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.*

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[2] Describe GOLD classification for COPD

Table 74-1 The GOLD Classification of Severity of Chronic Obstructive Pulmonary Disease (COPD)

STAGE	CHARACTERISTICS
I: Mild COPD	$FEV_1/FVC < 70\%$ $FEV_1 \geq 80\%$ of predicted Symptoms may or may not be present
II: Moderate COPD	$FEV_1/FVC < 70\%$ $50\% \leq FEV_1 < 80\%$ predicted Usually symptomatic with SOB on exertion or acute exacerbations or both
III: Severe COPD	$FEV_1/FVC < 70\%$ $30\% \leq FEV_1 < 50\%$ predicted Increasingly symptomatic with frequent exacerbations and deleterious effects on quality of life
IV: Very severe COPD	$FEV_1/FVC < 70\%$ $FEV_1 < 30\%$ of predicted or $FEV_1 < 50\%$ with chronic respiratory failure ($Pao_2 <$ 60 mm Hg ± $Paco_2 >$ 50 mm Hg) May or may not have clinical signs of right-sided heart failure

Adapted from Rabe K, Hurd S, Anzueto A, et al: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med 176:532, 2007.
FEV_1, forced expiratory volume in 1 second; *FVC*, forced vital capacity; *$Paco_2$*, arterial partial pressure of carbon dioxide; *Pao_2*, arterial partial pressure of oxygen; *SOB*, shortness of breath.

[3] List factors of decompensation or triggers of an AECOPD

Box 74-1 Causes of Acute Decompensation in the Patient with Chronic Obstructive Pulmonary Disease

Acute Exacerbations

Infectious

1. Viral
   Rhinovirus, respiratory syncytial virus, coronavirus, influenza virus
2. Bacterial
   Haemophilus influenzae, Streptococcus pneumoniae, Moraxella (Branhamella) catarrhalis, Pseudomonas aeruginosa
3. Atypical bacteria
   Chlamydia pneumoniae, Legionella

Air Pollution

1. Nitrogen dioxide
2. Ozone
3. Particulates, dust

Other Critical Events

1. Pneumothorax
2. Pulmonary embolism
3. Lobar atelectasis
4. Congestive heart failure
5. Pneumonia
6. Pulmonary compression (e.g., obesity, ascites, gastric distention, pleural effusion)
7. Trauma (e.g., rib fractures, pulmonary contusion)
8. Neuromuscular and metabolic disorders
9. Unrelated treatable chronic pulmonary disease (bronchiectasis, tuberculosis, sarcoidosis)
10. Noncompliance with prescribed treatment regimens
11. Iatrogenic
    a. Inadequate therapy
    b. Inappropriate therapy (e.g., deleterious drugs)

[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

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

ABC, airway, breathing, and circulation; *MDI*, metered dose inhaler.
It is important to consider an inciting or aggravating factor and provide specific therapy as discussed in text.

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:

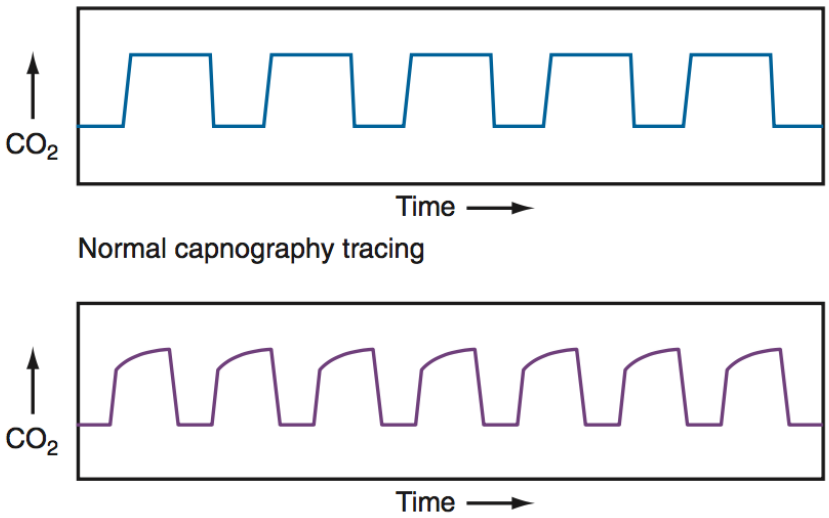

Figure 74-2. Capnography tracing in bronchospasm and obstruction. The upslope at the beginning of the expiratory phase is blunted because of expiratory airflow limitation.

[8] List indications and contraindications to NIPPV in COPD

Table 74-3 Suggested Selection and Exclusion Criteria for the Use of Noninvasive Ventilatory Support

SELECTION CRITERIA (ONE OR MORE MAY BE PRESENT)	EXCLUSION CRITERIA (ANY MAY BE PRESENT)
Moderate to severe dyspnea with use of accessory muscles and paradoxical abdominal motion	Respiratory arrest
Respiratory rate 25 breaths/min	Cardiovascular instability
Moderate to severe acidosis (pH < 7.35) and hypercapnia ($Paco_2$ > 45 mm Hg)	Uncooperative patient (agitated or severely somnolent)
	Upper airway obstruction
	High aspiration risk
	Recent facial or gastroesophageal surgery
	Craniofacial trauma, fixed nasopharyngeal abnormalities
	Nonfitting mask

Adapted from Rabe K, Hurd S, Anzueto A, et al: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med 176:532, 2007; and Soto F, Varkey B: Evidence-based approach to acute exacerbations of COPD. Curr Opin Pulm Med 9:117, 2003.

$Paco_2$, 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 hypercarbia (from baseline)
Inability to cope at home or insufficient home resources

Adapted from Rabe K, Hurd S, Anzueto A, et al: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med 176:532, 2007.

[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)*

Adapted from Rabe K, Hurd S, Anzueto A, et al: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med 176:532, 2007.
NIPPV, nasal intermittent positive-pressure ventilation.
*For several of these parameters, criteria are deliberately imprecise; clinical decisions must be individualized in each case.

Wise Cracks

[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