Acute Exacerbations of COPD

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Introduction

• Definitions
  – COPD
  – Exacerbations
• Causes of Exacerbations
• Assessment of Severity
• Management
  – Acute
  – Ongoing
Chronic Obstructive Pulmonary Disease

- ‘Response to inhaled toxins in susceptible individuals – cigarette smoke’

- <2% COPD in non-smokers

- Orthodoxy – 10-15% smokers get COPD
  - Recently challenged – 40-50%
Chronic Obstructive Pulmonary Disease

• Title of convenience
  – Chronic – progressive and relentless increase in airflow Obstruction affecting all Pulmonary compartments

• Multi-system disease
  – Osteoporosis, ‘sarcopaenia’, cachexia, systemic inflammation, ischaemic heart disease
Proportion of 1965 Rate

Coronary Heart Disease: -59%
Stroke: -64%
Other CVD: -35%
COPD: +163%
All Other Causes: -7%

Source: NHLBI/NIH/DHHS
Chronic Obstructive Pulmonary Disease

• **Inflammatory disease**
  – CD8 +ve T lymphocyte, neutrophilic inflammation in airways and tissues
  – Release of tissue-destructive enzymes, reduced inhibitors

• **Colonisation of normally sterile lower airways with bacteria**
  – Haemophilus Influenzae
Acute Exacerbation

• Contentious
  – Clinical trial versus clinical experience

• Acute change from the stable condition in a patient with known COPD
  – Symptomatic
  – Physiological (unhelpful unless recorded)
Acute Exacerbation

• **Symptoms**
  – Dyspnoea
  – Exercise Tolerance
  – Cough 86% (64%)
  – Sputum 84%
    • Volume (51%)
    • Purulence (41%)
  – Chest Pain 28%
Acute Exacerbation: Inflammation

• **Systemic**
  – CRP increased in 86%, IL-6, IL-1

• **Airways**
  – Cells, cytokines
Causes of Acute Exacerbation of COPD: Infection

• Infection
  – Viral up to 60%
  – Bacterial up to 70%
  – Co-infective common ? Synergistic

• Sero-shift in colonising bacteria
  – 50% high bacterial load during exacerbation
Causes of Acute Exacerbation of COPD: Pollution

- May increase or worsen infection
- Especially in regions of high fossil fuel use
- Poor air quality may predict exacerbation in a given patient
Causes of Acute Exacerbation of COPD

• 30% unknown
Immediate management

Clinical assessment

1. Oxygenation

2. Life threatening
   - Airway, Breathing, Circulation
     * Risk of dying $\propto$ acidosis, co-morbidity, need for ventilation

• Immediate management
  - Supplemental oxygen
Immediate assessment

- Admission
  - Medical ward
  - ITU
- Supported Discharge
- Alternative / co-morbid diagnoses
Clinical assessment

• History –
  – Confirm COPD
  – Normal functional status
  – Co-morbid conditions
  – ? Symptom deterioration

• Examination
  – Mental status, Respiratory, Cardiovascular
Admission

- ICU – severe acute exacerbation
  - Mental status – GCS
  - Persistent low $P_aO_2$
  - Mechanical ventilation
  - Haemodynamic status
- Pre-morbid details important
  - Functional status
  - Oxygenation
Investigation

• Pulmonary assessment
  – Arterial Blood Gases
    • Helpful if old gases available
  – Chest X-ray/ ECG
  – Peak flow
    • Little practical use in acute setting

• Inflammation/ Infection
  – Full Blood Count
  – CRP
Management 1

• **Oxygen**
  – Maintain Sat O$_2$ 90 – 94%
  – Continuous Sat O$_2$ monitoring
  – Be aware of hypercapnia

• **Corticosteroids**
  – Oral / Intravenous
  – 7 – 10 days 30 – 40mg prednisolone
Management: Bronchodilators

• **Increase from normal use**
  – eg. via nebuliser
  – $\beta_2$ agonists
  – Anti-cholinergics

• **If poor initial response**
  – Consider intravenous +/- aminophylline
  – Significant adverse event profile
Management: Antibiotics

• **Antibiotics if:**
  - Increase in dyspnoea +
    - Increase in volume and purulence of sputum
  - Any change in presence of change in purulence

• **Empirical:**
  - Strep. Pneumoniae, Haemophillus Influenzae, Moraxella catharallis
  - Recent hospital admission
Management: Co-morbidities

- **Related conditions:**
  - Pneumothorax – chest drainage
  - Cor-pulmonale – diuresis
  - Pneumonia
Management

• Failure to respond/ deterioration
• Consider alternative diagnoses
  – Heart Failure
    • Wheeze, JVP, ankle oedema
  – PTE
    • Persistent hypoxia, hypotension
• Increase treatment intensity
  – eg. Intravenous administration
  – Mechanical ventilation
Mechanical ventilation: non-invasive

• Failure to respond
  – Always use bronchodilators

• Evidence of hypercapnia or acidosis
  – Not best treatment for type I respiratory failure

• Exclusions – dangers of tight fitting mask
Ongoing management

- Plan discharge early – often delayed
- Monitor exercise capacity / spirometry / reliever inhaler use
- Consider supported discharge
  - Loan of a nebuliser
- 4 -6 week follow up
• Acute exacerbations are the point of greatest impact of health care in COPD

• Assess and record severity at admission

• Be alert for co-morbidities
Summary 2

• Cautious use of supplemental oxygen

• Corticosteroids + bronchodilators are mainstay

• Antibiotics - controversial
Summary 3

• Escalate treatment intensity with as much involvement of the patient as possible:
  – Supported ventilation
  – ICU management