded that reversible inferior lead T-wave abnormalities may occur in severe acute asthma and appear to be related to the severity of the attack, which is similar to our study.

## CONCLUSION:

Our study concluded that at the time of admission, children with acute severe bronchial asthma exhibited abnormal CPK-MB in 21.6%, abnormal trop-I in 58.8%, and abnormal BNP in 11.6%. The most common ECG finding in these patients was sinus tachycardia.

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