

**Serum Biomarkers in Children with Acute Severe Bronchial Asthma**Shaik Ameer Malik<sup>1</sup>, Ajay Kumar<sup>2</sup>, Vibha Uppal<sup>3</sup><sup>1</sup>MD, Resident, Department of Paediatrics, VMMC & Safdarjung Hospital, New Delhi, India<sup>2</sup>MD, Director Professor, Department of Paediatrics, VMMC & Safdarjung Hospital, New Delhi, India<sup>3</sup>MD, DNB Professor, Department of Biochemistry, VMMC & Safdarjung Hospital, New Delhi, India

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Conflict of interest: Nil

**Abstract:**

**Background:** The National Institutes of Health (NIH) describes asthma as a "Chronic-inflammatory" disease that affects the airways. Over the past few years, there has been an increase in the prevalence of asthma not just in our nation but also globally. Asthma affects the lungs, as we all know, but in cases of acute severe asthma, a hypoxic state can also impact other important organs. In a 2018 study, cardiac biomarkers in acute severe asthma were assessed by a tertiary care hospital of Northern India. Studies on adults with acute severe asthma have demonstrated the involvement of other essential organs as well, although there is insufficient evidence for the paediatric age range. Therefore, the purpose of this study is to assess the dysfunction of the heart, liver, and kidneys in patients with acute severe bronchial asthma based on the baseline biomarker levels at admission and determine whether or not this dysfunction persisted even after the patient's stabilization.

**Methods:** An observational, prospective study conducted over 18 months. The children of age group 3-12 years who presented with acute severe bronchial asthma (pulmonary score > 6) were enrolled into the study after taking informed consent from the parents/guardian and carefully ruling out exclusion criteria. The objective of the study was to evaluate the involvement of vital organs in acute severe asthma in children through estimation of serum levels of cardiac biomarkers [Troponin I, CPK-MB], liver biomarkers [ALT and AST] and kidney biomarkers [Blood urea nitrogen and serum Creatinine]. Blood sample was taken within 12 hours of admission, serve as baseline investigation. Another sampling was done at 48 hours of admission, and these investigations compared with the baseline investigations

**Result:** A total of 65 patients with asthma severity score more than 6 were included in the study. Majority (43.1%) of the children were of the age group of 8-10. The mean age was 10.3 years. Majority (55.38%) of the children were male. 83.07% of the cases had oxygen saturation below 90%, 10.7% had oxygen saturation between 91-95% and 6.1% had oxygen saturation above 95%. There was no statistically significant difference found between the values at the time of admission and 48 hours later in the level of cardiac, liver and kidney biomarkers.

**Conclusion:** This study concludes that in acute severe asthmatics children between 3-12 years of age, there was no association found in the serum biomarkers level of vital organs like Heart, Liver and Kidney at the time of admission and after 48 hours.

**Keywords:** Serum Biomarkers; Children; Acute Severe Asthma; Vital Organs.

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**Introduction**

According to the National Institute for Health (NIH), Asthma is a "chronic-inflammatory" condition affecting the airways, where various cells like eosinophils and the mast-cells, along with others like neutrophils and the macrophages play a role and in people who are susceptible can lead to recurrent episodes of breathlessness with cough and wheeze along with tightness of the chest often seen at the nights or in the early-mornings [1]. Not only in our country but also around the world the prevalence of Asthma is on a raise since last few years [2].

The condition is characterized by lower respiratory tract inflammation which occurs due to a

combination of multiple factors like genetics, environmental factors and in most cases characterized by type-2 inflammation, associated with IL-4,5 and 14, and cells like Type 2 T helper cells, eosinophils and basophils and the mast-cells and also IgE immunoglobulins. Tissue-remodeling (pathological alterations) that occurs in the mucosa and the sub-mucosa of lower respiratory tract includes the hyperplasia of the epithelial cells and the goblet-cell metaplasia and increase mucus production, smooth muscles of the sub-mucosa undergoing hypertrophy with collagen deposition, large mucous glands leading to narrow lumen for air

passage [3]. When bronchial obstruction is severe and does not resolve or gets worse even after receiving appropriate conventional therapy, it is referred to as Acute Severe Asthma, which can result in respiratory failure [4]. Adverse childhood asthma is presently the most frequent cause of paediatric medical emergencies, accounting for around 500,000 PICU admissions annually [5,6]. Viral respiratory infections are a major trigger for acute asthma and occur in approximately 80% of children.

It is known that lungs are severely affected in asthma but in acute severe asthma other vital organs may be affected due to hypoxic state. One study (M Jain et al, 2018) from Banaras Hindu University evaluated the cardiac biomarkers in acute severe asthma and they found that the use of beta 2 agonists in acute severe asthma may increase myocardial workload and cause myocardial infarction in addition to hypoxia and tachycardia. To identify myocardial damage, cardiac biomarkers such as brain natriuretic peptide (BNP), troponin I (TnI), and creatinine kinase muscle/brain (CK-MB) were utilized. In adults, studies have shown that there is involvement of other vital organs as well in acute severe asthma but there was paucity of studies among paediatric age group [7,8]. Another study done by Kapil Meena et al to evaluate the role of cardiac biomarkers and ECG changes in assessing the cardiac dysfunction in children with acute severe asthma and found that during the admission children with acute severe asthma exhibited abnormal CPK-MB as well as abnormal Trop I and the most common ECG finding was sinus tachycardia [9]. So, this study was planned to evaluate the vital organs (heart, liver, kidneys) dysfunction in patients of acute severe bronchial asthma based on their respective biomarker levels at admission as baseline and to see whether this dysfunction was transient or persistent even after the stabilization of the patient.

## Methods

This was an observational, prospective study conducted over 18 months. The children of age group 3-12 years who presented with acute severe bronchial asthma (pulmonary score > 6) [Table 1] to the paediatric emergency ward of a tertiary care hospital in New Delhi were enrolled into the study after taking informed consent from the parents/guardian and carefully ruling out exclusion criteria e.g. child with congenital heart disease OR acquired heart disease, tuberculosis, pneumonia and other chronic lung disease, previous kidney disease and previous liver disease. The ethical clearance was taken by institute ethical committee (S. No. IEC/VMMC/SJH/Thesis/2023-03/CC-229 dated 27.03.2023). The primary objective of the study was to evaluate the involvement of vital organs in acute severe asthma in children through estimation of serum levels of cardiac biomarkers [Troponin I,

CPK-MB], liver biomarkers [ALT and AST] and kidney biomarkers [Blood urea nitrogen and serum Creatinine]. The secondary objective of the study was to assess correlation of duration of hospital stay with serum biomarkers of heart, kidney and liver. 2 ml of blood sample for Troponin I, CPK-MB, Blood urea nitrogen and creatinine, AST and ALT were taken within 12 hours of admission, serve as baseline investigation. Another sampling was done at 48 hours of admission, and these investigations compared with the baseline investigations [Figure 1].

The sample size calculation is based on a study by Jain et al (2018), according to which the expected proportion of deranged markers is 50%. The sample size has been calculated according to the formula given by Lemeshow et al, 1990:

$$\text{Sample size } N = \frac{(z_{(1-\alpha/2)})^2 \times p \times (1-p)}{\delta^2}$$

**Expected Proportion:**  $p = 0.5$  (50%)

**Precision:**  $\delta = 0.125$  (12.5%)

**Type I error:**  $\alpha = 0.05$  (5%),  $(z_{(1-\alpha/2)}) = 1.96$ , Power = 80%. Based on the formula and values given above:  
**Sample size required**  $N = [1.96^2 \times 0.5 \times (1-0.5)] / 0.125^2 = 61.46 \approx 62$ .

Thus, to correctly estimate the desired proportion with a 95% confidence interval (margin of error) of  $\pm 12.5\%$ , the minimum sample size required for the study is 62.

Statistical analysis of the collected data was done. Data was entered in MS excel and analyzed using SPSS 22 version software. Qualitative data was presented in the form of proportions and pie diagrams. Bar charts were used to represent graphically. Quantitative data was presented as mean, standard deviation. Student's t test was the test of significance for quantitative data and chi-square test was the test of significance for qualitative data. P value <0.05 was considered as statistically significant.

## Results

A total of 65 patients with asthma severity score more than 6 were included in the study. Most of the children were of the age group of 8-10 years comprising 43.1% followed by 5-7 years (35.4%) and 11-12 years (21.5%). The mean age in this study was found to be 10.3 years. Majority (55.38%) of the patients were male followed by females (44.62%). According to modified Kuppaswamy scale for socioeconomic classification, out of total 65 patients, majority (30) of the patients belonged to middle-lower class, followed by lower-upper class (16), middle-upper class (14), lower-lower class (4) and upper class (1). In this study 83.07% of the cases had oxygen saturation below 90%, 10.7% had

oxygen saturation between 91-95% and 6.1% had oxygen saturation above 95%.

**Cardiac Biomarkers:** In this study only 7 cases out of total 65, were found to have a positive troponin I at the time of admission with a mean of  $0.188 \pm 0.29$  and a mean of  $0.06 \pm 0.015$ , 48 hours later. There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.32 [Table 2 & Figure 2]. 8 cases were found to have a positive CK-MB at the time of admission with a mean of  $39.875 \pm 3.83$  and 48 hours later a mean of  $38.5 \pm 4.38$ . There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.34 [Table 3 & Figure 3].

**Liver Biomarkers:** In this study 5 cases out of total 65, were found to have a raised ALT values at the time of admission with a mean of  $60 \pm 4.9$  and 48 hours later a mean of  $38.5 \pm 5.24$ . There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.11 [Table 4]. 6 cases were found to have a raised AST values at the time of admission with a mean of  $55.83 \pm 3.3$  and 48

hours later a mean of  $55.8 \pm 2.23$ . There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 1 [Table 5 & Figure 4]. 3 cases were found to have a raised ALP values at the time of admission with a mean of  $390 \pm 10$  and 48 hours later a mean of  $388.34 \pm 18$ . There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.75 [Table 6].

**Renal biomarkers:** In this study only 4 cases out of 65, were found to have a raised serum creatinine values at the time of admission with a mean of  $1.8 \pm 0.36$  and 48 hours later a mean of  $1.75 \pm 0.129$ . There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.35 [Table 7 & Figure 5]. 3 cases were found to have a raised blood urea levels at the time of admission with a mean of  $28.5 \pm 1.26$  and 48 hours later a mean of  $27.25 \pm 2.22$ . There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.40 [Table 8 & Figure 6].

**Table 1: Asthma Severity Score (Pulmonary Score) (Table 1)**

Score	Respiratory rate/min		Wheezing	Accessory muscle use
	<6yrs	>6yrs		
0	<30	<20	None	No apparent activity
1	31-45	21-35	Terminal expiration with stethoscope	Questionable increase
2	46-60	36-50	Entire expiration with stethoscope	Increase apparent
3	>60	>50	During inspiration and expiration without stethoscope	Maximum activity

Score 0-3: Mild, 4-6: Moderate, >6: Severe

If wheezing absent (due to minimal flow or silent chest), score = 3

**Table 2: Comparison of patients with raised troponin I during admission and 48 hours after admission (Table 2)**

Trop I at admission	Trop I after 48 hrs	P value
0.86	0.05	0.32
0.06	0.06	
0.07	0.05	
0.09	0.06	
0.08	0.08	
0.09	0.06	
0.07	0.09	

**Table 3: Comparison of patients with raised CK MB during admission and 48 hours after admission (Table 3)**

CK MB at the time of admission	CK MB after 48 hours	P value
40	45	0.34
38	36	
40	38	
42	40	
36	38	
45	38	
40	40	
38	33	

**Table 4: Comparison of patients with raised ALT during admission and 48 hours after admission (Table 4)**

ALT Level At Admission	ALT Level After 48hrs	P-Value
56	55	0.11
56	54	
60	51	
68	67	
60	58	
60	58	

**Table 5: Comparison of patients with raised AST during admission and 48 hours after admission (Table 5)**

AST level at the time of admission	AST level after 48hours	P value
	56	1
55		
62	60	
56	56	
52	54	
55	54	
55	55	
55	55	

**Table 6: Comparison of patients with raised ALP during admission and and 48 hours after admission (Table 6)**

ALP Level at The Time of Admission	ALP Level After 48hrs	P-Value
380	375	0.75
390	380	
400	410	

**Table 6: Comparison of raised levels of Serum Creatinine at the time of admission and after 48 hours after admission (Table 7)**

S. Creatinine level at the time of admission	S. Creatinine level after 48 hours	P value
1.9	1.8	0.35
2	1.9	
1.5	1.6	
1.8	1.7	

**Table 7: Comparison of raised levels of Blood Urea at the time of admission and after 48 hours after admission (Table 8)**

Blood Urea level at the time of admission	Blood urea level after 48 hours of admission	P value
28	30	0.40
30	28	
28	26	
27	24	

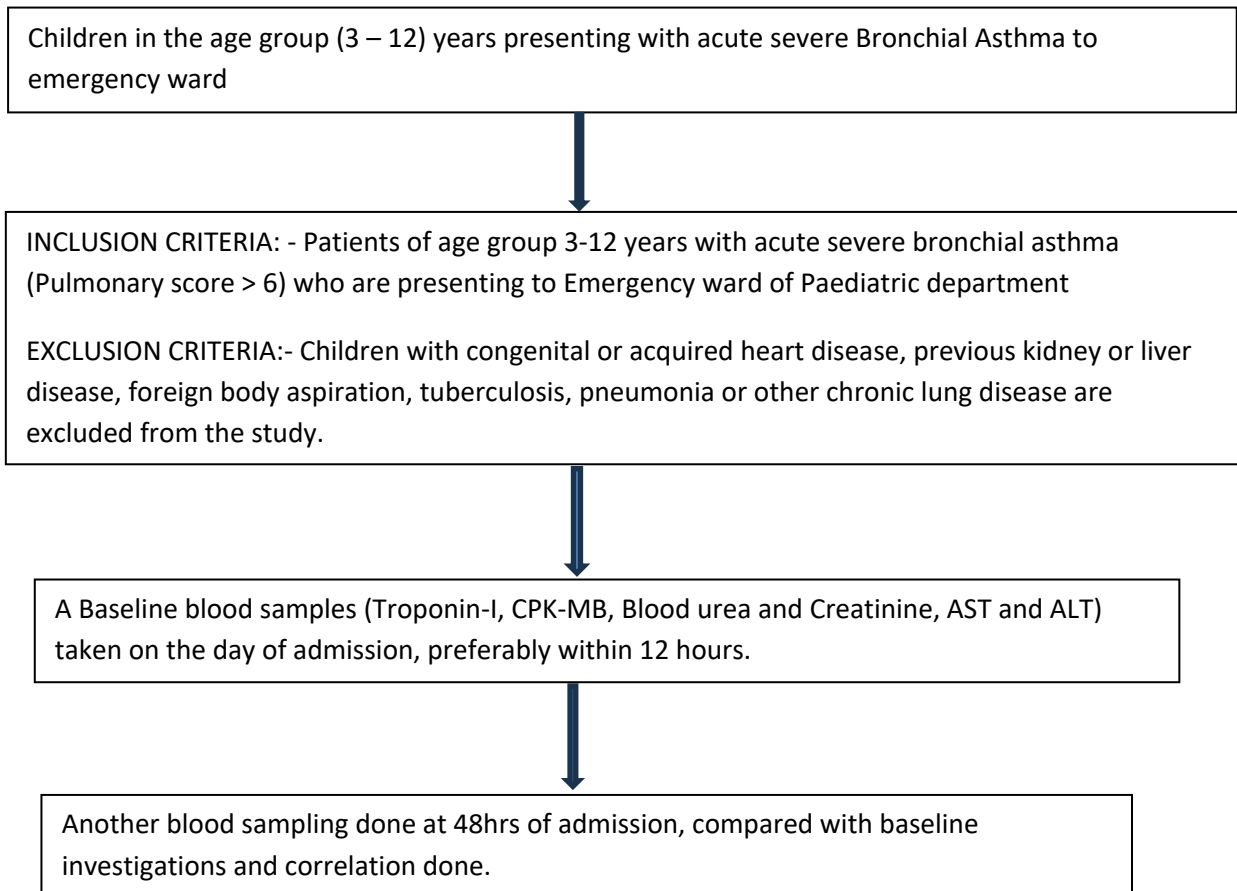


Figure 1: Study Flow

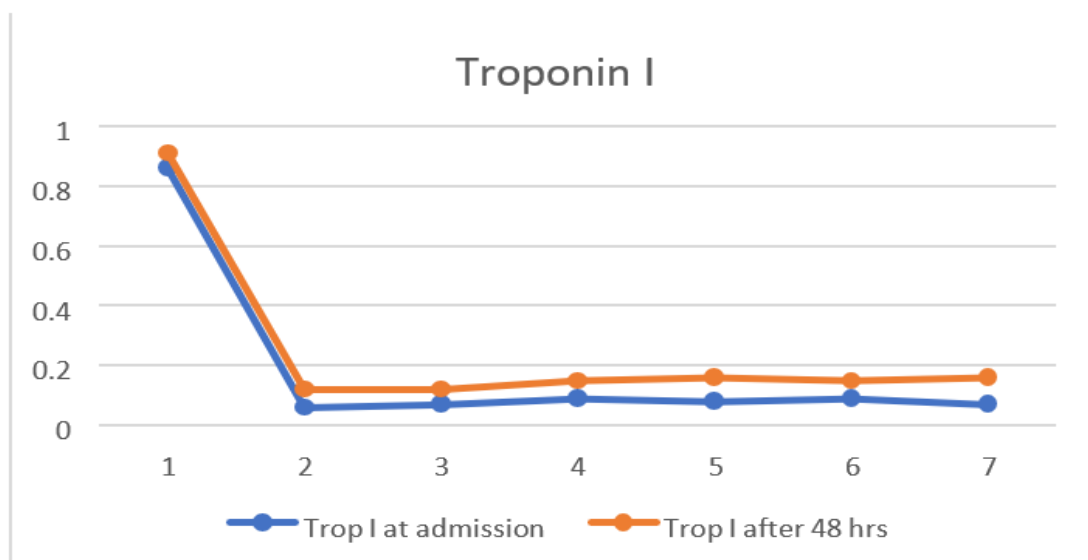


Figure 2: Comparison of patients with raised troponin I during admission and 48 hours after admission

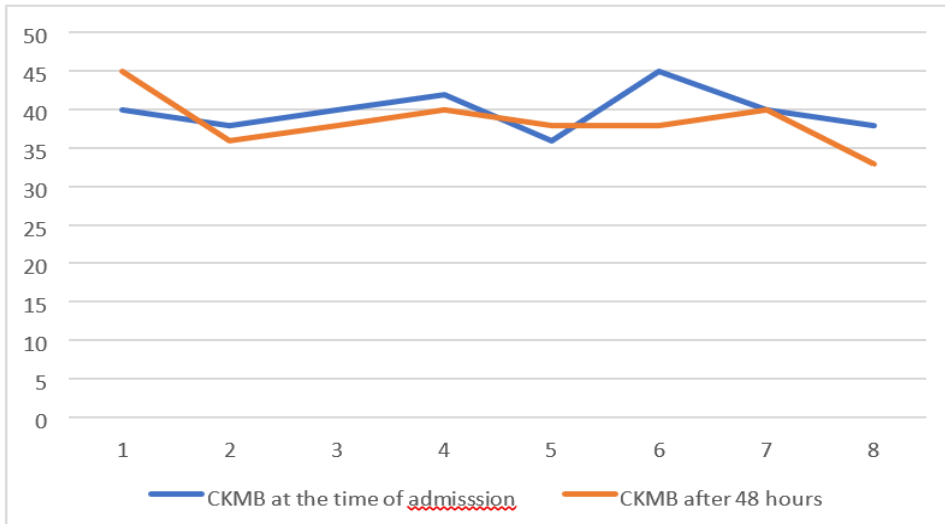


Figure 3: Comparison of patients with raised CK MB during admission and 48 hours after admission

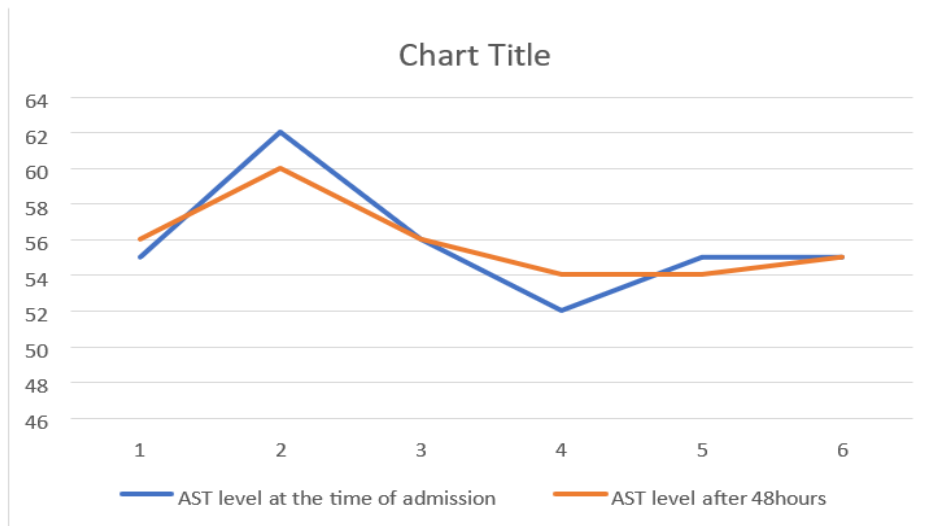


Figure 4: Comparison of patients with raised AST during admission and 48 hours after admission (Figure 4)

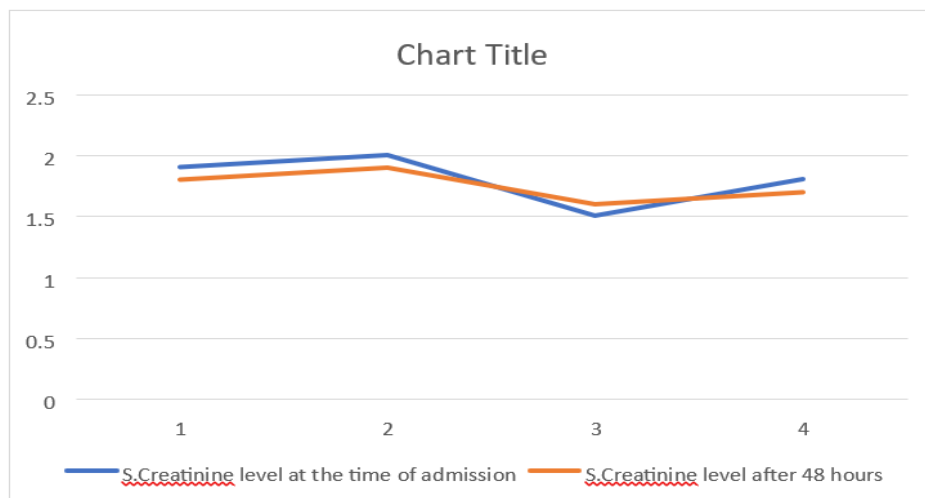
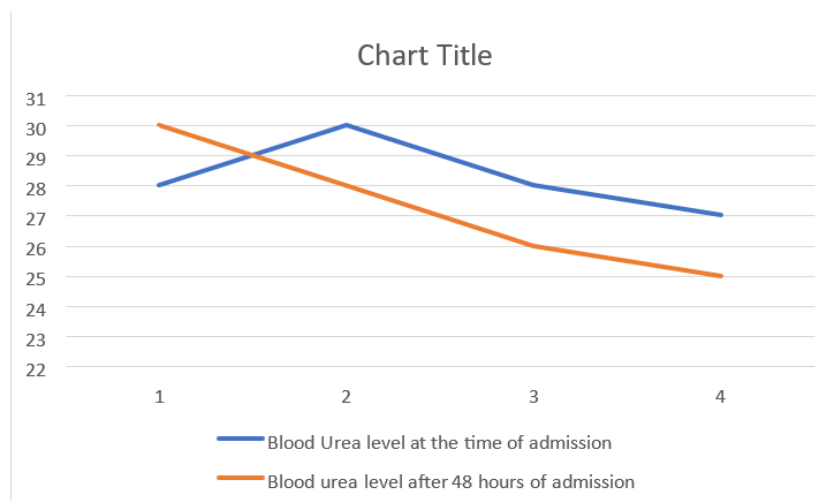


Figure 5: Comparison of raised levels of Serum Creatinine at the time of admission and after 48 hours after admission



**Figure 6: Comparison of raised levels of Blood Urea at the time of admission and after 48 hours after admission**

### Discussion

One of the most prevalent chronic illnesses in children, asthma has become more widespread during the last three decades. Among Indians, asthma was responsible for thousands of deaths and 27.9% of disability-adjusted life years (DALYs). According to a recent study, 7.9% of children have asthma, with boys having a higher frequency than girls. Asthma sufferers might have mild to severe symptoms, but regardless of the severity, they are always susceptible to an immediate, severe exacerbation of their condition. When bronchial obstruction is severe during an acute asthma exacerbation and does not improve or gets worse while receiving adequate standard medication, the condition is known as acute severe asthma, which can result in respiratory failure [10].

In this study most of the children were of the age group of 8-10 years comprising 43.1% followed by 5-7 years age range comprising 35.4% and 11-12 years comprising 21.5%. The mean age in this study was found to be 10.3 years, while study done by M Jain et al the mean age of the patients was 10.28 years and most of the cases (65%) were males, whereas in this study majority of the patients were male comprising 55.38% followed by females accounting for 44.62%. In Kapil Meena et al study the mean age of the study participants was  $8.8 \pm 2.5$  years with majority (32) being males and 19 females [8,9].

In this study, according to Modified Kuppuswamy scale for socioeconomic classification majority of the patients belonged to middle-lower class (30), followed by lower-upper class (16), followed by middle-upper (14), lower-lower (4) and upper (1) whereas in M Jain et al study majority belonged to middle – lower (46.6%), followed by lower-upper and middle-upper each comprising 23.3% followed by lower-lower and upper each comprising 3.3% [8].

M Jain et al study showed that the cardiac biomarker upon admission were elevated, 15% of the cases had abnormal CK-MB levels, 8.3% had abnormal BNP levels, and 50% of the cases had abnormal Tn I levels [8]. In Kapil Meena et al study out of 51 participants, 11 had abnormal CPK MB at the time of admission. The mean CPK MB at admission and at discharge was  $23.3 \pm 17.4$  U/L and  $14.9 \pm 10.2$  U/L respectively, whereas in our study the mean CPK MB at admission and 48 hours later was  $39.87 \pm 3.83$  and  $38.5 \pm 4.38$  respectively [9]. Lovis C et al 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 [11].

In this study 7 cases were found to have a positive troponin I at the time of admission with a mean of 0.188 with a standard deviation of 0.29 and a mean of 0.06 and standard deviation of 0.015. There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.32. 8 cases were found to have a positive CK MB at the time of admission with a mean of 39.875 and a standard deviation of 3.83 and a mean of 38.5 and standard deviation of 4.38. There was no statistically significant difference between the values at the time of admission and 48 hours later as the p value was found to be 0.34.

In research conducted by J. Efthimiou et al. on patients with acute severe bronchial asthma, twenty-two (34%) individuals displayed inferior lead T-wave inversion on ECGs taken within one hour of arrival. With the exception of sinus tachycardia, this was the most typical ECG irregularity. Similar to our investigation, they came to the conclusion that reversible inferior lead T-wave abnormalities may develop in severe acute asthma and appear to be connected to the intensity of the attack [12].

Aysegul Dogan Demir et al in their study looked into the chemicals that cause renal tubular injury as well as the renal tubular functioning of kids who have mild to moderate asthma. It was shown that while renal tubular function had not declined in the research group, children with asthma had higher urine N-acetyl- $\beta$ -d-glucosaminidase (NAG) levels, an early indicator of kidney impairment. In the control and study groups, serum electrolyte and creatinine levels also stayed within normal ranges. Other kidney damage markers, such as urine kidney injury molecule-1 (KIM-1) and microalbumin levels, did not change. Similarly, there were no distinctions seen between children with mild and moderate asthma [13]. They came to the conclusion that children with asthma who had higher urine NAG levels may be better able to identify subtle changes in kidney integrity early on. A straightforward and safe technique for identifying renal tubular injury caused by hypoxia and chronic inflammation is urinary NAG testing. Although the exact source of this impairment is unknown, persistent hypoxia and inflammation may be important contributors. In this study we found out the serum creatinine and blood urea at admission and 48 hours after admission. There was no statistically significant difference found between the values at the time of admission and 48 hours later in serum creatinine and blood urea as the p value was found to be 0.35 and 0.40 respectively.

A Rascu et al study in adult patients found that patients suffering from severe bronchial obstruction due to asthma may be hypoxemic, which lowers the synthesis of enzymes that cause damage to the liver. They came to the conclusion that serum enzymes are crucial indicators for prognosis and diagnosis. The biological relevance keeps growing. More severe asthma is substantially correlated with lower serum levels of AST and ALT [14]. While in this study we found no statistically significant difference between the values at the time of admission and 48 hours later in ALT, AST and ALP as the p value was found to be 0.11, 1.0 and 0.75 respectively.

The strength of this study was being prospective in nature and three vital organs (Heart, Kidney and Liver) studied simultaneously while limitation was small sample size and sample taken from the tertiary care hospital only.

### Conclusion

This study was done to find out the effects of acute severe asthma in major vital organs like Heart, Liver and Kidney in children between 3-12 years of age. The serum biomarkers for these vital organs at the time of admission and after 48 hours was measured and found that there was no association between the values. Hence this study conclude that the findings were transient and was normalized once the patient was stabilized.

### References

1. National Asthma Education and Prevention Program, Third Expert Panel on the Diagnosis and Management of Asthma. Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma. Bethesda (MD): National Heart, Lung, and Blood Institute (US); 2007
2. Dharmage SC, Perret JL, Custovic A. Epidemiology of Asthma in Children and Adults. *Front Pediatr*. 2019
3. Song P, Adeloje D, Salim H, Dos Santos JP, Campbell H, Sheikh A et al. Global, regional, and national prevalence of asthma in 2019: a systematic analysis and modelling study. *J Glob Health*. 2022.
4. Ricklefs I, Barkas I, Duvall M, Cernadas M, Grossman NL, Israel E et al. ALX receptor ligands define a biochemical endotype for severe asthma. *JCI Insight* 2017.
5. Agnihotri N. T., & Saltoun C. (2019). Acute severe asthma (status asthmaticus). *Allergy and Asthma Proceedings*, 40(6), 406–409.
6. Corbridge T, Corbridge S. Acute severe asthma. In: Grammer LC, Greenberger PA, editors. *Patterson's allergic diseases*. 8th ed. Philadelphia, PA: Wolters Kluwer, 2018.
7. Wasserfallen JB, Schaller MD, Feihl F. Sudden asphyxic asthma: a distinct entity? *Am Rev Respir Dis*. 1990.
8. Jain M, Jain D, Das BK, Prasad R, Sihag BK. Evaluation of cardiac biomarkers in children with acute severe bronchial Asthma-A prospective study from tertiary care center in northern India. *Indian Heart J*. 2018 Dec.
9. Meena K, Sehra R N, Meena C L. To evaluate cardiac biomarkers and ECG changes in assessing cardiac dysfunction in children with acute severe asthma. 12(3) DOI: 10.36106/ijrs; 2023
10. Daniel RA, Aggarwal P, Kalaivani M, Gupta SK. Prevalence of asthma among children in India: A systematic review and meta-analysis. *Lung India*. 2022.
11. Christian Lovis; François Mach; Pierre-François Unger; Muriel Bouillie; Jean-Claude Chevrolet. (2001). Elevation of creatine kinase in acute severe asthma is not of cardiac origin, 27(3), 528–533.
12. Efthimiou J, Hassan A B, Ormerod O, Benson M K. Reversible T-wave abnormality in severe acute asthma: an electrocardiographic sign of severity. *Respiratory Medicine*. May 1991, 85(3); P195-202
13. Demir AD, Goknar N, Oktem F, Ozkaya E, Yazici M, Torun E et al. Renal tubular function and urinary N-acetyl- $\beta$ -d-glucosaminidase and kidney injury molecule-1 levels in asthmatic children. *International Journal of Immunopathology and Pharmacology*.

2016;29(4):626-631.  
doi:10.1177/0394632016651448

14. Rascu A, Arghir O, Naghi E, Otelea M. Serum Aminotransferases and the Severity of Asthma. Rev Chim. 2018;1;69:1200–2.