PICU Asthma Quick Guide

Pathophysiology

- Pathophysiology of Asthma: narrowing of the airways secondary to smooth muscle spasm/bronchoconstriction, mucous plugging, and airway edema/inflammation.
    - Inflammation - mast cells, T lymphocytes, and eosinophils release cytokines (histamine, leukotrienes, and platelet-activating factor).
        - Various triggers: Allergens, infections, irritants (smoke), emotional stress, reflux, exercise, and medications.
    - Mucous Plugging: Goblet cells → hypersecretion of mucus
    - Bronchospasm - parasympathetic NS → Acetylcholine → narrowing of the airway.
    - Results in reduced airway diameter, severe airflow obstruction, and increased airway resistance.
- Lung Mechanics
    - Asthma = obstructive lung disease. Lungs are hyperinflated with air trapping.
    - Hyperinflation of alveoli causes V/Q mismatch → see arterial O2 desaturation.
- Cardiopulmonary Effects
    - Right ventricular afterload increases - secondary to hypoxic pulmonary vasoconstriction, acidosis, and lung inflammation → increased pulmonary vascular resistance and RV dilation
    - Left Ventricular output decreases due to a decrease in LV preload.
        - Dehydration + collapse of systemic veins as they enter thorax) + bowing of the dilated RV into the LV.
    - Increased LV Afterload: due to increased negative intrapleural pressure.
    - Pulsus paradoxus: an exaggerated (>10mmHg) drop in systolic BP with inspiration.
- Metabolism: V/Q mismatch leads to hypoxia, increased work of breathing and dehydration lead to increasing lactate production, and dehydration also leads to ketone production → Metabolic acidosis

Assessment:

- Assessment for Impending Respiratory Failure: mental status (concerning if lethargic/agitated), inability to speak, absence of wheeze or poor air movement, cyanosis, inability to lie down (prefers sitting up), marked pulsus paradoxus or even absent.
    - CXR - Not routinely indicated in asthma but can aid in identifying other causes of respiratory distress and wheezing → pneumonia, pneumothorax, pneumomediastinum, etc.

Treatments: Escalation of routine Floor Management:

- Oxygen: aids in V/Q mismatch, hypoxemia worsens pulmonary vasoconstriction
    - Many different modalities to give - Simple mask, HFNC, NIV.
- Beta-Agonists - stimulate Beta 2 receptors → bronchodilation via smooth muscle relaxation (Ex: Albuterol, epinephrine, isoproterenol, terbutaline)
    - Transition from intermittent to Continuous
    - Albuterol Dosing: Continuous 5-20 mg depending on weight/severity; Intermittent 5mg to 15 mg depending on weight/severity.
- Anticholinergics - Atrovent (Ipratropium) blocks the action of acetylcholine in bronchoconstriction from vagal impulses.
    - Dose = Inhalation of 250 - 500 µg q6
    - Most effective if added in combination with albuterol.
- Systemic Steroids: Transition from PO Dexamethasone to IV Methylprednisolone - Bolus dose 2 mg/kg then 0.5 mg to 1 mg/kg q6hr MAX 80 mg/day . Essential to decreasing inflammation.
    - Increase beta receptors on cell membrane.
    - Side Effects - hyperglycemia, hypertension, acute psychosis, secondary infections. Steroids contraindicated in active or recent exposure to chickenpox.
- Fluids: Most patients are dehydrated, using NS or LR bolus will aid in preload. Overhydration can lead to pulmonary edema. Severe asthma can have SIADH.
- Magnesium: Results in smooth muscle relaxation by blocking calcium uptake → bronchodilation.
    - Dose 50 mg/kg (Max 2g) over 20 minutes, Monitor for hypotension.

<u>Treatment: Escalation to ICU Management with drips</u>

- Terbutaline: IV Beta Agonist - Bolus with 10 mcg/kg and maintenance drip start at 0.4 and increase by 0.2 (usual dose 0.2 -2 µg/kg/min (MAX 5µg/kg/min. Consider when unresponsive to albuterol
    - Side Effects of Beta Agonists - Tachycardia, Agitation/tremor, Hypokalemia. Isoproterenol can cause myocardial ischemia, rare with terbutaline.
- HFNC: Washes out dead space, increases O2 in conducting airways & less rebreathing of CO2. Can add in continuous albuterol in-line.
- Non-Invasive Ventilation: CPAP/BiPAP: Reduces work of breathing, improves atelectasis, & Reduces airway resistance by stenting open distal airways to allow for improved beta 2 agonist delivery.
- Consider Heli-Ox - reduces resistance to airflow by decreasing turbulent airflow and increasing laminar flow. Need at minimum 60% He (70%-80% is best) therefore cannot require > 0.40 FiO2.
- Intubation - BE CAUTIOUS
    - Absolute Indications: Cardiac arrest, Respiratory arrest, Severe hypoxia, rapidly deteriorating mental state
    - Why Caution with intubation?
        - Tracheal foreign body (ETT) can activate bronchospasm.

        - Positive pressure ventilation increases risk for barotrauma & hypotension. 50% of mortality of severe asthma occurs during or immediately after intubation.
    - Preparation to Intubate:
        - Preoxygenation, volume resuscitation, place OG tube to decompress stomach.
        - Premedicate.
            - +/- Lidocaine 1 mg/kg (helps blunt bronchospasm)
            - Ketamine 1-2 mg/kg
                - Direct bronchodilator that is also a dissociative anesthetic & analgesic. Can also be used for sedation with continuous infusion (5-20 mcg/kg/min).
            - Rocuronium 1mg/kg for neuromuscular blockade. Don't use succinylcholine as it results in histamine release.
        - Ventilator Settings:
            - Permissive hypercarbia required to avoid complications.
            - Allow for small tidal volumes (6- 7cc/kg) due to high resistance
            - Lower PEEP of 4-6 to allow for improved expiratory flow resistance, triggering, and work of breathing.
            - Low Rate is better (typically 10-14)
            - Long expiratory times are best - I:E ratios of 1:4 to 1:6 are necessary to allow adequate ventilation and prevent air trapping (Trying to mimic the prolonged expiratory phase that asthmatics have)

<u>Refractory Asthma Treatment for Severe Cases</u>

- Aminophylline & Theophylline IV - Bronchodilator (currently not used at DCMC)
    - Works as a phosphodiesterase inhibitor to cause bronchodilation. Also blocks TNF alpha and leukotrienes.
- Inhalational Anesthetics (rarely really used at DCMC)
    - Sevoflorane - has bronchodilator effects. Can cause hepatotoxicity & malignant hyperthermia. Needs special room, and continuous gas analysis.
    - Isoflurane - similar effects
- ECMO
    - Venovenous bypass for life support in severe asthma refractory to all other treatments. The membrane lung is extremely efficient at CO_2 clearance and low flow.

Author: Natalie Willis
11/2023