Acute COPD

Initial management

- COPD is characterized by chronic airflow limitation due to impedance to expiratory airflow, mucosal oedema, infection, bronchospasm, and bronchoconstriction due to low lung elasticity.

- Smoking is the main cause, but other causes are chronic asthma, α-1 antitrypsin deficiency, and chronic infection (eg bronchiectasis)

History

Exertional dyspnoea, cough, and sputum are usual complaints.

Ask about:

- *Present treatment*: including inhalers, steroids, antibiotics, theophyllines,

nebulizers, opiate analgesia, and home O2

treatment.

- *Past history*: enquire about previous admissions and comorbidity.
- *Exercise tolerance*: how far can they walk on the flflat without stopping?

How many stairs can they climb? Do they get out of the house?

History

- *Recent history*: ask about wheeze and dyspnoea, and sputum volume and

colour. Chest injuries, abdominal problems, and other infections may

cause respiratory decompensation.

- *Read the hospital notes*: have there been prior ICU assessments? Has the

respiratory consultant advised whether ICU would be appropriate?

Examination

- Examine for dyspnoea, tachypnoea, accessory muscle use, and lip-pursing.
- Look for hyperinflflation (‘barrel chest’), and listen for wheeze or coarse crackles (large airway secretions).
- Cyanosis, plethora (due to secondary polycythaemia), and right heart failure (cor pulmonale) suggest advanced disease.
- Look for evidence of hypercarbia: tremor, bounding pulses, peripheral vasodilatation, drowsiness, or confusion.

Investigations

- SpO2 , RR, pulse rate, BP, T°, and peak flow (if possible).

- CXR (look for pneumothorax, hyperinflation, bullae, and pneumonia).

- ECG.

Investigations

- ABG (or VBG), documenting the FiO2 . Use pCO2 to guide O2 therapy.

- FBC, U&E, glucose, theophylline levels, and, if pneumonia is suspected

and/or pyrexial, blood cultures, CRP, and pneumococcal antigen.

- Send sputum for microscopy and culture if purulent.

Treatment

Give oxygen Remember that hypercapnia with O2 is multifactorial.

The aim is to maintain SpO2 of 88–92% without precipitating respiratory acidosis or worsening hypercapnia.

If the patient is known to have COPD and is drowsy or has a documented history of previous hypercapnic respiratory failure, give an FiO2 of 28% via a Venturi mask and obtain an ABG. Titrate up the FiO2 with serial ABG sampling until the minimum FiO2 that achieves SpO2 of 88–92%.

Reduce inhaled O2 concentration if SpO2 is >92%.

Treatment

Give bronchodilators and steroids

- Give nebulized salbutamol 5mg or terbutaline 5–10mg.
- Consider adding nebulized ipratropium 0.5mg.
- Use O2-driven nebulizers unless the patient has hypercapnic, acidotic COPD, in which case use nebulizers driven by compressed air, supplemented by O2 via nasal prongs at 1–4L/min.

- Give steroids (eg prednisolone 30mg PO stat, then continued once daily for 7 days). Use hydrocortisone 100mg IV if the patient cannot take prednisolone PO.

Treatment

Other drug treatments

- Give antibiotics (eg amoxicillin, doxycycline, or clarithromycin) if the patient reports highly purulent sputum or there is clinical evidence of pneumonia and/or consolidation on CXR.
- Only consider IV aminophylline if there is an inadequate response to nebulized bronchodilators. Beware interactions with other drugs and the potential for toxicity if the patient is already taking oral theophylline.
- Consider naloxone if the patient is taking an opioid analgesic that may cause respiratory depression.

Treatment

Non-invasive ventilation

NIV is standard early therapy for hypercapnic ventilatory failure during exacerbations of COPD.

NIV improves blood gas measurements in the ED and lower intubation rates, mortality, and length of hospital stay. Ensure patients started on NIV have a plan in the event of deterioration.