# embocs The EM Educator Series

The EM Educator Series: The Sick Adult Asthma Patient Author: Alex Koyfman, MD (@EMHighAK) // Edited by: Brit Long, MD (@long\_brit) and Manpreet Singh, MD (@MprizzleER)

**Case#1:** A 48-year-old male presents with diffuse wheezes and elevated respiratory rate. He is in respiratory distress. His wife says he has a history of severe asthma, and he has not been able to utilize his controller medications in the last week.

**Case#2:** A 29-year-old female comes in with somnolence and decreased air movement bilaterally. Per EMS, she has a history of severe asthma.

## **Questions for Learners:**

- 1. What conditions can mimic asthma?
- 2. What are red flags in the history and exam for severe asthma, as well as mimics?
- 3. What other medications should you consider beyond nebulizers and steroids? What place do magnesium, ketamine, and epinephrine have?
- 4. Is NIPPV effective for respiratory distress in asthma?
- 5. How can ultrasound help?
- 6. While you try to avoid it if possible, how do you optimize intubation?
- 7. What ventilator settings should you use after intubation? What is permissive hypercapnia?
- 8. What should you consider and do you do when the patient crashes after intubation?

### **Suggested Resources:**

- ✓ Articles:
  - o <u>emDocs Mimics</u>
  - o <u>LITFL Severe Asthma</u>
  - o <u>CoreEM Life-threatening asthma</u>
  - o First 10 EM
- ✓ Podcasts:
  - o <u>REBEL EM Crashing Asthmatic</u>
  - <u>REBEL EM Obstructive Physiology</u>
  - o <u>EMCrit Severe Asthmatic</u>
  - <u>EMCrit Finger Thoracostomy</u>

## **Answers for Learners:**

### 1. What conditions can mimic asthma?

**Anaphylaxis:** This condition presents with similar pathophysiology as asthma with hyperactive immune response and bronchoconstriction. It accounts for close to 1% of ED visits, though onset of symptoms is more acute than in asthma with history of exposure to an allergen. Anaphylaxis involves two organ systems or a known allergic exposure with low blood pressure. Exam usually reveals urticaria (10-20% will not have rash), and nausea, vomiting, or diarrhea may also be observed. Treatment requires immediate resuscitation with epinephrine, steroids, intravenous (IV) fluids, and histamine antagonists. Any concern for anaphylaxis warrants intramuscular epinephrine.

**COPD:** COPD classically has been thought to be irreversible and associated with tobacco use. Like asthma, it is an obstructive airway disease. The CHAIN study described Asthma-COPD Overlap Syndrome (ACOS or ACO). In this study, 15% of patients with COPD have features of asthma, while 15% of patients with asthma have COPD. Official diagnosis is often done by pulmonologist, though asthma is usually diagnosed in young adulthood and COPD usually after age 40 with history of bronchodilator responsiveness, though these are not completely reliable.5 Initial treatment of COPD is similar to asthma, though long term medication management differs (such as Long Acting Beta Agonists (LABA) in COPD improving mortality). In asthma, LABA use may increase mortality if used without inhaled steroids.

**Heart Failure:** Heart failure may result in wheezing with lung fluid accumulation. Some patients may not be diagnosed with heart failure upon initial presentation, and patients may present acutely with respiratory distress or with chronic worsening orthopnea and dyspnea (the most common presentation). Flash pulmonary edema (the acute version) can be due to ischemia, acute valvular pathology, cardiomyopathy, and other conditions. Exam may reveal rales, S3 heart sound, elevated JVP, and edema. Patients may have a history of dyspnea with exertion or with lying flat. Acute exacerbation with pulmonary edema requires venodilators and positive pressure ventilation (CPAP or BiPAP). However, treatment should be tailored to the patient. Diuretics can be used after venodilation. The underlying etiology should be treated as well.

**PE:** PE is not always an easy diagnosis, though emergency physicians evaluate patients with potential PE on a regular basis. Close to 1 in 10 patients with PE may have wheezing on exam.9 This may be due to vasoactive mediators released in response to the embolus.10 The key is to evaluate for PE risk factors in the history. If patients do not improve with standard asthma medications, PE should also be considered. Evaluation includes risk stratification such as Wells criteria, with D-dimer and CT pulmonary as needed. Significant PE requires anticoagulation.

**Foreign body aspiration:** A small object in the bronchioles from aspiration can result in focal lung finds, and worse, respiratory distress. Patients with greatest risk for this include children, older adults, and those mentally disabled. Patients may present with acute cough or wheezing. The classic history is lack of preceding upper respiratory symptoms or prior wheezing. Choking followed by cough is present in 75% of pediatric patients with foreign body aspiration. Chest x-ray can help, erect and lateral, which may show hyperinflation. Bronchoscopy is required if concern for aspiration, even with negative chest x-ray.

**Angioedema:** "Angioedema" refers to localized swelling in locations with loose connective tissue, including the face, oropharynx, bowel wall, extremities, and genitalia. There are many causes. Primary

pathology includes mast cell-mediated and bradykinin-mediated. Mast-cell mediated angioedema can have other signs of allergic reaction such as urticaria. Bradykinin-induced angioedema is not associated with urticaria and pruritis, as it is not allergic. This form is the cause in ACE-inhibitor-induced angioedema and Hereditary Angioedema (HAE). Angioedema associated with systemic findings such as upper airway swelling, wheezing, abdominal pain, nausea, and vomiting should be managed as anaphylaxis until proven otherwise and treated with epinephrine, antihistamines, and corticosteroids. Rapid airway protection with intubation may be required, and a difficult airway should be assumed with adjuncts available.

**Drug Related Wheezing:** This is most commonly associated with aspirin. Aspirin Exacerbated Respiratory Disease, or Samter's Triad, consists of asthma, nasal polyps, and sensitivity to aspirin or other NSAIDs. Non-selective beta-blockers can precipitate wheezing in patients with underlying disease (such as asthma). ACE-inhibitors, while commonly causing dry cough, may result in bronchospasm and wheezing. Many drugs can cause asthma symptoms, or worsen asthma in patients with known disease. The risks and benefits of continuing aspirin or an NSAID must be strongly considered, or an alternative can be used.

**Central Airway Obstruction:** Obstruction in the trachea and main stem bronchi include bronchogenic carcinomas, tracheal strictures, sarcoidosis, and goiter. Though these usually do not present acutely, patients may notice other symptoms including weight loss. If the obstruction is fixed, wheezing will likely be constant and fixed. Asthmatic wheezing usually occurs after exposure to allergen or upper respiratory infection. Wheezing due to fixed obstruction may be localized on exam. Further evaluation and testing relies on the history and exam. Risk factors for cancer, symptoms consistent with hyperthyroidism, or a history of intubation should be obtained. Chest CT will most likely identify the obstruction, though the patient must be able to lie supine. Further consultation is required if diagnosed, with bronchoscopy as well.

**Vocal Cord Dysfunction:** Paradoxical, involuntary closure of the vocal cords during inspiration, (or edema of the vocal cords) may result in wheezing and acute respiratory distress. Wheezing in this condition is usually inspiratory or inspiratory and expiratory (asthmatic wheezing is mostly expiratory). This is due to forced expiration stenting open the cords and decreasing the obstruction. Patients often present soon after exposure to a stressor, inhaled irritants, or exercise. Risk factors include prior intubation or neck surgery (leading to possible damage to the recurrent laryngeal nerve). Diagnosis is often confirmed through laryngoscopy. Treatment of vocal cord dysfunction includes several maneuvers. Panting, which can abduct the vocal cords through activation of the posterior cricoarytenoids, breathing through a straw, patient jaw thrust while breathing, nose to mouth breathing, administration of a helium-oxygen mixture such as heliox, or continuous positive airway pressure (CPAP) can be used. The condition is often self-limited and usually does not require other invasive interventions.

# 2. What are red flags in the history and exam for severe asthma, as well as mimics?

The approach for asthma and asthma mimics is to first assess for life threats, provide emergent interventions, and then obtain history and focused exam. Rapid treatment with bronchodilator and corticosteroids is required. Non-Invasive Positive Pressure Ventilation (NIPPV) should be considered. This modality decreases mortality in COPD and CHF, though in asthma more literature is needed.

Once treatment and initial stabilization have been completed, focused history and exam should be conducted to evaluate for mimics. As discussed, there are many causes of wheezing, and these conditions must be considered to prevent anchoring bias. Red flags for other conditions include absence of asthma history, prior history of mild asthma, or poor response to therapy. Most patients with asthma improve with treatment, as suggested in a retrospective study of over 13,000 patients.

Patients who do not improve with asthma treatment including anticholinergic/beta-agonist nebulizer treatment, corticosteroids, magnesium, and further resuscitation measures require more evaluation such as ECG, imaging (x-ray, CT, or US), and laboratory studies. These measures may allow physicians to diagnose and treat another underlying disease.

# 3. What other medications should you consider beyond nebulizers and steroids? What place do magnesium, ketamine, and epinephrine have?

- Consider using IM Epinephrine 0.3 0.5mg of 1:1000. Severe asthmatics will have a hard time getting inhaled beta agonists into the small airways, but parenteral epinephrine will get into the circulation and get to where it needs to provide support.
- Non-Invasive Positive Pressure Ventilation (NIPPV) can help decrease work of breathing and help push beta agonist into the smaller airways.
- Give IV magnesium sulfate for its bronchodilator effect, but don't just give 2g IV x1. Instead give 2g IV and repeat it upto 2 more times over an hour.
- Give IVF at a dose of 30cc/kg because patients with acute asthma exacerbations because patients will have insensible losses. Also if you are going to intubate patients with asthma, once they are intubated due to the positive pressure ventilation they will have a decrease in preload and IVF will help maximize preload.
- Sub-dissociative dose IV Ketamine (0.1 mg/kg followed by IV infusion of .5 mg/kg/hour for 3 hours) may be helpful to facilitate use of BiPAP in a hypoxic/combative patient. If you have gotten to this step you should start setting up for intubation at this point in case the patient continues to deteriorate.
- Intravenous epinephrine can be considered as well. In hypotensive patients, it is possible that the IM epinephrine is just not circulating. REMEMBER we want to use the 1:10,000 concentration here and even think about push dose epinephrine.

# 4. Is NIPPV effective for respiratory distress in asthma?

If the patient is not improving with maximal medical management, it is time to start thinking about positive pressure ventilation and intubation. A common teaching is: when thinking about intubating an asthma patient, wait, and then wait some more, and then continue to wait, but don't wait too long. If you are considering intubation, BiPAP should almost certainly be tried first. Remember that putting a piece of plastic in the trachea does nothing to help these patients. In fact, it increases airway resistance and dead space. The reason you considering intubation is because of respiratory fatigue and BiPAP can provide exactly the pressure support that these patients need.

# How to use NIPPV

- Constantly reasses these patients
- Have all intubation equipment ready at the bedside
- The benefit is all in the pressure support. Start around 8-10mmHg

• Set the PEEP very low (1-2), or none at all if your machine will allow

Most importantly, continue providing beta2 agonists and the full kitchen sink of medical management. NIPPV only allows the patient to temporarily rest their respiratory muscles, it does not solve the underlying asthma pathophysiology

# 5. How can ultrasound help?

Evaluate for asthma mimics (See section above for details) in use of using A vs B lines.

# 6. While you try to avoid it if possible, how do you optimize intubation?

- Don't forget NO DESAT (Nasal Oxygen During Efforts Securing A Tube). This will buy you a longer apneic period while trying to intubate.
- Ketamine 1 2 mg/kg IV is the ideal induction agent due to its bronchodilatory effects. Remember you already used a sub-dissociative dose in your medical management so why change to a different agent.
- Rocuronium 1.2 mg/kg IV is the ideal paralytic agent as it will keep the patient paralyzed close to 90 minutes which can help with ventilator management.

# 7. What ventilator settings should you use after intubation? What is permissive hypercapnia?

- Permissive Hypercapnia allows for CO2 to rise and pH to drop in order to avoid auto-peep and barotrauma.
  - Respiratory Rate of 6 8 breaths/min
  - Tidal Volume of 6cc/kg of IDEAL BODY WEIGHT
  - Peak Flow 90 120 L/min
  - All of the above increases the Inspiration/Expiration time to allow patients to not auto-peep
- If still having issues be sure to trouble shoot the ventilator.
  - Remember Peak Pressure is the maximum amount of pressure sensed by the ventilator in the ventilator circuit and doesn't reflect what is going on in the lungs
  - We care about Plateau Pressure which is the pressure in the lower airways. Shoot for a Plateau Pressure of <30 mmHg. (If you hit the expiratory pause button on the ventilator, this will give you the plateau pressure)
- Ventilator Asynchrony occurs due to patients being tachypneic and breathing against the ventilator and start to breath stack and develop more auto-peep. The solution for this is to keep the patient paralyzed and appropriately sedated. (Remember these patients are on high doses of steroids and critical care myopathy and long term paralysis is a real thing, so don't keep them paralyzed for more than 2 – 3 hours).
- If Plateau pressures are still running high you can consider a couple other things as final ditch efforts:
  - Inhalational Anesthetics by anesthesiology in the operating room due to their bronchodilatory effects
  - ED ECMOV-V ECMO will help get oxygen into the bloodstream (Checkout edecmo.org)

# 8. What should you consider and do you do when the patient crashes after intubation?

- If the patient is having hemodynamic instability, 1st disconnect them from the ventilator (removes equipment failure from the equation) and forcefully exhale the patient by pushing down on their chest softly (this can take 20 30 seconds), finally think about the DOPES mnemonic:
  - o Displacement of endotracheal tube
  - $\circ \quad \text{Obstruction of endotracheal tube}$
  - o Pneumothorax
  - Equipment Failure
  - Stacked Breaths