Research Article

# Evaluation if Risk Factors, Resource Utilization and Outcome of Critical Acute Asthma in PICU

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**ABSTRACT**

**Background:** Childhood asthma is the most common chronic illness in children, affecting approximately 10% of the pediatric population worldwide. Critical asthma, defined as acute severe asthma requiring PICU admission due to deteriorating clinical status and inadequate response to initial therapy, is a life-threatening condition.

**Aim:** To evaluate risk factors, resource utilization, and outcomes of critical acute asthma in pediatric patients who are admitted to intensive care units.

**Methods:** This case-control study was conducted in the Pediatric Intensive Care Units of Cairo University Children's Hospital (Abou El-Reesh) between April 2024 and August 2025. The study included 82 children aged 1-14 years with confirmed asthma diagnosis, divided into two groups: 41 children with critical acute asthma requiring PICU admission (cases) and 41 children with well-controlled asthma attending outpatient clinics (controls). Comprehensive data collection included demographic information, asthma history, medication adherence, environmental exposures, clinical examination, laboratory investigations, and virological screening. Asthma control was assessed using the validated Asthma Control Questionnaire (ACQ-6). Healthcare resource utilization and outcomes were documented for PICU patients.

**Results**: Cases and controls were comparable regarding age, sex, disease duration, and age at asthma onset ($p>0.05$). Critical acute asthma was significantly associated with family history of asthma/allergy ($p=0.046$), previous PICU admissions ($p=0.007$), frequent emergency room visits ($p<0.001$), poor controller therapy adherence ($p=0.036$), inadequate inhalation technique ($p=0.034$), lower leukotriene receptor antagonist use ($p<0.001$), obesity ($p=0.023$), eczema ($p=0.019$), allergic rhinitis ($p=0.023$), and poorer asthma control (ACQ-6 score; $p<0.001$). Environmental factors including allergy, air pollution, passive smoking, animal exposure, and seasonal symptom variation were also significantly associated with critical acute asthma ($p<0.05$). Among PICU patients, 43.9% had positive viral screening, predominantly respiratory syncytial virus. High-flow nasal cannula was the most commonly used respiratory support modality (51.2%). Clinical improvement occurred in 92.7% of cases, while mortality was 4.9%.

**Conclusion:** Critical acute asthma in children is strongly associated with poor asthma control, inadequate treatment adherence, allergic comorbidities, recurrent healthcare utilization, and environmental exposures. Despite generally favorable outcomes, significant healthcare resources are required for management. Early optimization of asthma control, adherence, and trigger avoidance may reduce the risk of severe exacerbations requiring PICU admission.

**Keywords:** Critical Asthma, Pediatric Intensive Care Unit. Resource Utilization, Medication Adherence.

## Introduction

Asthma is the most common chronic disease in children and one of the most significant chronic respiratory conditions affecting the pediatric population worldwide, causing substantial health, economic, and social challenges for patients, families, and healthcare systems [1, 2]. Pediatric asthma is characterized by variable expiratory airway limitation and persistent respiratory symptoms, including wheezing, coughing, shortness of breath, and chest tightness, with airway hyperresponsiveness ranging from intermittent symptoms to potentially life-threatening airway compromise [1].

The progression from routine asthma management to the need for critical care represents a significant increase in disease severity with important implications for patient outcomes and healthcare resource utilization [3]. Children with critical acute asthma account for approximately 20% of admissions to the pediatric intensive care unit (PICU) [1]. Severe acute asthma is an asthma attack that is unresponsive to repeated doses of inhaled β-agonists, oral or intravenous steroids, and oxygen therapy, representing the most extreme manifestation of asthma exacerbation [4].

Critical Acute Asthma is a severe, life-threatening exacerbation of asthma characterized by rapid onset of respiratory distress, hypoxemia, and poor response to second-line asthma treatment. These patients require intensive therapy such as continuous bronchodilator infusions, non-invasive or mechanical ventilation, and close

monitoring in a pediatric intensive care unit. Without medical intervention, this condition can progress to respiratory failure, making early recognition and management essential to prevent morbidity and mortality **[5]**. Pediatric mortality related to asthma is estimated at approximately 10.6 deaths per million **[6]**. Higher mean heart rate and respiratory rate, along with lower $SpO_2$ within the first hour of emergency department presentation, have been identified as key severity indicators in asthmatic children for PICU admission, with a higher proportion of admissions observed among those presenting with tachycardia and tachypnea **[7]**.

Risk factors that predispose to severe asthma exacerbation requiring PICU admission include: age (preschool children have significantly higher risk compared to older children aged 5-14 years), preceding upper respiratory tract illness, malnutrition, comorbidities such as gastroesophageal reflux and obesity, poor asthma control, and previous PICU admission [4].

The outcomes of critical acute asthma in children range from complete recovery to serious complications. Outcomes can be favorable if prompt and effective management is provided. However, PICU admission carries medical, financial, and social burdens for both the patient and their family, depending on the severity of presentation, early initiation of treatment, and effectiveness of therapeutic interventions. Critical acute asthma can lead to several serious complications, including respiratory failure, pneumothorax, and the need for mechanical ventilation. These complications can prolong PICU stays, increase the risk of long-term pulmonary issues, and significantly impact quality of life [8, 9]. So, this study aimed to evaluate risk factors, resource utilization, and outcomes of critical acute asthma in pediatric patients admitted to intensive care units.

## Patients and methods

This case-control study was conducted in the Pediatric Intensive Care Units (PICUs) of Cairo University Children's Hospital (Abou El-Reesh) between April 2024 and August 2025 to compare children with critical acute asthma and children with well-controlled asthma attending the outpatient clinic. The study included male and female children aged 1-14 years with a confirmed diagnosis of asthma, either previously established or newly diagnosed based on clinical evaluation. The critical acute asthma group consisted of patients presenting with severe exacerbations characterized by marked respiratory distress, respiratory rate >40 breaths/min, oxygen saturation <90%, and accessory muscle use requiring immediate medical intervention and PICU admission if unresponsive to initial treatment, while the control group included patients attending routine follow-up without an acute attack. Patients with other causes of wheezing, cystic fibrosis, congestive heart failure, chronic obstructive pulmonary disease, chronic neuromuscular disorders, or severe developmental delay were excluded.

## Methods

A comprehensive history was obtained from all participants, including demographic data (age and sex), current asthma symptoms, indicators of poor asthma control, adherence to maintenance therapy, and assessment of asthma control using a validated questionnaire. Detailed asthma-related information was also collected, including disease duration, age at symptom onset, triggering factors, association with exercise, seasonal and diurnal variation, response to asthma treatment, changes during travel, exposure to passive smoking, presence of allergies and comorbidities, family history of asthma or allergic diseases, animal exposure, long-term medication use and inhaler technique, as well as the frequency of asthma exacerbations and hospitalizations during the previous 12 months.

Clinical Examination included assessment of respiratory rate, heart rate, oxygen saturation ($SpO_2$), wheeze intensity on auscultation and blood pressure. Laboratory and radiological investigations were performed according to clinical indications. Virological screening for respiratory viruses, serum potassium measurement, respiratory panel testing, arterial blood gases, complete blood count (CBC), C-reactive protein (CRP) and blood cultures were carried out only in patients with critical acute asthma when infection or other relevant conditions were suspected. These investigations were not routinely performed in the control group unless specifically warranted by clinical assessment. Chest radiography was obtained in both groups only when clinically indicated, such as in cases of suspected complications, including pneumonia, rapid clinical deterioration, or the development of new or worsening respiratory symptoms.

## Respiratory Support Documentation (for PICU patients)

For children admitted to the PICU with critical acute asthma, detailed information regarding respiratory and supportive management was recorded. Data included the need for mechanical ventilation and the type of respiratory support provided, whether invasive mechanical ventilation with endotracheal intubation or non-invasive modalities such as high-flow nasal cannula (HFNC), continuous positive airway pressure (CPAP), and

bilevel positive airway pressure (BiPAP). Ventilator parameters, including $FiO_2$, respiratory rate, peak inspiratory pressure (PIP), and positive end-expiratory pressure (PEEP), were also documented. In addition, the requirement for other supportive measures, including inotropic support for hemodynamic instability, potassium replacement for electrolyte disturbances, management of acute kidney injury, and sedation, was recorded to assess the overall severity of illness and support needs of PICU patients.

## Assessment of Disease Control

Disease control was assessed using an asthma control questionnaire that evaluated the frequency of daytime symptoms, nocturnal awakenings, use of rescue medications, and activity limitation. Poor asthma control was defined by the presence of daytime asthma symptoms more than twice weekly, nighttime awakening due to asthma symptoms more than twice monthly, use of quick-relief medication more than twice weekly, or restriction of normal physical activities because of asthma symptoms [10].

The Asthma Control Questionnaire (ACQ-6), a validated tool for assessing asthma control in children, was administered to all participants by trained personnel during routine clinic visits. The questionnaire consists of six items, each scored from 0 to 6, with higher scores indicating poorer asthma control; the overall score is calculated as the mean of the six items. Participants were assessed regarding asthma symptoms during the preceding week, including nocturnal awakenings, morning symptoms, activity limitation, and shortness of breath.

## Risk Factor Analysis

Potential risk factors for critical acute asthma were evaluated in all participants. These included age at presentation, history and frequency of previous asthma exacerbations, presence of allergies or other atopic conditions, exposure to environmental tobacco smoke, adherence to prescribed asthma medications, and the adequacy of prophylactic treatment administration techniques. These factors were analyzed to identify possible contributors to the development of severe asthma exacerbations requiring intensive care admission.

## Resource Utilization Recording (for PICU patients)

Healthcare resource utilization was documented for all patients admitted to the PICU with critical acute asthma. Recorded data included the use of medications such as bronchodilators, corticosteroids, and magnesium sulfate, the duration of PICU stay in days, and any additional supportive measures or therapeutic interventions required during admission. These parameters were assessed to evaluate the intensity of care and healthcare resources utilized in the management of critically ill asthmatic children.

## Follow-up and Outcome Assessment

All patients admitted to the PICU were followed until hospital discharge to evaluate clinical outcomes, including mortality, morbidity, and length of hospital stay. The primary outcomes were the identification of risk factors associated with critical or fatal asthma and the assessment of mortality among patients with critical acute asthma. Secondary outcomes included hospital-related outcomes such as morbidity, asthma control, and activity limitation, as well as healthcare resource utilization, including medications, therapeutic interventions, mechanical ventilation requirements, and duration of PICU stay. Asthma control was additionally evaluated using the Asthma Control Questionnaire (ACQ-6).

## Sampling Technique

A convenient sampling method was employed. Patients meeting the inclusion and exclusion criteria were consecutively enrolled until the target sample size was reached. Cases were matched to controls for comparison.

## Sample size

The sample size was determined based on the methodology and risk factors identified in a prospective multicenter case-control study on PICU admission in children with severe acute asthma **[11]**. Based on a two-sided significance level of 0.05 and 80% power, a total of 82 asthmatic children were required. Participants were recruited using a consecutive convenience sampling method and matched 1:1 into two groups: 41 children with critical acute asthma (cases) and 41 children with non-acute asthma (controls).

## Ethical Considerations

The study was conducted after obtaining approval from the Research Ethics Committee, Faculty of Medicine, Cairo University, under protocol code MD-71-2024, with approval dated 11 May 2024. The approved protocol was entitled “Evaluation of risk factors, resource utilization and outcome of critical acute asthma in PICU”. Written informed consent was obtained from the parents or legal guardians of all participating children after a full explanation of the study procedures. All collected participant information was treated confidentially, and numerical codes were used instead of participants’ names to protect their identity and privacy. The study procedures involved the collection of clinical, demographic, laboratory, treatment, and outcome data as described in the approved protocol. The study was approved for implementation by the Research Ethics Committee, and the researchers complied with the requirements of the ethics committee regarding the conduct and reporting of the study.

## Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 22. Descriptive statistics were used to summarize the data, with qualitative variables presented as frequencies and percentages, while quantitative variables were expressed as mean±standard deviation for normally distributed data or median and interquartile range for non-normally distributed data. Inferential statistical analyses included the Chi-square test for categorical variables, Student’s t-test for normally distributed continuous variables, and Mann-Whitney U test for non-normally distributed continuous variables. A p-value ≤ 0.05 was considered statistically significant.

## Results

Table (1) summarizes the baseline demographic and clinical characteristics of the study population. Age (7.49±2.99 vs 8.40±2.91 years; p=0.166), age-group distribution (p=0.407), sex (males: 53.7% vs 46.3%; p=0.579), disease duration (3.83±2.17 vs 4.70±2.33 years; p=0.086), and age at onset (3.60±1.72 vs 3.46±1.70 years; p=0.721) did not differ significantly between cases and controls. However, cases were significantly more likely to have a family history of asthma/allergy (65.9% vs 43.9%; p=0.046), prior PICU admissions in the last 12 months (1.85±1.05 vs 0.46±0.98; p=0.007), ≥2 PICU admissions (41.5% vs 19.5%; p=0.031), prior ER visits (4.33±2.30 vs 1.88±2.23; p<0.001), and ≥3 ER visits (43.9% vs 22.0%; p=0.034).

Table (2) presents asthma control, medication adherence, and comorbidities. Compared with controls, cases had significantly lower compliance with controller therapy (14.6% vs 39.0%; p=0.036), less regular LTRA use (34.1% vs 73.2%; p<0.001), and less frequent correct inhalation technique (75.6% vs 92.7%; p=0.034). Inhaled glucocorticoid use (58.5% vs 63.4%; p=0.651) and long-acting beta-2 agonist use (68.3% vs 73.2%; p=0.627) were similar between groups. Obesity (22.0% vs 4.9%; p=0.023), eczema (26.8% vs 7.3%; p=0.019), and allergic rhinitis (22.0% vs 4.9%; p=0.023) were significantly more common among cases. ACQ-6 scores were markedly higher in cases (3.19±2.01 vs 1.01±1.67; p<0.001); no case was well controlled compared with 36.6% of controls, and 39.0% of cases were extremely poorly controlled compared with 0% of controls (p<0.001).

Table (3) explains environmental triggers and temporal patterns. Cases were significantly more likely to report allergy (46.3% vs 22.0%; p=0.020), air pollution (39.0% vs 17.1%; p=0.027), passive smoking (24.4% vs 4.9%; p=0.012), animal contact (29.3% vs 9.8%; p=0.026), and seasonality of symptoms (87.8% vs 65.9%; p=0.019). Exercise-induced symptoms (78.0% vs 82.9%; p=0.577), cold/flu triggers (85.4% vs 85.4%; p=1.000), and circadian variations (56.1% vs 56.1%; p=1.000) were comparable between groups.Table (4) summarizes vital signs, clinical, radiological, laboratory, and microbiological findings during admission among patients with critical acute asthma (n=41). Mean heart rate was 110.43±17.54 beats/min, respiratory rate was 37.15±6.34 breaths/min, systolic blood pressure was 102.33±8.04 mmHg, diastolic blood pressure was 63.43±9.39 mmHg, and $SpO_2$ was 89.78±3.79%. Laboratory values included hemoglobin 11.98±1.56 g/dL, lymphocytes 25.16±8.42%, WBCs 6.77±3.33 $\times 10^9$/L, pH 7.37±0.07, $PCO_2$ 40.08±9.53 mmHg, $HCO_3^-$ 19.40±3.23 mmol/L, base deficit 5.36±2.77 mmol/L, and CRP 9.29±9.33 mg/L. Wheeze was severe in 61.0% of patients and silent chest was present in 14.6%. Chest X-ray was normal in 43.9%, showed hyperinflation in 19.5%, increased bronchovascular markings in 31.7%, and pneumonia in 4.9%. Salbutamol-associated low potassium occurred in 12.2%, and blood cultures were positive in 9.8%, with E. coli, Pseudomonas, Salmonella, and Staphylococcus spp. each isolated in 2.4% of patients.Table **(5)** reports virological findings, medication use, and non-invasive mechanical ventilation after PICU admission (n=41). Virology screening was positive in 43.9% of patients; RSV was detected in 29.3% overall (66.7% of positive cases), rhinovirus and influenza in 4.9% each, and adenovirus and COVID-19 in 2.4% each. During the first hour, continuous SABA was used in 70.7% and intermittent SABA in 29.3%. Steroids were used in 65.9%, IV $MgSO_4$ in 51.2%, and mean SABA frequency after the first hour was 2.8±1.02. Among steroid-

treated patients, hydrocortisone was used in 63.0% and methylprednisolone in 37.0%, with a mean steroid dose of 53.73±34.8 mg and duration of 1.82±0.79 days. Non-invasive support included HFNC in 51.2%, CPAP in 24.4%, and BIPAP in 22.0%; none/invasive ventilation or missing data accounted for 2.4%.

Table (6) presents treatment response, outcome, and post-discharge compliance. Improvement was observed in 92.7% of patients, while 7.3% did not improve. Overall, 95.1% improved and were discharged, and 4.9% died. Post-discharge compliance data were reported for 41 patients: 20 (48.8%) were compliant and 21 (51.2%) were non-compliant.

Table (7) examines the association between medication use and non-invasive mechanical ventilation (MV) duration. Systemic corticosteroid use was not associated with MV duration (2.88±1.28 vs 3.00±1.47 days; p=0.797). $MgSO_4$ use was associated with significantly shorter MV duration (2.45±1.32 vs 3.35±1.37 days; p=0.038), as was continuous SABA use (2.61±1.20 vs 3.67±1.37 days; p=0.026).

**Table 1.** Baseline Demographic and Clinical Characteristics of the Study Population

| Parameter | Cases (n=41) | Controls (n=41) | Test statistic | p-value |
|---|---|---|---|---|
| Age (years), Mean±SD | 7.49±2.99 | 8.40±2.91 | t=−1.40 | 0.166 |
| **Age groups, n (%)** | | | | |
| 3–5 years | 8 (19.5%) | 4 (9.8%) | $\chi^2$=1.79 | 0.407 |
| 5–10 years | 22 (53.7%) | 24 (58.5%) | | |
| ≥10 years | 11 (26.8%) | 13 (31.7%) | | |
| **Gender, n (%)** | | | | |
| Males | 22 (53.7%) | 19 (46.3%) | $\chi^2$=0.31 | 0.579 |
| Females | 19 (46.3%) | 22 (53.7%) | | |
| **Disease duration** (years), Mean±SD | 3.83±2.17 | 4.70±2.33 | t=−1.75 | 0.086 |
| **Age at onset** (years), Mean±SD | 3.60±1.72 | 3.46±1.70 | t=0.36 | 0.721 |
| **Family history of asthma/allergy**, n (%) | 27 (65.9%) | 18 (43.9%) | $\chi^2$=3.99 | 0.046 (S) |
| **Prior PICU admissions** (last 12 mo), Mean±SD | 1.85±1.05 | 0.46±0.98 | U=574.50 | 0.007 (S) |
| ≥2 **PICU admissions**, n (%) | 17 (41.5%) | 8 (19.5%) | $\chi^2$=4.66 | 0.031 (S) |
| **Prior ER visits** (last 12 mo), Mean±SD | 4.33±2.30 | 1.88±2.23 | U=312.00 | <0.001 (S) |
| **≥3 ER visits**, n (%) | 18 (43.9%) | 9 (22.0%) | $\chi^2$=4.47 | 0.034 (S) |

(S)=statistically significant (p<0.05). t=independent-samples t-test; $\chi^2$=Chi-square test; U=Mann–Whitney U test.

**Table 2.** Asthma Control, Medication Adherence, and Comorbidities

| **Parameter** | Cases (n=41) | Controls (n=41) | Test statistic | p-value |
|---|---|---|---|---|
| **Compliance to controller therapy, n (%)** | | | | |
| **Compliant** | 6 (14.6%) | 16 (39.0%) | $\chi^2$=6.61 | 0.**036** (S) |
| **Non-compliant** | 33 (80.5%) | 25 (61.0%) | | |
| **Not prescribed** | 2 (4.9%) | 0 (0.0%) | | |
| **Regular use of LTRA, n (%)** | 14 (34.1%) | 30 (73.2%) | $\chi^2$=12.56 | <0.**001** (S) |
| **Inhaled glucocorticoid use, n (%)** | 24 (58.5%) | 26 (63.4%) | $\chi^2$=0.21 | 0.651 |
| **Long-acting beta-2 agonist use, n (%)** | 28 (68.3%) | 30 (73.2%) | $\chi^2$=0.24 | 0.627 |
| **Correct inhalation technique, n (%)** | 31 (75.6%) | 38 (92.7%) | $\chi^2$=4.48 | 0.**034** (S) |
| **Associated comorbidities, n (%)** | | | | |
| **Obesity** | 9 (22.0%) | 2 (4.9%) | $\chi^2$=5.14 | 0.**023** (S) |
| **Eczema** | 11 (26.8%) | 3 (7.3%) | $\chi^2$=5.51 | 0.**019** (S) |
| **Allergic rhinitis** | 9 (22.0%) | 2 (4.9%) | $\chi^2$=5.14 | 0.**023** (S) |
| **ACQ-6 score, Mean±SD** | 3.19±2.01 | 1.01±1.67 | t=5.34 | <0.**001** (S) |
| **ACQ interpretation, n (%)** | | | | |
| **Well controlled** | 0 (0.0%) | 15 (36.6%) | $\chi^2$=31.00 | <0.**001** (S) |
| **Adequately controlled** | 12 (29.3%) | 6 (14.6%) | | |
| **Partly controlled** | 9 (22.0%) | 12 (29.3%) | | |

| | | | | |
|---|---|---|---|---|
| **Not well controlled** | 4 (9.8%) | 8 (19.5%) | | |
| **Extremely poorly controlled** | 16 (39.0%) | 0 (0.0%) | | |

LTRA=leukotriene receptor antagonist; ACQ-6=Asthma Control Questionnaire. (S)=statistically significant.

**Table 3.** Environmental Triggers and Temporal Patterns

| Parameter, n (%) | Cases (n=41) | Controls (n=41) | Test statistic | p-value |
|---|---|---|---|---|
| **Allergy** | 19 (46.3%) | 9 (22.0%) | $\chi^2$=5.42 | **0.020 (S)** |
| **Air pollution** | 16 (39.0%) | 7 (17.1%) | $\chi^2$=4.89 | **0.027 (S)** |
| **Passive smoking** | 10 (24.4%) | 2 (4.9%) | $\chi^2$=6.25 | **0.012 (S)** |
| **Animal contact** | 12 (29.3%) | 4 (9.8%) | $\chi^2$=4.97 | **0.026 (S)** |
| **Exercise-induced symptoms** | 32 (78.0%) | 34 (82.9%) | $\chi^2$=0.31 | 0.577 |
| **Cold/flu triggers** | 35 (85.4%) | 35 (85.4%) | $\chi^2$=0.00 | 1.000 |
| **Seasonality of symptoms** | 36 (87.8%) | 27 (65.9%) | $\chi^2$=5.55 | 0.**019** (S) |
| **Circadian variations** | 23 (56.1%) | 23 (56.1%) | $\chi^2$=0.00 | 1.000 |

All variables analyzed using Chi-square test. Percentages are column percentages based on n=41 per group. (S)=statistically significant (p<0.05).

**Table 4.** Vital Signs, Clinical, Radiological, Laboratory, and Microbiological Data of Patients with Critical Acute Asthma During Admission (n=41)

| Parameter | Mean±SD (Min–Max) or n (%) | % of cases (n=41) |
|---|---|---|
| HR during attack (beats/min) | 110.43±17.54 (42–145) | — |
| RR during attack (breaths/min) | 37.15±6.34 (28–55) | — |
| Systolic BP (mmHg) | 102.33±8.04 (85–120) | — |
| Diastolic BP (mmHg) | 63.43±9.39 (47–89) | — |
| $SpO_2$ (%) | 89.78±3.79 (79–98) | — |
| **Laboratory data** | | |
| Hemoglobin (g/dL) | 11.98±1.56 (9–15) | — |
| Lymphocytes (%) | 25.16±8.42 (10–43) | — |
| WBCs ($\times 10^9$/L) | 6.77±3.33 (2–14) | — |
| pH | 7.37±0.07 (7.25–7.54) | — |
| $PCO_2$ (mmHg) | 40.08±9.53 (25–69) | — |
| $HCO_3^-$ (mmol/L) | 19.40±3.23 (16–34) | — |
| Base deficit (mmol/L) | 5.36±2.77 (2–15) | — |
| C-reactive protein (mg/L) | 9.29±9.33 (0.0–50.0) | — |
| **Wheeze intensity, n (%)** | | |
| Mild | 1 | 2.4% |
| Moderate | 9 | 22.0% |
| Severe | 25 | 61.0% |
| Silent chest | 6 | 14.6% |
| **Total** | **41** | **100.0%** |
| **Chest X-ray findings, n (%)** | | |
| No abnormality | 18 | 43.9% |
| Hyperinflation | 8 | 19.5% |
| Increased bronchovascular markings | 13 | 31.7% |
| Pneumonia | 2 | 4.9% |
| **Total** | **41** | **100.0%** |
| **Low K by salbutamol, n (%)** | | |
| Yes | 5 | 12.2% |
| No | 36 | 87.8% |
| **Total** | **41** | **100.0%** |
| **Blood culture, n (%)** | | |
| Positive | 4 | 9.8% |
| Negative | 37 | 90.2% |

| | | |
|---|---|---|
| **Total** | **41** | **100.0%** |
| **Organism isolated, n (%)** | | |
| E. coli | 1 | 2.4% |
| Pseudomonas | 1 | 2.4% |
| Salmonella | 1 | 2.4% |
| Staphylococcus spp. | 1 | 2.4% |

WBCs=white blood cells; CRP=C-reactive protein; HR=heart rate; RR=respiratory rate; BP=blood pressure; $SpO_2$=oxygen saturation.

**Table 5.** Virological Findings, Medication Use, and Non-Invasive Mechanical Ventilation Following PICU Admission Among Patients with Critical Acute Asthma (n=41)

| Parameter | n (%) or Mean±SD (Range) | % of total cases (n=41) where applicable |
|---|---|---|
| **Virology screening (n=41)** | | |
| Positive virology | 18 | 43.9% |
| Negative virology | 23 | 56.1% |
| **Virus detected (n=41)** | | |
| RSV positive | 12 | 29.3% (66.7% of positives) |
| Rhinovirus positive | 2 | 4.9% (11.1% of positives) |
| Influenza positive | 2 | 4.9% (11.1% of positives) |
| Adenovirus positive | 1 | 2.4% (5.6% of positives) |
| COVID-19 positive | 1 | 2.4% (5.6% of positives) |
| **Total positive** | **18** | **43.9%** |
| **Medications after PICU admission (n=41)** | | |
| Continuous SABA in 1st hour | 29 | 70.7% |
| Intermittent SABA in 1st hour | 12 | 29.3% |
| Steroid use | 27 | 65.9% |
| IV $MgSO_4$ use | 21 | 51.2% |
| SABA frequency after 1st hour | 2.8±1.02 (1–6) | — |
| **Steroid therapy (n=27)** | | |
| Hydrocortisone | 17 | 63.0% of steroid-treated |
| Methylprednisolone | 10 | 37.0% of steroid-treated |
| **Total** | **27** | **100.0%** |
| Steroid dose (mg) | 53.73±34.8 (0–100) | — |
| Steroid duration (days) | 1.82±0.79 (1–3) | — |
| **Non-invasive mechanical ventilation (n=41)** | | |
| BIPAP | 9 | 22.0% |
| CPAP | 10 | 24.4% |
| HFNC | 21 | 51.2% |
| None/invasive ventilation or missing | 1 | 2.4% |
| **Total** | **41** | **100.0%** |
| **BIPAP settings (n=9)** | | |
| $FiO_2$ (%) | 58.44±16.30 (45–100) | — |
| Rate (breaths/min) | 27.78±4.41 (23–34) | — |
| PIP ($cmH_2O$) | 20.67±1.00 (20–23) | — |
| **CPAP settings (n=10)** | | |
| $FiO_2$ (%) | 56.00±12.43 (40–80) | — |
| Pressure ($cmH_2O$) | 5.20±0.42 (5–6) | — |
| **HFNC settings (n=21)** | | |
| $FiO_2$ (%) | 49.95±18.77 (21–100) | — |
| Oxygen flow (L/min) | 8.00±1.73 (5–10) | — |

BIPAP=bilevel positive airway pressure; CPAP=continuous positive airway pressure; HFNC=high-flow nasal cannula; $FiO_2$=fraction of inspired oxygen; PIP=peak inspiratory pressure; RSV=respiratory syncytial virus; SABA=short-acting beta-2 agonist.

**Table 6.** Treatment Response, Outcome, and Post-Discharge Compliance Among Critical Acute Asthma Patients

| Parameter | n (%) | % of total cases (n=41) |
|---|---|---|
| **Treatment response (n=41)** | | |
| Improved | 38 | 92.7% |
| Not improved | 3 | 7.3% |
| **Outcome (n=41)** | | |
| Improved and discharged | 39 | 95.1% |
| Died | 2 | 4.9% |
| **Compliance after discharge (n=41)** | | |
| Yes | 20 | 48.8% |
| No | 21 | 51.2% |

**Table 7.** Relation Between Medication Use and Non-Invasive Mechanical Ventilation Duration in Patients with Critical Acute Asthma

| Medication group | n | MV duration, Mean±SD (Range) | Test statistic | p-value |
|---|---|---|---|---|
| **Systemic corticosteroids** | | | | |
| Yes | 27 | 2.88±1.28 (1–5) | t=−0.26 | 0.797 |
| No | 14 | 3.00±1.47 (1–6) | | |
| **$MgSO_4$** | | | | |
| Yes | 21 | 2.45±1.32 (1–5) | t=−2.14 | 0.038 (S) |
| No | 20 | 3.35±1.37 (1–6) | | |
| **Continuous SABA** | | | | |
| Yes | 29 | 2.61±1.20 (1–5) | t=−2.34 | 0.026 (S) |
| No | 12 | 3.67±1.37 (1–6) | | |

MV=mechanical ventilation; $MgSO_4$=magnesium sulfate; SABA=short-acting beta-2 agonist; (S)=statistically significant ($p<0.05$). Counts were corrected to match Table 5 and to sum to n=41. Independent-samples t-tests were used.

## Discussion

The aim of work of the current study was to evaluate risk factors, resource utilization, and outcomes of critical acute asthma in pediatric patients admitted to intensive care units. To clarify this aim, 41 children (males and females) with acute severe asthma who were admitted at Pediatric Intensive Care Units (PICUs) and 41 well-controlled asthmatic children following in the pediatric clinic of Cairo University Children Hospital were included in this study.

In the present study, the majority of children in both cases and controls fell within the 5–10-year age range, with 53.7% of cases and 58.5% of controls belonging to this category. The mean age was comparable between groups (7.49±2.99 years in cases versus 8.40±2.91 years in controls), and no significant difference was observed in gender distribution, with males comprising 53.7% of cases and 46.3% of controls. These demographic findings align with those reported by Boeschoten et al. (2020), who conducted a prospective study in the Netherlands involving 110 PICU patients and 111 general ward patients and identified that PICU children were significantly older (median age 8 years) compared to general ward children (median age 6 years) **[11]**.

The mean disease duration in the present study did not differ significantly between cases and controls (3.83±2.17 years versus 4.70±2.33 years, p=0.086), nor did the age at onset (3.60±1.72 years versus 3.46±1.70 years, p=0.721). These observations suggest that the duration of asthma diagnosis alone may not be a primary determinant of critical exacerbation risk, a finding consistent with Selmanoglu et al. (2025), who in their retrospective study of 83 patients admitted to PICU due to severe asthma exacerbation found that 44.6% of patients required escalation in GINA treatment steps during long-term follow-up ($p<0.001$) **[12]**.

In the current study, compliance with controller therapy before admission was significantly lower among cases, with only 14.6% demonstrating adherence compared with 39.0% of controls. Non-compliance rates were substantially higher in the cases group, representing a statistically significant difference (p=0.036). These findings demonstrate the role of medication adherence in preventing acute severe asthma exacerbation.

These results are supported by the recent work of Arcoleo et al. (2025), who demonstrated in their school-based asthma therapy study involving 633 children in Columbus that improving medication adherence through direct administration of daily controller medications by school nurses significantly reduced healthcare utilization. Their findings showed remarkable improvements: emergency department visits were reduced by 49%, urgent care visits by 41%, hospitalizations by 50%, and pediatric intensive care unit admissions by 71% ($p<0.001$) within one year of enrollment. Additionally, their intervention achieved Asthma Control Test improvements of 37% overall ($p<0.001$). These data directly parallel our observation that non-compliant patients were more likely to experience critical episodes requiring intensive care admission [13].

Correct use of inhalation technique was significantly lower among cases than controls (75.6% versus 92.7%, $p=0.034$), suggesting that poor technique may contribute to inadequate medication delivery and subsequent disease control failure. Volerman et al. (2021) similarly emphasized in their comprehensive review on improving inhalation technique in children that poor inhaler technique is highly prevalent, with observational studies showing only 8–22% of children with asthma using their inhalers correctly, and that improper inhaler use contributes to poorly controlled asthma with resultant exacerbation, acute care utilization, and hospitalizations [14].

The present study found a highly significant disparity in leukotriene receptor antagonist (LTRA) use, with only 34.1% of cases using these medications regularly compared with 73.2% of controls ($p<0.001$). In contrast, no significant differences were observed between cases and controls regarding the use of long-acting beta-2 agonists (68.3% versus 73.2%, $p=0.627$) or inhaled glucocorticoids (58.5% versus 63.4%, $p=0.651$). This suggests that leukotriene receptor antagonists may confer additional protective benefits against severe exacerbation beyond those provided by inhaled corticosteroids alone. However, contrasting evidence comes from Price et al. (2011), who conducted two parallel trials published in the New England Journal of Medicine and found that at 2 months, LTRA showed equivalence to LABA as add-on therapy, suggesting that while LTRAs may be effective, the superiority over other add-on therapies in preventing severe exacerbations specifically may vary depending on patient phenotype and adherence patterns [15].

Our results showed that cases were significantly more likely than controls to report passive smoking exposure (24.4% versus 4.9%, $p=0.012$), allergy (46.3% versus 22.0%, $p=0.020$), animal contact (29.3% versus 9.8%, $p=0.026$), and air pollution (39.0% versus 17.1%, $p=0.027$) as triggers. These findings highlight the substantial burden of environmental and atopic risk factors among children who develop critical acute asthma.

The association between passive smoking and severe asthma outcomes in the current study aligns with the prospective findings of Boeschoten et al. (2020), who demonstrated that environmental tobacco smoke exposure was a clinically relevant strong risk factor for PICU admission [11]. Regarding pet exposure, Ojwang et al. (2020) studied 3,781 children and found that having a pet in the house during the first year of life was associated with increased risk of asthma (HR 0.60; 95% CI 0.38–0.96, $p<0.05$), allergic rhinitis (OR 0.72; 95% CI 0.53–0.97, $p<0.05$), and atopic sensitization (OR 0.77; 95% CI 0.63–0.96, $p<0.05$) [16]. Their findings suggest that the timing of pet exposure may be crucial: early-life exposure during infancy may offer protection, while ongoing exposure in sensitized older children (as in our study population with mean age 7–8 years) may trigger exacerbation, particularly among those already sensitized to animal allergens.

The present study found that a significantly higher proportion of cases reported seasonality in their symptoms compared with controls (87.8% versus 65.9%, $p=0.019$), indicating that seasonal factors may contribute more prominently to critical exacerbation. The relationship between symptoms and exertion was similarly common in both groups (78.0% of cases versus 82.9% of controls, $p=0.577$), and circadian variation did not differ significantly between cases and controls (56.1% versus 56.1%, $p=1.000$).

Our results showed that type of feeding (breastfeeding, formula, or mixed) and history of low birth weight did not differ significantly between cases and controls ($p=0.131$ and $p>0.05$, respectively; data not shown), suggesting that these early-life factors may not be primary determinants of critical asthma risk later in childhood. Abate et al. (2025) revealed in their systematic reviews and meta-analyses that non-exclusive breastfeeding increases the risk of childhood asthma by 20% (pooled OR 1.20, 95% CI 1.12–1.29, $p<0.001$) and asthma exacerbations by 15% (OR 1.15, 95% CI 1.06–1.24, $p=0.001$). While breastfeeding has been associated with reduced asthma severity, our study findings indicate its protective effect may be overshadowed by other more proximal risk factors such as medication adherence, environmental exposures, and baseline disease control in determining which children progress to critical illness [17]. Contrasting evidence regarding severe exacerbations specifically comes from Dogaru et al. (2014), who performed a systematic review and meta-analysis of 117 studies and found strong protective associations for breastfeeding against asthma incidence but inconsistent effects on asthma severity markers [18].

In the present study, cases demonstrated significantly higher rates than controls of obesity (22.0% versus 4.9%, p=0.023), eczema (26.8% versus 7.3%, p=0.019), and allergic rhinitis (22.0% versus 4.9%, p=0.023). These findings indicate a stronger burden of atopy and metabolic dysfunction among children who develop critical acute asthma.

The association between obesity and severe asthma is supported by the work of Ross et al. (2019), who analyzed data from the National Survey of Children's Health and identified distinct obese-related and allergic-related asthma phenotypes among children across the United States [19]. Their analysis of 4,427 children with asthma demonstrated that the phenotype with the highest risk for more severe compared to mild asthma was the obese-and-allergic asthma phenotype (p<0.001). They found that obesity-related asthma (with or without allergic components) comprised approximately one-fifth of asthma phenotypes in the cohort, and children with both obesity and allergic asthma were most likely to have severe disease. The clustering of atopic conditions including eczema and allergic rhinitis among our cases suggests that children with multiple manifestations of allergic disease may be at heightened risk for severe asthma exacerbation.

The present study identified a statistically significant association between family history of asthma/allergy and critical acute asthma, with 65.9% of cases reporting positive family history compared with 43.9% of controls (p=0.046). The higher prevalence of positive family history among cases in our study suggests that children with familial predisposition may require more aggressive preventive strategies and closer monitoring for early signs of poor control.

This finding is supported by Al Ghadeer et al. (2024), who studied 124 asthmatic children and found that 58.9% had a family history of asthma. However, their study found that family history was not significantly associated with emergency room visits (p=0.817) or hospitalizations (p=0.441), suggesting that while family history may be common among asthmatic children, it may not independently predict acute severe exacerbation requiring hospitalization [20]. Similarly, Selmanoglu et al. (2025) found that while 20.5% of PICU patients had a positive family history of atopic disease, family history was not identified as a significant risk factor for GINA step escalation [12].

The present study found that children with critical acute asthma presented with elevated heart rates (mean 110.43±17.54 beats per minute), increased respiratory rates (mean 37.15±6.34 breaths per minute), and reduced oxygen saturation (mean 89.78±3.79%). These clinical parameters reflect the severe physiological derangement characteristic of critical asthma episodes. In agreement with our findings, Freedman & Forno (2023) demonstrated in their retrospective observational study of 5,185 emergency department encounters that initial vital signs were predictive of subsequent PICU admission in pediatric patients presenting with asthma exacerbation. They found that PICU admission increased 70% for each 10 beats per minute higher heart rate (OR 1.70, 95% CI 1.54–1.87, p<0.001), 125% for each 10 breaths per minute higher respiratory rate (OR 2.25, 95% CI 1.93–2.62, p<0.001), and 34% for each 5% lower oxygen saturation (OR 1.34, 95% CI 1.18–1.52, p<0.001) [7].

In the present study, the majority of children with critical acute asthma presented with severe wheeze (61.0%), while 14.6% exhibited a silent chest, an ominous sign indicating minimal airflow. Chest radiograph abnormalities were present in 56.1% of patients, with increased bronchovascular markings (31.7%) and hyperinflation (19.5%) being the most common findings. Pneumonia was detected in 4.9% of cases. Eusebe et al. (2025) reported similar clinical presentations in their ICU-3A study [21].

Blood cultures were positive in 9.8% of patients, with diverse organisms identified including Escherichia coli, Pseudomonas, Salmonella, and Staphylococcus species. While the rate of bacterial co-infection was relatively low, the identification of these pathogens in critically ill children highlights the importance of considering secondary bacterial infection in patients with severe asthma exacerbations. Mirhosseini et al. (2024) found that mixed bacterial infections, particularly involving Staphylococcus aureus, were common triggers of asthma attacks, emphasizing the need for vigilance regarding bacterial co-infection in children with prolonged symptoms or incomplete response to standard bronchodilator therapy [22].

Hypokalemia attributable to salbutamol administration occurred in 12.2% of cases in the current study. This finding aligns with several recent studies that have reported hypokalemia following salbutamol use in children with acute asthma. Hung et al. (2020), in a study of 433 hospitalized children, found that 38.8% developed hypokalemia after salbutamol administration [23]. Similarly, Singh et al. (2024), in a study of 67 children, demonstrated a significant reduction in serum potassium levels following repeated nebulized doses [24]. These findings emphasize that hypokalemia is a relatively common side effect, particularly with high or repeated doses, highlighting the importance of close clinical and laboratory monitoring during intensive bronchodilator therapy.

The present study found that viral testing was positive in 43.9% of children with critical acute asthma, with respiratory syncytial virus being the most frequently detected pathogen (29.3% of total cases; 66.7% of positive cases). Other respiratory viruses including adenovirus (2.4%), rhinovirus (4.9%), influenza (4.9%), and COVID-19

(2.4%) were identified less commonly. These findings are consistent with the work of Boeschoten et al. (2020), who included viral infections as secondary risk factors in their prospective study. However, they found that viral susceptibility was not a significant risk factor for PICU admission when comparing PICU patients to ward patients ($p>0.05$), suggesting that while viral infections may trigger exacerbations, they do not necessarily distinguish between severity levels requiring intensive versus general ward management. This contrasts with our higher detection rate of viral pathogens in PICU-admitted children (43.9%), potentially reflecting differences in testing protocols, study populations, or the temporal evolution of viral epidemiology between study periods [11].

The present study found that children in the case group had substantially higher rates of prior PICU admissions compared with controls (mean 1.85±1.05 versus 0.46±0.98, $p=0.007$) and emergency room visits (mean 4.33±2.30 versus 1.88±2.23, $p<0.001$) in the preceding 12 months. When categorized, 41.5% of cases versus 19.5% of controls had ≥2 PICU admissions ($p=0.031$), and 43.9% of cases versus 22.0% of controls had ≥3 ER visits ($p=0.034$). These findings indicate that critical acute asthma often occurs in children with a history of frequent severe exacerbations, suggesting a pattern of inadequate disease control. In agreement with our results, Johnson et al. (2022) showed that a school-based asthma program in Columbus, Ohio, led to a 49 percent reduction in emergency visits, a 50 percent decrease in hospitalizations, and a 71 percent decrease in PICU admissions within one year [25].

In the present study, the majority of children with critical acute asthma required continuous short-acting beta-2 agonist nebulization during the first hour of admission (70.7%), with a mean of 2.8±1.02 additional doses administered beyond the initial hour. Systemic corticosteroids were used in 65.9% of cases, and intravenous magnesium sulfate was administered to 51.2% of patients. Among those receiving corticosteroids, hydrocortisone was the most commonly used agent (63.0%), with a mean treatment duration of 1.82±0.79 days and mean dose of 53.73±34.8 mg.

These management patterns align with current evidence-based recommendations and are supported by recent systematic reviews. Maue et al. (2025) conducted a comprehensive systematic review and meta-analysis of 23 studies evaluating protocols and pathways in pediatric critical asthma and found that standardized approaches were significantly associated with reduced hospital length of stay (mean difference -0.77 days, 95% CI -0.81 to -0.73) and PICU length of stay (mean difference -0.31 days, 95% CI -0.37 to -0.26). Additionally, use of clinical pathways decreased the need for respiratory support escalation (OR 0.59, 95% CI 0.39–0.89) [3]. Similarly, Taher et al. (2022) found that patients receiving magnesium sulfate infusions ≥4 hours received significantly longer continuous albuterol therapy and higher cumulative corticosteroid doses [26]. However, Riggins et al. (2025) found that lower starting doses of continuous albuterol (10 mg/h) produced comparable clinical outcomes to standard doses (15 mg/h), suggesting that lower initial dosing may optimize resource utilization without compromising outcomes [27].

The present study found that patients treated with intravenous magnesium sulfate had significantly shorter mechanical ventilation duration compared with those who did not receive this therapy (2.45±1.32 days versus 3.35±1.37 days, $p=0.038$). Similarly, continuous short-acting beta-2 agonist administration was associated with reduced ventilation duration (2.61±1.20 days versus 3.67±1.37 days, $p=0.026$). Systemic steroid use did not significantly influence mechanical ventilation duration in our cohort (2.88±1.28 days versus 3.00±1.47 days, $p=0.797$).

The beneficial effects of magnesium sulfate observed in our study are supported by Ambrożej et al. (2024), who conducted a systematic review with meta-analysis and demonstrated that intravenous magnesium sulfate administration led to approximately 85% reduction in the odds of hospitalization (OR 0.15, 95% CI 0.05–0.42, $p<0.001$) and improved peak expiratory flows compared to placebo or other second-line intravenous treatments [28]. Thani et al. (2024) specifically evaluated prolonged magnesium sulfate infusions in 100 children with asthma exacerbations and found the treatment to be both safe and effective, with 89% of patients recovering with no other bronchodilator infusions required, and 94% did not require respiratory support escalation [29].

The present study found that non-invasive respiratory support was frequently utilized in children with critical acute asthma, with high-flow nasal cannula being the most commonly employed modality (51.2%). Children requiring bilevel positive airway pressure support (22.0%) needed relatively high settings, with mean inspired oxygen fractions of 58.44±16.30% and peak inspiratory pressures averaging 20.67±1.00 $cmH_2O$. Children managed with CPAP (24.4%) received a mean $FiO_2$ of 56.00±12.43% and a CPAP level of 5.20±0.42 $cmH_2O$.

Non-invasive respiratory support has gained increasing attention in the management of children with severe acute asthma, particularly as a strategy to avoid invasive mechanical ventilation. Mangkuliguna et al. evaluated 14,606 pediatric patients and reported that HFNC use was associated with improvement in clinical respiratory scores; however, no significant reduction was observed in PICU admission rates or need for escalation of respiratory support compared to standard oxygen therapy [30]. Rogerson et al. (2025) conducted a matched

cohort study of 443 children treated with early HFNC and 443 controls receiving standard oxygen therapy for severe acute asthma and found that HFNC use led to improvement in heart rate, respiratory rate, and Pediatric Asthma Severity Score within the first 24 hours [31]. This highlights the importance of individualized respiratory support strategies based on patient severity and response to therapy.

The present study found that the vast majority of children with critical acute asthma demonstrated clinical improvement following intensive management (92.7%), with 95.1% being discharged from the PICU. Mortality was 4.9%. These favorable outcomes reflect the effectiveness of contemporary critical care management for pediatric asthma, including aggressive bronchodilator therapy, systemic corticosteroids, magnesium sulfate, and respiratory support.

In agreement with our findings, Arcoleo et al. (2025) reported a 37% increase in well-controlled asthma based on ACT scores and a 56% improvement according to healthcare provider assessment, with significant reductions in healthcare utilization [13]. In contrast, Mukherjee et al. (2022) reported 15 deaths among 2,195 PICU admissions for asthma in England (mortality rate 0.68%), with deaths being most frequent in the 10–14 years age group and no deaths occurring in children less than 5 years of age. This age-related mortality pattern differs from our findings and suggests that adolescents may face unique risks during critical asthma episodes that warrant further investigation [32]. Maue et al. (2025) similarly reported that standardized protocols and pathways in pediatric critical asthma have been associated with improvements in outcomes [3], and Eusebe et al. (2025) found that most children had favorable outcomes, with no deaths even among those needing mechanical ventilation [21].

The present study found that asthma control questionnaire scores were significantly higher in cases compared with controls (mean 3.19±2.01 versus 1.01±1.67, $p<0.001$), indicating substantially poorer disease control among children who experienced critical exacerbation. None of the cases achieved well-controlled status compared with 36.6% of controls, while 39.0% of cases were classified as extremely poorly controlled compared with none of the controls ($p<0.001$). This stark contrast underscores the relationship between inadequate baseline asthma control and the risk of critical exacerbation. In agreement with our findings, Liu et al. (2010) evaluated 343 children aged 4–11 years using the Childhood Asthma Control Test and reported that children with C-ACT scores <20 had significantly poorer asthma control, with sensitivity of 68% and specificity of 74% for detecting uncontrolled asthma [33].

In the present study, 48.8% of children with critical acute asthma reported good adherence to prescribed treatment after discharge, while 51.2% remained non-compliant. This persistence of non-adherence even after a life-threatening exacerbation is concerning and highlights the ongoing challenge of medication compliance in pediatric asthma. Arcoleo et al. (2025) specifically addressed this challenge in their school-based intervention study, demonstrating that supervised medication administration through school nurses significantly improved adherence rates. Their findings suggest that traditional approaches relying on patient and family self-management may be insufficient for children with a history of critical disease, and that more intensive support strategies may be required to ensure adequate medication adherence following PICU discharge [13].

## Conclusion

This study demonstrated that critical acute asthma requiring PICU admission is primarily associated with modifiable factors related to poor disease control, including inadequate adherence to controller therapy, incorrect inhaler technique, frequent previous emergency visits and PICU admissions, and suboptimal asthma control scores. In addition, family history of asthma/allergy, environmental exposures such as passive smoking, air pollution and animal contact, together with atopic comorbidities including obesity, eczema and allergic rhinitis, were significantly more prevalent among critically ill patients. Despite the substantial healthcare resources required, including intensive pharmacological treatment and non-invasive respiratory support, the majority of children showed favorable clinical outcomes with a high rate of recovery and discharge. These findings emphasize that improving long-term asthma control, optimizing adherence and inhalation technique, and reducing exposure to environmental triggers may substantially decrease the risk of life-threatening exacerbations and PICU admission.

## Limitations

This single-center case-control study included a relatively small sample, which may limit the generalizability of the findings. The study design identifies associations rather than causality, and the use of convenience sampling may introduce selection bias. In addition, adherence, environmental exposures, and asthma control relied partly

on caregiver reporting, which may be subject to recall bias. Finally, the lack of long-term follow-up prevented assessment of future exacerbations and sustained asthma outcomes after discharge

## Recommendations

Based on the study findings, regular assessment of asthma control, medication adherence, and inhaler technique should be incorporated into routine pediatric asthma care, particularly for children with a history of frequent exacerbations or healthcare utilization. Early identification and management of associated allergic conditions and obesity, alongside interventions to minimize exposure to passive smoking, pollution, and other environmental triggers, are strongly recommended. Structured asthma education programs for patients and caregivers, coupled with close follow-up after hospital discharge, may improve long-term compliance and reduce recurrence of severe attacks. Furthermore, implementation of standardized PICU management protocols and multicenter prospective studies with larger populations are recommended to further define risk factors and optimize strategies for preventing critical acute asthma in children.

**Conflict of interest**
All authors have no conflicts of interest that are directly relevant to the content of this review.
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