IAEM Clinical Guideline

# Management of Acute Exacerbation of Chronic Obstructive Pulmonary Disease in the Emergency Department

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**DISCLAIMER**

IAEM recognises that patients, their situations, Emergency Departments and staff all vary. These guidelines cannot cover all clinical scenarios. The ultimate responsibility for the interpretation and application of these guidelines, the use of current information and a patient's overall care and wellbeing resides with the treating clinician.

| Revision History | Section | Summary of Changes | Author |
|---|---|---|---|
| V1.0 | All | Final version | OK/PN/BMacC/ SS/GMcM |

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## GLOSSARY OF TERMS

| | |
|---|---|
| ABG | Arterial Blood Gas |
| ACS | Acute Coronary Syndrome |
| AECOPD | Acute Exacerbation of Chronic Obstructive Pulmonary Disease |
| BTS | British Thoracic Society |
| CNS | Clinical Nurse Specialist |
| COPD | Chronic Obstructive Pulmonary Disease |
| $CO_2$ | Carbon Dioxide |
| CXR | Chest X-Ray |
| ED | Emergency Department |
| EPAP | Expiratory Positive Airway Pressure |
| $FiO_2$ | Fraction of Inspired oxygen |
| HSE | Health Service Executive |
| ICU | Intensive Care Unit |
| IPAP | Inspiratory Positive Airway Pressure |
| MDI | Metered-Dose Inhaler |
| NIBP | Non-Invasive Blood Pressure |
| NIV | Non-Invasive Ventilation |
| OECD | Organisation for Economic Cooperation and Development |
| $PaCO_2$ | Partial Pressure of Carbon Dioxide |
| PE | Pulmonary Embolism |
| POCUS | Point of Care Ultrasound |
| VBG | Venous Blood Gas |

# Management of Acute Exacerbations of Chronic Obstructive Pulmonary Disease in the Emergency Department

## INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is characterised by partially reversible and usually progressive airflow obstruction. It is an ambulatory care sensitive condition, i.e. early intervention and appropriate primary care can reduce complications and disease progression, ultimately avoiding hospital admission, which is naturally of benefit to both patients and the healthcare system.[1] Acute exacerbations of COPD (AECOPD) are discrete episodes characterised by acutely worsening symptoms of breathlessness, cough, or sputum production[2] and are important determinants of a patient's health status, as well as independent predictors of mortality.[3]

The mainstay treatment for AECOPD is $\beta_2$-agonists, steroids and antibiotics. Up to 80% of exacerbations can be treated on an outpatient basis.[4] Earlier recognition (or reporting) of exacerbations and prompt pharmacological treatment is associated with improved outcomes compared with delayed diagnosis and management.[5] The HSE acknowledges the importance of early intervention in their acute care bundle for COPD. It recommends time limits are applied to their listed interventions, with $\beta_2$-agonists to be administered in the first 30 minutes and steroids within the first 120 minutes.[9] Acute hypercapnic respiratory acidosis will develop in approximately 20% of patients attending the Emergency Department (ED) with AECOPD.[6] The use of non-invasive ventilation (NIV) has revolutionised care for these patients. Early intervention before severe acidosis, can reduce mortality, intubation rates, and failure of treatment.[7] British Thoracic Society (BTS) guidelines recommend commencing NIV within 60 minutes of a blood gas result that indicates the need for NIV, or within 120 minutes of arrival to hospital.[8]

A true incidence and prevalence rate of COPD in Ireland is not available, however it is estimated that there are between 380,000 – 500,000 people living with COPD in Ireland, with 270,000 of those yet to be diagnosed. Ireland has the highest age-sex standardised hospitalisation rate for COPD amongst Organisation for Economic Cooperation and Development (OECD) countries.[1, 9] Ireland also has a relatively high prevalence of hereditary alpha-1 antitrypsin deficiency, which affects 15,000 people, and another 250,000 carriers at risk of lung and liver disease.[10]

From an economic perspective, COPD is responsible for a significant financial burden, claiming 6% of the annual healthcare budget of the European Union, totalling €36.8 billion.[11] Data from adult acute hospitals reveals that patients with a primary diagnosis of COPD accounted for 4.1% of inpatient hospitalisations and 4.5% of bed days in 2016.[12] This burden of disease has not gone unnoticed by the HSE, and The National Clinical Programme for COPD published 'End to End COPD Model of Care' in December 2019.[9] This aims to provide an integrated model of care for COPD patients, ensuring they receive the right treatment, at the right time, in the right place.

The severity of the burden of this disease, on both the patient and healthcare system, mandates a standardised approach to management in the ED.

## PARAMETERS

| | |
|---|---|
| **Target audience** | Emergency Medicine clinicians and nurses working in adult or mixed Emergency Departments. |
| **Patient population** | Those with a confirmed diagnosis of COPD, or presumed diagnosis based on history and clinical examination presenting with all or some symptoms of increased breathlessness, cough and sputum production. |
| **Exclusion criteria** | Patients with asthma without a concomitant COPD component, or those with other diagnoses. |

## AIMS

To provide a standardised, structured, and evidence-based approach to the management of patients presented with AECOPD to Irish Emergency Departments.

## PRESENTATION

An AECOPD is defined as ‘an event characterised by dyspnoea and/or cough, and sputum that worsen over ≤14 days, which may be accompanied by tachypnoea and/or tachycardia and is often associated with increased local and systemic inflammation caused by airway infection, pollution, or other insults to the airways.[13]

As patients with COPD frequently have other co-morbidities and are at increased risk of cardiovascular events, other causes of such symptoms must be out ruled, including but not limited to pneumonia, pulmonary embolism (PE) and heart failure, which can confound and contribute to the presentation. By definition, if the severity of symptoms has necessitated an ED visit, and certainly an admission, it is classified as a severe exacerbation. An admission for a COPD exacerbation confers a poor long-term prognosis with a 5-year mortality rate of almost 50%.[14, 15]

## INVESTIGATIONS

All patients presenting with breathlessness should have all vitals checked at presentation. Accurate oxygen saturation measurement is paramount. A physical examination will reveal work of breathing and severity of presentation and will guide further investigations.

ECG, venous blood gas (VBG), laboratory blood tests, and CXR will all likely be necessary. D-dimer testing is not recommended as a routine test, however may be useful in acute breathlessness without signs of airway obstruction on examination or a clear cause on plain film radiograph. NT-proBNP if appropriate can also be considered.

The VBG will give a good indication as to the level of type 2 respiratory failure. If presenting with acidosis (pH <7.35) or hypercarbia (compared with baseline or >60mmHg), an ABG may be performed. If oxygen saturations are >88%, and no type 2 respiratory failure on VBG, an arterial sample is unlikely to provide useful additional acute clinical information.

## MANAGEMENT

See Figure 1 for a template of a management proforma. Patients presenting in extremis should be managed in a high acuity setting such as resus.

Important points to consider:

- Those likely to need NIV will require continuous ECG, oxygen saturations and NIBP monitoring.
- Treatment with bronchodilators and supplemental oxygen can be started immediately prior to full history and physical examination. Careful note should be taken of pre-hospital medications administered.
- Supplemental oxygen should be administered to maintain oxygen saturations between 88-92% to prevent hypercapnia and profound hypoxaemia.

Initial management is centred around early administration of **$\beta_2$-agonist treatment**, with or without short-acting anticholinergics.

- Metered-dose inhalers (MDI) can be used for patients with milder exacerbations.
- Patients' long-acting regular inhalers should be continued during the exacerbation period.
- in the setting of an acute presentation of breathlessness where inspiratory capacity is compromised, nebulisation may be the preferred method of administration.

*Figure 1. Management Proforma for AECOPD*

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# ACUTE EXACERBATION OF COPD PATHWAY

## INITIAL INVESTIGATIONS

**Bloods:** FBC, U&E, LFT, CRP, VBG
**Cultures:** Blood cultures if febrile, sputum cultures if expectorating
**Other:** CXR, ECG

Consider ABG ***only if*** severe symptoms **AND** respiratory acidosis
Administer local anaesthetic prior to performing ABG to reduce patient discomfort

## MANAGEMENT

**Oxygen** — Target $SpO_2$ of 88-92%

**Bronchodilators** — If no oxygen requirement, consider MDI with a spacer

| Drug | Inhaled Dose | Nebulised dose |
|---|---|---|
| Salbutamol | 12 puffs (1 dose) every 20 mins x3 doses | 5mg every 20 mins x3 doses |
| Ipratropium (if not given pre-hospital) | 8 puffs (1 dose) every 20 mins x3 doses | 500 micrograms Single dose |

**Steroids**
Prednisolone 40mg PO
OR
Hydrocortisone 100mg IV (***only if*** unable to tolerate PO)

**Antibiotics** — ***Only if*** clinical / radiological / laboratory evidence of infection

**NIV**
To be considered if:
Acute hypercapnic respiratory failure (pH <7.35, $PaCO_2$ >6.0 kPa)
OR
Hypoxic respiratory failure (e.g. cardiogenic pulmonary failure)

## ADMISSION REQUIRED IF ANY OF THE FOLLOWING

- Inability to cope at home
- Severe breathlessness
- Poor / deteriorating condition
- Poor level of activity / confined to bed
- Worsening peripheral oedema
- Cyanosis
- Lack of social support
- Acute confusion
- Rapid rate of onset
- Significant comorbidities
- $SaO_2$ <90%
- Impaired level of consciousness
- On long-term oxygen therapy
- New changes on CXR
- Use of NIV on this presentation
- Arterial $PaO_2$ <7kPa

## DISCHARGE CHECKLIST

Referral to respiratory CNS if available
Prescription to complete five day course of steroids (prednisolone 40mg PO daily x4 days)
Prescription for antibiotics (if indicated)
GP letter completed and provided / sent
Inhaler technique checked
Smoking cessation discussed with patient

Designed by B. MacCarthy, O. Kelly & T. Moriarty, May 2024. Version 1.3 Review date: May 2025
Based on guidelines from www.goldcopd.org and www.nice.org.uk

### Systemic corticosteroids

Systemic corticosteroids improve lung function, reduce risk of early relapse, and reduce hospital length of stay. Duration of treatment is recommended for 5 days. Oral steroids should be used preferentially over the intravenous route unless patient is unable to swallow.

### Antibiotics

Antibiotics confer similar benefits. As the patient may be suffering from a non-infective exacerbation of COPD, antibiotics should not be given indiscriminately to all AECOPD patients, but rather according to clinical likelihood of concomitant bacterial infection. Their indications include the presence of all 3: increased dyspnoea; increased sputum production and increased sputum purulence. As with steroids, their treatment time should also be for 5 days unless exceptional circumstances.[17] The choice of antimicrobial agent should be selected based on local sensitivities, therefore local guidelines should be consulted.

### Non-invasive Ventilation

NIV has been shown to reduce mortality and intubation rates in patients requiring hospitalisation for AECOPD. Early blood gas analysis will guide the need for NIV. Arterial sampling is useful to determine the severity of respiratory failure if the initial venous sample is abnormal.

Indications include:

- Respiratory acidosis ($PaCO_2 \geq 6.0$ kPa and arterial pH ≤ 7.35)
- Severe dyspnoea with signs of muscle fatigue
- Persistent hypoxaemia despite maximum medical management

Contraindications include:

- Presence of a pneumothorax without intercostal drain in place
- Vomiting patient
- Loss of airway protection reflexes
- Confusion/agitation
- Recent facial or upper airway surgery or trauma
- Recent upper GI surgery
- Fixed airway obstruction

Patients with AECOPD have deficiencies in both oxygenation and ventilation and therefore most frequently require bi-level non-invasive ventilation.

Reasonable initial settings:

- **EPAP 4-5cm $H_2O$**
- **IPAP at 10cm $H_2O$**

IPAP can then be gradually increased, not to exceed 30cm $H_2O$. $FiO_2$ should be set to target oxygen saturations of 88-92%. Once commenced, further blood gas analysis, initially taken at an hour post commencement of treatment can guide future ventilatory settings. Ongoing acidosis due to $CO_2$ retention prompts increasing IPAP and hypoxemia necessitates increasing EPAP (to a maximum of 8cm $H_2O$). The rate of increase and maximum pressure of IPAP can also be limited by patients' tolerance.

Patients not responding to conventional treatment should prompt clinicians to reassess for different diagnoses, including but not limited to PE, acute cardiogenic pulmonary oedema, ACS and anaphylaxis. If diagnosis is certain and no improvement is made with NIV and maximum medical therapy, discussion with the patient in conjunction with ICU regarding intubation may be appropriate on a case-by-case basis.

If available, patients should be referred to local respiratory services for follow-up, regardless of admission or discharge. Inhaler technique should be assessed, and smoking cessation discussed with all patients, particularly if being discharged.

## SPECIAL CONSIDERATIONS

### Pneumothorax

Patients with COPD often have underlying pulmonary emphysematous changes, increasing the likelihood of pneumothoraces. All patients should be assessed for pneumothorax prior to administration of NIV to prevent the development of a worsening or tension pneumothorax. Assessment can be performed by POCUS (performed by an accredited physician) or CXR. Large bullae may mimic a pneumothorax in this patient cohort, so comparison with previous imaging or cross-sectional imaging is advised if the diagnosis is unclear. A sudden deterioration of a patient on NIV should prompt suspicion for an acute pneumothorax.

### Magnesium

IV magnesium, often used in severe asthma as a bronchodilator, does not have robust evidence for its use in COPD. A recent Cochrane review[16] noted that it may reduce hospital admissions, length of stay and result in improved dyspnoea scores, however more robust trials are needed to assess its use in this patient cohort. Caution is advised when administering to patients in need of NIV, as rapid infusion can result in hypotension, which can be confounded by the increased intrathoracic pressure and reduced venous return associated with NIV.

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