Project: Ghana Emergency Medicine Collaborative

Document Title: Acute Asthma in Adults

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Background & Epidemiology

• Defined as a chronic inflammatory disorder characterized by increased airway responsiveness to multiple stimuli

• In susceptible people this results in recurrent episodes of wheezing, breathlessness, chest pain/tightness and or coughing
Background & Epidemiology

• Again as I have no epi data in Ghana it’s unclear what the burden on this disease is.
• In the U.S. asthma is the number 1 chronic disease of childhood
• Affects 4-5% of the population
• Roughly 50% of the cases develop before the age of 10
Background & Epidemiology

• In childhood there is a male predominance of 2:1
• This equalizes by age 30
• Although the developed nations have seen a steady increase in the numbers of cases
• The number of hospitalizations and deaths have seen a steady decline since the mid 1990’s
Pathophysiology

• This chronic inflammatory disease is characterized by abnormal accumulation of
  – Eosinophils, lymphocytes, mast cells, macrophages, dendritic cells and myofibroblasts

• The hallmark is the reduction of airway diameter that is caused by:
  – Smooth muscle contraction, vascular congestion, bronchial wall edema, and thick secretions
Pathophysiology

• This all results in the increased work of breathing and altered pulmonary function
• There is also a redistribution of pulmonary blood flow related to this inflammatory process
• The current theory is that there are three phases of the airway inflammation
Pathophysiology

• Acute phase:
  – There is early recruitment of cells to the airway
  – Antigens come in contact with mast cells in the submucosa and lead to elaboration of inflammatory mediators
    • Such as histamine, leukotrienes, chemokines and pro-inflammatory cytokines and multiple interleukins

• This leads to increased mucus production, decreased ciliary action and of course bronchoconstriction and vascular congestion
Pathophysiology

• Sub acute/ Late phase:
  – The eosinophils, platelets and polymorphonuclear leukocytes that are recruited, become activated
  – IgE response is also activated by the contact with the antigen

• This all creates a more persistent pattern of inflammation
Clinical Features

• Typically the symptoms consist of a triad of dyspnea, wheezing and cough
• Many patients will relay a history of asthma on presentation
• Early in the process they may describe the discomfort as a chest tightness or constricted feeling
Clinical Features

• As the exacerbation progresses the wheezing become evident
• Expiratory phase become prolonged
• Accessory muscle use may become evident
Physical Examination

• The physical exam can vary greatly depending on the patient and phase of presentation
• Some maybe in obvious respiratory distress with tachypnea and loud wheezing
• Others may present with persistent cough and mild end-expiratory wheeze
• The use of accessory muscles indicates diaphragmatic fatigue
Physical examination

• Signs of impending respiratory failure include
  – Paradoxical respirations
  – Alterations in mental status
    • Hallucinations, agitation, confusion
  – “silent” chest
Physical Examination

• Findings may reveal
  – Hyper resonance to percussion
  – Decreased air movement
  – Prolonged expiratory phase
  – Usually wheezes
  – Pulses Paradoxus
  – Tachypnea
  – Tachycardia
Evaluation

• Bedside spirometry provides a rapid objective assessment
• Can serve as a guide to the effectiveness of your therapy
• Can be measured as the Forced Expiratory volume in 1 minute (FEV1) or as the Peak Expiratory Flow Rate (PEFR)
Evaluation

• Chest X-ray
• Arterial Blood gas
• CBC
• Electrolytes
• Other studies
Management

• The primary goals of therapy for acute severe asthma are the rapid reversal of airflow obstruction and the correction.

• Airflow obstruction is most rapidly alleviated by the combination of repeated administration of inhaled bronchodilators and early institution of systemic glucocorticoids.

• Until their respiratory distress has abated, patients should receive close monitoring, including serial measurements of lung function (FeV1 or PEFR), to assess the response to treatment.
Management

• Inhaled beta agonists:
  • Are the mainstay of bronchodilator treatment.
    – short-acting beta-2-selective adrenergic agonists, such as albuterol, levalbuterol.
• The standard regimen for initial care in the emergency department has become albuterol 2.5 to 5 mg by continuous flow nebulization every 20 minutes for three doses
• Then 2.5 to 10 mg every one to four hours as needed.
• For critically ill patients, some clinicians prefer continuous nebulization, administering 10 to 15 mg over one hour.
Management

• Inhaled anticholinergics
  — The most recent guidelines for asthma management prepared by the Expert Panel of the National Asthma Education and Prevention Program (Expert Panel Report III) recommend the addition of ipratropium for patients with severe exacerbations who are in the emergency department.
• The adult dosing of ipratropium for nebulization is 500 mcg every 20 minutes for three doses.
• Alternatively, ipratropium can be administered by MDI at a dose of eight inhalations every 20 minutes, then as needed for up to three hours.
• Some investigators have reported that the combination provides greater bronchodilation than beta agonists alone, particularly among patients with the most severe airflow obstruction.
Management

• Systemic glucocorticoids
  – Continued wheezing and shortness of breath despite intensive bronchodilator therapy most likely represents persistent airflow obstruction on the basis of airway inflammation and intraluminal mucus plugging.
  – Studies show that among patients with significant airflow obstruction despite intensive treatment with bronchodilators, systemic glucocorticoids speed the rate of improvement.

• Current guidelines encourage early systemic glucocorticoids for all patients who have a moderate (peak expiratory flow <70 percent of baseline) or severe exacerbation (peak expiratory flow <40 percent of baseline).

• Oral vs. IV administration?
Management

• Orally
  – Prednisone 40-60mg

• IV
  – Methylprednisone 60-125mg IV
  – Hydrocortisone 100-200mg IV
Management

• Magnesium sulfate —
  – Intravenous magnesium sulfate (2 gm infused over 20 min) has bronchodilator activity in acute asthma.
  – two systematic reviews concluded that it is helpful in the subgroup of patients with severe attacks.

• This agent is suggested for patients who have life-threatening exacerbations or whose exacerbation remains severe
Older Alternatives

• Methylxanthines —
  – The use of alternative bronchodilators such as intravenous theophylline or aminophylline, in addition to beta-agonists, is not recommended in the treatment of acute exacerbations.
  – These agents are not as potent as the beta agonists when used alone for the treatment of asthma, and provide no further bronchodilation beyond that achieved with inhaled beta agonists alone.
  – In addition, these agents appear to increase the incidence of adverse effects when combined with bronchodilators.
  – Studies extending over several hours of emergency department care have also failed to show a benefit of theophylline therapy in terms of course or duration of hospitalization.
  – For patients who are taking oral theophylline at presentation, we typically continue maintenance oral therapy (ideally after checking a blood level.)
Management of asthma exacerbations: emergency department and hospital-based care

**Initial assessment**
Brief history, physical examination (auscultation, use of accessory muscles, heart rate, respiratory rate), PEF or FEV₁, oxygen saturation, and other tests as indicated.

**FEV₁ or PEF ≥40 percent (mild-to-moderate)**
- Oxygen to achieve SaO₂ ≥90 percent
- Inhaled SABA by nebulizer or MDI with valved holding chamber, up to 3 doses in first hour
- Oral systemic corticosteroids if no immediate response or if patient recently took oral systemic corticosteroids

**FEV₁ or PEF <40 percent (severe)**
- Oxygen to achieve SaO₂ ≥90 percent
- High-dose inhaled SABA plus ipratropium by nebulizer or MDI plus valved holding chamber, every 20 minutes or continuously for 1 hour
- Oral systemic corticosteroids

**Impending or actual respiratory arrest**
- Intubation and mechanical ventilation with 100 percent oxygen
- Nebulized SABA and ipratropium
- Intravenous corticosteroids
- Consider adjunct therapies

**Repeat assessment**
Symptoms, physical examination, PEF, O₂ saturation, other tests as needed

**Admit to hospital intensive care (see box below)**

**Moderate exacerbation**
FEV₁ or PEF 40-69 percent predicted/personal best
Physical exam: moderate symptoms
- Inhaled SABA every 60 minutes
- Oral systemic corticosteroid
- Continue treatment 1-3 hours, provided there is improvement; make admit decision in <4 hours

**Severe exacerbation**
FEV₁ or PEF <40 percent predicted/personal best
Physical exam: severe symptoms at rest, accessory muscle use, chest retraction
History: high-risk patient
No improvement after initial treatment
- Oxygen
- Nebulized SABA + ipratropium, hourly or continuous
- Oral systemic corticosteroids
- Consider adjunct therapies
**Good response**
- FEV₁ or PEF ≥70 percent
- Response sustained 60 minutes after last treatment
- No distress
- Physical exam: normal

**Incomplete response**
- FEV₁ or PEF 40-69 percent
- Mild-to-moderate symptoms

**Poor response**
- FEV₁ or PEF <40 percent
- PCO₂ ≥42 mmHg
- Physical exam: symptoms severe, drowsiness, confusion

Individualized decision re: hospitalization

**Discharge home**
- Continue treatment with inhaled SABA
- Continue course of oral systemic corticosteroid
- Consider initiation of an ICS
- Patient education
  - Review medications, including inhaler technique
  - Review/initiate action plan
  - Recommend close medical followup

**Admit to hospital ward**
- Oxygen
- Inhaled SABA
- Systemic (oral or intravenous) corticosteroid
- Consider adjunct therapies
- Monitor vital signs, FEV₁ or PEF, SaO₂

**Admit to hospital intensive care**
- Oxygen
- Inhaled SABA hourly or continuously
- Intravenous corticosteroid
- Consider adjunct therapies
- Possible intubation and mechanical ventilation

**Discharge home**
- Continue treatment with inhaled SABAs
- Continue course of oral systemic corticosteroid
- Continue on ICS. For those not on long-term control therapy, consider initiation of an ICS
- Patient education (eg, review medications, including inhaler technique and, whenever possible, environmental control measures; review/initiate action plan; recommend close medical followup)
- Before discharge, schedule followup appointment with primary care provider and/or asthma specialist in 1–4 weeks

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FEV₁: forced expiratory volume in 1 second; ICS: inhaled corticosteroid; MDI: metered dose inhaler; PCO₂: partial pressure carbon dioxide; PEF: peak expiratory flow; SABA: short-acting beta₂-agonist; SaO₂: oxygen saturation.

<table>
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<th>Medication</th>
<th>Dosage form</th>
<th>Adult dose</th>
<th>Comments</th>
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<td>Methylprednisolone</td>
<td>2, 4, 8, 16, 32 mg tablets</td>
<td>7.5-60 mg daily in a single dose in am or qod as needed for control</td>
<td>For long-term treatment of severe persistent asthma, administer single dose in am either daily or on alternate days (alternate-day therapy may produce less adrenal suppression). If daily doses are required, one study suggests improved efficacy and no increase in adrenal suppression when administered at 3:00 pm (Beam et al 1992). Short courses or &quot;bursts&quot; are effective for establishing control when initiating therapy or during a period of gradual deterioration. The burst should be continued until patient achieves 80 percent PEFR personal best or symptoms resolve. This usually requires 3 to 10 days but may require longer. There is no evidence that tapering the dose following improvement prevents relapse.</td>
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<td>Prednisolone</td>
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PEFR: peak expiratory flow rate; qod: every other day.
Severe asthma exacerbation in adults: Rapid overview

Clinical Danger Signs
- Use of accessory muscles of respiration; brief, fragmented speech; inability to lie supine; profound diaphoresis; agitation
- Life-threatening airway obstruction can still occur when these signs are NOT present
- Inability to maintain respiratory effort, cyanosis, and depressed mental status portend imminent respiratory arrest

Assessment
- Measurement of expiratory airflow is the best measure of severity; peak expiratory flow rate < 40 percent predicted indicates severe obstruction
- Severe hypoxemia (SpO2 < 95% despite high flow O2 by nonrebreather mask) portends imminent respiratory arrest; continuous pulse oximetry should be performed
- Arterial blood gas evaluation does not assist management; it can aid assessment if intubation is not an immediate concern
- Chest radiograph is generally unhelpful; obtain if complications suspected, diagnosis in doubt, or patient is high-risk

Standard treatments
- Inhaled beta agonist (albuterol)
- Oxygen
- IV access, normal saline
- Ipratropium bromide
- Corticosteroids
- Magnesium sulfate

Additional treatments
- Terbutaline
- Epinephrine
- Heliox (helium and oxygen)

Endotracheal intubation and ventilation
- Decision to intubate during first few minutes of severe asthma attack is clinical
- Goal of mechanical ventilation is to maintain adequate oxygenation and ventilation while minimizing elevated airway pressures
## Predicted average peak expiratory flow for normal males (L/min)

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These values represent average normal values within 100 L/min. Predicted values for African American and Hispanic minorities are approximately 10 percent lower.

## Predicted average peak expiratory flow for normal females (L/min)

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These values represent average normal values within 80 L/min. Predicted values for African American and Hispanic minorities are approximately 10 percent lower.

## Predicted average peak expiratory flow rates for normal children

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<th>Height (inches)</th>
<th>PEFR (L/min)</th>
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