Status Asthmaticus & COPD with Respiratory Failure

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North York EMU Europe - Oct 2009

Outline

- Cases to discuss approach to acute severe asthma & COPD with respiratory failure
- Treatment Options
- Diagnostics
- Questions

Learning Objectives

- Outline aggressive treatment strategies to avoid intubation in the patient with Status Asthmaticus & decompensated COPD
- Review intubation techniques & ventilation settings in the event intubation is required in these patients

Disclosures

- None
Asthma Severity: CAEP

Severe
- Laboured respirations
- Agitated, diaphoretic
- Difficulty speaking
- Tachycardic
- No prehospital relief with ß agonist
- FEV₁, PEFR - unable or <40% predicted or best
  - FEV₁ < 1.6L PEFR < 200L/min O₂ saturation < 90%

Asthma Severity: CAEP

Near Death
- Exhausted, confused,
- Diaphoretic, cyanotic,
- Silent chest + poor resp. effort
- Falling heart rate
- O₂ saturation < 90% (despite supplemental O₂)

The Problem

Treatment Goals:
- Correct hypoxia
- Reverse airflow obstruction
- Treat underlying inflammatory response
OXYGEN

- Will not suppress respiratory drive in acute asthma
- Start high: FiO2 40-100%
- Achieve SaO₂ of 92-95%

Initial Treatment Options

- Epi:
  - Dosing:
    - 0.5 mg 1:1000 IM
    - 0.5-1 cc 1:10000 IV q2-3 minutes to effect

- Continuous nebs:
  - Ventolin 5 mg
  - Atrovent 0.5 mg X 3

- Steroid if not already given
- MgSO₄ 2-4 g IV over 15 minutes
- IV Ventolin 4 mcg/kg IVP q 15 min
Alternative Drugs

Heliox:
- Mixture of helium and oxygen
- Low-density gas mixture which may reduce turbulent airflow
- Must be at least 60% helium which presents a problem in hypoxic patients
- Evidence is limited
- Can be considered in a limited group of non-hypoxic severe asthmatics

Now What to Do if all That Fails...

Intubation technique:
- Keep patient sitting
- Ketamine 1-2 mg/kg IV
- Succinylcholine 1.5 mg/kg or Rocuronium 1 mg/kg

Forced Exhalation
- Slow BVM ventilation and or very conservative initial ventilator settings

Ventilatory Strategies:
- Cautious CO2 reduction with permissive hypercapnea until lung function improves
- Slow RR (6-8 breaths/min) to reduce barotrauma & volutrauma
- Low tidal volumes (6-8 mL/kg)
- Low I:E ratios
- Bicarb as needed to keep pH>7.2
- Frequent suctioning of mucous secretions

Ventilator Settings
- May need to switch to a pressure control mode if peak airway pressures or plateau pressures are consistently higher than 40 or 50
Now what if you can’t get the pressures down or you can’t oxygenate the patient?

- Consider barotrauma!!!!
- Maintain paralysis
- Inhalational anesthesia

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**COPD: Definitions**

**Chronic Bronchitis:**
- Chronic productive cough for 3 months in each of 2 successive years in a patient in whom other causes of chronic cough have been excluded

**Emphysema:**
- Abnormal permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis

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**Causes of AECOPD**

- Superimposed respiratory infection
- Cardiovascular deterioration
- Smoking
- Noncompliance with meds
- Environmental exposures
- Meds: e.g. β-blockers, benzos, narcotics
- Misuse of oxygen therapy
- Metabolic derangements

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**Clinical Presentation**

**Acute exacerbation of COPD:**
- Patients present complaining of:
  - Worsening dyspnea
  - Increased sputum volume
  - Increased sputum purulence
  - Hypoxemia, tachypnea, cyanosis, agitation, tachycardia, hypertension, add mm use, pursed-lip exhalation, “sitting up leaning forward” posture
  - Hypercapnea may result in confusion, tremor, decreased LOC
  - Respiratory failure
DDX AECOPD:
- Pneumonia
- IHD
- CHF
- Asthma
- PE
- Pneumothorax
- Etc.

Diagnostic Tests
- **CXR:**
  - Almost always abnormal, comparisons with prior exams should be made
  - Helpful in the diagnosis of complications such as pneumothorax, pneumonia, pleural effusions, pulmonary neoplasia

Management: AECOPD
- **Goals of therapy:**
  - Relieve bronchoconstriction
  - Improve oxygenation
- **Approach to treatment:**
  - Multi-modal
  - Be cognizant of previous disease pattern

Management: AECOPD
- **Oxygen:**
  - Target SaO$_2$ > 90%
  - Be aware that patients known to be CO2-retainers may require controlled oxygen therapy (SaO$_2$ 88-91%)
Management: AECOPD

**β₂-agonists:**
- COPD patients tend to have some reversibility to their airflow obstruction that can effectively be relieved by inhaled short acting β₂-agonist therapy
- Long acting β₂-agonist therapy should be reserved for chronic management only
- No evidence that one specific agent has any greater efficacy than any other
- Little evidence regarding timing of administration (q60 min vs. q20 min etc.)

Management: AECOPD

**Anticholinergics:**
- Slower onset of action than β₂-agonists
- Thought to inhibit vagal stimulation of the bronchi → promoting smooth muscle relaxat
- Most common agent is ipratropium bromide (Atrovent® q4-6h by neb or MDI)

Management: AECOPD

**Corticosteroids:**
- Conflicting results in the literature
- In acute exacerbation, there is likely a role for systemic steroids, but not for inhaled
- Steroid response is more likely a continuum than an “all or none” phenomenon

Management: AECOPD

**Magnesium:**
- Studied primarily in asthma
- One study showed benefit in COPD, used as 1-2g IV over 20 min

**Heliox:**
- Same issues as in asthma
Antibiotics in AECOPD

Current best evidence is that patients presenting with infectious symptoms:
- Fever
- Increased sputum production
- Change in character of sputum
Will have a better outcome with the use of empiric antibiotic therapy

Antibiotics in AECOPD

Current CHR recommendations:
- ≤4 exacerbations/year:
  - Amoxicillin 500mg po tid x 7-10d
  - Doxycycline 200mg po x 1d then 100mg po od x 7-10d
  - TMP/SMX 1 DS tablet po bid x 7-10d

Antibiotics in AECOPD

CHR recommendations:
- ≥4 exacerbations per year or failure of first line agent or Abx last 6 weeks:
  - Cefuroxime axetil 250-500mg bid x 7-10d
  - Amoxicillin-clavulanate 875mg po bid x 7-10d
- For PCN allergic patients:
  - Azithromycin 500mg x 1d then 250mg po od x 4d
  - Clarithromycin 250-500mg po bid x 7-10d
  - Levofloxacin 500mg po od x 5-10d
  - Moxifloxacin 400mg po od x 5-10d

Mechanical Ventilation

Decision to begin assisted ventilation is a clinical one
Noninvasive ventilation (BiPAP):
- BiPAP works by providing bilevel positive airway pressure.
- Response is usually seen within the first 20 minutes if it’s going to work
Mechanical Ventilation

Selection criteria for NPPV (any two):
- Moderate to severe dyspnea with use of accessory muscles & paradoxical abdominal motion
- Moderate to severe acidosis (pH 7.3-7.35) and hypercapnia (PaCO2 45-60)
- Respiratory frequency > 25 breaths/min

Exclusion Criteria for NPPV (any one):
- Respiratory arrest
- Cardiovascular instability (hypotension, dysrhythmias, AMI)
- Somnolence, impaired mental status, uncooperative patient
- High risk of aspiration
- Viscous or copious secretions
- Recent facial or gastroesophageal surgery
- Craniofacial trauma with fixed nasopharyngeal abnormalities
- Extreme obesity

Benefits of NIV
- Preserves pt’s ability to speak, eat, & cough
- Less invasive nature:
  - ↓‘s need for sedation
  - no airway trauma
  - ↓‘s risk of nosocomial pneumonia
- Increases FRC & helps recruit underventilated or collapsed alveoli
- Decreased work of breathing

Continuous Positive Airway Pressure (CPAP)
- Positive pressure 5-15 cm H₂O applied throughout respiratory cycle
- Reduces
  - Right ventricular preload
  - Left ventricular wall tension
- Improves:
  - Functional residual capacity
  - Lung compliance
  - Opens underventilated & collapsed alveoli
BiLevel Ventilation (BiPAP)
- Provides different levels of pressure during inspiration & exhalation
- Decreases
  - Neuromuscular drive
  - Inspiratory muscle effort
  - Work of breathing
- Increases alveolar ventilation
- IPAP usually 4-10 cm H₂O, EPAP 12-20 cm H₂O

Summary - NIV
- NIV is beneficial in COPD exacerbations:
  - Beginning to be considered standard of care
  - Reduces mortality, intubation rate, length of hospital stay
  - Less severe exacerbations show less benefit
  - More severe exacerbations show more benefit
  - Reasonable to consider a trial in all patients presenting with exacerbation of COPD who do not have contraindications

Clinical Benefits
- Decreased risk of pneumonia as compared with ETMV
  - Decrease in biofilms
  - Decrease in oropharyngeal secretions around tube
- Decreased risk of mechanical complications (e.g. vocal cord damage, tracheal stenosis)
- Decreased use of medications (sedative agents, analgesics, & neuromuscular blocking agents)
- Decreased use of invasive devices (central venous catheters, nasogastric & tracheostomy tubes)
- Able to be rapidly stopped & started
- Preserve the ability to eat, speak, & think

Reasons for Failure of Conservative Management
- GCS <13 at admission
- pH < 7.35 after 1 hour of NIV
- Resp rate >20 bpm after 1 hour of NIV
Indications for Invasive Mechanical Ventilation in AECOPD

- Severe dyspnea with accessory muscle use & paradoxical abdominal motion
- RR>35
- Life-threatening hypoxemia (PaO2<40)
- Severe acidosis (pH < 7.25) and hypercapnea (PaCO2 > 60)
- Respiratory arrest
- Somnolence or impaired mental status
- Cardiovascular complications
- Other complications (sepsis, pneumonia, PE...)
- Failure of NPPV

Reasons for Intubation

Intubate if:
- Respiratory or cardiac arrest
- Hemodynamic instability
- Life threatening arrhythmia
- Inability to clear secretions
- Face mask intolerance
- Worsening of level of consciousness
- Progressive worsening of dyspnea
- Worsening of ABG values

Intubation Technique

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References

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