Review Article

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Management of Acute Severe Asthma (Status Asthmaticus)

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Abstract

Asthma is a chronic disease of the respiratory tract characterized by recurrent episodes of airway inflammation and obstruction, showing evidence of reversibility of obstruction. Despite advances in the understanding of asthma and development of effective medical interventions for treatment of asthma (and for prevention of morbidity), children continue to present to the emergency departments of health care institutions with acute asthma exacerbations. An accurate assessment of the severity of the acute attack is essential to ensure optimal treatment. Asthma exacerbations are defined as 'acute or sub-acute episodes of progressively worsening shortness of breath, cough, wheezing, and chest tightness – or some combination of these symptoms.' The pathophysiology of an asthma exacerbation includes airway edema, mucus secretion and smooth-muscle spasm (bronchospasm). The foundations of the treatment of acute asthma are bronchodilators, corticosteroids and oxygen. Before the initiation of therapy, and as therapy is initiated, a brief, focused history and physical examination should be obtained. Escalation of the therapy (aminophylline, magnesium sulfate, rarely mechanical ventilation, etc) may be necessary in severe exacerbations. This review details the assessment and treatment of acute episode of asthma in children.

Keywords: Aminophylline; Asthma; Beta-2- agonists; Children; Hyperinflation; Pulmonary; Ronchi; Salbutamol; Steroids; Terbutaline.

Introduction

Asthma is the most common chronic disease of childhood and the leading cause of childhood morbidity from chronic disease (as measured by school absence, emergency department visits, and hospitalisations).[1] Despite the advances made in the management of asthma and the improved use of inhaled medications, asthma exacerbations still continue to pose a management dilemma for the pediatricians. Martinez points out that asthma exacerbations are a major component of asthma morbidity in both preschool and school-aged children.[2] Asthma is defined as a disease with variable airway inflammation and airflow obstruction. Asthma exacerbations are defined as 'acute or subacute episodes of progressively worsening shortness of breath, cough, wheezing, and chest tightness'– or some combination of these symptoms.[3,4] The pathophysiology of an asthma exacerbation includes airway edema, mucus secretion and smooth-muscle spasm (bronchospasm).

Status asthmaticus/acute severe asthma is an acute exacerbation of asthma that remains unresponsive to conventional therapy and may progress to respiratory failure.[5] It is essential to assess the severity of an acute attack of asthma so that appropriate management can be instituted. The signs that should be assessed include- pulse rate, respiratory rate, breathlessness (ability to talk and feed), use of accessory muscles of respiration, extent of wheezing, level of consciousness and presence of agitation (suggesting hypoxemia).[6]

Complications of acute asthma include atelectasis, air leaks and in severe cases -

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respiratory failure.[7] Frequency and severity of exacerbations help define the severity of asthma. The patterns of exacerbations of asthma vary among patients, but they tend to be similar in the same patient.[7] Exacerbations may be of acute onset or may occur over a period of few days. Abrupt-onset exacerbations occur mostly due to extreme airway hyper-reactivity and susceptibility to airways closure.[7,8,9] Extreme cases may have severe bronchospasm and asphyxia.[8,9] These patients present with high arterial pCO2 levels and may require brief periods of mechanical ventilation. Some asthma exacerbations present over a few days and are due to obstruction resulting from progressive inflammation, epithelial sloughing and cast impaction of small airways.[7,10] Such patients, in extreme cases, present with respiratory failure due to fatigue, necessitating mechanical ventilation for many days.[7,10]

Quick/Rapid Assessment of the Patient (on Admission/Presentation to Emergency Room)

The clinical findings, supplemented by objective tests, should be used to assess the severity of an acute asthma exacerbation. Clinical evaluation should be repeated frequently during the management of an acute asthma exacerbation to assess response to therapy.[11] A quick assessment on admission involves a focused history and clinical assessment.

The *focused history* should include details about the onset of current exacerbation, frequency and severity of day-time and night-time symptoms and activity limitation, frequency of rescue bronchodilator use, current medications (obtain a detailed list of medications being taken at home and the timing and dosage of the medications), known potential triggers and history of atopy /other allergies.[11,13] The *risk factors* for developing severe or persistent status asthmaticus include - history of increased use of home bronchodilator treatment without improvement, history of previous intensive care unit (ICU) admissions (with or without endotracheal intubation and mechanical ventilation), asthma exacerbation despite recent or current use of corticosteroids, frequent emergency department (ED) visits and/or hospitalization (implies poor control) in recent past, and history of syncope or seizures during the acute exacerbation.[14]

Clinical Assessment of a patient with severe asthma should include physical examination, pulse oximetry, and lung function study.[4,15] Physical examination involves assessment of the following parameters - vital signs, pulsus paradoxus, breathlessness, air-movement, use of accessory muscles, retractions, anxiety level and alteration in mental status.[4,11,13] Children with status asthmaticus may appear dehydrated as a result of poor fluid intake, vomiting and increased work of breathing.[16] In the early stages of acute asthma exacerbation, tachycardia and tachypnea may be observed. As the episode progresses, the tachycardia and tachypnea may worsen. With worsening hypoxemia, hypercarbia, marked air trapping and hyperinflation, the stroke volume is compromised. Hypotension and bradycardia may be observed at this stage. Most patients have hypoxia and decreased oxygen saturation due to ventilation-perfusion (V/Q) mismatch. Use of accessory muscles has been shown to correlate with severity of airflow obstruction. Retractions (i.e. intercostal, subcostal, or use of abdominal muscles) may be observed. An abnormally prolonged expiratory phase with audible wheezing can be observed. Mental status ranges from wide-awake to lethargic or agitated to comatose. As hypoxemia progresses, the lethargy progresses to agitation caused by air hunger. Both hypoxemia and hypercarbia can lead to seizures and coma and are late signs of respiratory compromise. Pulse oximetry should be used for assessment in all the patients. Pulsed oxygen saturation (SpO2) of 92% or less on presentation (before oxygen or bronchodilator treatment) is associated with higher morbidity and greater risk for hospitalization.[17] Lung function tests - a peak flow meter assesses airflow obstruction and provides an objective assessment of disease severity. However, children younger

Table1: Drugs used in treatment of acute asthma[4,6,7,12,13,16]

Note: A/E = adverse effects; SABA = short acting beta agonist; MDI = meter dose inhaler; IV = intravenous; SC = subcutaneous; IM = intramuscular; NS = normal saline; DTR = deep tendon reflexes; GI = gastrointestinal; GER = gastro-esophageal reflux; PVC = premature ventricular contractions.

No	Name	Dose	Formulations	Side effects and special remarks
1	Salbutamol (Short acting beta-2 agonist) Nebulizer:	0.15 mg/kg/dose (minimum: 2.5 mg) every 20 min for 3 doses as needed, then 0.15-0.3 mg/kg up-to 10 mg every 1-4 hr as needed. upto 0.5 mg/kg/hr by continuous nebulization.	Nebulizer solution (5 mg/mL concentrate; 2.5 mg/2.5 mL respule).	Tachycardia, hypokalemia and metabolic acidosis may be seen on continuous therapy.
	Salbutamol MDI (100 microgm/ puff):	2-8 puffs up to every 20 min for 3 doses as needed, then every 1-4 hr as needed.	MDI	
	Salbutamol - intravenous	5-15 microgm/kg/dose intermittently OR 1 microgm/kg loading dose followed by 0.2 microgm/kg/min infusion		
2	**Levo-Salbutamol:** R-Isomer of salbutamol	0.075 mg/kg (minimum: 1.25 mg) every 20 min for 3 doses, then 0.075-0.15 mg/kg up to 5 mg every 1-4 hr as needed, or 0.25 mg/kg/hr by continuous nebulization	Respules : 0.63 mg/2.5 mL, 1.25 mg/ 2.5 mL.	0.63 mg is equivalent to 1.25 mg of salbutamol for both efficacy & side effects
3	**Ipratropium** (Anticholinergics) Bronchodilator & Mucolytic. Onset of action-20 min; Peak effect 60 min.	250 microgm every 20 min for 3 doses, then every 2-4 hourly as required	Nebulizer solution- 0.25 mg/mL; Respule-0.5 mg/ 2 ml.	Add to SABA therapy for children with moderate and severe exacerbations. A/E: Dryness of mouth, paradoxical increased wheezing.
4	**Corticosteorids**	0.5-1 mg/kg every 6-12 hr for 48 hr, then 1-2 mg/kg/day bid (maximum: 60 mg/day). IV Methyl-Prednisolone : 2 mg/kg foll. by 1 mg/kg 6 hrly. IV Hydrocortisone : 10 mg/kg stat foll. by 5 mg/kg 6 hrly.	Prednisolone: 5-mg tablets; syrup-5 mg/5 mL & 15 mg/5 mL IV-500mg/vial Methylprednisolone: 2, 4, 8, mg tabs IV- 100 mg/ vial	Short-course "burst" steroids: 1-2 mg/kg/day; 3-7 days. I.V. steroids used if sustained respiratory distress or unable to tolerate orals.
5	**Terbutaline** (beta 2- agonist)	Subcutaneous: 0.01 ml/kg of 1:1000 solution. max dose 0.3-0.5 ml. Continuous IV infusion: 2-10 microgm/kg loading dose, followed by infusion at 0.1 to 0.4 microgm/kg/min, increased by 0.1-0.2 microgm/kg/min every 15-30 min up-to 10 microgm/kg/min.	IV- 1mg/ml	Used in extreme circumstances (e.g., impending respiratory failure, non-cooperative children- anxiety/ altered sensorium, apnea). A/E: tremor, tachycardia, palpitations, arrhythmia, hypertension, headaches, nervousness, nausea, vomiting, hypoxemia.

No	Name	Dose	Formulations	Side effects and special remarks
6	**Epinephrine**	SC or IM: 0.01 mg/kg of 1:1000 (max dose 0.5 mg); may be repeated after 15-30 min.	1 ampoule = 1 ml of 1:1000 soln.	Used in severe bronchospasm. A/E- Nausea, tachycardia, tremors, palpitations, agitation, rarely hypertension & ventricular arrythmias.
7	**Magnesium Sulfate** Bronchodilator (Ca-antagonist). Rapid onset of action.	IV infusion in NS over 30 min: 50-75 mg/kg (max 2gm) every 4-6 hours. OR 50-75 mg/kg loading dose followed by 10-20 mg/kg/hr continuous infusion.	25% Inj. (250 mg/ml) & 50% Inj. (500mg/ml)	Side Effects: Nausea, vomiting, facial flushing, dry mouth, hypotension, tachycardia. Toxicity: Loss of DTR, muscle weakness, respiratory depression, cardiac conduction defects.
8	**Aminophylline:** Bronchodilation (without affecting V-Q matching), diuretic effect (reduce alveolar fluid & microvascular permeability)	IV Dose: 5 mg/kg loading over 20-30 min followed by 1 mg/kg/hr continuous infusion.	Inj. 250 mg/10 ml	Useful in situations of impending respiratory failure. Maintain serum levels between 12-20 microgm/ml. S/E: 15-25 microgm/ml - GI upset, GER, diarrhea, nausea, vomiting, abdominal pain, headache, insomnia, nervousness, agitation, dizziness, muscle cramps, tremors. 25-35 microgm/ml - tachycardia, PVC. > 35 microgm/ml - ventricular tachycardia, PVC, seizures.

than six years may not be able to cooperate with peak expiratory flow rate (PEFR) assessments and severely ill children may not be able to stand and provide three consecutive recordings by forced exhalation (as is generally recommended for performing the test).[18] Also, PEFR mainly measures larger airway function and does not accurately reflect smaller airway bronchospasm.[4] In addition, the reading is most helpful when the child's personal best PEFR measurement is known. Hence the information obtained from PEFR has its own limitations.

Various *classifications* have been described for the initial assessment of the severity of asthma. The National Asthma Council (Australia)[19] classifies acute asthma into mild, moderate and severe by using the following parameters – sensorium, oxygen saturation, able to speak or not, pulse rate, cyanosis, wheeze intensity and PEF, FEV1. The Pulmonary Index Score (PIS) is an asthma score based on five clinical variables: respiratory rate, degree of wheezing, inspiratory to expiratory ratio, accessory muscle use and oxygen saturation. Each variable is assigned a score from 0 to 3. Total scores range from 0 to 15. A score of 7 to 11 indicates an exacerbation of moderate severity and a score of more than/equal to 12 indicates a severe attack.[20] Other clinical scores include the Clinical Assessment Score[21] and

the Pediatric Respiratory Assessment Measure (PRAM)[22] - both used to assess the severity of an acute asthma exacerbation and are sensitive to changes in clinical status of the patient.

Drugs Used in Management of Acute Asthma

The goals of therapy for acute severe asthma include - rapid reversal of airflow obstruction, correction of hypoxemia and/or severe hypercapnia and reduction of likelihood of recurrence by intensifying baseline therapy.[11] The initial therapy in management of acute asthma involves use of - Oxygen, Inhaled SABA (short acting beta-2 agonists) and Systemic Steroids.[16] If there is an incomplete response, then the therapy can be further intensified using continuous Beta-2 agonists and Ipratropium, subcutaneous (SC) /intravenous (IV) Terbutaline, IV Magnesium Sulfate, IV Aminophylline and SC Epinephrine.[11,23] Some patients may require management in a Pediatric Intensive Care unit (PICU) and assisted mechanical ventilation. Other therapies tried include use of ketamine, Helium - Oxygen mixture and volatile anesthetics like Isoflurane.[24] The details of various drugs used in treatment of acute asthma are summarized in Table 1.

Home Management of Acute Asthma Attack

Home management of acute exacerbation of asthma includes evaluation of early clinical signs and symptoms of airway inflammation. Beginning treatment at home may avoid treatment delays, prevent exacerbations from becoming severe and also adds to patient's sense of control over their asthma.[13] Accurate evaluation of symptom severity by parents and children will assist to avoid delays in care and inappropriate home management.[25] The degree of care provided at home depends on the patient's (or parent's/ caretaker's) abilities and experience and on the availability of emergency care.[26] Patients with asthma should have a written home action plan to guide early recognition and management of acute exacerbation of asthma.[16,27] This is known to reduce the risk of asthma death by 70%.[7] The patient should be started on immediate treatment with "*rescue*" therapy, which includes inhaled short acting beta-2 agonists (SABA)-up to 3 doses can be given in one hour.[28] SABA have a rapid onset of action (<5 min) and duration of action is around 4-6 hrs. SABA act by causing bronchodilation by inducing airway smooth muscle relaxation, reducing vascular permeability and airway edema and by improving mucociliary clearance. The adverse effects of SABA include tremor, irritability, tachycardia, hypokalemia, palpitations, arrhythmias, hypoxemia, and muscle cramps.[28] A good response to SABA implies resolution of symptoms within one hour, no further symptoms over the next 4 hours and improvement in PEFR (80% of personal best).[4,7] An incomplete response is defined as persistent symptoms and/or a PEFR value < 80%.[7] In such situations, oral steroids should be added to the SABA therapy. The physician should be immediately contacted for further instructions. In the Indian context, patient should immediately visit the health facility after 2-4 puffs of SABA. Immediate medical attention is necessary in situations such as severe exacerbations, persistent respiratory distress, lack of expected response or lack of sustained improvement, further deterioration, or presence of high-risk factors.

Emergency Department (ED) Management

The primary goals of management of acute asthma in Emergency Department are - correction of hypoxemia, rapid improvement in airflow obstruction and prevention of further progression or recurrence of symptoms.[7,28] Interventions should be based on - the clinical severity on arrival, response to initial therapy and the presence of risk factors.[7,29]

a. Treatment in the Emergency Department

The initial treatment includes supplemental oxygen, inhaled beta-agonist therapy (SABA)

every 20 minutes for one hour and use of systemic steroids.[4,13] Inhaled ipratropium should be added if there is no significant response or incomplete response to SABA.[30] Subcutaneous injection of epinephrine or beta-agonist can be given in cases of severe exacerbation.[11,31] The clinical status of children who are being treated for acute asthma should be monitored frequently. The frequency of monitoring varies depending upon the severity of illness and response to initial therapy.[11] Monitoring includes-assessment of clinical status, hydration and oxygenation.[4,32] Poor response to first hour of treatment generally implies that exacerbation will not remit quickly.[7]

b. Discharge from ED

Continuous assessment following treatment should be done in the emergency room. Patients can be discharged home if the need for beta 2-agonists is reduced after 4 to 8 hours of conventional treatment, a reading of SpO2 at/above 94% on room air, minimal or no signs of respiratory distress with an improved air entry.[7,28] Discharge medications should include inhaled SABA up to every 3-4 hours and a short course of oral steroid for 3 to 7 days.[11,28,33] Addition of inhaled corticosteroids (ICS) to oral steroids helps optimize controller therapy and reduces risk of further exacerbations.[7,32]

Hospital Management

A *hospital admission* should be considered for patients with moderate to severe exacerbations if the following are present - no adequate improvement within 1-2 hours of initial intensive treatment, cyanosis, an ongoing need for supplemental oxygen, persistently increased work of breathing, child is unable to speak or drink or is breathless, repeated ED visits over several days, oxygen saturation (when breathing room air) is less than 92%, patients with high-risk features for asthma morbidity or death.[11,28,34,35] Other criteria may also be taken into consideration (like distance between home and health care facility, number of previous severe/life-threatening exacerbations and other co-morbid conditions).[35] Indications for *admission to intensive care unit* include-severe attack with poor response to bronchodilators, cyanosis and hypoxaemia ($PaO_2 < 60$ mm Hg) unrelieved by oxygen, $PaCO_2 > 40$ mm Hg, PEFR <30% of predicted or personal best, minimal chest movement or 'silent' chest, severe retractions, deteriorating mental status, confusion, drowsiness, or coma, and concerns for potential respiratory failure/arrest.[4,7,16]

a. Treatment in the Hospital

Treatment should be continued on oxygen therapy and frequent/continuous administration of inhaled bronchodilators and systemic corticosteroids should be given.[16] SABA can be delivered frequently (every 20 minutes to 1 hour) or continuously (at 5-15 mg/hr).[7,36] Continuous inhaled SABA can lead to significant systemic absorption. This obviates the need for intravenous beta-agonist.[7] Patients with acute asthma have hypoxemia as a result of ventilation-perfusion (V/Q) mismatch. Beta-2 agonists may worsen this mismatch by causing pulmonary vasodilation in areas of the lung that are poorly ventilated.[37] Humidified oxygen should be provided as needed to maintain an oxygen saturation of more than/equal to 92 percent.[38] Continuous pulse-oximetry monitoring is advised.[7,39] Inhaled ipratropium has a synergistic effect with SABA in relieving severe bronchospasm and is especially beneficial in patients with mucous hypersecretion or those who are on beta-blocker therapy.[7,30] Additional evaluations include- a complete blood count, arterial blood gas analysis, serum electrolytes and chest radiograph.[4,28] The patient should be simultaneously monitored for respiratory insufficiency, co-morbidities, infections, air-leaks, etc.[7] Infants with increased respiratory rate have increased insensible water loss. This high respiratory rate coupled with decreased oral intake carries a higher risk for dehydration. Hence, the hydration status of the patients should be monitored. Fluids

may be given at or slightly below maintenance.[7,16] Some patients are critically ill and at risk for respiratory failure; these cases are potential candidates for endotracheal intubation and mechanical ventilation.[7]

b. Adjunctive Therapies

These include parenteral (SC/IV) epinephrine or SC terbutaline sulfate or methylxanthines.[4,11] They may be effective in life-threatening airway obstruction not responding to high doses of inhaled SABA.[7,40] A mixture of helium and oxygen theoretically may enhance beta 2-agonist delivery because the lower gas density would result in decreased flow resistance.[24] In patients with acute exacerbation who have been maximized on standard therapy, intravenous magnesium sulfate has been shown to reduce hospitalizations and to improve lung function.[23] However, the use of methylxanthines and magnesium sulfate requires monitoring of serum levels and cardiovascular status.[4,7,41]

c. Mechanical Ventilation

Indications for intubation and mechanical ventilation include - apnea or respiratory arrest, diminishing level of consciousness, impending respiratory failure marked by significantly rising PCO2 with fatigue, decreased air movement and altered level of consciousness and significant hypoxemia that is poorly responsive or unresponsive to supplemental oxygen therapy alone.[7,16] Positive pressure ventilation in a patient with asthma is complicated by severe airway obstruction and air trapping, this results in hyperinflated lungs and high risk of barotrauma.[7,42] Mechanical ventilation should be anticipated and patients at risk for respiratory failure should be managed in the ICU.[7] More than 50% of the complications associated with ventilation in children with asthma occurs at the time of or immediately after intubation.[4,42] Elective tracheal intubation with rapid-induction by sedatives and paralytic agents is safer than emergency intubation.[4,7] If the decision of intubation is made, ketamine is the induction agent of choice because of its bronchodilatory action.[43,4] Asthma is a disease of airway obstruction (i.e. increased airway resistance), resulting in prolongation of the time constant (the time needed for lung units to fill and empty). Hence the strategy for mechanical ventilation in status asthmaticus is controlled hypoventilation i.e. low tidal volume, low rate, long expiratory times, and minimizing the extrinsic PEEP. Patient should be delivered adequate oxygen while tolerating mild to moderate hypercapnia (PaCO2 50-70 mm Hg).[4,44,45]

Outcome in Acute Asthma

The outcome in a particular case is determined by the nature of asthma exacerbations leading to respiratory failure. Patients with rapid/abrupt onset of asthma exacerbations tend to resolve quickly, while those that progress gradually to respiratory failure can require days to weeks of mechanical ventilation.[7] Patients with frequent exacerbations and prolonged treatment are complicated by muscle atrophy and corticosteroid-induced myopathy.[7] Management of severe exacerbations in medical centers is usually successful, even in extreme cases.[7] Asthma deaths in children are rare in medical center, most deaths occur at home or in community settings.[7] Several *risk factors* for mortality in asthma have been identified, mainly biological, environmental, economic and psychosocial.[4,7] *Biological risk factors* include - previous severe exacerbations (ICU admission/mechanical ventilation), sudden asphyxic episodes, two or more hospitalizations in the past year, three or more ED visits in past year, hospitalization/ED visit/s in past one month, increasing and large diurnal variation in peak flows, use of more than two canisters of SABA per month, poor response to systemic corticosteroid therapy, male gender, low birth-weight, and non-white (especially black) ethnicity.[4,7] The *Environmental risk factors* are allergen exposure, environmental tobacco smoke exposure, air

pollution exposure and urban environment.[7] The *Economic and Psychosocial risk factors* include poverty, overcrowding, mother <20 years old, mother with less than high school education, inadequate medical care, psychopathology in the parent/child, poor perception of asthma symptoms or severity and alcohol/substance abuse.[7]

Acute Asthma- Therapies NOT Recommended

Some therapies have little or no benefits in the management of acute asthma and hence they are not recommended. These include inhaled corticosteroids, long acting beta agonists (LABA) and leukotrienes, and anxiolytic/hypnotic drugs.[11] Chest physiotherapy, incentive spirometry and mucolytics should not be used routinely for acute asthma exacerbations as they can trigger bronchospasm or worsen cough or air flow obstruction during an acute asthma attack.[26]

Assessment for Discharge

It is recognized that asthma is a chronic disease. Hence discharge planning is intended to assist the transition from the acute exacerbation to chronic management, identifying factors within the chronic action plan that may need adjusting to prevent future exacerbations and improving long-term patient outcomes.[13] Ongoing assessment will provide the patient information needed to determine the readiness for discharge. *Discharge readiness* usually includes the following - child stable on therapies that can be administered at home, home environment that is able to safely fulfill discharge plan, sufficient knowledge of asthma in the patient/parent to manage care at home or seek help if symptoms worsen, and when arrangements for any special medications or equipment required for home therapies are completed.[13] The patient can be discharged home if the following criteria are met - sustained improvement in symptoms and bronchodilator treatments are at least 3 hours apart, physical findings are normal, PEFR >70% of predicted or personal best and oxygen saturation >92% (in room air).[7] It is recommended that before the patient is discharged from the ED or the in-patient unit, asthma education be provided that is tailored to the identified needs, beliefs, and learning styles of the patient and family and addresses the identified patient-desired outcomes.[46,47] Depending on the severity of the illness at presentation, a complete 3 to 5-day course of oral steroids (prednisolone) should be administered in all the patients having an acute exacerbation.[11,28,33] It is necessary to review the techniques for using inhaled asthma medications as well as for cleaning/maintaining the inhaler device.[48] Every effort should be made to ensure proper follow-up and to implement a long-term plan with the patient's primary care physician or asthma specialist within two to four weeks of discharge from the ED/hospital.[46]

Asthma Education

Asthma education programs includes the following *components*- etiology, prognosis, and risk factors emphasizing chronicity of condition, medication purpose, when and how to use medications and provision or updating of written asthma plan.[13,49] Patient and parent *education* should include instructions on how to use medications and devices (like spacers, nebulizers and metered-dose inhalers [MDIs]).[48] The patient's MDI technique should be assessed at every office visit.[48] It is important to discuss the management plan, which includes instructions about the use of medications, precautions with drug and/or device usage, monitoring symptoms and their severity (peak flow meter reading) and identifying potential adverse effects and the necessary actions.[46,50] The patient should be provided with a written two-part asthma management plan, which includes a plan for daily management and also for acute exacerbations.[7] The plan should discuss, in details, a rescue plan for an acute episode/exacerbation. This plan should include instructions for identifying signs of an acute

attack, using rescue medications, monitoring, and contacting the asthma care team.[13] The parents/caretakers should understand that asthma is a chronic disorder with acute exacerbations and hence continuity of management with active participation by the patient and/or parents and interaction with the asthma care medical personnel is extremely important.[13] The importance of adherence to treatment needs to be emphasized. The factors contributing to asthma severity (such as environmental exposures and co-morbid conditions) need to be investigated and treated as well.[51] The concept of expecting full control of symptoms, (including nocturnal and exercise-induced symptoms) should be incorporated in the management plans and goals (for all but the most severely affected patients). The child should be able to participate in recreational activities/sports and to attend school as usual. Studies have demonstrated a reduction in subsequent ED visits and hospital admissions in those receiving intensive education.[52] Parental attitudes towards and parental knowledge of asthma (pathophysiology, medications, action plans, and environmental triggers) have been shown to influence the adherence to prescribed asthma medications and action plans in several studies.[53] The importance of regular follow-up visits should be emphasized repeatedly. Follow up visits should be at least twice in a year (more often if asthma not well-controlled) and should include lung function monitoring annually.[7,28] The purpose of follow-up appointment should be to explore action plan, evaluate patient goal attainment, identify barriers to meeting activity goals, identify potential treatment adjustments to help meet goals and prevent future exacerbations.[46,54]

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References

1. Masoli M, Fabian D, Holt S et al. The global burden of asthma: executive summary of the GINA Dissemination Committee report. *Allergy.* 2004; 59: 469-78.
2. Martinez FD. Managing childhood asthma: challenge of preventing exacerbations. *Pediatrics.* 2009; 123: S146-50.
3. Global Strategy for Asthma Management and Prevention 2009 (Update). Available from www.ginasthma.org.
4. Lodha R, Walia M. Acute exacerbation of asthma. In: Kabra SK, Lodha R (editors). Essential Pediatric Pulmonology, 2nd edition. New Delhi: Noble Vision; 2010, 175-185.
5. Cohen NH, Eigen H, Shaughnessy TE. Status asthmaticus. *Crit Care Clin.* 1997; 13: 459–76.
6. Robinson PD, Van Asperen P. Asthma in Childhood. *Pediatr Clin N Am.* 2009; 56: 191–226.
7. Liu AH, Covar RA, Spahn JD, Leung DYM. Childhood Asthma. In: Kliegman RM, Stanton B, Geme JS, Schor N, Behrman RE (editors). Nelson Textbook of Pediatrics, 19th edition. Philadelphia: Elsevier; 2012, 780-81.
8. Strunk RC. Identification of the fatality-prone subject with asthma. *J Allergy Clin Immunol.* 1989; 83: 477-485.
9. DeNicola LK, Monem GF, Gayle MO, Kissoon N. Treatment of critical status asthamaticus in children. *Pediatr Clin North Am.* 1994; 41: 1293-324.
10. McFadden ER, Jr, Warren EL. Observations on asthma mortality. *Ann Intern Med.* 1997; 127: 142-7.
11. National Asthma Education and Prevention Program: Expert panel report III: Guidelines for the diagnosis and management of asthma. (NIH publication no. 08-4051). Bethesda, MD: National Heart, Lung, and Blood Institute, 2007.
12. Wheeler DS, Page K, Shanley TP. Status asthmaticus. In: Wheeler DS, Wong HR, Shanley TP (editors). The Respiratory Tract in Pediatric Critical Illness and Injury. London: Springer-Verlag; 2009, 168-93.
13. Acute Asthma Guideline, Cincinnati Children's Hospital Medical Center: Evidence-based care guideline for management of acute

asthma exacerbation in children Asthma Exacerbation in Children Pediatric Evidence Based Care Guidelines, Cincinnati Children's Hospital Medical Center, Guideline 4, pages 1-35, September 16, 2010. www.cincinnatichildrens.org/svc/alpha/h/health-policy/ev-based/asthma.htm.

14. Belessis Y, Dixon S, Thomsen A, et al. Risk factors for an intensive care unit admission in children with asthma. *Pediatr Pulmonol.* 2004; 37: 201-9.
15. Canny GJ, Reisman J, Healy R, et al. Acute asthma: observations regarding the management of a pediatric emergency room. *Pediatrics.* 1989; 83: 507-12.
16. Kling S, Goussard P, Gie RP. The treatment of acute asthma in children. *Current Allergy & Clinical Immunology.* 2011; 24: 22-6.
17. Geelhoed GC, Landau LI, Le Souëf PN. Evaluation of SaO2 as a predictor of outcome in 280 children presenting with acute asthma. *Ann Emerg Med.* 1994; 23: 1236-41.
18. Gorelick MH, Stevens MW, Schultz T, Scribano PV. Difficulty in obtaining peak expiratory flow measurements in children with acute asthma. *Pediatr Emerg Care.* 2004; 20: 22-6.
19. Powell CV, Kelly AM, Kerr D. Lack of agreement in classification of the severity of acute asthma between emergency physician assessment and classification using the National Asthma Council Australia guidelines (1998). *Emerg Med.* 2003; 15: 49-53.
20. Scarfone RJ, Fuchs SM, Nager AL, Shane SA. Controlled trial of oral prednisone in the emergency department treatment of children with acute asthma. *Pediatrics.* 1993; 92: 513-8.
21. Parkin PC, Macarthur C, Saunders NR, Diamond SA, Winders PM. Development of a clinical asthma score for use in hospitalized children between 1 and 5 years of age. *J Clin Epidemiol.* 1996; 49: 821-5.
22. Chalut DS, Ducharme FM, Davis GM. The Preschool Respiratory Assessment Measure (PRAM): A responsive index of acute asthma severity. *J Pediatr.* 2000; 137: 762-8.
23. Rowe BH, Bretzlaff J, Bourdon C, Bota G, Blitz S, Camargo CA. Magnesium sulfate for treating exacerbations of acute asthma in the emergency department. *Cochrane Database Sys Rev.* 2000; (2): CD001490.
24. Kim IK, Phrampus E, Venkataraman S et al. Helium/oxygen-driven albuterol nebulization in the treatment of children with moderate to severe asthma exacerbations: a randomized, controlled trial. *Pediatrics.* 2005; 116: 1127-33.
25. Garbutt J, Highstein G, Nelson KA, Rivera-Spoljaric K and Strunk R. Detection and home management of worsening asthma symptoms. *Annals of Allergy, Asthma, & Immunology.* 2009; 103: 469-73.
26. Expert Panel Report 3 (EPR-3): Guidelines for the Diagnosis and Management of Asthma-Summary Report 2007. *Journal of Allergy & Clinical Immunology.* 2007; 120: S94-138.
27. Bhogal S, Zemek R, Ducharme FM. Written action plans for asthma in children. *Cochrane Database Sys Rev.* 2006;(3) CD005306.
28. O Ortiz-Alvarez, A Mikrogianakis. Canadian Paediatric Society, Acute Care Committee. *Paediatr Child Health.* 2012; 17: 251-5.
29. Yock Corrales A, Soto-Martinez M, Starr M. Management of severe asthma in children. *Aust Fam Physician.* 2011; 40: 35-8.
30. Plotnick LH, Ducharme FM. Acute asthma in children and adolescents: should inhaled anticholinergics be added to beta 2-agonists? *Am J Respir Med.* 2003; 2: 109-115.
31. Browne GJ, Penna AS, Phung X, Soo M. Randomised trial of intravenous salbutamol in early management of acute severe asthma in children. *Lancet.* 1997; 349: 301-5.
32. Sin DD, Man SF. Low-dose inhaled corticosteroid therapy and risk of emergency department visits for asthma. *Arch Intern Med.* 2002; 162: 1591-5.
33. Qureshi F, Zaritsky A, Poirier MP. Comparative efficacy of oral dexamethasone versus oral prednisone in acute pediatric asthma. *J Pediatr.* 2001; 139: 20-6.
34. Kelly AM, Kerr D, Powell C. Is severity assessment after one hour of treatment better for predicting the need for admission in acute asthma? *Respir Med.* 2004; 98: 777-81.
35. Global Initiative for Asthma. Global strategy for the diagnosis and management of asthma in children 5 years and younger, 2009. Available from www.ginasthma.com/download.asp?intId=380.
36. Camargo CA, Spooner C, Rowe BH. Continuous versus intermittent beta-agonists for acute asthma. *Cochrane Database Syst Rev.* 2003; (4): CD001115.
37. Ballester E, Reyes A, Roca J, Guitart R , Wagner

PD, Rodriguez-Roisin R. Ventilation-perfusion mismatching in acute severe asthma: effects of salbutamol and 100% oxygen. *Thorax*. 1989; 44: 258-67.

38. Rodrigo GJ, Rodriquez Verde M, Peregalli V, Rodrigo C. Effects of short-term 28% and 100% oxygen on PaCO2 and peak expiratory flow rate in acute asthma: a randomized trial. *Chest*. 2003; 124: 1312-7.
39. Sole D, Komatsu MK, Carvalho K V, Naspitz C K. Pulse oximetry in the evaluation of the severity of acute asthma and/or wheezing in children. *J Asthma*. 1999; 36: 327-33.
40. Silverman R. Treatment of acute asthma. *Clin Chest Med*. 2000; 21: 361-79.
41. Alter HJ, Koepsell TD, Hilty WM. Intravenous magnesium as an adjuvant in acute bronchospasm: a meta-analysis. *Ann Emerg Med*. 2000; 36: 191-7.
42. Carroll CL, Smith SR, Collins MS, Bhandari A, Schramm CM, Zucker AR. Endotracheal intubation and pediatric status asthmaticus: Site of original care affects treatment. *Pediatr Crit Care Med*. 2007; 8: 91-5.
43. Williams TJ, Tuxen DV, Scheinkestel CD, Czarny D, Bowes G. Risk factors for morbidity in mechanically ventilated patients with acute severe asthma. *Am Rev Respir Dis*. 1992; 146: 607-15.
44. Cox RG, Barker GA, Bohn DJ. Efficacy, results and complications of mechanical ventilation in children with status asthamaticus. *Pediatr Pulmonol*. 1991; 11: 120-6.
45. Tuxen DV. Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airflow obstruction. *Am Rev Respir Dis*. 1989; 140: 5-9.
46. Zorc JJ, Scarfone RJ, Li Y. Predictors of primary care follow-up after a pediatric emergency visit for asthma. *J Asthma*. 2005; 42: 571-6.
47. Mansour ME. How do we support follow-up with the primary care provider after an emergency department visit for asthma? *Pediatrics*. 2009; 124: 1206-7.
48. Hussain-Rizvi, A, Kunkov S, Crain EF. Does parental involvement in pediatric emergency department asthma treatment affect home management? *J Asthma*. 2009; 46: 792-5.
49. Boyd, M, Lasserson TJ, McKean MC, Gibson PG, Ducharme FM, Haby M. Interventions for educating children who are at risk of asthma-related emergency department attendance. *Cochrane Database of Sys Rev*. 2009; (2): CD001290.
50. Goldberg S, Springer C, Avital A, Godfrey S, Bar-Yishay E. Can Peak Expiratory Flow Measurmenets Estimate Small Airway Function in Asthmatic Children? *Chest*. 2001; 120: 482-8.
51. Lanphear BP, Aligne CA, Auinger P, Weitzman M, Byrd RS. Residential Exposures Associated with Asthma in US Children. *Pediatrics*. 2001; 107: 505-11.
52. Boyd M, Lasserson TJ, McKean MC, Gibson PG, Ducharme FM, Haby M. Interventions for educating children who are at risk of asthma-related emergency department attendance. *Cochrane Database Sys Rev*. 2009; (2): CD001290.
53. Jones KG, Bell J, Fehrenbach C, Pearce L, Grimley D, McCarthy TP. Understanding patient perceptions of asthma: Results of the Asthma Control and Expectations (ACE) survey. *International Journal of Clinical Practice*. 2002; 56: 89-93.
54. Flores G, Abreu M, Tomany-Korman S, Meurer J. Keeping children with asthma out of hospitals: parents' and physicians' perspectives on how pediatric asthma hospitalizations can be prevented. *Pediatrics*. 2005; 116: 957-65.