Chapter 74 – Chronic Obstructive Pulmonary Disorder (COPD)

Episode Overview

1. Define acute exacerbation
2. Describe GOLD classification for COPD
3. List factors of decompensation or triggers of an AECOPD
4. Name 4 mimics for AECOPD
5. What are the clinical features used to diagnose AECOPD?
6. Describe the ED management of AECOPD.
7. What does the end tidal tracing look like in COPD?
8. List indications and contraindications to NIPPV in COPD
9. Which patients with AECOPD should be treated with antibiotics?
10. Which patients with AECOPD require admission?
11. List indications for intubation for AECOPD

Wise Cracks

1) List 4 CXR and 3 ECG findings in COPD

Rosens in Perspective

Pathophysiology of COPD

According to Global Initiative for Chronic Obstructive Lung Disease (GOLD), COPD is:

“a preventable and treatable disease with some significant extra-pulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible…the airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.”

This used to include elements of reversible airflow limitation (i.e. asthma) and chronic bronchitis (increased sputum for x 3 months for last 2 consecutive years, +/- airflow limitation)

Don't make the rookie mistake of mixing up COPD and emphysema: The latter is a destructive process. Classically centrilobular emphysema is a component of COPD. Alternatively, severe panacinar emphysema is associated with α1-antitrypsin deficiency, an enzyme that inhibits neutrophil elastase (ie stops your neutrophils from melting your lung parenchyma) (Big shout out to all my friends with this disease, you know who you are!).

Elements of COPD: Comes down to Airway obstruction and Airway obliteration

1. Chronic inflammation from trachea down to alveolar.
   a. Neutrophils/CD8+/Macrophages/Lymphocytes
   b. Differs from ASTHMA which is primarily eosinophils
2. Mucous plugging
a. Increase in goblet cell proliferation and size, increase mucous production and plugging

3. Endothelial barrier damage
   a. Mucociliary response inhibited

4. Centrilobar emphysema
   a. Loss of connective tissue and subsequent airway patency/stenting through expiration via radial support (OBLITERATION)

- In the end patients end up with Type 1 & Type 2 respiratory failure (Hypoxemia <<< Hypercapnia)
- DECREASE in pulmonary vascular bed w/ chronic hypoxia = thickening of the vessel walls.
- Net result = pulmonary hypertension, polycythemia: right- sided heart failure (cor pulmonale)

[1] Define acute exacerbation

According to GOLD:

“An event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough, and/or sputum that is beyond normal day-to-day variations, is acute in onset, and may warrant change in regular medication in a patient with underlying COPD”

Remember viral induced tend to be protracted course. Common viruses include rhinovirus, respiratory syncytial virus, coronavirus, and influenza virus

Bacterial pathogens: controversial of acute versus chronic versus acute on chronic. Bacteria to consider include: H. influenzae; M. catarrhalis; S. pneumoniae; P. aeruginosa.

[2] Describe GOLD classification for COPD

<table>
<thead>
<tr>
<th>STAGE</th>
<th>CHARACTERISTICS</th>
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<tbody>
<tr>
<td>I: Mild COPD</td>
<td>FEV1/FVC &lt; 70% &lt;br&gt;FEV1 ≥ 80% of predicted &lt;br&gt;Symptoms may or may not be present</td>
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<tr>
<td>II: Moderate COPD</td>
<td>FEV1/FVC &lt; 70% &lt;br&gt;50% ≤ FEV1 &lt; 80% predicted &lt;br&gt;Usually symptomatic with SOB on exertion or acute exacerbations or both</td>
</tr>
<tr>
<td>III: Severe COPD</td>
<td>FEV1/FVC &lt; 70% &lt;br&gt;30% ≤ FEV1 &lt; 50% predicted &lt;br&gt;Increasingly symptomatic with frequent exacerbations and deleterious effects on quality of life</td>
</tr>
<tr>
<td>IV: Very severe COPD</td>
<td>FEV1/FVC &lt; 70% &lt;br&gt;FEV1 &lt; 30% of predicted or FEV1 &lt; 50% with chronic respiratory failure (Pao2 &lt; 60 mm Hg ± Paco2 &gt; 50 mm Hg) &lt;br&gt;May or may not have clinical signs of right-sided heart failure</td>
</tr>
</tbody>
</table>

FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; Pao2, arterial partial pressure of carbon dioxide; Paco2, arterial partial pressure of oxygen; SOB, shortness of breath.
[3] List factors of decompensation or triggers of an AECOPD

### Causes of Acute Decompensation in the Patient with Chronic Obstructive Pulmonary Disease

<table>
<thead>
<tr>
<th>Acute Exacerbations</th>
<th>Infectious</th>
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<tbody>
<tr>
<td>1. Viral</td>
<td>Rhinovirus, respiratory syncytial virus, coronavirus, influenza virus</td>
</tr>
<tr>
<td>2. Bacterial</td>
<td><em>Haemophilus influenzae, Streptococcus pneumoniae,</em>  <em>Moraxella (Branhamella) catarrhalis, Pseudomonas aeruginosa</em></td>
</tr>
<tr>
<td>3. Atypical bacteria</td>
<td><em>Chlamydia pneumoniae, Legionella</em></td>
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<table>
<thead>
<tr>
<th>Air Pollution</th>
<th>Surgical and chemical events</th>
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<tbody>
<tr>
<td>1. Nitrogen dioxide</td>
<td></td>
</tr>
<tr>
<td>2. Ozone</td>
<td></td>
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<tr>
<td>3. Particulates, dust</td>
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</table>

<table>
<thead>
<tr>
<th>Other Critical Events</th>
<th>Other causes of acute exacerbations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pneumothorax</td>
<td>Congestive heart failure, lobar atelectasis</td>
</tr>
<tr>
<td>2. Pulmonary embolism</td>
<td>Acute exacerbations in patients with respiratory disease</td>
</tr>
<tr>
<td>3. Lobar atelectasis</td>
<td>Noncompliance with prescribed treatment regimen</td>
</tr>
<tr>
<td>4. Congestive heart failure</td>
<td>Unrelated treatable chronic pulmonary disease</td>
</tr>
<tr>
<td>5. Pneumonia</td>
<td>Dysrhythmias</td>
</tr>
<tr>
<td>6. Pulmonary compression (e.g., obesity, ascites, gastric distention, pleural effusion)</td>
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<tr>
<td>7. Trauma (e.g., rib fractures, pulmonary contusion)</td>
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<tr>
<td>8. Neuromuscular and metabolic disorders</td>
<td></td>
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<tr>
<td>9. Unrelated treatable chronic pulmonary disease (bronchiectasis, tuberculosis, sarcoidosis)</td>
<td></td>
</tr>
<tr>
<td>10. Noncompliance with prescribed treatment regimen</td>
<td></td>
</tr>
<tr>
<td>11. Iatrogenic</td>
<td>a. Inadequate therapy</td>
</tr>
<tr>
<td></td>
<td>b. Inappropriate therapy (e.g., deleterious drugs)</td>
</tr>
</tbody>
</table>

[4] Name 4 mimics for AECOPD

- Pneumonia
- Congestive heart failure (CHF)
- Pneumothorax
- Pulmonary embolism (PE)
- Lobar atelectasis (plugging / mass)
- Pleural effusion
- Dysrhythmias

[5] What are the clinical features used to diagnose AECOPD?

The old school description of blue bloater (polycythemia, core pulmonale from chronic obstructive bronchitis) and pink puffer (crazy V/Q mismatch from emphysema leading to increased RR to compensate minute ventilation) have gone the way of the dodo bird. But they still have some utility for pattern recognition.
On History and Physical, look for:

**History**
- Cough with/without expectoration
- Increased WOB or air hunger

**Physical**
- Wheeze
- ALOC “Irritable Somnolence” (hypercapnea: often has asterixis with it)
- Right heart failure: JVD and peripheral edema

[6] Describe the ED management of AECOPD

Remember: Beta agonists, anticholinergics and corticosteroids are our workhorses for AECOPD.

![Table 74-2 General Therapeutic Guidelines for Chronic Obstructive Pulmonary Disease Exacerbations](image)

<table>
<thead>
<tr>
<th>LIFE-THREATENING</th>
<th>MODERATE OR SEVERE</th>
<th>MILD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Address ABCs</td>
<td>Oxygen to maintain $O_2$ saturation near 90%</td>
<td>Oxygen to maintain $O_2$ saturation near 90%</td>
</tr>
<tr>
<td>Bag-valve ventilation, preoxygenation</td>
<td>Nebulized beta-agonist, anticholinergic</td>
<td>MDI or nebulized beta-agonist, anticholinergic</td>
</tr>
<tr>
<td>Intubation with or without rapid sequence technique</td>
<td>Noninvasive ventilation if severe</td>
<td>Consider oral or intravenous corticosteroid</td>
</tr>
<tr>
<td>In-line beta-agonist, anticholinergic</td>
<td>Intravenous corticosteroid</td>
<td>Consider oral antibiotic on discharge</td>
</tr>
<tr>
<td>Intravenous corticosteroid</td>
<td>Intravenous antibiotic</td>
<td></td>
</tr>
</tbody>
</table>

Little or no evidence:
- Nebulized saline
- Chest physiotherapy
- Heliox
- Oral expectorants

[7] What does the end tidal tracing look like in COPD?

Remember Lipp’s top hat description for capnometry? Well instead of Abe Lincoln, we have Jaws. Think shark tooth for airway obstruction:
[8] List indications and contraindications to NIPPV in COPD

![Capnography tracing](image)

**Figure 74-2.** Capnography tracing in bronchospasm and obstruction. The upslope at the beginning of the expiratory phase is blunted because of expiratory airflow limitation.

<table>
<thead>
<tr>
<th>Table 74-3</th>
<th>Suggested Selection and Exclusion Criteria for the Use of Noninvasive Ventilatory Support</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SELECTION CRITERIA (ONE OR MORE MAY BE PRESENT)</strong></td>
<td><strong>EXCLUSION CRITERIA (ANY MAY BE PRESENT)</strong></td>
</tr>
<tr>
<td>Moderate to severe dyspnea with use of accessory muscles and paradoxical abdominal motion</td>
<td>Respiratory arrest</td>
</tr>
<tr>
<td>Respiratory rate 25 breaths/min</td>
<td>Cardiovascular instability</td>
</tr>
<tr>
<td>Moderate to severe acidosis (pH &lt; 7.35) and hypercapnia (Paco₂ &gt; 45 mm Hg)</td>
<td>Uncooperative patient (agitated or severely somnolent)</td>
</tr>
</tbody>
</table>


\( \text{Paco}_2 \), arterial partial pressure of carbon dioxide.

[9] Which patients with AECOPD should be treated with antibiotics?

i) Any patient who gets snorkelled: all intubated and NIPPV patients

ii) Increased sputum purulence with:

   A) Increased dyspnea, OR
   B) Increased sputum volume
**Note** Patients with clinical pneumonia without radiographic evidence may benefit as well.

**Double Note:** Drug selection should target local sensitivities to:
- S. pneumoniae,
- H. influenzae,
- M. Catarrhalis.

Five days course of respiratory fluoroquinolones > 7-10 days with beta lactams and tetracycline

[10] Which patients with AECOPD require admission?

**Box 74-3** General Guidelines for Admission of the Patient with Chronic Obstructive Pulmonary Disease

- Significant worsening of symptoms from baseline
- Inadequate response of symptoms to emergency department management
- Significant comorbid condition (e.g., pneumonia, heart failure)
- Worsening hypoxia or hypercapnia (from baseline)
- Inability to cope at home or insufficient home resources


[11] List indications for intubation for AECOPD

**Box 74-2** Proposed Indications for Mechanical Ventilation

- Respiratory arrest
- Worsening level of consciousness despite maximal therapy*
- Cardiovascular instability (shock, heart failure)*
- NIPPV failure or exclusion criteria (see Table 74-3)
- Severe dyspnea with use of accessory muscles and paradoxical abdominal motion*
- Severe tachypnea*
- Life-threatening hypoxia
- Severe acidosis and hypercapnia*
- Other complications (metabolic abnormalities, sepsis, pneumonia, pulmonary embolism, barotraumas, massive pleural effusion)*


*NIPPV, nasal intermittent positive-pressure ventilation.
*For several of these parameters, criteria are deliberately imprecise; clinical decisions must be individualized in each case.
[1] List 4 CXR and 3 ECG findings in COPD

CXR:
- Hyperinflated lungs
- Decreased vascular markings
- Small cardiac silhouette OR late stage cardiomegaly
- Increased vascular markings
- Bullae may be many or may be large, mimicking pneumothorax.

ECG:
- P pulmonale: Peaked P waves in leads II, III, and aVF
- Low QRS voltage (hyperinflated chest)
- Clockwise rotation & poor R wave progression in the precordial lead (classically described from RV hypertrophy or dilatation, but this is non specific)
- Tachydysrythmias: AFIB/Multifocal atrial tachy