Project: Ghana Emergency Medicine Collaborative

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COPD
in the emergency department

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BACKGROUND

epidemiology

- one of the top causes of death worldwide
- 7th most common cause of disability by 2030
- billions of dollars per year in treatment and lost productivity
- under-reported (only 50% see a physician for an exacerbation)
- 2% of all hospitalizations, 20% >65 years

pathophysiology

- chronic airway inflammation as in asthma
  - asthma: eosinophils
  - COPD: neutrophils, CD8+ lymphocytes, & macrophages
- lung parenchyma damaged by TNF, leukotriene B4, & interleukin 8
  - hence, poorer response to anti-inflammatory treatments than asthma
- COPD = chronic bronchitis + emphysema

chronic bronchitis

- progressive scarring and narrowing of airways → obstruction
- increase in globlet cells → mucus plugs
- epithelial damage → mucociliary impairment → decreased clearing of bacteria and mucus

emphysema

- destruction of alveoli
- loss of elasticity
- collapse of small airways
- chronic air trapping/hyperinflation
- prolonged expiratory phase
  - decreased FEV1 (forced expiratory volume in 1 second)

natural course

- chronic bronchitis + emphysema → decreased size of pulmonary vascular bed → capacity for gas exchange
- chronic hypercapnia and hypoxemia result
- lung vessels thicken, hemoglobin increases → increased pulmonary vascular resistance → pulmonary hypertension → R-sided heart failure

etiology

- smoking, pollution (incl indoor cooking), occupational exposures, genetic factors
- negatively affect body's natural oxidant/ antioxidant and protease/antiprotease balances
- secondary effects:
  - weight loss, muscular wasting, metabolic derangement, depression etc.

staging

- stage I – mild
  - FEV1 > 80% predicted, little to no symptoms
- stage II – moderate
  - FEV1 50-80%, shortness of breath on exertion, occasional exacerbations
- stage III – severe
  - FEV1 30-50%, shortness of breath at rest, frequent exacerbations
- stage IV – very severe
  - FEV1<30%, ↓pO2,↑pCO2, cor pulmonale

CLINICAL

physical exam

- chronic bronchitis - “blue bloater”
  - chronic respiratory failure, cor pulmonale, polycythemia, hypoxia
  - cyanosis, facial plethora, edema, JVD, frequent cough
- emphysema - “pink puffer”
  - thin, anxious, dyspneic, barrel chested, uses accessory muscles, pursed lip exhalation (auto-PEEP)
- most patients exhibit elements of both

exacerbation

- acute and unusual change in baseline dyspnea, cough, or sputum production
- warrants change in baseline medication
- more common in winter
  - suggests relation to seasonal viruses (RSV, coronavirus, influenza, rhinovirus)
- bacteria isolated in 50% of exacerbations
  - also isolated in a similar proportion of COPD patients without exacerbation

comorbidities

- patients with COPD are also at much higher risk for:
  - CAD/acute coronary syndrome (smoking)
  - pulmonary embolism (sedentary)
  - pneumonia (decreased ciliary function)
  - congestive heart failure (R-sided)
  - pneumothorax (ruptured bullae)
  - arrhythmias (atrial tachy)
- be careful to avoid premature closure
  - “just a COPD exacerbation”

diagnostic approach

- pulse oximetry – compare to baseline
- ABG – limited utility, mgt guided by exam
  - acute resp acidosis, compensatory metabolic alkalosis – values don't predict outcome
- ECG – ACS, arrhythmia
- CBC – limited value (↑WBC, ↑Hb)
- BNP – may help rule out CHF
- d-dimer – may help rule out PE

imaging

- CXR – atelectasis, PNA, CHF, pneumothorax, bullae, pericardial/pleural effusions, pulmonary fibrosis, etc.
- US – CHF, PE, pericardial effusion, pneumothorax, pleural effusion
  - careful with interpretation of large RV
- CT – definitive diagnosis of PE, bullae vs. pneumothorax, atx vs. PNA, etc. in unclear clinical situations

MANAGEMENT

long term

- chronic inhaled steroids
- albuterol as needed
- influenza and pneumonia vaccines
- home oxygen when necessary
- smoking cessation

case

- 63 yo M with a history of COPD p/w shortness of breath, cough
- started out as a cold with worsening over 3 days
- increasingly productive cough
- more frequent albuterol use
- VS: P104, BP166/94, R26, T37.4, 89% RA
  - appears anxious, dyspneic
  - poor air movement on auscultation

acute exacerbation

- A – airway
  - intubation if obtunded, severe respiratory distress, or clinically tiring out (RSI vs. nasal)
- B – breathing
  - inhaled albuterol/ipatropium
  - non-invasive positive pressure ventilation
  - oxygen
- C – circulate medications
  - antibiotics
  - corticosteroids

airway

- indications for intubation
  - severe respiratory distress
  - agitation, obtundation
  - fails NIPPV
  - shock
  - respiratory arrest
- methods of intubation
  - RSI – takes away respiratory drive, familiar
  - nasal – preserves ventilation, less familiar

breathing

- oxygen (as little as possible for sat ~90%)
  - monitor respiratory rate, intervene if slow
- albuterol 2.5 mg neb, titrate to effect
  - beta 2 agonist → bronchodilation
- ipatropium 0.5 mg neb, repeat x3
  - anticonlinergic → inhibits smooth muscle contraction, decreases secretions
- non-invasive positive pressure ventilation
  - provides extrinsic PEEP to ↓work of breathing

circulate medications

- corticosteroids
  - 60 mg prednisone PO x1, 40 mg daily x4
  - 125 mg solumedrol IV x1
- antibiotics
  - objective evidence of pneumonia
  - increased sputum production
  - critically ill patients (ventilated)

disposition

- significant worsening from baseline
- poor response to ED treatment
- significant co-morbid diseases
- hypoxia
- unable to care for self
  - can they get around their house?
  - can they keep down fluids?
  - who will call for help if they get worse?

SUMMARY

in conclusion…

- it's not always “just” a COPD exacerbation
- treat with beta agonists, anticholinergics, and steroids
- try NIPPV before intubating
- re-evaluate frequently
- make then prove to you that they can go home

QUESTIONS