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Original Research Article

A STUDY OF CLINICAL PROFILE, EPIDEMIOLOGICAL FACTORS, SOCIAL IMPACT AND OUTCOME IN CHILDREN ADMITTED WITH ASTHMA EXACERBATION AT A TERTIARY CARE HOSPITALIN CHENNAI

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ABSTRACT

Background: Severe childhood asthma causes frequent hospitalisation and significant clinical, social, and economic burden, requiring a better understanding to improve prevention and management. This study aimed to assess the clinical profile, epidemiological factors, social impact, and outcomes of children admitted with asthma exacerbations at a tertiary care hospital in Chennai. **Materials and Methods:** This hospital-based observational study was conducted in the Department of Paediatrics, Government Stanley Hospital, Chennai, from April 2021 to August 2022. A total of 130 children admitted with moderate to near-fatal asthma exacerbations were enrolled. Clinical details, triggers, inflammatory markers, hospital course, and social impact were analysed using descriptive statistics and Fisher's exact test. **Result:** Most children were aged 6–9 years (54.6%) with a slight female predominance (53.1%). Acute severe exacerbation was the most common presentation (58.5%), followed by life-threatening exacerbation (33.8%). Oxygen therapy via a non-rebreather mask was required in 91.5% at admission. Hospital stay was commonly 3–7 days (70%), while 20.8% stayed longer than 7 days. Viral upper respiratory tract infection (23.1%) and cold, dry air exposure (19.2%) were the leading triggers. Elevated serum IgE levels (1000–5000 IU/mL) were observed in 50%, and absolute eosinophil counts of 1000–3000 cells/µL in 56.2%. Higher IgE and eosinophil levels were significantly associated with greater exacerbation severity ($p < 0.001$). Most children had fewer than two hospitalisations per year, yet activity limitation (65.4%), academic impact, and significant financial burden were common. **Conclusion:** Severe paediatric asthma is closely linked to allergic inflammation and common environmental triggers. Despite limited hospitalisations, the disease imposes a considerable functional and economic burden, highlighting the need for early risk identification and comprehensive long-term care.

INTRODUCTION

Asthma is a long-term airway disease with chronic inflammation. It causes symptoms like wheeze, breathlessness, chest tightness, and cough that change over time and severity. These symptoms are linked with variable expiratory airflow limitation. This definition comes from the Global Initiative for Asthma.[1] Asthma is one of the most common chronic airway disorders and is known for recurrent symptoms, airflow obstruction, bronchial hyper responsiveness, and underlying airway inflammation.[2] Asthma develops due to genetic susceptibility and environmental exposure. Triggers include allergens, respiratory infections, and air pollution. These factors affect disease severity, comorbidities, progression, and treatment response.[3] Patients with similar clinical and biological features are grouped into asthma phenotypes.[4] The commonly described phenotypes are allergic asthma, non-allergic asthma, and adult-onset asthma. Allergic asthma usually starts in childhood and is often linked

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to a history of allergic conditions such as eczema, allergic rhinitis, or food allergy in the child or family members.[2] Allergic asthma is driven by type 2 inflammation. It involves Th2 cells, ILC2 cells, eosinophils, and basophils. These cause mucus excess, airway swelling, narrowing, and remodelling. It is linked with IgE sensitisation and high serum IgE, increasing persistent wheeze and childhood asthma risk.[5,6]

Asthma affects nearly 300 million people worldwide, and childhood asthma is a major cause of emergency visits, hospital admissions, and school absenteeism, particularly in developing countries.[7] The International Study of Asthma and Allergies in Childhood showed wide differences in asthma prevalence among children across different regions.[8] Recent study have reported higher asthma prevalence in rural areas, which challenges the earlier belief that asthma is mainly an urban disease.[9] In India, more than 15 million people are affected by asthma, and childhood prevalence varies between 2% and 18.2%.[10] Study from Tamil Nadu has shown that around 21% of children have asthma-like symptoms, while about 5% have a confirmed diagnosis of asthma based on community-based studies in rural Coimbatore.[11]

Asthma is diagnosed clinically based on symptoms and evidence of variable airflow limitation. In children who require hospital admission, acute asthma exacerbations indicate severe disease. These episodes are often triggered by viral infections, environmental factors, or poor adherence to controller medications.[12] Exacerbations lead to worsening respiratory symptoms, reduced lung function, and frequently require hospital treatment. Asthma exacerbations cause significant clinical, social, and economic problems for children and their families. These include missed school days, restricted physical activity, emotional stress, and financial burden.[13] Indian hospital-based studies integrating clinical severity, inflammatory biomarkers, and social impact in children admitted with asthma exacerbations remain limited. Therefore, this study aims to assess the clinical profile, epidemiological factors, social impact, and outcomes of children admitted with asthma exacerbations at a tertiary care hospital in Chennai.

MATERIALS AND METHODS

This prospective observational study included 130 children admitted with asthma exacerbation in the Department of Paediatrics, Government Stanley Hospital, Chennai, and was conducted from April 2021 to August 2022. Ethical committee approval was obtained, and written informed consent was taken from parents or legal guardians of all participants.

Sample size calculation: Sample size was calculated using data from the American Lung Association with 95% confidence and 1% absolute precision. Mortality proportion was taken as 0.3%. The calculated sample size was 115, which increased to 127 after adding 10% non-response. Finally, 130 children were included. The formula used was $N = Z^2pq / d^2$.14

Inclusion and exclusion criteria

Children aged 4–12 years admitted with asthma exacerbation diagnosed according to GINA 2020 guidelines were included.

Children aged ≤3 and those with wheeze or respiratory distress due to airway anomalies, pneumonia, foreign body aspiration, cardiac, renal, metabolic or systemic illnesses, or immunocompromised status were excluded.

Methods: Clinical data and detailed histories of all enrolled children were collected using a structured pro forma. The proforma included clinical profile, epidemiological factors and social impact variables such as frequency of hospitalisation, school absenteeism, academic performance, relationship with peers, limitation in physical activities, history of domestic violence in the family and socioeconomic burden. Laboratory investigations included serum IgE levels and absolute eosinophil count (AEC) for all children. Basic tests like blood sugar and serum electrolytes were done as part of routine care and not included in the final analysis, as no significant abnormality related to outcomes was noted. Data were collected by trained paediatric residents using a standardised proforma to minimise observer variability.

Triggering factors for asthma exacerbation, such as viral infections, environmental exposure, allergens and comorbid conditions, were recorded. Type and severity of exacerbation, oxygen requirement and duration of hospital stay were documented for each child. The respective unit treated all children as per institutional protocol. No extra cost was charged to patients for investigations. Outcome of treatment, including recovery and discharge status, was recorded for all participants.

Statistical analysis: Data were entered and analysed using SPSS v29. Continuous variables, including age, serum IgE levels, and AEC, were summarised using medians with interquartile ranges. Categorical variables were presented as frequencies and percentages. Associations between exacerbation severity and serum IgE or AEC, as well as between triggering factors, exacerbation type, and duration of hospital stay, were analysed using Fisher's exact test. A p-value < 0.05 was considered statistically significant. There were no missing data for the variables analysed.

RESULTS

A total of 130 children admitted with asthma exacerbation during the study period were screened and included in the analysis, with no exclusions after enrolment. The majority were aged 6–9 years, 71 (54.6%) and were female (69, 53.1%). Acute severe exacerbation was the most common presentation, 76

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(58.5%), followed by life-threatening exacerbation, 44 (33.8%). Most children required oxygen via a non-rebreather mask at admission, 119 (91.5%). Hospital stay was predominantly 3–7 days in duration, 91 (70%), while 27 children (20.8%) required hospitalisation for > 7 days [Table 1].

Table 1: Baseline demographic and clinical characteristics

Parameter	Category	N (%)
Age group (years)	4–5	20 (15.4%)
	6–9	71 (54.6%)
	10–12	39 (30%)
Gender	Male children	61 (46.9%)
	Female children	69 (53.1%)
Type of exacerbation	Moderate	6 (4.6%)
	Acute severe	76 (58.5%)
	Life-threatening	44 (33.8%)
	Near fatal	4 (3.1%)
Severity of illness at admission	Oxygen via NRM (Non-Rebreather Mask)	119 (91.5%)
	Oxygen via JR (Jackson–Rees circuit)	7 (5.4%)
	No oxygen	4 (3.1%)
Duration of hospital stay (days)	< 3	12 (9.2%)
	3-7	91 (70%)
	> 7	27 (20.8%)

NRM: non-rebreather mask; JR: Jackson–Rees circuit.

Viral upper respiratory tract infection was the most common trigger for asthma exacerbation, 30 (23.1%), followed by cold, dry air exposure, 25 (19.2%). Elevated inflammatory markers were frequent, with most children showing serum IgE levels between 1000 and 5000 IU/mL 65 (50%) and AEC between 1000 and 3000 cells/µL 73 (56.2%) [Table 2].

Table 2: Triggering factors and inflammatory biomarker profile

Variable	Category	N (%)
Triggering factor	Viral URTI	30 (23.1%)
	Cold, dry air	25 (19.2%)
	Food allergy/food substance	18 (13.8%)
	Air pollutants (tobacco smoke, CO_2, CO, NO, SO_2, firewood smoke)	16 (12.3%)
	Strong odour (perfume, hair spray, cleaning agents, talcum, incense sticks, mosquito coils)	16 (12.3%)
	Indoor allergens (animal dander, dust, dust mites, cockroach debris, mould, stuffed pets)	13 (10%)
	Exercise/emotion (laughter, crying, hyperventilation)	5 (3.8%)
	Comorbidities (rhinitis, sinusitis, GERD)	5 (3.8%)
	Occupational exposure (farm barn exposure, formaldehyde, paint)	2 (1.5%)
Serum IgE (IU/mL)	230–1000	45 (34.6%)
	1000–5000	65 (50%)
	>5000	20 (15.4%)
Absolute eosinophil count (cells/µL)	300–1000	49 (37.7%)
	1000–3000	73 (56.2%)
	>3000	8 (6.2%)

Most children had fewer than two hospitalisations per year 85 (65.4%). Limitation of daily activities was common, 85 (65.4%), and academic performance was predominantly average, 69 (53.1%). A significant economic burden was observed, with over two-fifths of families spending > 3% of their annual income on asthma-related care 53 (40.8%) [Table 3].

Table 3: Social, educational, and economic impact

Variable	Category	N (%)
Frequency of hospitalisation (times/year)	<2	85 (65.4%)
	≥2	45 (34.6%)
School absenteeism	<3 days/year	58 (44.6%)
	>3 days/year	46 (35.4%)
	>3 days/6 months	26 (20%)
Academic performance	Good	39 (30%)
	Average	69 (53.1%)
	Poor	22 (16.9%)
Relationship with peers	Good	110 (84.6%)
	Being left out	16 (12.3%)
	Being bullied	4 (3.1%)
Limitation in activities	Yes	85 (65.4%)
	No	45 (34.6%)
Domestic violence in the family	Yes	20 (15.4%)

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	No	110 (84.6%)
Economic burden (annual income)	<1%	31 (23.8%)
	1–3%	46 (35.4%)
	>3%	53 (40.8%)

Acute severe exacerbations were most common in children with serum IgE levels of 230–1000 IU/mL (24, 53.3%) and 1000–5000 IU/mL (42, 64.6%), while near-fatal exacerbations occurred only in those with IgE levels >5000 IU/mL (4, 20%), showing a significant association ($p < 0.001$). Similarly, acute severe exacerbations predominated in children with AEC of 300–1000 cells/µL (43, 87.8%), whereas life-threatening (40, 54.8%) and near-fatal exacerbations (4, 50%) were more frequent at higher eosinophil counts, with a significant association ($p < 0.001$) [Table 4].

Table 4: Association of serum IgE levels and AEC with severity of asthma exacerbation

Biomarker	Category	Moderate	Acute severe	Life-threatening	Near-fatal	Fisher's exact test value	p value
Serum IgE (IU/mL)	230–1000	6 (13.3%)	24 (53.3%)	15 (33.3%)	0	22.076	<0.001
	1000–5000	0	42 (64.6%)	23 (35.4%)	0		
	>5000	0	10 (50%)	6 (30%)	4 (20%)		
Absolute eosinophil count (cells/µL)	300–1000	6 (12.2%)	43 (87.8%)	0	0	81.627	<0.001
	1000–3000	0	33 (45.2%)	40 (54.8%)	0		
	>3000	0	0	4 (50%)	4 (50%)		

Viral upper respiratory tract infection was the most common triggering factor and was mainly associated with acute severe exacerbations 16 (21.1%) and a hospital stay of 3–7 days 21 (23.1%). Cold, dry air was more frequent among children with higher serum IgE levels (>5000 IU/mL: 7 [35%]) and was associated with longer hospital stay (>7 days: 6 [22.2%]). No significant association was found between triggering factors and serum IgE levels ($p = 0.415$), type of exacerbation ($p = 0.99$), or duration of hospital stay ($p = 0.415$) [Table 5].

Table 5: Association of triggering factors with serum IgE levels, exacerbation severity, and duration of hospital stay

Variable		Triggering factor for exacerbation									Fisher's exact test value	P value
		Viral URTI	Cold, dry air	Food allergy/ Food substance	Air pollutant	Strong odour	Indoor allergen	Exercise, Emotion	Comorbids	Occupational exposure		
Serum IgE	230-1000	14 (31.1%)	6 (13.3%)	5 (11.10%)	8 (17.8%)	7 (15.6%)	3 (6.7%)	1 (2.2%)	1 (2.2%)	0	15.703	0.415
	1000-5000	14 (21.5%)	12 (18.5%)	11 (16.9%)	4 (6.2%)	7 (10.8%)	8 (12.3%)	4 (6.2%)	3 (4.6%)	2 (3.10%)		
	>5000	2 (10%)	7 (35%)	2 (10%)	4 (20%)	2 (10%)	2 (10%)	0	1 (5%)	0		
Type of exacerbation	Moderate	2 (33.3%)	1 (16.7%)	1 (16.7%)	1 (16.7%)	0	1 (16.7%)	0	0	0	11.803	0.99
	Acute severe	16 (21.1%)	14 (18.4%)	11 (14.5%)	8 (10.5%)	11 (14.5%)	9 (11.8%)	3 (3.9%)	3 (3.9%)	1 (1.3%)		
	Life threatening	11 (25%)	8 (18.2%)	5 (11.4%)	7 (15.9%)	5 (11.4%)	3 (6.8%)	2 (4.5%)	2 (4.5%)	1 (2.3%)		
	Near fatal	1 (25%)	2 (50%)	1 (25%)	0	0	0	0	0	0		
Duration of hospital stay (days)	< 3	4 (33.3%)	2 (16.7%)	1 (8.3%)	1 (8.3%)	1 (8.3%)	1 (8.3%)	0	1 (8.3%)	1 (8.3%)	15.703	0.415
	3-7	21 (23.1%)	17 (18.7%)	14 (15.4%)	12 (13.2%)	10 (11%)	9 (9.9%)	5 (5.5%)	2 (2.2%)	1 (1.1%)		
	> 7	5 (18.5%)	6 (22.2%)	3 (11.1%)	3 (11.1%)	5 (18.5%)	3 (11.1%)	0	2 (7.4%)	0		

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DISCUSSION

This study found that school-aged children commonly presented with severe asthma attacks requiring oxygen and short hospital stays. Viral infections and cold air were frequent triggers, with many children showing high IgE and eosinophil levels. Although hospitalisations were usually limited, asthma significantly affected daily activities, school performance, and family finances. Higher inflammatory markers were linked to more severe exacerbations, highlighting the burden of childhood asthma beyond hospital care.

Similarly, Major et al. found that the mean age at admission was 10.3 ± 3.4 years, indicating school-age predominance, with a near-equal sex distribution (56.9% male). All children had severe asthma exacerbations; 13.9% required mechanical ventilation. Median hospital stay was 5 days (IQR 3–7) and ICU stay 1 day (IQR 1–3).[15] Alherbish et al. reported that among 906 ED visits for moderate-to-severe asthma, 157 (17.3%) required admission; hypoxemia and tachypnea were key admission predictors.[16] Thus, similar age groups, gender, severe asthma attacks needing oxygen or ventilation, and comparable hospital stays, confirming that our patient profile and disease severity match those seen in other paediatric asthma settings. Variations across studies may reflect differences in study design, trigger ascertainment methods, timing of biomarker measurement, and healthcare settings.

In our study, viral infections and cold air were common triggers, with frequent eosinophilic and IgE-mediated inflammation among affected children. Similarly, Trivedi and Patel found in a paediatric cohort, 86.2% had elevated serum IgE, increasing with severity to 1399 ± 693 IU/mL in moderate and 2031 ± 553.9 IU/mL in severe asthma. Elevated AEC occurred in 56%, reaching 1147.7 ± 893.1 cells/µL in severe disease ($p < 0.0001$).[17] Zhao et al. reported that viral antigens were detected in 44% of wheezing children. RSV-associated attacks were moderate–severe in 82% versus 36% with influenza A ($p < 0.05$). Severe RSV exacerbations showed higher eosinophil counts, supporting viral-triggered eosinophilic inflammation.[18] These findings showing that viral infections commonly trigger severe asthma attacks and are linked with higher IgE and eosinophil levels, confirming that inflammation plays a key role in worsening asthma, similar to what we observed in our patients.

In this study, most children had limited hospitalisations but significant activity restriction, moderate academic impact, and significant financial burden from asthma care. Similarly, Sharifi et al. found that over one year, children had 0–3 emergency visits (mean 0.07) and 0–11 hospital days (mean 0.17). School absence ranged 1–34 days (mean 1.7). Mean annual asthma cost was USD 368, higher in 7–11-year-olds ($p = 0.008$), confirming functional and economic burden.[19] This showing that even with few hospital visits, asthma still disrupts daily activities, affects school attendance, and creates financial strain for families, confirming that the overall burden of childhood asthma extends beyond hospitalisation alone.

In our study, higher asthma severity clustered with higher IgE and eosinophil levels, indicating a strong link between inflammatory burden and exacerbation severity. Similarly, Trivedi and Patel reported that mean serum IgE increased with asthma severity from 231.9 ± 182.6 IU/mL (intermittent) to 2031 ± 553.9 IU/mL (severe) ($p < 0.0001$). Mean AEC rose from 411 ± 299 to 1147.7 ± 893 cells/µL with increasing severity ($p < 0.0001$).[17] Bossley et al. found that children with severe therapy-resistant asthma had higher serum IgE (median 386 IU/mL vs 40 IU/mL in controls; $p < 0.001$) and significantly increased airway eosinophils (BAL eosinophils 2.7% vs 0%; $p < 0.001$), supporting more severity with elevated IgE and eosinophilic inflammation.[20] Therefore, children with more severe asthma have higher IgE and eosinophil levels, confirming that stronger allergic and eosinophilic inflammation is closely linked to worse asthma attacks, as seen in our study.

Our study showed that viral and cold air triggers demonstrated trends with severity and hospital stay, but no significant associations were identified. Similarly, Khetsuriani et al. found that respiratory viruses were detected in 63.1% of children with acute asthma versus 23.4% with controlled asthma (OR 5.6, $p < 0.001$). Viral positivity did not vary with severity indicators or hospitalisation history (all $p > 0.05$), supporting a lack of association with length of stay.[21] Ahn and Choi show that cold air–related symptoms occurred in 34.7% of children. Total IgE did not differ between cold dry air–positive and negative groups ($p = 0.704$), and no associations were found with exacerbation severity, lung function, or clinical outcomes (all $p > 0.05$), supporting the lack of trigger–severity correlation.[22] Although viral infections and cold air are common asthma triggers, they do not consistently predict attack severity or hospital stay, confirming that trigger exposure alone does not determine how severe an asthma episode becomes.

Strengths of this study include prospective data collection, comprehensive clinical and inflammatory profiling, and inclusion of social and economic outcomes.

Clinical implications: These findings suggest that early identification of children with high inflammatory burden and targeted trigger avoidance may help inform optimisation of asthma management in similar clinical settings. Future studies should include multicentre cohorts with long-term follow-up to guide targeted prevention, early intervention, and personalised asthma management strategies.

Limitations: This was a single-centre study; the results may not reflect all settings. Some information depended on parental recall. Measurements were taken only at specific times, and the lack of long-term

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follow-up limits understanding of ongoing asthma outcomes.

CONCLUSION

Severe asthma in children is commonly seen in school-aged groups and is often associated with viral infections, cold air exposure, and allergic inflammation. Most children required oxygen therapy but had relatively short hospital stays. Elevated IgE levels and eosinophilia were linked with more disease severity. Hospital admissions were limited, asthma significantly affected daily activities, schooling, and family finances. These findings highlight the need for early recognition of high-risk children and focused long-term asthma management.

REFERENCES

1. Goldin J, Cataletto ME. Asthma. StatPearls, Treasure Island (FL): StatPearls Publishing; 2025. https://www.ncbi.nlm.nih.gov/books/NBK430901/.
2. Custovic A, Johnston SL, Pavord I, Gaga M, Fabbri L, Bel EH, et al. EAACI position statement on asthma exacerbations and severe asthma. Allergy 2013;68:1520–31. https://doi.org/10.1111/all.12275.
3. Apter AJ. Advances in adult asthma diagnosis and treatment in 2012: potential therapeutics and gene-environment interactions. J Allergy Clin Immunol 2013;131:47–54. https://doi.org/10.1016/j.jaci.2012.11.020.
4. Lötvall J, Akdis CA, Bacharier LB, Bjermer L, Casale TB, Custovic A, et al. Asthma endotypes: a new approach to classification of disease entities within the asthma syndrome. J Allergy Clin Immunol 2011;127:355–60. https://doi.org/10.1016/j.jaci.2010.11.037.
5. León B. Understanding the development of Th2 cell-driven allergic airway disease in early life. Front Allergy 2022;3:1080153. https://doi.org/10.3389/falgy.2022.1080153.
6. Wenzel SE. Asthma phenotypes: the evolution from clinical to molecular approaches. Nat Med 2012;18:716–25. https://doi.org/10.1038/nm.2678.
7. Martinez FD, Vercelli D. Asthma. Lancet 2013;382:1360–72. https://doi.org/10.1016/S0140-6736(13)61536-6.
8. Mallol J, Crane J, von Mutius E, Odhiambo J, Keil U, Stewart A, et al. The International Study of Asthma and Allergies in Childhood (ISAAC) Phase Three: a global synthesis. Allergol Immunopathol (Madr) 2013;41:73–85. https://doi.org/10.1016/j.aller.2012.03.001.
9. B M R, Shailaja S P, B M S, S P V. Pediatric asthma: Prevalence and Socio-cultural factors affecting asthma management in a rural area of northern Karnataka. Indian J Community Med 2021;46:24–9. https://doi.org/10.4103/ijcm.IJCM_85_20.
10. Daniel RA, Aggarwal P, Kalaivani M, Gupta SK. Prevalence of asthma among children in India: A systematic review and meta-analysis: A systematic review and meta-analysis. Lung India 2022;39:357–67. https://doi.org/10.4103/lungindia.lungindia_706_21.
11. Balakrishnan S, Karunanidhi R, Pandian R, Ramamurthy S, Gurusamy S, Chacko TV. Prevalence of asthma and its associated risk factors among children aged 5-10 years in a rural area of Coimbatore: A community-based cross-sectional study. Cureus 2024;16:e69329. https://doi.org/10.7759/cureus.69329.
12. Graham BL, Steenbruggen I, Miller MR, Barjaktarevic IZ, Cooper BG, Hall GL, et al. Standardisation of spirometry 2019 update. An official American Thoracic Society and European Respiratory Society technical statement. Am J Respir Crit Care Med 2019;200:e70–88. https://doi.org/10.1164/rccm.201908-1590ST.
13. Merghani MM, Mohmmed RGA, Al-Sayaghi KM, Alshamrani NSM, Awadalkareem EM, Shaaib H, et al. Asthma-related school absenteeism: Prevalence, disparities, and the need for comprehensive management: A systematic review and meta-analysis. Dialogues Health 2025;7:100239. https://doi.org/10.1016/j.dialog.2025.100239.
14. Asthma and children fact sheet. American Lung Association. Available from: www.https://www.lung.org/lung-health-diseases/lung-disease- lookup/asthma/learn-about-asthma/asthma-children-facts-sheet. Accessed 02 Dec 2021.
15. Major S, Vézina K, Tse SM. Lung function of children following an intensive care unit admission for asthma. Pediatr Allergy Immunol Pulmonol 2021;34:1–6. https://doi.org/10.1089/ped.2020.1271.
16. Alherbish M, Mobaireek KF, Alangari AA. Admission predictability of children with acute asthma. Ann Thorac Med 2018;13:36–41. https://doi.org/10.4103/atm.ATM_197_17.
17. Trivedi PP, Patel AH. Serum immunoglobulin E and absolute eosinophil count as markers of severity in childhood asthma. Int J Contemp Paediatrics 2020;7:413. https://doi.org/10.18203/2349-3291.ijcp20200120.
18. Zhao J, Takamura M, Yamaoka A, Odajima Y, Iikura Y. Altered eosinophil levels as a result of viral infection in asthma exacerbation in childhood: Eosinophil levels in virus-induced asthma exacerbation. Pediatr Allergy Immunol 2002;13:47–50. https://doi.org/10.1034/j.1399-3038.2002.00051.x.
19. Sharifi L, Dashti R, Pourpak Z, Fazlollahi MR, Movahedi M, Chavoshzadeh Z, et al. Economic burden of pediatric asthma: Annual cost of disease in Iran. Iran J Public Health 2018;47:256–63. https://pmc.ncbi.nlm.nih.gov/articles/PMC5810389/.
20. Bossley CJ, Fleming L, Gupta A, Regamey N, Frith J, Oates T, et al. Pediatric severe asthma is characterised by eosinophilia and remodelling without T(H)2 cytokines. J Allergy Clin Immunol 2012;129:974-82.e13. https://doi.org/10.1016/j.jaci.2012.01.059.
21. Khetsuriani N, Kazerouni NN, Erdman DD, Lu X, Redd SC, Anderson LJ, et al. Prevalence of viral respiratory tract infections in children with asthma. J Allergy Clin Immunol 2007;119:314–21. https://doi.org/10.1016/j.jaci.2006.08.041.
22. Ahn JY, Choi BS. Application of a cold dry air provocation test in pediatric patients with asthma. Children (Basel) 2022;9:920. https://doi.org/10.3390/children9060920.

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