Emergency department treatment of asthma in children: A review

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Abstract
Asthma is the most common chronic illness in children, with >700,000 emergency department (ED) visits each year. Asthma is a respiratory disease characterized by a combination of airway inflammation, bronchoconstriction, bronchial hyperresponsiveness, and variable outflow obstruction, with clinical presentations ranging from mild to life-threatening. Standardized ED treatment can improve patient outcomes, including fewer hospital admissions. Informed by the most recent guidelines, this review focuses on the optimal approach to diagnosis and treatment of children with acute asthma exacerbations who present to the ED.

KEYWORDS
asthma, child, emergency service hospital, emergency treatment, respiratory disorders

INTRODUCTION

Asthma is the most common chronic illness in children, affecting >6 million children in the United States.1 In 2014 to 2015, of all emergency department (ED) visits in the United States, 9.5% had an asthma diagnosis documented in the medical record; the largest proportion of these patients were children aged 5 to 17 years (13%).2 Each year, asthma accounts for >700,000 ED visits by children and 2% to 5% of all pediatric hospitalizations.3,4

In this review, we provide an up-to-date overview of the ED diagnosis and treatment of children with acute asthma exacerbations. Optimal treatment in the ED can improve patient outcomes, including both reducing ED length of stay and hospital admission rates.5-8 The review focuses on ED treatment and reflects the literature and guideline recommendations through 2019. We begin with a brief discussion of the clinical presentation of asthma in children, the differential diagnoses, and the scoring tools available to aid in the assessment of severity. We then review the ED treatment of asthma, with a focus on the most well-supported therapies. We conclude with general guidelines to determine patient disposition.

CLINICAL PRESENTATION

Definition

Asthma is a chronic lower respiratory tract disease characterized by a combination of airway inflammation, bronchoconstriction, bronchial hyperresponsiveness, and variable outflow obstruction. In patients with asthma, inflammatory cell activation leads to airway edema and hypersecretion of mucus, and, if sufficiently chronic and severe, may progress to airway remodeling and persistent narrowing.9 An acute, severe asthma exacerbation unresponsive to repeated administration of inhaled β-agonists may be life-threatening if untreated.
2.2 | Diagnosis

Young children can be particularly difficult to diagnose with asthma because wheezing can be caused by a variety of etiologies, most commonly respiratory viral infections. Rather than formally diagnosing children with asthma, ED clinicians are tasked with determining when to initiate treatment for a possible or likely asthma exacerbation. ED clinicians must use a combination of history and physical exam findings to determine when to start treatment while looking for alternative diagnoses if patients are not improving.

2.2.1 | History

A hallmark of asthma is recurrent exacerbations. Characteristic features of exacerbations include wheezing, cough, shortness of breath, and chest tightness. Caretakers may report a worsening of symptoms at night and fatigue or poor sleep, especially in school-age children. Exacerbations are often triggered by respiratory infections, creating considerable overlap between symptoms of an isolated viral lower respiratory tract infections and asthma. Other common triggers include inhaled irritants such as tobacco smoke, exposure to environmental allergens, or changes in weather.

2.2.2 | Physical exam

Wheezing is the most common physical finding associated with asthma and is included on almost all published scoring tools for the assessment of asthma in children. Wheezing should not be used in isolation. Wheezing is notably absent in many severe exacerbations as a result of a lack of airflow. Wheezing may also be caused or mimicked by other disease processes. Wheezing is a high-pitched, continuous noise that is most often expiratory. Wheezing should not be confused with stridor, which is also high pitched but primarily inspiratory, or stertor, which is more variable in pitch and respiratory phase and is primarily associated with nasal congestion and discharge. Wheezing has good reliability in controlled studies of children with asthma and pneumonia. In clinical practice, however, the assessment of wheezing is likely to be less valid and reliable when using loudness and focality to determine severity.

Physical examination should focus on a child’s mental status and respiratory effort. Particular attention should be paid to a child’s mental status—a fussy, crying child is more reassuring than a quiet, listless child, which should alert the clinician to the possibility of more severe disease. On examination, children may have increased work of breathing which manifests as nasal flaring, facial pallor, grunting, head bobbing, and retractions. The expiratory phase may be notably prolonged and is usually associated with intercostal retractions and wheezing. In contrast, prolonged inspiration and sternal retractions are physical examination findings more consistent with an upper airway obstruction and should not be confused with a severe asthma exacerbation.

2.2.3 | Differential diagnosis

The differential diagnosis of acute asthma exacerbation is detailed in Table 1. Asthma may be challenging to diagnose in children and assessing the severity of an acute exacerbation may be particularly difficult. Any child with a reported asthma history unresponsive to asthma therapy requires reconsideration of the diagnosis.

3 | SCORING TOOLS AND DISEASE SEVERITY

Despite being the gold standard to diagnose asthma, pulmonary function tests and incentive spirometry lack reproducibility in young children and are unreliable during acute illness. Pulmonary function tests are also impractical in the ED and should not be used during an acute exacerbation. Clinical assessments of children with an asthma exacerbation may also be variable.

To standardize clinical diagnosis and deliver timely ED care, several pediatric asthma scoring tools have been developed (Table 2). These scoring tools are intended to guide treatment decisions during acute asthma exacerbations by determining the level of ED treatment, repeating scores to determine changes in treatment, and patient disposition. Each scoring tool has several examination findings scored on a 3-point, 4-point, or 7-point scale; higher numbers on each scale represent more severe disease. Examples of common examination findings are the presence of wheezing, respiratory rate, work of breathing, oxygen saturation, expiratory phase, and dyspnea.

Most scoring tools have demonstrated good interrater reliability, but studies comparing tools are limited. Asthma severity scoring tools have been associated with more timely administration of medications, reductions in ED length of stay, and reductions in admissions without a corresponding increase in return ED visits. Some tools can also predict the need for admission and level of inpatient care. Several institutions have developed asthma treatment pathways based on scoring tools to improve care through standardization.

4 | TREATMENT

For patients with mild asthma, first-line treatment is albuterol hydrofluoroalkane-pressurized (HFA) and an oral steroid, for example, dexamethasone, and may also include inhaled ipratropium. Patients with moderate to severe asthma exacerbations will typically receive inhaled albuterol and ipratropium, an oral or systemic steroid, and if symptoms are not improving intravenous magnesium. Patients with severe or life-threatening exacerbations will receive all of these treatments and one or more adjunctive treatments, which may include systemic epinephrine. Patients with severe asthma exacerbation that does not respond to treatment may also require non-invasive positive pressure ventilation (NIPPV) and monitoring for deteriorating mental status and ventilatory failure. Medication dosages and administration routes are detailed in Table 3.
### TABLE 1  Differential diagnoses for acute asthma exacerbation in a child

| Diagnosis                                      | Chronicity | Clinical features                                      |
|-----------------------------------------------|------------|--------------------------------------------------------|
| Anaphylaxis                                   | Acute      | Urticaria, facial/oral edema, emesis, abdominal pain   |
| Bronchiolitis                                  | Acute      | Fever, nasal congestion, rhinorrhea, coarse rales on auscultation |
| Foreign body (lung or esophagus)               | Acute      | History of choking, unilateral wheezing                |
| Pneumonia                                     | Acute      | Fever, focal wheezing, cough, fatigue                  |
| Aspiration syndromes                          | Chronic    | Coughing or choking with feeding, recurrent pneumonia, cough |
| Anatomic abnormalities (eg, malacias, external compression) | Chronic | Recurrent pneumonia, fixed wheezing                   |
| Bronchopulmonary dysplasia                    | Chronic    | History of prematurity, history of oxygen requirement  |
| Cystic fibrosis                               | Chronic    | Frequent pneumonia, failure to thrive, persistent cough |
| Primary ciliary dyskinesia                    | Chronic    | Frequent pneumonia, recurrent sinusitis and otitis, cough |
| Asthma                                        | Acute and/or chronic | Diminished air flow, prolonged expiratory phase, cough |
| Heart disease                                 | Acute and/or chronic | Poor feeding or sweating with feeds, failure to thrive, cyanosis, tachycardia, hepatomegaly |

### TABLE 2  Severity scoring tools for pediatric asthma

| Asthma score (study)                  | Ages                     | Interrater reliability | Components (point range) | Score interpretation and when to use                                                                 |
|---------------------------------------|--------------------------|------------------------|--------------------------|--------------------------------------------------------------------------------------------------|
| PASS: Pediatric Asthma Severity Score | 1–18 years               | Good to excellent      | Degree of wheezing (0–2)  | Score > 2 predicts length of stay > 6 hours or hospital admission                                |
| (Gorelick et al)                      |                          |                        | Work of breathing (0–2)   | Use during initial assessment                                                                    |
|                                       |                          |                        | Prolonged expiration (0–2) |                                                                                                 |
| PRAM: Pediatric Respiratory Assessment | Original 3–6 years;      | Good                   | Pulse oximetry value (0–2)| Mild: 0–1                                                                                         |
| Measure (Chalut et al)                 | subsequent validation    |                        | Intensity of air entry (0–3)| Moderate: 4–7                                                                                     |
|                                       | from 18 months to 18     |                        | Degree of wheezing (0–3)  | Severe: 8–12                                                                                     |
|                                       | years                    |                        | Suprasternal retractions (0–2)| Use during initial assessment and to assess response to treatment                                |
|                                       |                          |                        | Scalene retractions (0–2) |                                                                                                 |
| AAIRS: Acute Asthma Intensity Research  | 5–18 years               | Good                   | Retractions (0–6)         | Mild: 1–6                                                                                        |
| Score (Arnold et al)                   |                          |                        | Characteristics of air entry (0–3)| Moderate: 7–11                                                                                  |
|                                       |                          |                        | Degree of wheezing (0–3)  | Severe: 12–16                                                                                   |
|                                       |                          |                        | Pulse oximetry value (0–3)| Use during initial assessment to determine pediatric ICU admission and to assess response to treatment |
|                                       |                          |                        | Prolonged expiration (0–3) |                                                                                                 |
| PAS: Pediatric Asthma Score (Kelly et  | 2–18 years               | Not reported           | Respiratory rate (1–3)    | Mild: 5–7                                                                                        |
| al)                                    |                          |                        | Oxygen requirements (1–3)  | Moderate: 8–11                                                                                   |
|                                       |                          |                        | Auscultation (1–3)        | Severe: 12–15                                                                                   |
|                                       |                          |                        | Retractions (1–3)         | Use during initial assessment and to guide management                                            |
|                                       |                          |                        | Dyspnea (1–3)             |                                                                                                 |

*Score developed for the emergency department.

### 4.1  Albuterol

Albuterol is one of the two mainstays of acute asthma treatment. Albuterol is a long-acting β2 receptor agonist, producing bronchodilation through smooth muscle relaxation. Albuterol’s onset of action is <5 minutes, and duration of action is 3 to 6 hours. The most common side effects with albuterol are vomiting, tremor, and tachycardia attributed primarily to peripheral vasodilatation. Albuterol is most commonly administered via HFA metered dose devices. Most young children cannot create a seal around the mouthpiece of the HFA and will need a mask and spacer to improve medication delivery. Albuterol can also be administered via nebulization, but albuterol HFA with a spacer delivers an equivocal dose compared with nebulizers. Albuterol HFA also results in shorter ED length of stay, fewer hospital admissions, and no differences in oxygen saturations. Albuterol HFA is also the preferred mode of administration for patients under airborne precautions, such as for severe acute respiratory syndrome coronavirus 2.

In severe asthma exacerbations, the use of continuous, nebulized albuterol is recommended. A recent study of continuous albuterol in pediatric patients found no optimum weight-based dose for decreasing the length of hospital stay. This study suggests that lower doses of
TABLE 3  Medications for the emergency department management of asthma exacerbations

| Medication name          | Route    | Typical dose                  | Typical maximum dose |
|--------------------------|----------|-------------------------------|----------------------|
| Primary medications      |          |                               |                      |
| Albuterol sulfate        | HFA      | 4–8 puffs                     |                      |
|                          | Nebulized | 2.5–5 mg                     |                      |
|                          | Continuous | 5–20 mg/hour                |                      |
| Ipratropium bromide      | HFA      | 4–8 puffs                     |                      |
|                          | Nebulized | 0.25–0.5 mg                  | 1.5 mg/hour          |
| Dexamethasone            | PO, IV, IM | 0.6 mg/kg                    | 16 mg                |
| Prednisone               | PO       | 2 mg/kg                       | 60 mg                |
| Prednisolone             | PO       | 2 mg/kg                       | 60 mg                |
| Secondary medications    |          |                               |                      |
| Magnesium sulfate        | IV       | 25–75 mg/kg                   | 2 g                  |
| Epinephrine              | IV, IM   | <12 years 10 mcg/kg/dose every 15 minutes for 2 doses >12 years 0.25 mg/dose every 15 minutes for 2 doses | 250 mcg/dose         |
| Terbutaline              | SC       | 2–10 mcg/kg loading dose followed by infusion 0.1–0.4 mcg/kg/min | 3 mcg/kg/min         |
| Ketamine                 | IV       | 2 mg/kg loading dose followed by 20–60 mcg/kg/min |                      |

HFA, albuterol hydrofluoroalkane-pressurized; IM, intramuscular; IV, intravenous; PO, orally; SC, subcutaneous.

Continuous albuterol may be just as effective and potentially decrease cost and unwanted side effects.38

4.2  Ipratropium

Ipratropium, a muscarinic receptor antagonist, also produces bronchodilation as well as decreasing mucous production. Ipratropium’s onset of action is 15 to 30 minutes and the duration of action is 3 to 5 hours.39 Common side effects of ipratropium are dry mouth, headache, and nasal congestion.

Ipratropium is typically added to albuterol in moderate or severe exacerbations. The addition of ipratropium to albuterol has been associated with reduced hospital admission in pediatric patients with moderate and severe asthma exacerbations.40 Early initiation of ipratropium in the ED has been shown to prevent hospital admission, but little benefit has been reported in continued use in the critical care or floor setting after admission.41

4.3  Systemic steroids

Systemic steroids are the other mainstay of acute asthma therapy. Multiple studies have shown early steroid administration in the ED reduces hospitalization.6,42 Recent literature supports dexamethasone as the preferred systemic steroid. Dexamethasone is a long-acting steroid that can be given orally or intramuscularly. Dexamethasone’s onset of action when given orally is 1 to 2 hours and 30 to 120 minutes after intramuscularly administration. The half-life is 2 to 9 hours, and commonly reported side effects include nausea, vomiting, restlessness, and fatigue.43 A 2016 study reported that 1 dose of dexamethasone was not inferior compared with 3 days of prednisone in terms of hospital admission rate or unscheduled return visits to health-care providers.44 In a study comparing a 2-dose regimen of oral dexamethasone to a traditional 5-day course of prednisone, similar efficacy with increased compliance was reported.40,45 In a separate study, children with acute asthma treated with 1 dose of dexamethasone had a 36% relative risk reduction in 72-hour ED return rate compared with children treated with a 3-5-day course of oral prednisone.46 Dexamethasone is also more cost-effective and tastes better, and patients are more likely to complete treatment when compared with prednisolone.47

The literature comparing single-dose dexamethasone to multiple doses of dexamethasone is limited. Most studies suggest single-dose versus a daily dose for 2 days.49 More research is needed to determine if providing a second oral dose will aid in pediatric exacerbation relapses as well as more investigation of the effect of this regimen in the hospitalized pediatric patient.47

Prospective randomized trials show no clinical difference between oral and intramuscular steroid administration.50–52 Intramuscular administration may be used in patients who are unable to tolerate oral steroids. Parents reported a preference for a single dose of intramuscular dexamethasone compared with 5 days of oral prednisone.51
4.4 | Magnesium

Magnesium is a second-line therapy in children presenting with moderate to severe asthma exacerbation. Magnesium is administered intravenously, competing for calcium channels with resultant smooth muscle relaxation and bronchodilation. Magnesium also decreases histamine release from mast cells and produces an anti-inflammatory effect by decreasing super-oxide production by neutrophils. Magnesium’s onset of action is immediate and given over 15 to 60 minutes. Magnesium is well tolerated and has few side effects. Hypotension, dry mouth, nausea, flushing, hyporeflexia, and respiratory depression are reported but rare.

Magnesium should be considered in patients insufficiently responsive to first-line treatment or for those with more severe presentations on arrival. In a 2016 Cochrane review of 5 randomized, placebo-controlled studies, children treated with intravenous magnesium were 68% less likely to be admitted to the hospital. There were insufficient data to evaluate the effects of magnesium administration on secondary outcomes, including ED treatment durations, ICU admission rates, hospital length of stay, and adverse events. There is no conclusive evidence on the impact of inhaled versus intravenous magnesium.

4.5 | Epinephrine and terbutaline

Epinephrine and terbutaline are adjunctive therapies for patients with severe exacerbations who fail to improve after first-line treatment and magnesium. These patients will also typically be candidates for admission to the pediatric ICU, and concern for respiratory failure may be high.

Epinephrine is both an α-agonist and β-agonist and induces smooth muscle relaxation as a result of systemic β-agonist effects and can be administered intramuscularly, subcutaneously, intravenously, or through nebulization. Epinephrine is typically administered intramuscularly in the ED because of more rapid availability and ease of administration, although the literature is mixed on improved efficacy of intravenous β-agonists. Onset of action is 5 to 10 minutes if subcutaneous and immediate when given intravenously. The duration of action is <5 minutes intravenously. Most common side effects are anxiety, tremors, agitation, and sweating.

Terbutaline is pure β-agonist and is an option for hypertensive patients who may not tolerate epinephrine. Subcutaneous dosing is only for children older than 12 years of age. Side effects of terbutaline include tachycardia, arrhythmias, hypokalemia, and rarely myocardial ischemia.

4.6 | Ketamine

Ketamine is an N-methyl-D-aspartate receptor antagonist that has dissociative, amnestic, and analgesic properties. Ketamine has been shown to promote bronchodilation and prevent bronchospasm. Children with severe asthma in the ED who received a 2-hour ketamine infusion did not have improvement in respiratory rate, oxygen saturation, hospital admission rate, or need for mechanical ventilation when compared with normal saline. However, ketamine has been shown to increase pulmonary compliance in mechanically ventilated patients.

4.7 | Heliox

Helium-oxygen mixture (heliox) is a potential adjunct for severe asthma exacerbations refractory to first-line and other adjuncts, in particular in patients at risk for respiratory failure. Heliox’s potential benefit is indirect—it enhances the delivery of other inhaled treatments through its lower gas density by decreasing flow resistance, lowering viscosity, and increasing laminar flow. Heliox is available in concentrations of 80% helium/20% oxygen and 70% helium/30% oxygen. If the patient requires >40% oxygen, the potential beneficial effect is diminished. Ideally heliox should be started early in the patient’s presentation. Heliox has limited use past the first 24 hours of disease and in patients with hypoxia.

The literature on heliox in children is limited to case reports or case studies and has shown conflicting results. One study demonstrated no significant improvement in the recovery of pulmonary function in children treated with heliox. However, a 2014 meta-analysis examining 113 children found that the use of heliox in delivering continuous albuterol therapy was associated with a significant improvement in peak expiratory flow.

4.8 | High-flow nasal cannula

High-flow nasal cannula (HFNC) provides heated and humidified gas at a rate greater than typical inspiratory flow and is well tolerated by most patients. In addition, HFNC reduces anatomic dead space and potentially provides some degree of positive end-expiratory pressure. The degree of positive end-expiratory pressure provided by HFNC is not as consistent as that provided by NIPPV.

HFNC is primarily used to treat respiratory distress/failure with bronchiolitis, and the literature on HFNC in asthma is limited. One retrospective, single-center study of HFNC in 73 children with status asthmaticus demonstrated HFNC was feasible and likely safe, with only 2 instances of treatment failure—pneumothorax in 1 patient and another requiring escalation of therapy to NIPPV.

4.9 | Non-invasive positive pressure ventilation (NIPPV)

Asthma, as a result of bronchiolar narrowing/closure, results in air-trapping within the alveoli, leading to dynamic hyperinflation. The patient must generate a higher negative inspiratory force to overcome the gradient to initiate inspiratory flow. If the patient cannot generate sufficient negative force to overcome the gradient, then
hypercarbia may ensue, leading to altered mental status, severe acidosis, and respiratory depression. This sequence of events is the most feared result of severe asthma and puts the patient at risk of cardiac arrest.

NIPPV refers to either bilevel positive airway pressure (BiPAP) or continuous positive airway pressure devices. Continuous positive airway pressure delivers a single degree of pressure support throughout the respiratory cycle, whereas BiPAP provides pressure support during both inspiration (inhalation positive airway pressure [IPAP]) and expiration (exhalation positive airway pressure). IPAP reduces the work by respiratory musculature. Exhalation positive airway pressure allows for pressure equilibration between the mouth and alveoli, reducing the amount of force needed to generate inspiratory flow.

NIPPV is primarily indicated with severe asthma either insufficiently responsive to initial treatment or with extreme respiratory distress/ventilatory failure on arrival. The goal of NIPPV is to provide the patient with pressure support, ease the work of breathing/patient distress, and forestall or treat ventilatory failure. In a child, BiPAP and continuous positive airway pressure can be provided through a nasal mask or a full-face mask, although use may be limited by patient size and equipment availability. Typical settings for BiPAP are an IPAP of 10 cm H₂O and an exhalation positive airway pressure of 5 cm H₂O, with or without a backup ventilation rate. Because these supports require NPO status, intravenous administration of glucose-containing fluids should be initiated in infants and considered in all patients. Children may require anxiolysis and other supportive measures to tolerate NIPPV. Allowing the child to sit on a caregiver’s lap and involvement of a child-life specialist can ease the transition to respiratory support.

A 2016 Cochrane review evaluated 2 randomized controlled trials that investigated the efficacy of BiPAP in children presenting with asthma exacerbations. Both studies report that BiPAP improves respiratory rate, accessory muscle use, and dyspnea as compared with standard therapy, but the effect on overall outcomes are unclear.

Despite NIPPV use, 7% of patients will progress to require mechanical ventilation.

### 4.10 Endotracheal intubation

Endotracheal intubation and mechanical ventilation may be indicated rarely for severe asthma, but the decision should not be taken lightly. Avoiding intubation is the primary goal of the other treatments for severe asthma. Patients who are candidates for intubation are already at risk for complications, including respiratory and cardiac arrest, and intubation only increases that risk. The apnea associated with medication-assisted approaches to intubation inevitably worsens hypercarbia and respiratory acidosis. The reported rate of complications of endotracheal intubation is as high as 26%, including hypotension, pneumothorax, subcutaneous emphysema, and cardiac arrest.Reported mortality after intubation is as high as 8%. Worsening hypercarbia after the initiation of treatment is ominous, but hypercarbia in itself does not necessitate intubation. With aggressive therapy, intubation can be avoided.

Criteria used to consider endotracheal intubation include poor response to maximum therapy, hypercarbia (pCO₂ > 50 mmHg), severe hypoxemia (PO₂ < 60 mmHg), worse mental status, impending respiratory arrest indicated by respiratory depression or bradycardia, worse metabolic acidosis, and cardiopulmonary arrest.

Before the start of intubation attempts, asthma therapy should be maximized and a clear plan in place for cardiac arrest, with code doses of epinephrine and the defibrillator prepared. Ketamine is associated with bronchodilating properties and is a good choice for induction. As noted, the decision to use a paralytic medication specifically should be made with extreme caution and likely in consultation with critical care. As patients with severe asthma exacerbations will be intolerant of the resultant apnea; respiratory acidosis will worsen, maybe critically and irreversibly. If a paralytic medication is given, succinylcholine and rocuronium have rapid onset and are effective. The patient should be oxygenated as well as possible before intubation. Cardiac arrest will often be preceded by worsening of hypoxemia and bradycardia, and loss of pulses may be subtle in a patient already in extremis. The care environment should therefore be as quiet as possible, with clear roles and responsibilities and anticipation of deterioration.

Immediately, post intubation, it is paramount to use adequate deep sedation in these patients as it can prevent patient–ventilator asynchrony. Deep sedation should be achieved with propofol or ketamine. Once intubated, general ventilation parameter goals include an oxygen saturation > 91%, permissive hypercarbia, and a pH > 7.2. Permissive hypercarbia is achieved through controlled hyperventilation and lower tidal volumes to prevent hyperinflation and minimize barotrauma while providing adequate ventilation and oxygenation. Despite the inclination to remedy the patient’s hypercarbia, it is vital to remember that permissive hypercarbia is preferable to excessive pressures that can lead to pneumothoraces and breathing collapse, which can lead to circulatory collapse.

Mechanical ventilation can also be very challenging in severe asthma, and coordination of care with a pediatric intensivist is strongly recommended. A pressure support approach is preferred, allowing the patient to set their own rate and tidal volume, although studies have looked at other common modes such as pressure support and volume control. Ventilatory settings should use low tidal volumes (5–10 mL/kg), lower respiratory rates for age, inspiratory-to-expiratory time rates of 1:4 to 1:5, and plateau pressures < 35 cm H₂O. The goal of prolonged expiratory times is to avoid breath stacking.

If there is hemodynamic instability after endotracheal intubation, in particular bradycardia and/or hypotension, there is a step-by-step approach to troubleshoot the issue. First, disconnect the patient from the ventilator to allow his or her chest to naturally recoil; blood pressure should improve immediately. This hypotension is attributed to the decrease of systemic venous return (preload) as a result of worsening hyperinflation brought on by mechanical ventilation. If this maneuver is successful, the tidal volume and respiratory rate are too high and mechanical ventilation can be restarted at lower settings. Confirm endotracheal tube placement and ensure there is no dislodgement and suction the endotracheal tube to guarantee there is no obstruction. Consider use of a bedside ultrasound or chest X-ray to rule out the
Methylxanthines, such as aminophylline and theophylline, were historically used for acute asthma exacerbations. In recent years, the National Asthma Education and Prevention Program Expert Panel recommended against the use of methylxanthines for treatment of hospitalized children with status asthmaticus. However, there has been some literature to suggest that there may be an indication for theophylline in the hospitalized pediatric ICU asthmatic patient. Leukotriene-modifying agents, such as montelukast, are not indicated in the treatment of an acute asthma exacerbation in the ED, although they are often used as adjunct therapies for maintenance in the outpatient setting in addition to an inhaled corticosteroid. The efficacy of inhaled nitric oxide in severe asthma exacerbations is unknown.

5 | DISPOSITION

The ED disposition of a child with an asthma exacerbation is dependent on his or her projected clinical stability and response to treatment. The aforementioned asthma severity scores may aid in prognostication of the need for hospital admission (Table 2). Some EDs offer observation units for prolonged monitoring before discharge. Recent internally validated asthma prediction models found several risk factors were strongly associated with hospital admission: oxygen saturation <94%, respiratory rate >31/min, history of pneumonia, and past ED visit for asthma in the prior 12 months. Hospitalization or observation should be strongly considered for any child with (1) persistent hypoxemia despite treatment and observation, (2) mental status/ inability to drink well worsening, (3) respiratory distress requiring bronchodilators every 2 hours or more, or (4) social or environmental factors that could limit a caregiver’s ability to provide treatment. Children requiring ED escalation of care beyond nebulized short-acting β-agonist treatments every 2 hours may require ICU admission.

Children with improving asthma symptoms may be discharged safely from the ED if they have (1) demonstrated manageable respiratory distress with short-acting β-agonist bronchodilators every 3 to 4 hours or more during their ED course, (2) do not pose imminent risk of clinical decompensation, and (3) have feasible asthma action plans in place with reliable caregivers.

Discharge medications for outpatient prescription include appropriately dosed inhaled bronchodilators with spacer as well as counseling to continue the child’s pertinent daily home medications including allergy medications and controller therapy with inhaled steroids. Children with little controller therapy are at higher risk for admission.

In 2018, Parikh et al examined the value of the following 4 general discharge practices: (1) asthma education; (2) medications provided at discharge, including spacer, β-agonist, controller medication, and oral steroids for current or future exacerbation; (3) primary doctor contact by phone and with scheduled follow-up to manage longer term asthma management plans; (4) other post-discharge components including follow-up phone call to caregivers, home visit referrals, and environmental mitigation program referrals. When evaluated individually, providing comprehensive education resulted in a 6% to 7% reduction in readmission rates at 3 months. Bundling of measures further reduced readmission rates. Similar studies report mixed results, suggesting that a follow-up phone call may reduce ED revisits after hospitalization.

In studies of caregivers of children with asthma, 90% believed that instruction regarding follow-up appointments, medications, reasons to seek medical care, and education in general were important aspects of the discharge process, preferably with live demonstration. Factors associated with an increased risk of return ED visits within the year include younger age, lower socioeconomic and educational status, and chronic obstructive pulmonary disease. In addition, previous critical care admission for asthma increased a child’s risk for readmission for an asthma exacerbation.

Consideration must be given to social determinants of health, as these may play a critical role in both effective treatment of individual patients and in eliminating disparities in care. Interventions supporting asthma education and counseling should address a spectrum of contextual and psychosocial factors that may not be included in historical or more traditional materials. Examples include inner-city dwelling, exposure to air pollution, presence of pests, stress in the home, low income and other limitations affecting access to care should be considered prior to discharge. Physician counseling, coordination with primary care from the ED, and social work consult from the ED visit may support a transition to outpatient care. Perhaps most important, effectively addressing the social determinants of health will almost certainly require multidisciplinary, institution-level commitments to initiatives that partner with community members and both government and non-governmental groups.

6 | CONCLUSION

Asthma exacerbation in children is a common presentation in the ED often requiring escalation of care and hospital admission. Clinical scoring tools are important ED adjuncts to guide treatment, prognosticate disposition, and inform admission. Albuterol, ipratropium, and dexamethasone are the mainstays of acute treatment of severe acute asthma exacerbations in children. All 3 agents have been shown to prevent hospitalization and improve other short-term outcomes. More severe asthma exacerbations should be treated with magnesium. Endotracheal intubation should be approached with extreme caution given the high mortality rate and complications.

CONFLICT OF INTERESTS

The authors have no conflicts of interest to disclose.

AUTHOR CONTRIBUTIONS

Moon O. Lee, Cherrelle Smith, Nicholas Pokrajac, Shyam Sivasankar, and Angela Lumba-Brown have made substantial contributions to...
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